THE UNIVERSITY *of* EDINBURGH

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LIST OF ACRONYMS USED

ACS	American Cancer Society
AHA	American Heart Association
ASH	Action on Smoking and Health
ASO	Association for the Study of Obesity
BBPS	Build and Blood Pressure Study
BDA	British Diabetic Association
BMJ	British Medical Journal
BMI	body mass index
BMR	basal metabolic rate
BNF	British Nutrition Foundation
CBT	cognitive behaviour therapy
CCF	Center for Consumer Freedom
CDC	Centers for Disease Control and Prevention (of the US govt)
CPG	Coronary Prevention Group
CPS	Cancer Prevention Study (I & II)
CHD	coronary heart disease
COMA	Committee on Medical and Nutritional Aspects of Food
CVD	cardio vascular disease
DALY	disability adjusted life years
DHSS	Department of Health and Social Security (now Dept of Health)
DoH	Department of Health
FAO	Food and Agriculture Organization
FDA	Food and Drug Administration (of the US govt)
GBD	Global Burden of Disease
HDL	high-density lipoprotein
ISCSH	Independent Scientific Committee on Smoking and Health
IOTF	International Obesity Taskforce
LDL	low-density lipoprotein
LSHTM	London School of Hygiene and Tropical Medicine
MAFF	Ministry of Agricultural Food and Fisheries (now Dept of Environment Food and Rural Affairs)
MRC	Medical Research Council
MONICA	Multinational Monitoring of Trends and Determinants in Cardiovascular Disease
MRFIT	Multiple Risk Factor Intervention Trial
NAAFA	National Association for the Advancement of Fat Acceptance
NCD	non-communicable diseases
NGO	non-governmental organisation
NHANES	National Health and Nutrition Examination Survey
NHES	National Health Examination Survey
NHI	National Heart Institute
NHS	Nurses Health Study
NICE	National Institute for Health and Clinical Excellence
NIDDM	non insulin-dependent diabetes mellitus
NIH	National Institutes of Health (of the US govt)
NPHI	National Public Health Institute (of Finland)
OA	Obesity Association (of Great Britain)
OHE	Office of Health Economics

OPCS	Office of Population Censuses and Surveys
PAF	population attributable fraction
PAL	physical activity level
QUALY	quality adjusted life years
RCP	Royal College of Physicians
RMR	resting metabolic rate
SIGN	Scottish Intercollegiate Guidelines Network
SOS	Swedish Obese Subjects study
SSK	sociology of scientific knowledge
SSRI	selective serotonin re-uptake inhibitor
TMRU	Tropical Medicine Research Unit
TOPS	Take Pounds off Sensibly
TPRT	Tobacco Products Research Trust
TR	technical report
VLCD	very low calorie diet
WHO	World Health Organization
WHR	waist to hip ratio

CHAPTER 1: INTRODUCTION

1.1 Introduction

In 2000, the World Health Organisation published a technical report that labelled obesity as an epidemic and therefore, an important global health problem (WHO, 2000). This report described how average body weights were increasing globally due to a combination of inappropriate diet and sedentary lifestyles, and it outlined the negative consequences of such increases in terms of health and economic costs. It marked a significant and important shift in biomedical understandings of body weight and its relationship to health. This thesis sets out to analyse the development of this shift and its implications for modern public health policy.

This introductory chapter situates my thesis within several different existing literatures. Firstly, as a way of introducing the topic, I discuss historical accounts of post-war public health and nutrition science, and recent sociological accounts of obesity. Then I briefly outline recent sociological analyses of public health, which I will go on to show, in the body of this thesis, are directly pertinent to analysing the development of thinking on obesity as a public health problem. I go on to highlight three theoretical approaches within Science and Technology Studies that I have used in my research, and link them to writing on health policy. I describe my research methods and some of the issues that have arisen from doing document based research. Finally, I conclude with a brief outline of the following chapters.

1.2 Historical accounts of public health

This section contains an account of historical writing on post-war public health, largely focusing on development in the UK, but also including some material discussing the WHO. It then briefly outlines Thomas McKeown's concept of the epidemiological transition and presents a critique of this concept.

1.2.1 Historical accounts of post-war public health

This section discusses and reflects upon historical accounts of twentieth century British public health, in order to provide a context for my analysis of the development of obesity science. It relies on two principal sources: *Marketing Health: Smoking and the Discourse of Public Health in Britain* (Berridge, 2007a) which uses the history of government policy on smoking as a lens to examine post-war British public health; and *What Price Community Medicine?; The Philosophy, Practice and Politics of Public Health since 1919* (Lewis, 1986), an analysis of the deleterious effects of the foundation of the NHS and its re-organisation in 1974 on the discipline of public health. These two sources provide the most complete accounts of the changes in post-war British public health as a profession, an academic discipline and a wider topic of concern.

The history of post-war British public health has focused on the NHS, the majority provider of healthcare. However, while the recent history of public health in Britain is necessarily entwined with that of the NHS, public health has its own independent identity and status as a profession and academic discipline which an exclusive focus on NHS public health history risks masking. Moreover, despite the foundation of the NHS being described as 'a major public health achievement' (Baggott, 2000: 45), writing on the twentieth century history of British public health (for example Lewis, 1986, Porter, 1999, Berridge, 2007a) has largely taken place outside the study of the NHS. The topic is not covered in depth in any of the mainstream academic histories of the NHS: Rudolf Klein's (1995) history of the organisation does not explicitly discuss public health and Charles Webster's (2002) account is restricted to a description of the negative effects of the 1974 NHS restructuring on the profession. For this reason, Martin Gorsky (2008: 449) describes the history of public health as a 'subaltern narrative' within the historiography of the NHS. Berridge's (1999a) *Health and Society in Britain since 1939* is a notable exception to this pattern. In it she provides a four-stage chronology of British public health in the twentieth century that describes a shift from an early environmental focus in the 1950s; a focus on personal prevention from the 1960s onwards; decline in the professional standing of the discipline after the 1974 NHS re-organisation; and a subsequent rise in its status beginning in the late 1980s.

As I argue later on (see section 1.8.3), there is a very limited amount of available secondary material covering the history of the WHO. Moreover the existing histories (for example Siddiqi, 1995, Lee, 2009) do not explicitly frame it as a public health organisation. This is despite the fact that the WHO developed out of international public health collaborations, such as the International Sanitary Conferences and the League of Nations Health Organization (Lee, 2009: 2), and that much of the work the organisation carries out - immunisation campaigns, health promotion and outbreak monitoring - is part of mainstream public health practice. It is, therefore, not clear why it has not been considered in this light.

Accounts of the foundation and early structure of the NHS provide an important context for any history of twentieth century British public health. I will highlight three aspects of the history of the NHS that are directly relevant to the history of public health – the role of the medical profession, the dominance of hospital-based curative medicine, and recurring attempts by British governments to cut the costs of healthcare. Mainstream academic accounts of the history of the NHS (for example Berridge, 1999a , Klein, 1995, Webster, 2002) all include discussions of these three topics.

Different groups within the medical profession managed to secure varying concessions in the protracted and fraught negotiations that led up to the foundation of the organisation in 1948 - GPs resisted becoming government employees and retained their independent contractor status and hospital consultants retained the right to take on private work – but their medical expertise gave them an authority within the organisation other healthcare professions lacked (Klein, 1995: 17). In describing the period until the 1980s, Klein argues that

> The internal political history of the NHS in the 20th century was, for most of the period, the history of the relations between the government of the day and the medical profession….[It] appeared to offer a paradigm of "oligarchic elitism", reflecting the fact that it was the child of a union between technocratic paternalism and professional interest. Political ideology intruded from time to time…but for much of the time arguments about policy were a duet between the government and the medical profession (Klein, 1995: 231).

At its foundation, the NHS was a healthcare service dominated by hospital medicine (Berridge, 1999a: 29, Webster, 2002:29, 38-46). It was established by nationalising the existing hospital system and bringing it under central government control. A tripartite structure with different systems of administration for hospitals, primary care, and public health and social welfare was created (Webster, 2002). The historical unwillingness of the medical professional to accept local authority control (Klein, 1995: 70) was one important cause of this centralised system and also had a profound effect on the discipline of public health (see below). This partly reflected the power of the medical profession: 'much of the profession itself was at this stage at best indifferent, and at worst actively hostile, to preventive medicine, social medicine and public health' and combined with the administrative structure of the NHS led to the creation of a service 'which favoured central rather than local control; curative over preventive and social medicine and public health; and hospitals over health centres, and, indeed, primary care in general' (Stewart, 2008: 463). This model continued to operate throughout the 1960s and 1970s, when the Hospital Plan

> consisted of, essentially, a technocratic approach which strongly favoured curative, and consequently also hospital-based, medicine. In new, and very large, hospitals heroic surgery and newly synthesised drugs were to be the solution to individual and national health problems (Stewart, 2008: 462).

Since its establishment the NHS has operated under significant financial constraints. It cost more than had been anticipated in its first two years (Webster, 2002: 31) and, despite its relative cheapness, its perceived expense continued to be a politically contentious issue throughout the 1950s and 1960s (Webster, 2002: 32-35). Even in the 1960s, when the economy was growing and health expenditure was rising, population change and developments in medical technology meant that 'increased expenditure was barely keeping pace with changing circumstances' (Stewart, 2008: 462). Such a system of state-funded hospital-based health care, thought to be short of resources, made the financing of public health and preventive medicine politically problematic:

> And it is clear that while the early NHS did at least acknowledge the need for a preventive programme, it was taken aback by the demands on its curative services which the advent of "free" healthcare engendered. However, pursuing a social/preventive/public health agenda would have required a much more radical approach than that adopted in the post-war era…addressing health issues from a preventive standpoint involves not only political will, but also a

> recognition that its benefits may take a considerable time to come to fruition. To put it another way, it is politically more attractive to increase short-term hospital capacity than to institute a preventive programme via, say, health centres (Stewart, 2008: 469).

In a healthcare system dominated by hospital consultants and funded through public taxation, it can be much more politically expedient to promise funding for the building of hospitals, the provision of new treatments or even the reduction of waiting lists, rather than funding for preventive programmes whose effects will not be apparent for many decades.

Prevention of disease was crucial for social medicine, a branch of academic medicine which developed in the 1930s, and became very influential in British universities in the 1940s. Its concerns overlapped with those of public health in its aim to analyse the causes of disease in populations:

> The international social medicine movement before the Second World War aimed to create a new social role for medicine in order to grapple with epidemiological transition created by economic and social developments in the twentieth century. The interdisciplinary program between medicine and social science would provide medicine with the intellectual skills needed to analyze the social causes of health and illness in the same way as the alliance between medicine and the laboratory sciences had provided new insights into the chemical and physical bases of disease (Porter, 2006: 1668).

Thus, social medicine was an important body of ideas arguing for change in public health in the 1930s and 1940s: 'In Britain, the ideas of social medicine were taken up by academics in medicine and the social sciences and the ideas developed as part of the more general discussion of health planning and reconstruction during the war.' (Berridge, 2007a: 29). In 1942 John Ryle was given the chair in Social Medicine at Oxford University, and in 1948 the MRC funded the Social Medicine Research Unit which became an institutional home for the research of Richard Titmuss and Jerry Morris. Between 1942 and 1944, Titmuss and Morris had published three papers on the epidemiology of juvenile rheumatism, the history of rheumatic heart disease and the epidemiology of peptic ulcer that Ryle described as 'the first practical example of social medicine' (quoted Oakley, 1997: 90). In this research social medicine was, 'part of a wide and ambitious project …to reshape both health policy and medical

practice: the end result was to be a profound health-promoting impact on the welfare of the community at large' (*ibid.*).

However, social medicine was a poorly defined concept and competing definitions existed (Porter, 1997a: 104). Titmuss and Morris had broad interests in the relationship between ill-health and social environments, whereas Ryle's narrower understanding of social medicine saw it 'as a kind of statistical addendum to the clinical model of health and disease' (Oakley, 1997: 92). The location of social medicine also led to a narrowing of its intellectual concerns:

> Increasingly, the concept of social medicine was narrowed in order to stake a claim to academic respectability. Ryle's own work increasingly emphasized not only the links with clinical medicine and epidemiology at the expense of social science and health policy, but also the importance of the study of "social pathology" – the quantity and cause of disease – at the expense of the more radical and difficult aim of promoting health (Lewis, 1986: 39).

As Berridge explains: 'Social medicine in its Oxford variant and also in Birmingham under the leadership of Thomas McKeown increasingly came to mean medical statistics' (Berridge, 2007a: 31). This narrowing further increased the discipline's isolation:

> The isolation of social medicine was enhanced by the fact that its epistemological agenda narrowed. The study of 'population health and disease' was vastly reduced in scope from the holistic union of theory and practice in the establishment of scientific humanism as a new social ethic. As social medicine developed in Britain after the Second World War the influence of social science became limited to the empirical methods of quantitative social research (Porter, 1997a:113).

Proponents of social medicine also took care to differentiate it from the theories and practice of public health, and, consequently, public health doctors' initial enthusiasm for the approach was replaced by outright rejection (Lewis, 1986: 41-2). The academic nature of social medicine and its resulting disconnection from public health practice (Lewis, 1986: 8-9) meant that it could not be used as a theoretical bulwark for public health:

> the development of social medicine was conditioned by its location within the universities and, in the search for academic credibility, it moved further away from a concern with health policy and social science….In part because of this and in part because of their own narrowness of vision, public health practitioners did not take up the idea of social medicine. This resulted in a damaging rift between the teachers and practitioners of public health (Lewis, 1986: 37).

The longer term result of the weakness of social medicine was that few departments of social medicine were set up, and it failed to have an impact either on the medical curriculum or on medical practice (Berridge, 2007a: 30). This weakness may also have resulted from its unavoidable entanglement in wider debates about the nature of health and how it should be achieved:

> The tension between biological and social explanations which gave birth to social medicine was not new…..It could be argued that part of the failure of social medicine to survive and transform itself into the study of the social relations of health envisaged by Titmuss and others derived precisely from its enclosure within the debate about two opposing models of how health is determined (Oakley, 1997: 94).

Social medicine was an international movement (Porter, 2003: 58-62, Lee, 2009: 5-9). Its aims and ideals developed out of exchanges between Soviet doctors and public health experts, and European and American social reformers, public health professionals and academic researchers (Porter, 2003: 60). One of the first chairs of social medicine was created in 1945 at the University of Brussels for the Belgian doctor, Rene Sand, who was also active in the League of Nations Health Organization and the WHO (Lee, 2009: 8). The WHO was founded in 1948 when social medicine was an active and influential body of thought, and its first director general, Brock Chisholm had links to British social medicine (Brown et al., 2006: 64). The adoption of a broad definition of health as 'a state of complete physical, mental and social wellbeing and not merely the absence of disease or infirmity' within the WHO's constitution is evidence of the influence exerted by proponents of social medicine in the organisation's early years (Lee, 2009: 16).

In the UK, public health – both as an occupation and as a scientific discipline - was in a state of flux in the 1950s (Berridge, 2007a: 26). Public health and general practice

were both 'severely demoralized' after the foundation of the NHS (Lewis, 1999: 337). The profession of public health, in particular

> had failed to capitalize on the coming of the NHS in 1948….The pre-war public health "empire" in the local authorities had seen public health doctors running hospitals and a wide range of services. But this empire began to disintegrate post-war. Lewis has argued that MoHs bear some responsibility for this outcomes, having previously been happy to extend their activities in whatever direction offered, without a distinct version of what "public health" was about (Berridge, 2007a: 26).

MoHs (or MOsH – usages vary) stands for Medical Officers of Health. The Public Health Act of 1872 made the appointment of Medical Officers of Health by local authorities a statutory necessity in England and Wales: 'MOsH were charged with enforcing the public health acts in the communities, for inspecting food, sanitation and housing, and for publishing an annual report on their activities and the state of public health in their communities' (Lewis, 1986: 5).

In the 1950s and 1960s, MOsHs were in a difficult position as both GPs and social workers encroached on their professional autonomy. A concern with personal hygiene had led to campaigns in the 1920s and 1930s to reduce infant mortality: 'Public health justified its increased emphasis on clinic work with mothers and children as "applied physiology", a new kind of preventive clinical medicine. But this philosophy could not survive the transition to the NHS and the subsequent claim of GPs to do the work' (Lewis, 1986: 6). There had been hostility between GPs and MOsH since the 1930s: 'Independent practitioners regarded MOsH as a threat because of their position as salaried employees'(Lewis, 1986: 10). After the formation of the NHS, public health doctors shared non-hospital medical work with GPs, and in the 1950s 'GPs and public health doctors continued to vie with one another in their claim to do personal prevention work' (Lewis, 1986: 58, see also Lewis, 1999). This was one aspect of a wider problem for MOsH, as the foundation of the NHS had removed many of their responsibilities, and left them with a poorly defined administrative role that largely involved planning the delivery of a wide range of health and welfare services (see below)

> The logic on the one hand of strengthening the GP and promoting him as the focus of the preventive community health services and, on the other, of promoting social workers and social service departments as the means of developing community care, proved the downfall of the MOsH and the public health departments (Lewis, 1986: 92).

Public health in the late 1940s and 1950s consisted of vaccination against infectious diseases, such as tuberculosis and polio, and the clean air campaign which had successfully lobbied for government legislation to reduce levels of air pollution (Berridge, 1999a: 48-9). Vaccination was carried out by the public health profession, but the clean air campaign was not, and so anticipated later single issue campaigns conducted by campaigning groups rather than the public health profession (Berridge, 1999a: 49). This was part of a wider shift in public health practice (see below) that can be related both to the success of the earlier sanitarian approaches and to wider changes in causes of disease and ill-health:

> The traditional focus of the public health practitioner had been the outbreak or the epidemic: public health practitioners looked back to the great days of environmentalism and the fight against cholera and typhoid in the mid nineteenth century. But this pattern of disease and disease-related mortality began to change in the middle of the twentieth century. Between the 1840s and 1971, three-quarters of the improvements in death rates had been due to a decline in infectious disease with non-infectious disease accounting for the remaining quarter. But this pattern changed after the Second World War. The old "public health" diseases like TB or diphtheria were in decline…As the population lived longer so non-infectious causes such as heart disease, strokes and cancer grew in importance…These changes in the patterning of disease were important for the new role of public health, for they, along with the modes of explanation, prefigured the important changes in the nature and focus of public health activity in the next half-century (Berridge, 2007a: 28-9).

The British tradition in statistics was an important influence on epidemiology in the 1950s (Berridge, 2007a: 32). The MRC Statistical Research Unit at LSHTM was internationally influential in the 1940s and 1950s, and was the base for Richard Doll and Austin Bradford Hill's studies into the link between smoking and lung cancer. This research extended and refined quantitative techniques such as large population based surveys, case control and prospective studies, and marked a shift away from earlier studies focusing on social inequality or environmental influences (Berridge, 2007a: 31). The increasing use of social surveys during this period was another methodological development which also replaced explanations framed in terms of

social structural inequality with those that emphasised social behaviour (Berridge, 2007a: 32). Smoking was the first health behaviour to be studied in this fashion, and it became a template for research within chronic disease epidemiology on other risk factors, such as diet or physical activity:

> Smoking was a pioneer issue, but others followed. The connection between diet and heart disease began to be outlined in the 1950s. Exercise and fitness also started to come on to the agenda. Jerry Morris' paper of 1953, which showed through epidemiological methodology, the differential susceptibility to heart disease of sedentary bus drivers and active conductors, was symbolic of the old and the new – an occupational study which showed the importance of lifestyle factors (Berridge, 2007a: 50).

Despite its lack of institutional success, social medicine had remained influential and was important in the development of chronic disease epidemiology. Researchers who were part of its network were among the first to work on the health effects of risk factors such as smoking and physical activity (Berridge, 2007a: 30 & 50). The new focus on individual habits meshed well with the quantitative methods increasingly favoured by researchers working in this area: 'The behavioural model of lifestyle prevention became indistinguishable from social medicine as an academic discipline throughout this period as it was increasingly grounded in a methodologically individualist epidemiology as the dominant science of the etiology of chronic disease.' (Porter, 2006: 1670).

The 1974 NHS re-organisation had a detrimental effect on the already embattled public health profession. In the early 1960s, 'public health doctors were deemed to be doing primarily administrative, routine work and were paid accordingly' (Lewis, 1986: 61) according to discussions of medical salaries, and, therefore, public health doctors 'experience considerable tension in reconciling their role in local government with their professional aspirations' (Lewis, 1986: 63). Several different health-related occupational groups – sanitary inspectors, health visitors and social workers - were also asserting their professional autonomy at this time (Lewis, 1986: 65-6). By the late 1960s, public health was seen to be failing and 'the material collected by the Seebohm Committee seemed to provide more evidence of the essential weakness of public health departments and their failure to analyse or to deal effectively with the

new health problems manifested in the changing patterns of mortality and morbidity' (Lewis, 1986: 100-1). [1]

Berridge also discusses the decline of public health as a profession:

> Public health as a formal medical profession had been at a cross roads at the end of the 1960s, with the old role of the MoH in the local authorities on the point of disappearing. The new role in the reorganised NHS was yet to be established. Many in the public health profession welcomed the new changes which offered the apparently tangible advantages, to a medically based profession, of incorporation within mainstream specialist services within the hospital, and of consultant status (Berridge, 1999a: 85).

In the 1960s, local authority health and welfare departments had expanded and as these departments were often run by MOsH this 'further enhanced the authority of public-health doctors' (Webster, 2002: 124-5). However, local government re-organisation and the creation of unified personal social services departments proposed by the Seerbohm Committee in 1968 meant that MOsH would lose most of their responsibilities and the majority of their staff. These changes 'raised doubt about the survival of public health as an independent medical speciality' (Webster, 2002: 125). Three separate bodies addressed this problem and evolved a 'rescue package' that involved 'proposals for resuscitating public health as the new speciality of "community medicine"'(Webster, 2002: 125). As a result, the Faculty of Community Medicine was established in 1972 and changes in training 'emphasised the reorientation of this medical subgroup towards specialist status and towards the concept of medical administration' (Berridge, 1999a: 86)**.** The 1974 re-organisation removed public health from local authority control and, therefore, dissolved the relationship between public-health doctors and environmental health officers (Webster, 2002: 104). Integration into the NHS was described as in the interests of both public health professional and administrative efficiency:

> Proponents of community medicine used the argument that the barrier between prevention and cure was crumbling to promote the integration of public health into the NHS, with the community physician providing the information for the

[1] The Seebohm Committee was an inquiry into the future of personal social services. Its 1968 report recommended the removal of social workers from local authority public health and the setting up of separate social work departments (Berridge, 1999a: 44).

> efficient and effective administration of the service and co-operating closely with clinicians (Lewis, 1986: 11).

However, because of this, 'the speciality was born largely of administrative fiat, with its identity and future bound closely to that of the reorganized health service' (Lewis, 1986: 101). The concept of community medicine was not the product of medical advances or disciplinary strength, rather it had been developed by academics in the field of social medicine – such as Morris[2] - 'who believed that the practice of public health had to be reformed if it were to survive' (Lewis, 1986: 100). In addition, there was an important and destabilising uncertainty about the role of community physicians (Berridge, 2007a: 106, Lewis, 1986: 12): should they act as administrators ensuring efficient provision of health services or as watchdogs who analysed the health of local populations?

> Public health doctors accurately foresaw the problems inherent in the new NHS structure, in particular the possibility of tension between allegiance to their local communities and the NHS bureaucracy, and between the demands exerted by the hospital and by the community outside it (Lewis, 1986: 102).

None of the groups involved in planning the re-organisation – academics, civil servants or MOsH - had thought in depth about what community physicians would actually do, and so their workload was largely dictated by their place in the new NHS structure, meaning that predictions about their conflicting roles proved accurate (Lewis, 1986: 102). Thus the profound disillusionment of MOsH after re-organisation had several causes:

> the transition for many, especially the more senior public health doctors, who comprised the vast majority of new community physicians was far from easy; secondly, the NHS was almost immediately beset by a severe financial crisis, and community physicians found themselves at the centre of conflicts over cutbacks; thirdly, community physicians were neither understood nor respected by their clinical colleagues…the community physician inherited the low status of the public health doctor; and fourthly, community physicians experience considerable difficulty in defining both their tasks and the aim of the new speciality (Lewis, 1986: 125).

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[2] Jerry Morris was an influential critic of the work of public health departments and believed that 'public health practice should be grounded more firmly in the principles of modern epidemiology' (Lewis, 1986: 103)

The result of the re-organisation was that public health as an occupation was in a state of disarray during the 1970s (Berridge, 2007a: 161). Another NHS re-organisation took place less than 10 years later, in 1982, which removed the middle tier of NHS administration and renamed community physicians with management responsibilities district medical officers (Lewis, 1986: 126). This further upheaval led to polarisation within the profession and a fifth of public health doctors subsequently took early retirement (Berridge, 1999a: 86). Furthermore, in the 1980s, Lewis argues that 'the management role of the community physician has been eroded and with it the importance of the community physician in the eyes of the government' (Lewis, 1986: 127). According to her analysis the specialist advisor and management roles proved impossible to combine (Lewis, 1986: 142).

Webster argues that in the longer term, the attempt to reform public health was largely unsuccessful and partly responsible for the continuing decline of the profession:

> The rescue effort was not entirely successful; community medicine failed to achieve the status intended by its architects and recruitment fell off. The limited success of community medicine was reflected in the failure to reshape the health service according to the governments stated priorities – for instance, by redirecting resources into community care, preventive, and promotive medicine. Also the responsibility for public health and other functions formerly important to the MOH became fragmented with unfortunate consequences, as demonstrated in the long and disquieting run of public-health alarms during the 1980s and 1990s (Webster, 2002: 126)

The increasing prominence, from the early 1960s, of smoking as a public health issue, exemplifies a shift in the theory and practice of the discipline toward personal responsibility (see also Berridge, 1999b, Berridge, 2003b, Berridge, 2006, Berridge, 2007b, Berridge and Loughlin, 2006). The publication of the 1962 Royal College of Physicians report *Smoking and Health*,

> encapsulated a number of significant developments in science and in policy. For science, it endorsed the role of epidemiology and a change of scientific "gaze" from direct biomedically influenced causation to statistical inference. For policy, it helped initiate a post-war health policy based round the notion of individual responsibility and personally avoidance of risk, a policy which skilfully combined elements of morality with the concepts of science (Berridge, 1999a: 50).

This approach developed through the 1960s and, from the 1970s, individual lifestyle change was central to public health practice and health promotion initiatives. In the UK it was put forward as government policy in the 1976 DHSS report *Prevention and Health: Everyone's Business*. Such approaches differed sharply from both the traditional environmental of early public health, and the focus on structural causes of ill-health contained in social medicine and health inequalities research (Berridge, 1999a: 93-6):

> The new individualisation of health issues continued. "Single issues" were highlighted rather than broader concern for social context. Individual action could remedy health ills; the role of women as mothers was given special attention. These ideas were a natural reaction to a period when scientific, high-technology medicine had been centre stage. They drew on long-standing beliefs about health which stressed the value of a health regimen – diet, exercise and moderate living – and intermingled powerfully with lay beliefs about moral responsibility for health and healthy living (Berridge, 1999a: 88)

A stress on personal responsibility was combined with mass communication techniques to produce a new approach to public health, epitomised by health education and later health promotion:

> This new public health/health promotion constituency stressed population-based interventions, taxation as a public health tool, with a focus on control of advertising and on public information and mass advertising to achieve objectives (Berridge, 1999a: 89)

In the 1970s and 1980s, the media techniques of the 1960s were developed and expanded e.g. in the advertising for the Health Education Council produced by Saatchi and Saatchi (Berridge, 1999a: 90). As I discuss below (see section 1.3) diet and nutrition once again became politically controversial in the 1970s and 1980s with publication of the NACNE guidelines in 1983 and the second COMA report in 1984, and CHD prevention campaigns such as 'Look After Your Heart' (Berridge, 1999a: 90). However, smoking was the first target for such approaches and anti-smoking health education campaigns epitomised the new approach to public health:

> the 1970s was a crucial decade when a new public health style and agenda emerged …this was the decade when public health developed its twin-track emphasis on the evidence base and health services and on the lifestyle version

> of public health. Smoking was the crucial "tracer issue" for the latter, the template for other public health issues subsequently (Berridge, 2007a: 161).

This focus on lifestyle and health behaviour meant that psychology and psychological models also became important within public health (Berridge, 2007a: 109-10). Another discipline that became incorporated into public health at this time was economics. The oil crisis of 1973 meant that the costs of health care services became an increasingly important issue which led to the growth and development of the discipline of health economics (Berridge, 2007a: 109). It became 'a central investigative tool for health in the technocratic era of the 1970s'(Berridge, 2007a: 128). Berridge argues that ideas from health economics influenced arguments about anti-smoking policies, which increasingly came to be framed in economic terms, and also that research into the link between increases in taxes and cigarette prices and reductions in smoking rates 'helped turn public health in a different direction - towards high taxation policies aimed at abstention from smoking' (Berridge, 2007a: 130).

Significant institutional changes took place within British public health in this period. The late 1960s and early 1970s have been labelled as a period of 'technical public health' (Berridge, 2007a) which was one aspect of a wider technocratic approach to decision making prevalent within the NHS at this time (Klein, 1995). Expert committees had been a feature of the WHO's work since its foundation, but in the 1970s they became an increasingly important part of British public health:

> The way in which researchers related to government and the mechanisms which brought the two together became more formal and framed by government interests. The idea of rationality, that there could be a rational relationship between research and policy, was high on the agenda. This was a technocratic message…The rise of the expert committee was a feature of this period. Also characteristic of this emphasis on the bringing of expertise into policy making were the moves to develop "rational" policies on a cross-departmental basis, which are seen as characteristic of the late 1970s (Berridge, 2007a: 105).

The early 1970s re-organisation of the NHS had led to a re-assessment of the network of NHS advisory committees, and the development of new expert committees covering specific areas, such as the Advisory Committee on Alcoholism and the

Advisory Council on the Misuse of Drugs. This marked a shift in relations between government, scientific research and the medical profession, and a new way for individual clinicians and researchers to employ their technical expertise:

> Doctors were moving from influence through "outside" organizations (like the Royal College of Physicians (RCP) committee in the case of smoking), into positions of scientific influence which operated at the boundaries between government and the professions. The new expert committees were more clearly organizations which linked expertise within and outside government and which were founded on ideas of technical expertise and of the role of research (Berridge, 2007a: 134).

Other key developments in 1970s public health included the growth in activity of new style pressure groups such as Action on Smoking and Health (ASH) and the rise of state responsibility for health education (via the Health Education Council) (Berridge, 2007a: 161). These aspects were apparent for smoking and other public health concerns such as diet and heart disease and alcohol consumption. New activist groups, who campaigned on a single public health issue like smoking or diet and heart disease, began to be set up in the 1960s and their numbers increased in the 1970s. The activities of ASH exemplified this wider trend in public health and demonstrated how 'the "health pressure group" largely replaced the formal public health occupation as a source of public pressure on health issues'(Berridge, 2007a: 164)**.** Such groups largely operated on the national level using the national media:

> However, their role went further than simply conveying health education. Their 'new radical/new social movement' style in fact masked close relationships with government, both strategic and through government funding. The groups, ASH in particular, were part of an essential policy-balancing act for government. These were government funded pressure groups as well as new social movements and part of the "policy community" which composed the network of influence (*ibid.*).

Berridge argues that these institutional shifts combined to create groups of people working on public health topics, such as smoking or diet, whose membership had few connections to the existing profession of public health:

> The raft of new expert committees which came into being in the 1970s and after provided a means of cementing "communities of interest" between government and public health-medical expertise. The new health pressure

> groups, moving away from a mass membership model to a media and government-based (and funded) one, also symbolized the changing relationship between voluntarism, expertise and the state. A cadre of public health activists who had little relationship to the formal, medical public health profession in health services was emergent (Berridge, 2007a: 282).

In the 1970s, the social medicine approach became dominant once again within the WHO. Despite the early influence of social medicine, for much of the 1950s and 1960s, the work of the WHO had been dominated by technologically-driven disease eradication campaigns, such as the Malaria Elimination Programme (Siddiqi, 1995). Political developments in the 1970s, such as the emergence of the newly independent African countries and the growth of the Non-Aligned Movement, contributed to a major shift in the WHO's priorities away from 'vertical' disease control approaches (Brown et al., 2006: 66) . Instead the focus shifted to primary health care, as epitomised by the goal of 'Health For All in the Year 2000' announced at the Alma-Ata conference in 1978. The Alma Ata Declaration redefined the role of the governments towards their populations by making it 'abundantly clear' that the state was responsible for the health of its citizens, a 'redefinition of the norms and expectations of the state role with regard to health' (Kickbusch, 2000: 981) . Kickbusch argues that the WHO, therefore, can be said to have 'invented' international health policy at this time by defining both a problem and the organisation's role in providing a solution:

> Just as the World Bank institutionalized and internationalized the concern for global policy and made it an inextricable part of what development was…so did WHO for health (*ibid.*).

The Health For All agenda was criticised as idealistic and impractical shortly after Alma-Ata, and an alternative programme of selective primary health care focusing on the key techniques of growth monitoring, oral re-hydration, breastfeeding and immunisation was adopted the next year (Brown et al., 2006: 67, Lee, 2009: 75-81). Moreover, although it was designed to provide basic healthcare for all populations, irrespective of income, Health For All had most impact in the WHO Europe region as it required a well-developed health infrastructure, something lacking in most developing countries (Kickbusch, 2000: 891).

In the first half of 1980s the British public health profession was still seen as in a dire state, but, beginning in the late 1980s, its standing began to improve. Writing in the mid 1980s, Lewis was not optimistic about the future of community medicine:

> the policy documents of the 1980s – *Patients First*, which signalled the second reorganization of the health service and the Griffiths Report on the management of the NHS – have shown little awareness or appreciation of the community physician's contribution. As a result the position of community medicine is currently uncertain (Lewis, 1986: 149).

Other authors describe a crisis in community medicine that led to the setting up of a committee of inquiry chaired by the Chief Medical Officer, Donald Acheson (Baggott, 2000: 47). Baggott lists four key problems for public health at that time: confusion about the multiple roles of health doctors; a lack of consistent collaboration with the other professions working within public health; the reduced independence of public health; and the low status of and low morale within the discipline (Baggott, 2000: 87-90). This analysis echoes many of the points made by Lewis about public health in the previous two decades (see above).

However, Berridge describes signs of a revival in 'formal' public health developing from the 1980s onwards (Berridge, 2007a). This was partly due to important developments in both epidemiology and government concerns:

> The policy and scientific community changed in the late 1970s and early 1980s. Concerns about the environment re-emerged as part of public health rather than separate from it. Epidemic disease, previously consigned to the "dustbin of history", suddenly made a reappearance. New alliances emerged within the science of public health; epidemiology was no longer proof enough and gained greater legitimacy through support from biomedicine and the science of psychopharmacology. Occupational health revived as a public health matter (Berridge, 2007a: 208).

Some of these new concerns - especially those about the environment – were developed by campaigning groups outside of public health (*ibid.*). Policy discourses about passive smoking exemplified these trends as they focused on the effects of the environment on individual health and considered environmental tobacco smoke as 'an infective agent for the population at large' (Berridge, 2007a: 209).

This renewed focus on the environment and infection within public health was accompanied by a new militancy both in the UK and at the international level (*ibid*.). From the late 1960s, there had been a developing internationalism in public health illustrated by the World Conferences on Smoking and Health and the subsequent involvement of the WHO European Regional Office in the development of smoking policy: 'This type of manoeuvring at the international level and through international organizations was to become commonplace over the following decades; but in the early 1970s it was something relatively new and a way of building a consensus and an alliance for action.' (Berridge, 2007a: 163) . In the 1980s, continued collaboration with the WHO led to the development of extensive and influential international networks in smoking and other policy areas such as alcohol and drugs (Berridge, 2007a: 235). Further networks were established from 1987 onwards by the WHO Europe Office's Healthy Cities programme, an extension of its earlier work with cities, local authorities and universities. This aimed to disseminate the Health For All approach and build 'a strong lobby for public health at the local level' (Kickbusch, 2003: 385).

In the UK, the 1988 Acheson Report 'made a determined effort to upgrade the status of public health' by proposing the appointment of regional Directors of Public Health in England and the renewed publication of annual public health reports (Berridge, 1999a: 87). However, Berridge (1999a: 86) argues that this revival left many of the issues about the role of the community physician (outlined above) unresolved. Moreover, even though ideas about the nature of public health had broadened in the 1970s and 1980s as did interest in the field, the profession failed to respond to these changes:

> The public health community was much broader by the 1980s; and environmental health officers, health promotion officers and GPs all maintained that they, too, had a role in formulating health public policies. However, the official public health response remained a medicalised one and attempts to broaden the Faculty of Public Health Medicine were largely failures. Arguably primary care and the pivotal role of the GP were to an extent taking over the community role of the old public health physician.(Berridge, 1999a: 92).

As a result of the weakness of public health, the rivalry between GPs and public health doctors (see above) seemed to be over,: in early 1980s, the idea of GP-led primary care was attractive as it demonstrated 'the government's commitment to filling the vacuum left by the collapse of the public-health medicine speciality, many of the functions of which…could be designated as part of the expanded scope of the primary-care team' (Webster, 2002: 178-9).

An important development of the 1980s was the renewed attention paid to health inequalities research. The Black Report, *Inequalities in Health,* was published in 1980, and became the subject of controversy as the Conservative government initially tried to restrict the number of copies available (Berridge, 1999a: 93). However, it was subsequently re-printed and the controversy contributed to a body of research and debate which 'continued to grow and expand throughout the 1980s and early 1990s' (Berridge, 2003a: 8). This became crucial to the re-emergence of the issue as an important policy concern in the 1990s, which culminated in the 1998 Acheson enquiry into inequalities. Although the controversy around the Black Report was a particularly highly-charged episode in the relationship between government and health inequalities researchers, it did not lead to the cessation of government funding of such research. Sally Macintyre and others (Macintyre, 2003, Webster, 2003) argue that it was part of a long tradition of such research that had been conducted throughout the twentieth century, and continued throughout the 1980s and 1990s. Much of this research was done outside the public health profession (e.g. Webster, 2003: 93) – by social science researchers and campaigners - but it was sometimes used strategically by them to argue for the importance of the discipline. For example, in the 1980s, public health doctors promoted claims about the negative health effects of unemployment as a way of demonstrating their knowledge about the hidden costs of government economic policies and, thus, improving their status, particularly in respect to health economists (Bartley, 1994, Bartley, 1996).

In the 1990s, government policy began to re-focus on the health of the population with the setting of specific targets in the 1992 *Health of the Nation* White Paper (Lewis, 1999: 339), although these were criticised for omitting controversial aspects that related directly to health inequalities research (Berridge, 1999a: 94). At this time,

public health policy also began to draw upon both pharmaceutical treatments and genetic research as ways of bolstering its authority:

> As the twentieth century drew to a close, smoking epitomized the conflicting tendencies within public health and the changes in its knowledge base, the general tensions between environmentally conscious health promotion ideas and the growing influence of pharmaceutical imperatives which stressed vaccination and or drug interventions as preventive measures…The rise of the concept of addiction to nicotine as a policy fact signified the enhanced role of pharmaceutical interests, the role of treatment and of medicalized ideas; treatment became a public health strategy (Berridge, 2007a: 241).

The environment also remained an important concern, within smoking policy, illustrated by the continued focus on the effects of environmental tobacco smoke and increasing regulation of public spaces (Berridge, 2007a: 281). This expressed in a new form the ongoing tension between individual and population levels that has been central to twentieth century public health.

The 1980s and 1990s were also a period of crisis for the WHO, which was confronted with an unpopular director general, a diminishing budget, and encroachment into its area of activities from the World Bank and later public-private partnerships such as the Global Alliance for Vaccines (Lee, 2009: 99-115, Brown et al., 2006: 67-8). In the early 1990s, 'WHO began to re-fashion itself as a co-ordinator, strategic planner and leader of "global health" initiatives' (Brown et al., 2006: 69). Kickbusch (2000: 983) argues that the promotion of a new method of mapping and measuring the world's health problems - the 'global burden of disease' – was key to this process of shaping global health policy, as like the Health For All agenda it provided 'arguments and evidence for the need to act jointly in view of increased common threats and the need for common action.'

Many of the shifts that I have outlined above are still visible in British public health of the 1990s:

> By the end of the century, many of the public health tactics and concepts that were so unusual in the 1950s and 1960s were so commonplace as to be unremarkable. People expected doctors to give advice on lifestyle conditions and politicians to give advice on how the population should eat and drink'. Government itself had become a public health activist. The re-orientation of

> epidemiology post-war to concepts of risk in relation to chronic disease were part of everyday currency, although they had been novel and lacking in legitimacy in the 1950s (Berridge, 2007a: 281-2).

This historical literature provides an important context for my analysis of the development of obesity science. Histories of the NHS describe the dominance of curative, hospital medicine and the professional interests of consultants, at the expense of preventive medicine and the interest of other groups, like public health doctors who practiced medicine outside of the hospital system. The work of Lewis and Berridge documents the weakness of the British public health profession in this post-war period. This weakness meant that issues about the relationship between diet, bodyweight and health could be addressed by other groups of professionals, including the group of policy-orientated nutrition researchers and their allies, whose activities I outline in later chapters.

The 1960s and 1970s are a particularly well documented period. Berridge's account of developments in this period points to the increasing importance of expert committees, whose reports form the majority of my primary sources, in providing technical advice to governments. She, and later Kickbusch, also describes the development of international networks in areas such as smoking or diet and health, created by researchers' involvement in the work of bodies such as the WHO which are also important to my research. Furthermore, Berridge and others describe a key shift away from environmental approaches and towards personal prevention, that began in the 1960s with the focus on smoking as a public health issue and developed through the 1970s to include many other everyday activities, especially those, such as diet and exercise, that were thought to be associated with an increased risk of coronary heart disease. These accounts describe the ways in which the concept of risk factor from chronic disease epidemiology was incorporated into public health practice to produce an approach to disease prevention that relied on educating individuals to change their behaviour.

Accounts of developments in the 1980s and 1990s are less comprehensive, but they show a renewed interest in the topic of public health and a revival in the discipline, and maybe also in the profession of public health. Berridge also describes the incorporation of environmental concerns into public health discourses in the 1990s

which is reflected in obesity sciences' use of environmental explanations in this period. Finally, the writing of Webster and Macintyre shows that although there was active research into health inequalities throughout this period, it was largely conducted outside of the profession of public health. Due to its links with the issue of poverty, research into health inequalities was also often a source of contention between politicians and researchers in this field.

1.2.2 Ideas of the demographic or epidemiological transition

In chapter 2 I will describe, in broad outline, the changing patterns of health and illness that are understood to have occurred in rich, industrialised countries since the beginning of the twentieth century. In contemporary medical history and sociology these changes are well recognised and often labelled the epidemiological, demographic or simply health transition. Until the late 1990s, these terms do not appear to have been used in British epidemiological and public health writing. Earlier commentators on secular changes in population health discussed increasing rates of chronic disease and linked it to 'modern' ways of life, such as increasing consumption of fatty and sugary foods and increasing workplace mechanisation. These authors spoke of 'modern epidemics' (Morris, 1964) caused by the 'prosperous industrial society' (OHE, 1969), the 'affluent society' (Craddock, 1978) or the 'affluent lifestyle' (WHO, 1986). As Rosenberg has argued, there has long been an ongoing discourse of the negative effects of economic development on human health – what he labels 'pathologies of progress' (Rosenberg, 1998). These labels, especially ideas of diseases of affluence, draw on this long enduring strain of thought. However, these authors also convey an implicit narrative of improvements in average health status due to economic development and social progress or modernity that has been shaped by ideas of a demographic transition. Thus, there are two overlapping and partially contradictory narratives in play - of social progress as, alternatively, improving health or leading to disease - that are often present in discussions of chronic disease and obesity/overweight, but rarely acknowledged.

A conventional account that draws on the first of these narratives, stresses the role of innovations in medical practice and treatment in the late nineteenth and early twentieth century decrease in mortality, increase in life expectancy and increase in

population in the UK. From the mid 1950s Thomas McKeown, professor of social medicine at Birmingham University, began producing a series of articles and books challenging these sorts of accounts (McKeown, 1976, McKeown, 1979, McKeown and Brown, 1955). His argument has two major elements. First, he used British mortality statistics and dates of important treatment innovations to argue that death rates from the major infectious diseases – tuberculosis, typhus, typhoid, diphtheria, scarlet fever, pneumonia – were already in decline before effective medical treatments existed:

> I conclude that immunization and treatment contributed little to the reduction of deaths from infectious disease before 1953, and over the period since cause of death was first registered they were much less important than other influences' (McKeown, 1979: 77).

In the second part of his argument, McKeown uses statistics of improving food supply, and an assumption that fertility had reached its maximum, to argue that decreasing mortality and population growth in population were largely due better resistance to disease deriving from improved nutrition:

> the influences responsible for the decline of mortality were environmental, behavioural [the declining birth rate] and therapeutic. They became effective in the eighteenth, nineteenth and twentieth centuries, and their order in time was also that of their effectiveness(McKeown, 1979: 78)

Arguing that economic growth led to higher standards of living, better nutrition, and so reduced susceptibility to disease was a comprehensive rebuttal of one of the central myths of professional medicine. One historian of medicine baldly summarises McKeown's argument as 'the modern demographic revolution was agricultural, not social or medical' (Bynum, 2008: 645).

McKeown's arguments, although highly publicised (one of his books was a best seller), were iconoclastic and controversial: they did not completely replace the mainstream medical account, but they epitomise a particular faith in economic development as the key to improving population health, and thus were politically very useful (see below). It was not until the 1990s that McKeown's arguments begin to be cited and discussed in epidemiological or public health textbooks (Beaglehole et al.,

1993, Connelly and Worth, 1997). McKeown's is a specific argument from historical demography, so it is not surprising that these texts do not discuss his ideas directly, but, they are consonant with more general ideas the role of modernity or civilisation that do inform much of post-war British chronic disease epidemiology.

The historian, Simon Szreter has argued against McKeown's approach (Szreter, 2007). Szreter discusses McKeown's arguments in the context of the development of ideas of the demographic transition:

> The notion that modern economic growth has invariably been associated with a "demographic transition" of beneficent mortality and fertility declines has been one of the most important ideas in the liberal social sciences throughout the entire period since 1945, endorsed in their plans by many national governments and by all the principal global government institutions, from the U.N. to the World Bank. It has formed an important part of the justification for the general policy presumption that national economic growth is an invariably and automatically desirable goal and should be encouraged everywhere as much as possible. (Szreter, 2007: 3)

Szreter argues that McKeown's thesis supported this orthodoxy: McKeown's argument that improvements in mortality were due to improved nutrition levels brought about by increased standards of living readily translated into a policy recommendation that improvements in health derive directly from capitalist economic growth (Szreter, 2007: 5). His critique of McKeown's argument questions the simple causality at the core of the narrative of the demographic transition and re-introduces a role for collective action to combat disease, such as the work undertaken by the sanitary reformers of the nineteenth century (Szreter, 2007). Specifically, Szreter argues that McKeown's interpretation of the nineteenth century epidemiological evidence is mistaken and that evidence of an early decline in tuberculosis mortality – a key element in McKeown's argument – is not supported by the data. Moreover, Szreter proposes that any such decline would have been counteracted by a strong rise in bronchitis-type infections due to declining urban air quality.

Szreter's alternative interpretation of the 19th century British mortality statistics narrative is that they show a worsening of the health of urban populations due to increasing congestion and lack of infrastructure, which was remedied by social interventions such as municipal sanitation: 'the decline in mortality, which began

noticeably in the 1870s, was due more to the eventual successes of the politically and ideologically negotiated movement for public health than to any other positively identifiable factor' (Szreter, 2007: 125). Such public health work was pioneered by the municipal authorities of the rapidly growing new urban industrial centres of Birmingham, Manchester, Liverpool and Glasgow. It initially included provision of clean water, paved streets, refuse collection and a mains sewage system, and, at the beginning of the twentieth century, expanded to cover minimum housing quality standards, the health visitor system and regulation of the milk supply (Szreter, 2007: 125-9). Szreter argues that unchecked economic growth is profoundly disruptive for individuals and populations –'setting in train a socially and politically dangerous, destabilizing, and health-threatening set of forces' – and leads to the "four Ds" of disruption, deprivation, disease, and death (Szreter, 2007: 204). It is only collective action, as undertaken by local and then central government in Britain in the late nineteenth and early twentieth centuries, that is capable of transforming economic growth into sustained social development that improves the living standards and health of populations (Szreter, 2007: 235). This argument contradicts McKeown's conclusions, and with them the more general neo-liberal faith in economic development as the only factor necessary for improvements in health.

Szreter thus mounts a comprehensive challenge to widespread assumptions that 'modernity', equated simply to economic growth, leads to improvements in health status. And in doing so, he implicitly challenges also the idea that, accompanying this general improvement in health, we see the emergence of new 'diseases of modernity' that can be understood as unfortunate side-effects of economic growth and improved material standards of living. In the course of my thesis, I will examine the construction of an account of one particular 'disease of modernity', namely obesity. And I will show that this construction was shaped by precisely the kinds of assumptions about the beneficial and harmful consequences of economic growth that Szreter seeks to undermine. In particular, I will show that that account was constructed in the context of a developing field of 'policy science' which was in turn shaped by wider socio-economic factors including the institutional and political marginalization of environmentalist approaches to public health.

1.3 Historical accounts of nutrition science

Historians of medicine have analysed the ways in which, since its beginnings in the late nineteenth century, nutrition science has been used in the making of public policy.

Nutrition science has been described as a complex and heterogeneous field:

> "Nutrition" covers a very wide subject area, from sociology to molecular biology, and similarly, "nutrition scientist" can embrace a remarkably wide range of people. The membership of the Nutrition Society (founded in 1941) has included, for example, chemists, biochemists, physiologists, medical and veterinary practitioners, agricultural scientists, food scientists, dieticians, sociologists, psychologists and administrators, as well as "nutritionists". Nutrition is also an area in which many wider interests intersect: the implications of nutritional research potentially impinge, not only upon the practices of doctors, dieticians and veterinary practitioners, but also on the policies of central and local government, the agricultural, food and pharmaceutical industries, and the domestic habits of the population (Smith, 1997: 1).

As it overlaps with individual daily experience, '"nutrition experts" abound, and nutrition scientists face more acute problems that many other scientists in establishing their expertise' (*ibid.*). These multiple and contested forms of expertise, combined with the effects of strong commercial interests, makes nutrition a particularly valuable case through which to study the relationship between scientific expertise and public health policy.

From the beginnings of the discipline in the late nineteenth century, nutrition researchers have also attempted (often successfully) to use their expertise in the development of public policy. Naomi Aronson describes how the construction of nutrition as a social problem resulted from 'the entrepreneurial claims of an emerging scientific discipline' and demonstrates how the first generation of U.S. nutrition scientists 'recruited support for their research by linking nutrition to the labor problem, one of the most visible social issues of the time' (Aronson, 1982: 483)**.** In the late nineteenth century, American industrialisation was accompanied by labour unrest. Liberal reform movements promoted the scientific management techniques of Frederick Taylor to solve the problems of industrial capitalism.

> economists and statisticians hoped to end class conflict by developing objective criteria for the adequacy of wages. They established a base line, termed the "standard of living", for the quality of food, clothing and other items needed to live adequately…At this point the questions of political economy and nutrition research coincided: what were the nutritional requirements for human subsistence? (Aronson, 1982: 477).

The Massachusetts Bureau of Labor Statistics sponsored the first American nutritional study in 1885 and, as part of it, Wilbur O. Atwater conducted a study of the food consumption of factory workers. From this study he concluded that existing wages would be adequate if workers learnt to eat according to the scientific principles of nutrition:

> Optimal nutrition would increase productivity, thereby increasing the total social wealth to be divided between the classes. At the same time, the application of sound nutritional principles would reduce worker expenditures for food, thereby effectively increasing the buying power of existing wages…Atwater concluded that the problems of poverty and labor unrest could be solved by teaching the masses to shop and cook economically (Aronson, 1982: 478-9).

In America, from the late 1880s onwards, there was a 'broadly based nutrition research movement' including prominent individuals, philanthropic organisations and home economists who lobbied Congress for federal funding. However, as Aronson points out, the scientific claims of nutrition researchers were not providing a neutral solution to the problem of low wages but providing a rationale for prevailing wage levels. Moreover, such surveys contained a moralistic and judgmental attitude towards the eating habits of poorer families but not those of affluent families. Nutrition researchers' recommendations were often disadvantageous to poor families as they saw the consumption of oranges and green vegetables as an unnecessary luxury, and wives were advised to spend more of their time preparing and cooking family meals (Aronson, 1982: 481-2).

This definition of nutrition as a social problems was gradually dismantled in the US between 1900 and 1915 due to increased private research funding - which meant that nutrition researchers did not have to make claims of social utility to secure government funding - and by the discovery of vitamins which 'undermined the scientific basis for the nutrition researchers social program by showing that there were

essential foods that workers couldn't afford' (Aronson, 1982: 483 see also Kamminga, 2000). Although the increasingly biomedical focus of nutrition research meant that later researchers could avoid earlier social policy orientated activities which were continued instead by home economists (Aronson, 1982: 483) such entrepreneurial activities continued to be a recurring aspect of British and American nutrition science in the first half of the twentieth century.

The health problems associated with poor diet of the urban working class had also been a subject of British scientific and political discussion since the 1890s (Smith and Nicolson, 1995). There were often two contrasting explanations put forward:

> On the one hand, nutritional problems have been ascribed to economic handicaps, to a substantial proportion of the working class simply not being able to afford and adequate diet. Alternatively blame has been placed upon the perceived prevalence of educational, behavioural and moral failings among the urban poor, especially working-class mothers…those who sought to deny that insufficient income was the fundamental cause of inadequate diet have routinely advocated improvements in education as the most effective remedy for the dietary ills of the less well-off. On the other hand, those on the opposite side of the argument, who espoused various degrees of political and economic restructuring, have often chosen to dismiss the question of nutritional education as merely a distraction from more important issues (Smith and Nicolson, 1995: 290).

As an example of the second type of argument, witnesses to the Interdepartmental Committee on Physical Degeneration in 1904 'complained that working-class housewives were idle, indifferent and unduly inclined to spend their meagre resources in fish and chip shops or on tinned food' and 'stressed ignorance and degenerate habits as causes of ill-health and inferior physique among the poor much more strongly than poverty *per se*'(Smith and Nicolson, 1995: 292). Twenty years later, in the mid 1920s, the debate was still being conducted in broadly similar terms when Edward Cathcart, Professor of Chemical Physiology at Glasgow University argued that inadequate diet was caused by 'bad buying and bad cooking' (Smith and Nicolson, 1995: 297). Such arguments, closely related to those of Wilbur Atwater (see above), were made regularly throughout the 1930s in British debates on diet and poverty. John Boyd Orr, who published *Food Health and Income* in 1936, was one of the few high-profile and influential writers to make the opposite argument that the poor diet of the working classes was due to lack of money rather than ignorance:

> His unequivocal conclusion was that it was income, or the lack of income, which was overwhelmingly the key factor in determining whether or not working-class people obtained a healthy diet. The income of the poorer sections of the population was simply not sufficient to enable them to purchase adequate quantities of nourishing food (Smith and Nicolson, 1995: 300).

Debates about the nutritional status of the poor during the 1930s were complicated by the recent discovery of the necessity of vitamins and other micronutrients for health – often labelled the 'newer knowledge of nutrition'. Medical Research Council (MRC) research into 'protective foods' became a source of potential public controversy in a period of economic recession:

> The practical application of many of the principles of the "newer knowledge of nutrition" in the 1930s was controversial because, at a time of mass unemployment, low-wage labour and fiscal retrenchment, government ministers and senior civil servants were desperately concerned to disprove links between malnutrition, ill-health and low income. Financial considerations determined economic and social policies, which in turn were promoted as being compatible with the new science. Optimism about the nation's improving health characterized official statements in the press and parliament, despite private unease at the Ministry of Health (Mayhew, 1988: 462).

Research showing the importance of expensive foods such as milk and vegetables was, therefore, difficult politically, but also scientifically, since 'complete scientific and medical consensus as to the practical importance of vitamins and minerals in human diets had not been achieved' (Smith, 1995: 280). Despite this uncertainty, vitamin researchers in the 1930s participated in debates about nutritional policy and the links between poverty and ill health through deficient diet, and this dissemination and popularisation had the effect of 'hardening' vitamins as they became incorporated into diverse bodies of knowledge (Kamminga, 2000). The food industry also popularised knowledge about vitamins as companies began to develop and market a range of fortified foods, such as infant formula and margarine (Horrocks, 1995, Horrocks, 1997). The 'newer knowledge of nutrition' was thus very rapidly mobilised by a wide range of groups other than nutrition researchers.

In the case of dietary standards and other forms of nutritional guidelines, the committee room is an important site of scientific knowledge-generation (Smith, 1995:

280 see also Berridge, 2005). The claims produced such contexts can be addressed to several audiences simultaneously and perform several different functions:

> scientists who meet in committees to establish dietary standards may address themselves towards both the "research community" and nonspecialist audiences at the same time. The knowledge generated may be subsequently used, not only in policy-making, but also in future research….knowledge-construction and knowledge-deployment are not always easily separated. (Smith, 1995: 281).

Smith and other authors have analysed the workings of several British nutrition committees between the 1930s and the 1950s (Smith, 1995, Bufton et al., 2003, Smith and Bufton, 2004), including the two 1934 Joint Conferences involving the Ministry of Health's Advisory Committee on Nutrition and the Nutrition Committee of the British Medical Association (BMA) (Smith, 1995). In 1933 there had been controversy arising from the publication of the BMA's calorie and protein recommendations which were more generous than those of the Ministry of Health. The 1934 joint conferences were a 'damage-limitation exercise' resulting from this conflict between nutrition experts, and the new recommendations for calorie and protein requirements were minor modifications of generally accepted data that 'protected the public image and credibility of the parties involved' (Smith, 1995: 299). This example shows both the recurring possibility of public controversy when expert committees set nutritional limits (see below) but also the fact that conflict between groups of expert can often be resolved by minor reformulations of existing knowledge rather than wholesale revision of technical standards.

By the 1930s international organisations had also started to develop expertise in nutrition research. Throughout the 1920s, bodies such as the League of Nations Health Organisation (LNHO) and the International Labour Office, had moved from famine relief work to supporting nutrition research programmes (Weindling, 1995). Although research was being conducted in several European countries and nutrition had obvious relevance to the LNHO's social understanding of health: 'it was not easy to funnel these into a tangible international scheme without infringing on perceived national perogatives' (Borowy, 2009: 381) But for Ludwik Rajcman, the director of the LNHO:

> Nutrition was to become a prime example for establishing scientifically based policies that would transcend the sectional interests of member governments while disseminating scientifically innovative food policies to a wider world (Weindling, 1995: 321).

From this perspective, nutrition research was able to establish the dietary requirements compatible with 'optimum health' and this technical knowledge could be used to advise governments. Rajcman considered convening an expert committee in 1927, but it was not until after the appointment of a young nutrition researcher, Wallace R Ackroyd, in 1931 that regular meetings began to take place. These meetings discussed technical topics such the uses of statistical surveys and methods for assessing malnutrition, (Borowy, 2009: 381-5). After 1933, 'nutrition grew into one of the largest international health study projects of the 1930s.... producing an impressive number of publications' (Borowy, 2009: 386). Economic crises made such research an urgent political priority. In an approach that parallels modern appeals to establish global food security, the use of the technical knowledge of nutrition science was seen by those involved in the LNHO as a way of preventing another world war by mitigating the effects of widespread hardship resulting from worldwide recession (Weindling, 1995: 325). However despite these activities, which included the publication of a best selling report in 1937, the LNHO's nutrition activities were ultimately unsuccessful in influencing national policies. This was partly due to the unwillingness of governments to follow the organisation's recommendations and enact legislation aimed at minimising social inequalities (Borowy, 2009: 391-2).

In the same period, friction developed between the British Ministry of Health and nutrition experts in the MRC (Mayhew, 1988, Smith, 1995). British nutrition researchers, such as Edward Mellanby and John Boyd Orr, gained legitimacy in this institutional conflict by using the new standards and perspectives developed by the LNHO, whilst also being heavily involved in their creation:

> The MRC, in its quest not only to gain prestige and influence by fostering high quality nutrition research but also to place social policy and lifestyles on scientifically rational foundations, began to exercise a decisive influence in determining the direction of the LNHO's nutrition programme (Weindling, 1995: 322).

This echoes the way in which later British researchers gained legitimacy from the standards developed by WHO expert committees whilst being active members of these committees (see chapters 4, 7 and 8)

The BMA produced another important report on nutrition in the late 1940s, as a response to controversy about 'the adequacy of the nation's food supply generally and more particularly to what extent people were consuming enough calories for growth and work' (Bufton, 2005: 127). As part of the controversy government departments had been accused of not releasing the results of anthropometric surveys and food expenditure data (Smith and Bufton, 2004). The BMA Nutrition Committee[3] was, therefore, convened to produce a report on the health effects of food rationing but by the time the report was released the controversy had begun to abate, and the report was considered a success because it did not lead to further negative press coverage. Despite their official titles, 'observers' from the Ministries of Health and of Food actively participated in the production of this report:

> the government officials who served on the committee, ostensibly as observers, played important roles in providing and writing information and in writing the report. The interaction between officials and outsiders that took place and the release of data that was involved formed part of the process of transition from the aftermath of war to routine peacetime government and contributed to a modus operandi for nutrition science (Smith and Bufton, 2004: 244).

These officials ensured that a 'nominally independent' report 'largely reflected official agendas' and 'contributed to a consensus, not only among but also beyond scientists and doctors that the wartime and early postwar diet, although boring, had been reasonably adequate' (Smith and Bufton, 2004: 271). Smith and Bufton argue that the processes of 'political disengagement' that took place within British nutrition science in the late 1940s established a precedent for many subsequent report of the topic of nutrition and health, and this success may be an important reason why, despite its lack of official status, the BMA nutrition committee 'guided policy for nearly twenty years' (Bufton, 2005: 126).

[3] Members of this committee included Jack Drummond (Director of Research at Boots), VH Mottram (former Professor of Physiology at King's College of Household Science), Dr Harriette Chick (of the Lister Institute) and Dr John Yudkin (Mottram's successor at King's College)

In 1949, before the report could be finalised, there was a new controversy about protein requirements in the Committee that mirrors the 1934 controversy outlined above (Bufton et al., 2003). This controversy was difficult to manage because it involved a large element of scientific uncertainty but was also obviously closely connected to the controversial issue of meat rationing:

> The problem of the level of protein requirement facing the BMA committee in 1949, it might be suggested, falls into the category of "trans-scientific" questions…According to Weinberg, such questions are those that that can be asked, but cannot be answered, by science. He pointed out that in debates about trans-scientific questions, credibility becomes as important as scientific competence… None of our scientific actors was really sure what to recommend about protein requirements. None was specially competent to settle the question, and the dispute and its settlement became concerned largely with the credibility of alternative formulations (Bufton et al., 2003: 488).

Weinberg's account is plausible, especially when combined with Jasanoff's argument that British policy participants are often chosen for their personal credibility, not just their technical expertise (see section 1.6.2 below).The protein controversy was fuelled by the political inclinations of individual committee members, but these were also tempered by their professional ambitions and knowledge of the potential damaging effects of a public controversy (Bufton et al., 2003: 491-2). As in 1934, consensus was eventually reached in order to preserve the credibility the report and the committee producing it.

In the interwar period, malnutrition amongst colonial populations had become recognised as an important problem for the British government (Worboys, 1988). Worboys argues that it was the use of the established technique of the nutrition survey in a new context that led to this 'discovery':

> The "discovery" of colonial malnutrition was a result of the direct transfer of the "dietary survey" from the centre to the periphery. The important point was that the transfer was direct and did not involve the creation of an exceptionalist, tropical nutrition science. Rooted in the basic sciences of physiology and biochemistry, nutritional science allowed a common approach to problems worldwide (Worboys, 1988: 222).

Despite this reliance on biomedical research data to establish the existence of a problem, malnutrition became officially defined as an economic, rather than a health, concern: 'when the British government officially took up the matter of nutrition in the colonies it defined the issue structurally, as one of agricultural and economic development, rather than of medical services and public health' (Worboys, 1988: 216-7). This framing resulted in part from the government's priorities in the context of a worldwide recession, but it may also reflect the ambiguous status of nutrition science itself.

> The claims made [about malnutrition] signalled the impending emergence of nutrition as a discipline in its own right and showed that a number of people were thinking about new professional identities for nutrition within public health. However, the professional identity and allegiance of "nutritionists" was by no means clear for it was maintained that the subject was "an economic, agricultural, industrial and commercial problem, as well as a problem of physiology". At an intellectual level nutrition was clearly interdisciplinary in character, embracing these and other fields. But at a practical and political level, it was intricately bound up with economic concerns and political interests that the idea of disinterested professional expertise was always questionable (Worboys, 1988: 213-4).

This intellectual and practical intertwining of the scientific and the economic within the discipline of nutrition allowed the emergence of two distinctive understandings of the problem of malnourished colonial populations to emerge. This split between economic and health framings of under- and mal-nutrition is also illustrated by the earlier interventions made by nutritionists, such as Atwater, into debates on wages levels.

The research outlined above also shows that setting recommended levels of protein consumption had involved nutrition researchers in controversies since the end of the nineteenth century. Sufficient protein, the most expensive and desirable macronutrient, was seen as an important benchmark of an adequate diet. From the 1950s onwards, as British researchers started to study malnutrition in more detail, protein deficiency became framed as an urgent global threat. This framing was largely due to the work of the Protein Advisory Group, a network of nutrition researchers who became influential in three UN agencies: the Food and Agriculture Organisation (FAO), the World Health Organisation (WHO) and the United Nations Children's

Fund (Unicef) (Ruxin, 2000: 151). The PAG was partially funded by US philanthropy and depended on an important breakthrough in nutrition research:[4]

> In 1957, research by J C Waterlow and Nevin Scrimshaw helped consolidate the protein field by showing that kwashiorkor in Africa and Latin America were indistinguishable…Fuelled by data indicating that kwashiorkor was prevalent and preventable, WHO's commitment to protein began to shape something of a priesthood of nutritionists, exercising substantial influence over policy (Ruxin, 2000: 153).

This 'priesthood' became very influential within the UN:

> PAG and its associates made remarkable progress in encouraging interest in protein among policy makers and politicians. In 1963, FAO concluded from its Third World Food Survey that since in developing countries "the level of animal protein intake is only one fifth of that of the more developed areas" world food supplies would have to rise by 50 per cent by 1975 (Ruxin, 2000: 155).

By the late 1960s the problem of how to fill "the protein gap" had become a critical issue for the PAG and associated UN bodies, and two years later a UN report written by members of PAG called for 'the last great push for protein' (Ruxin, 2000: 156). However, not all UN agencies accepted these claims. Despite the organisation's earlier support, FAO officials often resisted attempts by PAG to expand their expertise. By the early 1970s, they did not fully accept the term "protein gap" because FAO surveys showed that there was more protein in the world than was required to meet basic human needs (Ruxin, 2000: 157-8).

In 1974, concern about a global food crisis made the protein gap seem like a less important issue, and subsequent discussions focused on food production and hunger rather than specific nutritional deficiencies. Critics within nutrition science also argued against the emphasis put on protein malnutrition and kwashiorkor at the expense of other forms of malnutrition:

> McLaren published a stinging condemnation of protein policies in *The Lancet* entitled "The Great Protein Fiasco" in 1974. He argued that there was no

[4] The Josiah Macy Foundation sponsored a joint FAO/WHO conference on protein malnutrition in 1953 and the research of a sub-group of the PAG – the Committee on Protein Malnutrition – was jointly funded by the Rockefeller Foundation and Unicef (Ruxin, 2000: 153).

> "protein gap" and that PAG had been perpetuating a myth…decades of protein obsession (Ruxin, 2000: 159).

The disavowal of claims about widespread protein deficiency led to a wider mistrust of nutrition researchers:

> In the minds of many policy makers, the nutritionists had been unable to guide or create successful projects. Knowledge of the complexities of good nutritional status was increasing but there was a feeling that past results of nutrition programmes had not fulfilled expectations. On protein policy makers felt misled (*ibid.*).

Over the next two years, the PAG lost support and in 1977 it was replaced by the Advisory Group on Nutrition (AGN). Ruxin (2000: 164) argues that this organisational shift 'symbolised a fundamental shift downwards of the status of the outside nutrition expert'. This downward shift presumably affected the work of nutrition experts in WHO committees on the relationship between diet and chronic disease that began to operate from the late 1960s onwards (see section 7.2)

The work of other British expert committees in the post-war period, such as the Committee on Medical Aspects of Food Policy (COMA) and the National Advisory Committee on Nutritional Education (NACNE) has also been analysed by historians of medicine (Bufton and Berridge, 2000, Bufton, 2005). Bufton and Berridge analyse the post-war work of COMA (Bufton and Berridge, 2000). They outline how British food and nutrition policy was seen to have improved the population's health during WW2 (see above) and how after rationing ended there were significant increases in levels of sugar and fat consumption. By 1954 the government was aware of this trend and linked it to increasing rates of heart disease:

> Explanations for the rise, whether real or apparent, began to focus on diet. The explanation which was to emerge as dominant was that of dietary fat, and saturated fat in particular, was a central cause. This first emerged in the early 1950s through the work of Ancel Keys (Bufton and Berridge, 2000: 209).

In a 1952 paper in *The Lancet,* Keys linked age-related increases in heart disease in US men to increasing blood cholesterol levels, comparing them to lower levels in

Italian men, and five years later he suggested that substituting vegetable for animal fats might lower cholesterol levels:

> Keys' research was influential in an emergent international consensus. During the 1960s, a number of western governments commissioned expert committees to investigate the causes of coronary heart disease…Most advocated that individuals should lower their sugar and saturated fat intake and increase their polyunsaturated fat intake and it was a Scandinavian report which helped to trigger developments in the UK. The diet and heart disease thesis gained credibility (Bufton and Berridge, 2000: 210).

Other nutrition researchers, including John Yudkin in the UK, contested Key's claims, but 'the diet and heart disease thesis gained ground in many industrialised countries around the world with various expert committees giving support to the idea.' (Bufton, 2005: 131). In response to this increased interest in diet and nutrition, in 1957 the UK government revived the Standing Committee on Nutritional and Medical Problems, renaming it the Committee on Medical and Nutritional Aspects of Food Policy (COMA). It was chaired by the Chief Medical Officer. In 1959 COMA investigated the fat content in milk in relation to coronary heart disease (CHD) and ten years later formed a panel on diet and heart disease to investigate the issue further. The panel considered 400 articles over 4 years and produced its report in 1974:

> Its basic recommendations were that the amounts of saturated fat and sugar in the diet should be reduced. These recommendations were quite limited and basic because the panel members had found it difficult to agree. Yudkin had disagreed with the most basic of recommendations…He had argued that his colleagues had exaggerated the role of dietary fat in causing heart disease and underestimated the role of sucrose (Bufton, 2005: 131-3).

Many of the members did not accept the Report's recommendation that individuals should reduce the amount of fat they consume, and John Yudkin disagreed with this so strongly that he wrote a note of reservation, arguing that the report minimised the role of sucrose in causing heart disease (Bufton and Berridge, 2000: 212). He also gave newspaper and television interviews to outline his disagreement with the developing consensus:

> Those scientists, like Yudkin in the case of diet and heart disease, whose theories were not adopted as part of the agreed consensus, increasingly used

> the media to advance their scientific case; and the media was important, too, when committee findings were ignored by government. (Berridge, 2005b: 25)

This recourse to the media meant that journalists became key intermediaries in the science/policy process. Such a lack of consensus amongst the panel was one of the reasons why the report had minimal effects:

> The main impact of its report seems to have been on advertising. Here it was used as a reference point. If a proposed advertisement made health claims, unsupported by the conclusions of the COMA report then it was turned down (Bufton and Berridge, 2000: 212).

In the second half of the 1970s the issue of diet and heart disease again began to attract political and media attention. The RCP and British Cardiac Society produced a joint report on the topic in 1976. This was not an official report but as it was sent to all doctors, it 'did in all probability influence the dietary advice some doctors gave to their patients' (Bufton, 2005: 133). It also contained advice to substitute polyunsaturated fats for some saturated fat that contradicted the COMA report's recommendations. The Coronary Prevention Group was also formed in 1976 and began to 'campaign for greater public awareness of coronary heart disease and policy initiatives from government to reduce its incidence' (Bufton, 2005: 134).[5] Such initiatives led to the establishment of a second COMA panel on diet and health. By the early 1980s there were two expert committees operating in this area: the second COMA panel and the National Advisory Committee on Nutritional Education (NACNE) which had been established in 1979. NACNE has been described as 'quasi-official' (Bufton, 2005: 134) whereas COMA was an 'insider' body whose approach has been contrasted with NACNE's more 'policy-activist' orientation (Berridge, 2005b: 25). A few individuals, such as Philip James and JL Mann, were members of both committees and the CPG.

The 1983 NACNE report recommended significant dietary change, notably that individuals should reduce their average total fat intake and their consumption of sugar, salt and alcohol. This received a hostile reaction from the DHSS and food industry groups (Cannon, 1987) and through the British Nutritional Foundation (BNF)

[5] Berridge (2005) argues that the Coronary Prevention Group was explicitly modelled on ASH (see section 3.2).

the food industry tried to block its publication (Bufton, 2005: 134). Government ministers were also accused in the medical and popular press of obstructing the report's production and restricting its circulation (Bufton and Berridge, 2000: 214). A year later, COMA set up an expert panel to update the findings of its 1974 report, and Philip James, who was chairman of the NACNE report committee, argued that this was to provide a mechanism for the government to distance itself from the NACNE findings, and that 'the COMA panel felt under pressure to produce a report acceptable to the government' (*ibid.*). COMA took four years to produce this report which included the review of 600 scientific articles and preparation of 40 working papers and like the other committees, this panel also had difficulty in reaching consensus. Bufton (2005: 135) describes its recommendations to individuals as 'more circumscribed than those of NACNE' and they focused on improved food labelling in the hope that consumers would purchase lower fat foods. There was controversy around the advice to reduce consumption of saturated fats as one committee member, JRA Mitchell, thought this was unsupported by the available evidence: as active members of the CPG, Philip James[6] and JL Mann disagreed. The viewpoints these expert committee members might have reflected their disciplinary backgrounds so the epidemiologists were more in favour of the diet-heart disease hypothesis than cardiologists and physicians as Bufton (2005: 139) argues. However, this does not explain the energy and activism of James (a nutrition researcher) and Mann (a physiology researcher).

Despite the fact that the 1984 recommendations were similar to those of NACNE, the COMA report was released without a significant controversy. According to Bufton and Berridge, there were a number of reasons for this change: the chairman and committee producing the COMA report wanted it to make an impact; the earlier highly publicised controversy around the NACNE report had rehearsed many of the arguments and alerted the press to the possibility of government hostility; and its recommendations also fitted well with a new emphasis on prevention within primary care. The report was also congruent with a new approach in public health:

[6] 'James was considered by one COMA panel member to be fanatical about the diet-heart disease thesis, and one former government minister thought him a maverick.' (Bufton, 2005: 135)

> The diet and heart disease consensus was also part of a wider public health consensus established in these years round the role of individual lifestyle and the need for population-based policy interventions. Policy based on such an approach rather than government attempts to regulate food consumption more directly, is also likely to be less threatening for the food industry and may even create new opportunities (Bufton and Berridge, 2000: 216).

In this period members of the COMA panel regularly appeared in television programmes discussing the relationship between diet and heart disease giving the debate a high public profile.

> The scientists on COMA panels during the 1980s frequently appeared on television arguing for their interpretation of the facts or contesting the science. Some, such as W.P.T James and G. Rose were also involved in voluntary pressure groups such as the CPG. James, who some saw as mercurial and outspoken, was prominent in putting forward his views. This contrasts with some other policy arenas where those who were outspoken, used the media and held dissident views did not get access to expert medical committees and were effectively excluded from the corridors of expert power (Bufton, 2005: 139).

Despite this level of activity (and activism) it is unclear what effect researchers' campaigning efforts had in this area: 'Scientists became activists, but it is difficult to gauge if any of this influenced national policy formation.' (Bufton, 2005: 140).

The next COMA report in 1994 made similar recommendations without dissenting experts or a widely publicised disavowal by the Department of Health: 'the report was more detailed and closely argued than the previous reports. However, unlike earlier panel reports, there seems to have been more consensus around the recommendations and less dissent about dietary factors and their relationship to heart disease.' (Bufton, 2005: 138). Another mark of increasing scientific consensus in this area was that by 1994 four voluntary groups in the area of diet and health - the British Cardiac Society, British Heart Association, British Hypertension Society and British Diabetic Association - who had previous been unable to agree on the content of dietary advice for the public, had developed a common set of dietary recommendations (Bufton and Berridge, 2000: 217). Bufton and Berridge argue that differing responses to these reports were 'not just a matter of "delay" in responding to scientific "truth"'(*ibid.*) but rather the outcome of complex processes involving the workings of expert committees, including the influence of individual members, reporting in the popular

press and long-term changes in public health policy. The process of producing these reports had also become professionalized – it was more complex and time consuming, more transparent and individuals were now paid for their work, and therefore had more to lose by becoming embroiled in protracted controversies. However, the consensus in this area was still a partial one:

> Researchers are still publishing strongly argued papers and books questioning the strength of the diet and heart disease thesis. Indeed one recent article reviewed Yudkin's theory in new form. This would seem to suggest that unanimity on the issues is still not complete. (Bufton, 2005: 138)

The controversy over the nutritional value of high protein diets, and the Atkins diet in particular, suggests that this consensus can be challenged by strong scientific or commercial interests. Nutrition researchers were engaged in the making of public health policy from the very beginnings of the discipline and this engagement resulted in important controversies. Some of these, like the one over appropriate levels of protein in the diet, recur regularly throughout the post-war period. During this period a consensus developed about the relationship between consumption of high levels of dietary fat and an increased risk of heart disease, but it took a great deal of work to achieve (Garrety, 1997). Perhaps, this investment of time and effort was one of the main reasons why, despite recurring criticism, the diet-heart disease consensus has remained largely intact within academic medicine and public health practice from the 1970s to the present day.

This body of research demonstrates some of the recurring and difficult features of incorporating nutrition science into British public health policymaking. Firstly, there was very little consensus amongst experts in this field – controversies regularly broke out in crucial areas such as protein requirements or the role of fat in causing heart disease. Secondly, because of widespread public interest in such debates, individual researchers with unorthodox points of view often used the media to gain more credibility and enhance their ability to negotiate in private. Finally, as Worboys points out (Worboys, 1988) there were frequently two distinct models of undernutrition circulating within these debates: an economic model which saw it as an issue of industrial development and a health model which saw it as an issue of medical provision and public health.

1.4 Social science accounts of obesity

This section describes a collection of recent social science writing about obesity that has informed my work. Just as medical and health policy discourses have increasingly focused on obesity and overweight in the last ten years, a social science commentary has developed that analyses the biomedical claims made about obesity and the implications of such claims, both for public policy and wider society. Between the 1970s and the mid 1990s, the main non-medical accounts of social understandings of excess body weight came from feminists and historians of eating or the body. The subsequent development of social science writing on the topic has led to a growing literature that analyses the media coverage of the 'obesity epidemic' and discusses the extent and implications of medical accounts of large body size. It has also generated a new sub-discipline called critical obesity studies or critical weight studies[7] that rejects many of the claims made by biomedical discourses about the extent of excess body weight and its negative effect on health.

As I mentioned above, feminist authors have been writing about eating disorders, body weight and body image since the 1970s (Orbach, 1982, Chernin, 1981, Wolf, 1990, Bordo, 1993), analysing obesity as the result of disordered eating deriving from social pressure for women to maintain an acceptable body size and shape. Susie Orbach, a practicing psychotherapist, sees obesity as an unconscious rebellion against such pressures and a way of maintaining an alternative identity out-with the narrow range prescribed within orthodox femininity (Orbach, 1982: 18-21). Naomi Wolf argues that eating disorders are caused by dieting and that the ideal of thinness is a political reaction to women's increasing autonomy and political power (Wolf, 1990: 196-7). Susan Bordo sees eating disorders as a psychological consequence of the contradictions of modern societies: 'the coexistence of anorexia and obesity reveals the instability of the contemporary personality, the difficulty of finding homeostasis between the producer and the consumer sides of the self'(Bordo, 1993: 201). Jeanine C Cogan (1999) echoes this earlier body of writing. She calls for a paradigm shift

[7] Some authors use the term 'critical weight studies' to describe their approach because 'obesity' is a medicalised term that is rejected by some activists, 'fat' is offensive to some, and their work is concerned with many types of bodies (Monaghan et al., 2010).

away from 'the weight-centred approach toward health', contending that current cultural meanings of obesity and eating disorders are based on a selective focus on key pieces of scientific research that ignore the potential health benefits of moderate obesity and the long-term failure rates of diets. Cogan argues that a rejection of the current 'thinness ideology' is necessary to 'prevent another generation of children, women and men from developing eating problems, loathing their bodies, engaging in risky weight loss strategies, and dying to be thin' (Cogan, 1999: 202).

Related to and overlapping with feminist accounts, there is also a much smaller body of writing that comes out of the fat oppression and size acceptance movements. Explicitly modelling itself on other rights-based movements, it describes and argues against the discrimination against people of large body weight, and challenges medical orthodoxy on the health consequences of obesity (Schoenfielder and Wieser, 1983). Sociologists such as Sobal (1999b) have also examined the significant stigmatisation experienced by those considered to be overweight or obese.

The most descriptive accounts in this area are historical or geographical surveys of the phenomenon of obesity, overweight and large body size which describe changing conceptions of body size in different countries and historical periods (Schwartz, 1986, De Gaurine, 1993, Sobal, 1999a, Stearns, 2002).[8] These authors describe social contexts where large body size is valued as a sign of health and prosperity, and the development of contemporary norms of slenderness that largely pre-date the establishment of contemporary obesity science.

After 2000, the existence of an 'obesity epidemic' was widely reported in the British and American popular press. The topic was used in many news stories and became the subject of much commentary. Media studies researchers, particularly those in America, have analysed this reporting. In one of the earliest analyses of the media coverage of the obesity epidemic in the US, Lawrence (2004) argues that a vigorous framing contest took place between 1985 and 1996 between arguments emphasising

[8] Jeffrey Sobal is a sociologist who has been writing about obesity, weight and nutrition since the late 1980s, and has co-authored articles with medical experts such as Albert Stunkard, a psychiatry professor at the University of Pennsylvania, and Kelly Brownell, a professor of epidemiology and public health at Yale University (see section 5.4).

personal responsibility and those emphasising the 'social environment' which, for her, results from corporate and public policy. As reflected in news discourse

> popular understanding of the causes of obesity has moved from the individualized and medical realms of biology and personal behaviour toward the realm of environmental causation (Lawrence, 2004: 69).

I would argue that the shift that she identifies derives largely from the sort of policy documents that I analyse in the following chapters. Some of them, in particular the WHO technical report on obesity as global epidemic, were extensively cited in both the popular and medical press (Fletcher, 2007). In contrast Samantha Kwan's (2009b) interview-based research suggests that ideas of individual responsibility and moral models of fatness remain dominant, despite alternative accounts that stress the role of a 'toxic food environment' (Brownell and Horgen, 2004). However, the difference between these two sets of results is probably due to their differing methods.

Abigail Saguy and Kevin Riley (2005) also analyse claims made in the media distinguishing between four different groups – antiobesity researchers, antiobesity activists, fat acceptance researchers and fat acceptance activists – and describing how each framed the causes of obesity differently. For Saguy and Riley, struggles over framing and morality are actually conducted through arguments about scientific method and facts:

> in the case of obesity, debates over the nature of the condition have largely hinged upon underlying moral assumptions about fat individuals and their behaviours. To date, *medical* arguments about the health risks of obesity have been effectively used to stymie *political* arguments about rights for fat individuals (Saguy and Riley, 2005: 871, emphasis authors' own).

Natalie Boero (2007) argues that there has been a 'remarkable' increase in scientific research on obesity and its treatment since 1990 which has led to new forms of medicalisation where 'common knowledge' about the causes of excess body weight has been co-opted by the medical profession (Boero, 2007: 53). According to Boero,

> The obesity epidemic is not a traditional epidemic of contagion and mass death. Rather it is what I call a "post-modern epidemic", one in which unevenly medicalized phenomena lacking a clear pathological basis get cast in

> the language and moral panic of more "traditional" epidemics (Boero, 2007: 41).

The concept of moral panic (Cohen, 2002 [1972]), is one that recurs in critical analyses of obesity epidemic discourses. For example, Paul Campos and his co-authors rebut claims that obesity is epidemic, that obesity is contributing significantly to increased mortality rates, or that long-term weight loss is medically beneficial (Campos et al., 2006). For them and other authors working in the field of critical obesity studies (see below), the obesity epidemic is a moral panic rather than a genuine public health problem. Such media analyses point to the moral and political assumptions embedded in public health discourses about weight.

Several recent accounts use ideas of medicalisation (Conrad, 1992) to focus on the implications of defining obesity as a disease, rather than an alternative body size or a risk factor for other diseases. Annemarie Jutel (2006) argues that 'overweight' can be distinguished from 'fatness' by the use of measurement. As a result of contemporary medicalisation, obesity is now quantified and 'Weight becomes the diagnosing tool, but…it also becomes the basis for a pseudo-disease in and of itself' (Jutel, 2006: 2270). According to Jutel, this change is due to a convergence of a belief in the neutrality and objectivity of practices involved in counting and measuring bodies, and the strength of normative ideas about modern conceptions of health. She describes this change as a process of 'creating a non-disease' and concludes that

> Overweight is not a disease anymore than slenderness is an indication of health. Like baldness it is a description of physical appearance. It may or may not be the cause of, related to, or a risk factor. It is not, on its own and outside of the context of individual risk factors, beliefs and practices, meaningful (Jutel, 2001: 2275).

Such accounts of medicalisation can be linked to the more historical approaches with which I began. Jeffery Sobal argues that the medicalisation of obesity has been taking place since the beginning of the twentieth century, and that

> fatness has moved from a moral conception of fat as badness, to the medicalization of obesity as sickness, to the demedicalization of large body size as politically acceptable (Sobal, 1995: 67).

However, in analysing the development of public health policy a more fine grained approach is necessary. For example, Chang and Christakis (2002) assess the claim that obesity has been progressively medicalised using a content analysis of entries on obesity in a bestselling American medical textbook. They argue that between 1927 and 2000 these entries show a change in 'the social appraisal of obesity'

> Obese individuals are progressively held less responsible for their condition in successive editions of the text. Initially cast as societal parasites, they are later transformed into societal victims (Chang and Christakis, 2002: 151).

They question whether this amounts to medicalisation since research has shown that both patients and doctors are sceptical of the value of medical interventions. Commenting on the work of one critic of theories of medicalisation, they argue that

> Strong (1980) found that physicians generally dislike treating alcoholics, preferring to manage problems that are more straight-forwardly 'biological', or easily susceptible to abstraction from social context, problems for which they have a clear-cut expertise in etiology, diagnosis and effective treatment. Given that obesity shares many of these features with alcoholism on the physician side, and given the emergence of resistance in the lay populace against the medical management of obesity on the patient side…one might follow Strong's reasoning on the limits of medical imperialism and hypothesise that the medicalisation of obesity is subject to important doctor and patient constraints (Chang and Christakis, 2002: 167).

Given the difficulties of treatment, the parallel between obesity and alcoholism is persuasive. Such arguments lead to the questioning of simple claims about the medicalisation of obesity, and show the necessity of further empirical research to establish in what ways obesity is being medicalised, and how these processes may be resisted by both doctors and patients.

Economic historian Avner Offer (2001, 2006) provides an alternative account of increasing average body weights in the US and UK that focuses on the role of declining self-control. He argues, against theorists such as Norbert Elias and Pierre Bourdieu, that, in the second half of the twentieth century, affluence, or economic development, is leading to decreased self-control and, therefore, increased ill-health: 'If affluence is generally associated with increasing well-being, the increase in bodyweight above normative levels presents an unwelcome paradox.' (Offer, 2001:

79). The concept of myopic choice is important to his argument that many people irrationally privilege short-term rewards over the (health, economic and psychological) advantages of delayed gratification:

> Our hypothesis is that myopic choice accounts for the reversal of the historical trend towards self-control….Affluence may be characterized as a flow of new and inexpensive rewards. If these rewards arrive faster than the disciplines of prudence can form then self-control will *decline* with affluence (Offer, 2001: 85).
>
> New rewards were thrown up by affluence faster than it took to learn to cope with the previous ones, so that overall, despite growing wealth, self-control declined. Obesity shows how abundance, through cheapness, variety, novelty and choice, could make a mockery of the rational consumer, how it enticed only in order to humiliate (Offer, 2006: 169).

Offer identifies many important trends found in the public health literature: increasing availability of food, the manufacture of a wider variety of more highly processed foods (including snack foods and fast food) and more opportunities to eat away from the home. He also identifies the ways in which a reliance on individual self-control may actually reinforce health inequalities, by arguing that the resources an individual has at their disposal affect their capacity to adapt their behaviour:

> As body weight began to rise, it stimulated an effort to recapture self-control. As in other dimensions of self-control, those with more at stake, and with more resources, have been more successful. Women, with more at stake than men, maintained lower weights; the well-off were more successful than the poor. The repertoire of reactions included food choice, exercise, eating disorders, normative defiance, and acceptance (Offer, 2001: 100)

However, his argument cannot adequately explain differential rates of weight gain among different social groups – the concept of 'mate competition' which is central to his argument cannot adequately explain why women from affluent social groups find it more possible to exercise self-control than their male partners, or other groups of women. It is also inadequate to argue that high rates of male incarceration and employment in the military leads to reduced competition for men explains higher rates of overweight/ obesity amongst African-American women (Offer, 2006: 166). This limitation arises partly because his argument is explicitly based on a mainstream economic model of individuals as rational consumers who attempt to maximise their

resources. There is little attempt in Offer's writing to understand the complex mixtures of individual and social factors that lead individuals to make 'irrational' or 'myopic' choices.

Since 2005 a group of social scientists have developed an alternative account of the obesity epidemic that they call critical obesity studies. This body of writing brings together many of the strands I have outlined above, especially a concern with processes of medicalisation, and a focus on the potential for discrimination against those who are labelled overweight or obese by the 'rhetoric' of an obesity epidemic. One author, Lucy Aphramor, an NHS dietician as well as a social scientist, questions the research linking weight loss with improvement in health, and argues that the energy balance model omits too many variables to satisfactorily explain individual weight gain. In particular, she questions whether it is ethical to recommend weight loss treatments, given a 95% failure rate (Aphramor, 2005: 319). Another sociologist, Lee Monaghan, argues that the concept of 'excess' weight 'does not correspond with epidemiologic evidence' since the association of 'overweight' with excess mortality is equivocal (Monaghan, 2005: 305). He contends that an important reason for this inadequacy is methodological inadequacy on the part of existing research that does not adequately account for the effects of physical activity on morbidity and mortality (Monaghan, 2005: 307). For Monaghan

> the highly publicized war against fat is about moral judgements and panic (manufactured fear and loathing). It is about social inequality (class, gender, generational and racial bias), political expediency and organizational and economic interests. For many everyday people, including men and boys (but more often women), it is about striving to be considered good or just plain acceptable in a body-orientated culture (Monaghan, 2005: 309).

In a more recent discussion Monaghan and his co-authors have developed the concepts of *obesity epidemic entrepreneur* (and *entrepreneurship*) in order to analyse the actors, interests and practices involved in 'constructing medicalised fatness as a social issue or crisis' (Monaghan et al., 2010: 38). According to this account, 'creators' such as epidemiologists and public health scientists draw on a wider cultural fear of fatness to 'actively define and redefine the "benchmarks" of "excess" weight, which leads to obesity being constructed as a "chronic disease" that has reached "crisis" proportions' (Monaghan et al., 2010: 47). A combination of

professional interests and 'highly lucrative connections between obesity experts, organizations like the IOTF[9] and the pharmaceutical industry' (Monaghan et al., 2010: 49) has led to the science behind the obesity epidemic being contaminated by commercial interests and moral concerns. One example of the 'fragility and morality' of this science is the downward revision of American body mass index (BMI) thresholds in 1998 by National Institutes of Health (NIH) which has been attributed by Campos (2004) to the influence of vested interests in the weight loss industry.

Others also describe how biomedical narratives dominate discussions of obesity, despite the large uncertainties in the basic scientific research. This dominance, they argue, leads to a lack of discussion about the effects of such medicalised understandings of body weight on individuals.

> This omission constitutes an 'exclusion fallacy' where what we 'choose' not to discuss…is assumed to have no bearing on the issue. In other words, the stereotyping of fat, the feelings of guilt and shame that are produced through this discourse, and the tendencies towards a culture of healthism[10] and individualism, are regarded as secondary to the primary concern to develop scientific evidence to understand the causes of and treatment for the obesity epidemic (Rich and Evans, 2005: 344).

The most significant piece of writing that has come out of critical obesity studies is a book by two physical education academics containing a very detailed critique of the concept of an obesity epidemic (Gard and Wright, 2005). These authors analyse material from the scientific publications, the mass media and popular science accounts of the obesity epidemic. In the process they assess the current state of 'obesity science' including the use of the body mass index as an index of overweight, and the epidemiological evidence linking large body size with negative health consequences (see chapter 4). Gard and Wright largely reject the arguments of obesity science, on the grounds of scientific adequacy and social equity. They argue that when scientists attempt to explain the cause of the obesity epidemic in terms of changing diets or childhood inactivity, they 'draw on often conservative and age-old ideas about social and moral decline'. They continue:

[9] IOTF stand for the International Obesity Taskforce (see sections 3.5 and 7.2).
[10] Robert Crawford (1980) defines healthism as a new form of medicalisation that has resulted from an increasing stress on preventive behaviour and the increasing popularity of alternative health treatments that see health as the result of individuals choosing to lead healthy lifestyles.

> Despite an almost complete dearth of compelling evidence, obesity scientists regularly propose wide-ranging measures to 'cure' this decline, from what children should learn at school, how parent's should manage children's lives, to the ways in which buildings should be constructed. In short there appear to be few areas of our lives which obesity scientists would leave untouched in the "war on obesity" (Gard and Wright, 2005: 15).

They use analyses from feminism and the sociology of health and illness to provide alternative approaches to the obesity epidemic, and conclude by arguing that addressing the problem of the body weight of Western populations involves an overtly moral and ideological project that would need to tackle issues such as 'economic disadvantage, the workings of capitalism, increasingly deregulated labour markets and the imperative of companies, particularly, but not only, those that sell food, to be profitable' (Gard and Wright, 2005: 190).

An earlier article by Bryn Austin also critically analyses the 'obsessive concerns with food, fat and diet' found in nutritional science and public health discourses (Austin, 1999). Austin argues that

> Contemporary nutritional public health's profound allegiance to the biomedically conceived body and naivete about the centrality of cultural meaning-making regarding eating, food, fat and gender do more harm than simply hobble efforts to promote more healthful behaviour. By failing to consider the intersection of food, bodies and diet in its cultural complexity, public health gives scientific credibility to our society's obsession with dieting and loathing of fat and is implicated in the promotion of a cultural climate that generates eating disorders (Austin, 1999: 245-6).

Austin's analysis takes him into the processes of modern epidemiological fact construction to argue that non-scientific beliefs about appropriate body weight are distorting the epidemiological analyses, such as the link that has made between increasing risk of cardiovascular disease (Austin, 1999: 255 - 8). In addition, critical obesity studies, like the earlier feminist writing, begins to address the power relations involved in public health discourses around excess body weight.

In my own work, I have not been able to consider some of these issues directly because of the nature of the documents I analyse, and I briefly want acknowledge two

important areas of omission. The first is the voices of individuals labelled as overweight and obese. They are not included in the reports or textbooks that I analyse. Secondly, feminist analyses also point to contradictory gender aspects to contemporary obesity science. Due to the predominance of male doctors and female patients, obesity science is part of a long tradition of male medical advice aimed at women.[11] More importantly, twentieth century concern about increasing rates of heart disease, out of which concern for excess body weight partially developed (see chapter 2), was framed as a problem of excess heart disease among men (Riska, 2000). It is still not usually understood as a disease that affects women, despite, for example, being the leading cause of death for women in the US and the UK (Pollock, 2010: 79-81). Women were thus being expected to practice preventive strategies initially designed to address the health problems of (middle aged) men.

1.5 Sociological accounts of the 'new public health'

This section outlines a further body of literature relevant to the construction of ideas of an obesity epidemic which provides an influential critique of the focus on prevention within modern public health.

Medical sociology has considered the post-war growth in preventive medicine largely through analysis of the activities of health education and health promotion. A related approach that is more directly relevant to my research is a critique of the 'new public health'(Lupton, 1995, Petersen and Lupton, 1996). The 'old public health' is defined as the 19th century sanitary reforms that attempted to control infectious disease, whereas 'new public health' is a late 20th century individualised approach that uses techniques such as health promotion, social marketing, diagnostic screening and community participation in the attempt to control health problems seen as deriving from 'lifestyle' factors such as diet, exercise and smoking (Lupton, 1995). Deborah Lupton describes how 'the new public health' is

> typically represented as a reaction against both the individualistic and victim-blaming approach of health education and the curative model of biomedicine. It is heralded as a return to the concern with environmental factors that first

[11] Men are seen to be more prone to obesity but less prone to overweight.

> generated the public health movement of the nineteenth century' (Lupton, 1995: 50).

Alan Petersen and Deborah Lupton draw on the work of Michel Foucault and Ulrich Beck to argue that the contemporary focus on lifestyle and the management of risk, embodied in health promotion, is 'at its core a moral enterprise that involves prescription about how we should live our lives and conduct our bodies, both individually and collectively' (Petersen and Lupton, 1996: 174). In particular they discuss the development of the idea of the 'entrepreneurial self': 'the self who is expected to live life in a prudent and calculating way, and to be ever vigilant of risks' (Petersen and Lupton, 1996: xiii), arguing that such a self is the product of neo-liberal discourses about the duties of citizenship. They conclude that, 'The new public health can be seen as but the most recent of a series of regimes of power and knowledge that are oriented to the regulation and surveillance of individual bodies and the social body as a whole' (Petersen and Lupton, 1996: 3).

Petersen and Lupton also discuss the application of epidemiological knowledge in the development of public health policies: 'Given its close alignment with policy processes, it is surprising that epidemiology as a discipline has remained generally impervious to the type of critical scrutiny to which other sciences have been treated by sociologists of science' (Petersen and Lupton, 1996: xiii – xiv). Their description of the socially constructed nature of epidemiological data is heavily indebted to theoretical approaches from STS: they are particularly interested in the role of quantification in the construction of epidemiological facts (echoing Hacking, 1990, Hacking, 2006b, Hacking, 2006c), and the relationship between epidemiology and risk discourses. Two important issues for analyses of this kind of scientific research emerge from their account. Firstly the sheer complexity of the physiological and social processes being studied makes epidemiological research very complex: because of this they argue that it is 'beset by its reliance on probabilities and post-hoc observational studies that attempt to relate health outcomes … to exposure to hypothesised "risk factors" that precede the outcome'(Petersen and Lupton, 1996: 45). Secondly, by definition epidemiological research applies to populations, yet much of the new public health attempts to apply this knowledge to changing the behaviour of individuals. People who are the target of such initiatives are well aware of this gap in

scientific knowledge and often have their own lay epidemiological explanations which may or may not overlap with 'scientific' epidemiology (Petersen and Lupton, 1996: 50-52, see also Davison et al., 1992).

One weakness from which this analysis of the 'new public health' suffers is a deficit of empirical evidence demonstrating the consequences of such changes in discourse. Nor does it explain the historical development of these changes in public health (see chapter 10). The analysis of 'the new public health' as a moral enterprise, however, is particularly relevant to the topic of obesity since moralistic tendencies have never entirely disappeared from advice about appropriate body weight (Austin, 1999, Gard and Wright, 2005). Another important aspect of this approach is its focus on changing definitions of health and disease. In preventive medicine, individuals are no longer categorised into either the sick or the healthy, instead there has been a blurring of these categories and the development of a new category of individuals who are deemed 'at risk': 'The notion of "health" therefore has become somewhat of an abstract and liminal category in epidemiology, as all people, whether or not they are experiencing symptoms, may harbour "risk factors" potentially leading to illness' (Petersen and Lupton, 1996: 48). As we will see in the course of this thesis, this aspect of the emergence of the 'new public health' is clearly evident in the case of changing definitions of obesity.

1.6 Theoretical approaches from Science and Technology Studies (STS)

The media analyses and critical obesity studies approaches that I outline in section 1.4 above analyse the ways in which scientific claims are used in the construction of social problems. In this sense they are social constructivist accounts. However a critical weakness of this body of literature is that it does not analyse 'science' and 'the social' symmetrically: scientific knowledge is either 'black boxed' so that its content is not subjected to sociological analysis or it is implied better and more objective science could exist, if all of the social contaminants were removed.

The key contribution of my thesis is to address these weaknesses in the existing literature in this field by applying STS theory as an analytic framework through which to analyse the content of policy-oriented biomedical accounts of excess body weight.

The crucial insights offered by STS are that that all knowledge is produced within, and contains the traces of, particular social contexts and that it should be analysed on this basis. In this section I will discuss four themes from STS that are important to my research: the sociology of scientific knowledge (SSK) which I have used as an umbrella approach that analyses scientific knowledge in terms of the operation of social interests; Thomas Kuhn's concept of scientific paradigms; Sheila Jasanoff's understanding of the distinctive character of policy science and Thomas Gieryn's concept of boundary-work. I will then outline two previous STS case studies of nutrition science and conclude by defining how I use the term 'obesity science' in the chapters that follow.

1.6.1 The Sociology of Scientific Knowledge, social interests and paradigms

Within STS, the strong programme in the sociology of scientific knowledge (SSK) represents a central theoretical approach to the analysis of the construction of scientific knowledge (Bloor, 1976). The central contention of SSK is that, because scientific accounts and judgments are inevitably underdetermined (i.e. it is always possible to produce more than one account of an empirical phenomenon), the role for sociological analysis is to explain why some knowledge claims – rather than others - become established as scientific 'fact' (Barnes et al., 1996). This interest in 'causality' represents one of the four basic tenets of SSK.

The other three key tenets are that, in developing sociological explanations for the development of scientific knowledge 'impartiality', 'symmetry' and 'reflexivity are vital (Bloor, 1976: 4-5). Impartiality and symmetry require that all knowledge is explained on the same terms disregarding labels such as 'true' and 'false 'or 'rational' and 'irrational'. The relativism which follows from SSK's emphasis on impartiality and symmetry is particularly important when studying processes of concept formation. It permits the development of an analysis of the construction of pieces of scientific knowledge using largely actor's categories and prevents any essentialist presuppositions about what constitutes obesity, as well as ensuring that the past is not analysed as though the present state of knowledge were inevitable. Finally, the notion

of reflexivity means that the tenets of SSK apply to sociological knowledge as well as scientific knowledge.

Canonical SSK studies utilise historical scientific controversies and offer explanations for the 'closure' of these controversies (i.e. the establishment of particular knowledge claims as objective fact) in terms of the social interests of the actors concerned. However, the use of interest-based explanations by authors such as Shapin (1982) has been criticised on the basis of the difficulties of identifying members of particular social groups and the impossibility of demonstrating causal links between actors' beliefs and actions (Sismondo, 2004: 46-7). However, when an analysis of social interests takes place mostly at the level of a 'professions's' rather than an 'individual's' interests (as in the case of my research), these criticisms have much less force. They do, however, act as a reminder to use interest-based explanations with care, rather than automatically 'reading off' an individual's motivations from their occupation.

Unlike canonical SSK studies, my research analyses the contemporary history of policy science, and the development of a scientific consensus rather than a controversy.[12] Nonetheless, for the reasons that I have outlined, it is a useful theoretical approach in the context of my research. Steve Shapin argues that

> An empirical sociology of knowledge has to do more than demonstrate the underdetermination of scientific accounts and judgements; it has to go on to show why particular accounts were produced and why particular evaluations were rendered; and it has to do this by displaying the historically contingent connections between knowledge and the concerns of various social groups in their intellectual and social settings (Shapin, 1982: 164).

My research has begun to do this for contemporary public health policy in the area of diet and nutrition. In the following chapters I will show why particular accounts of obesity were produced and what models of health they contained. I will also describe the contingent connections between scientific knowledge about obesity and the concerns of the different social groups and settings involved in its production.

[12] As I have described (see section 1.4), there is controversy about the nature of obesity and existence of an epidemic of obesity. However, this has formed a small part of my thesis.

Although interest-based explanations allow for the influence of wider social processes on the formation of scientific knowledge, the actors who are considered in most detail in such historical case studies are usually individual scientists and their peer groups. This narrow focus is problematic in the context of the present study, because, as I will go on to illustrate, scientific knowledge about obesity is constituted through several different fields of science, including biomedical laboratory research, clinical knowledge and large-scale epidemiological studies. My research thus requires me to draw upon additional theoretical approaches which broaden SSK's conceptual focus to consider how and why the relationships between these different fields of research are implicated in the construction of obesity science.

One useful method of conceptualising these links is to employ Thomas Kuhn's concept of a paradigm (Kuhn, 1970). Kuhn describes scientific research as consisting of periods of 'normal science' where activities are guided by a shared model of the phenomena being studied and the methods of study, interspersed with scientific revolutions where paradigms break down and have to be replaced. I have described the two models of obesity that I identify and analyse in the following chapters as paradigms because I think the concept captures the combination of theoretical and practical knowledge contained within these models of excess body weight. The individual and environmental models of obesity are simultaneously shared bodies of knowledge about the condition and its causes, which link to laboratory research science, and collections of standard treatments or policy solutions.

Kuhn is acknowledged to have used the term paradigm in many different senses. According to Margaret Masterman (1970: 65) these different usages can be labelled as philosophical (a set of beliefs), sociological (a universally recognised achievement), or as an artefact or a construct, (an actual textbook or piece of work). I use the term in the following chapters, largely, in her second sense of a recognised scientific achievement. Each of the models of obesity that I outline is a widely accepted body of knowledge about the condition. However, these two models are not incommensurable in the way that paradigms are sometimes understood to be (Sismondo, 2004: 17-18), and, in fact, they currently co-exist. This can occur because the causes of obesity they discuss, and the solutions they advocate, operate at different levels of social organisation, namely the individual and the population. Finally, these

paradigms develop incrementally (Barnes, 1982) – there have been no revolutions in twentieth century public health policy in the area of diet and nutrition. Instead each piece of writing, a report or a textbook, can be seen as developing the models of obesity.

1.6.2 Regulatory science and boundary-work

Another way in which the field of science that I am studying differs from canonical SSK studies is that it concerns 'policy science'. Policy science is a distinctive form of scientific knowledge in that it is explicitly oriented towards problems requiring some kinds of government solution and it is largely a product of appointed experts and expert committees. Sheila Jasanoff has written extensively on the relationship between science and policy processes (Jasanoff, 1990, Jasanoff, 2005). She explicitly rejects technocratic models of the science policy relationship that separate technical considerations from social or political ones:

> The notion that the scientific component of decision making can be separated from the political and entrusted to independent experts has effectively been dismantled by recent contributions to the political and social studies of science (Jasanoff, 1990: 16).

Advisory (or policy-orientated or regulatory science) is, therefore, 'a hybrid activity that combines elements of scientific evidence and reasoning with large doses of social and political judgement' (Jasanoff, 1990: 229). For Jasanoff, such scientific advice is a process of negotiation and boundary-work (Gieryn, 1999). In outlining constructivist approaches to the study of science, Thomas Gieryn describes how

> the boundaries of science are episodically established sustained, enlarged, policed, breached, and sometimes erased in the defense, pursuit, or denial of claims of epistemic authority. As knowledge makers seek to present their claims or practices as legitimate (credible, trustworthy, reliable) by locating them within "science," they discursively construct for it an ever changing arrangement of boundaries and territories and landmarks, always contingent on immediate circumstances (Gieryn, 1999: xi).

Gieryn labels the activities involved in building, maintaining, adapting and policing these discursive perimeters 'boundary-work': 'Boundary-work occurs as people

contend for, legitimate, or challenge the cognitive authority of science' (Gieryn, 1995: 405). His historical case studies illustrate the argument that such tasks are fundamental to the credibility of scientific knowledge. [13]

Such boundary-work is central to Jasanoff's understanding of the interaction between scientific expertise and policymaking. She argues that there is a fluid boundary between science and policy, and that scientists draw and re-draw this boundary in order to exempt 'technical' matters from political control:

> By drawing seemingly sharp boundaries between science and policy, scientists in effect post "keep out" signs to prevent non-scientists from challenging or reinterpreting claims labelled as "science". The creation of such boundaries seems crucial to the political acceptability of advice. When the boundary holds, both regulators and the public accepts the experts' designation as controlling, and the recommendations of advisory committees, whatever their content, are invested with unshakeable authority (Jasanoff, 1990: 236).

However, this fluid boundary can also be used by expert scientists to define the limits of their authority and avoid their recommendations becoming politically controversial. Scientific advice about diet and body size has often been highly charged due to the operation of extensive commercial interests in this area and the potentially wide ranging political implications of public health policy. Researchers serving on expert committees in this field have adopted differing definitions of the boundaries of science (and therefore their expertise) in different contexts. For example, as they have come to understand the causes of excess bodyweight as resulting from a damaging environment, rather solely from individual failings, nutrition experts have acquired the authority to condemn the un-healthiness of many aspects of modern industrialised life (see section 8.2).

As I will go on to illustrate through my empirical analysis, the obesity epidemic provides an important example of policy institutions as knowledge makers – mandated or regulatory knowledge called into being for statutory purposes (Jasanoff, 1990). It is an example of knowledge making in the committee room, rather than the

[13] One case study is of the botanical research of Albert and Gabrielle Howard who developed the Indore system of composting – the horticultural compost heap - and are considered founders of modern organic farming (Gieryn, 1999).

lab or the clinic (see section 1.3 above). The knowledge created in these processes of policymaking has a distinctive character:

> What emerges from a successful recourse to scientific advice, then, is a very special kind of construct: one that many, perhaps most, observers accept as science, although it both shapes and is shaped by policy. That such constructs sometimes breakdown under political pressure is hardly surprising. Their frequent durability is the greater puzzle, for they are founded neither on testable, objective truths about nature, as presupposed by the technocratic model of legitimation, nor on the kind of broadly participatory politics envisaged by liberal democratic theory (Jasanoff 1990: 234).

This is partly because of the many overlapping roles that individual members and committees can play whilst providing expert advice to governments:

> Protected by the umbrella of expertise, advisory committee members in fact are free to serve in widely divergent professional capacities: as technical consultants, as educators, as peer reviewers, as policy advocates, as mediators, and even as judges. Though their purpose is to address only technical issues, committee meetings therefore serve as forums where scientific as well as political conflicts can be simultaneously negotiated. When the process works, few incentives remain for political adversaries to deconstruct the results or to attack them as bad science (Jasanoff, 1990: 237).

The activities of post-war British advisory committees on nutrition that I outlined in section 1.3 provide an illustration of many of these different roles and the processes of negotiating scientific and political conflicts that Jasanoff outlines in this paragraph. According to Jasanoff participants are aware that they are making claims to expertise that go beyond the purely scientific: 'the experts themselves seem at times painfully aware that what they are doing is not "science" in any ordinary sense, but a hybrid activity that combines elements of scientific evidence and reasoning with large doses of social and political judgement' (Jasanoff, 1990: 229). Reading reports on obesity and overweight does not give a sense of 'painful' awareness, but does demonstrate the hybrid nature of such knowledge as authors attempt to combine their research results with an awareness of both the politics of food production and the effects of industrialisation and modernisation on daily life.

Jasanoff also argues that there are distinctive aspects to British policy making and expert knowledge creation. As it often takes place in private, British policy making is

a ‘more insulated process’ that places greater faith in the trustworthiness of recognised experts (Jasanoff, 2005: 262) than the US political system. Secondly, in the UK policy making involves a specific kind of expert who has earned their status through sustained work on public issues:

> To a remarkable extent British expertise remains tied to the person of the individual expert , who achieves standing not only through knowledge and competence, but also through a demonstrated record of service to society. It is as if the expert’s function is to discern the public’s needs and to define the public good as much as it is to provide appropriate technical knowledge and skills for resolving the matter at hand. In this cultural setting individuals ranging from Prince Charles and Baroness Warnock to Julie Hill of the Green Alliance and various academic social scientists can all emerge as authoritative policy actors (Jasanoff, 2005: 268).

The relatively private nature of British policy making combined with a reliance on personal credibility means that, in some situations, expert scientists can credibly make recommendations going well beyond the areas of their research expertise (see chapter 8).

1.6.3 Previous STS case studies of nutrition science

As noted in the introduction to this section, the key novel contribution of my thesis is to apply STS theory to an analysis of obesity science. However, in concluding this discussion of my utilisation of STS it is important to draw attention to two important pieces of STS writing which overlap with the empirical focus of my research. Few STS authors have addressed nutrition science or the relationship between diet and health. Two authors that do write about this topic are Stephen Hilgartner and Karin Garrety. Garrety (1997) discusses the development of a consensus in American health policy of the 1970s, about the links between cholesterol and heart disease. She describes the complex negotiations that resulted in accepted knowledge about the links between coronary heart disease, cholesterol and dietary fat, in the absence, she argues, of ‘definitive’ experimental evidence:

> Even before the new knowledge escaped from the laboratory, actors in several social worlds promoted conditions which helped the initially unstable hypothesis to survive and thrive. They created both the social problem of heart disease and its solution – a massive medical research effort (Garrety, 1997: 757).

The organisers of the 1984 Consensus Conference on Lowering Blood Cholesterol to Prevent Heart Disease recommended that the entire US population over the age of two should reduce its consumption of dietary fat and cholesterol, despite results from a ten year trial that were only marginally statistically significant, and criticised within the medical literature (Garrety, 1997: 732). In describing how an influential and relatively durable consensus was achieved, Garrety demonstrates how different actors such as the American Heart Association, physiological and epidemiological research scientists, journalists from the non-scientific press, the food industry, the American Medical Association, politicians and the FDA became involved in the processes of creating knowledge about diet and health:

> Between 1960 and 1984, while scientists were endeavouring to construct the definitive evidence, many social worlds continued to use and shape various interpretations of the cholesterol hypothesis…the cholesterol hypothesis became increasingly popular in many social worlds. Most medical organizations, Western governments and numerous food industry and consumer groups supported dietary change. There were only a few individuals and groups interested in pointing out the "gaps in knowledge" (Garrety, 1997: 746).

She argues that the collection of knowledge claims linking dietary fat, cholesterol and heart disease acted as a boundary object (Gieryn 1999) 'with different meanings uses and implications in different social worlds' (Garrety, 1997: 755). Her conclusion is that the popularity of the cholesterol hypothesis led to boundary struggles as its opponents attempted to reverse the scientific consensus. Attempts to adjudicate these opposing claims led to sites where 'the worlds of science and policy merged' as the 'proponents of dietary change eventually jettisoned their requirements for "definitive evidence"' (Garrety, 1997: 757).

Stephen Hilgartner also writes about the public role of science (Hilgartner, 2000, Hilgartner and Nelkin, 1987). He has used a description of the public controversies around three of the National Academy of Science reports on diet and nutrition to illustrate his analysis of scientific advice as a form of performance.

> The ability to offer authoritative advice is obviously not an entitlement, automatically bestowed on any group that seeks it, but something that advisors

> must actively assert, cultivate and guard – sometimes in the face of intense opposition. How do advisory bodies lay claim to the cultural authority of science? How do they cast themselves as trustworthy advisors? And how do they create credible voices for themselves? (Hilgartner, 2000: 5).

In this way he addresses both the development of a consensus and the outbreak of a controversy, since for each report he shows how the credibility of scientific advice was produced, challenged and sometimes sustained. His detailed description of the production and reception of these three reports demonstrates the techniques of persuasive rhetoric, stage management and information control that underpin successful production of scientific advisory reports:

> this study suggests that struggles over the enclosure and disclosure of information play a far more important role in stabilizing (and destabilizing) scientific texts and knowledge than most recent work in science and technology studies has recognized (Hilgartner, 2000: 149).

All attempts at authoritative public statement of science-based advice will face similar problems. Consequently, Hilgartner's analysis is relevant to the reception of, for example, the obesity policy advice which I analyse in this thesis. However, Hilgartner's research was carried out in America, where policy processes are relatively transparent, and after a high profile public controversy where material was leaked to the popular press. This gave him access to a lot of 'backstage' material about the production of each report which is not available in all institutional contexts (see section 1.8.3).

1.6.4 Defining obesity science

In the case of public health policy on diet and health the resulting scientific knowledge is a new construct that draws from many areas of biomedical research – physiological research into the regulation of body weight in rodents and humans, clinical research into the effectiveness of specific weight reduction treatments and large scale epidemiological studies into the links between lifestyle factors such as diet, physical activity and smoking and chronic disease. As Jasanoff (1990: 227) argues it is a hybrid form that is both profoundly shaped by public health policy and has an important influence on subsequent policy**.** Drawing on these ideas of policy science as specific kind of construct, throughout the following chapters, I use the term

'obesity science' to refer to this particular body of knowledge that is the product of scientific advisory processes involving a variety of different social actors and incorporating results from many different areas of contemporary biomedical research, as well as other disciplines such as health economics and social psychology.

1.7 The making of health policy

Because of their focus on shared knowledge and the social processes that establish and maintain it, STS approaches mesh well with empirical analyses of the making of health policy that highlight the role of networks and communities. Gill Walt writes about the role of policy communities which are

> networks of individuals from various institutions, disciplines, or professions, and in the health field may be practitioners (health professionals) researchers (academic epidemiologists or parasitologists) or commentators (medical journalists). The health policy community might also include pharmaceutical companies, hospital administrators, any interest groups and members of government (Walt, 1994: 110).

She describes how policy communities form a nexus for the exchange of information, some of which will reach government policy makers:

> Policy communities provide a number of different fora in which the early stages of opinion formation and consensus building among experts takes place (scientific meetings, journals, newspapers) although it may take years for ideas to diffuse broadly, especially when they are critical of current policy (Walt, 1994: 110-1).

These policy communities may come together to form larger groupings that she labels policy networks, citing the example of the international network of development economists, family planning advocates and health professionals who developed and promoted the idea of primary health care (Walt, 1994: 111). Other authors extend this terminology to talk about policy sub-systems, issue networks and 'iron triangles'[14] (Buse et al., 2005: 111). Membership of the larger discourse or epistemic communities that form interest networks is defined by 'shared political values and a shared understanding of a problem, its definition and its causes, though usually

[14]The authors define 'iron triangles' as very stable small sub-systems of such networks.

marked by detailed disagreements about policy responses' (*ibid.*). On the international scale there are also global policy networks and which unite the domestic bureaucrats, elected officials and interest groups of the iron triangle with international organisations and global civil society (Buse et al., 2005: 153). Analysing the development of public health policy in these terms encourages a focus on coalitions that form around particular health issues, and the shared ways in which members of these coalitions frame their concerns and make their arguments.

1.8 Methodological issues

This section begins with a narrative of the ways my methodology evolved during my research. I then discuss my use of documentary sources, outline the criteria I used in the selection of my sources and conclude by describing some of the issues I encountered when studying the WHO.

1.8.1 Narrative of an evolving methodology

The starting point for this research had been my sense that the term 'obesity epidemic' marked a new way of understanding the health consequences of increasing rates of excess body weight. Therefore, the first stage of my research involved mapping the usage of the term 'obesity epidemic' – who used it and where? – and beginning to locate the documents that would become my key sources. The use of documents as my primary source material seemed appropriate at this stage as it would enable me to produce a detailed account of specific stages in the development and spread of this new body of knowledge. It would also provide this knowledge within its contemporary context, rather than recounted from the context of the present. I began by searching the popular and scientific press[15] and looking for the sources of their coverage of this topic. I quickly realised that both the popular press and the scientific press were reflecting and recycling the language of official reports and policy documents, most notably the WHO technical report of 2000. This was the first stage in my understanding that the discourse of an obesity epidemic was a body of knowledge created in the making of public health policy – it was not coined by the popular press re-interpreting the technical language of the scientific press, nor did it

[15] The results of some of these searches are discussed in chapter 3

arise directly out biomedical research. The obesity epidemic discourse was also a scientific consensus that very rapidly came to be part of a taken-for-granted public discourse.

Using library searches and following citations, I used a snowballing strategy to gather more published sources and become familiar with this body of literature. I was struck by the increasing importance given to excess body weight within chronic disease epidemiology and public health policy. Judging only by the conditions of their production and their contents, the sources I have assembled demonstrated that, between the 1970s and the 1990s, excess bodyweight became more intensively studied and more widely accepted as a significant public health problem. At the same time I realised that twentieth century medical interest in excess bodyweight initially derived from its role as risk factor for coronary heart disease (see chapter 2). This led me to investigate earlier official reports on coronary heart disease and historical accounts of the development of chronic disease epidemiology in the first half of the twentieth century, in order to understand the pre-history of obesity science.

I drew up a timeline to key events and documents in order to understand the development of both chronic disease epidemiology and obesity science. This led me to understand obesity science and discourse within a broad periodisation whereby obesity and overweight were first studied within chronic disease epidemiology as a risk factor for heart disease, then, as obesity science developed, as a condition in their own right, and finally as a rapidly increasing and globally threatening disease. As my research progressed, I learnt that the distinction between a disease and a risk factor can be more fluid than I first understood it to be. However, this does not fundamentally invalidate my description of the changing status of excess bodyweight or the observation that these changes were closely associated with the increasing attention paid to the problem of excess bodyweight within British public health policy in the 1990s.

In the course of this mapping process, I found that certain names recurred. There were a few very active individuals – George Bray, Philip James and John Garrow – and a wider group of regular participants that evolved gradually over the thirty year period, as individuals retired or focused on other aspects of their research. I began to see that

a concern with excess bodyweight had united a group of researchers in the fields of nutrition and chronic disease, and that these researchers had formed a network or coalition (see section 3.2) to persuade others of the importance of this new public health problem and lobby governments for action in this area. By using key word title searches of library catalogues and databases (such as Ingenta and Web of Knowledge), checking bibliographies, references to previous reports and the activities of key individuals, I continued to assemble the collection of key sources listed in Appendix 1 (see also section 1.8.3). This process was aided by witnessing a presentation by Philip James at a national conference in 2003 where he listed all the reports on obesity that he had been involved in producing over the last 30 years.

Philip James was a key participant in the network or coalition that developed around excess bodyweight but I did not restrict my searches to reports he had worked on. I also followed the activities of other researchers. As part of this tracking process, I examined the continuities between these reports and the earlier ones into coronary heart disease and diet-related chronic disease – both in terms of individual participants and framings of the material in the reports. I used obituaries and accounts of particular projects, such as the Seven Countries Study or the WHO MONICA project (see section 2.5), as a way of finding out more about the work of particular individuals and groups.

In order to analyse the developing understanding of excess bodyweight in this period, I decided to analyse the documents I had assembled in more detail. The background reading I had done in the early stages of my thesis research led me to outline a series of key changes that had taken place in understandings of excess bodyweight during this period. These changes took place in the areas of definition, measurement, health effects, causes, treatment and economic costs of excess bodyweight. I began to see that these two models relied on very different kinds of scientific data and had very different implications for treatment and prevention recommendations. Because of these differences and the fact that they largely operate on different levels, I argue that collectively they amount to a shift from one paradigm of excess bodyweight to another (see section 8.8). I have tried to encapsulate these shifts in the six questions listed below:

1. What is the prevalence of excess bodyweight - relatively low and stable or high and increasing?[16]
2. How is excess body weight defined and measured - using Metropolitan Life Insurance Company ideal weight charts and a range of indices or using the Body Mass Index (BMI) and standard cut-off points?
3. What are given as the health consequences of excess body weight –a relatively short list of complications, such as an increased risk of diabetes, CHD and mechanical problems such as osteoarthritis, or a much longer list of conditions, including some cancers, where the relative risks are elevated?
4. What are seen as the causes of excess bodyweight – is it the result of individual failings such as over-eating and inactivity or is it the symptom of the widespread adoption of inappropriate diet and sedentary lifestyle?
5. Should the therapeutic focus mostly be on individual treatments such as weight loss diets and drugs, or on population-level preventative measures such as taxing/banning certain foods or redesigning urban environments, aimed at populations?
6. Who pays for obesity – are the costs of obesity primarily born by individuals or by healthcare systems?

Because of their either/or format these questions are obviously simplified and schematic, but they were helpful as a framework to analyse the contents of my chosen sources and my results are summarised in the table provided in appendix 2. These results show a pattern of incremental change as successive enactments of this body of knowledge shifted the categories used, incorporated more research results and enlarged the scope of the pubic health problem of excess bodyweight and the potential solutions offered.

In the final stages of my research, I went back to my library catalogue and database searches to make sure that I hadn't missed any important reports. Once I was sure I had achieved saturation, I stopped searching. In addition, I searched a complete list of the WHO technical reports in order to check my understanding of the timing of its activities in the area of chronic disease epidemiology. I also decided that for periods such as the mid 1970s and late 1980s, when there were no reports on obesity published I would analyse the contents of textbooks as they can be considered as a comparable source of authoritative knowledge (Kuhn, 1970).

[16] This is my interpretation of the epidemiological data provided: all the authors I studied agreed that excess body weight was an important public health problem, and because of this basic understanding of the problem, they argued that rates are high and increasing even in the absence of large-scale prevalence data demonstrating such a pattern.

At this point, I also began to think more concretely about the status of the knowledge that I had derived from these documents. Analysing this collection of documents has demonstrated the evolution of a body of policy-orientated biomedical knowledge about the causes, consequences and possible policy responses to increasing rates of excess bodyweight. It showed a shift between two understandings of excess bodyweight that I label the individual and the environmental paradigms of obesity. Each of these texts can be considered as an enactment of a body of shared knowledge, as a policy science paradigm. Each contains an authoritative and contemporary account of knowledge about this subject – they were usually consensus statements of what could be said about particular topics or areas of research – and therefore functions as an exemplar within this area of research. Successive documents re-iterated this body of knowledge and developed it so that the understanding of excess bodyweight mobilised by obesity science slowly evolved. By the 1990s this evolution had led to the development of a new understanding of obesity and overweight as a population health problem with environmental causes, rather than an individual problem with solely behavioural causes.

However, there are important limitations of such documentary based analysis. Firstly analysing documents can give very little information about the conditions of their production. One way to overcome this would have been to combine such an analysis with archival research or interviews of key participants. At the time of my fieldwork I was unaware of the existence of the possible UK-based archives, such those of the Department of Health, and so did not consult them. For the WHO documents, archival research would have been of limited use since the organisation has a twenty year embargo in place. I did conduct a few interviews in the early stages of my research but I used them to inform my research rather than a source of research data. On reflection, I eventually realised that it might be useful and interesting to produce a more detailed account of the negotiations behind scientific consensus around the obesity epidemic. But I also realised that this would require other methods and further research. Interviews would have potentially offered a valuable source of additional data. They could have provided information about the negotiations involved the production of these reports, which would have usefully supplemented my textual analysis. This layering of perspectives would have amounted to a form of triangulation (Ritchie, 2003: 43-44), and added depth to my findings (see also section

1.8.4 below). However, by that time I had more than enough research material for a PhD and insufficient time to interview such elite government scientists.

1.8.2 Use of Documentary Sources

There are several methods I could have used to investigate the changing discourses around obesity. I considered carrying out my research by interviewing research scientists and epidemiologist to analyse whether their understandings of obesity have changed and what effect these changes might have. Instead, I have chosen to make my research a primarily document-based case study that analyses writing about this issue.[17] Documents[18] and interviews can be seen as epistemologically equivalent forms of evidence. That is to say neither provides a complete and authoritative account of a social phenomenon such as obesity science (Kvale, 1996, Platt, 1981). Like interviews, documents represent a partial account of social phenomena which must be analysed within the social and historical context where they are produced and the uses to which they were intended to be put.

I chose the method of a document-based case study because I was interested in providing a detailed account of the development of a body of shared and widely accepted knowledge. I believe that document-based research is particularly useful in tracing processes of concept formation and development. Actors' recollections of even the recent past can be inaccurate due to the effects of overlaying present ideas onto past events; consequently, interview evidence about past events, and especially about past thought processes, can often be unreliable. Moreover, recollection of scientific developments, in particular, tends to be heavily influenced by positivistic ideas of truth and discovery (Lowy, 1990). Document-based analysis can overcome this effect, and therefore provide a more nuanced account of how concepts developed and specific details of how they were mobilised at particular times and in particular contexts.

[17] I have conducted interviews with 6 individuals – epidemiologists, public health workers and social scientists who have conducted research in the area of obesity. These were helpful in developing my knowledge of more recent framings of specific issues, but I did not make use of them directly in the following chapters.

[18] I define document as including writing in the scientific and popular press, official reports and government publications and the contents of relevant websites.

Writers on documentary analysis argue that the major issues to be considered by researchers are authenticity, credibility and representativeness (Platt, 1981, Scott, 1990). My research is based on contemporary documents and so authenticity is not a problem. Expert committee reports and other policy science publications are produced with the aim of being authoritative accounts of particular bodies of knowledge, and this gives a particular status to the knowledge contained within them, and a high degree of credibility. These reports are consensus documents (see below) and are, therefore, central to the charting and explanation of changes in the policy science consensus about the obesity epidemic. Textbooks were a less important source in this context but I used them to supplement the available reports because they are considered paradigmatic accounts of disciplinary knowledge (Kuhn, 1970: 43). Meeting or symposium proceedings and reports (in this field at least) function in a similar fashion. All these publications are repositories of a common body of knowledge about a particular topic, which makes them central to the analysis of the development of a shared body of consensus knowledge. Interviews with participants, such as expert scientists or civil servants and archive research would have enabled me to describe the micro-politics behind the policies being recommended but would have also diluted my focus on the development of a shared body of scientific knowledge about obesity and overweight.

I have treated the reports I study as 'consensus reports'. Hilgartner's description of the production of National Academy of Science reports explains how such reports are produced:

> Each consensus report is prepared by an expert panel, expressly convened for that purpose. These expert committees do not perform new research, but review the literature, hold workshops and symposia, and consult with relevant specialists. The Academy instructs its committees to strive to reach unanimous agreement on their findings and recommendations (Hilgartner, 2000: 23).

In my analysis of the reports I consider, I have assumed that they are produced in a similar fashion, so that they have been jointly authored and produced on a consensus basis. I think they can be considered, in a very specific sense, as a lowest common denominator - a body of consensus knowledge that can be agreed upon by all participants. I address issues of representativeness in the next section.

1.8.3 Selection of Sources

I have focused on policy writing in English. Despite the parallels that are drawn between increasing rates of obesity and overweight in Britain and America, I have looked at the development of obesity science primarily from a UK perspective, only using US and other international sources where these represent an important input into or extension of the activities of UK obesity scientists. This was for both practical and academic reasons: I thought a wider perspective would hugely increase the size of my data sample, and I did not think I had sufficient knowledge of American policy processes and institutions, at this stage in my career, to provide a properly nuanced analysis of such material.

Obesity science is also an international discourse and some of my material reflects this, such as the WHO technical reports and the heavy reliance on American data in earlier publications (see chapter 3). Given the international nature of contemporary biomedical science, this is to be expected, and it also means that a strictly geographical analysis is not possible. Medical writing in English is an international discourse, although much of it still appears in journals published in the US and UK. However, international professional networks and research collaborations mean these articles are reporting on research that is carried in many different countries. Such considerations mean that it would be difficult to use geographical categories to exclude authors or research in any meaningful fashion. Therefore, I have analysed this discourse as one entity using its existence in English as a method of demarcation, rather than any hypothetical country of origin. Moreover, it would be counterproductive to attempt to exclude American research from my analysis, since it was such an important resource for British authors.

A bibliography on the topic of obesity published in 1974 (Whelan and Silverstone, 1974) begins with a publication from 1964, but the majority of its entries date from after 1969. This is consistent with my searches of UK academic libraries. From the late 1960s onwards, a small number of publications begin to appear; several of these are reports of symposium proceedings, but there is also one publication from the Office of Health Economics and a couple of textbooks (see below). A search of the

COPAC database of British research library catalogues [19] using the keyword 'obesity' gives 8 publications from the 1950s, 20 publications from the 1960s and 80 publications from the 1970s. Because of this pattern of growth, I have used two different criteria in my selection of sources. For the 1960s, because writing on this topic is relatively uncommon, I have tried to find early reports, conference reports and textbooks.[20] From 1970 onwards, as there is much more material available, I selected all the official reports on the topic of obesity and textbooks produced by high profile individuals who were regularly participating in the production of obesity science, usually as members of expert committees.

In 1969 the Office of Health Economics (OHE)[21] published the earliest British report on the topic of obesity I have been able to locate. This report was produced when relatively little was being written on the topic of obesity and appears to have been largely ignored by researchers and practitioners. I have considered it, therefore, not as an example of an influential source, but to provide an example of what could be said about the topic at this time. The OHE produced another report on obesity in 1994, again as part of their series on the economics of diseases which now appears to be moribund. Many of these seem to be areas where new drug treatments could be expected to be developed, and in this context their 1969 report seems like an opportunistic, and unsuccessful, attempt to contribute to a developing discourse.

The next report produced jointly in 1977 by the Department of Health and Social Security (DHSS) and the Medical Research Council (MRC) considered research into obesity. It was a survey of an emerging field that focused heavily on physiological research into the human metabolism and endocrine system. In 1983, the Royal College of Physicians produced a report on the topic that demonstrated increasing interest by clinical medicine practitioners in this area. Then there was a gap of 11

[19] This count omits duplicates, non-English writing, unpublished theses and self-help/diet books in order to try and give an estimate of scientific writing on the topic in English. Using a keyword search should compensate for the fact that some writing about obesity is given titles referring to advances in nutrition, dietary therapy or energy balance, rather than obesity, and it shows a similar pattern of increasing publications on the topic.

[20] An earlier symposium on obesity took place in 1963 at the Royal College of Surgeons but I have not been able to locate a copy of the proceedings.

[21] The OHE is a research organisation founded in 1962 by the Association of the British Pharmaceutical Industry. Its mission is to 'Support better health care policies by insightful economic and statistical analysis of critical issues' by providing 'authoritative resources, research and analyses in health economics and health policy' (OHE, 2009a).

years (from 1983 to 1994) in the sequence of reports on obesity. However, in this period scientific research on excess bodyweight and the links between diet and chronic disease was well established, even if obesity was not seen as a politically important health problem. Moreover, policy concern about healthy eating kept ideas about the relationship between diet, health and body weight circulating in the non-scientific press. In 1994 the UK government set up two taskforces on nutrition and physical activity and in 1995 a report on obesity was published by the Department of Health. In 1996 the Scottish Intercollegiate Guidelines Network produced a report on the topic and the British Nutrition Foundation produced one in 1999.

The WHO reports I have included form a different series with a different rationale behind their selection. The technical report of 2000, *Obesity: Preventing and Managing the Global Epidemic* was the starting point of my research. It was a widely cited authoritative source for the idea that obesity had become a major public health problem. Having identified this and heard Philip James give a presentation about the reports on obesity he had been involved in producing, I tracked back to the earlier British reports that I have noted above. However, as my research progressed, I realised that earlier WHO technical reports about cardiovascular disease and the links between chronic disease and health had also been incorporated into obesity science and I included technical reports from 1982, 1986 and 1990 on these topics. The 1995 technical report on anthropometry was included because Philip James participated in its production and because its treatment of the topic of excess body weight is very close to that of the 2000 report.

Finally I want to note that this series of publications illustrates the success of the public health coalition that formed around obesity and overweight. The first report was produced by a think-tank and ignored, and in the 1970s and 1980s the topic was written about by an ad-hoc committee and professional medical organisations. However, by the mid 1990s, reports were being published by the Department of Health and, in 2000, by the WHO. This ascent continued in the next decade as obesity became a policy issue spanning many UK government departments (Government Office for Science, 2007).

1.8.4 Issues in studying the WHO

In some senses the WHO is a well-documented organisation. It has always produced reports of its activities and much of this output is well-documented, readily available and now often online. Three official histories of the organisation have been published (WHO, 1958, WHO, 1968, Litsios, 2008) covering the period 1948 to 1978. However, official histories give an authorised account of events that does not dwell on internal politics or controversial aspects of the organisation's activities. There is also some scholarly writing analysing the founding of the organisation and its history (Siddiqi, 1995, Lee, 1998, Staples, 2006, Lee, 2009). These authors have provided much useful information about the workings of the organisation's structure, and the history of high profile campaigns such as the Malaria Eradication Programme (Siddiqi, 1995, Lee, 2009). However, I have been unable to find any secondary sources describing the production of technical reports in general, or specific technical reports such as the one on obesity published in 2000. Walt (1994: 144-5) writes of the existence of international policy networks and the role of international non-governmental organisations - such as the International Obesity Taskforce – but in the absence of information about the production of these reports it is possible only to speculate about the influence of particular individuals and groups.

I have also not been able to gather much about the organisation's work in the area of chronic disease epidemiology, beyond what is outlined in the official histories referred to above and an official history of the WHO Regional Office for Europe (Kaprio, 1991). This is because the WHO, like other UN bodies, does not yet appear to function in a very transparent manner. For example, unlike in government or academia, it is often difficult to establish where even relatively high profile individuals are currently employed within the organisation, or what posts they have held in the past. Again some of these issues derive from my use of exclusively documentary sources, rather than supplementing them with interviews. Because the WHO is such an opaque organisation, and the development of health policy in Britain has traditionally relied on the recommendations of expert committees who deliberate in private (Jasanoff, 1997: 228), access to the kind of 'backstage' material that Hilgartner (2000) uses would have involved a different research approach based on interviews and archive material. Interviews with key participants would have provided me with greater understanding of the processes involved in the production of

these documents (see section 1.8.1 above) and the additional perspectives generated by such interviews would have allowed for triangulation of my data (Denzin cited in Lewis and Ritchie, 2003: 276). However, as I outlined above (see section 1.8.2 above), I initially set out to examine an agreed on body of policy science knowledge and how it changed over time, so, at first, I was less interested in the kind of 'backstage' detail that would have been available from interview-based research.

1.9 Childhood obesity

I have explicitly excluded consideration of childhood obesity from this thesis. Since 2000, rising rates of excess body weight amongst children has become a high profile issue in health and education policy (SIGN, 2003, WHO, 2006). It is also an issue that was discussed in some of the reports I analyse in later chapters (DHSS/MRC, 1976, RCP, 1983, SIGN, 1996, WHO, 2000). Excess body weight in children was seen as important on the grounds that fat babies become fat children and then fat adults. However, reports from the 1970s described the evidence for this assertion as 'fragmentary and sometimes conflicting' (DHSS/MRC, 1976: 11, see also RCP, 1983: 23). But by the late 1990s, the authors of another report could describe infancy and childhood as one of the 'critical period' for the development of obesity and cite data demonstrating the link between obesity in childhood and as an adult (BNF, 1999: 49). Moreover, the definition and measurement of overweight and obesity amongst children and young adults was a contested issue. In 1983, one author argues that there is a need for a standard measure using a reference population so that studies of different populations can be compared. The WHO had collected such data for the American population, but there was still the problem of how to express individual children's weights and define obesity and overweight (RCP, 1983: 18-19). By the mid 1990s, British reference growth charts for children had been developed and limited data suggested that 20% of children and adolescents in one area of Scotland might be overweight (SIGN, 1996: 4,9). However, at the end of the decade, a WHO report stated that there 'has not been the same level of agreement over the classification of overweight and obesity in children and adolescents as there has been in adults' (WHO, 2000). Part of the problem in drawing up such classifications was that, unlike in adults,

> In children….BMI changes substantially with age, rising steeply in infancy, falling during the preschool years, and then rising again during adolescence and early adulthood. For this reason, child BMI need to be assessed using age-related reference curves (WHO, 2000: 12).

Differences between populations and lack of national data were also confounding the task of constructing a globally applicable childhood reference population (WHO, 2000: 12-3). Despite these problems, the report's authors argued that

> whatever method is used to classify obesity, studies of this disease during childhood and adolescence have generally reported both a high prevalence and rates that are increasing (WHO, 2000: 32).

The results of available research studies showed that the prevalence of overweight had doubled amongst 5 to 24 year olds in Louisiana between 1973 and 1994; the rates of obese schoolchildren in Japan aged 6 to 14 had increased from 5% to 10% between 1974 and 1993; and the prevalence of obesity among Thai children aged 6 to 12 increased from 12% in 1991 to 16% in 1993 (*ibid.*). This was similar to the situation for adult rates of overweight and obesity (see chapter 4) and, in this case, these very scattered results provide a weak evidential basis for inferring the existence of a global public health problem.

Because of their dependency on adults for food and the structuring of their time by compulsory education, the causes of overweight and obesity amongst children were framed differently those for adults. They were discussed in terms of infant feeding patterns (DHSS/MRC, 1976: 18-19), low levels of physical activity (BNF, 1999: 90, 120-2, WHO, 2000: 121), television viewing (WHO, 2000: 121) and food advertising (WHO, 2000: 133). And finally, due to their ongoing development, the suggested treatments for overweight and obese children and young people were also different from those for adults. Because they are still growing, children need adequate nutrition, which is difficult to reconcile with treatment for excess weight. One author considered that between the ages of 5 and 10, measures to limit weight gain rather than lose weight would be sufficient due to further growth (BNF, 1999: 145). A later report accordingly describes treatment as involving reducing energy intake, improving dietary quality, increasing physical activity and reducing sedentary behaviour – although pharmaceutical and surgical treatments could be considered for children with

'potentially fatal complications of obesity' (WHO, 2000: 226-8). These are measures that are considered to prevent weight gain rather than promote weight loss, as would be appropriate for adults, but they are also described as preventive of adult weight gain since 'childhood obesity substantially increases the risk of adult obesity' (*ibid.*).

1.10 Practical Issues and Limitations

The main constraint that I have encountered has been obtaining information of processes within the WHO (see above). If I had managed to interview high profile participants, like Philip James or John Garrow, this might have been less of a problem, but I would have had the additional issue of integrating their retrospective accounts with the material from my documentary sources. This would be an important and interesting task, possibly for future research now that I have a firm grasp on the development of the field of obesity science. Other limitations of this research are that it focuses on a literature of public health policy advice and recommendations contained within it. Such writing cannot be assumed to reflect clinical medical practice or the planning of health care. Again further research would be required to establish whether such writing has concrete effects, and what they are in different areas of medicine.

Primarily, I intend this to be a piece of research for its own sake that adds to knowledge in my chosen field. I did not plan to carry out the kind of research that feeds directly into policy making. As it provides a historical account, rather than an analysis of contemporary initiatives, it may also be of less interest to policy makers, despite arguments for the use of history in this context (Berridge, 2000, Berridge, 2008). However, obesity science and the links between diet, nutrition and health remain a topic of both political and media interest and so I anticipate that this research may be seen to have some relevance for public health policy. If that is the case, then I hope that my research can provide a different perspective on the health implications of excess body weight, and demonstrate links between this and other contemporary policy issues, such as health inequalities.

1.11 Conclusion

To conclude this introduction I will give a brief outline of the chapters to follow. In chapter 2, I outline the history of post-war chronic disease epidemiology which developed out of changing patterns of population health and a growing concern with rising rates of coronary heart disease. I show how this led to a new understanding of chronic diseases, the risk factor approach. In chapter 3, I discuss the formation from the 1950s of a public health coalition around one such risk factor – smoking – and describe how nutrition researchers and their allies began to develop another coalition around the topic of excess body weight. I do this by analysing a series of reports and textbooks on the topic of obesity that were published between 1969 and 2000. I outline this coalition's initial model of the condition, which I label the individual paradigm. In chapters 4 and 5, I describe two important areas of obesity science – the development of the BMI and BMI-based models of overweight and obesity, and the energy balance model and its explanations of the causes of obesity. Chapter 6 analyses these topics in more detail, discussing the advantages of BMI for obesity science and using approaches from medical sociology to outline the limitations of the energy balance model. In chapter 7, I move to the international forum and discuss the development of knowledge about chronic diseases and excess body weight in World Health Organization (WHO) publications from the early 1980s onwards. I demonstrate how increasing incorporation of the results from epidemiological studies and a new institutional context led to changes in the shared model of obesity and overweight. Chapter 8 analyses a WHO technical report of 2000 on obesity as a global epidemic, explaining further changes in the understanding of obesity that constitute a new model I label the environmental paradigm. In chapter 9, I analyse this new model, discussing the framing of obesity as a global health problem despite the paucity of data and the implications of explanations that rely so heavily on accounts of the effects of modernisation, rather than other plausible causes such as poverty. Chapter 10 concludes with a discussion of the conclusions, both empirical and theoretical, that can be drawn from this case study of the development of public health policy knowledge.

CHAPTER 2: HEART DISEASE AND THE EMERGENCE OF RISK FACTOR RESEARCH C.1945-1980

2.1 Introduction

This chapter discusses a profound shift that took place in the medical research and practice of industrialised countries after the Second World War. It describes the growth of concern about rates of chronic disease, such as heart disease and cancer, and the new research methods that were developed in order to understand the causes and treatments of these conditions. The most important of these new methods was the large scale prospective epidemiological study, as exemplified by the Framingham Heart Study which was set up in 1948 to investigate the prevalence and development of heart disease (Dawber, 1980: 239). This study is still running and has had a major impact on contemporary medical understandings of the causes of heart disease.

Using a model borrowed from the insurance industry, Framingham researchers developed an understanding of heart disease as a multi-factorial condition that was caused by a set of lifestyle-related attributes such as high blood pressure, cigarette smoking and elevated blood cholesterol levels. These attributes quickly became known as risk factors and this approach became a crucial element of the development of the post-war epidemiological paradigm of coronary heart disease (CHD) (Aronowitz, 1998: 113). This paradigm, and the wider field of chronic disease epidemiology, is one important location from which much biomedical concern with excess body weight – usually labelled as overweight or obesity – developed. Overweight and obesity became the subject of twentieth century medical research due to their status as risk factors for heart disease. This chapter, therefore, functions as an outline of the immediate context from which British (and American) obesity science developed.

2.2 The epidemiological transition and the epidemic of heart disease

Mortality statistics collected in developed countries, such as the UK and the US, show a striking change in disease prevalence from the middle of the nineteenth century onwards. Overall mortality rates declined, due to a decrease in death rates from

infectious diseases such as cholera, typhus, typhoid, diphtheria and tuberculosis that had been the major cause of mortality and morbidity. However, this decline was perplexingly associated with an increase in rates of chronic or non-communicable diseases (NCDs). Certain types of these diseases, including heart disease, stroke, cancer and diabetes, became seen as important sources of mortality and morbidity. Discussing the natural history of chronic diseases – 'the term that has become used, rather loosely, for the malignant, metabolic and mental disorders that present mostly in the second half of life' - one British epidemiological textbook explains this new understanding as follows:

> In the ageing population of an affluent society, which has mastered many of the infections and malnutrition, and has high standards of maternal and child care, these chronic diseases increasingly dominate the practice of medicine (Morris, 1964: 133).

This change is currently understood to have taken place across many different countries and has been labelled the 'epidemiological transition' (Omram, 1971, Susser, 1985b: 149-51, Szreter, 2007: 4-5). During this transition, societies are understood to have shifted from a pattern of high mortality and high rates of infectious diseases to one of low rates of mortality and higher rates of chronic diseases (see section 1.2.2). Over a period of a hundred years, between the early nineteenth and early twentieth centuries, such a shift was associated with dramatic increases in average life expectancies in the industrialising economies of the UK, USA and Western Europe.

As rates of infectious disease fell, widespread medical (and public) concern developed about high rates of heart disease amongst certain populations: 'the great epidemic disease of the twentieth century' (Rothstein, 2003: 191). Rothstein argues that despite changes in classification and inconsistency in death certificates, the diagnosis of coronary heart disease was rare in the late nineteenth century, and only became sufficiently common to be included in medical textbooks in the 1920s: 'Expressions of concern and confusion during the 1920s indicated that they viewed coronary heart disease as a new and growing problem in medicine' (Rothstein, 2003: 199, see also Lawrence, 1997). In the next thirty years, coronary heart disease was to become recognised as an increasingly common cause of mortality and morbidity, especially

amongst middle aged men. By the 1950s chronic diseases were seen as a major health problem in the US, and there was a developing consensus that the British population was also suffering from rising rates of coronary heart disease:

> It is widely accepted by those involved in health education, community medicine and even medical sociology that since the end of the Second World War, Britain has suffered an epidemic of heart disease. Epidemiologists tell us that "coronary" or "ischaemic" heart disease is both the largest single category of fatal illness among men and also the disease responsible for most of the inequality between social classes in life expectancy (Bartley, 1985: 289).

This new concern with heart disease as an important health problem redirected medical attention from infectious to chronic disease, leading to the production of further evidence for the growing prevalence of CHD. The evidence derived from two series of autopsy studies. One series took place in the 1930s, as doctors tried to gather information about the prevalence of heart disease and the reliability of pre- versus post-mortem diagnoses (Rothstein, 2003: 200 - 202). Such studies aimed to improve diagnosis in the living, but a later series of post-mortems carried out in 1953, on soldiers killed in the Korean War, was conducted in order to establish the prevalence of atheroschlerosis (narrowing of the arteries, seen to be a precursor of CHD). The results showed that the majority had some degree of hardening, which had been not expected in such a group of relatively young men (Atrens, 1994).

> Thus a large number of studies using different methods and populations all found a real increase in coronary heart disease death rates that began about 1920. The increase also occurred at the same time in other westernized countries. Unquestionably, at that time coronary heart disease began its rise to become the great pandemic disease of the twentieth century in all advanced countries (Rothstein, 2003: 203).

From the early days of cardiology, middle aged male businessmen had been seen as particularly vulnerable to heart disease: 'Business men leading lives of great strain, and eating, and drinking, and smoking to excess, form the largest contingent of angina cases' (William Osler [1914] quoted in Rothstein, 2003: 206).[22] Such accounts focused on the dangers of affluent living and the stresses and strains of modern city life. Studies based on Metropolitan Life Insurance Company data from the 1920s and

[22] These ideas fed into theories of the Type A personality that were developed by American sociologists from the late 1950s onwards (Riska, 2000).

1930s showed that coronary heart disease rates were higher for men than for women with a ratio that decreased with increasing age (Rothstein, 2003: 206). However, despite doctors' willingness to believe that coronary heart disease was a disease of the affluent, American and British studies in the 1950s showed that lower socio-economic groups had higher rates of heart disease: 'Coronary heart disease was similar to practically all other major causes of death in attacking the poor with greater frequency and severity than the rich' (Rothstein, 2003: 208). But the stereotype meant that high mortality from heart disease was seen as an important economic problem, since it killed middle aged men who were key wage earners.

2.3 Investigating the causes of chronic disease

Mid-twentieth century doctors and policymakers proposed various explanations for the epidemiological transition. The decline in infectious disease was usually attributed to a combination of medical and social progress: improvements in public health, such as sanitation and clean water, improvements in medicine, such as vaccination and new drugs like sulphanilamide and penicillin, and rising standards of living were all seen to have played a role (Susser, 1985b: 150). During this period, doctors and medical scientists had developed a clear explanatory paradigm for infectious diseases based on laboratory-based scientific knowledge of the infectious and pathological role of bacteria and viruses. In the second half of the nineteenth century, the work of Joseph Lister, Louis Pasteur and Robert Koch changed understanding of disease (Worboys, 2000) and ultimately led to the development of effective treatments for many of the major infectious diseases. An important element of this new understanding of medicine and sickness was the idea of specific causes of disease, since it built on existing 'germ' theories to argue that infection by specific micro-organisms caused disease (Weindling, 1993:193), rather than the environment or the patient's individual disposition. This meant that the search for the causes of, and cures for disease, should be conducted in bacteriological laboratories. Because this 'bacteriological revolution' was so successful, it eclipsed other explanations for disease for much of the next fifty years: 'The cultural power of the bacterial model within medicine was such that alternative, nonbacterial theories of infection ceased to be articulated' (Worboys, 2000: 280). Even in disciplines such as epidemiology, which had strong links to

public health and traditions of social medicine, bacteriological explanations were dominant in this period (Amsterdamska, 2005).

In contrast, the causes of chronic diseases were not known. Because such diseases develop over several decades, researchers could not readily identify specific aetiological factors as had been the case for infectious diseases (Rothstein, 2003: 5). Elizabeth Fee describes the post-war situation as one of confusion and feuding between medicine and public health:

> The problem was that nobody knew how to prevent cancer or heart disease. There was little agreement or clarity about the relevance of nutritional, occupational or environmental health – or about any other aetiological factors. The only approach to prevention upon which everyone could agree was the need for screening and early diagnosis. The chronic diseases – cancer, hypertension, diabetes and others – could neither be prevented nor cured on the older public health and medical models; at best, they could be controlled through screening, education and medical supervision (Fee, 1996: 250).

This explanatory gap meant that doctors tended to invoke other explanations, especially ones that involved assumptions about medical and social progress, though in often ambivalent or contradictory ways. The rise in chronic diseases was often attributed to the fact that, due to the decline in rates of infectious disease, people lived longer. Conditions such as heart disease and other cardiovascular diseases were simply the result of older bodies or individual organs wearing out – hence the label of 'degenerative diseases'. Distinguishing ageing from the diseases that often accompanied it led to the hope that these conditions might be treatable:

> Increasingly chronic disease was thought to result from specific and not necessarily inevitable individual and environmental factors…This conceptual change from degeneration to specific mechanism, along with the growing optimism that specific policy and clinical interventions might prevent or ameliorate the onset and course of chronic disease, prepared the way for embarking on costly, large-scale population studies of CHD risk (Aronowitz, 1998: 122).

An alternative, more pessimistic, understanding drew on narratives of the problems of social progress - what Rosenberg (1998) labels the 'pathologies of progress' and others 'diseases of civilisation' – to argue that diseases such as CHD resulted from changes in lifestyle resulting from increased wealth – hence the term 'diseases of

affluence' (Morris, 1964). The excesses of eating drinking and smoking that Osler described as habits of wealthy businessman were thought to be becoming commonplace, due to economic growth and increasing prosperity.

2.4 The development of risk factor research

Meanwhile, certain strands of research were developing new ways of investigating the aetiology of chronic diseases, especially CHD, which replaced laboratory research. A key resource was the actuarial investigations that had been taking place within the American insurance industry. In the early twentieth century, American insurance companies began to develop mortality tables, in order to be able to insure individuals at above average risk, known as "impaired lives" or "substandard risks" (Rothstein, 2003: 63). Due to increasing prosperity, life insurance was a rapidly expanding industry during the late nineteenth and early twentieth centuries and increasingly precise physical examination was also being used by other institutions such as railroads, schools, police forces and the army (Davis, 1981: 393). Insurance companies employed local doctors as medical examiners who were instructed to measure and examine potential policyholders with a variety of tools, including (in 1891) a set of scales, tape measure, ophthalmoscope, stethoscope, urinary test apparatus and a sphygmomanometer (Davis, 1981: 398). It is therefore not surprising that these companies 'most systematically and effectively accumulated relevant physical examination data and related to the health and disease potential of the largest number of people in the United States prior to World War I' (Davis, 1981: 394). Insurance companies, especially the Metropolitan Life Insurance Company, had the most comprehensive collection of data on trends in body weight and its relationship to health until at least the 1960s, when governments started to collect such information (Oddy et al., 2009: 225).

In 1903 the *Specialized Mortality Investigation* was published by the Actuarial Society of America. This investigation of the feasibility of insuring 'high risk' or 'impaired' policyholders was based on data from 38 countries. The Joint Committee on Mortality, another insurance company body, was founded in 1909 and published a series of studies between 1912 and 1939 that identified new medical factors which increased the risk of mortality. Rothstein argues that these studies are the origin of the

modern concept of risk factors for diseases such as heart disease and cancer. The following excerpt summarises this process for one particular risk factor, body weight, which is central to my research:

> The most unexpected finding of the original 1903 study was the discovery of a strong relationship between mortality and 'build', a construct that combined height and weight. Using industry data gathered between 1909 and 1928 for all male policyholders ages 40 to 49 and taking 100 as the average mortality rate, those who were at least 25% overweight for their height had a relative mortality rate of 141, those of average weight had a relative mortality rate of 86, and those who were 5% to 14% underweight had a relative mortality rate of 77…..The low mortality rates of slightly underweight policyholders astonished physicians who were busy treating gaunt patients sick or dying of tuberculosis, the "wasting disease". They viewed ruddy and rotund persons as the epitome of good health and underweight ones as suspect. Although the medical directors had no theory of disease etiology that explained the statistical relationship, they accepted it unequivocally and made it a key factor in selection [of policyholders] (Rothstein, 2003: 64).

Medical directors of insurance companies were more concerned with profits than aetiology and so they accepted this new and unexpected relationship between build and increased mortality. Their use of weight in medical examinations, the selection of policyholders and the construction of mortality tables, made weight, or build, one of a set of important medical risks that also included high blood pressure, diabetes and kidney disease.

2.5 The American heart lobby and the Framingham Heart Study

A major research effort was seen as necessary to investigate the precise causes of the epidemic of CHD and provide information on how to prevent cases in the future. The actuarial method, with its focus on identifying risk, was recognised as a powerful new method for investigating heart disease. During the 1940s, the American Heart Association drew on fears of the growth of heart disease to raise funds and win resources, leading in 1948 to the passage of the National Heart Act and the creation of the National Heart Institution (NHI) within the National Institute of Health (NIH) (Fye, 1996). The AHA had been founded in 1924, as the professional organisation of the emerging speciality of cardiology. By the early 1940s the organisation was well established but had chronic financial problems, and had limited success in raising

research funds from the insurance industry. In 1937, the National Foundation for Infantile Paralysis had successfully used celebrity endorsements and political allies in the White House to raise enormous sums of money in its March of the Dimes campaign (Fye, 1996: 94-7). The success of this model of fund raising, combined with the founding of competing organisations in the area of heart disease and hypertension, led to the transformation of AHA into a voluntary health organisation that raised funds for research into heart disease:

> After World War II, AHA leaders joined with a group of legislators, government officials and concerned citizens to form the "heart lobby" which used a military metaphor to describe their goal. They told the American people and their leaders that heart disease could be *conquered* if enough money was given to support cardiovascular research. The success of the Manhattan project to build an atomic bomb and recent medical advances, such as antibiotics, helped them make their case (Fye, 1996: 86-7).

In the 1930s and 1940s there was a significant increase in funding for medical research, firstly from private bodies such as the Rockefeller Foundation, and, later, from the US government:

> The AHA leaders looked beyond public-fundraising, corporate donations, and philanthropists to support their twin causes: cardiovascular research *and researchers*. They knew that the federal government was the most promising source for research funding…Gradually a coalition of politicians, federal officials, scientists and academic physicians succeeded in getting the government to liberally fund research projects in private institutions (Fye, 1996: 102).

Leaders of the AHA were aware that they were competing with groups lobbying for funding for other diseases, especially cancer. After the National Cancer Institute Act was passed in 1937, the heart lobby used cancer lobbying as a model (Fye, 1996: 103). They recruited allies, such as Mary Lasker the wife of a rich industrialist, and several politicians including the Surgeon General, Leonard Scheele. As a result of intensive lobbying, the National Heart Institute (NHI) was founded in 1948 and the First National Conference on Cardiovascular Diseases (sponsored by the AHA and NHI) took place in Washington in 1950. The NHI continued to lobby Congress for increased funding. In 1953, its director told Congress that 10 million Americans suffered from cardiovascular diseases and that '[H]eart disease exacts an enormous

economic cost in medical and institutional care, in military management, and in industrial production' (quoted Fye, 1996: 160). In such lobbying, the newly established heart lobby presented cardiology research as the search for a cure for heart disease, an approach that united the academic and practitioner groups within the AHA. This increased funding for medical research post-WW2 led to a dramatic expansion in cardiovascular research activity, and US cardiology continued to expand throughout the 1950s and 1960s (Fye, 1996: 181).

Also in 1948, the NHI and the University of Boston set up the Framingham Heart Study. The researchers decided to locate the study in Framingham, a small town in Massachusetts, because of its size and proximity to medical facilities in Boston, its homogeneous population[23], the existence of an annual census and previous successful participation in a tuberculosis study between 1917 and 1923 (Dawber, 1980). In the 1950s Framingham researchers borrowed the idea of risk factors from the insurance industry, and from 1961 the terms 'factors of risk' or 'risk factor' were being used in study publications (Rothstein, 2003: 283). The first risk factors identified were age and sex; subsequently, already identified risk factors of hypertension, raised blood cholesterol, increased body weight and smoking were also re-identified (Dawber, 1980), and the researchers of the Framingham study were 'among the first to emphasize the dangers of smoking for coronary heart disease' (Rothstein, 2003: 283).

An international consensus about the relevant risk factors to investigate began to develop relatively fast. A 1957 WHO technical report on atheroschlerosis and heart disease discussed the role of environmental factors such as calories, obesity and overweight (labelled as dietary factors), physical activity, stress, alcohol and tobacco and infections (WHO, 1957). In this report, the authors state that 'obesity in itself is not a primary factor in producing ischaemic heart disease' (WHO, 1957: 17). In 1962, in another technical report on the prevention of arterial hypertension and ischaemic heart disease, factors indicating 'exposure to increased risk' were listed as high blood pressure, high blood cholesterol, obesity, diabetes, heavy cigarette smoking and a family history of cardiovascular disease (WHO, 1962: 19).

[23] According to Rothstein the population was largely of European descent, more than half of whom were of either Italian or Irish ancestry (Rothstein, 2003: 280).

The link between increased risk of heart disease and hypertension, raised blood cholesterol and smoking seemed relatively straightforward, but the relationship between diet and blood cholesterol was contested (Dawber, 1980: 141), and in turn the link between raised blood cholesterol and increased body weight was found not to be strong (Dawber, 1980: 129). Although men 20% or more above the median weight had double the risk of heart disease (Dawber, 1980: 146), this uncertainty about the relationship between raised blood cholesterol, diet and weight, gave overweight and obesity an uncertain status as a risk factor for heart disease. This uncertainty persisted in later publications where obesity is described as undesirable as it is associated with more important risk factors (DHSS, 1974: 11, RCP/BCS, 1976: 2).

The success of the Framingham researchers in identifying risk factors for heart disease led to the setting up of other large scale prospective epidemiological studies. In 1958 Ancel Keys of University of Minnesota, with support from the WHO and, once again, the NHI, set up The Seven Countries Study. Seven Countries developed from earlier prospective studies into the risk factors for heart disease among middle aged men, including a study of Minnesota business men begun in 1948 and two studies on the epidemiology of coronary heart disease that began in Italy and Spain in 1952 (Keys, 1980b: 1). The study focused on diet as a risk factor for cardio-vascular disease: building on his previous research, Keys and his collaborators hypothesised that 'differences among populations in the frequency of heart attacks and stroke would occur in some orderly relation to physical characteristics and lifestyle, particularly composition of the diet, and especially fats in the diet' (Blackburn, n.d.). The final version of the study included eighteen cohorts of men aged 40 to 59 of varied occupations living in rural and urban areas, from Finland, Greece, the Netherlands, Italy, Japan, the US and the former Yugoslavia. These areas were selected for a combination of pragmatic reasons, such as research contacts who were willing to collaborate at low cost, and dietary or epidemiological variability: Japan had very low rates of heart disease at the time and Finland very high ones, and Greece had high levels of consumption of olive oil (Keys, 1980b: 7).

Later high-profile studies that also adopted this new research method included the Nurses Health Study and the Multiple Risk Factor Intervention Trial (MRFIT). The Nurses Health Study was set up in 1976 by the NIH and the Harvard School of Public

Health, initially to investigate the health effects of long term usage of oral contraceptives, and is currently still running having recruited a new younger cohort of participants. MRFIT began in 1972. It was funded by the National Heart Blood and Lung Institute (previously the NHI) to test the effect of interventions on several different heart disease risk factors. This very costly study is widely agreed to have shown that the expensive treatments given to the 'special intervention' group had only a marginal effect on cardiovascular mortality rates. The organisers relied on participants presenting themselves at screening centres, and volunteers in such studies are invariably healthier than non-volunteers: 'Thus a study designed to lower mortality rates in a high-risk population used a sample with much lower-than-average mortality rates'(Rothstein, 2003: 309). Rothstein argues that this high profile failure was an example of a more general problem in clinical trials of risk factor behaviour changes as they often relied on atypical and highly motivated participants.

The increased amount of American research funding available in the 1950s and 1960s (Fye, 1996) meant that this first group of studies were organised by American research institutions. However, in the 1970s, European bodies, such as the WHO Europe Office, began to build on their involvement in the international projects to set up their own studies. The most famous of these was the North Karelia Project, a collaboration between the Finnish government, the Finnish Heart Association, the University of Kuopio and the WHO, which ran from 1972 to 1977. This project extended the methods of studies such as Seven Countries[24] by including investigations into the effects of interventions on key risk factors. An important component of this study was an attempt to reduce the prevalence of risk factors, and therefore cardio-vascular disease, in a population chosen because they had very high rates of those diseases. The risk factors identified were smoking, blood cholesterol levels (or diet) and hypertension. The project strategy was explicitly based on risk factors as the following except makes clear:

> 1. There is no natural limit between a normal and an abnormal risk factor level. The risk for coronary attack increases as the level of any risk factor increases. One feature of the Finnish and North Karelian population was that the average level of all risk factors was high (Rimpela et al. 1974). Practically none of the adult population had a "safe" level of all risk factors.

[24] North Karelia was one of the Finnish regions included in the Seven Countries study.

> For this reason, the intervention aimed at general changes of the risk factors among the population (i.e. changes in the distributions and means) instead of measures restricted to "high risk" groups (National Public Health Laboratory of Finland, 1981: 18).

The North Karelia Project is still routinely described as one of the very few successful health interventions in the area of cardiovascular disease.[25] Its outcome is summarised in the project report as follows: 'The estimated CHD risk reduction from all risk factors in North Karelia was 17.4% among men and 11.5% among women. The greater change among men is in accordance with project goals.' (National Public Health Laboratory of Finland, 1981: 303)

A final large-scale epidemiological study that falls directly in this tradition of research is the WHO MONICA project (MONitoring of trends and determinants in CArdiovascular diseases). MONICA was a European prestige project set up to rival Framingham: it was a self-conscious attempt by the European research community to conduct a large prospective epidemiological study into cardiovascular risk factors, modelled on the studies that had been running in the US since the 1950s. It began in 1978 and ran for 23 years, collecting data on 38 populations in 21 countries. Like North Karelia, MONICA had strong links with the WHO Regional Office for Europe in Copenhagen, although the project director was Professor Hugh Tunstall-Pedoe, a cardiologist based at Dundee University.[26] One commentator has described the establishment of the 'first global epidemiological study' as 'an amazing feat' and states that 'it is almost unimaginable that an enterprise of this scale will ever be conducted again' (McKee, 2003: 613). I will discuss MONICA at greater length in Chapter 8 as it was a very important source of data on increasing average body weights in European populations.

2.6 Risk factor research and causal explanation

Risk factor analysis represented a new paradigm in research into disease and ill-health, displacing laboratory investigations as the main mode of research and criterion

[25] It has even been regularly described as the only successful public health intervention aiming at reducing body weight, even though this was not one of the project's aims and there is no available data to demonstrate this success.
[26] One of the other members of the MONICA steering committee was Professor Jaakko Tuomilehto who was centrally involved in the North Karelia Project.

of proof. Its development in the UK in the 1950s has been described as a "paradigm shift" that led to the 'growing acceptability of epidemiological rather than the biomedical, laboratory-based mode of proof'(Berridge and Loughlin, 2006: 957). Partly, this was due to the exemplary status of Framingham: 'The probabilistic yet "hard" data produced by studies such as Framingham and their wide dissemination in a variety of clinical, public health, cardiology, and other journals...beginning in the mid-1950s played an important part in gaining visibility and acceptance for risk factor ideas' (Aronowitz, 1998: 121). Individual susceptibility to heart disease could still not be satisfactorily explained and Aronowitz argues that this is another reason for the spread of the risk factor approach:

> Risk factors provided a new scientifically rationalized framework for managing the increasing uncertainty associated with the occurrence of CHD by providing an overarching, consoling, meaning-giving framework. Risk factors provided a reassuring explanatory framework because they gave some sense of who was at greatest risk and what one might do to decrease risk. At the same time, risk factors embodied the cultural and medical ideals of precision, specificity and quantification (Aronowitz, 1998: 125).

Risk factor analysis, being based on the identification of statistical correlation, does not identify causes *at all* – merely associations. In particular, it does not throw any light on biological causes. Luc Berlivet outlines the use of epidemiological evidence in the 1950s debate on the links between smoking and lung cancer:

> In the absence of comprehensive and accurate laboratory explanations, the statistical-probablistic approach worked like a "black box" – the input i.e. smoking, and the output, i.e. cancer of the lung, were associated through a method that could not shed any light on the biological processes which lay behind (Berlivet, 2003: 52).

Moreover, a number of historians have noted that the identification of risk factors as causes has also tended to be distinctly selective. This warrants some explanation.

In principle, risk factor analysis is open to the possibility that multiple causes or predisposing factors – including social and environmental factors – may contribute to the production of disease and that such causes may interact holistically. This kind of analysis was apparent for instance in early life insurance industry work to identify risk factors. Prospective epidemiological studies such as Framingham borrowed the

concept of 'risk factor' from the life insurance industry which had been analysing information on the mortality rates of their policyholders in terms of 'risk factors' since the beginning of the twentieth century. Rothstein argues that

> Several basic differences existed between the old life insurance risk factor and the new medical risk factor popularized by the Framingham study. The life insurance risk factor was conceived in terms of a gradient of risk depending on its level, while medical risk factors were often dichotomized into healthy and unhealthy levels. Each life insurance risk factor was related to all other risk factors, while each of the new medical risk factors was considered separately. Last, the life insurance risk factor emphasized both the social and the medical characteristics of the applicant, while the medical risk factor was restricted to medical characteristics (Rothstein, 2003: 285).

However, Rothstein notes that subsequent risk factor research, from the Framingham study onwards, epidemiologists of heart disease have tended to write out social and environmental risk factors.

> Despite some promising early findings, they disregarded social characteristics such as education, income, occupation, living conditions, usual sources of health care, marital status, place of birth, and family structure. Yet social characteristics are as important as physiological ones in clinical decisions…The narrow focus of this pioneering study established an unfortunate precedent for most subsequent studies (Rothstein, 2003: 285).

How, and why, this occurred remains somewhat unclear, however.

Aronowitz attributes it in part to historical accident, resulting from the initial framing of the Framingham study. One of the important and novel features of Framingham was that it became a prospective study i.e. one looking for the factors that preceded the development of heart disease. As originally conceived, the aims of the study were far less ambitious than they subsequently became, and were oriented towards identifying risks in the sense of clinically useful measurements that could be used to predict the likelihood of heart disease in particular individuals (Aronowitz, 1998:120) – i.e. not concerned with larger social and environmental factors. Only subsequently was it decided to turn Framingham into a prospective study to identify risk factors more generally. And although the study period was extended beyond its initial duration of five to ten years for this purpose, the individualised perspective on risk factors was retained. Despite the fact that the methodological innovations making it

into a large scale cohort study relating exposure to outcome of chronic disease were later additions to the study, they were to be highly influential.

Additionally, Aronowitz identifies in the Framingham study a tendency towards biological reductionism, in the sense of a tendency to see risks as real only insofar as they can be explicated in terms of underlying biological causal processes

> But not every association can be expressed in risk factor terms. Putative risk factors need to meet certain conditions. They need to be measurable and specific characteristics of individuals in order to fit into the risk equations that express the results of epidemiological trials…Nonspecific and less individualistic variables, even if they could be measured and manipulated as if they were specific characteristics of the individual, have not been easily assimilated into mechanistic models of disease and mainstream clinical and public health approaches. Such variables generally lack a direct biological mechanism by which coronary artery pathology develops in the individual. Although knowledge about risk factors is almost entirely developed from epidemiological observation, risk factors have been understood – and legitimated – only as they contribute to the specific, localized pathophysiological processes that result in disease (Aronowitz, 1998: 132-3).

Indeed, identification of risk factors for heart disease commonly led to clinical and laboratory research aimed at identifying causal biological processes – e.g. Ancel Keys conducted research on the human physiology of cholesterol consumption, and Pekka Puska on the relationship between nutrition and hypertension, focusing on the effect of blood lipids.

At the same time, this biological understanding of risk factors facilitated their incorporation, at least in certain instances, into clinical medicine, and indeed to certain risk factors coming increasingly to be seen as diseases in their own right. Within clinical medicine, the acceptance of risk factor frameworks was less related to overall funding and more to individual doctors' economic and professional interests – the availability of specific tests (and payment for performing those tests), the availability of drug treatments, expert committee reports producing guidelines for best practice, and campaigns by governments and health groups promoting preventive health (Aronowitz, 1998: 127). A case in point is hypertension, long recognised as a risk factor in heart disease. With the advent of effective drugs to treat hypertension, clinicians began to pay greater attention to the condition. Initially, only those patients

showing unmistakable and acute symptoms were treated (Timmermann, 2006). But as the idea of risk factors became increasingly widespread within the medical profession and in policy circles, hypertension was redefined with the establishment of the concept of "malignant hypertension" not just a risk factor, but as itself an occult form of pathology, defined by guideline thresholds and made visible through routine surveillance with the sphygmomanometer (Timmermann, 2006: 245-6). Similarly, routine screening and treatment for hypercholestrolaemia in the US only developed in the 1980s, despite the medical profession having recognised its importance as a risk factor since the 1950s. This change resulted from campaigns by the National Institutes of Health and the American Heart Association who had developed consensus recommendations and treatment protocols, and pharmaceutical companies who had developed new drug therapies (Aronowitz, 1998: 127).

Ironically, this narrowing of the scope of what kinds of factors can be considered causal has actually weakened the explanatory power of the risk factor approach. As a number of epidemiologists have pointed out, risk factor analysis, as it has developed since Framingham, is only capable of explaining a certain proportion of variation in rates of heart disease. In the 1970s, one American epidemiologist argued that current risk factors were not capable of explaining the patterning of heart disease distribution by geography or gender (Winkelstein quoted Aronowitz, 1998:133). The disregarding of wider social and environmental causes of disease, such as the effects of bad housing, may be one reasons why the risk factor approach is acknowledged to be able to explain only a proportion of the variation of heart disease rates within populations (Rose and Marmot, 1981).

Berlivet suggests that this exclusionary approach to risk factors is deeply embedded in the methodological suppositions on which current risk factor research is founded, particularly the criteria for attributing causation that were articulated by the British statistician Austin Bradford Hill in the 1950s. At the end of his discussion of the development of ideas of causality in chronic disease epidemiology, he asks

> Could poverty be a "cause" of disease? Many, including several outstanding epidemiologists would agree; still, [Bradford] Hill's criteria [of causation] accommodate more easily tobacco smoke, infectious agents or chemical carcinogens than social and economic deprivation (Berlivet, 2003: 61).

But others attribute it to a systematically *political* orientation permeating biomedical and especially epidemiological thought. Bartley describes how problems which are 'too big to solve' (Bartley, 1992: 220) can end up being sidelined or ignored in the policy process. Due to their focus on a limited number of behavioural causes, risk factor approaches to heart disease (and other chronic diseases) are one way that health policymakers can avoid this problem of 'bigness'. Such approaches have the double advantage of seeming suitably scientific, but, at the same time, rendering problems of chronic disease more manageable in policy terms, because, in terms of both treatment and prevention, they allow the targeting of individual risk factors, often by manageable methods such as drug treatments or behaviour change.

These processes of defining diseases and their causes demonstrate ways in which 'medicine individualises disease and writes-out social deprivation and inequality from the description of illness' (Bartley, 1985: 292). Relating health inequalities to socio-economic class or race can operate a form of black-boxing that obscures the mechanisms creating these negative health effects.

> Epidemiological approaches to managing race, class and sex/gender distill the effects of *social* and *relational* ideological, structures and practices organised around such differences into characteristics of discrete and self-contained individuals……Epidemiology thereby renders invisible the very social relations of power structuring material and psychic conditions and life chances that contribute to the stratification of health and illness (Shim, 2002: 134).

This is a theme to which I will return throughout my thesis, by looking in particular at how the social has been omitted from explanations of eating habits and patterns.

2.7 Has there been an 'epidemic' of coronary heart disease in the UK?

More radically, Bartley (1985) has challenged aspects of the idea of the demographic transition itself, and specifically the idea that there was an increase in the incidence of coronary heart disease after the Second World War. Bartley's re-interpretation of mortality statistics, using arguments about changing disease classifications, 'throws considerable doubt on the idea of *one* clearly distinguishable and temporally located "modern epidemic of heart disease"' (Bartley, 1985: 293, italics in the original).

Instead she argues that the health of certain groups within the British population, notably middle-aged working class men, has ceased to improve in the last 50 years. Earlier epidemiologists were aware of differential declines in mortality rates but ascribed this to changing disease patterns:

> During the nineteenth century death rates in middle age in England and Wales were high…but about the turn of the century sanitary reform and the rise in the standard of living began to show results in this age group. Death rates began to fall, both in men and women, and they continued to fall until the early 1920's. Then rather abruptly, there was a change. Mortality in women kept its downward course; but the decline in male mortality slowed, and for the last twenty years has hovered around 22 per 1,000. A hundred years ago the death rate among middle aged men was about 15 per cent higher than in women, after the first world war it was about 33 per cent higher, now the male rate is twice the female.….[gains from advances in medical science have been counteracted by other changes]…The most important is that two diseases, affecting men in middle age far more than women, and highly lethal, have emerged from obscurity to become exceedingly common: "coronary thrombosis" or ischaemic heart disease, and cancer of the bronchus (Morris, 1964: 1-2).

Bartley argues that this apparent post-war rise in coronary heart disease was actually due to a reclassification of forms of disease that were already prevalent among middle-aged men. From the 1920s onwards a conceptual framework was developed by British and American doctors that understood hardening of the arteries, or arteriosclerosis, leading to heart failure as a major cause of death (Rothstein, 2003: 286-94). Bartley tracks the changing terminology used to describe these conditions and argues that in the 1930s, a large proportion of heart disease was still classified as 'degenerative' – that is, not caused by the blockage of coronary arteries. She combines the mortality rates for 'non-coronary'(degenerative) and 'coronary' heart disease to show that between 1921 and 1939 rates of heart disease in men 45 to 64 increased by 85%, and then by 12% to 1951 and by 20% to 1971 (Bartley, 1985: 300). This means that, contrary to narrative of a post-war increase in heart disease, the increase in the interwar period was greater than that of 1950s onwards, and, in the early post-war period there was continuing uncertainty as to whether rates for heart disease were still rising. From her re-reading of contemporary accounts Bartley argues that there is no 'consistent evidence to the effect that the coronary arteries of the nation deteriorated over the period we are examining, let alone that such deterioration was more prevalent in those being classified at death as cardiac victims'

(Bartley, 1985: 302). Her conclusion is that ultimately, 'we can take as our problem, not the "epidemic of heart disease", but rather, the failure of the health of men (particularly working class men) in later working life to improve appreciably in the last fifty years' (Bartley, 1985: 309).[27]

Such a conclusion runs counter to the conventional narrative of British chronic disease epidemiology, and especially those presented by public health policy textbooks and policy reports from the 1960s onwards. This is not, as far as I can judge, a widely accepted argument, even within Bartley's home discipline of medical sociology. However, such an analysis is important because it leads to questions about the distribution of the improvements in health between different sections of the population. In arguing that the high post-war rate of heart disease among middle-aged working class men is not new, but rather is continuous with the ill health they suffered between the wars, Bartley is arguing, in effect, that the health improvements enjoyed by the rest of the population since WWII have largely by-passed this group. And if that is the case, it raises questions about why this group, in particular, should have missed out on the benefits that others have enjoyed. Bartley does not offer an alternative explanation of this phenomenon. Any causal explanation would need to include social factors, since what is at issue is a differential distribution of health advantages and disadvantages across social groups. But, the risk factor approach, as it has developed since Framingham, is ill-suited to clarify the role of such factors, since it focuses on individual biological indicators of risk.

2.8 Conclusion

The idea of an epidemic of heart disease emerged in America and Britain during the years following World War 2. Faith in modern medicine and biomedical research accompanied by economic growth, led to significant increases in funding for health care and for medical research in the US, and to a lesser extent the UK, for the next 25 years. Since increasing levels of heart disease had become a focus of medical and political concern, some of this new funding was used to conduct research into its

[27] Aronowitz states that one of the reasons prospective studies such as Framingham were undertaken was the realisation that the positive effects of the epidemiological transition had by-passed the middle-aged (Aronowitz, 1998: 123).

causes. Using the new technique of the large scale prospective epidemiological study, this research programme developed and consolidated the risk factor approach to understanding heart disease. Such development included an increasing tendency to regard specific risk factors, for example hypertension and raised blood cholesterol, as diseases in their own right. However, the risk factor approach to heart disease was selective in the factors it considered as causes of disease. This meant that chronic disease epidemiology, and indeed even the assumption that heart disease was a peculiarly post-war public health problem, tended to obscure the social determinants of heart disease.

The following chapters will build on these arguments by looking specifically at the identification and definition of obesity as a risk factor for heart disease, then increasingly as a health problem in its own right. In so doing, these chapters will throw additional light on how the risk factor approach was articulated and disseminated after its initial pioneering formulation in Framingham. And ultimately, they will also lead to a further consideration of how that approach may have helped to obscure the social determinants of ill-health.

CHAPTER 3: THE DEVELOPMENT OF PUBLIC HEALTH COALITIONS 1950 - 1999

3.1 Introduction

The previous chapter describes the post-war growth of a major research programme into the causes of coronary heart disease (CHD), a condition which was seen as an important and growing public health problem. This was a crucial element in the more general re-focusing of medical attention towards chronic disease. The risk factor approach to chronic disease that developed out of this research programme meant that excess body weight became an object of increased medical interest. Excess body weight was seen as one of a group of newly identified risk factors for heart disease that also included smoking, hypertension, raised blood cholesterol, stress and physical inactivity. Its status was uncertain (see section 2.5) but the links between raised blood cholesterol, poor diet, excess body weight and increased risk of heart disease were sufficiently convincing to generate further research in these areas.

At the same time British nutrition researchers were switching focus away from under-nutrition and malnutrition and towards over-nutrition. A coalition of medical researchers and practitioners interested in the topic of obesity and overweight began to emerge in the late 1960s as nutrition scientists began to establish links with diabetes researchers and those interested in the links between diet and heart disease. At first this seems to have been a small offshoot of larger groups, but the success of the anti-smoking public health coalition provided a role model, and gradually the activities of the obesity science coalition expanded beyond their own professional organisations to include collaborations with government departments and bodies such as the Royal College of Physicians (RCP). In the course of this expansion scientific knowledge about increased body weight grew into a new understanding that I label the individual paradigm of obesity.

3.2 The development of an anti-smoking public health coalition in the UK

Smoking was the first of the newly identified risk factors around which a policy coalition developed. The framing of smoking as a medical and public health problem

began in the late 1950s (Berridge, 1999b, Berridge, 2003b, Berridge, 2005a, Berridge, 2006, Berridge, 2007b, Berridge and Loughlin, 2006). Virginia Berridge (2003b) provides a chronology of smoking policy in the UK, dividing it into four periods: the 1950s and 60s when the first epidemiological evidence of the link between smoking and lung cancer was published; the 1970s when the government relied on voluntary industry regulation in the face of increasing health activism; the 1980s when the discovery of passive smoking shifted understandings of smoking from an individual to an environmental issue; and the 1990s when a 'medicalized public health' focused on pharmaceutical treatments for nicotine addiction. In each of these eras, the policy situation was characterised by complex and overlapping networks of politicians, industry representatives, voluntary and professional organisations and expert government advisors.

In the first of these periods, Berridge argues that there was genuine uncertainty about the status of epidemiological evidence of the association between smoking and lung cancer. The results of the Doll and Hill study showed an association between smoking and lung cancer, and the authors argued that this meant that smoking was the most likely cause of lung cancer (Doll and Hill, 1950). Although this study is currently seen as a paradigmatic example of risk factor research, contemporary critics argued that 'correlation should not be taken as proof of causation' (Berridge, 2006: 1192, see also Parascandola, 2004). The nature of public health was changing in this period as it re-oriented toward chronic disease (Berridge, 2006: 1191). In the UK, this conceptual change occurred at the same time as an institutional change, as public health moved out of local authority control to become part of the NHS (Lewis, 1986). Such factors contributed to what Berridge labels a 'fluid policy situation', and although acceptance of the link between smoking and lung cancer was later seen as a crucial episode in chronic disease epidemiology (Susser, 1985a: 163), it was not until the 1960s that British government policy began to change: 'The establishment of this new epidemiological risk-focused way of explaining disease was a gradual process of what can be seen as "scientific claims building". It did not automatically lead to translation into policy.'(Berridge, 2003b: 65). One of the reasons for this slowness was that this approach to public health required new ways of addressing different sections of the public:

> Smoking was a difficult issue in terms of policymaking. It did not "fit" with what was traditionally considered appropriate as public health intervention. Much public health concern had been for the containment of epidemics of infectious, not chronic disease. Health advice about individual behaviour modification, where it was given, had usually been aimed at women and children rather than at men, yet the latter formed the majority of smokers in the 1950s (Berridge, 2003b: 68).

The personal smoking habits of ministers and their scientific advisors, the importance of tobacco revenues to the Treasury and the long term nature of anti-smoking advice (which would not produce results for thirty to forty years) also led to political delay and weak policy responses. In the 1950s, smoking was a widely accepted and normal activity, and this shaped its regulation. Smoking rates began to decline in the 1970s, from 51 per cent of men and 41 per cent of women in 1974 to 28 per cent of men and 26 per cent of women in 1998, and this has resulted in the 'increased marginalization of smoking and its gradual closer association with poorer groups in society, both men and women' (Berridge, 2003b: 63). This marginalisation made intervening in personal habits more acceptable, and the declining importance of tobacco revenues also meant that measures that would restrict consumption became more politically possible (Berridge, 1999b: 1188).

The 1962 Royal College of Physicians (RCP) report, *Smoking and Health,* marked the development of a public health policy community around smoking as medical practitioners and researchers began to adopt a new understanding of their role in providing the public and politicians with information. A policy community was developing and lobbying for change. This community consisted of representatives of chest medicine, cancer and epidemiology and was now based in the RCP, whose 1962 report had provided an agenda for action by this community:

> This was a new era in the role of scientific argument and medical lobby. For the first time, science was reaching out to the public and using the full panoply of marketing and consumer oriented techniques which were then emergent in a post-rationing society (Berridge, 2006: 1203 - 4).

Smoking became symbolic of a new role for medicine in explaining health risks and advising individuals on their management (Berridge, 2006: 1202). The 1962 report

was an important episode in the relationship between the medical profession and the government as it

> gave public significance to a new type of public health and to different scientific ways of studying it. The new epidemiology of the 1950s and the new focus on risk of chronic disease were translated into a wider public and policy agenda (Berridge, 2007b: 289).

A focus on preventive health and individual responsibility for avoiding health risks – an approach that has been labelled 'new public health'(Petersen and Lupton, 1996) – was becoming increasingly important in public health policy from the late 1960s onwards. In the 1950s and early 1960s, British public health was '"on the cusp", moving away from the mass campaign service-focused public health ethos of the inter-war years towards a new type of "healthism" epitomized by the concern about smoking'(Berridge, 2003b: 69). This shift was not fully apparent until the 1970s, but it originated in the 1950s epidemiological "paradigm-shift" exemplified by widespread acceptance of the link between smoking and lung cancer (Berridge, 2003b: 73).

Although the anti-smoking coalition had formed primarily around this link between smoking and lung cancer, it soon came also to focus on the role of smoking as risk factor for heart disease, which had been demonstrated by results from Framingham (Dawber, 1960). This link had been known of previously (Aronowitz, 1998: 97-8, Rothstein, 2003: 239) but the formation of anti-smoking collations in the UK (and the US) gave it special salience. In this manner, these coalitions contributed to the consolidation of the narrower or mono-causal approach to risk factors described in chapter 2.

In 1971, after the publication of the second Royal College of Physicians (RCP) report *Smoking and Health Now*, voluntary agreements between government and industry on regulation of advertising and sponsorship and the use of health warnings were drawn up. But throughout the 1970s, policy positions on smoking continued to diverge. An anti-smoking policy coalition developed around Action on Smoking and Health (ASH) and the Health Education Council, which differed from the harm reduction approach favoured by the tobacco industry. ASH had been formed in 1971, with funding from the Department of Health, as an external body that would put pressure

on the government to act on the issue (Berridge, 2003b: 73). Initially ASH focused on policy development and educating smokers, however under a new director in 1973, the organisation adopted a more high-profile campaigning style that used the media to create public awareness of the case against smoking. It was 'a new style of activism in health, drawing on models developed elsewhere in social campaigning' (Berridge, 2005a: 106). As Berridge points out, this gave ASH a dual status - it operated simultaneously as an external pressure group and as an insider member of the policy network – and it was useful to government ministers who wanted to take action on smoking (*ibid.*). Until the 1980s, British government policy on smoking was largely made in co-operation with the tobacco industry, paralleling the model used in regulating the pharmaceutical industry (Berridge, 1999b: 1187). This situation has been described as consisting of a 'producer network' of tobacco industry and retail interests with links to government, and an 'issue network' of outside public health professionals and anti-smoking organisations (Berridge, 2003b, Berridge, 2005a). However, an analysis of the work of ASH, the Independent Scientific Committee on Smoking and Health (ISCSH) and the Tobacco Products Research Trust (TPRT) shows that the networks in the area of smoking policy were more complex, and that public health scientists, particularly those on expert committees, had links with all three bodies (Berridge, 1999b: 1187, Berridge, 2005a: 117). In this period, links with industry were still normal, and not damaging to the credibility of advisors and policymakers.

However, the anti-smoking coalition was vehemently opposed to the harm reduction or 'safer smoking' approach preferred by industry. From the 1970s onwards, it campaigned for an abstentionist agenda based on public education, increased taxation and restrictions on smoking in public places (Berridge, 1999b: 1187-8). Such an approach was given increased legitimacy in 1981 by the publication of a study showing that the non-smoking wives of heavy smokers had a higher risk of lung cancer. There had been early evidence of harm to unborn children which led to anti-smoking campaigns targeting pregnant women, but this was the first evidence of smoking as harmful to the general population, rather than just the individual. The existence of 'innocent victims' such as women and children 'widened the debate and provided a more powerful engine for driving policy' (Berridge, 1999b: 1190). Again the evidence for re-defining the understanding of the risks associated with smoking

came from epidemiology, but it also relied on the development of biochemical markers that could be used to judge levels of exposure to tobacco smoke. Berridge describes the new science of passive smoking as 'science waiting to happen' because 'the policy objectives of the anti-smoking alliance had already begun to shift in the direction to which the scientific concept implied policy should go' (Berridge, 1999b: 1191). This new scientific fact led to a breakdown in the relationship between orthodox science and the tobacco industry and the entry of new actors such as the British Medical Association into the anti-smoking coalition.

> Passive smoking and the policies it helped initiate essentially combined the individualism of the 1970s public health paradigm with the environmentalism of the 'new public health', with its resonance with the image of nineteenth century public health. It was environmental individualism, an alliance of individual and environmental approaches, just as the science itself married different technical approaches into a reformulation of the original epidemiological case (Berridge, 1999b: 1192).

As I have outlined above, these processes ultimately led to a new set of relationships between the medical profession and government.

> The RCP report of 1962 was the forerunner of later College reports on smoking and a host of other health-related subjects, all of which were aimed at both the government and the public. The "medical voice" developed important relationships with both government and the public in areas that would not previously have been considered the province of either. In the 1970s this insider/outsider relationship for medicine developed further into a host of expert committees with close relationships within government (Berridge, 2007b: 310).

These relationships between the medical profession, the government and the public would also become important in the area of diet and health (see below).

3.3 The early years of the obesity coalition

Smoking was the first risk factor for heart disease around which a policy coalition formed, and in the next twenty years, researchers and practitioners working on the relationship between diet, nutrition and chronic disease used the successful creation of the anti-smoking policy community as a model for the development of a similar community around diet and heart disease.

> Smoking was the major issue which marked the redefinition of public health around lifestyle issues. The "new public health" policy programme focused on fiscal (taxation) and media strategies (advertising bans and mass media campaigns), with a new and distinctive role for "health activist" groups with a strong anti-industry stance, like ASH. This was a model of public health activism that was replicated in other areas, for example, diet and heart disease (Berridge, 2003b: 81).

Obesity and overweight were often listed as subsidiary risk factors for heart disease (see section 2.3 above) and the public health coalition around them appears to have developed out of a shared interest in the topic between researchers interested in diabetes, nutrition and heart disease modelled on the anti-smoking coalition, and partly overlapping with the diet and heart disease coalition. It started off as a much smaller off-shoot group, but its framing of increasing body weight as a major public health problem has been so successful that, by the beginning of the twenty first century, obesity had become a mainstream political issue (see chapter 10).

The late 1960s mark the start of this important period for British biomedical research into body weight and health. A burst of activity around the topic occurred between the late 1960s to the late 1970s and gave rise to a series of symposiums and conferences, a couple of reports and textbooks. The newly formed Obesity Association held its first symposium in October 1968 (Butterfield, 1969: 1). A 1969 textbook on the subject of obesity was described as the first 'comprehensive' treatment of the topic for more than a decade, whereas by the third edition the author was stating that 'more than a dozen books' had been published on the subject between 1973 and 1977 (Craddock, 1978: vii). The First International Conference on Obesity was held in 1975, and in the late 1970s another author referred to 'several major international conferences' on the topic in the previous four years and a 'recent explosion of literature in the field'(Garrow, 1978: 1). However, the topic of obesity was framed in a seemingly contradictory fashion: although the condition was widely accepted to be an important health problem, the consensus was that very little was known about it and more research was required:

> whilst the indictment against obesity appears to grow in strength each year enormous gaps in our knowledge of the subject remain. There is great uncertainty regarding both an appropriate definition and possible methods of

> measuring obesity and consequent lack of information on its prevalence in the United Kingdom. This paucity of fundamental data makes it extremely difficult to assess the precise role of obesity in disease patterns (OHE, 1969: 3).

This juxtaposition of great uncertainty about the definition, incidence and health consequences of obesity, with a clear sense that it is an important health problem, both for individuals and society, was typical of obesity science writing in this period. Despite its apparent contradictoriness, it allowed excess body weight to be presented as an important issue in health policy, whilst at the same time calling for more research funding in this area.

Participants in the early period of obesity science mainly came from two medical specialities - diabetes and human nutrition. Specialists from other areas, such as heart disease, also participated but they were heavily outnumbered by these two other groups (see table 3.1). Those who attended the first symposium organised by the Obesity Association in 1968 were largely clinicians and researchers from London universities and teaching hospitals. An interest in the topic brought together those interested in research into diabetes and its treatment, those studying human nutrition and pharmacologists investigating treatments for obesity. For many of these participants, obesity was interesting primarily as it related to other conditions rather than as a condition in its own right. The significant number of diabetes researchers in this group shows that it was another important area for the development of knowledge about obesity, since the link between overweight and type 2 diabetes had been known since the mid nineteenth century (Schwartz, 1986: 100) and many of these individuals were also present at a meeting of the British Diabetic Association in 1970 which included six presentations on the subject of obesity (BDA, 1970). The first President of the Obesity Association and chair of the symposium was Professor John Butterfield, whose central research interest was diabetes, including basic physiological research, epidemiological surveys and diagnostic criteria. He was later a member of the Committee on Medical Aspects of Food Policy and the World Health Organization's expert committee on diabetes. As an acknowledged expert in his own field, his reputation would have added to the legitimacy of this new professional body.

The nutrition researchers present at the OA symposium, such as Professor John Yudkin and Dr I. Howard Baird, began their careers researching deficiency diseases and the effects of malnutrition. In the immediate post-war period human nutrition research focused on protein malnutrition in developing countries. Because nutrition research in developed countries focused so strongly on animal nutrition, researchers interested in human nutrition had been forced to switch their interests to malnutrition in developing countries (James, 2006: 83). The identification of one form of malnutrition, kwashiorkor, in the mid 1930s, along with the food shortages associated with World War II, had also led to nutrition researchers shifting into the area of deficiency diseases in order to identify their causes and, therefore, preventive measures. According to another researcher in the area, protein malnutrition – 'the protein gap' - was seen as a major public health problem until the late 1960s (Payne, 2010).[28]

The second report *Research into Obesity* (henceforth the 1977 DHSS/MRC report) was produced by group from the Department of Health and Social Security and the Medical Research Council chaired by Professor John Waterlow of the London School of Hygiene and Tropical Medicine (LSHTM) who also worked in the area of

First symposium of Obesity Association, 1968	**MRC/DHSS Report, 1977**	**Royal College of Physicians report, 1983**
Dr Ian Mclean Baird West Middlesex Hospital, (human nutrition)	**Prof John Butterfield** University of Cambridge, (diabetes)	**Sir Douglas Black (chair)** Royal College of Physicians (health inequalities)
Prof John Butterfield (chair) University of Cambridge (diabetes)	**Dr J Durnin** University of Glasgow (physiology)	**Dr Gordon M Besser** St Bart's Hospital, London (diabetes)
Dr Alan Howard University of Cambridge (human nutrition)	**Dr John Garrow** MRC Clinical Research Centre, Harrow (human nutrition)	**Dr Charles Brook** Middlesex Hospital, London (paediatric endocrinology)
Professor Alan Keswick West Middlesex Hospital, (human nutrition)	**Professor Nick Hales** The Welsh National School of Medicine (diabetes)	**Dr Denis Craddock** GP/textbook author, (obesity)

[28] This author also referred to the 'great protein fiasco' when researchers based at LSTHM participated in a FAO/WHO consultation in the mid 1970s and successfully argued for a reduction in the accepted levels of protein requirements (Payne, 2010).

Dr June Lloyd University of London/Great Ormond St Hospital (paediatric endocrinology)	**Dorothy Hollingsworth** British Nutrition Foundation (nutrition policy)	**Dr John Garrow** MRC Clinical Research Centre, Harrow (human nutrition)
Dr Derek Miller University of London (food history)	**Dr WPT (Philip) James** MRC Dunn Nutrition Unit Cambridge (human nutrition)	**Dr Derek Hockaday** Radcliffe Infirmary, Oxford (diabetes)
Dr Galwan Pawan West Middlesex Hospital, London (human nutrition)	**Dr June Lloyd** University of London/Great Ormond St Hospital (paediatric endocrinology)	**Dr WPT (Philip) James** MRC Dunn Nutrition Unit Cambridge (human nutrition)
Dr Trevor Silverstone St Batholomew's Hospital, (pharmacology)	**Prof John Waterlow (chair)** London School of Hygiene and Tropical Medicine (human nutrition)	**Dr Barry Lewis** St Thomas's Medical School, London (heart disease)
Dr Steve Szanto University of London (human nutrition)		**Dr J L Mann** University of Oxford (diabetes)
Dr Paul Turner St Batholomew's Hospital, London (pharmacology)		**Dr Derek Miller** University of London (food history)
Professor John Yudkin University of London (human nutrition)		**Dr TRE Pilkington** St George's Hospital Medical School, London (diabetes)
		Dr Trevor Silverstone St Batholomew's Hospital, London (pharmacology)

Table 3.1 Committee members for three early obesity reports showing their institutional affiliations and research areas.

malnutrition.[29] His research focused on protein turnover, nitrogen balance and childhood malnutrition as a public health problem. In 1956 he had established the Tropical Metabolism Research Unit (TMRU) at the University of the West Indies in Jamaica to study malnourished children, identifying that they were suffering from kwashiorkor which resulted from infective conditions such as diarrhoea and developing a treatment programme (Millward, 2010). Dr (later Professor) W.P.T. (Philip) James who compiled the DHSS/MRC report, and was at that time based at the MRC Dunn Nutrition Unit in Cambridge, had also worked on malnutrition at the TMRU in the early 1960s, but by the 1970s had become interested in coronary heart

[29] Information about the careers of individuals provided in this section comes from the bibliography of obesity (Whelan and Silverstone, 1974), PubMed/Medline and *The Lancet.*

disease and obesity. Dr John Garrow was another nutrition researcher who worked at the TMRU in the 1950s and moved into the study of overweight and obesity in the 1960s. By the late 1960s he was working at the MRC Clinical Research Centre, Harrow and he ultimately became a Professor of Human Nutrition at the University of London, as well as consulting at several London teaching hospitals. He wrote on a wide range of topics around human nutrition and dietetics, including measuring obesity/body composition/adiposity, energy expenditure and the effects of diet and exercise, including text books such as *Energy balance and obesity in man* (Garrow 1978*), Treat Obesity Seriously: A Clinical Manual* (Garrow, 1981), *Obesity and related diseases* (Garrow, 1988) and *Human nutrition and dietetics* (Garrow and James, 1993). In the UK he became a prominent advocate of both the energy balance model and the BMI as a means of measuring and classifying overweight and obesity (see chapters 4 to 6). Another notable participant was Dr J. Durnin from the Department of Physiology at Glasgow University whose research areas were physical activity, energy expenditure, dietary intake and body fat measurements. In 1971 Durnin had been part of an FAO/WHO ad hoc committee of experts on energy and protein, and in 1987 he took part in a consultation on chronic energy deficiency with Philip James and John Waterlow. From the outset, therefore, members of the emerging obesity science coalition already had a well developed professional network as well as links with UN bodies such as the FAO and the WHO. Despite the shift in focus towards over-nutrition, there was continuing research and policy interest in under-nutrition.

As I have outlined, by the late 1960s there appears to have been a major shift in British nutrition science as researchers shifted their focus towards problems associated with over-nutrition, such as heart disease and overweight. One explanation for this shift may be that whilst it was acceptable to study malnutrition in poorer countries, the growing prosperity of the post-war economic boom made it unfashionable to study malnutrition in rich industrialised countries. From the early days of their discipline, nutrition researchers have become involved in the framing of policy and debates on issues such as wage levels, rationing requirements and the relationship between poverty and malnutrition (Aronson, 1982, Mayhew, 1988). Nutrition researchers were often highly entrepreneurial scientists, who sought to use their expertise in outside arenas. It is possible that as malnutrition became a less

fashionable research topic, researchers based in developed countries saw obesity as an area, with potential policy relevance, which they could claim as their own.

The DHSS/MRC report was a step up the institutional ladder for the obesity coalition. Their early activities consisted of establishing professional organisations, organising meetings and publishing collections of research articles and textbooks. However, the 1977 report was the first time they had collaborated with a government department, and this marks an important step in the acceptance of their arguments that obesity was a significant public health problem. The increasing profile of obesity research was further enhanced when, in 1983, a working party of the Royal College of Physicians chaired by Sir Douglas Black produced the report, *Obesity.* Lord Black, who was president of the organisation at the time, had produced the famous 1979 report into health inequalities, which contained substantial discussions on heart disease and the role of lifestyle factors in its high rates. Controversy around the newly elected Conservative government's attempt to suppress this report had made Sir Douglas Black one of the most high profile medical experts of the period and his involvement, as president of the RCP, must have enhanced the visibility of obesity science. But this recognition appears to have been short-lived as there were no more reports on obesity until the mid 1990s.

The honorary secretary of the RCP report was Philip James, and another member was Professor Barry Lewis, a high profile researcher on coronary heart disease, who went on to be chair of the International Task Force for Prevention of Coronary Heart Disease at its foundation in 1987. At this time, Philip James was also involved in the Coronary Prevention Group (CPG). The CPG aimed to operate in the style of a campaigning pressure group, such as ASH, and had been involved in the production of a well publicised report on policies for the prevention of heart disease, known as the Canterbury Report (Rose et al., 1984). Thus there was still overlap between obesity science and the wider field of coronary heart disease research. However, a coalition of researchers interested in obesity, hitherto regarded as one of the lesser risk factors for heart disease, was beginning to establish the field of obesity science as separate from mainstream heart disease research through strategic alliances with other groups of researchers and with bodies such as the MRC and the RCP.

Policy making in the area of diet and heart disease showed many parallels with that in the area of smoking, including the problems of dealing with scientific uncertainty and the deployment of this new medical voice, particularly in the form of advice from expert committees. The gradual acceptance of the link between high fat diets and increased rates of heart disease by the Committee on Medical and Nutritional Aspects of Food (COMA) demonstrates these processes. The committee's panel of diet and heart disease moved from complete lack of agreement consensus in 1974 (resulting in no report) to consensus in the 1984 and 1994 reports which advised individuals to reduce their average total fat consumption by 10 per cent, as well as reducing their consumption of sugar, salt and alcohol (Bufton and Berridge, 2000).

3.4 The coalition's initial model of obesity: the individual paradigm

In this section I will outline the main features of what I label the individual paradigm of obesity. This paradigm was an emerging set of assumptions and claims about obesity that circulated, largely unchallenged, within the obesity coalition. It developed alongside the institutions of early obesity science: the formation of the obesity coalition and the articulation of the individual paradigm were effectively two halves of the same coin.

This model of the condition largely defined it and measured it using the tables of ideal weight produced by the Metropolitan Life Insurance Company (see chapter 2). It relied on small-scale surveys to estimate the condition's prevalence, and saw excess body weight as largely caused by overeating, best treated by weight loss diets and associated with heart disease, diabetes and osteoarthritis.

3.4.1 Defining and measuring obesity

In this early period of obesity science, defining obesity objectively was often seen as difficult: 'Many clinicians have argued that the definition of obesity must remain subjective' (OHE, 1969: 4). This led to problems for research: 'the great paucity of studies to establish the incidence of obesity probably almost as much reflect the problem of definition as the early lack of concern with the condition' (OHE, 1969: 7).

This persistent problem of definition was related to understandings of the causes of obesity, as it was not yet understood as a unitary entity:

> Obesity is neither one condition nor one disease. The fat baby, the fat adolescent girl, the woman who gets fatter after each pregnancy, the traditional example of the fat business executive – these all have fatness in common, but it is very doubtful if the aetiology and natural history are the same in all of them (DHSS/MRC, 1976: 1).

However, despite this uncertainty, in order to frame it as a public health issue, it was strategic to provide an appropriate numerical measure so that the incidence of obesity could be measured. Throughout this period, statistical methods were being increasingly used within medicine (Rothstein, 2003) and numerical indices were becoming important in the framing of convincing arguments for the existence of a health problem. However, competing definitions and measures of obesity, some of which were difficult to convey concisely, made this difficult for early obesity science writers.

Often obesity was defined using ideal weight tables produced by the American insurance industry. These were based on US data from 26 large companies collected over 20 years from nearly 5 million people (OHE, 1969 : 4 - 5). In particular, the Metropolitan Life Company used relative weight[30] to produce a range of categories: average weight, overweight (greater than 110% of average weight), and obese (greater than 120% of average weight). These weight ranges were then incorporated into charts including height and frame sizes and correlated with mortality data to give ranges of "ideal weights" – for a 5 foot 6 inch woman the desirable weight range was 124 to 156 pounds, depending on whether they were small, medium or large framed (*ibid.*). However, the link between 'above average weight' and being 'too heavy' was seen as unclear and methods such as under-water weighing and measurements of skin fold thickness had to be used to determine total body fat (OHE, 1969: 4). To counter accusation of the narrowness of these ranges, another author argued that 10% excess weight 'allows a 76kg (168lb) man to accumulate 7.5kg (16lb) of fat without being obese!' (Craddock, 1978: 2). Worrying about the stringency of recommended weight ranges was not a feature that recurred in these sources.

[30] This is weight relative to height.

In addition to the widely used Metropolitan Life tables, there were several other definitions of obesity used in these texts. One author defined obesity as weighing 10% above 'normal' or 'desirable' weight[31], and 'excessive' obesity as weighing 20% above the desirable weight, but mentioned others using 30% as an appropriate figure (Craddock, 1969: 2 - 3). Another referred to Seltzer's 'ponderal index'[32] which was proposed in 1966 but does not seem to have become widely used, but also used tables from Framingham that framed risks of heart disease in terms of relative weight (Baird, 1969: 17-19). Nearly ten years after the OHE report, a later textbook was still using an approach derived from insurance company tables:

> For practical clinical purposes it is convenient to take the range of "desirable weight" from life insurance experience, from the lower end of the small frame to the upper end of the large frame (since frame size is undefined), and accept that people above this weight are obese (Garrow, 1978: 149).

Garrow also referred to body mass index (BMI)[33] as an alternative formulation of an acceptable weight range, implying that it was known about, but had not yet become the standard index.

The 1977 DHSS/MRC report is the first to make a distinction between two different ways of measuring obesity – relative weight and numerical indexes, such as W/H^2. The latter express the relationship between weight and height and are more absolute measures because they do not refer to population averages. A fundamental problem with relative weight measures using insurance company data was that the selection of reference standard was arbitrary, since it was based on a particular and unrepresentative sub-group of the population (DHSS/MRC, 1976: 3 - 4). The authors did discuss W/H^2 in this report, but in the context of the Metropolitan Life Company ideal weights: 'A cut-off point often used for separating obese from non-obese is a relative weight 120% of the "desirable" weight. This corresponds to values for W/H^2 of 27.5 for men and 27.0 for women of medium frame size and 29.9 for men and 29.5 for women of large frame size' (DHSS/MRC, 1976: 4). The main definition of obesity

[31]However, he admitted that others used a figure of 15% or 20% above desirable weight.
[32] This is defined as the ratio of the height in inches divided by the cube root of the weight in pounds – one of a number of precursors to the BMI (see chapter 4).
[33] Body mass index equals weight divided by height squared or W/H^2.

was based on percentage over desirable weights from the Metropolitan Life Insurance Company tables. Other methods of measurement were being discussed, but there were several competing alternative measures rather than one agreed alternative to desirable weights.

3.4.2 Estimating the prevalence of obesity in the population

At this time there were no large scale studies to give an accurate picture of the prevalence of obesity within the British population, and such health statistics were not yet collected by government departments. Researchers seeking to frame obesity as a public health problem were, therefore, forced to refer to the American insurance company data mentioned above, and extrapolate from the small number of British surveys that had been conducted. These authors strategically used the available information to argue that a problem existed, while at the same time pointing out the inadequacy of this evidence, and thus stressing the need for further research.

To illustrate the incidence of obesity, one author referred to Metropolitan Life data demonstrating that one in five men and one in four women aged 20 or over was at least 10% over the average weight, and one in twenty and one in nine at least 20% over (OHE, 1969: 7). American data were cited because 'No comparable study has been undertaken in this country, and we are therefore left with less precise data on which to base an assessment of the prevalence of obesity' (*ibid.*). However, the results of various smaller studies 'would seem to indicate a picture similar to that found in America with about one in five adults considered to be clinically obese' (OHE, 1969: 10).

A 1969 textbook referred to only three small scale UK studies of the weights of general practice patients (Craddock, 1969: 3 - 5), but a later edition, although mentioning a lack of national data, was also able to cite a 1968 study of 11000 BP workers, and two studies of London patients. Using this new data, the author concluded that 'it is likely that up to one half of the women over 30 in Great Britain are at least 10 per cent overweight and a similar proportion of men' (Craddock, 1978: 4). An earlier author states that the incidence of obesity in Britain was not accurately known but, after citing the same studies, went on to argue that 'at least half the

middle-aged persons in this country are overweight' (Baird, 1969: 15-16). The DHSS/MRC report referred to the same studies as the other sources, but came to a different conclusion, arguing that 'at all ages and in both sexes, there has been a small increase [in the prevalence of obesity] since the war time surveys of 1943' (DHSS/MRC, 1976: 9). This conclusion was strangely at odds with the sense of urgency conveyed by other sections of the report (see section 3.6 below).

3.4.3 The health consequences of obesity

In the 1960s the relationship between obesity and mortality was still contentious, and the case had to be made for a causal relationship (OHE, 1969: 15). The results of the 1959 Build and Blood Pressure Study[34] showed that mortality amongst men 10% above average weight was 20% higher than that of those of average weight. However, it was possible that 'obesity does not lead to the complications which cause death but is simply a consequence following from the significant disease. If this is so, reduction in weight would not influence survival' (OHE, 1969: 17). This author also cited evidence from the same study to show that weight reduction lowered mortality suggesting that there is a causal relationship. Such discussions further demonstrate the heavy reliance on statistics from life insurance companies, despite the acknowledged problems with their generalisability, since the insured population was generally more affluent and in better health than the general population (see also Baird, 1969: 16, DHSS/MRC, 1976: 20).

Until the 1980s, the list of negative health consequences given in these sources was relatively short and based on understandings of the relationship between obesity, heart disease (which was still controversial), diabetes and mechanical problems such as joint pain and breathing difficulties. One such list split the consequences into the mechanical - effects on joints, obstruction of breathing, hernia and varicose veins - and the metabolic - diabetes, raised cholesterol, gallstones and atheroma[35] and CHD (OHE, 1969: 15 - 17). Another author listed the health consequences as diabetes, coronary artery disease, osteoarthritis, hypertension (although this is still not definite)

[34] This study pooled data on 4.9 million policy holders from 26 life insurance companies in Canada and the US, and one author argues that Metropolitan Life used it to produce a new table of reduced recommended weight ranges in 1960 (Glaesser, 2002: 46).
[35] Atheroma is a disease resulting from fatty deposits in the linings of the arteries.

and varicose veins (Butterfield, 1969: 4-5). Later on the same author used early Framingham data to argue that obesity was also associated with an increased risk of heart disease (although the relationship was not clear and several possible mechanisms were proposed), angina, impaired pulmonary function and diabetes (Baird, 1969: 17-21).

In the 1970s, heart disease was becoming seen as an increasingly important health consequence of obesity. However, the attribution of a causal role for obesity was described as 'controversial' (DHSS/MRC, 1976: 22), due to contradictory data from two of the large scale epidemiological studies: results from Framingham showed that reduced weight led to a reduced risk of heart disease, whereas those from the Seven Countries study appeared to show no effect.[36] Other negative consequences highlighted at this time included hypertension, diabetes, damage to weight bearing joints, obstetric and post-operative complications and reduced physical fitness (DHSS/MRC, 1976: 22 - 25). By the late 1970s, authors quoted Framingham researchers arguing that 'If everyone were at optimum weight we would have 25 per cent less coronary heart disease and 35 per cent less congestive heart failure and brain infarction' (quoted in Craddock, 1978: 6). Moreover, Craddock's 1978 discussion of the health consequence of obesity was more detailed than in 1969 when it relied heavily on 1923/4 mortality data from Metropolitan Life, and he divided the 'complications, hazards and disadvantages of obesity' into three categories – increased mortality, increased morbidity, and physical discomforts and disadvantages. The increased mortality derived from increased rates of cardiovascular, respiratory insufficiency, diabetes, gall stones and liver damage (Craddock, 1978: 6 - 8). The evidence for these increased risks came from Framingham, Metropolitan Life Company and three other named studies. Examples of morbidity cited included hernias, colon cancer, diverticular disease, arterial disease, haemorrhoids, varicose veins, thrombophlebitis[37] and complications of pregnancy (Craddock, 1978: 8).

[36] The authors of the DHSS/MRC report argued that these apparent contradictory results were due to the difference in the two study populations - the Framingham study population was aged from 30 to 59 whereas Seven Countries one was aged 40 to 59. As the effects of obesity on mortality were more marked among the young, it would be expected that by not studying this age group the Seven Countries Study would be less likely to show a correlation between obesity and heart disease mortality (DHSS/MRC, 1976: 23).

[37] Diverticular disease is another name for diverticulitis or inflammation of diverticula in the intestinal tract and thromboplebitis is inflammation of a vein associated with the formation of a blood clot.

In his textbook of the same year, Garrow also used data from early large scale epidemiological studies. He lists increased risk of coronary heart disease as the main mortality risk and diabetes, hypertension, gall bladder disease, plus endometrial cancer, osteoarthritis of the knees, risks to mother and child in pregnancy and poor lung function as associated morbidities using data from American insurance companies, a Finnish study of 5000 insured men, the Seven Countries study and a study of 73, 522 members of an American slimming club known as TOPS[38] (Garrow, 1978: 146 - 152). As prominent researchers in the field, Garrow and the committee producing the DHSS/MRC report, would be expected to include recently published data from prestigious research, such as the large scale American epidemiological studies. These novel sources of information were beginning to be cited, but they would not lead to an expanded understanding of the health consequences of obesity until later reports in the 1990s (see chapter 7).

3.4.4 The causes of obesity

These authors largely framed the causes of obesity in individual terms. There was a partial acknowledgement of the social factors involved in eating behaviour and body size: occasionally, these discussions were even framed in terms of social class or gender (see below). However, the most commonly cited and best described causes were psychological or behavioural ones. These included a tendency for the obese to eat in response to external cues such as time of day or anxiety, rather than internal cues of hunger; their greater preference for sweet foods, and the relationship of obesity to conditions such as neuroticism and depression. As well as containing an appealing element of common sense, these explanations also fitted well with individualised, biomedical accounts of overweight and obesity. They drew on psychiatry, a discipline with a long standing interest in eating disorder that had become part of orthodox medical practice and research by the early twentieth century,

[38] TOPS stands for Take Pounds Off Sensibly. This study involved analysing questionnaires asking for information about diagnoses of particular conditions such as diabetes, hypertension, heart disease and anaemia and relating this information to degree of overweight. The most overweight women were found to have significantly higher rates of diabetes, high blood pressure and gall bladder disease (Garrow, 1978: 150 -1).

unlike social sciences such as sociology or anthropology which have also analysed eating practices in considerable depth (see chapter 6).[39]

In discussions of the causes of obesity, there were further attempts to sort out whether there was more than one kind of obesity. The majority of the overweight were thought to suffer from 'simple obesity' caused by eating too much and insufficient physical activity, rather than obesity resulting from psychiatric 'abnormalities', genetic factors or damage to the hypothalamus (demonstrated in rats to lead to obesity) (OHE, 1969: 11). Although Silverstone saw obesity as a normal response to ageing:

> Most people tend to put on weight as they grow older (due to a reduction of energy expenditure without a concomitant reduction in calorie intake), hence the increase in the prevalence of obesity with age, but against this there are considerable social pressures acting on individuals in the upper social classes to take remedial action – that is to diet – whereas such social pressures are probably much weaker among lower social classes, hence the increased prevalence of obesity in this group (Silverstone, 1969: 47).

He labelled this 'maturity-onset obesity' and separated it from 'early-onset, neurotic obesity' for which the causes included eating to manage anxiety, disturbed body image and disassociation between eating and physiological hunger (Silverstone, 1969: 48 - 51).[40] This categorisation of obesity as an eating disorder is illustrated by the way textbooks at the time (e.g. Bruch, 1974) routinely dealt with both topics.[41] In his summary of the psychological aspects of obesity Craddock argued that,

> The majority of obese people probably have no obvious psychological factors affecting their tendency to obesity. Many of them are people who eat under the minor stresses of everyday life instead of, or as well as smoking, drinking alcohol or tea or biting their nails. They are often hypersensitive individuals whose reaction to life is passive rather than active, they have a genetic

[39] The DHSS/MRC report was unusual in that it stated that information on the 'social factors' involved in the development of obesity was 'limited' and argued for more collaborative research into eating patterns involving nutritionists and social scientists (DHSS/MRC, 1976: 65). In a more standard mode, Butterfield discusses the use of 'psychosocial workers' to help with communication with patients and work with teenagers and the middle aged to prevent the development of obesity (Butterfield, 1969: 8-9).

[40] He also has a third category of reactive obesity – those who become obese in later life due to severe stress (Silverstone, 1969: 54) – which was understanding obesity as an eating disorder with an identifiable psychological or psychiatric cause.

[41] The boundary between obesity and eating disorders is fluid and has shifted in the last thirty years, partly due to different groups of clinical practitioners and biomedical scientists claiming the subject as their own.

> tendency to put on weight easily and they have been conditioned to eating as reward since early childhood. They therefore eat when under stress, whereas most people are less hungry and lose weight when under stress owing to increased adrenalin production which mobilises fat and glucose. Many of them do not bother to consult their doctors about the problem of overweight for a variety of reasons. Many of them are not distressed physically by their overweight and are not worried enough about their looks to consider the discipline of serious dieting. Many of them appear to overeat instead of getting depressed or neurotic and this is often a reasonable compromise for them. Those who express a neurosis by over-eating instead of in other ways usually have difficulty in losing weight (Craddock, 1969: 100).

This excerpt may reflect his background in general practice, where he would see fewer seriously overweight patients, rather than hospital medicine. The matter-of-fact description of the causes of obesity, and many individuals' acceptance of excess body weight was not found in other writing of the period, and is not found in contemporary writing on the topic. But his account of the psychological aspects of obesity, focusing on patterns of compensatory and compulsive eating, was very standard.

Anticipating later concerns, one author argued that: 'The problem of inadequate exercise is probably a growing one. The development of a prosperous industrial society means that many of the essential physical activities of life have been removed'(OHE, 1969: 13). Another made the point in more detail:

> the increasing prevalence of obesity in Western Societies can be attributed to under-exercise and over-eating. It is emerging that the former is really more important than the latter, and that, as a race, we have inherited appetites commensurate with unmechanised agricultural communities. We have not yet adapted to the luxury of steady supplies of calories in our markets all year round. Nor have we mastered the inevitable laziness, in terms of muscular effort, which follows the exploitation of the world's energy supplies (Butterfield, 1969: 3).

This thread of nostalgia for an idealised healthier past remained important in obesity science writing (see chapter 8). However, Garrow criticised arguments that obesity is caused by physical inactivity since no studies have been able to find significant differences between the levels of physical activity of obese and lean subjects (Garrow, 1978: 105). There is a tension in this writing between assertions that can be backed up by research evidence, and those, like the statements above, which are more general

and tap into fears about the effects of modernisation and industrialisation (see chapters 8 and 9).

A common approach of these authors was to discuss the causes of obesity in terms of genetic, environmental factors and finally social factors: 'Obesity is a product of plenty, but social factors play a big part in determining the relative influence in differing groupings' (Craddock, 1969: 22). Under the heading of social factors topics as varied as social class, national habits, eating habits, smoking and pressures affecting women were noted in different combinations by different authors. The 'social factors in obesity' could also include food consumption, eating patterns, physical activity (obese people show reduced physical activity, but it cannot be established whether this is cause or effect), social class, individual attitudes and psychiatric and behavioural aspects (DHSS/MRC, 1976: 13 - 18). The causes of obesity were coming to be labelled as complex and multi-factorial, and this rapidly became conventional wisdom in obesity science

> It seems probable that in the great majority of cases the cause of obesity is multifactorial: its onset is determined by a combination of genetic, psychological, metabolic and endocrine factors, and we are unable to disentangle the relative importance of each component. These are the cases sometimes referred to as "simple" or "idiopathic" obesity because there is no obvious cause (DHSS/MRC, 1976: 2).

Describing obesity as a complex and multi-factorial condition put it in the same category as many chronic diseases, notably heart disease, treating it as condition in its own right, rather than merely a risk factor for other conditions. It also implied that more research would be required in order to fully understand these causes, and that it might be a difficult condition to treat. This in turn gave status to the professionals whose research sought to understand these causes and whose clinical practice attempted to treat the obese.

3.4.5 Treatments for obesity

Between the 1950s and the 1980s, the standard treatments for obesity were restricted diet (of various types although the basic elements are fairly stable), exercise (with reservations about its effectiveness), behavioural therapy and drugs. Surgery was a

last resort treatment, reserved for severely overweight patients who have failed to lose weight by other means. These treatments were largely based on existing, clinical knowledge about the efficacy of such techniques:

> There are virtually no published trials of different therapeutic regimes in properly matched groups of obese patients studied over a period of long enough for at least some of the patients to achieve normal weight. The proportion of people in the community who, by their own endeavours, reduce weight to normal and then maintain weight by conscious dieting remains unknown. In hospital practice, however, it is evident that all forms of medical treatment for gross obesity have a low "cure" rate; long term follow up shows that many of those who have achieved normal weight by slimming relapse (DHSS/MRC, 1976: 57).

Not only had many of these techniques not been formally trialled, but even the best recognised treatment, weight-loss diets, was accepted to be ineffective in many cases.[42] This was not a new or uncommon understanding: 'Despite the availability of several useful dietary aids, the long-term treatment of obesity can best be achieved by a re-education of the patient in his dietary habits' (Howard, 1969: 109) – that is, not by short-term restrictive diets. Despite this knowledge a reduced calorie diet remained the main treatment recommended in all of these texts: the 'obvious' and common sense cure for obesity was to reduce the amount of calories consumed, and a common recommendation is for a diet of 1000 calories a day (OHE, 1969: 21). Restriction of food intake to reduce weight was seen to be the method favoured by doctors and the general public. Presumably, as a GP, Craddock would have been familiar with dietary treatments, and he outlined a range of options, including low carbohydrate, high fat or high protein and calorie controlled versions, various type of meal replacements and substitute foods, and he gave detailed diet sheets for nine different diets that included daily meal plans detailing appropriate portion sizes for particular food items (Craddock, 1969). In the short term, weight loss was seen as a matter of the overweight individual finding sufficient motivation and a palatable diet. Different macronutrient compositions were possible and Yudkin's high protein, low fat and low carbohydrate diet fitted the criteria that a diet should be 'calorically reduced, nutritionally adequate, socially acceptable, economically feasible, and potentially

[42] This was not an isolated finding (for a discussion of the topic see Ernsberger and Haskew, 1987 and Campos 2004).

permanent' (Yudkin, 1969: 92). Yudkin argued that a low fat diet was unpalatable and not satisfying to most people, and so his low-carbohydrate diet was 'a most effective way of losing excessive weight and losing it permanently' (Yudkin, 1969: 94). Other options discussed included starvation therapy and high protein low calorie diets (Howard, 1969: 96 - 105).

At first the role of exercise was seen as problematic since increased activity can increase appetite and caloric requirements:

> There is an obvious relationship between reduction of caloric intake and an increase in calorie expenditure resulting from increased activity. Unfortunately whilst both activity and resultant heat loss owing to muscular inefficiency dissipate body energy, they also lead to increase in both caloric requirements and appetite (OHE, 1969: 22).

Craddock, on the other hand, argued that the role of exercise had been underestimated 'because of theories as to the amount of exercise needed to "work off" a certain weight of fat, which have not taken into account the increased metabolic rate produced by exercise' (Craddock, 1969: 92). Therefore, he recommended that exercise should be part of every treatment programme unless medically contra-indicated. Yudkin also stated that an extra 30 minutes walking a day (with no increase in food intake) would lead to weight loss of about a pound a month (Yudkin, 1969: 92). He also rejected the argument that increased levels of physical activity led to increased appetite and therefore food intake. For these reasons he favoured his patients undertaking regular frequent 'mild activity'. However he admitted that most patients rejected this advice and bemoans that 'Much in our affluent lives is directed to reducing our activity rather than to increasing it – not only motor cars and television and washing machines, but plastic surfaces, non-polish floors, cars with automatic gears and even electric toothbrushes'(*ibid.*).

The author of the OHE report admitted that 'most efforts to reduce weight, whether or not taken under the supervision of a doctor are unsuccessful' (OHE, 1969: 24). Such low levels of success often led to the prescription (and abuse) of drugs, such as amphetamines, about which the BMA was becoming more cautious at this time (OHE, 1969: 25). Drug treatments at that time fell into two categories – bulking

agents (which were not recommended) and appetite suppressant drugs such as amphetamines, fenfluaramine and diguanides which were used to treat diabetes, but caused loss of appetite in both diabetics and non-diabetics (Turner, 1969: 112 - 4). The accepted approach was that drugs should only be prescribed when dieting had been first tried and that they should be used to improve adherence to a dietary regime, rather than as a weight loss method on their own. Another author divided the drugs used for weight reduction into five categories – anorectics, oral hypoglycaemic agents, metabolic stimulators, laxative and diuretics – and of these recommended only the anorectic drugs (Craddock, 1969: 70).[43]

Behavioural therapy was occasionally discussed as another way of ensuring that patients adhered to restrictive diets (DHSS/MRC, 1976: 60, Garrow, 1978: 172 - 3). Garrow hoped that 'a means of permanently rendering obese patients more amenable to dietary restriction will be found, but no sure method of achieving this objective has been published so far' (Garrow, 1978: 173). The best treatment results were thought to occur when the individual followed a low calorie diet, and was seen and weighed regularly by the same person (DHSS/MRC, 1976: 57 - 60). Simple surveillance was the most effective method of ensuring patient compliance.

Surgery was very much seen as a last resort. Intractable obesity that was 'resistant to normal methods of treatment' (Craddock, 1969: 133) should be treated by fasting (short and long term), intensive out-patient support and a low calorie diet, surgery (reduction of specific areas or intestinal by-pass), group therapy or hypnotherapy. In the mid 1970s surgery was discussed under the heading of 'extreme measures' which included in-patient treatment by starvation and surgical therapies such as jejunum-ileal by-passes or dental splints[44] (DHSS/MRC, 1976: 61 - 62).

Thus, there was a growing body of clinical knowledge about appropriate treatments for overweight and obese patients that included weight loss diets and drugs as well as additional treatments such as monitored fasts and surgery for extreme cases. There

[43] The 1969 edition contains a great deal of detail about the various drugs available – including the chemical structure, mode of action and possible side effects. This is almost entirely omitted from the 1978 edition, possibly due to change fashions in obesity treatment.

[44] A jejunum-ileal by-pass was a surgical procedure in which all but 30cm to 45cm of the small bowel were detached in order to significantly decrease food absorption, and dental splints is another name for jaw wiring which means that the patient can only consume liquids.

were debates about the role of exercise and the most effective and palatable diet, but, in spite of the low rates of success, there was already a large measure of agreement about the 'rational treatment of obesity' (Craddock, 1969: vii).

3.4.6 Costs to healthcare systems

There was virtually no consideration of the economic costs of obesity in early obesity science. Only the author of the OHE report discussed the issue, as this was in keeping with the organisation's remit to examine the economic issues around healthcare. It is also a pharmaceutical industry funded organisation and pharmaceutical treatments for overweight and obesity have always been very profitable. In contrast to later accounts of the expense of treating obesity, the author of the OHE reports described the costs of obesity to the NHS as relatively insignificant, as they derived from visits to GPs, some out-patient appointments and pharmaceutical treatments. Obesity was suggested to result in a small proportion of GPs work – 1.5% of consultations in the UK – and the total costs of obesity treatments were calculated as £3.5 million, although the author did acknowledge that the retail market for over the counter weight-loss products and dietary aids was significantly larger (OHE, 1969 : 26).

Medical textbooks and reports outlining research findings did not include discussions of the costs of healthcare at this time, which may be why many of my sources did not discuss these issues. Health economics was also a relatively underdeveloped discipline (Ashmore et al., 1989: 5 - 6). But, from the mid 1960s there was concern about the increasing costs of the NHS, particularly the rising costs of drugs (Webster, 2002: 46-7). This concern increased after the UK economy slumped following the oil crisis of 1973, and again after the industrial unrest of the Winter of Discontent in 1978/9 (Berridge, 1999a: 55). Discussions of the economics of particular conditions, and the development of new treatments, therefore, took place in a context of severe economic constraints. However, as many of the individuals in this coalition were employed in the relatively well-funded hospital sector of the NHS, these considerations may have seemed less important to them.

In 1983, the wider clinical medical community claimed ownership of the problem of obesity when the Royal College of Physicians produced a report on the topic. This

report was broadly similar in content to the other sources discussed above and can be considered as the culmination of the individual paradigm of obesity. The report's authors discussed the BMI, but defined overweight and obesity as greater than 120% of the upper limit in 1960 Metropolitan Life tables. They considered research into a range of endocrinological and metabolic causes of excess body weight, but concluded that in the majority of cases such changes were secondary to weight gain. Their account was firmly based in Garrow's work on the energy balance model (see chapter 4), and considered the physiological mechanisms underlying food intake, the role of declining levels of physical activity and (in much less detail) 'social factors' such as parental pressure, food availability and customary eating patterns. For the report's authors the management of obesity involved unsupervised slimming, dietary treatment under medical supervision, behavioural therapy (the results of which were sometimes 'disappointing') and appetite suppressing drugs.

However, there were three important areas of difference between this report and earlier publications. Firstly, the RCP report was the first to use larger scale British data on body weight. In the past 'Selected studies have been undertaken in different parts of the country but the criteria for assessing overweight and obesity have varied' (RCP, 1983: 20). In this report, the authors could cite prevalences derived from a 1981 OPCS survey which was designed to be representative of the adult British population, and which used BMI cut-off points to define overweight and obesity. According to this new data 6% of men and 8% of women were obese and 34% of men and 24% of women were overweight (RCP, 1983: 21). Apart from a 15% increase in numbers of overweight men, these figures were very close to the Metropolitan Life figures quoted at the beginning of section 3.4.1.

Secondly, the RCP report used data from both Framingham and the American Cancer Study (ACS) (ACS, 2010)[45] to discuss the health consequences of obesity. Data from

[45] This was a prospective mortality study of nearly a million adult men and women who were enrolled in 1959 and 1960, and followed up until 1972. Participants came from 25 US States, having been recruited in household units by ACS volunteers. On enrolment 'each participant completed a four-page baseline questionnaire providing information on height, weight, demographic characteristics, personal and family history of cancer and other disease, menstrual and reproductive history (women), occupation, diet, alcohol and tobacco, and physical activity' (ACS, 2010). Supplemental questionnaires were sent to participants in 1961, 1963, 1965, and 1972, and there were annual follow-ups to check whether participants were still alive. If they died death certificates were used to establish cause of death.

Framingham and other American studies had been used before, but the ACS study was a new resource and it led to an expansion in the number of conditions associated with excess body weight. Specifically, it led to several cancers being included in the list of negative health consequences of overweight and obesity (see chapter 7). And finally, the report's authors discussed the importance of preventing obesity: 'The high prevalence of overweight in the community means that public health measures as well as individual health education and medical advice are needed' (RCP, 1983: 52). Suggested interventions included breeding animals for less fatty meat, doorstep delivery of low fat milk, the manufacture and promotion of low fat and low sugar convenience foods, increasing consumption of cereals fruit and vegetables, financial measures to decrease consumption of alcohol, provision of advice on behavioural change, and standard nutritional labelling of food. In addition, catering organisations should provide more varied and healthier menus and doctors should provide information on appropriate diets to parents of overweight babies and children (RCP, 1983: 50 - 51). Although firmly based on an individual behavioural change model this was a call for wide-ranging government action in the areas of food production and health that overlapped in several places with proposals in the Canterbury Report on preventing heart disease (see section 3.2). It does not, however, seem to have been influential.

This report was the last in the early series of reports: the next report specifically on the topic of obesity was not published until 1994. It is not clear why the topic of obesity was less of a priority for the British government in the late 1980s and early 1990s. A partial explanation may be that one of the key individuals in this field – Professor Philip James – began to work on WHO publications rather than UK government ones. After 1983 he worked on a technical report on prevention and control of cardiovascular disease (WHO, 1986) wrote a report on healthy eating for the WHO European Office (James et al., 1988) and was involved in producing the technical report on nutrition and the prevention of chronic disease (WHO, 1990). Following the political controversy around the report of National Advisory Council on Nutritional Education (NACNE, 1983), he may have found the subject of nutrition

and health less politically charged in WHO than in the UK (Cannon, 1987)[46]. Despite Professor James' administrative energy and engagement with the topic of obesity, an explanation that depends on the absence of one individual is not entirely satisfactory and further research is required to explain this finding.

3.5 The obesity coalition goes mainstream

In this section I will describe how the public health coalition around obesity successfully developed its activities in the late 1980s and throughout the 1990s. The first two sections draw on my Master's research and uses surveys of writing about the obesity epidemic and contemporary obesity organisations to develop an initial mapping of the policy arena of obesity and overweight. The final section gives more details about the documents analysed in chapters 4 to 6.

3.5.1 Mapping the usage of the term 'obesity epidemic'

Between June 2006 and January 2007 I conducted retrospective searches of Medline, Web of Knowledge and other scientific journal databases for the term 'obesity epidemic' (Fletcher, 2007). I was investigating the usage and spread of the term 'obesity epidemic' as it seemed to mark a new understanding of the increasing incidence of an important public health problem. Combining Medline and Web of Knowledge gave the widest range of results, and further searches produced increasing duplication of results. The search results from these two databases were, therefore, combined and the results cleaned up by removing duplicate and non-relevant results to give the results in table 3.2 below. These results show a sharp increase in the use of this term in the scientific press from 1999 onwards.

YEAR	NUMBER OF ARTICLES
1996	**2**
1997	**3**
1998	**5**
1999	**10**

[46] Chaired by WPT James, NACNE produced a report in 1983 that was alleged to have been suppressed by the Conservative government at the time.

2000	**12**
2001	**26**
2002	**34**
2003	**48**
2004	**87**
2005	**101**
2006	**114**

Table 3.2

Before 1996 the term 'obesity epidemic' was used only occasionally and often interchangeably with 'epidemic obesity'. As table 3.1 shows, usage of the term in scientific journals increased slowly between 1996 and 2000, and then after 2000 increased very rapidly. In parallel to this increase in the scientific literature, a search of LexisNexis showed that there was an increasing usage of the term 'obesity epidemic' in the popular press. I tracked this increase by searching the content of two British daily newspapers, the *Daily Mail* and the *Guardian* for the same period: the former is politically centre right and socially conservative the latter politically centre left and socially liberal, but both regularly contain detailed coverage of health issues. The results of that search were very similar to those in the table above and showing a pattern of increasing usage of the term after 2000. In this period, there were also a number American authors publishing popular science books about nutrition science and the public health aspects of increasing rates of excess bodyweight (Fumento, 1997, Pool, 2001, Critser, 2003, Shell, 2003, Brownell and Horgen, 2004, Oliver, 2006). Some of this writing was reproduced in the British popular press, expanding the size of the coverage and, occasionally, the terms of the debate, by unpacking the wider moral and political implications of these technical debates. This popular science coverage fed off the growing scientific and policy discourse about the health consequences of rising rates of excess body weight, but also provided a rare opportunity for critics of the obesity epidemic discourse to provide sustained counter-arguments, for example against the linkage made between excess bodyweight and ill-health or the use of BMI-based definitions of overweight and obesity.

Contrary to my expectations, and popular discourse, there was no controversy amongst the various professional groups, who might be thought to have a stake in this discourse, about the rapidly increasing usage of the term 'obesity epidemic'. For example, I thought that epidemiologists might object to this new application of the

term to a non-infectious risk factor for chronic disease, rather than an infectious disease like influenza. In fact, I would argue that the rapid spread of this usage demonstrates the existence of a previously developed consensus (or at least lack of controversy) around this usage.

The most heavily cited articles containing the term 'obesity epidemic' came from journals such as the *Journal of the American Medical Association*, *Science*, the *New England Medical Journal* and *The Lancet*. Articles in these leading journals were often written by individuals who contributed to reports, such as the WHO Technical Report 894 *Obesity: Preventing and Managing the Global Epidemic*, who were thus simultaneously developing scientific knowledge and public health policy. In this period British and American research into the health effects of excess body weight was characterised by 'scientific entrepreneurs', high profile scientists who used their status to publicise a particular problem and 'promote it both within and beyond the parameters of science' (Hannigan, 1995: 153). These scientific entrepreneurs were publicising their ideas by writing in high profile general science journals, and their articles were widely cited both by other researchers and by journalists from the scientific and popular press.

However, the journals that contained most number of articles using the term were different from these high prestige general science ones: they were journals such as the *International Journal of Obesity and Related Metabolic Disorders*, *Obesity Research*, *The Journal of the American Dietetic Association*. These are the specialist journals in which researchers developing a detailed body of knowledge write. It also did not seem that this usage spread from a particular group of specialist journals: the pattern I found seemed to be more one of sporadic uses of the term 'obesity epidemic' that became increasingly frequent after the publication of the WHO Technical Report 894 in 2000. This term derived much of its initial legitimacy from the activities of expert committees developing public health policy and this legitimacy was then re-appropriated by several different communities of research scientists who used it in article titles and abstracts as a way of highlighting the importance of their ongoing research in the areas of diet, nutrition and chronic disease.

3.5.2 Contemporary British obesity organisations

The most active British obesity organisations between 1996 and 2006 were professional medical groups such as the Association for the Study of Obesity (ASO). ASO was set up in 1967 and describes itself as 'the UK's foremost organisation dedicated to the understanding and treatment of obesity' (ASO, 2011). It is an organisation for medical professionals working in the area of diet, nutrition and body weight. High profile committee members include Dr Susan Jebb of the MRC Human Nutrition Unit in Cambridge and Dr Peter Kopelman, a diabetes specialist based at St George's Hospital London. ASO organised the First International Congress on Obesity (ICO), held in London in 1974, and also publishes the *International Journal of Obesity* (from 1977). The impetus for the1974 London conference and the journal both came out of the work done to prepare for the first Fogarty Center International Conference on Obesity in 1973 in Bethseda. The second ICO and the second Fogarty Center International Conference on Obesity were both held in Washington DC in 1977. The ICO is still held every three years, the last one being in held in 2010 in Stockholm.

In the 1980s obesity science became more established and the organisation expanded geographically and diversified its activities. The North American Association for the Study of Obesity (NAASO) was founded in 1982, the International Association for the Study of Obesity (IASO) in 1985 and the European Association for the Study of Obesity (EASO) in 1986. IASO now acts as an umbrella organisation, currently for 52 national obesity associations. It also publishes a range of journals including the *Obesity Newsletter* (from 1986), *Obesity Reviews* (from 1988*)* and the *International Journal of Pediatric Obesity* (from 2005). The annual (or sometimes biennial) European Congress on Obesity also began in 1988 with a meeting in Stockholm.

In 2002, IASO merged with the International Obesity TaskForce (IOTF), founded in 1995 by Philip James as a 'policy and advocacy "thinktank"'. According to official IASO history 'the IOTF prepared the first scientific research report on the global epidemic of obesity, which served the basis of the first WHO expert consultation on obesity held in Geneva in 1997' (IASO, 2011). As part of IASO, the IOTF now functions as **'**a global network of expertise, a research-led think tank and advocacy

arm of the International Association for the Study of Obesity' (IOTF, 2011). Individual members of the IOTF, such as Philip James and other Rowett Research Institute researchers, were centrally involved in the production of the WHO Technical Report 894, *Obesity: Preventing and Managing the Global Epidemic*. The IOTF has subgroups working in the areas of childhood obesity, obesity prevention, obesity management and the economic costs of obesity. Its main activities seem to consist of producing research reports and organising conferences in conjunction with other bodies such as the World Heart Organisation. [47]

Other obesity organisations were formed by or aimed at other groups of healthcare practitioners, members of the public and patients. In the UK, the National Obesity Forum (NOF) was set up in 2000 by a group of GPs 'to raise awareness of the growing health impact that being overweight or obese was having on patients and the National Health Service (NHS)' (NOF, 2011). It is an umbrella campaigning group that tries to enrol both healthcare professionals and members of the public in a campaign against increasing excess bodyweight. The NOF has a long list of partner organisations that includes ASO, drug companies, food companies and diet industry companies. It does not seem to contain any particularly high profile committee members. The All Party Parliamentary Group on Obesity, a subdivision of the NOF, was set up in 2001. It was chaired by Dr Howard Stoate MP and Vernon Coaker MP, neither of whom are particularly well known outside parliament, and produced thirteen reports on various aspects of obesity, but seems to have been inactive since 2008, despite a re-launch in 2006. Despite their attempts to become involved in the growing obesity epidemic discourse, neither of these organisations became as influential as the elite practitioner groups, such as ASO, IASO and the IOTF.

By 2005, there were a couple of patient organisations operating in this area. The most high profile was The Obesity Awareness and Solutions Trust (TOAST) set up in 1998 with the stated aim of enabling people whose lives were affected by obesity to influence both policy and treatments. However, its activities were limited and it went

[47] As well as the conferences organised by organisations such as IASO/IOTF and ASO there are also regular obesity conferences organised by commercial companies, such as Childhood Obesity or the Annual Obesity Europe Conference . These are hard to document since they do not last online: once the conference is over its details disappear, and advertising starts for next year's event.

bust in 2006 amid accusations of fraud. Another patient group, the British Obesity Surgery Patient Association (BOSPA) was set up in 2003 and is still active. Neither of these groups managed to become high profile participants in the debates around obesity taking place in the British scientific and popular press between 2000 and 2005.

Other private organisation who became more opportunistically involved in the discourse about obesity and the obesity epidemic included the International Life Sciences Institute (ILSI), a food-industry-funded body that has identified overweight/obesity as one of four Global Science Issues (www.ilsi.org). Another was the RAND Corporation whose senior economist wrote regularly on the health risks of obesity and the links between obesity and chronic ill-health (Sturm, 2002, Sturm, 2008, Sturm, 2007, Sturm et al., 2007, Sturm and Cohen, 2009). This increasing level of activity demonstrates how the obesity epidemic discourse was becoming widely distributed and a resource to which new, non-medical groups could contribute. However, this expanding group of contributors did not diminish the authority of professional medical groups, such as ASO, IASO and the IOTF: they remained the most legitimate creators of knowledge about the individual and social consequences of increasing rates of excess body weight.

3.5.3 Principal sources for chapters 4, 5 and 6

In these chapters I consider nine further publications: four reports on the topic of obesity and three textbooks by key researchers. The first textbook is a special edition of an endocrinological periodical edited by Philip James, with contributions from academic medical researchers from the UK, North America and Europe. The North American contributors included two high profile American researchers, George Bray (see below) and F. Xavier Pi-Sunyer (see section 6.3)[48], and other, less well known researchers from institutions such as Harvard School of Public Health. The British contributors were from teaching hospitals such as St Georges in London and Ninewells in Dundee, and the Dunn Nutrition Laboratory in Cambridge, and the

[48] Both these individuals are endocrinologists specialising in diabetes and obesity At the time of publication, Dr Bray taught at the School of Medicine in the University of South California and Professor Pi-Sunyer was associate professor of medicine at the College of Physicians and Surgeons in Columbia University New York.

European contributors were from universities in Lausanne, Geneva and Paris. This is similar in composition to the earlier symposia described in section 3.3, apart from the wider geographic spread of participants.[49]

The second textbook is an edited collection resulting from an international meeting on body weight control that took place in Montreux, Switzerland in April 1985. Participants who have already been referred above included George Bray, John Garrow, Philip James, Derek Miller and Trevor Silverstone. Other high profile participants included Albert Stunkard, professor of psychiatry at the University of Pennsylvannia; Audrey Eyton, founder of *Slimming Magazine* and author of *The F-Plan Diet*; Theodore B VanItallie, professor of medicine at Columbia University and co-director of its Obesity Research Center; Geoffrey Rose, professor of epidemiology at the London School of Hygiene and Tropical Medicine, and Per Bjorntorp, professor of medicine at Gotenberg University, Sweden. Other participants came from universities and hospitals in France, the Netherlands, Germany and Switzerland and seven of the thirty two participants were from Finland, mostly from the National Public Health Institute (NPHI) in Helsinki. The Finnish group included Audilikki Nilesen co-principal investigator of the North Karelia project, Pekka Puska, professor and director of the epidemiology department of the NPHI and Jakko Tuomilehto, head of the MONICA data centre, also at the NPHI. The presence of such a large and distinguished Finnish group shows the ongoing links between research into heart disease and obesity science (see chapter 2)[50]. One of the editors, Arnold Bender, was an emeritus professor of nutrition at Queen Elizabeth College and, therefore, a former colleague of John Yudkin (see section 3.3). The final textbook was written by John Garrow (see section 3.3) and was one of a series he produced during between 1978 and 2000.

The first of the reports is a 1994 Office of Health Economics report titled *Obesity* written by Richard West. I have been unable to find out anything about this author apart from that he worked for the OHE. The second report, *Obesity: Reversing the*

[49] This presumably partly reflects the reduced cost of requesting the contribution of an article rather than attendance at an event.
[50] Pekka Puska, George Rose and Philip James had previously worked together on a WHO technical committee responsible for compiling technical report 732, *Community prevention and control of cardiovascular diseases* (see section 7.2).

Increasing Problem of Obesity in England, published in 1995*,* was part of the Health of the Nation programme and came out of a symposium on obesity held in February 1994 by two Department of Health bodies, the Nutrition and Physical Activity Task Forces. Neither of these bodies appears to be currently active. The chairman of both taskforces was Philip James and, as well as John Garrow and Peter Kopelman, members included civil servants (especially the secretariat which was mostly drawn from the Department of Health), academics from psychiatry, physical education and nutrition departments and representatives of various organisations. These included industry groups such as the National Dairy Council, Slimming Magazine Clubs, and the British Retail Consortium, professional groups such as the Association for the Study of Obesity, the Coronary Prevention Group, the British Nutrition Foundation and the British Dietetic Association and quangos like the Health Education Authority and the Sports Council. The political momentum necessary for the organisation of the symposium that produced this report ultimately seems to have derived from the target set out in the 1992 DoH *Health of the Nation* White Paper for the reduction of obesity by 25% for men and 33% for women as part of a wider set of targets to reduce the levels of mortality and morbidity cause by CVD (DoH, 1992: 55). In the late 1990s or early 2000s, when it became apparent that obesity rates were rising rather than falling, these targets appear to have been quietly abandoned.

The Scottish Intercollegiate Guidelines Network publication *Obesity in Scotland: Integrating Prevention with Weight Management* was published a year later in 1996. This document was published by the RCP in Edinburgh, and demonstrates the shift towards evidence based medicine since the authors stipulated the type of evidence used for each of their recommendations and graded it using a system developed by the US agency for Health Care Policy and Research (SIGN, 1996). As a way of acknowledging previous policy concern about obesity, the report's introduction referred to the 1977 DHSS/MRC report, the 1983 RCP report, as well as targets included in the 1992 Health of the Nation White Paper and the 1993 *Scottish Diet* report (Scottish Office, 1993). This later reference appears to be mostly rhetorical since the Scottish Diet report focused on health eating and did not discuss the subject of obesity in any detail.

The final report was produced by the British Nutrition Foundation (BNF). The BNF describes its function in the following terms:

> The Foundation promotes the nutritional wellbeing of society through the impartial interpretation and effective dissemination of scientifically based knowledge and advice on the relationship between diet, physical activity and health. It works in partnership with academic and research institutes, the food industry, educators and government. (BNF, 2004a)

It is open only to corporate members and seems to be a food industry funded organisation that works to publicise nutritional research by means of organising conferences, seminars and lectures, developing educational material, funding research, producing reports on particular topics and providing nutritional information to journalists and broadcasters (*ibid.*). Judging by its publications, the organisation has existed since the late 1990s and to date it has produced eight taskforce reports on topics such as cardio-vascular disease, trans-fatty acids, oral health and healthy ageing. These publications 'are written by internationally recognised experts' to 'provide a comprehensive and authoritative review of a particular area of nutrition science' (BNF, 2004b). The remit of the task force that produced the BNF's report on obesity was to review 'the present state of knowledge of the causes, consequences, prevention and treatment of obesity' (BNF, 1999: xi) in order to make policy recommendations and identify future areas of research. This is very similar to the remit for the expert committee that produced the WHO technical report on obesity (see chapter 8). The chair of the taskforce was John Garrow, who was now apparently retired. Other taskforce members include Dr Ann Fehily from H J Heinz Company Ltd; Dr David Mela from Unilever Research; Peter Kopelman (see above); Professor Mike Stock, a physiologist from St George's Medical School, London; Professor Jane Wardle, a health psychologist from University College London and Professor Andrew Prentice, a nutritionist from the MRC International Nutrition Group at the LSHTM. Observers came from the DoH, MAFF and the Department of Dietetics and Nutrition at Leeds General Hospital. Named contributors to the report included Dr Tim Cole a biostatistician from the Department of Epidemiology in the Institute of Child Health, London; Gail Goldberg from the MRC Dunn Clinical Nutrition Centre in Cambridge; Dr Susan Jebb from the MRC Human Nutrition Research Unit, Cambridge; Dr James

Stubbs, a behavioural researcher from the Rowett Research Institute in Aberdeen[51], and two members of the faculty of Agriculture and Biological Sciences at the University of Newcastle. This is a much wider academic network than existed for other British obesity reports, both geographically and institutionally, since it went beyond London hospital based biomedical researchers to include representatives from other institutions, including three different MRC funded nutrition research centres and a department of agricultural science.

This body of material demonstrates the increasing success of British obesity science as reports on obesity were now being produced by increasingly influential bodies – including taskforces convened specially by the Department of Health – and involving greater numbers of participants from a wider range of organisations.

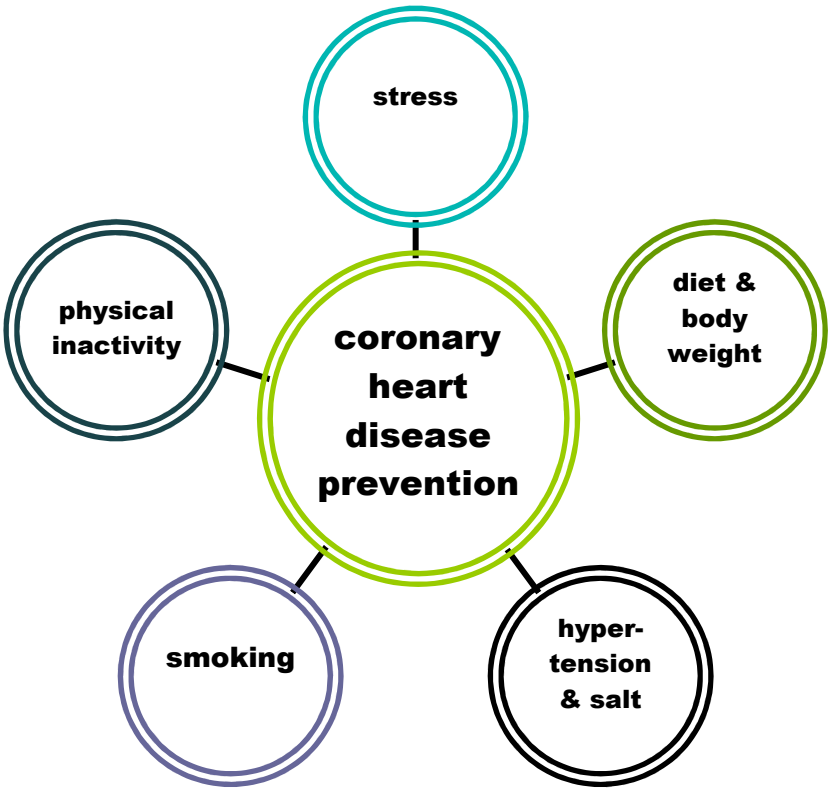

Diagram 3.1 the policy coalitions that developed around each of the identified risk factors for coronary heart disease in British chronic disease epidemiology, 1970-2000

[51] At this time Philip James was Director of the Rowett Research Institute which conducts research into human and animal nutrition. It was founded in 1913 and its first director was Dr John Boyd Orr (later Lord Boyd Orr).

3.6 Conclusion

> We are unanimous in our belief that obesity is a hazard to health and a detriment to well-being. It is common enough to constitute one of the most important medical and public health problems of our time, whether we judge importance by a shorter expectation of life, increased morbidity or cost to the community in terms of both money and anxiety (DHSS/MRC, 1976: 1).

The authors of the DHSS/MRC report produced no evidence for this dramatic assertion and it contradicted their evidence of a small average increase body weight having taken place since the 1940s (see section 3.4.1). As a pharmaceutical industry funded body the OHE had an obvious economic interest in disseminating knowledge about "new" health problems. But the financial interests of a government department such as the DHSS would have gone in the opposite direction, since a widespread and hard to treat new health condition could lead to significant increases in health expenditure. They legitimated their choice of topic by referring to 'widespread anxiety among the general public' on the subject of obesity (DHSS/MRC, 1976: ix). Such reports are exercises in framing arguments as well as fact gathering (Hilgartner, 2000), and so it should not be assumed that such public anxiety existed, but it was a useful justification for a technical piece of writing on medical research findings.

In this chapter, I have outlined the formation of the obesity coalition, modelled on the anti-smoking coalition. I have briefly described the articulation by that coalition of a set of largely unchallenged claims about the nature and public health significance of obesity that I identify as the "individual paradigm" and that serve to justify further research into obesity, especially its causes and prevalence. I have illustrated how a limited amount of American evidence, on changing average body weights and the link between obesity and heart disease, was picked up by an emerging British public health coalition and made the basis of expansionist claims-making about obesity. Obesity was seen as an individual condition, of limited prevalence in the population

which resulted from eating too much due to lack of self-control, had a limited range of health effects that were largely related to heart disease, diabetes and the mechanical effects of heavier body size, was treated by diet, and which cost healthcare services relatively little (compared to the sums spent on commercial dietary services and productions).

In the next few chapters I will go on to document further how key aspects of these claims were elaborated and substantiated through further definitional work and research. This will develop my account of the individual paradigm as I move from looking largely at the articulation of a set of claims about obesity, to also include fundamental practices such as the measurement of obesity and the assessment of different treatments.

CHAPTER 4: THE DEVELOPMENT OF THE BODY MASS INDEX 1972-1995

4.1 Introduction

In the previous chapter, I referred to the competing definitions and measures of overweight used in early obesity science. Often these were based on Metropolitan Life Insurance Company statistical tables of ideal weights. These were developed from height and weight tables (classified by frame size) correlated with mortality statistics and percentages of average weight to define categories of excess weight (overweight was 10% above average weight, and obesity as 20% above, for example). In this chapter I will discuss the problems of defining obesity and overweight in greater depth before describing the development a new index – the body mass index (BMI) - that combined a measurements and definition of obesity and overweight and rapidly became the standard measure throughout American and British obesity science. Eventually the BMI would become a global standard when it was (partially) adopted by the World Health Organization (WHO) in the mid 1990s.

4.2 The measurement of body fat

The basic definition of obesity is an excess of body fat (for example, see Garrow, 1988: 1). This is relatively easy to identify qualitatively – standards for judging bodyweight vary across time and place, but can often be applied without any medical training. But the precise percentage of body fat is very difficult to measure accurately. Moreover, the relationship between body fat (often understood as the body's energy stores) and body weight is not straightforward:

> Body weight is easy to measure to one hundredth of one per cent with a simple beam balance, but the weight of the body bears no simple relationship to the size of the energy stores, nor is a change in energy stores necessarily reflected in a change in weight (Garrow, 1988: 25).

Despite this complex relationship, all but the most detailed and laboratory based approaches to measuring body fat use relative body weight as a proxy measure for fat, because of the difficulty of measuring exact body composition, which makes it

impractical for use in ordinary clinical practice. Body weight and height are simple to measure and routinely collected in primary health care, and so an index based on these measures is more economical to collect at the population level.[52]

A small number of laboratory-based studies have sought to calculate the precise proportion of body fat through the dissection of dead human bodies. Nonetheless, such studies still rely on the assumption that the fat and non-fat components of the body can be separated unproblematically, and that 'the body consists of fat and fat-free tissue of constant composition' (Garrow, 1988: 29), even though research into the physiological effects of weight loss, amongst other processes, has shown this not to be the case (see below). Despite this, Garrow (1978: 115) produced a table of the chemical composition of the human body by compiling the results of 1950s British and American chemical analyses of a very small sample of dead bodies.

> Our understanding of the composition of the human body is based on chemical analyses of 6 cadavers which were performed between 1945 and 1956. Mitchell et al (1945) analysed the body of a 35-year-old white male who died suddenly of a heart attack. Widdowson et al (1951) analysed 2 adults and 1 child; the adults were a man of 25 who died of uraemia and a woman of 42 who drowned herself. Forbes et al (1953) analysed a man of 46 who died of a fractured skull, and Forbes et al (1956) reported two more analyses: one a Negro (*sic*) male with bacterial endocarditis who died aged 48, and the other a man of 60 who was found dead, presumably of a heart attack (Garrow, 1988: 26).

Even within this small set of results, the parameters varied widely: the proportion (in g/kg) of water varied from 674 to 775 and that of protein varied from 165 to 238, apparently without being related to age. The figures for potassium also varied but within a smaller range (see Garrow, 1988: 27). These variations are important because such data has been used in physiological research to estimate both body composition and the effects of weight changes on it. When the practicalities of such an analysis are considered[53], it is perhaps not surprising that so little data exists, but it leads to the conclusion that this area of research in human physiology relies on a surprisingly

[52] A similar case has been made for the use of waist circumference and waist to hip ratios (WHR) (see section 3.2 below).

[53] Such an analysis involves dissecting the cadaver and reducing it to its constituent parts by scraping the bones clean and extracting the fat by boiling or dissolving it in ether.

small and selective set of crucial data. As part of his wider discussion of metrology, O'Connell (1993) outlines a larger history of the measurement of body composition that includes chemical analyses of 3, 6 and 25 cadavers performed between 1902 and 1950, hydrostatic weighing by submersion in water, and the development of portable measurement instruments used in health clubs and weight-loss centres. These developments in measurement technologies mean that

> A body must still be dissected to realize its composition directly, but body composition has been translated into more easily measured quantities that have been accepted as representations of it (O'Connell, 1993: 130-1).

Present-day methods of indirectly estimating the relative proportions of lean body mass (also often known as fat free mass or FFM) and fat mass are technically complex and involve measuring body density, its water content or its potassium content, or using CAT and MRI scanners or dual-energy X-ray scanning (BNF, 1999: 17 - 19). All of these methods are both time-consuming and expensive, and these authors therefore recommended more practical clinically applicable methods such as skinfold measurement, weight and height and combined with waist, hip and thigh measurement, bio-electrical impedance measurement and near infra-red reactance[54] (BNF, 1999: 19). A study of 106 adult women showed that '92% of the variation in body weight is explained by the variation in body fat' (BNF, 1999: 18)[55] which was judged to be a high enough percentage for body weight to be used as a reliable estimate of body fat (with a few well known exceptions, see section 4.6). Studying the effects of weight loss diets required knowledge of their effects on body composition – diets that result in weight loss mostly due to water loss or loss of lean tissue were widely recognised to be both dangerous and ineffective (see for example Garrow, 1988). However,

[54] Skinfold measurement relies on the fact that most body fat is stored immediately under the skin, and the amount of this fat is measured using calipers. However, despite the apparent simplicity of this method, there has been a significant amount of research investigating the necessary number of measurements and the best sites on the body to select in order to minimise potential measurement errors and discrepancies (Garrow, 1988: 37 – 39). Bioelectrical impedance uses a measure of the body's conductivity to assess the relative proportions of water and fat, and near infra-red reactance show the amount of fat present in subcutaneous tissue which is combined with anthropometric data to give an estimate of body composition.

[55] Other sources of such variation include the necessary assumptions made about the density, water content or potassium content of lean body mass. For example, fluid retention leads to a lowered potassium level which in turn means that the FFM is under-estimated and fat over-estimated (BNF, 1999: 18).

> it is more difficult to measure change in body composition with treatment. The same amount of weight loss (and hence decrease in BMI), brought about by two different treatments may signify different proportions of loss of fat, lean tissue and water. At present, there are no methods that will reliably measure loss of fat in an individual with an accuracy of better than 1kg (BNF, 1999: 21).

Given that the approximate weight of fat in an average human body has been given earlier as twelve kilograms (BNF, 1999: 17), one kilogram is a significant margin of measurement error, although if it was expressed as percentage of fat (which is used for other measurement methods, such as density and skinfolds), it will be much larger for a thin person than for a fat person (Garrow, 1988: 29). In spite of such error margins, measurements of body fat were significant because of their use in estimating body composition and, therefore, in judgements about the healthiness (or not) of individual physiques.

4.3 Ancel Keys and George Bray redefine W/H²

As I described in chapter 3, early obesity science writing used the Metropolitan Life ideal weight tables. But by the 1990s, these tables were replaced by the BMI which has become the standard method of both measuring and defining obesity. In comparison to the previous diversity of definitions that I outlined in chapter 3, it is striking how, after the mid 1980s, reports on obesity and textbooks use BMI to define obesity and classify obesity in a highly standardised fashion.

W/H²[56] was first developed by the Belgian statistician and astronomer, Adolphe Quetelet for use in comparing populations. Quetelet was important in the history of human statistics because he was the first person to argue that human physical characteristics such as weight and height follow a normal or Gaussian distribution (Desrosieres, 1998, Hacking, 1990). However, as Quetelet's interest was in comparing populations, he did not use W/H² to assess individuals (Desrosieres, 1998: 73 - 77).

[56] W/H² (weight divided by height squared) is the mathematical formula used to calculate BMI.

In 1972 Ancel Keys was one of the authors of an article arguing that W/H^2 was the most useful of the available indices of relative weight for indicating the relative proportion of body fat in individuals, and suggested that it be re-named the body mass index or BMI (Keys et al., 1972). Keys was the lead investigator of the Seven Countries Study (see section 2.5) and although it is not mentioned in the article the data used in the 1972 article came from this study. The authors compared the usefulness of W/H^2 to that of W/H, the ponderal index (W/H^3), and percentage above average weight, and argued that W/H^2 was superior on two counts. First, W/H^2 was less sensitive than W/H to variations in population height, provides a better measure of overweight rather than simply of stature. Secondly, W/H^2 was found to have a high correlation with skinfold thickness and body density (which were used to estimate body fat). BMI was thus found to be, 'if not fully satisfactory, at least as good as any other relative weight indicator as an indicator of relative obesity' (Keys et al., 1972: 339). While this was not a particularly enthusiastic endorsement, Keys noted that W/H^2 had one additional advantage as an indicator of obesity: it provided both a very clear index of change in the weight of an individual, and an effective means of comparing the obesity of individuals of different heights but the same weight:

> The important difference between the properties of the ponderal index and those of the ratios W/H, W/H^2 and W/H^3 is apparent when calculations are made with increasing weights at constant height. A given increase in weight with constant height will produce exactly the same percentage increase in the values of all the ratios W/H, W/H^2 and W/H^3 but a much smaller increase in the ponderal index. Consider a man 1.70m who weighs 60 kg and then gains 15kg. He gains 25% in weight; his value of W/H changes in the same proportion, his body mass index W/H^2 changes from 20.76 to 25.95, i.e. it increases by 25% also. But his ponderal index changes only from 2.3029 to 2.4807, an increase of only 7.7 per cent. Now consider two persons of the same weight of 60kg, one is 1.70m tall, the other 1.45m in height. The ponderal index of the shorter person is (2.700)(2.303) = 117.2 per cent that of the taller person, while the percentage comparisons using W/H, W/H^2 and W/H^3 are 117.2, 137.4 and 161.1 respectively. Of the various indices considered, the ponderal index is the least sensitive to differences in weight (Keys et al., 1972: 340).

This was, therefore, a relatively sensitive and discriminating measure of relative body fat that could be usefully applied in situations of increasing average body weights both to describe population changes and to compare the relative fatness of different individuals.

A classification for body weights based on BMI values was proposed by George Bray at the first international conference on obesity in 1979, organised by the NIH and held in Bethesda, Maryland. The results of this conference were published under the title *Obesity in America* (Bray, 1979a) and the introductory overview chapter contains a nomogram[57] which includes BMI classification system for men and women that is summarised in table 4.1

	women	**men**
obese	BMI > 30	BMI > 30
overweight	BMI > 23.5	BMI > 25
acceptable	BMI > 18.5	BMI > 20

Table 4.1 (adapted from Bray, 1979b: 6)

This classification was not discussed in the text of the introductory chapter but a subsequent chapter did refer to the difference between relative weight definitions of obesity and measures that combine height and weight

> Various indexes involving height and weight have also been tested. However, they can never provide anything more than an index of overweight, since they falsely suggest that a muscular football lineman is obese and they fail to characterize a patient with atrophic muscle mass and increased body fat. The so-called body mass index (weight/height^2) … has the highest correlation with independent measures of body fat; but in some series this may be as low as 0.6 or less (Sims, 1979: 24).

Evidently, the contributors to the 1979 conference were aware that their chosen index of obesity was imperfect, but a numerical index such as BMI had a variety of useful functions (see chapter 6), and so they made a pragmatic choice from the available options.

4.4 The British adoption of BMI: Garrow's definitions

[57] A nomogram is a diagram used for calculating a variable. The simplest version is a chart consisting of three parallel lines which are scaled. The scales represent three related quantities and the third quantity is derived by joining the two known quantities with a straight line, and reading off the value where it intersects the third line.

Subsequent versions of the BMI-based definition of overweight and obesity with different cut-off points were developed by John Garrow in his series of textbooks. In his 1978 textbook, Garrow referred to BMI in a discussion of ideal weight range linking them to mortality rates, but he did not explicitly define obesity or overweight using BMI cut-off points. Table 4.2 shows the classification that first appears in his 1981 textbook along with a graphic representation in both metric and imperial units. This material appeared unaltered in his 1988 textbook.

Grade III	$W/H^2 > 40$
Grade II	W/H^2 30-40
Grade I	W/H^2 25-29.9
Grade 0	W/H^2 20-24.9

Table 4.2 (adapted from Garrow, 1981: 2)

Grade 0 obesity was previously referred to as normal weight, and as such this re-labelling was an extension of the range of obesity science – now it was not only concerned with the moderately and seriously heavy (grades 1 to 3), but also with the normal weight. This basic system remained intact for at least a decade. Later publications, such as the 1999 British Nutrition Foundation report, with which Garrow was associated, continued to use this classification system citing his earlier textbook.

Garrow was also one of the few authors in this period who discussed frame sizes, but only to argue against their use:

> Life insurance tables may distinguish between large and small frame sizes, but all attempts to refine the estimation of fatness from weight and height by adding various body diameters have failed … There is no advantage in specifying different weight ranges for people of large, medium or small frame if there is no usefully accurate way in which frame size can be measured (Garrow, 1988: 3).

This was a means of distancing his research from the earlier and very influential work of the insurance industry. As a means of increasing the credibility of obesity science, he was demonstrating the superiority of biomedical categories over actuarial ones.

Because of Garrow's advocacy, most of the British publications discussed below cite him as the source of this classification scheme. For example, Philip James used Garrow's classification when writing in 1984 (James, 1984: 636), but when writing in a later collection refers to cut-offs based on the American NHANES data (see below) and percentages of ideal weights taken from the Metropolitan Life tables (James, 1988: 90 - 94). Presumably, he used the definitions embedded in the different sets of data and this switch reflects the use of American data for an American readership. This shows that in the 1980s different classification systems were still circulating. James is unusual in that he explicitly addressed the effect of wider social context on defining obesity, by arguing that an overweight individual in a country like India (where average weights of adults were lower than those of the UK and there was the possibility of periodic famine), might live longer than a person of similar weight in the UK:

> It should be recognized, therefore that any definition of overweight and obesity is an operational one which requires an understanding of the morbidity and mortality associated with varying degrees of weight and/or adiposity in each country. If this were established for each society one could then judge which weights to specify as inappropriate (James, 1988: 90).

However, James went on to argue that such a socially embedded definition is a 'council of perfection', and that a simple definition covering all developed countries is required, such as obesity as >120% of the optimal weight for height ranges in the 1959 Metropolitan Life tables: 'Obesity is therefore, considered in anthropometric terms and is defined on an actuarial basis' (*ibid.*). This meant that although he included BMI ranges at the bottom of his tables of guideline weights, he did not use them as the primary definition of overweight and obesity.

4.5 The British adoption of BMI: other authors use Garrow's definitions

Garrow's grades of obesity		
Ungraded	BMI < 20	Underweight
Grade 0	BMI 20-24.9	Desirable weight
Grade 1	BMI 25-29.9	Overweight
Grade 2	BMI 30-40	Obese
Grade 3	BMI > 40	Severely Obese

Table 4.3 (adapted from West, 1994: 7)

The 1994 Office of Health Economics report on obesity, gave a definition of obesity based on Garrow's 1981 classification (see table 4.3 above) with added labels, and a couple of examples to show how the BMI was calculated. However, the author qualified this definition by arguing that

> In practice the grades of obesity should not be used as rigidly as the classifications may suggest since body frame and build should also be considered, thus, the categories in reality will have an element of overlap and flexibility. It has been suggested that a different BMI scale be used for women, with BMI 18-23 regarded as desirable, BMI 23-28 considered overweight and BMI over 28 judged to be obese (Bray 1979) … A clear case can be made for the interpretation of obesity changing with increasing age. Among Finnish men over 80 years the highest five-year survival was among those with a BMI of over 30 (West, 1994: 7).

This quote shows the complexity of merging Bray and Garrow's classification schemes with data from epidemiological research, as well as debates about whether the same limits should apply to men and women, or to different age groups. Later on similar arguments were made about different definitions for different ethnic groups (see below and chapter 8).

By the mid 1990s overweight and obesity was defined and measured solely in terms of a simplified version of the BMI classification in Garrow's 1988 textbook (DoH, 1995: 3, BNF, 1999: 6-7). There was little discussion of the relationship between body fat and obesity or of alternatives to BMI, which was described as a 'relatively simple index of body fatness' that is useful because 'in general [it] is relatively height-independent, ie short and tall people of similar proportions but very different weight have similar BMIs' (DoH, 1995: 3). This consensus was reflected in the partial adoption of BMI-based definitions of obesity and overweight by the WHO (see chapter 7). Moreover, by the late 1990s the consensus around the use of BMI was well enough established that its history was being outlined in reports:

> There is international consensus that tables showing weight-for-height can conveniently be replaced by a single index. The Belgian astronomer Quetelet observed in 1869 that, among adults of normal body build, weight was proportional to the square of height; in other words weight in kilograms (kg)

> divided by the square of height in metres (m)² was constant. Keys *et al*. (1972) made a similar observation and named the relationship Body Mass Index (BMI) (BNF, 1999: 4).

The BMI was seen to derive prestige both from its origins in Enlightenment science and its re-invention by modern epidemiological research.

It is worth noting that BMI cut-off points had an explicit age-related normativity built into them. They were based on the average weights of young adults, and the fact that ageing often led to weight gain was not taken to mean that standards should be adjusted to reflect this. Two American obesity researchers explained this in their discussion of the relationship between BMI cut-off points and population average weights. Their data[58] used population-based definitions of overweight and obesity, so that overweight was defined as a body mass index of higher than that of the 85th percentile for the surveyed population of men and non-pregnant women aged 20 to 29, and obesity was defined as a body mass higher than that of the 95th percentile:

> The 20-29-year-old group was used as a reference population because young adults are relatively lean and the increase in body weight which ordinarily occurs in men and women during ageing is almost entirely due to fat accumulation (VanItalllie and Woteki, 1987: 40).

Translating these percentile based cut-offs into equivalent BMIs meant that for men overweight was a BMI of greater than 27.8 and severe overweight was greater than 31.1. For women the equivalent cut-off points were 27.3 for overweight and 32.3 for severe overweight (*ibid.*). These alternative cut-off points were used in US government publications until the late 1990s (see section 4.7 below).

British authors also discussed whether recommended BMIs should increase with age. The BMI associated with lowest mortality increases from 21.4 among men aged 20-29 with successive decades from 21.6, 22.9, 25.8 to 26.6 for those aged 60-69 (for women the comparable figures are 19.5 aged 20-29 then 23.4, 23.2, 25.2 and 27.3 at age 60-69). Despite these data, Garrow argued against allowing for an increase in the desirable weight ranges at later ages:

[58] This data comes from the American NHANES II (National Health and Nutrition Examination Survey) which took place between 1976 and 1980. See chapter 7 for more discussion of the use of data from this series of surveys.

> A striking feature about the curves relating QI[59] to mortality ratios is that they become very much flatter in older age groups … In the younger age groups there is a sharp decrease in mortality ratios around or somewhat below average weight, with a high mortality ratio in the most overweight individuals, but in the older age groups relative weight has less and less influence on mortality ratios (Garrow, 1988: 3).

In his judgement, the practical problems of excess weight in old age out-weighed the decreased effect on mortality:

> It cannot therefore be concluded that there is no disadvantage to an old person being overweight, since exercise tolerance and mobility may be greatly impaired by excess weight in an elderly person with degenerative disease of weight-bearing joints. In practice, therefore, the classification given above of grades of obesity serves quite well, at least over the range 20-65 years (*ibid.*).

Later authors continued to make the point that ideal body weights are in this range of BMI 20 to 25, irrespective of gender or age. The British Nutrition Foundation report (produced by a committee chaired by Garrow) stated that, despite the fact that the BMI at which mortality is lowest increased from 20 at aged 20 to 25 at aged 50, this did not mean that there was a health benefit from weight gain by adults: 'In fact there is good evidence that, for an individual, minimum mortality is associated with a constant weight between the ages of 20 and fifty years' (BNF, 1999: 4). This links to the importance of weight stability which was beginning to be discussed at this time (see section 5.6).

From the mid 1990s authors also began to discuss another aspect of obesity – body shape – and another measurement – the waist to hip ratio (WHR). Often this was done using the image of apple- and pear-shaped individuals. A French endocrinologist, Jean Vague, appears to have been one of the earliest researchers to argue for the link between body shape and health, and to classify his patients accordingly (Vague,

[59] QI stands for Quetelet Index. Garrow tried to popularise this name in preference to BMI 'out of respect for our European anthropometric forefathers' (Garrow, 1988: 2). His attempt failed as he was the only person who used it.

1968), but this topic was not regularly discussed in English language writing for another 30 years.[60]

> It is thought that people's shape as well as their weight is an important factor with regards to potential hazards to health. A measure of "central" fat distribution can be obtained by calculating the waist/hip ratio (WHR), by dividing the waist measurement by the hip measurement. Patients with a high WHR are the "wrong" shape since they are "apples" rather than "pears". A waist/hip ratio of above one in men and 0.85 in women has been identified as a meaningful cut-off point associated with increased health risks (Bray, 1993). Many women are pear-shaped, with fat on the hips and legs, whilst an "apple" shape with fat around the middle appears to be the harmful form of obesity, leading to diabetes and heart disease … Those with excess abdominal fat and a BMI of over 27 may be a greater health risk than those adults with a BMI of over 30 but who have their fat peripherally distributed (West, 1994: 7 - 8).

The BMI cut-off point of 30 was by now very standard, but this paragraph demonstrates a further elaboration of the ideal body – it had to be a particular shape as well as a certain size. And although this quote appears to relax the criteria for good health using the distinction of body shape, the author stressed that the 'pear-shaped' individual was still at risk of the mechanical complications of obesity and, ultimately, used body shape to argue for more stringent BMI categories. Sorting out how these two methods of assessing risk relate to each other was difficult:

> Recent studies have shown that the distribution of fat on the body is at least as relevant to the risk of disease as total body fatness (Björntorp 1990). Excess abdominal fat with a BMI of only 26 or 27 may lead to greater risk than a BMI of over 30 in a person whose fat is more evenly distributed (eg Filiporsky et al 1993). Women tend to have more subcutaneous fat than men but most of this tends to be accumulated in the hips and extremities and is not associated with an increased risk of cardiovascular disease (Krotkiewski et al 1983). In contrast men tend to accumulate fat around the waist … Men who have thin limbs but a 'pot belly' are at especially high risk. Pear-shaped men who deposit their fat on their hips have a low waist-hip ratio and are at lower risk. Women with a "male" pattern of fat distribution ("apples") have been shown to have the same risk of CHD and diabetes (Larsson et al, 1992) (DoH, 1995: 4).

[60] I think WHR was a measure used in clinical assessments of risk of heart disease for diabetes patients, in which case these discussions would illustrate the complexities of aligning medical knowledge from different research areas. These discussions were attempting to integrate clinical data from different areas of practice with other data that also derived from different (although overlapping) fields of research. However, more historical research is required in this area.

These authors reported that a relationship had also been found between drinking alcohol and increased abdominal obesity (*ibid.*). Research was attempting to tease out the complex relationships between related risk factors for heart disease, social norms around diet and alcohol consumption, and physiological differences between men and women.[61]

Despite defining obesity using BMI, the authors of another report on clinical guidelines for treatment argued that the waist-hip-ratio – 'the traditional method' of identifying individuals at increased risk – was sufficient, since the allowance for variation in height introduced by BMI was unnecessary:

> New research indicates that measurement of waist circumference alone is preferable for most purposes and best reflects the intra-abdominal fat mass without any need to adjust for height. The waist circumference should be measured at a specific level, viz. half-way between the superior iliac crest and the rib cage in the midaxillary line (SIGN, 1996: 3).

These instructions show that even such a seemingly straightforward activity as measuring the waist can be rendered as a medical procedure. This report gave a table of gender-specific waist measurement and their correlations with increased risks of CHD that is reproduced as table 4.4 (below).

	Increased Risk	**Substantial Risk**
Men	≥ 94 cm (≈ 37 inches)	≥ 102 cm (≈ 40 inches)
Women	≥ 80 cm (≈ 32inches)	≥ 88cm (≈ 35 inches)

Table 4.4 (SIGN, 1996: 4)

A twelve year long Swedish study had also found that a high WHR was a more important risk factor than BMI, but the causality was still seen to be uncertain: 'it is not yet known to what extent the health risk is a direct effect of the visceral fat, or if the disease risk and visceral fat are both indicators of other risk factors' (BNF, 1999:

[61] The protective effects against heart disease of hormones such as oestrogen for women of reproductive age have been researched in depth, but I am unaware of historical or sociological writing on this topic.

16). Moreover, problems of universal applicability with these cut-off points were similar to those for the BMI. After stressing that women with the same waist measurements are at the same risk as men, one author introduced an important exception by arguing that 'adults from the Indian subcontinent are particularly prone to abdominal fat deposition on weight gain, are very susceptible to glucose intolerance and diabetes, and are more prone to coronary heart disease than Caucasians' (SIGN, 1996: 4). This was part of an ongoing attempt to sort out the differences between population groups that formed an important element of later reports (see chapter 8).

In the writing of this period, BMI-based definitions of obesity and overweight became standard. However, due to the overlap between obesity science and different areas of clinical practice, other measurements of cardio-vascular risk, such as WHR and waist circumference continued to be discussed. These two indices were both considered as clinically significant and, despite the dominance of BMI, both were the subject of ongoing discussions as authors analysed new research findings.

4.6 The relationship between BMI and mortality

The BMI cut-off points that were used to define overweight and obesity were derived from plots of mortality rates against BMI which often had a bathtub or a J-shaped curve where BMI 30 or 31 marked a point of significantly increased risk as the curve became much steeper.[62] In the 1970s and 1980s the source of this evidence was still American research, often the large epidemiological studies discussed in chapter 2. For example, one discussion of the relationship between BMI and mortality used data from the Build and Blood Pressure studies of 1959 and 1979 and the American Cancer Study of 1979 (see chapter 7). The authors described the limitations of the Build and Blood Pressure Study (BBPS) data[63], but argued that the American Cancer

[62] Hacking argues that the cut-off point of 25 between acceptable weight and overweight derives from increases in other risk factors that occur at this point, and he regards it as an illegitimate distinction: 'the obesity label for BMI over 30 does, in crude but useful terms, indicate an objective point of increased mortality. *But the overweight label for BMI does no such thing*' (Hacking, 2007: 20 italics in the original). He cites textbooks referring to this issue, but my sources do not discuss it.

[63] The reasons given were that because it comprised of those who bought health insurance it was an unrepresentative sample of the American population – more male, affluent and healthy than the average – and also it relied on self-reported data which was deemed unreliable, especially for weight.

Study showed the same curvilinear relationship between mortality and relative weight or BMI that was shown in the older BBPS study (Hautvast and Deurenberg, 1987). Figures 4.1 and 4.2 show these graphs.

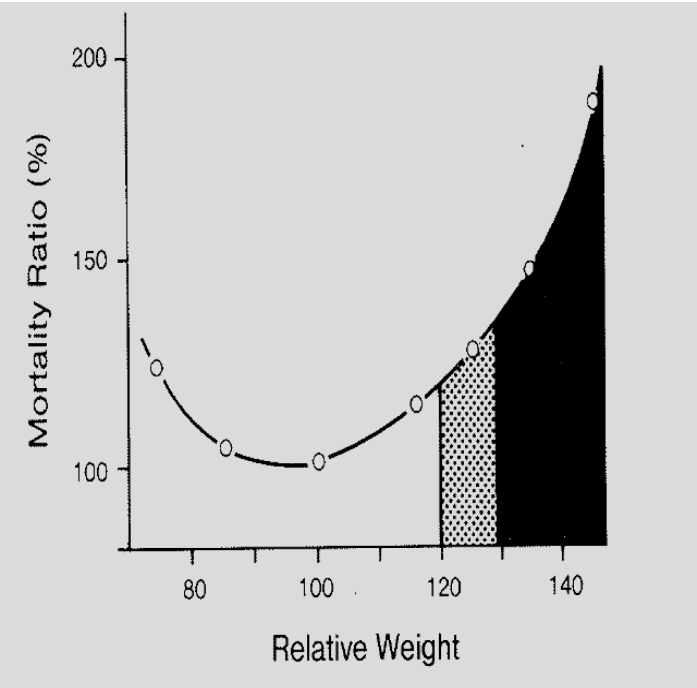

Figure 4.1 The relationship between mortality rate (%) and relative weight as found by the American Cancer Study (Hautvast and Deurenberg, 1987: 66)

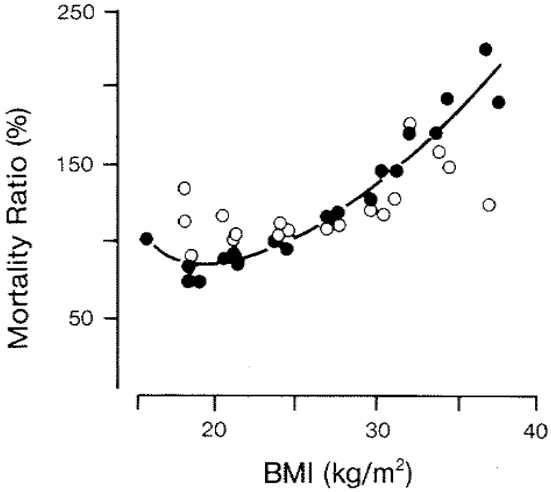

Figure 4.2 The mortality rate (%) in relation to the body mass index (kg/m²) in different age categories (Hautvast and Deurenberg, 1987: 67)

In the 1988 edition of his textbook, Garrow referred to a Consensus Conference organised by the National Institutes of Health in 1985 to provide expert evidence for his claim that obesity is associated with high blood pressure, high blood cholesterol, diabetes and cancer. He argued that

> Medical experts have been curiously slow to realise that obesity impairs health and reduces longevity through an effect on related diseases. Since the first actuarial investigation of mortality in 1903, life insurance companies have been aware that overweight people tended to die young, and hence were less profitable to insure (Garrow, 1988: 9 -10).

Garrow thought that this lag was due to a number of factors: the effects of obesity on mortality take many years to become apparent; the development of conditions like diabetes and cancer leads to weight loss, and so obscures the effect of obesity; and the adverse health effects of smoking confound the correlation between obesity and mortality, since 'smokers tend to be lighter than non-smokers and to die younger, so when studied in a population mixed with non-smokers they distort the true relationship of weight to mortality' (Garrow, 1988: 11). In fact, the relationship between smoking and reduced body weight was widely accepted, and Garrow had to argue against the idea that ideal weight ranges for smokers should be different than for those of non-smokers:

> It has been suggested than the desirable weight-for-height for cigarette smokers differ from that of non-smokers, but there is no reasonable basis for this. Smokers tend to be lighter than non-smokers, and have a greater risk of early death, but when the relation of weight-for-height to mortality is examined for smokers and non-smokers separately the curves are virtually identical in shape for both men and women, with the smokers showing a higher mortality at each weight (Garrow, 1988: 4).

This was part of the continuing analysis of what factors were relevant in the specification of 'ideal' body weights and what could be legitimately ignored. This issue of confounding – sorting out the true causes of disease due to the presence of distorting factors – became widely discussed in this period.

In the 1990s American data were still being used in the framing of such arguments. The author of one report also cited studies based on the American Cancer Study data (see section 3.4) to argue that all-cause mortality in both men and women 'showed a

gradual rise from a BMI of 25 to an almost 2.5-fold higher risk at a BMI of 40'. Furthermore:

> Severe obesity (BMI over 40) is associated with a 12 fold increase in mortality in persons aged 25 to 35 years as compared to those with a BMI 20-25 and BMI 30-40 with a two fold mortality increase (West, 1994: 14).

But there were also British data including a seven year study of 18 400 civil servant which showed that for men 'at age 45 BMI over 30 carries about three times the mortality risk of BMI 20-25' (*ibid.*). Again, the author compared the health consequences of smoking with those of overweight and obesity, by describing how the presence of smokers affected analysis of the relationship between mortality and body weight:

> A non-smoker, for example with a BMI 20-25 would have to increase his or her weight to BMI over 30 in order to experience the same mortality risk as a person with BMI 20-25 who smokes 20 or more cigarettes a day (West, 1994: 13).

Later on, to reinforce this message, he argued that for those with a BMI of over 40, their obesity 'is a greater threat to health than that of being within the desirable weight range but smoking 20 cigarettes a day' (West, 1994: 19). In this argument the health consequences of obesity and smoking were seen as directly comparable. This comparison was presumably made partly as a result of the growing success of the anti-smoking public health coalition (see section 3.3).

Discussion of the relationship between body weight, smoking and mortality continued in these reports, with evidence being used to argue for stringent standards of ideal body weight:

> Epidemiological studies which do not take account of smoking behaviour may not reflect the risk associated with being overweight because overweight non-smokers survive better than thin smokers. Smokers die early and account for much of the increased mortality of the thinnest people in the population. This has led to inaccurate suggestions that weight gain in middle age is conducive to better health (DoH, 1995: 4).

The argument about how smoking confounded the relationship between body weight and health was being used here to argue against data demonstrating that survival rates increased with a moderate increase in body weight in later life (see section 4.8).

Direct comparisons of the effects of obesity and smoking continued to be a routine element of these discussions. Another report argued that 'the mortality risk of a normal-weight adult smoker exceeds that of non-smokers with a BMI of 30-35' but continued: 'both smokers and non-smokers, considered separately, show the lowest mortality rates in the 18.5-24 range of normal BMI. Long-term follow-up of non-smoking adults suggests an optimum BMI of 20 or less' (SIGN, 1996: 10). The comparison was made only for illustrative purposes, rather than as part of a treatment argument, since in the same paragraph the authors argued that stopping smoking was a priority even if it resulted in weight gain.

A final example of these discussions began with an illustration of the J-shaped curve of plot of BMI age mortality ratio showing the BMI at which average mortality is lowest. Figure 4.3 shows this graph.

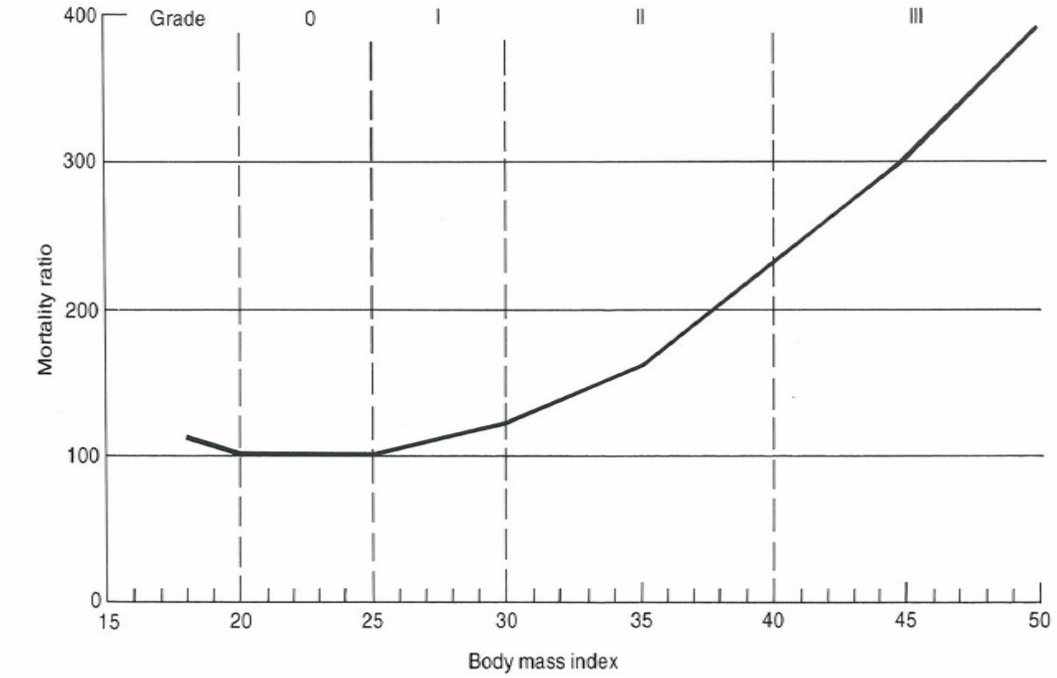

Figure 4.3 (BNF, 1999: 5)

Describing this plot, Garrow argued that:

> Between the ages of 20 and 50 years, [the BMI at which mortality is lowest] increases linearly from a BMI of 20 to a BMI of 25, which in a person 1.73m tall, implies an increase in body weight from 60-75kg. This does not mean, as has been suggested, that there is a health benefit from a gain of 15kg during adult life…..In fact, there is good evidence that, for an individual, minimum mortality is associated with a constant weight between the ages of 20 and 50 years (BNF, 1999: 5).

There were seen to be a series of confounding factors in operation – some very overweight individuals would be dead by 50, chronic degenerative diseases take years to develop and so the life expectancy of younger individuals was more likely to be limited, and conditions such as cancers and chronic infections caused weight loss and increased the mortality rates for lower BMIs. Moreover, data from the Nurse Health Study[64] showed a different relationship since, when smokers and ex-smokers were taken out, the J-shape of the curve disappeared: 'Minimum mortality risk is present in the lowest BMI groups and there is a significant increase in risk at and above a BMI of 27' (*ibid.*). Therefore, 'the J-shaped curve, with a nadir (lowest point of the curve) of a BMI of about 25, is caused by deaths among smokers or among those with pre-existing disease' (*ibid.*). The shaping of the standard curves was defined as an artefact resulting from confounding factors, rather than an expression of a 'true' relationship.

The parallels that were drawn with smoking performed two important functions in these discussions. Firstly, repeated comparison with an increasingly stigmatised and marginalised habit that was framed as the result of individual choice (see section 3.4) invited the inference that weight gain was also voluntary and resulted from individual ignorance and poor food choice. Secondly, the logic of confounding factors allowed researchers to argue that deaths among the underweight were due to smoking or underlying disease (see chapter 6).

4.7 Prevalence and incidence: estimating the extent of obesity and overweight

[64] This study of 121 700 American registered female nurses began in 1976.

By the late 1980s, as well as data from American studies, data from large-scale British research was also becoming available. One of these was Rosebaum's 1985 survey of approximately 10 000 members of the British adult population selected from the electoral register (Garrow, 1988: 5). Using the classification from Table 3.3, the results showed that 40% of men and 32% of women are 'obese to some extent' (*ibid.*). In discussing the effect of age on BMI Garrow argued that,

> Among both men and women the proportion in the range QI 30-35 increases from about 2% at age 16-20 to about 10% at 60-64. Among men there are relatively few over QI [i.e. BMI] 35, but among women the prevalence increases with age to about 4% at age 60 years. Grade III obesity is relatively rare: in this survey there were less than 0.5% of men in this grade at any age group, but among women age 40 and more years about 1% were grade III. We can calculate for the whole population about 0.1% of men and 0.3% of women are in grade III, and these are mainly over 40 years old (Garrow, 1988: 6 - 7).

The relatively low levels of obesity cited for each age group contrast with the figures given in modern discussions of the issue. Comparative international data on prevalence rates also existed for the Netherlands, Norway, Australia, Canada and the US. This shows rates of grade 1 obesity/overweight ranging from 46% for Dutch men aged 50-64, to 11% for Norwegian women aged 20-24, and those for grade II obesity/obesity ranging from 24% for Norwegian women aged 60 -64 and 1% for Norwegian men aged 20-24 (Garrow, 1988: 8). The variation within populations seems to be as large as that within populations, but Garrow's overall summary was that: 'the prevalence of obesity seems higher in North American than in European countries, with the UK in an intermediate position' (Garrow, 1988: 9). Comparison between populations was of more immediate concern than comparison within populations.

Other authors discussed American data from the 1976-80 NHANES II survey which gave the prevalence of overweight among American women as 27.1% compared to a prevalence of 24.2% among American men (VanItalllie and Woteki, 1987: 47). Using BMI cut-offs of 27.8 for overweight and 31.1 for severely overweight for men and 27.3 and 32.3 for women, meant that 34 million men and women were defined as overweight, and of them 12.5 million were severely overweight (VanItalllie and Woteki, 1987: 40). However, breaking these figures down by age, socioeconomic

status and ethnicity, showed that 'several categories of people have a special propensity to become obese; namely black women, women below the poverty line and people with a large frame and/or large musculature' (VanItalllie and Woteki, 1987: 50). The inclusion of this last group begs the question of what was being measured in these surveys; as well as being a proxy for fat, weight may also be acting as a proxy for frame size or musculature, as critics of the BMI argued (see section 4.8 below).

In the late 1990s, the cut-off points used by the US government were changed. Using the above criteria on the NHANES III [65] data, the prevalence of overweight among the adult population was 33.3% for men and 36.4% for women. When Bray's and Garrow's now standard cut-off points were applied to this data the prevalence figures became 59.4% for men and 50.7% for women. As two contemporary commentators put it

> By simply changing the overweight cutoffs, the estimated number of overweight adults increases from 61.7 million (BMI $\geq$ 27.8 and 27.3) to 97.1 million (BMI $\geq$ 25.0), representing a difference of 35.4 million overweight adults. This example calls attention to the actual effect that a shift in BMI criteria can have on determining the population at risk (Kuczmarski and Flegal, 2000: 1078).

An increase of over 35 million adults is a major shift and, although this quote appeared in a clinical nutrition article about twentieth-century American weight classification, it has become part of the wider public discussion about obesity as it is regularly cited by critics of the obesity epidemic to highlight the arbitrary and constructed nature of such criteria (Oliver, 2006: 22, Bacon, 2008: 148-51). However, as this article shows, researchers working in this field were very aware of this point.

Evidence for increases in average body weights started to appear in British government reports: 1991 data from a DoH report gave rates of obesity as 13% for men and 15% for women and rates of overweight as 40% for men and 29% for women, which suggested that over 14 million UK adults were overweight and 6 million obese (West, 1994: 11). The rates found in the UK were seen to be typical compared to those of other developed countries, including the US: 'America has

[65] NHANES III took place between 1988 and 1994.

similar overall rates but a higher proportion of men in the more serious grades of obesity' (West, 1994: 13). In fact the highest rates cited in the 1994 OHE report were from Canada. The Health of the Nation report on obesity argued that there had been a 'marked increase' in the prevalence of obesity amongst UK adults in the last 15 years using figures from four different studies, including one by the OPCS in 1984 (DoH, 1995: 5, see table 3.4 below). The authors also predicted that by the year 2005 average BMIs for both men and women would be 27.5 and obesity rates would be 18% for men and 24% for women which was similar to 'the pattern observed in the United States'(*ibid.*). The situation in the US was beginning to be framed as a warning of future problems in Britain. This process was aided by the increasing production of projected trends in average body weights.

Year	**1980**	**1986/7**	**1991/2**	**1993**
Men				
Mean BMI	24.3	25.0	25.7	25.9
Overweight (BMI 25-30) %	33	38	42	44
Obese (BMI > 30) %	6	7	12	13
Total (BMI > 25) %	39	45	54	57
Women				
Mean BMI	23.9	24.7	25.4	25.7
Overweight (BMI 25-30) %	24	24	29	32
Obese (BMI > 30) %	8	12	16	16
Total (BMI > 25) %	32	36	45	48

Table 4.5 (DoH, 1995: 5)

The authors of this report also gave projected graphs of the rate of increase in mean BMI and secular trends in BMI for both men and women between 1980 and 2010 – the mean BMI for both sexes was given as 28 and the prevalence of obesity for men was forecast to be 21% and for women 27% by 2010 (DoH, 1995: 6). These show how growing collection of data was being used to frame obesity as a problem that had been increasing since the 1980s and would carry on increasing at the same rate, for the next two decades.

The Scottish Intercollegiate Guidelines Network (SIGN) report on obesity used different data because it was produced by a Scottish rather than UK body, and quoted prevalences of 44% of adult men as overweight and 14% as obese, and 32% of adult

women as overweight and 17% as obese (SIGN, 1996: 8). Because this was a document aimed at practitioners, these percentages were then translated into a 'clinical load': 'in a general practice of 10,000 patients about 1,600 men can be expected to be in need of assessment for their overweight and a further 360 need management for their obesity. Similarly, about 1,300 women may be overweight and a further 700 obese' (*ibid.*). Prevalence figures were beginning to move into the arena of health services planning. The authors also quoted rates from regional surveys that varied widely, from a 2% rate of obesity among 18-23 year old men in Glasgow to a 51% rate of overweight among 45-64 year old women in Dumfries and Galloway. However, these figures were described as illustrative since comprehensive data from the Scottish Health Survey did not yet exist (SIGN, 1996: 9).

By the late 1990s, the authors of the British Nutrition Foundation report on obesity could list 38 studies from 1971 to 1996 that provided data on adult body weight in the UK. They gave the location of each study and brief details of the participants e.g. '10,482 male steelworkers aged 20-64 years' (BNF, 1999: 23). In this data, BMI was found to increase with age until 64 for both men and women and then decrease slightly. In particular, data from the 1996 Health Survey for England showed that 'The mean BMI of the age group 55-64 years was 27.6 for men and 27.7 for women; this compared to a BMI of 23.4 in men and 23.5 in women, in the age group 16-24 years' (BNF, 1999: 24). So, in agreement with earlier data, excess body weight might have been a problem in the older age group, but was not a problem for young adults.

The existence of a shared definition of obesity was important in the collection and analysis of this prevalence data. The early documents analysed in chapter 3 contain frequent references to the problems of comparing data from differing surveys because they used differing definitions of overweight and obesity. The adoption of BMI and increasingly standard categories meant that this was less of a problem. However, the majority of the studies cited by these authors were still relatively small scale, and apart from the OPCS study of 1985, there appears to be no government collection of such information in this period. It is not until the 1990s that British and European researchers had access to large scale epidemiological data on body weights as a result of studies such as WHO MONICA (see chapters 7 and 8). The prevalence figures given in these reports do appear to show that rates of overweight and obesity were

increasing, but it is by no means obvious that, in the UK at least, they constituted a significant public health problem.

4.8 Contemporary criticisms of the usefulness of BMI

Contemporary critics from the fields of obesity science or the wider area of chronic disease research were rare. One of the very few critical articles published by researchers who could be described as working in the field of obesity science was written by Dr Paul Ernsberger, a neurologist from Cornell University and Dr Paul Haskew, a nutritionist specialising in eating disorders from the University of Connecticut. Their criticisms focused on the question of what the BMI actually measured, and how well it correlated with health. The overall argument of their article was that medical understandings of obesity and overweight represent 'an unbalanced view of adiposity and health' since elevated risk factors associated with above average weight do not translate into high mortality rates and, ultimately, 'the net adverse effects of adiposity are relatively modest and may be partly attributable to the hazards of treatment ' (Ernsberger and Haskew, 1987: 59). In particular, these authors questioned what was measured by BMI:

> Obesity, as defined by increased weight for height, is not a unitary phenomenon. In some cases only adipose tissue is increased, while in others increased lean tissue mass may make a major contribution to overweight. Both BMI and overweight are highly correlated with measures of body fat ($r = 0.7$ for data from the NHANES survey).However, BMI is almost as highly correlated with lean body mass ($r = 0.6$), or with bony chest breadth ($r = 0.5$). The latter is a major determinant of the mesomorphic or muscular build. A variety of evidence indicates that excess lean body mass or muscle is more hazardous than excess fat (Ernsberger and Haskew, 1987: 86).

Their argument undermined the correlation between BMI and body fat, and, more radically, argued that excess lean body mass might also be a cause of hypertension and other cardiovascular diseases (Ernsberger and Haskew, 1987: 86 - 93). Body shape was also important in their discussion and they concluded that 'mesomorphy, upper body obesity, and excess lean body mass may be interrelated conditions that signal high-risk obesity' and 'individuals biologically disposed towards adiposity, such as the rounded endomorphs, may tolerate fatness better than naturally muscular mesomorphs' (Ernsberger and Haskew, 1987: 91). The evidence they cited for such

arguments was that the Pima Indians have their lowest mortality rates at BMIs of between 35 and 40 and the Maori population, 'a stocky Polynesian people' who show no consistent relationship between BMI and mortality (*ibid.*) – a further unpicking of ideas about a single relationship between obesity, body composition and mortality.

These authors argued for adaptation of the BMI cut-off points, using the J-shaped curves of bodyweight plotted against mortality as evidence that obesity should be defined as consisting of a BMI greater than 30 since this is the point at which 'excess' body weight starts to result in greater risk of illness and death. They quoted another researcher's conclusion that: 'The obese appear to be a greater risk for some forms of death yet at lesser risk for others' (Ernsberger and Haskew, 1987: 37). Ernsberger and Haskew accepted the cut-off point at BMI 30 as meaningful – it did demonstrate a greater risk of some conditions – but argued that this understanding should be balanced by an awareness of a reduced risk for other conditions (see below).

A related element of their argument was a re-framing of existing epidemiological evidence on the relationship between mortality and weight. Data from the Seven Countries study showed the lowest mortality rates in men who were 'somewhat over the average in relative weight or fatness' (quoted in Ernsberger and Haskew, 1987: 5). A follow-up of the study had confirmed this finding, and it was also supported by findings from the 'world's largest epidemiological study to date' (*ibid.*) which collected data on 1.8 million Norwegians for 10 years. The results of the Norwegian study showed that

> Even women who weigh more than double actuarial standards, the so-called "morbidly obese" have a better chance of surviving to retirement age than the leanest women (BMI of 18 and below) … Current medical concepts of "desirable weight" are thus unrelated to epidemiologic estimates of risk, but may in fact be more closely related to current standards of attractiveness (Ernsberger and Haskew, 1987: 6).

Ernsberger and Haskew also argued that the results of the Pooling Project (a synthesis of data from Framingham and four other studies) showed a similar pattern of the underweight group having the highest mortality rates and the 'fattest' group having a

slightly lower rate than that of the 'desirable weight'[66] group. They drew support for this approach from the writing of Ancel Keys himself: 'Commenting on this research and similar findings of his own, Keys concluded that "underweight is a greater hazard than overweight"' (*ibid.*). The hypothesis of earlier writers, that the high mortality rates of the underweight were due to an unknown underlying illness, was also, they argued, not supported by the findings of several studies which showed that the majority of individuals with unexplained weight loss died within two years, and excluding them did not remove the excess mortality. In fact 'exclusion of deaths in the first five to ten years of follow-up had no effect on the increased mortality in thin persons in several studies' (Ernsberger and Haskew, 1987: 9).

Another confounding factor that Ernsberger and Haskew discussed was smoking:

> One study links the increased mortality in thin persons to the lower body weight of smokers…Yet moderate adiposity has been shown to protect both smokers and nonsmokers alike in seven separate controlled studies … Adjustment of mortality data in the Whitehall study for smoking behaviour had no perceptible effect on the U-shaped relationship between mortality and weight … A study of the Pima Indians, among whom smoking is rare, found lowest mortality in persons 45 to 90 per cent above actuarial standards (Ernsberger and Haskew, 1987: 9).

As well as arguing that mortality is lower in smokers with higher body weights, they also argued that the Framingham data showed that it was only 'light-to-moderate smokers who are thinner than average, heavy smokers' weight is similar to that of nonsmokers' (Ernsberger and Haskew, 1987: 10).[67] The complex relationship between smoking, bodyweight, social class - affluent smokers tended to be heavier than average compared to poor and middle class smokers who tended to be lighter (*ibid.*) – meant that it was difficult to analyse the precise nature of the relationships between risk factors and health outcomes. Furthermore they argued that 'Fatness may have an ameliorating influence when it coexists with major risk factors' (Ernsberger and Haskew, 1987: 25), having cited studies showing lower mortality rates in fat individuals with cancer (controlling for the confounding effects of weight loss by

[66] This was defined using insurance company categories.
[67] They suggested that this was because heavy smokers also tended to consume significant amount of alcohol.

excluding those with pre-existing disease), hypertension and cardiovascular disease.[68] Overall, evidence of the negative health consequences of obesity was not seen as convincing by these authors, since removing studies that analysed mortality due only to cardiovascular disease[69]

> leaves only the Framingham study as a prospective evaluation of the role of fatness in all-causes mortality in which the effect of obesity appeared to become stronger with increasing follow-up. Five additional studies covering twenty years or more do not support such a trend … Thus of the six prospective studies of body weight and all-causes mortality covering twenty years or more, two show a negative relationship … three show no consistent relationship … and one study has yielded conflicting reports (Ernsberger and Haskew, 1987: 10 - 11).

The author of a review of 16 studies analysing the relationship between bodyweight and mortality, Reubin Andres, was also quoted arguing that these studies did not show that obesity led to increased mortality and that 'not only does advice on the subject of obesity need reappraisal but research into the benefits of moderate obesity would be worthwhile' (quoted in Ernsberger and Haskew, 1987: 11).

Ernsberger and Haskew raised a number of serious questions about the validity of BMI both as a proxy for obesity and as an indicator of cardiovascular risk, but they were almost alone in raising such concerns. Other researchers within the obesity and chronic disease communities seem to have been remarkably unperturbed by their objections, and even those of a respected pioneer such as Ancel Keys. The use of BMI had become firmly entrenched within the practice of these communities, and technical criticisms such as those raised by Ernsberger and Haskew were insufficient, on their own, to challenge such practice.

4.9 Conclusion

Between the 1970s and the 1990s, BMI became the standard means of assessing obesity both in populations and in individuals. This occurred despite occasional

[68] The second set of data is from the Framingham study and showed that '77 per cent of lean men developing cardiovascular disease died from it, while only 39 per cent of the extremely fat men died from cardiovascular disease' (Ernsberger and Haskew, 1987: 26).
[69] 'Several other studies have shown increased cardiovascular mortality but unchanged or decreased overall mortality among heavy persons' (Ernsberger and Haskew, 1987: 10).

acknowledgements throughout this period that there were important technical shortcomings with using BMI as a proxy for obesity and as a measure of health risk. The reason why BMI gained such acceptance despite these shortcomings was primarily due to the utility of BMI for certain purposes. As Garrow had argued, BMI provided a useful index for observing obesity both in populations and in individuals since it was relatively independent of height and sensitive to changes in body weight. The utility of BMI as a measure not just of obesity but also of health risk subsequently received a strong endorsement when other researchers validated it with reference to other, already accepted data, especially the Metropolitan Life data. Garrow acknowledged that BMI was imperfect as an index of obesity and risk, but held that it was the most practicable such index based on readily available measurements and population data.

Garrow's influential position within the UK obesity coalition meant that his advocacy of BMI for this purpose was widely heeded, and a growing number of subsequent research projects began to measure BMI, either using existing data sets or through the relatively easy collection of new height and weight data. Moreover, this standardisation of practice around the use of BMI - and its independence of other population variables such as height - facilitated comparison between different studies. Increasingly, then, BMI became embedded in practice for practical and pragmatic reasons, both in terms of the ease of collecting the relevant data and the desirability that new studies should be comparable with previous ones. At the same time, further research made clear the complexity of the various factors besides BMI that could influence risk – but, at least for the time being, this was not sufficient reason to abandon use of the BMI as a key piece of information in the analysis of health risks and chronic illness.

CHAPTER 5: THE CAUSES AND TREATMENT OF OBESITY AND OVERWEIGHT

5.1 Introduction

Since the beginning of obesity science the main theoretical underpinning of attempts to understand the development of overweight and obesity has been the energy balance model. This model interpreted the human body as a homeostatic system where excess body weight resulted from a chronic energy imbalance brought about by persistent over-consumption and/or under-exertion. As well as explaining the causes of excess body weight, the energy balance model also provided an explanation of the standard treatments – to lose weight the individual had to create an 'energy deficit' by eating less, being more active or increasing their metabolism with stimulants. By the late 1990s, the acknowledged difficulties of treatment had led to a new approach of 'modest weight loss' which stressed the health benefits of losing 5 or 10kg, rather than aiming for a return to 'ideal' body weight.

The energy balance model was first outlined in the early period of obesity science, and was carried over into later writing largely intact. This chapter will begin with an outline of earlier approaches from the late 1960s until the mid 1980s, before sections 5.3 and 5.4 describe later accounts of the causes of overweight and obesity from the mid 1980s to the late 1990s.

5.2 Obesity as energy imbalance: theoretical understanding of body weight mechanisms in early obesity science

The concept of 'energy balance' was central to the most important model of the mechanisms of body weight maintenance in obesity science. This model interpreted the human body as a system attempting to achieve balance, where excess body weight was seen to result from a chronic energy imbalance brought about by persistent over-

consumption and/or under-exertion. It was usually justified with reference to the first law of thermodynamics[70] and represented in the form of a diagram such as this:

Total Body Calories = Calories IN – Calories OUT

(adapted from Miller, 1969: 59)

Ideas of energy balance were crucial in the understanding of how individuals gained weight, and also provided the explanation for particular treatments such as restricted calorie diets and increased activity levels. In early sources it was often invoked to counter the argument that there were different types of obesity with markedly different causes. For example one author argued

> There is, as yet, no convincing evidence that obesity is primarily a metabolic disorder of adipose tissue considered in isolation: it is ultimately a disorder of energy balance of the whole animal (*sic*) (Hanley, 1969: 34).

In this period, authors were still discussing the definition of obesity – what forms of excess body weight could be considered instances of the same phenomenon (see section 3.2), or which causes were relevant to their approach and which could be ignored as too esoteric or the territory of other specialists.

Once accepted, one of the early uses of the concept of energy balance was to differentiate between "dynamic" and "static" obesity: 'In the first there is an energy imbalance with progressive accumulation of fat, whereas in the second energy balance has been restored, but the subject remains obese' (DHSS/MRC, 1976: 30, see also Hanley, 1969: 36). The idea of static obesity related to the concept of set point (see below) and made sense in a context where average body weights were thought to be rising relatively slowly. An important and related aspect of earlier versions of energy balance theory was the idea that body weight was relatively stable. This was based on the discovery of mechanisms controlling appetite and satiety, and an 'appetite regulating centre' in the hypothalamus of both humans and animals, which if damaged resulted in 'voracious appetite'.

[70] The principle of conservation of energy states that energy can be transformed but it can neither be created nor destroyed.

> The mechanism for weight control is remarkably efficient in most people living normal, active lives, as the intake of merely 1 per cent of excess calories each day will lead to a weight increase of approximately 0.9 kg (2lb) in a year and 25.4 kg (56lb) in 30 years. The reason why weight is normally kept within such narrow limits is not fully understood and it may be due generally to an efficient mechanism for burning excess fuel rather than to controlled food intake (Craddock, 1969: 18).

However, large scale epidemiological study data showed that, in many individuals, body weight was not this finely controlled – in the Framingham study, individuals varied by an average of 5kg. Later accounts did not abandon energy balance models, but incorporated the existence of short-term fluctuations into a more sophisticated account of weight regulation: 'although daily variation in energy turnover may be considerable, there is, over a slightly longer time, a control system which matches intake and output' (DHSS/MRC, 1976: 28). It was now seen to occur over a longer time period.

Factors that were seen to influence caloric intake included body weight, climate, damage to the hypothalamus, stomach capacity, blood glucose levels, skin temperature, body fat, exercise and food palatability. On the last point an early author anticipated issues that were repeatedly raised in later reports:

> In times of food shortage man will eat almost anything simply to satisfy his calories needs, but in an affluent society his palate is tickled by such a wide range of flavour and texture in attractive dishes and convenience foods it is not surprising that he eats more than a bare minimum (Hanley, 1969: 65).

These authors did not appear to have a detailed understanding of the mechanisms of human appetite and most argued that a lot more research was necessary: 'There has been much research on the factors influencing appetite in animals but little work has been undertaken in man, and the degree to which appetite adjusts to energy needs has not been fully explored'. Moreover 'There is still no consensus on the degree to which appetite in man is important in maintaining energy balance'(DHSS/MRC, 1976: 32 and 33). In these arguments, laboratory research was framed as a means of increasing knowledge about the mechanisms of human appetite and improving clinical and public health practice.

A large element of the research took the form of psychological studies investigating differences in eating behaviour between the obese and the non-obese. However, this research had also failed to prove that the obese ate more than 'normal' individuals, although they had been found to be 'insensitive to "internal cues" which normally affect satiety or hunger[71]: 'a normal weight subject seems less influenced by his external circumstances and responds to some internal signs or signals which tend to reflect the state of energy balance' (DHSS/MRC, 1976: 34). This was part of a wider programme of research into whether the obese were different from non-obese individuals in significant ways: due to excess food consumption or pre-existing metabolic, endocrinological or other physiological differences. All of the report and textbook authors addressed some aspects of these areas of research. Craddock discussed the metabolic effects of exercise, overfeeding ('most are reversible'), cold and metabolic differences between the obese and the normal weight, concluding that:

> In the majority of people most metabolic differences between obese and normal people are ones of degree only and are due to adaptation to abnormal intake of food at some time (Craddock, 1969: 35).

Overall the consensus was that, despite the lack of available evidence, the differences in energy metabolism between obese and lean subjects do not lead to the development of obesity (DHSS/MRC, 1976: 47). For endocrine changes the conclusions were broadly similar: endocrine changes in obesity were discussed in terms of adrenal, thyroid and pituitary function and the role of insulin. Studies of overfed volunteers showed that 'many hormonal changes are secondary to the subjects' increased weight and are not necessarily an innate characteristic of obesity' (DHSS/MRC, 1976: 53). Changes in metabolism and hormone levels were seen as the result of excess weight rather than the cause of it.

An alternative approach, arguing that childhood obesity led to an increased number of fat cells – 'hypercellularity' – which made individuals prone to obesity later in life, was also discussed but described as unproven (DHSS/MRC, 1976: 48). This was similar to Craddock's distinction between the 'metabolic' obese and the 'normal' obese:

[71] Internal cue are feelings of hunger, such as a growling stomach, rather than habit or external cues such as time of day.

> The clinical findings that some obese patients can lose weight easily ('easy losers') while others genuinely find it difficult ('poor losers') is confirmed by the two distinct groupings found, even in children, as regards the serum insulin level and the differing response of the serum free fatty acid level to adrenaline or glucose. It is likely that most of these 'metabolic' obese have been overweight from childhood. Some obese patients therefore not only lay down fat more efficiently than others, but also mobilise it less efficiently (Craddock, 1978: 48-9).

Such an approach contradicted the others outlined above, but in this period there were several possible models co-existing, as researchers sought to further analyse a complex and poorly understood phenomenon, using research from several different kinds of research. Ideas of excess numbers of fat cells also recurred in a later distinction made between hyperplastic and hypertrophic obesities:

> Early stages of fat storage involve expansion of existing adipocytes (hypertrophy). Later stages involve the recruitment of new adipocytes (hyperplasia). Current evidence suggests that hyperplasia is difficult to reverse once it has occurred, thus emphasising the need for preventative strategies (BNF, 1999: 38).

Not only has hypercellularity now become part of the arguments for preventative strategies, but because these processes were not seen to occur only in childhood, such strategies have become applicable to a much wider section of the population (see chapter 8).

John Garrow's research was firmly rooted in the energy balance model – the title of his first textbook was *Energy Balance and Obesity in Man* (Garrow, 1978) – the first edition of which included an introduction of 38 pages to outline the concept of energy balance and

> persuade all these people [physicians, dieticians, biochemists, physiologists, psychologists, physiotherapists] to think in terms of energy balance, and not to suppose that there was "a cause" of obesity, like overeating, inactivity, or some genetic disposition (Garrow, 1978: 1).

Garrow's textbook contained detailed discussions of the factors affecting energy intake, energy output and energy stores (Garrow, 1978). In the chapter on energy

intake he outlined the role of the hypothalamus, intestinal factors such as gut hormones and endocrine factors such as insulin, referring to Bray's[72] review of the topic (Garrow, 1978: 53 - 6). However, a large proportion of this section was taken up by a description of the social causes of obesity:

> There is no shortage of factors which can be shown to influence food intake in one model system or another. To understand energy balance in man we need to know how the control system is integrated, and why the human race tends more often than most species to err in the direction of a positive balance with consequent obesity (Garrow, 1978: 56).

He labelled this 'the big head problem' - the fact that human intelligence leads to complex eating behaviour – but also discussed the social factors influencing taste, the effects of drugs and illness, childhood influences and set point theory.

Set point theory was the idea that individuals have a standard weight to which they are metabolically programmed to return (Garrow, 1978: 63 - 67). Garrow profoundly disagreed with the concept of a set point since short-term control of food intake in individuals was too erratic for a set point to exist, and moreover, the evidence of long term weight stability had been over-stated (Garrow, 1978: 64 - 7). Whilst he accepted that metabolic adjustment to both over and under feeding could occur, he argued this was largely limited to weight loss and gain, and so not evidence for a set point:

> nor is it necessary to postulate one to explain the observed stability of body weight in most individuals. Conscious control of food intake and energy expenditure is the most probable explanation for the observed oscillations in weight about a preferred level (Garrow, 1978: 144).

Garrow's ultimate conclusion on the subject of food intake was that:

> In man the control of food intake is very complex, and the primitive hypothalamus reflexes are buried under so many layers of conditioning, cognitive and social factors that they are barely discernible. In the short term the physiological control of food intake is very poor, and under laboratory test conditions he is easily misled by false cognitive clues. Body weight in men is not constant within 2kg, as has been stated, but usually fluctuates about 10kg

[72] George Bray was a prolific and influential American researcher in the areas of body composition, energy expenditure and the metabolic consequences of obesity. He developed the classification of obesity by BMI discussed in section 3.4.

> during the adult lifespan …The factors determining these short-term (i.e. year to year) oscillations are unknown, and virtually uninvestigated except on an anecdotal basis (Garrow, 1978: 76).

Whilst ostensibly stressing the social factors that affect eating behaviour (which often is how it is framed when quoted), Garrow's focus on the metabolic processes underlying energy balance meant that his conclusion also functioned as an argument for further physiological research into the control of food intake and body weight.

5.3 Physiological explanations of the causes of obesity in later sources

A 1984 collection of articles edited by Philip James summarised contemporary areas of biomedical research relevant to overweight and obesity. It included: two chapters on obesity in animals (laboratory mice) that covered dietary and genetic factors; one on the role of catecholamines in the regulation of thermogenesis[73]; one on brown adipose tissue activity in animals and man; one on the neural and endocrine control of energy balance, again in animals and man; one on the regulation of food intake in obese individuals; and two on the endocrinological aspects of obesity, one of which focused on the role of thyroid hormones (James, 1984). In addition to improving understandings of the underlying bodily processes, such research was still conducted with the aim of understanding in what ways the obese were different from the non-obese. For example, in the chapter on catecholamines, the authors described the existence of individuals with a 'thrifty' metabolism who do not burn excess energy as heat and are therefore liable to obesity (Landsberg and Young, 1984: 493), while one of the chapters on endocrinology stated that 'there is little to indicate that obesity depends on some underlying endocrine abnormality but much evidence to suggest that the endocrine changes are secondary to the abnormal nutritional state of the patient' (Jung, 1984: 609).

Two chapters in a 1987 collection of articles also discussed understandings of obesity rooted in energy balance models. The first outlined current understandings of energy metabolism in humans, discussing the three components of energy expenditure – basal

[73] Catecholamines are hormones such as adrenaline and noradrenaline that are produced by the adrenal glands and thermogenesis is the process of heat generation which occurs due to physical activity, diet and some drugs, one being caffeine.

metabolic rate (BMR)[74], thermogenesis and physical activity. The author focused on whether obese individuals expend less energy than lean individuals:

> Obesity arises when energy intake is chronically greater than energy expenditure, which leads to a positive energy balance. It is, however, difficult to establish whether this imbalance results from an excessive input or a defective output of energy. In some obese individuals hyperphagia is the obvious cause of excessive body weight, whereas in others an inability to adapt energy expenditure to a variable intake may play a role favouring energy gain (Jequier, 1987: 21).

As well as the practical ramifications of such issues, the author argued that the need to reconcile low levels of food consumption reported by some obese individuals with the basic theory of energy balance meant that more research into the regulation of energy intake and expenditure was needed. The second chapter, meanwhile, discussed the mechanisms of weight regulation primarily in terms of different aspects of the 'set point' of body weight (see section 5.2), including temporal variation and possible biochemical mechanisms, using findings derived from animal (mostly rat) and human research (Apfelbaum, 1987).

Garrow's 1988 discussion of the aetiology of obesity began with a section on the inviolability of the laws of thermodynamics, which meant that

> The search for aetiological factors in obesity can therefore be narrowed down to factors affecting energy input or output. Do obese people have a higher energy intake or a lower energy output than lean people? (Garrow, 1988: 102).

Referring to research into human subjects, he reviewed evidence concerning the resting metabolic rate of individuals of different weight, and their energy expenditure, to argue that, on average, obese individuals have higher energy expenditures than do lean, therefore they must have a higher energy intake in order to maintain their excess bodyweight (Garrow, 1988: 108). It had been thought previously that the obese had lower metabolic rates than the lean – that was why they put on weight – and so this was an important point. He also discussed research into the aetiology of human obesity using rats and mice which focused on the role of regulation of food intake by

74 Basal metabolic rate (BMR) is defined as the amount of daily energy expended while at rest in a neutrally temperate environment when the digestive system is inactive (which requires about twelve hours of fasting in humans).

centres in the hypothalamus, the adipocyte[75] hypothesis that the number of fat cells in an adult does not increase (see above), and the role of brown adipose tissue in thermogenesis. However, rodent models could often be misleading due to metabolic and/or physiological differences between rodents and humans (Garrow, 1988: 109 - 110). Much knowledge about the regulation of food intake and body weight still derived from rodent experiments, but integrating this research with data from human experiments could be problematic because human diets were more varied than those of laboratory rats, and human populations are more genetically diverse than those of laboratory rats (Garrow, 1988: 111).

Garrow's book also contained another discussion of the concept of 'set points' explaining that 'individuals are said to tend to revert easily towards their set point, but to oppose movement away from their set point by metabolic rate adaptations' (Garrow, 1988: 114). However, Garrow argued that the data used by proponents of this theory did not support such a hypothesis, rather it supported the idea of a '"buffer" control system' which 'tends to oppose and minimise any imposed weight change' (Garrow, 1988: 115). According to this logic, weight change in any direction is guarded against, rather than specifically weight loss.

Finally, Garrow also discussed the genetics of obesity, mentioning the 'thrifty gene' hypothesis, but arguing that 'it is a mistake to argue that characteristics that are shared by a family are necessarily genetically determined' (Garrow, 1988: 118). Using data from several large scale studies, including one on 540 Danish adoptees and another on metabolic rates of Pima Indians,[76] Garrow argued that there is a genetic component to obesity, but not so large that weight loss cannot be achieved:

> The problem is that inheritance may be genetic or cultural, and it is difficult to distinguish these possibilities. For example the total inheritance of percentage body fat, and of distribution of fat, may be 0.55 and 0.61 respectively, but some of this inheritance is due to a genetic-environmental interaction, and the true genetic inheritance is probably about 0.22 and 0.28 respectively (Garrow, 1988: 121).

[75] Adipocyte is another name for fat cells.
[76] See chapter 7 for further discussion of research into the Pima Indians.

Garrow's use of figures of percentage inheritance provided an additional aura of scientific authenticity to his discussion of the thrifty gene hypothesis. However the source of these figures was not provided. Other authors argued that Albert Stunkard's study of the weight of Danish adopted children and their parents showed a significant relationship between the weight of the biological mother and the adopted child, but that this relationship was strongest for thinness rather than fatness. Such research was often seen to demonstrate that 'The genes which lead to a tendency to weight gain are thought to be insufficient on their own to cause obesity and can be overcome by manipulating diet and lifestyle' (West, 1994: 16). Such studies lent themselves to diverging interpretations, as other authors framed the results of human twin and adoption studies very differently. In a discussion of the heritability of obesity, another author argued that 'Genetic influences appear to account for 50-70% of the difference in BMI in later life in both monozygotic and dizygotic twins brought up apart whereas the childhood environment had little or no influence' (BNF, 1999: 39). In contrast to earlier authors, this author also argued that adoption studies showed 'a strong relationship between the BMI of the biological parents and the adoptee for the whole range of body fatness… No relationship is shown, however, between the adoptive parents and the adoptee' (BNF, 1999: 40). However, this 'hard line' approach was softened by the author's stress on the complex aetiology of obesity. This then allowed them to argue more plausibly that the rapidly increasing prevalence of obesity in the UK over the last decade 'in a genetically stable population' confirmed the importance of environmental causes, and the necessity for further research into gene-environment interactions (BNF, 1999: 44).

By the 1990s, energy balance approaches were regularly drawn upon in government policy literature. The 1995 Health of the Nation Report contained a brief discussion of the concept of energy balance and the factors that were seen to affect it. Although Garrow's work was not directly cited - the studies cited came from Philip James and his collaborators (DoH, 1995: 12) - Garrow was a member of the taskforce responsible for the report, and the approach outlined was firmly based in the energy balance model. Unsurprisingly, given Garrow's chairmanship, the 1999 British Nutrition Foundation (BNF) report contained an extended discussion of the aetiology of obesity that was also firmly based on ideas of the role of energy balance (BNF, 1999). The results of further research into human metabolism meant that the main

components of energy expenditure could now be quantified - for sedentary individuals the approximate percentages were given as basal metabolic rate 65%, thermogenesis 10% and physical activity 25% - and the authors of the BNF report argued that the overweight and obese have higher basal metabolic rates and total energy expenditures because of their increased weight (BNF, 1999: 68). This was emphasised to refute earlier arguments that individuals gain weight due to lower metabolisms.

Physiological research had also taken place into the metabolic pathways of different macronutrients, such as carbohydrate, fat, protein and alcohol, that gave a picture of complex and overlapping metabolic pathways for the different 'fuels' burnt by the body. The tendency to store fat in the form of excess bodyweight was a consequence of the lack of an exact match between fat intake and its use, combined the fact that fat was the least preferred of the body's fuels (BNF, 1999: 71). The report's authors saw findings on the physiology of appetite regulation as supporting this approach. According to their interpretation of the research, human energy balance was regulated mostly through changes in food intake, and physiological regulation was 'often deeply embedded within social and environmental influences, which can readily lead to dysregulation' (BNF, 1999: 80). One example of this was the phenomenon of 'high fat hyperphagia' or 'passive over-consumption' where individuals were found to eat roughly the same amounts of food, even if the fat content was significantly increased, leading to energy imbalance. This pattern 'is readily reproducible in experimental settings and helps to explain the increasing prevalence of obesity' (*ibid.*) (see section 5.4 below).

An important new line of argument developed by obesity science authors in the 1990s was that a relatively small energy imbalance, if present over a long period of time, could explain increasing rates of obesity.

> The increase in average BMI and the prevalence of obesity in the UK, although marked, can be explained by a persistent energy imbalance, consistently positive, but of a very modest degree. Assuming the average height has not changed, the increase in average BMI since 1980 would be equivalent to an average increase in weight in men and women of about 4kg between 1980 and 1992. The energy content of excess weight amounts to about 7000 kcal/kg (29MJ/kg). Thus, if an adult puts on 4kg, a net gain of

> about 28 000 kcal will have occurred. Assuming there is a smooth, even gain in body energy and fat, this is equivalent to an excess of 6.5 kcal/day over 12 years. A 4kg weight gain is unusually large but represents a discrepancy between average intake and output of only about 4% per day over the year (DoH, 1995: 13)[77].

This way of understanding the long term processes of weight gain became an important element of what I have labelled environmental understandings of obesity, since it demonstrated how individual and population weight gain can occur largely unnoticed (see chapter 8). However, the fact that consuming as an excess of as little as a 6.5 kcal a day was thought to lead to significant weight gain was important, since it implied that weight gain was very easy to achieve with relatively small levels of 'over-consumption', and led to the question of why a greater proportion of the population was not overweight or obese. The authors of the 1996 Scottish Intercollegiate Guidelines Network (SIGN) report discussed processes of energy imbalance and weight gain in very similar terms:

> Body weight is regulated by powerful physiological signals which change appetite and satiety to a far greater extent than is recognized by patients. However, only a small (i.e. 2%) persistent discrepancy between daily intake and energy output is required to produce progressive and substantial weight gain. Metabolic responses to over-eating or semi-starvation play only a modest role in buffering changes in energy balance. The metabolic rate relates to body weight of an individual, but there are substantial differences (±20%) between individuals … A modest but persistent accumulation of only 50-200 kcal daily leads, over a 4-10 year period, to a slow but progressive weight increase of 2-20 kg before the metabolic and physical cost of maintaining this additional weight balances the additional intake. Body weight then stabilizes at this higher level (SIGN, 1996: 5).

The figures given in this report were different – a much lower percentage discrepancy was required than in the 1995 report, but a higher excess amount of calories was cited – however, the authors of the Health of the Nation and the SIGN reports both stressed the important role of small scale, chronic, over-consumption in the development of overweight and obesity.

Another theoretical approach being incorporated into obesity science from the mid 1990s was that of 'critical periods' for the development of obesity. These critical

[77] 6.5 kcal is the energy value of 100g (or roughly 3 sticks) of celery.

periods were listed in one publication as foetal growth, infancy and childhood, adolescence, reproduction (pregnancy and lactation) and middle age (especially the menopause for women) (BNF, 1999: 45). The author admitted that the significance of these critical periods was 'not universally accepted' and that evidence of susceptibility only applied to a 'small fraction' of individuals. I question the practical uses of such a list, since these critical periods could easily cover the majority of a woman's life, leading to the question of whether there were any non-critical periods. However, judging by the numbers of articles cited in this discussion, studies of *in utero* determinants of obesity were becoming an important area of obesity science. Such studies had addressed the effects of foetal over-nutrition (studying both diabetic and non-diabetic pregnancies), the effects of foetal under-nutrition (its effect on heart disease rates from both UK epidemiological studies and data from the Dutch famine of 1944-5), the relationship between foetal and maternal adiposity, and tracked weight/adiposity from birth until adulthood (BNF, 1999: 45-59 (chap 7)). The last of these studies was a continuation of the earlier studies of foetal over-nutrition on mothers with poorly controlled diabetes, which often results in large babies. One important source of data for this research was studies into high rates of chronic disease among the Pima Indians of Arizona:

> Pima Indians are probably the most intensively studied people with respect to longitudinal follow-up and development of obesity. However, this population is one in which the prevalence of both obesity and non-insulin-dependent diabetes mellitus (NIDDM), with its consequent gestational effects, are among the highest in the world. The findings may, therefore, be atypical (BNF, 1999: 46).

However, the evidence for the importance of these 'critical periods' appeared to be at best mixed. The author concluded that maternal obesity and gestational weight gain tended to produce bigger fatter babies, as did poorly controlled diabetes, but it was not yet known whether these fatter babies were more likely to be obese as adults (*ibid.*). However, such research on specific population groups such as the Pima was an important source of data for later reports (see chapter 8).

The accounts discussed in this section demonstrate the growth of abstract understandings of the causes of overweight and obesity based in basic metabolic, endocrinological and physiological research underpinned by the energy balance

theory of body weight regulation. They also show the effects of earlier increases in funding for laboratory research in these areas (Quirke and Gaudilliere, 2008) and the many different kinds of research that were being drawn upon in the development of obesity science.

5.4 Other explanations of the causes of obesity

This section considers another collection of explanations of the causes of overweight and obesity, also discussed in the 1980s and 1990s, which derive less from laboratory research into human and animal physiology, and more from other research traditions including psychology. These causes were often considered under broad headings, such as the role of physical inactivity, psychological factors (sometimes this included food choice but sometimes it was addressed under a separate heading) and socio-economic factors. The last of these categories often functioned as large and poorly-defined catch-all collection of information, often 'social' in nature that was deemed relevant to the topic.

The role of inactivity in the development of obesity, despite appearing quite obvious, was difficult to resolve, partly due to lack of data:

> It is very difficult to obtain data to show that inactivity really causes obesity, largely because habitual physical activity is so difficult to measure … Certainly very obese people tend to be inactive, because they have very low exercise tolerance, and cannot manage much exertion. On the other hand the exercise which obese people do undertake costs them more energy than the same activity undertaken by a thin person. Furthermore many non-obese people are also very inactive (Garrow, 1988: 121).

According to Garrow, studies had found that there was no difference in the physical activity patterns of the majority of the obese and the non-obese, since both groups had sedentary lifestyles, and so after describing the existing evidence as 'sketchy', he concluded that inactivity may be a 'minor contributing factor' (*ibid.*) in the aetiology of obesity. However, research into this topic carried on, despite these early disappointing results.

In later writing, it is notable how often the link between physical inactivity and the development of obesity was taken for granted, rather than demonstrated by the results of specific studies. For example, the authors of the 1995 Health of the Nation report quoted NHANES-1 (see section 4.5) data showing an inverse link between low recreational physical activity and weight gain, and three other studies showing that overweight children spend more time watching television (DoH, 1995: 16). This small amount of data did not appear to show any evidence of cause and effect, and did not rule out the possibility that increased television watching was a result of overweight rather than a cause of it. Nonetheless, the authors' conclusion was that

> It seems reasonable to conclude that not only is the overall population likely to get fatter as leisure activity declines but that individuals who are particularly inactive are much more likely to gain weight in both childhood and adult life (DoH, 1995: 17).

In the absence of any clear epidemiological or clinical research findings, this statement was presumably based on everyday common sense ideas about the link between declining levels of physical activity and increased weight. Researchers also sought to counter the concern that increased levels of physical activity would lead to increased appetite. Thus the BNF report of 1999 declared that there was 'no evidence to suggest that exercise results in more food than would be expected in order to compensate for the extra expenditure' and that 'the evidence is stronger that exercise may work to improve feeding regulation to better match energy expenditure' (BNF, 1999: 131). This demonstrates the persistence of earlier worries about the counter-productive effects of increased physical activity.

More complex thinking about the possible social causes of obesity was notably absent. Garrow's 1988 textbook on obesity was typical in this regard, devoting only a small proportion of his chapter on the causes of obesity to the social, cultural, psychological and cognitive factors that affected food intake. This section included a brief discussion of Jeffrey Sobal and Albert Stunkard's 1989 article[78] outlining the inverse relationship between obesity and social class found in developed countries

[78] The Sobal and Stunkard article reviewed 144 studies of the relationship between the incidence of obesity and socio-economic class, and was an important source and that had been regularly cited in obesity science writing since its publication (see also section 1.4).

which contrasted with the direct relationship found in less affluent countries such as India and Germany (*sic*), but Garrow concluded

> There is no obvious explanation for the increased prevalence of obesity in the poorer sections of many communities, nor is it clear if the poverty causes the obesity, or the obesity causes the poverty (Garrow, 1988: 126).

He also referred to Suzy Orbach's (1982) argument that obesity and other eating disorders in women resulted from a rejection of the norms of feminine behaviour around appearance, sexual relationships and domesticity. Somewhat missing the point, Garrow suggested that 'obesity also occurs in men, presumably for unfeminist reasons, so some obesity in women must occur for unfeminist reasons also' (Garrow, 1988: 128). This was, however, a rare reference to feminist criticisms of obesity science.

The author of the 1994 OHE report also used Sobal and Stunkard's article in his discussion of socio-economic and psychological factors in the development of obesity. Sobal and Stunkard had concluded that there was a strong inverse relationship between socio-economic class and obesity for women in affluent countries; a weaker inverse relationship for children and men in affluent countries; and a strong positive relationship between socio-economic class and obesity for women, children and men in developing countries (Sobal and Stunkard, 1989: 269). They thought that there were three ways of explaining this inverse relationship in developed societies – obesity could affect socio-economic status, socio-economic status could affect obesity, or they could both be affected by a third factor, such as heredity (Sobal and Stunkard, 1989). The causality was so uncertain, that the author of the 1994 report did not feel able to draw a definite conclusion:

> The fact that lower socioeconomic status tends to be associated with a higher prevalence of obesity in developed countries could be taken to give more credence to an environmental rather than inherited explanation for the cause of obesity although both factors may be important and the direction of causation may be reversed (West, 1994: 17).

Discussions of psychological factors were still seen as an important factor in the development of obesity. In his 1988 textbook, Garrow discussed evidence that obese

individuals eat in response to social and emotional pressures, but noted that the non-obese do so also. He also mentioned studies of the link between obesity and binge-eating, but suggested that binge-eating could be a consequence of dietary restriction rather than a cause of obesity in its own right. Ultimately, he argued that research has not been able to identify any psychological disorder that was a defining feature of the obese (Garrow, 1988: 129). He also argued that the existence of weighing devices such as bathroom scales, and the sale of diet books, showed that the majority of people take action to restore or maintain their weight within a 'desirable' range:

> If this is so then cognitive factors explain why many people maintain energy balance rather accurately over long periods, although they fluctuate in weight by many kilograms in the short term (Garrow, 1988: 132).

His conclusion was that many people are overweight or obese because they 'do not know, or care' and so need education by professionals in the realities of their situation *(ibid.)*.

Psychological factors remained an important explanation of the cause of obesity and overweight. Other authors in the 1990s discussed the concept of 'reactive eating', eating as a response to psychological distress (West, 1994: 18), or 'externality theory' which suggested that obese individuals were more responsive than average to food cues (food accessibility or attractiveness) and, therefore, eat more when food is readily available. This last theory had become less fashionable by the mid 1990s, but new evidence on the link between 'externality' in eating behaviour and BMI subsequently led to calls for more research in this area (BNF, 1999: 84 - 5). The concept of 'dietary restraint'- the idea that there is a link between dieting and abnormalities in the eating behaviour of the obese - was also discussed during the 1990s, but the British Nutrition Foundation report of 1999 noted that there was 'controversy' about the direction of cause and effect, and thus about whether restraint led to eating disorders or was a method for maintaining a healthy body weight (BNF, 1999: 85 - 6). In a similar fashion, the report noted that conditions such as binge eating disorder, emotional eating, food addiction and night eating syndrome tended to be defined as psychological disturbances associated with obesity rather than as the causes of it. However, the report concluded that:

> In general it has been difficult to separate cause from effect in the relation between obesity and emotional disorders. A current view is that personality and emotional factors play only a minor role in the aetiology of obesity, but may be important in relation to responses to treatment (BNF, 1999: 90).

Insofar as the report discussed the behavioural and social aspects of eating, it referred only to the externality theory and to ideas about the relationship between the sensory characteristics of food ('palatability') and the food preferences of the obese and the non-obese. The report argued that obesity and dietary restraint were understood to be linked to increased responsiveness to particular foods, and so 'Palatable, high fat, energy-dense foods may, therefore, provoke behavioural problems of weight control' (BNF, 1999: 114). The report's author, thus, reduced eating to an issue of individual choice that was most appropriately studied and treated by the discipline of psychology.

At the same time, the composition of the diet was also seen as an important cause of overeating. Whilst acknowledging the 'complex interplay of physiological, social and psychological factors influencing appetite, satiety and over-eating' (DoH, 1995: 14), the reports of the 1990s tended to identify the fat content of the diet as a key factor, since a high fat diet was thought to encourage increased energy consumption. The results of one study suggested that 'it is easier to overeat fat than carbohydrate and that there are fewer compensatory mechanisms when fat is used to boost energy intakes' (DoH, 1995: 14). Evidence for the role of artificial sweeteners in the reduction of energy intake was conflicting, and for alcohol the evidence on the relationship between intake and BMI was inconclusive (DoH, 1995: 15). Such research was part of a wider investigation in the different metabolic pathways of different foodstuffs (see section 5.3).

In the late 1990s discussions on dietary factors considered epidemiological evidence for secular trends in energy intake, such as the UK National Food Study. This study showed that, since the 1970s, there had been a decline in the overall per capita energy intake, and a shift in composition of the diet as the proportion of carbohydrate declined whilst that of fat had increased significantly (BNF, 1999: 92 - 3). Amongst other data sources, the author of the BNF report describes results of studies such as Seven Countries and MONICA (see section 2.5) as providing 'modest, but

inconclusive, evidence that high-fat/low carbohydrate diets favour the development of obesity' (BNF, 1999: 100). This was one way in which the enormous quantity of data generated by such research was incorporated into this body of knowledge (see also section 7.5).

Overall, discussions still stressed the complex causes of excess body weight, regularly quoting Garrow's summary: 'the aetiology of obesity in man has genetic, social, cultural and psychological components in different proportions in different people' (West, 1994: 18). Similar kinds of statements remained common throughout the 1990s:

> Recent increases in the prevalence of obesity show the importance of environmental factors in determining the onset of this condition. Many factors interact to induce weight gain, including behavioural, physiological, genetic, medical, therapeutic and psychological processes. To identify a single factor as the cause of obesity in a patient oversimplifies a complex process (SIGN, 1996: 5).

However, it is notable that many of the reports published at this time also tended to bracket out wider, what I have labelled 'environmental', causes of obesity and overweight, and give greater weight to individual or personal factors. Thus the SIGN report's discussion of particular factors conducive to weight gain focused on individual factors including pregnancy, medical and therapeutic causes (endocrine disorders and drug treatments) and genetic disposition (SIGN, 1996: 6-7). It was only in accounts of the behavioural causes of excess body weight that environmental factors were referred to, often obliquely, when the authors' described the role of physical inactivity, snacking, energy dense and highly processed diets and alcohol consumption (SIGN, 1996: 6). But, these practices were described as if they were largely the product of individual choices, rather than socially stratified differences in, for example, access to particular types of food or leisure activities.

This focus on individual behaviour was retained in the policy recommendations that were made in some reports published in the 1990s. For example, the Department of Health's Nutrition and Physical Activity Taskforces recommended a reduction in the average percentage of food energy derived from fat and an increase in average levels of physical activity. When these guidelines were translated into specific

recommendations they became uncontroversial suggestions to improve consumer knowledge through information and education, make low fat products more widely available and develop voluntary national guidelines for caterers (DoH, 1995: 19 - 20). The recommendations on physical activity became even more modest suggestions to encourage individuals who take no exercise to aim for one period of 30 minutes of moderate activity a week, and to increase both the number of individuals who take moderate activity 5 times a week and those who vigorously exercise (DoH, 1995: 20). The only other significant suggestion was 'a mass media campaign to improve awareness amongst professionals and the public of the benefits of physical activity – particularly moderate activity' (*ibid.*). During this period the approach of increasing physical energy expenditure in order to lose weight was no longer routinely described in terms of 'exercise' or 'physical exercise'. These terms carried connotations of drilling, training and the need for specialist equipment and facilities. In the 1990s, such behaviour was re-labelled 'physical activity', and its non-specialist, cumulative and mundane character was stressed. Activities such as walking, cycling, gardening and housework, that fitted this new definition, were also given an important role in preventative strategies (see chapter 8).

The relevant section of the BNF report began with an acknowledgement of the complexity of the causes of obesity:

> Obesity is a complex syndrome with multifactorial origins. Its aetiology can range from the purely molecular (e.g. Prader-Willi and other obesity syndromes) to the purely social (e.g. Sumo wrestlers). Most cases probably cluster towards the middle of this spectrum and can best be described as the result of an adverse 'obesogenic' environment working on a susceptible genotype (BNF, 1999: 37).

This shows the developing idea of environmental causes of obesity, and was the first reference to the concept of the 'obesogenic' environment in these documents. Wider environmental factors such as food industry marketing policies, government policies, the structure of food retailing, and changes in food composition (which refers to the use of artificial sweeteners and lower fat products) were discussed in this report. A very careful framing of food preference in the first half of the chapter (arguing that palatability has no effect on long term food choice) then allowed the authors to conclude that:

> There is no strong evidence to suggest that the marketing policies of the food industry directly impact an individual's predisposition to obesity … The main impact of the EU's Common Agricultural Policy on the UK diet has been via altering the relative price of major food product groups and has been broadly positive from a nutritional point of view (BNF, 1999: 114).

Presumably, the involvement of several representatives of major food manufacturing companies and government departments like MAFF had some influence on the formulation of these conclusions.

From the mid 1990s, obesity science authors start to provide more general explanations of the causes of obesity and overweight that began to include arguments for possible environmental causes, such as increasing consumption of processed foods and increasingly sedentary lifestyles. However, the policy recommendations, that authors also provided, largely still addressed individual behaviour, rather than wider 'environmental' causes.

5.5 Conventional treatments for obesity and overweight

As I described above, Garrow's reformulation and promotion of BMI-based definitions of overweight and obesity meant they had become the standard definitions used within obesity science (see section 4.3). The embedding of these BMI-based categories into clinical practice in turn contributed to their consolidation. For example, Philip James used Garrow's standard definition of obesity as a method of categorising potential patients, as shown by table 5.1 (below).

Grade of obesity	Body mass index	Management strategy
0	20 – 24.9	Patient 'normal' assess basis for anxiety (*sic*)
I	25 – 29.9	Community based group therapy
II	30 – 40	Individual attention, detailed management
III	> 40	Special measures e.g. surgery

Table 5.1 (James, 1984: 636)

James used this classification to outline what he saw as appropriate treatments for different groups of patients. Proposed treatment for patients with grade 1 obesity

involved advice to follow a low fat, high carbohydrate diet, attend a group such as Weight Watchers and have their weight monitored;[79] treatment for patients with grade 2 obesity involved the use of very low calorie diets based on liquid formulas, behaviour modification focusing on understanding the individual's eating habits and anorectic drugs such as fenfluramine; treatment options for patients with grade 3 obesity included those for grade 2 obesity plus jaw-wiring and abdominal surgery such as by-pass surgery or stomach stapling (James, 1984: 644 - 57). Unsurprisingly, given the time of writing, this list was not substantially different from the treatments recommended during the 1960s and '70s, as outlined in chapter 3. Garrow also categorised patients on the basis of BMI into grades of obesity and gave a breakdown of appropriate treatment strategies that I have reproduced in table 5.2.

Treatment strategy	**Grade III**	**Grade II**	**Grade I**	**Grade 0**
Diet				
Starvation	NO	NO	NO	NO
very low calorie	Possible	NO	NO	NO
conventional	YES (1)*	YES (1)	YES (1)	NO
Milk	YES (2)	YES (2)	Possible	NO
jaw wiring/cord	YES (3)	Possible	NO	NO
exclusion surgery	Possible	NO	NO	NO
Drugs				
Anorectic	Possible	Possible	NO	NO
thermogenic	NO	NO	NO	NO
Physical training	NO	Possible	YES	YES
Reassurance	NO	NO	Possible	YES

* these numbers refer to preferred treatment choices within each grade of obesity

Table 5.2 (Garrow, 1988: 185)

These treatment options were similar to the ones outlined by James. Garrow's basic argument was that the more overweight a patient, the greater the number of treatment options that should be considered: 'everything should be done to encourage the Grade III obese person to tackle the problem sooner rather than later, since with time the health risks will become greater and weight loss will become more difficult to achieve' (Garrow, 1988: 192). He ruled out starvation and thermogenic drugs for all

[79] This programme was extended by a further refinement based on the presence of a series of additional risk factors such as smoking, high blood pressure, high blood cholesterol, family history of CHD and other CVDs and central distribution of fat in patients with grade 1 obesity that led to more specific and tailored dietary and activity changes (James, 1984: 643).

of his patients, and described exercise as 'impractical and possibly dangerous' for this group of patients. Treatment for this group, therefore, should rely heavily on different kinds of dietary regimes. For grade 2 patients, Garrow saw specialist techniques such as jaw-wiring as a last resort, whereas he now recommended physical exercise. He stressed that this class of obesity should still be taken seriously:

> It is the job of the therapist to guide and encourage the patient, but with grade II or III obesity it is very rarely ethical to console the patient by saying that weight loss is not very important, so not to worry too much (Garrow, 1988: 202).

For Garrow, the selection of treatment options for grade 1 obese patients presented different problems than those of the other grades. Firstly, the large number of potential patients meant that treatment could not be offered on a one-to-one basis. Secondly some GPs resisted treating obesity as part of general practice due to the ineffectiveness of the available methods of treatment (see section 5.6). Garrow did not accept this argument, suggesting that it was easy to monitor a patient's weight: 'The doctor who does not keep an accurate record of the patient's weight change every 2-4 weeks cannot claim to be treating obesity seriously, and has no right to complain if the results are poor' (Garrow, 1988: 208). He thought that, despite their lack of consistent success, one solution to both these problems was the use of slimming clubs:

> a proper integration of non-profitmaking slimming clubs with professional dieticians as group leaders, backed up by a hospital service for special cases, is a practical and economical way of providing care for the large numbers of moderately obese people in the community (Garrow, 1988: 212).

Garrow saw this as more economical for the health service and, in terms of lost work hours (although not money), the patient. However, these would be slimming clubs run with the approval of medical and nutrition science, and not solely for profit. Other authors also framed the treatment of this category of patients as a largely non-medical activity:

> For those who are overweight (BMI 25 – 30) medical treatment is unlikely to be necessary, but it is important to prevent further weight gain and a conventional diet may be used in an attempt to reduce the BMI to 20 – 25 (West, 1994: 19).

For grade 1 obesity, the main treatment should be dietary, and Garrow gave an example of a commonly recommended 1000 kcal a day diet in the appendix. It was based on eating three meals a day and strict limits on the consumption of sugar, fat, sweetened drinks, alcohol, cereals and baked goods, some fruit and pulse and 'slimming foods'. Finally, his discussion of the treatment of grade 0 patients – he quoted a reference to 'thin fat people' – clearly refers to people who would now be described as suffering from disorders such as compulsive eating, bulimia and anorexia (Garrow, 1988: 217 - 20).[80] Although he recognised that the majority of these patients were female, and that compulsive eating was one common reaction to prolonged dietary restriction, Garrow explicitly denied that a link existed between weight reduction and eating disorders (Garrow, 1988: 219). Eating disorders were the professional concern of psychology not obesity science (see section 5.4).

The list of treatment options proposed by obesity science researchers throughout this period still included dietary methods, drug treatment and behaviour therapy and self-help groups. Surgical techniques were discussed more often than in early obesity science, but still seen as an uncommon option, a last resort for seriously obese patients (who had tried all other techniques unsuccessfully). There were many different forms of dietary treatment – including calorie-counted, carbohydrate restricted, fat-restricted and high-protein ones (Garrow, 1987: 111-4). Dietary restriction was still considered as 'the mainstay treatment for all grades of obesity' and could be supplemented at low cost by minimal intervention strategies e.g. verbal instructions from a GP on how to lose weight (West, 1994: 20).

Due to its intended audience of general practioners, the SIGN report focused largely on the treatment of obesity and overweight within primary healthcare: 'Given the very large number of overweight and obese patients in Scotland in need of medical care, it is inappropriate for obesity to be managed in a hospital setting on a routine basis' (SIGN, 1996: 16). The treatment methods that the SIGN report considered appropriate for primary health care included exercise, behavioural advice and weight loss diets. The report argued that support from the patient's family, health professional or community was an important additional component of such approaches (SIGN, 1996:

[80] Presumably these were the majority of grade 0 individuals that hospital practitioners, rather than GPs, would encounter.

23). Exercise combined with diet was seen by the authors of this report as more effective than either method in isolation for promoting fat loss, and therefore schemes to increase physical activity should be included in all weight management schemes (SIGN, 1996: 25). They divided the population into four categories depending on their activity levels (sedentary, irregular moderate, regular moderate and regular vigorous activity) and distinguished between moderate intensity activities (brisk walking, climbing stairs, heavy DIY and spring cleaning) and vigorous and intense activities (squash, brisk hill walking, running and heavy lifting). Their aim was to provide encourage individuals to increase their levels of physical activity: 'to convert inactive children and adults to a pattern of "active living" where they are on their feet for 4 hours daily' (SIGN, 1996: 67). Research had found that home-based, informal and unsupervised activities were most successful, and so the author suggested that 30 minutes of brisk walking every day should be promoted for the purposes of weight maintenance (*ibid.*).

The weight loss diets recommended in this report reflected orthodox nutritional advice to increase consumption of unrefined starchy carbohydrates and fruit and vegetables, and reduce consumption of fat, sugar and salt (SIGN, 1996: 57 - 59). Such 'moderate deficit diets' of 1200 or 1300 calories a day produced weight losses of half of a kilogram a week for 12 to 24 weeks: 'The average weight loss after one year is 6% of initial weight, but this depends on a support system and follow up to limit weight regain' (SIGN, 1996: 24). The report noted that very low-calorie diets (800 calories a day) had been shown to produce rapid weight loss in the short-term, but not result in long-term weight maintenance (SIGN, 1996: 25). This was part of an increasing emphasis on the importance of weight stability, as opposed to repeated attempts at weight loss followed by rapid regaining, which came to be labelled as 'weight cycling' in future reports (see chapter 8).[81]

Specific dietary treatments varied between different reports. The treatments recommended in the BNF report of 1999 were low-calorie diets (800 – 1500 kcal per day), very low calories diets (less that 600 kcal per day) and the milk diet (1.5 to 2 litre a day plus nutritional supplements, equivalent to 800kcal) (BNF, 1999: 151-7).

[81] Ernsberger and Haskew were the earliest of my sources to discuss this link arguing that 'Temporary weight loss may actually promote further accumulation of weight in the long run' (1987: 38).

Dietary treatments had been heavily researched and so the report's chapter on the topic contained discussions about the factors affecting compliance with particular diets and the role of psychological support and behaviour modification, and the authors addressed the argument that dietary treatments might do more harm than good. Having refuted arguments that dieting inevitably led to further weight gain or was linked to the development of eating disorders, the authors argued that the role of such diets should be shifted from a short-term approach of 'going on a diet' to a much longer-term one of 'weight management'.

> Any treatment programmes for obesity should address, not only the problem of weight reduction but should also include measures to help with the maintenance of lowered weight…. The ability of a treatment to maintain long-term weight reduction is as important as its ability to cause the initial weight loss (BNF, 1999: 145).

This would involve permanent changes in eating habits in order to lose weight and maintain this lower weight for the foreseeable future (BNF, 1999: 164). This framing was linked to a growing stress on the prevention of weight gain that occurred in this period (see chapter 8). The BNF report's chapter on physical activity also outlined a very similar approach to that of the SIGN report where:

> Physical activity should be included as an essential part of obesity treatment, with patients supported in their efforts to steadily build a daily routine involving moderately intense exercise, such as brisk walking, more incidental or opportunistic movement and physical work and reduced time in sedentary pursuits (BNF, 1999: 174).

Again, the report saw this process as long-term: the overweight and obese should be encouraged to make permanent changes in their pattern of physical activity. The report also included discussion of the environmental changes that could be made in order to increase levels of activity among the population – suggestions include 'traffic free' (i.e. car-less) routes to schools and shopping areas; greater access to parks and sport facilities; rewards for journeys undertaken on foot or by bike rather than by car; and alternatives to lifts and escalators (BNF, 1999: 175). This was part of the growing discussion of the environmental causes of overweight and obesity (see section 5.4).

By the 1980s, advocates of pharmaceutical treatments for obesity had proposed a number of different types of potential drug treatments, including biguanides which were related to diabetic treatments, bulking agents to compensate for lack of dietary fibre, thyroid hormones to increase metabolic rate, and anorectic drugs (relatives of amphetamine that suppressed appetite) (Simpson et al., 1987: 118-21). Obesity scientists were cautious about recommending pharmaceutical interventions, however. One influential survey of treatment options, for instance, recommended that only the anorectic drugs, such as diethylpropion, phentermine and fenfluramine, should be considered as treatments, and noted that most of them had significant side effects. Given that weight gain was normal after drug therapy, the same survey argued that the use of anorectics 'can only be justified if there is a clearly defined, short term need for weight loss' (Simpson et al., 1987: 123).[82]. But by the 1990s, some of these drugs had been withdrawn from the market due to their side effects and dexfenfluramine was the most widely used pharmaceutical treatment, although SSRIs such as fluoxetine were awaiting approval by the US Food and Drug Administration (West, 1994: 24).

In all of the above discussions, drug treatment was considered suitable only for those patients who had already attempted and failed to lose weight using the more orthodox methods of diet and exercise. However, the BNF report is more specific about the criterion:

> It may be appropriate to consider drug therapy for those patients with a BMI of 30 or greater who have failed to lose this amount of weight [10%], or whose weight is no longer decreasing after at least 3 months of structured dietary management (BNF, 1999: 182).

Given the low levels of success reported using dietary treatments, this formulation appears to allow for the prescription of such drugs to the maximum number of obese patients. The drugs recommended for such treatment fell into three categories – depending on whether they act on the serotoningenic pathways (fenfluramine and dexfenfluramine), catecholamine pathways (phentermine) or serotoningenic and noradrenergic pathways (sibutramine). Drugs previously prescribed, such as diuretics, amphetamines and thyroxine, were described as not appropriate in the treatment of

[82] This usually meant as preparation for elective surgery, but non-medical reasons such as being able to enlist in the armed services could also count.

obesity (BNF, 1999: 183). Drug treatment was described as short-term – initially for three months and only extended if the patient has achieved a cumulative 10% weight loss. However, the report warned that

> The absence of extensive safety data means that the use of anti-obesity drugs must be very closely monitored, with patients subject to very regular medical review (BNF, 1999: 186).

The withdrawal of fen-phen (a combination of fenfluramine and phentermine) in the 1990s after patients developed serious cardiac problems suggests that this injunction was not followed closely enough by some practitioners (Gard and Wright, 2005: 162).

Meanwhile, because patients found it hard to maintain weight loss using diets or drugs, making both techniques effective only in the short-term (if at all), some researchers explored other approaches in the hope of obtaining better results. These additional approaches included behavioural therapy and self-help groups. Albert Stunkard used the results of two clinical trials to argue that

> behaviour therapy can help obese patients to lose moderate amounts of weight and to maintain these weight losses for at least one year. The better maintenance of weight loss makes behaviour therapy appear superior to the other treatment modality for obesity – pharmacotherapy (Stunkard, 1987: 128).

As a psychologist (who conducted one of these trials) rather than a pharmacologist, Stunkard would be expected to argue along these lines. In his research, behaviour therapy consisted of self-monitoring so the patient kept precise records of what they ate, where, when and why they ate; control of the stimulus preceding eating by planning what to eat and minimising opportunistic eating; control of the act of eating using techniques such as chewing thoroughly, leaving food on the plate and not doing anything else whilst eating; reinforcement of prescribed behaviours with praise from friends and family and material rewards; cognitive restructuring to counteract negative monologues; nutrition education; and physical activity (Stunkard, 1987: 139). Whilst it might have been successful, it was also a demanding and time consuming process.

By the 1990s, behaviour therapy was seen by obesity science researchers as an important technique because it re-educated individuals' eating habits and allowed the maintenance of a new lower weight, and also because it was cheaper than other forms of treatment as it relied on group sessions with a dietician, rather than individual treatment by a doctor (West, 1994: 29). The tendency to recommend the use of behavioural techniques was boosted by research findings showing increased success with long term dietary modification using such techniques (SIGN, 1996: 25). All of the reports published in the mid to late 1990s period recommended some combination of the following practices: patient education about food labelling and preparation: self-monitoring and recording of eating behaviour; and understanding of the psychological and habitual elements involved in food consumption. Examples of the last category included eating as a form of self-medication and how to deal with situations that may give rise to 'inappropriate' food choices such as festivals or eating out (SIGN, 1996: 66). It was recommended that these kinds of techniques were used in group situations by health professionals such as dieticians, rather than by doctors.

These authors' accounts of behavioural treatments argued that obese individuals do not demonstrate an 'eating style' different from the non-obese. Despite this recognition, they still maintained that cognitive behaviour therapy (CBT) - self-monitoring, stimulus control and modifying self-defeating thought processes - had a significant role to play in individuals' attempts to lose weight. The initial success of such approaches had, however, led to unrealistic expectations concerning their long-term effectiveness.

> There was initially optimism that the weight loss achieved by CBT, although usually modest, would be long lasting, as it would permanently alter the patient's attitude to food. In fact, longer-term studies show that the affect of CBT alone disappears by five years after therapy (BNF, 1999: 181).

Research then focused on the possibility of using CBT to prevent weight regain, whilst recommending restrictive diets and exercise to achieve greater initial weight loss (*ibid.*). This pattern of treatment 'hype' followed by lowered expectations and finally incorporation into routine weight management practices, was similar to that which occurred when new pharmaceutical treatments were being promoted.

A related approach, which provided another kind of social support in the weight process, was the self-help group. However, Audrey Eyton, the founder of Weight Watchers, argued that the label self-help was not entirely appropriate.

> Although group therapy under a peer who serves as a role model is an essential part of the treatment, it appears to work well only when the leader follows a precisely structured policy. In many cases this policy has been developed by a highly professional commercial organization using all the facilities of modern market research, professional medical, nutritional and behavioural consultation (Eyton, 1987: 140).

We can read Eyton here as staking a claim to professional expertise in the area of obesity treatment whilst being careful to acknowledge that of other, more prestigious professional groups, and to counteract the negative image of the weight loss industry, particularly among medical practitioners, as unscrupulously profiting from people's desire to lose weight.

The final form of treatment considered was surgery. In the 1980s, a description of different types of surgical methods was labelled 'unusual methods of obesity control' (Gries, 1987: 147), and, in the early 1990s, techniques were still limited to different kinds of stomach stapling and jaw wiring (West, 1994: 25 - 27). By the late 1990s, new methods were being developed but the basic distinction was still between mal-absorption techniques such as jejuno-ileal or gastric bypasses, and techniques to restrict intake which include jaw wiring and stomach stapling or banding (BNF, 1999: 187-9). The long term effects of such surgery was also beginning to be studied in the Swedish Obese Subjects (SOS) study which monitored the health of approximately 2000 individuals who had undergone such surgery, compared to a similar number who underwent non-surgical treatment (BNF, 1999: 189). The BNF report summarised the findings of this research:

> In severely obese Swedish patients, gastric surgery causes massive weight loss, maximal about one year after operation, and this greatly improves the health and quality of life compared with weight-stable obese controls. Weight loss of this magnitude, attained by non-surgical methods, confers similar benefits…However, few patients achieve this level of weight loss, even with the assistance of drugs (BNF, 1999: 190).

Because of the complex nature of these operations, the BNF report argued that they should be performed by experienced surgeons in specialist centres, on patients who had been fully informed of the risks involved, and, even if successful, 'lifelong monitoring is required post-operatively' (*ibid.*). Such surgery was acknowledged, in this report, to have a significant risk of complications, including death, and to leave the patient at life long risk of side effects, that included involuntary regurgitation and malnutrition. This was one of the few acknowledgements of the hazards of weight loss treatments in this literature, although the side effects of drugs, especially those that had been withdrawn from the market, were occasionally discussed (OHE, 1969: 24-5, RCP, 1983: 48-9). On the other hand, Ernsberger and Haskell discussed this topic in detail:

> Because virtually all weight reduction methods are only temporarily effective, they can be hazardous indirectly due to the regaining phase. However, many of the most popular weight-control techniques have direct hazards also (Ernsberger and Haskew, 1987: 40).

Ernsberger and Haskell gave a chronological list of 'hazardous treatments for obesity', including a detailed description of the negative health effects of each treatment. This began with an early version of the low carbohydrate diet (from 1862), included the use of laxatives (from the 1920s), amphetamines (from 1937), total fasting as an in-patient (from 1959), intestinal bypass (from 1969) and jaw wiring (from 1974). Their conclusion was that

> In the light of the relatively mild and uncertain hazards associated with adiposity, and the potential contribution of iatrogenic disease to these hazards, the use of dangerous weight control measures appears to be highly questionable. We urge that hazardous treatments for obesity…be replaced with nutritional counselling and exercise training, supported by behaviour modification (Ernsberger and Haskew, 1987: 48).

Ironically, this critique was preceded by advice to adopt a diet low in saturated fat, cholesterol, salt, sugar and alcohol, along with increased physical activity to 'promote gradual and permanent, albeit modest, loss of weight' (*ibid.*), that was very similar to that given by the practitioners whose treatments they had just heavily criticised.

5.6 The difficulties of successful treatment and a new approach of 'modest' weight loss

The difficulty of successful treatment had been an ongoing subject of discussion in obesity science. From early on, it had been acknowledged that all the recommended treatments had relatively low success rates: 'it is evident obesity is difficult to treat' (DHSS/MRC, 1976: 63). Philip James subtitled his 1984 chapter on treatment 'The Constraints to Success' (James, 1984: 635) which suggests he did not have great confidence in the measures he was about to describe. On the success of dietary treatment, Garrow stated that:

> A jeremiad on the inefficacy of dietary treatment of obesity is often quoted: 'Most people will not stay in treatment for obesity. Of those who stay in treatment most will not lose weight, and for those who lose weight most will regain it' (Stunkard 1972). There is some truth in this aphorism, especially when the results of treatment are followed for several years. To some extent the prophecy of failure is a self-fulfilling one: if both patient and therapist expect to fail they will … The poor results for dietary treatment of obesity apply only to outpatient regimens: virtually any amount of weight can be lost by dietary methods given time and the complete control of the diet in a metabolic ward (Garrow, 1987: 115).

Garrow's parallel between unsuccessful attempts to lose weight and in-patient treatment does not adequately address the complex issues involved in long-term failure rates of dietary treatments (see chapter 6). Later in the same discussion, he argued that 'The vast majority of people who lose weight successfully do so by following diets they have read or heard about, and never appear in any statistics' (*ibid.*); ultimately, he implied, success depended on the motivation of the patient rather than medical treatment.

The 1995 Health of the Nation report argued that the targets for reducing the prevalence of obesity can be met in three different ways: by helping the obese to lose weight, by helping the overweight avoid becoming obese and by preventing the whole population becoming fatter. The report recommended focusing on the last two since, as Garrow had argued, obesity treatment in adults was 'limited in its success' (DoH, 1995: 19). In this respect, the Health of the Nation report reflected a wider shift toward prevention on the part of government departments and professional bodies

involved in the planning of health care services (see section 3.2), one facet of which was an acceptance of the limited possibilities of treatment. A similar acknowledgement occurred in the 1995 WHO report:

> Because a large proportion of the adult population in industrialized societies will be overweight or obese and because weight loss therapy is ineffective unless closely supervised and followed up, not all overweight or obese individuals will qualify for intervention. Priorities [*sic*] should be given to those at highest risk, with the primary focus on reducing the risk profile rather than on weight loss *per se* (WHO, 1995: 329).

Coming as it did from the WHO, this frank acknowledgement of the difficulties of weight loss therapy builds was framed primarily for an audience of readers with responsibility for planning health care and developing appropriate policies.

The difficulty of identifying successful treatment options also led to research into other treatment approaches. In the 1990s, the potential benefits of 'relatively modest weight loss' were first discussed. A 1992 study by Goldstein demonstrated that an individual with a BMI of 40 who lost 5% of his body weight (reducing his BMI to 38) would reduce his mortality risk by 12%, although the report's author was also careful to say that greater weight loss would lead to increased benefits, so as not to undercut the argument for achieving 'desirable weights' which were still one of the two main aims of treatment (West, 1994: 19).[83] Other reports also focused on the health benefits of 'modest' weight loss, usually defined as between 5 and 10 kilograms:

> A 10kg weight loss results in many benefits e.g. >20% fall in mortality, >30% fall in diabetes related deaths, >40% fall in obesity-related cancer deaths … [cites decreases in blood pressure, fasting glucose and blood cholesterol levels] … Modest weight reductions of only 5-10 kg improve back and joint pain, lung function, breathlessness, and reduce the frequency of sleep apnoea (SIGN, 1996: 12).

> A weight loss of between 5-10% of the initial body weight is associated with clinically useful improvements in terms of blood pressure, plasma cholesterol, triacylgyclerols and HDL cholesterol, and a significant improvement in diabetic control (BNF, 1999: 150).

[83] The other was weight stability (West, 1994: 20).

The growing importance assigned to weight stability also led to arguments for the benefits of 'modest' weight loss: 'a small sustained weight loss is preferable to large weight losses followed by weight regain' (West, 1994: 31). This argument built on data about the rarity of significant long-term weight loss from dieting and exercise alone, and anticipated later discussions of the dangers of repeated attempts at weight loss followed by rapid regaining which came to be labelled as 'weight cycling' in future reports (see chapter 8).

Combined with this new emphasis on modest weight loss was a more radical re-definition of the goal of treatment. This involved a shift from a focus on weight loss as an end in itself, the health benefits of which were assumed but not measured, to one that focused on demonstrable improvements in health risks.

> Overweight patients frequently assume, as do some health personnel, that the principal goal should be a return to an ideal weight. Yet, in medical terms, great health gains can be achieved if a patient's smoking, dietary and exercise habits are improved to reduce risk factors without weight reduction (SIGN, 1996: 20).

The authors of the 1995 WHO report (see chapter 7) shared this new approach: 'Weight loss is recommended but weight loss *per se* should not be the primary target of intervention' (WHO, 1995: 329). The reasoning behind this change was that:

> Until recently, the response to intervention was usually assessed in terms of attaining "ideal body weight" or reducing body weight to below a certain BMI cut-off or "percentage over ideal body weight". It is no longer clear that such goals are optimal. For some people they are unrealistic: in most obese individuals they imply large sustained weight losses, which few are able to achieve unless they are enrolled in long-term programmes with extensive follow-up (e.g.> 5 years). Moreover, substantial improvements in risk factor profiles have been documented in obese individuals who lost only moderate amounts of weight and would still be classified as overweight or obese… Finally, there is no evidence that large weight losses either have beneficial effects or reduce mortality, and the more extreme diets needed to produce large weight losses may increase the likelihood of relapse (WHO, 1995: 330).

These claims built on the approach of 'modest weight loss', but went much further by suggesting that there was no evidence for benefits to health or improvements in mortality rates from large weight losses.

5.7 Conclusion

In this chapter I have outlined developments in biomedical understandings of the causes of overweight and obesity as they were represented in largely British policy documents of the 1990s. Basic metabolic, endocrinological and physiological research continued to use the concept of energy balance as foundational, while the concept of set point for body weight was widely discussed at times. There was a new focus within this policy-oriented literature on small-scale chronic energy imbalances combined with low levels of physical activity leading to overweight and obesity. Obesity science accounts of the causes of obesity initially focused on the individual level, trying to understand the psychology of over-eating. Complementary accounts considered the social level, where the authors discussed the relationship between body weight and socio-economic class, dietary composition, food choice and physical activity. The idea of physical activity was adapted to include activities such as housework, climbing stairs and DIY which had not generally been included within the scope of "exercise". From the mid 1990s, these authors began to increase the range of causes they considered as the environmental or structural causes of overweight and obesity began to be discussed and the term 'obesogenic' environment was introduced.

A version of the BMI classification discussed in chapter 4 was widely used as the basis of policy recommendations about appropriate individual treatment for overweight and obesity. The recommended treatments in primary health care remained weight loss diets combined with exercise and, if possible, behavioural therapies. Supplementary pharmaceutical treatments might be offered to patients who did not initially manage to lose weight. In specialist clinics, it was recommended that surgery should be offered to the minority of patients whose weight was seen to pose such health risks that it outweighed the risks of such procedures. However, the stated aim of treatment was beginning to change towards to a much longer-term approach that focused on changing eating habits, improving levels of physical activity, and on the health benefits of 'modest' weight loss and subsequent stabilisation, rather than a return to 'average' or 'ideal' weights. Such an approach was at least partly justified by greater acceptance of the difficulties most individuals found in maintaining long-term

weight loss. Importantly, accompanying this was a shift from a policy focus on weight loss *per se*, to a focus on demonstrable improvements in risk status.

CHAPTER 6: ANALYSING OBESITY SCIENCE

6.1 Introduction

In the previous two chapters, I have outlined the development of British obesity science in the 1980s and 1990s; the way that a range of scientific findings were incorporated and represented in a series of policy-oriented documents. I have described how research into the physiology of appetite and digestion was combined with data generated by large scale epidemiological studies to create an argument about the increased mortality associated with excess body weight, and with clinical knowledge to generate recommendations about treatment options. This body of knowledge about the definition, causes, effects, treatment and, eventually, prevention of obesity and overweight was a hybrid entity, assembled from heterogeneous sources, often as part of government-appointed committees of expert medical professionals. The group of researchers and clinicians who formed a policy coalition around obesity and overweight in the late 1960s had become increasing successful in persuading governments to listen to their message. In the process, they created a body of policy-relevant science that framed excess body weight as a public health problem using standard measurement and models.

In the judgements about health that are a crucial part of obesity science there was an assumption that thinness equalled healthiness. Many critics have questioned this assumption (Ernsberger and Haskew, 1987, Bacon, 2008, Campos, 2004, Gaesser, 2002, Gard and Wright, 2005, Monaghan, 2005, Oliver, 2006). Those who come from a background in physical activity often point out that this approach privileges body size and shape over other indicators such as cardio-respiratory fitness, blood sugar levels or cholesterol levels (Gard and Wright, 2005). Research shows that such indicators can often be improved more easily than body weight (SIGN, 1996), and studies into the effects of physical activity on health and mortality have been conducted since the 1950s (Morris and Heady, 1953, Morris et al., 1966, Morris et al., 1973). However, research and policy focusing on body size and shape seems to have become more widely accepted than that focusing on physical activity and fitness, certainly until the end of the 1990s. This may be because of the widespread acceptance of dieting for weight loss within British and American popular culture

throughout the second half of the twentieth century (Schwartz, 1986, Stearns, 2002). It may also be connected with the 'obviousness' of body weight as a sign of ill-health. Without denying the complexities of agreeing standards relating health to body weight (see below), it is possible to suggest that a visual inspection of an individual's body seemed to be a much more straightforward procedure than assessing their physical fitness.

In the preceding chapters I have charted the development of two important aspects of the framing of body weight as a public health problem within obesity science: the body mass index and the energy balance model of weight regulation. In the present chapter, I will consider the important advantages of the BMI for obesity science. In particular, I will argue that without such a standardised measure, it would be difficult to talk meaningfully about an epidemic of obesity, whether or not such terminology is actually justified by the existing epidemiological data.

Then, I will switch focus to analyse the model of the body used in obesity science. In common with much of biomedicine, obesity science used a mechanistic understanding of human physiology which sees obesity and overweight as the result of a positive energy imbalance due to long-term consumption of more calories than are expended. The simplicity of this model allowed it to be shared by many different research areas, but also meant that it could not account for the low success rates for all conventional weight treatments. Explanations of eating and eating behaviour considered by obesity science were dominated by individualised accounts largely based on psychology or economics. This focus on the individual left very little room for more social or structural explanations and therefore gave a very impoverished account of individual eating behaviour and changing patterns of consumption.

6.2 The advantages of BMI for obesity science

STS researchers have written on the wider social effects of standards and classification systems (Bowker and Leigh Star, 1999). Critics of obesity science have also written extensively on the problems of using BMI-based definitions of overweight and obesity (Ernsberger and Haskew, 1987, Bacon, 2008, Campos, 2004, Gaesser, 2002, Gard and Wright, 2005, Monaghan, 2005, Oliver, 2006). I am not

going to add to these arguments because they are well-rehearsed, and because those working in the area are aware of the problems with the BMI (WHO, 2000). As I argued in section 4.3, public health statisticians were also highly aware of the effects their redefinitions of overweight and obesity had on the numbers of populations fulfilling these criteria (Kuczmarski and Flegal, 2000: 1078). Instead I want to examine the advantages of the BMI for obesity science, to explain why, in a relatively short period of time, it became the field's standard measurement and definition of the problem of excess body weight.

In chapter 4, I described how obesity and overweight were initially defined using a variety of classificatory schemes and indices. The most common of these was the Metropolitan Life Company's table of ideal weight for height categorised by frame sizes (often the 1959 version), or another definition based on a set percentage above average weight (usually 10 or 20%). Other indices such as W/H, W/H^3 (the ponderal or Rohrer index) and W = H-100 (the Broca index)[84] were also used. There seem to have been disciplinary and regional patterns in these usages: W/H^3 was used in paediatrics, while epidemiological researchers in mainland Europe used the Broca Index until relatively recently (Oddy et al., 2009). One account also states that the Broca index was the basis of the categories in American insurance companies' height and weight tables (Oddy et al., 2009: 7). Often authors used a definition based on the Metropolitan Life ideal weights and referred to other indices as they incorporated results from different studies into their research. These varying definitions and measurements caused several problems for epidemiology researchers. Firstly, varying definitions and measures (often in different units) made it hard to compare the results of different studies. Secondly, using percentage deviation from average body weight like the ideal weight tables meant that, if relative proportions stayed the same, then the same number of people would be defined as overweight or obese, even if the whole population gained significant amounts of weight. Such a relative index can be useful when the measured attribute is stable in the population, but an absolute measure (which does not vary with changing prevalence) would be more useful in a

[84] W = H-100 is the Broca index, and is a rough means of calculating ideal weight; the + 15% sets the threshold for obesity as 15% above this ideal weight.

context where the prevalence within a population is changing, especially if the rationale of such an index is to track this change.

As Ian Hacking describes, W/H^2 was first developed by the Belgian astronomer and statistician, Adolphe Quetelet in the early nineteenth century (Hacking, 2006c). Quetelet was interested in anthropometry and he developed W/H^2 as one way of illustrating differences between populations; it was part of his concept of the 'l'homme moyen' or average man (Hacking, 1990). The association with Quetelet was well known in obesity science and regularly referred to in textbooks. However, unlike these conventional accounts, Hacking tracks these changing meanings of 'the fraction' i.e. W/H^2:

> We should notice how the changes, in the ways that this fraction have been used, reflect a profound shift in interest. We began quantifying the human body and its excellences, and now regard it as a threat, an ever-shifting kaleidoscope of risk factors (Hacking, 2006c:1).

BMI, in its modern form, developed out of 1960s chronic disease epidemiology, but, according to Hacking, one of its first important uses was in a Norwegian survey collecting information about the relationship between levels of underweight and tuberculosis (Hacking, 2006c: 19). Only subsequently did it come to be used primarily to identify and monitor over-weight. This change of use parallels the shift from research into under-nutrition to research into over-nutrition described in section 3.3 (also see below). As I described in chapter 4, the name body mass index was suggested in 1972 by Ancel Keys (Keys et al., 1972), and gradually adopted, despite an alternative attempt to call it the Quetelet Index (Garrow, 1981: 3). Keys, Garrow and George Bray, who developed the first version of the cut-off points to define overweight and obesity, were central figures in the development of obesity science, as well as chronic disease epidemiology, and their recommendations carried considerable authority among obesity researchers.

Like many other indices of bodily measurement - such as blood pressure (Timmermann, 2006) - BMI functioned as both the measurement of a bodily attribute (weight related to height) and also the definition of a condition (obesity/overweight). This dual role makes an analysis of its function within obesity science crucial, since it

simultaneously measured and defined the modern problem of excess body weight. However, until the late 1990s, the combination of varying indices of measurement with varying definitions of overweight or obesity led to a large number of competing definitions – one article lists 18 different classifications that were used in the US between 1942 and 2000 (Kuczmarski and Flegal, 2000: 1076) and a wider search would certainly locate more examples.

The BMI-based definitions of overweight and obesity consist of two elements – the BMI index which I have just described, and the cut-off points which are used to define the levels at which body weight was considered to be excessive and a danger to health. Even in the 1980s and 1990s when the BMI had become widely used, the specific cut-off points used varied between studies. For example, an NIH consensus conference in 1985 decided on a cut-off point for obesity as BMI $\geq$ 27.8 for men and 27.3 for women (NIH Panel on the Health Implications of Obesity, 1985),[85] whereas most British research was then using overweight BMI $\geq$ 25 and obesity BMI $\geq$ 30 for both sexes. This lack of standardisation of BMI cut-off points was part of a much wider set of disagreements about how to define obesity, which includes not just BMI-based measures but also percentages above average body weight (hence the NHANES definitions) or increases in mortality risk (hence the Keys/Bray/Garrow definitions). The review article mentioned above, which was a retrospective account of these changes, also demonstrates some the work involved in trying to develop a consensus in this area, as the authors created an impression of continuity by stressing the similarities between the NHANES definitions to the older Metropolitan Life definitions and the newer lower numerical limits (Kuczmarski and Flegal, 2000: 1077)

As Hacking also points out, one advantage of the BMI was that it was based on readily available data that is relatively cheap to collect, particularly compared to measuring body fat which is difficult and technically complicated and therefore very expensive (see section 4.2).

[85] These were the sex-specific 85th percentile of the BMI distributions in NHANES II for the age group 20 to 29 and were very close to 20% above the 1983 Metropolitan Life medium frame desirable weights (Kuzmarkski and Flegal, 2000: 1076).

> Adiposity, the ratio of body fat to body mass, is the important health indicator, but is fairly expensive to measure by any current technique – and thus comparable to a personal DNA readout. But the BMI is very cheap: stand on a scale, stand under a device that measures height, press two buttons on a calculator (or use one of the innumerable online BMI calculators), and there you have your BMI … A national study of adiposity would have been more informative and cost about a million times more (Hacking, 2006a: 88).

The development of the BMI was an example of epidemiologists using readily available, and therefore cheap, data to create large data sets. In additions, because height and weight have been routinely collected in many areas of medical research, including epidemiology, existing data sets can be easily converted into this new index. Much of the data from large studies, such as Framingham, appears to be been re-worked using BMI cut-off points from the 1980s onwards. The adoption of this simple numerical index allowed epidemiologists and other researchers to directly compare the results of different research studies, and thus investigate, describe and quantify trends in changes in population weight between regions, countries and across time.

The development of classification schemes such as Bray's and Garrow's also included an increasingly large percentage of the population – in one of Garrow's schemes those of BMI 18.5 to 25 were no longer simply labelled 'normal' or 'ideal' weight but 'grade 0 obesity' (Garrow, 1988: 3). The distinction between "normal/ideal" and overweight is one of *kind*, whereas the distinction between grade 0 and grade 1obesity is one of *degree*, and implies a continuum or gradation from low to high risk. As such, it underwrites the idea that even those not "at risk" could be at risk of becoming at risk. This was an enormous expansion in the jurisdiction of obesity science, but it fitted well with the logic of the risk factor approach and the preventive medicine associated with it, which emphasised individual responsibility for minimising health risks by lifestyle changes such as healthy eating and increased activity levels (Petersen and Lupton, 1996).

6.3 BMI and the epidemicity of obesity

As I described in chapter 4, many of the early reports argue from a very limited evidence base that obesity was an important public health problem. Until the 1980s,

the most frequently cited empirical sources were the Metropolitan Life height and weight tables, the 1959 Build and Blood Pressure Study, and results from the Seven Countries study and Framingham, both of which were studies of risk factors for heart disease, rather than of body weight. This meant that, especially for British authors, there was a limited amount of large-scale data available to use in their arguments. Until the 1960s and 1970s, governments did not routinely collect statistics about the physical stature of their populations: 'In the middle of the twentieth century few countries could make general statements about trends in weight gain by their populations.' (Oddy et al., 2009: 225). The subsequent growth in collection of this information was a twentieth century continuation of the processes of nineteenth century government information collection (Desrosieres, 1998, Hacking, 1990), as well as a product of the increasing use of statistics within medicine (Rothstein, 2003).

Because of this lack of data, British authors made heavy use of American sources[86] combined with the limited number of small-scale British studies (which had often been carried out as part of various occupational health investigations). Whilst I argue that the claims of obesity science were developed on the basis of limited evidence, I am not putting forward a conspiratorial understanding of these processes. Claims-making is a normal aspect of biomedical research and clinical practice. Researchers and clinicians need to secure funding by demonstrating the importance of the health issue on which they work, and this involves using the available evidence. Such claims-making is a routine aspect of the activities of such coalitions and their attempts to influence the development of public health policy. However, in the case of obesity science, the timing of these efforts was significant. As I described in section 3.3, medical concern focused on under-nutrition in the immediate aftermath of the Second World War. In particular, protein malnutrition was seen as a pressing problem from the late 1940s and throughout the 1950s. However, the focus of medical concern seems to have switched, relatively rapidly, to a concern with over-nutrition at some point in the late 1950s or early 1960s. The timing of this transition varied between different countries (Oddy et al., 2009: 224-5), but seems to have occurred in many European countries between the late 1950s and late 1960s. This was a rapid change

[86] The Build and Blood Pressure Study, like the 1959 Metropolitan height and weight tables, was still being cited in the 1980s and 1990s.

since the aftermath of the Second World War had meant that, in the late 1940s and early 1950s, there were still food shortages in many European countries.

Despite the speed of this transition, obesity science had become well-established by the 1980s. Judging by the increasing institutional scope of their reports, in the 1980s and 1990s, the arguments of the public health coalition that had formed around obesity were becoming increasingly accepted. More results were being generated by large-scale epidemiological studies, and the WHO MONICA project was set up as a European version of American risk factor research. Results from NHANES II (see section 3.5), showing a big increase in the prevalence of obesity and overweight, were released in the mid 1990s (Kuczmarski et al., 1994). These figures were reported in the popular press and obesity started to be described as an epidemic in the medical and popular press (Pi-Sunyer quoted in Pringle, 1994). This new framing of obesity was largely made possible by the gradual adoption and standardising of the BMI definitions. With an easily handled numerical measure such as BMI, it was much simpler to construct prevalence rates for overweight and obesity, and, therefore, monitor secular changes within populations, and the differences between populations,.

One further aspect of defining and measuring obesity and overweight using this classification was that it facilitated the diagrammatic representation of increasing rates of overweight and obesity. The spread of infectious diseases had often been represented by mapping the geographical spread of numerical increases in prevalence rates. In the late 1990s this method was applied to increasing rates of obesity and overweight in a famous series of slides produced by the Centers for Disease Control (CDC).[87] This animation shows an increasing number of the American states going from blue to red over a period of 15 years, as average rates of obesity increased to levels above 30% (CDC, 2010). It has been argued that this memorable and widely publicised animation – first produced in 1999, but still readily available online – was one of the most important factors in establishing the 'fact' of obesity as an epidemic (Oliver, 2006: 40-3). It is a particularly vivid illustration of a more general co-production (Jasanoff, 2005) of, on the one hand, the BMI as measurement and definition of obesity, and on the other hand the 'epidemicity' of obesity. Without the

[87] This uses Behavioral Risk Factor Surveillance System Survey Data which seems to be equivalent to or incorporate the NHANES data.

BMI to provide a simple way of turning those data into a stark representation of obesity rates, it would have been much harder to argue credibly that obesity was increasing to the extent it justified the label of an 'epidemic'.

6.4 Limits of mechanistic models

As I outlined above, contemporary obesity science relies on a mechanistic model of human physiological processes of appetite and weight regulation. The metaphor of the human body as a machine has a long history within in physiological research (Rabinbach, 1990) and has been developed in various ways depending on the specific organ or processes under investigation. In the context of obesity science, the mechanistic model takes the form of the energy balance model, and one of the most influential British accounts was in the work of John Garrow (see sections 4.4 and 4.5). As I outlined in chapter 5, obesity scientists generally considered that weight stability occured when the energy consumed by an individual was equivalent to their expenditure of energy. Obesity and overweight arose from an energy imbalance due to excess consumption and insufficient energy expenditure. This framing was powerful because it tallied with common sense and, sometimes, everyday experience. Authors also argued that it was based on the first law of thermodynamics (Miller, 1969: 58-9, Jequier, 1987: 17),[88] thereby invoking the authority of physics and adding to the difficulty of questioning its accuracy as a model of individual weight regulation.

In analysing the problems of the energy balance model, I want to follow Gard and Wright (2005) and others, who argue that it is a necessary but not sufficient account of the processes of appetite and body weight regulation. Not only does it not consider the social aspects of these activities (see section 6.5 below) but its simplicity and standardised form does not allow for the consideration of variation either between individuals or specific population groups. Apart from the individual variation in susceptibility to weight gain that is apparent from everyday experience, two examples of such variability are age differences and sex differences. These are acknowledged to result in differences in metabolism, and average BMI is widely acknowledged to

[88] This states that matter can neither be created nor destroyed.

increase from the twenties until old age when weight loss can occur due to muscle wasting. However, this variability was ignored when it came to making recommendations about how to combat obesity. When BMI cut-off points were initially proposed there was discussion about allowing these limits to increase with age but this was ultimately decided against, partly on the grounds that weight stability was best for health (Garrow, 1978: 37, RCP, 1983: 5-6). Women were also understood to be particularly prone to weight gain under the critical periods model (see section 5.3) and physiologically different from men, both in their levels of body fat and the health risks that it gave rise to.[89] However, none of these differences were incorporated into the cut-off point classifications of overweight and obesity. The resulting inflexibility of these standards, combined with the idea that body weight is largely under individual control, does not allow for either the propensity to gain weight with pregnancy and increasing age, or the increasing difficulty that older and sick individuals may find in following weight loss advice**.**

The prevalence of the energy balance model in obesity science derived from the significant overlap between laboratory research into the physiology of appetite and body weight regulation on the one hand, and clinical research into the development of treatments for overweight and obese patients on the other. Physiological researchers need models to study complex systems such as human metabolism, and even with such models the complexity of the systems governing appetite and body weight was daunting. Obesity science combined this laboratory and clinical research with the findings of large-scale epidemiological studies. The simplicity of the energy balance model meant that it provided a common basis for interpreting all these diverse kinds of results.

As STS scholars have argued (Fujimura, 1992), research practices need to be standardised in order to be reproducible. The processes of creating simplified models and common sets of practice lead to the creation of ideal standards – as in the example of the BMI cut-off points discussed above. In the case of body weight regulation the studies that formed the basis of these standards were often done on relatively young,

[89] The data that was initially used to argue for the use of BMI came from the Seven Countries study whose subjects were all men. The differences between men and women are referred to in the quotes in my discussion on body shape in section 3.4, but this is an area that needs more research.

fit men (such as students or prisoners), and so their physiological capacities became seen as standard. The World War II Minnesota Starvation Experiment, supervised by Ancel Keys, took place in a laboratory setting over six months and involved 36 white male conscientious objectors aged 25 to 35, many of whom belonged to religious groups such as Quakers or Mennonites (Kalm and Semba, 2005). Although about under- rather than over-feeding, this experiment was canonical and was regularly cited in obesity science writing. Ethan Sim's experiments in over-feeding Vermont prisoners were less well-known but also regularly referred to, and described in a couple of popular accounts of obesity science (Pool, 2001: 70, Shell, 2003: 79-81). However, patients seeking treatment for weight problems were not usually young, fit men – they were often middle aged or older, female and with other long-term health problems. The discrepancy between research subjects and patients is problematic because patients are unlikely to achieve the weight loss that laboratory research subjects appear to be capable of, and, because of its extreme simplicity, the energy balance model does not acknowledge the differences between these groups. The energy balance model provides a rationale for assuming that findings produced in young men can be generalised to other groups, but it cannot deal with variability between or within groups - even amongst the younger male research subjects studied there was a wide variation in individual's capacity to gain and lose weight in the controlled conditions of a laboratory or a prison.

A final problem with this use of the energy balance model was that when it was transferred into a clinical context it was used with, and served to underwrite, the assumption that an individual's body weight was under their control. To an extent this is obviously the case – cessation of eating usually leads to weight loss – but the extent of individuals' control over their weight was typically exaggerated in discussions about weight loss. There are two facets to this exaggeration. First was a lack of consideration of the social aspects of eating that I address in section 6.6 (below). Secondly, insufficient recognition was given to the limits of individuals' abilities to undertake levels of physical activity that will have an impact on body weight. Voluntary activity only accounts for about a quarter of an individual's overall energy use (BNF, 1999: 61), although this amount can be increased by intense physical activity. Although writers such as Garrow recognised that physical activity could not be used as a treatment for obesity and overweight, they increasingly stressed its role

in prevention. Again, young fit men will be more able to increase their energy expenditure to a point where it will affect their body weight, but older individuals with other health conditions are unlikely to be physically capable of achieving the necessary levels of activity. The energy balance model helps to reinforce this perspective, by directing attention solely to the relationship between energy input and output, rather than to the social and other complexities surrounding both those processes.

Such input/output models exclude any consideration of regulatory mechanisms, but homeostatic models presuppose regulation around a set point. Considering the body as a homeostatic system hinges on the idea that there is a 'normal' state to which the body attempts to return. The failure of research to produce evidence of a set point (see chapter 5) suggests that this may not be the case. Karen Throsby and Celia Roberts contrast such homeostatic models with Ann Fausto-Sterling's model of the body as an allostatic system (Throsby and Roberts, 2010). An allostatic model is one that 'regularly changes activity to meet anticipated demands' rather than a homeostatic one that is 'constantly trying to achieve a "normal" state' (Throsby and Roberts, 2010: 87). Fausto-Sterling develops this framework in her discussion of the phenomenon of essential hypertension[90] found in black populations in the US. She links the higher blood pressures of these individuals to the chronic stress of living in a discriminatory environment, arguing that 'different life experience activates physiological processes common to all, but less provoked in some' (Fausto-Sterling, 2004: 26). In a similar fashion, 'normal' weight can be seen as the outcome of complex interactions between an external environment, personal medical history, and habitual behaviour. Such an approach provides a better explanation of the fact that so many people find it so difficult to lose significant amounts of weight over the long term. It would also allow health to be understood as cumulative, as the result of ongoing interactions that may profoundly alter the body's capacities to respond. This would fit with life course approaches within the sociology of health inequalities which study patterns of accumulation of health advantage and disadvantage in individuals' biographies (Bartley, 2004).

[90] Essential hypertension is defined as consistently raised blood pressure in the absence of an obvious physiological cause such as a constriction in a major blood vessel.

6.5 Confounding and similarity judgements

"Confounding" is currently defined as 'the error in the estimate of the measure of association between a specific risk factor and disease outcome which arises when there are differences in the comparison populations other than the risk factor under study' (Bhopal, 2002: 79). The term has been used in this sense in epidemiology since the late 1950s (Vandenbroucke, 2004: 317). In this section I will consider confounding sociologically as the result of a series of judgements that are made about the causes of disease. Confounding is important in epidemiology because it leads to mistaken inferences about the causes of disease. Epidemiologists endeavour to avoid this mis-attribution of causality by adopting appropriate research design and appropriate methods of statistical analysis. However, the description of discussions about the relationships between bodyweight, social class and smoking outlined in section 4.5 above, demonstrates that such analyses are the outcomes of complex judgements about behaviour and disease aetiology that are both biomedical and sociological.

Although epidemiology involves some laboratory research, it is primarily a field science in which researchers have limited control over causality. In the case of coronary heart disease, large-scale longitudinal epidemiological studies were initially set up to study risk factors for heart disease which were known to be varied and to operate over long timescales. However, such research gave rise to particularly difficult problems of confounding because of the number of potential variables involved and the complex interactions that could take place between them. This may be one of the reasons why discussions of confounding start to appear in epidemiological literature in the late 1950s; another was the controversy around the Doll-Hill study on the association between smoking and lung cancer (Berridge, 2003b, Vandenbroucke, 2004: 318). Decisions about whether confounding has taken place are essentially based on similarity judgements about whether one population is sufficiently similar to another in particular respects for the relevant outcomes to be considered comparable. To put it another way: researchers must consider in what ways particular populations differ, and whether these differences matter for the findings of their research. Smoking behaviour, for instance, needs to be considered in discussions of the relationship between body size and health, not only because it is a

cause of heart disease and other chronic diseases, but also because smokers have been found to be lighter, on average, than non-smokers.

Establishing the presence of confounding factors requires a sophisticated understanding of the health and behaviour of the populations being studied. For example, judgements of whether smoking was a confounding factor in the relationship between body weight and increased mortality had to take into account not only the fact that smokers tend to be both lighter and unhealthier than non-smokers, but also the social patterning of smoking behaviour, which has changed significantly in the last fifty years, and the use of smoking as a weight control technique by certain groups (usually young women). Because of this complexity such judgements are fragile and potentially unstable. They can be easily undermined by new information casting doubt on the similarity judgement: if more affluent smokers are actually heavier on average (Ernsberger and Haskew, 1987: 10) this alters the relationship between smoking and body weight on which early judgements of causality and confounding relied. It also requires a more complex analysis of the relationship between smoking, health and body weight since the effects of income level have to be incorporated.

This analysis demonstrates that confounding is important, sociologically, because it requires complex judgements about the relationship between health and structural factors such as gender, socio-economic class and age. It shows that researchers working in such areas were capable of dealing with such 'social' factors in a sophisticated manner, but that they chose to do this in a restricted fashion, in certain explanations of disease causality only.

6.6 The dominance of individual explanations: a further 'writing out' of the social

Individualism is an important element of post-war public health in developed countries (see sections 1.2.1 and 2.6). It marks a well-defended boundary between public health, on the one hand, and politics or social policy on the other. Claims to expertise in the area of diet and bodyweight have significant social and political implications, attract a lot of scrutiny and, therefore, require a great of deal boundary-

work to maintain the credibility of the people or institutions making them (see chapter 9). Individual behaviour has historically been seen as coming under medical authority, whereas the attribution of responsibility for social problems has often been more contested. The development of social medicine in Britain in the 1940s and 1950s was an attempt was made to shift the boundary between public health and social policy, but for a variety of reasons it was unsuccessful, and the work of social medicine became split between public health and medical sociology (see section 1.2.1). The growth of an individualist perspective in post- war public health can be attributed to the influx of hospital doctors and laboratory scientists into this field, both of whom were more inclined to adopt an individualist rather than a sociological or political view of illness.

Because of this individualism, the only explanations of eating behaviour that are considered at length in obesity science writing were based on either psychology or economics. The presence of psychological explanations derived from historical overlaps between obesity research and the treatment of eating disorders, whereas the prestige of economics and its closeness to public policy has meant it was regularly drawn upon in health policy writing. Individualistic accounts of behaviour, based on these two disciplines, are typical of policy literature (Steward, 2010), but they leave little room for more social and structural explanations of behaviours such as food consumption habits. Consequently, insofar as the obesity policy literature of the 1980s and 1990s makes any mention of social explanations of eating, these are typically cursory and tokenistic: brief references to outdated anthropological studies of 'fattening huts' for instance (WHO, 1995: 319), or to preference for larger body size in previous centuries or contemporary under-developed societies (West, 1994: 9-10, see also WHO, 2000: 127-8) that say more about the authors' attitudes than about contemporary eating behaviour. Often in the obesity science of that period, the 'social' functioned as a residual category, where seemingly relevant but currently unexplainable information was assembled and briefly outlined.

By contrast, recent sociological accounts of food and eating stress that such behaviour is strongly related to social norms around family conduct, gender roles, national, regional and ethnic identity and age (Atkins and Bowler, 2001, Caplan, 1996, Mennell

et al., 1992, Murcott, 1998, Warde, 1997). As Delormier and her co-authors argue in their summary of the field:

> The most important limitation of studying eating strictly as a behaviour under the control of an individual is that it exaggerates the extent to which rational choice drives what people choose to eat, and underestimates the extent to which eating is embedded in the flow of day to day life. People's eating patterns form in relation to other people, alongside everyday activities that take place in family groups, work and school. Eating does involve isolated choice, but it is choice conditioned by the context in which it occurs (Delormier et al., 2009: 217).

Classic examples of the effect of social norms on eating behaviour include the link between personal identity and religious practices of food exclusion, or gender norms around appropriate food for women and the effects of their role as family meal providers and preparers. The operation of these norms combines with more prosaic factors such as individual preference, economic resources and time constraints. Merely considering psychological attitudes to food and issues of food availability and price does not address this complexity in a nuanced enough fashion to understand why, despite the pressure of negative health messages about excess body weight and the necessity to lose weight, many individuals are unable to adapt their diet over the long term.

The rational choice model used in framing such dietary decision-making does not allow for the possibility that such pattern of eating may require choices that are not easy to make in particular social contexts. Elizabeth Shove discusses the same approach, which she labels the ABC (attitudes, behaviour, choice) model, in an article on climate change policy, arguing that

> the idea that desires and attitudes drive behaviour produces a blind spot at a particular crucial point, making it impossible to see how the contours and environmental costs of daily life evolve (Shove, 2010: 1277).

In a similar fashion, rational choice models obscure the structural factors influencing food choice. To continue the example above, women still do a disproportionate amount of the work involved in providing and preparing food, but do not necessarily have the authority to specify what food is eaten and often subordinate their

preferences and needs to other those of other family members (Charles and Kerr, cited in Nettleton, 2006: 51-2). Changes such as eating differently from family and friends, going against norms about accepting food and drink offered in hospitality and sharing, and abandoning familiar foods and patterns of eating are much easier to make in a social setting where the primacy of health-based or other nutritional arguments for food choice are generally accepted. Such arguments have been acceptable amongst affluent groups for much of this century, but, despite their increasingly wide social diffusion, it is not clear that they are equally acceptable among all social groups (Crotty, 1995, Levenstein, 2003).

Researchers into health inequalities have also considered the effect that 'the social' or 'culture' has on individual behaviour in order to explain the links between poor health, behaviour and socio-economic disadvantage (Bartley, 2004). The authors of *The Black Report* (see section 3.3) included 'behavioural/cultural' explanations of health inequalities in their typology and tried to go beyond the existing dichotomy between individual ignorance and irresponsibility and social structure (Townsend and Davidson, 1982). However, Bartley argues that very little health inequalities research has studied differences in health behaviour in terms of this anthropological sense of culture (Bartley, 2004: 69). One of the few studies that has been done, by Mildred Blaxter (1990), showed that although the patterns of behaviour she studied – smoking, drinking, diet and exercise – were patterned by class, gender and age, 'circumstances' (by which she meant both the external environment and the individual's psycho-social environment) had more effect on health than these behaviours. Another important finding was that

> Unhealthy behaviour does not reinforce disadvantage to the same extent as healthy behaviour increases advantage. This seems to suggest that the prior effect on health is the general lifestyle associated with economic or occupational position. Only in the more favourable circumstances is there 'room' for the considerable damage or improvement by the adoption of voluntary health-related habits (Blaxter, 1990: 203).

Such findings show the limitation of explanations based on individual behaviour and point to wider structural causes of ill-health. They also demonstrate how a stress on voluntary behaviour change can reinforce existing inequalities since those who are

already advantaged will not only have greater capacity to make such changes, but also, according to Blaxter's research, to benefit from them.

These areas of research show that it is necessary to go beyond 'rational choice' or ABC models to adequately explain individual eating behaviour and habits. The 'writing out' of the social that I have described here parallels that in risk factor approaches to understanding chronic diseases and has broadly similar effects. It has meant that the causes of variations in body size cannot be properly understood within obesity science. Without a sociologically informed approach that considers these causes fully, it is not be possible to understand the difficulties individuals have in losing weight over the long term.

6.7 Conclusion

This chapter has analysed two important components of obesity science: the BMI and the energy balance model. These are both important components of this body of knowledge and practice but their effects are very different. The BMI was based on understandings of body weight first developed in the insurance industry and then imported into chronic disease epidemiology. It has a complex role of simultaneously defining the problem of excess body weight in populations and individuals and allowing it to be measured, quantified and framed as an epidemic. The energy balance model derived from laboratory and clinical research into the physiological mechanisms underlying body weight regulation. Like risk factor epidemiology it involved a simplification and narrowing of understandings of human functioning, and also a writing out of social processes and structures, in this case in eating behaviour and habits. One problematic element of this model is its over-emphasis on individual control over body weight. This meant that it could not explain the widely acknowledged failure of conventional treatments for overweight and obesity (see section 5.6). Such failure is an example of the non-plasticity of human bodies. It demonstrates a social and physiological obduracy that results from factors that as well as age and sex, include individual variation, life history (including the embodiment of health inequalities), and differences between laboratory subjects and obese patients. Eating as part of everyday life is very different from eating as part of a research study.

CHAPTER 7: THE INTERNATIONAL ARENA: OBESITY AND OVERWEIGHT IN WHO REPORTS

7. 1 Introduction

In this chapter I discuss another arena in which researchers working on obesity science were active – the World Health Organisation (WHO). This discussion gives a brief outline of selected WHO publications in the area of chronic disease, diet and body weight in order to illustrate a development of ideas that paralleled processes taking place in the British obesity science reports discussed in the previous chapters. It shows how researchers participating in the production of these reports, like many officially recognised 'expert' scientists, were part of overlapping networks, both national and international. From the 1980s, obesity and overweight began to be discussed as part of an increasing focus on chronic diseases within the WHO, and from the mid 1990s, the topic began to be the focus of more sustained interest, culminating in the publication in 2000 of a widely publicised technical report labelling excess body weight as a global epidemic (WHO, 2000: 185).

I will give a brief history of such publications and the individuals involved in their production, and then discuss two areas of obesity science where the move into this wider international arena was associated with significant developments in obesity science – global estimates of prevalence and wider understanding of the negative health consequences resulting from it. As I will argue below, framings of obesity and overweight as a global phenomenon derived directly from its consideration in WHO publications. The wider understanding of its health consequences resulted from the continuing incorporation of large-scale epidemiological survey data into obesity science. This data was combined with developments in physiological research to explain the profound metabolic and endocrinological changes associated with excess body weight. This shift is analysed in this chapter because it started in the mid 1990s, and is best illustrated by discussions in the WHO reports. In order to demonstrate development in this area, my analysis is preceded by a brief outline of discussions of the negative health consequences from the earlier British reports (see chapters 4 to 6).

7.2 WHO reports on chronic disease, diet and body weight

In the WHO series of technical reports[91], publications on the topic of nutrition are relatively infrequent. Until the 1980s, those reports that dealt with nutritional issues mostly considered malnutrition, especially protein malnutrition, rather than over-nutrition. Protein malnutrition was considered in a series of reports produced by a joint FAO/WHO expert committee on nutrition, which met every two years in the 1950s and every five years after that. Philip James' career as an expert committee participant seems to begin in 1985, as a contributor to a related FAO/WHO/United Nations University report *Energy and Protein Requirements* (WHO, 1985), when he was still Assistant Director of the Dunn Nutritional Laboratory in Cambridge. This report was one of the first WHO publications to examine nutritional requirements without an explicit focus on malnutrition or deficiencies. It contained international standards for assessing the prevalence of obesity that would also be cited in later reports (WHO, 1986: 27). Other participants from the world of British obesity science also included J Durnin, who was now professor of the physiology department of Glasgow University, and John Waterlow who was now professor of human nutrition at London School of Hygiene and Tropical Medicine (LSTHM) (see section 3.3). Another notable member was Dr Anna Ferro-Luzzi (see below).

Beginning in the 1960s, WHO also published more infrequent technical reports on heart disease, but until the 1980s and *The Prevention of Coronary Heart Disease* (WHO, 1982) (henceforth TR 678), these did not discuss the different risk factors in any detail. The authors of TR678 discussed the issue of body-weight as one of a list of 'life-style' factors that included diet and blood cholesterol, blood pressure, smoking, physical activity, alcohol and oral contraceptives, but the section on body-weight was brief .They argued that the 'prevention or correction of obesity is of great importance' and referred to 'the mass nature of obesity and the profound

[91] The WHO technical report series currently consists of nearly 1000 reports. Approximately six such reports are produced every year. The first, a report on a session of the Expert Committee on the Unification of Pharmacopoeias was published in 1950 (WHO, 1950) and the most recent, number 957, is the Forty-fourth report of the WHO Expert Committee on Specifications for Pharmaceutical Preparations (WHO, 2010a). Many of the reports form long running series, such as on biological standardization, drug dependence and food additives, but others appear more subject to the vagaries of medical or policy fashions. Technical reports are designed to make 'available the findings of various international groups of experts that provide WHO with the latest scientific and technical advice on a broad range of medical and public health subjects' (WHO, 2010b). This fulfils the organisation's responsibility to act as a globally authoritative source of technical medical expertise (Lee, 2009, Siddiqi, 1995).

sedentariness of many modern cultures' (WHO, 1982: 22, 30), but framed the issue as one of persuading individuals to follow appropriate guidelines on diet and levels of physical activity (WHO, 1982: 31). This was compatible with approaches described in chapter 2 where bodyweight was understood as a relatively unimportant risk factor for heart disease: there was none of the sense of body-weight as an important issue in its own right that later documents display (e.g. WHO, 1995, WHO, 2000). Because this was a report on heart disease, and obesity science had already started to separate from this community (see section 3.3), none of the researchers discussed in earlier chapters were involved in the production of this report, although the chairman was Professor Geoffrey Rose from the Department of Epidemiology at the London School of Hygiene and Tropical Medicine (LSTHM) (see section 2.5).

Community Prevention and control of cardiovascular diseases (WHO, 1986) (henceforth TR 732) also focused on heart disease. It appears to have been the next WHO publication in which Philip James participated. Now the director of the Rowett Research Institute (see section 3.4), James was again a temporary advisor to the expert committee producing this report which also included Pekka Puska from the National Public Health Institute of Helsinki, (see section 2.5) and Geoffrey Rose of the LSTHM. The authors of this report described how

> The emergence of mass coronary heart disease has accompanied the increase in affluence in industrialized societies; affluence itself is not to blame for cardiovascular disease, but only certain specific components of the affluent life-style. Affluence generates powerful social forces which encourage the adoption of a lifestyle that includes these adverse elements. That association is not inevitable, however, since coronary heart disease death rates have been declining in some countries recently without any decline in affluence (WHO, 1986: 9).

This anticipated discussions in later reports (especially TR797 and TR 894) of the bodily harms associated with increasing affluence. However, teasing out of which aspects of the affluent lifestyle were harmful to health was proving difficult, as shown by the subsequent argument that higher mortality rates amongst the less affluent 'cannot be fully explained by the standard cardiovascular risk factors' (*ibid.*). The authors framed the problem of heart disease and stroke as one of 'nutrient excess' and argued for the 'avoidance of weight gain and obesity' by advising individuals to

'increase their physical activity and to reduce the energy-density of their diet by consuming more low-fat, low-sugar foods (WHO, 1986: 14).[92] This was very conventional advice at the time, as were the targets to reduce average saturated fat contents of diet, but the authors' suggestions that agriculture and food production should be altered in line with health policy to encourage the production and consumption of low fat dairy products and lean meats were more radical (WHO, 1986: 16-18). This was very close to the approach advocated in the UK government's Health of the Nation report on obesity published ten years later (see chapter 8). Judgements about the relative healthiness of a 'Mediterranean diet' (James et al., 1988: xii) containing significant quantities of fruit, vegetables, fresh fish, whole grains and olive oil were implied in the authors' recommendation of a largely plant based diet. Given the extent to which such recommendations conflicted with food production and processing commercial interests, the highly conditional and very technical way such recommendations were framed might be seen as method of avoiding the controversies that accompanied producing policy in the area of diet and health.

In the late 1960s, the WHO Europe regional office had developed a research programme into cardiovascular disease, particularly coronary heart disease (Lee, 1998: 94 - 95, Kaprio, 1991: 35-6)**,** and it began to move into the area of nutrition and health from the late 1980s (Baggott, 2000: 172). In this period, Philip James co-authored *Healthy Nutrition*, a report on preventing nutrition-related diseases for the WHO European regional office for Europe (James et al., 1988: 201). One of his co-authors was Dr Anna Ferro-Luzzi, director of the Unit of Human Nutrition at the National Institute of Nutrition in Rome, who worked for the FAO and the United Nations University. Baggott cites the publication of *Healthy Nutrition* as the beginning of the WHO's move into this area, describing how it was followed by the publication of a technical report on nutrition and chronic disease, the International Declaration on Nutrition in 1992 and a WHO Nutrition Programme in 1993 which declared the tackling obesity and diet-related disease as a priority, setting targets to reduce the proportion of fat in average diets (Baggott, 2000: 172).

[92] This approach is comparable to the 'old approach' which emphasised carbohydrate restriction and is no longer part of 'current nutritional thinking' since it is now considered to 'enhance rather than reduce the cardiovascular complications of obesity' (WHO, 1986: 27).

Healthy Nutrition was an overview of the links between diet and conditions such as CHD, diabetes, cancer, cirrhosis, bone disease and anaemia. The authors argued that from the mid-1960s 'evidence was beginning to emerge suggesting that diseases not normally associated with malnutrition had their origin in nutrition' - citing the example of heart disease - but by the 1980s obesity had become a 'major public health problem' (James et al., 1988: 12)**.** However, in this publication, obesity was discussed both under the heading of conditions that predispose to major health problems and as a major health problem in its own right, which demonstrates a certain amount of confusion about its status. The brief discussion used European and American data to make the same arguments regarding its prevalence and negative health consequences as earlier British reports (see section 3.4) (James et al., 1988: 55-60). European epidemiological data also cited used a variety of definitions of obesity including BMI, the Broca index[93] and Metropolitan Life recommended weights, showing that no one standard definition of obesity was used at the time. Although the causes of obesity were barely addressed the authors admitted that there were 'no satisfactory explanations for the marked differences in the prevalence of obesity in different parts of Europe' (James et al., 1988: 83). Since other section of the report documented significant variations in dietary composition throughout the European region and advocated a low-fat diet as a way of preventing obesity and other nutrition-related diseases, this was an important gap in their argument. Suggesting that obesity rates may be correlated with average fat consumption (*ibid.*) did not count as an adequate explanation of these differentials.

Anna Ferro-Luzzi was also a member of the study group for the publication, *Diet, nutrition and the prevention of chronic diseases* (WHO, 1990) (henceforth TR 797). Philip James chaired the study group which included nutrition researchers from North America, China, Egypt, Nigeria, the USSR and Japan. The aim of the report was

> to describe recent changes in dietary and health patterns of countries, define the relationship between the "affluent diet that typically accompanied

[93] In 1871 Dr. Pierre Paul Broca, a French physician, anatomist and anthropologist created the formula known as Broca's index where weight (in kg) *should equal* height (in cm) minus 100 (plus or minus 10% for men and 15% for women). According to the authors of *Healthy Nutrition*, obesity is defined as 20% or more above this value, (James et al, 1988: 55).

> economic development and the subsequent emergence of chronic diseases, and explore the need for national food and nutrition prevention policies to prevent or minimize costly health problems (WHO, 1990: 9).

The affluent diet was described as one 'characterized by an excess of energy-dense foods rich in fat and free sugars but a deficiency of complex carbohydrate foods' (WHO, 1990: 10-11). The report's authors described the relationship between diet and chronic diseases, such as CHD, hypertension and stroke, cancer and diabetes, as a 'recently identified problem' (WHO, 1990: 10). This framing of the 'affluent diet' is an intermediate stage between the labelling of chronic diseases as 'diseases of affluence' (see section 2.3) and later ideas that they are caused by the effects of modernisation (see section 8.2). In an argument that anticipates the later idea of the double burden of disease (see section 8.5), the authors stated that in countries with relatively modest average incomes[94] 'the burden of cardiovascular disease and cancers is nearly as great as in the very affluent countries with an income nearly three times as great' (WHO, 1990: 33). It was the beginning of a global understanding of chronic diseases affecting populations of developing countries as well as those of industrialised ones.

TR797 contains many references to obesity, both as a disease in its own right and as a result of rapid changes taking place in both diet and life conditions of many populations. The authors argue that obesity was a problem in developing as well as industrialized countries, mentioning very high rates amongst women in Trinidad (WHO, 1990: 28). Widely varying rates of obesity amongst different populations were argued to be the result of 'environmental factors' such as diet and levels of physical activity, rather than solely the result of variations in genetic susceptibility between populations, leading to an argument for public health measures to modify 'the population's environmental circumstances' (WHO, 1990: 69). The cause of obesity was understood to be an imbalance between energy intake and expenditure, but this imbalance was linked to economic development, since as well as unprecedented dietary changes, processes of industrialisation and urbanisation had led to reductions in levels of physical activity (WHO, 1990: 72). The overarching message of this report was of the health benefits of a high-carbohydrate, low-fat,

[94] They refer annual GNP per capita in the range of $3000 to $4000.

largely plant-based diet (WHO, 1990: 158), which was important in preventing many types of diet-related chronic disease as well as obesity and overweight. This too was part of the new understanding of chronic disease as a consequence of economic development (see section 5.5 and 8.2), which could be experienced by the poor as well as the rich.

The first report that contains a significant discussion of overweight (the authors refused to use the term obesity)[95] was *Physical Status: The Uses and Interpretation of Anthropometry* (WHO, 1995) (henceforth TR854). The expert committee responsible for compiling TR 854 included Anna Ferro-Luzzi and Jakko Tuomilehto of the Finnish National Public Health Institute who was part of the North Karelia project and MONICA (see section 2.5). The rest of the committee comprised epidemiologists and nutrition researchers from America, Australia, Brazil, Columbia, India, Netherlands and Nigeria, as well as advisors from the FAO and UNICEF. Philip James was also among a long list of individuals thanked for their extensive contributions (WHO, 1995: 412). The chapter of TR854 on overweight closely resembles the next relevant report which was entirely focused on the topic, *Obesity: Preventing and Managing the Global Epidemic* (WHO, 2000) (henceforth TR894). The publication of TR894 in 2000 was part of an important increase in activity around this area of health policy that began in the mid 1990s, and included several British publications (see chapters 4 to 6). In the international context, TR 854 was published in 1995, the consultation meeting for TR894 took place in 1997, the International Obesity Taskforce (IOTF) (see below) was set up in 1998 and TR894 was published in 2000. Two technical reports were produced in a couple of years - a relatively short period of time for a large bureaucracy like the WHO and, therefore, a significant burst of activity in the area of body weight and health.

Several researchers from the Rowett Research Institute participated in the production of TR894, presumably due to the presence on the secretariat of Philip James in his role as the chair of the IOTF. Two other individual from the Rowett served on the

[95] The authors defined normal weight and three grades of overweight using the standard BMI cut-off points for obesity and overweight with the following caveat: 'Because BMI does not measure fat mass or fat percentage and because there are no clearly established cut-off points for fat mass or fat percentage that can be translated into cut-offs for BMI, the Expert Committee decided to express differently levels of high BMI in terms of degrees of overweight rather than degrees of obesity (which would imply knowledge of body composition)' (WHO, 1995: 312).

secretariat, both of whom are also listed as members of the IOTF. The introduction of TR894 also describes the consultation process as 'undertaken in close collaboration with the Rowett Research Institute…and the International Obesity Taskforce (IOTF) chaired by Professor Philip James'(WHO, 2000: 1). This level of representation, and such an explicit acknowledgement of the role of the Rowett/IOTF staff, suggests that Philip James had an important role in the production of this report.

As is appropriate for a WHO consultation, other members of the expert committee for TR894 came from all regions of the world, including representatives from the US, Canada and Europe, China, Malaysia and Japan, India and Pakistan, and Nigeria and South Africa. One of the American members was George Bray (see section 4.4), the chairman was Professor James Hill of the University of Colorado and another was Dr William Dietz, a paediatrician from the North-east Medical Centre, Boston. Two other high profile members were Professors Arne Astrup of the Royal Veterinary and Agricultural University, Copenhagen and Per Bjorntorp of Gothenberg University, Sweden. Per Bjorntorp was a clinical researcher interested in metabolic problems related to type II diabetes, obesity and cardiovascular disease. Arne Astrup was a high profile nutrition researcher whose research interests included fat metabolism, energy expenditure and dietary management of obesity, and was also a member of the IOTF.

7.3 Obesity as a global health problem

Previous obesity science writing discussed prevalences of obesity and overweight in European and North American populations (see section 4.6). However, WHO is a global institution whose work has largely focused on health problems in developing countries. Presumably, this meant that it was important to frame obesity as global phenomenon. From the mid 1990s, obesity did become discussed as a global health problem. Standard definitions of overweight and obesity combined with an increasing amount of data permitted analyses of changing prevalence rates that were both global in scope and acknowledged the differences between populations. The 1995 WHO technical report on anthropometry (TR854) was the first to contain a detailed discussion of overweight as a global phenomenon, citing prevalence rate data from countries in Africa, South America, the Caribbean and Asia, as well as from more than twenty Pacific and Indian Ocean island populations. The data for these small

island populations showed a striking variation of levels of obesity within as well as between populations: the prevalence rates quoted for the population of Papua New Guinea varied from 4.7% for Highland men to 64.3% for urban women, and the rates for Chinese men in Mauritius were 2.1% compared with a rate of 20.7% among Creole women in Mauritius (WHO, 1995: 314-5). Overall, the global situation was summarised as follows:

> Overweight is a major public health issue. Grade 2 overweight…is relatively common in most industrialized societies and also in many less modernised cultures: data compiled recently show that the prevalence among 20-60 years olds is about 10-20% among whites in the USA and most countries of Europe…Prevalence is high (20 – 40%) among women in eastern European and Mediterranean countries and black women in the USA. Even higher prevalences are observed among American Indians and Hispanic Americans, and on the Pacific islands…, with probably the highest rates in the world among Melanesians, Micronesians, and Polynesians… In some African and Asian countries prevalence is much lower but in countries of South America and the Caribbean the prevalence of grade 2 overweight may be close to that in many European countries (WHO, 1995: 313).

This is obesity explicitly understood as a global public health problem and to reinforce this, the authors referred to 1980s national survey data for the US, Australia, Finland, the Netherlands, Sweden and the UK. As this data demonstrated that the prevalence of overweight 'remained stable or increased', it is debatable how much it contributed to their argument, but the authors still managed to incorporate it.

> Since then, there seems to be no indication of a decrease in the prevalence of overweight in these affluent countries despite increased commercial and other interests in promoting leanness; on the contrary, the prevalence of overweight may be increasing further (WHO, 1995: 317).

By the late 1990s, the authors of the technical report on obesity (TR894) had developed the framing of obesity as a global health problem and tried to provide a more complete, and therefore authoritative, picture of global trends. An important new aspect of TR894 was the use of prevalence data from the WHO MONICA project.[96] This information was crucial for the authors' framing of obesity as a global health problem and MONICA was described as the 'most comprehensive data on the

[96] The WHO MONICA (Multinational Monitoring of Trends and Determinants in Cardiovascular Disease) Project 1979-2002 was described as the world's largest and longest study of heart disease and stroke (Tunstall-Pedoe, 2003) (see also section 2.5).

prevalence of obesity worldwide' (WHO, 2000: 17 , see also Tunstall-Pedoe, 2003). Despite arguing that 'the prevalence of overweight and obesity [is] increasing worldwide at an alarming rate' (WHO, 2000: 16), the quality of the evidence cited to illustrate this dramatic claim varied considerably between different global regions.[97]

In the African region, the authors admitted that the principal focus of research had been on undernutrition and food security so that increases in obesity rates were documented for only a few countries and information of current prevalences was 'fragmentary and limited'. One study conducted in Mauritius showed the same upward trends as the other WHO regions, and five other small scale studies were used to demonstrate the current prevalence rates of obesity in African countries, that ranged from 0.6% in Tanzanian men in 1986-9 to 44% among black South African women in 1990 (WHO, 2000: 21). There was thought to be a significant difference between rural adults in developing countries who maintained their weight and urban dwellers who did not: 'with improvements in socio-economic status and increasing changes due to rapid urbanization, the prevalence of obesity amongst some groups of black women has risen markedly to levels exceeding those in populations in industrialized countries' (*ibid.*)[98]. In this report, a total of six small scale studies, on populations who were unlikely to be representative, were cited as evidence for changes in the average weights of the populations of an entire continent.

For the Eastern Mediterranean region, the authors admitted that good quality nationally representative secular trend data was not available, but the limited data that existed 'indicate that the prevalence of adult obesity in countries in the Region is high and that women in particular are affected' (WHO, 2000: 26). For example, a 1990-3 cross-sectional survey of 13177 adult Saudis showed a prevalence of obesity amongst women several times higher than current rates in developed countries and higher than that of Saudi men (*ibid.*). Similarly, 'Good quality, nationally representative secular

[97] The six WHO regions are Africa, the Americas, the Eastern Mediterranean (most of the Middle Eastern countries plus Afganistan, Dijbouti, Pakistan, Somalia and Sudan), Europe (which includes Israel, all of the former USSR countries and Turkey), the Western Pacific (which includes China and Japan) and South East Asia (which includes India and Bangladesh). This division resulted from a mixture of geographical and political considerations (Siddiqi, 1995) and it operated as an important structuring device for the epidemiological data considered below.

[98] This relates to discussions about the role of industrialisation and modernisation in increasing average body weights (see chapter 8).

trend data for countries in the South-East Asia Region were unavailable' (WHO, 2000: 23). Two small scale studies of Thai government officials and affluent urban dwellers showed that in 1985 2.2% of men and 3.0% of women were obese, and in 1991 3.0% of men and 3.8% of women were obese (*ibid.*). However, these groups were unlikely to be representative of the wider population of this region. Furthermore, because these prevalence rates were notably low and South-East Asia contains a large fraction of the world's population, the absence of such data was a significant omission, weakening the report's argument that obesity should be considered as a global public health problem.

For the Americas region, secular trend data showed obesity rates increasing in both developed countries, like the US and Canada, and in developing countries, like Brazil (WHO, 2000: 21). Figures for the US derived from a series of national health surveys[99] demonstrated that 'obesity is an escalating problem in the USA' (WHO, 2000: 22): between 1960 and 1994 the prevalence among men increased from 10.4 to 19.9%, and among women it increased from 15.1% to 24.9%, and these rates of increase accelerated in the 1980s. At the time of the report's publication 20% of American men and 25% of women and 15% of Canadian men and women were considered obese and black women and other minority groups in the US were understood to have 'particularly high rates of obesity' (WHO, 2000: 23). The current prevalence in Brazil was given as 6% among men and 13% among women, and obesity was also seen as a major public health problem in parts of the Caribbean (*ibid.*).

Data from the MONICA study was an important source of information for the Europe Region. However, the report's authors argued that MONICA populations were not necessarily representative of host countries, and so the best data on secular trends in obesity came from national surveys which were available for England, Finland, Germany, the Netherlands and Sweden. These surveys showed that 'the prevalence of obesity has increased by about 10-40% in the majority of European countries in the past 10 years' and the most dramatic increases occurred in England where rates had

[99] These were NHES and NHANES (see section 4.4) which took place between 1960-2, 1971-4, 1976-80 and 1988-94. The data derived from these surveys shows slight increases in prevalence between the first three surveys and much larger increases between last two.

doubled in the last ten years (WHO, 2000: 24). Obesity was seen as relatively common in Europe:

> The average prevalence of obesity in European centres participating in the WHO MONICA study between 1983 and 1986 was about 15% in men and 22% in women, although there was great variability both within and between countries (WHO, 2000: 25).

The existence of great variability between populations suggests that excess bodyweight might be a problem of specific populations rather than all European countries (see section 9.2) but this is not an approach that was discussed here.

In the Western Pacific region data was available showing increasing prevalence of obesity amongst Australian, Samoan, Japanese and Chinese populations (WHO, 2000: 27). The current prevalence among the white population of Australia and New Zealand was given as 10-15%, but in the aboriginal populations it was thought to be either much higher or much lower depending on location (*ibid.*). The prevalence figures given for Japan were 2% for men and 3% for women, and for China 1% for men and 2% for women with studies finding that obesity is commoner in women than in men and also in urban rather than rural areas (WHO, 2000: 29). This was evidence of very low rates in another large country, containing a significant proportion of the global population, which again undermines claims of a global epidemic. In contrast to these very low rates, very high prevalence rates of obesity had been found in the Pacific Island populations of Melanesia, Micronesia and Polynesia (WHO, 2000: 30). The most extreme example given was urban Samoa where a marked increase in the prevalence of obesity had been observed between 1978 and 1993, and the prevalence of obesity was given as over 75% in women and approximately 60% in men. However, there is an important caveat attached to these figures:

> Polynesians seem leaner than Caucasians at any given body size so the prevalence of obesity in Polynesian populations may not be quite as high as is currently estimated using Caucasian-derived classifications based on BMI (*ibid.*).

The emphasis on this point is part of the ongoing debate about the applicability of standard BMI cut-off points (see section 4.4 and 7.4).

Despite the centrality of increasing prevalences to the argument that obesity was a significant global health problem, the amount of available data for different regions of the world varied widely and so did the quoted incidences of obesity - from 0.8% for both men and women in Mali and 1% for women and 2% for men in China, to 15% for men and 17% for women in England, 20% for men and 25% for women in the US and 58% for men and 77% for women in urban Western Samoa. However, despite the patchiness of the available information, prevalences were framed in both reports as uniformly rising. In TR894, current prevalences and secular trends for each region of the world were discussed, despite the fact that the US and countries such as England, Finland and the Netherlands were unusual in having national survey data demonstrating increasing prevalences; for most African, Asian and Middle Eastern countries such data was very fragmentary or simply did not exist. These gaps demonstrate that the framing of obesity as a global public health problem seems to have largely preceded the data.

7.4 Particular populations as exemplars

As the discussion of the prevalence data given above suggests, some populations were seen as particularly prone to both obesity and overweight and the physiological consequences of excess weight. These populations functioned as exemplars within obesity science, warnings of future possibilities for societies currently with lower rates, and ways of understanding the causes of excess body weight and the chronic diseases associated with it. The Pima Indians (see section 5.3) and the Samoans were regularly referred to in such discussions. For example, in TR854 analyses of populations among whom overweight was now very common - the Pima Indians in the US, Fijians, Maltese, Melanesians, Nauruans and Samoans - suggested that ‘the condition [of obesity] was uncommon before the adoption of sedentary lifestyles and high-fat diets’ (WHO, 1995: 317). Because they referred to particular populations, such discussions could readily shift from the role of economic development to genetic explanations. In this way, descriptions of the effects of increasing affluence on average body weight were combined with explanations focusing on the role of genetic causes in differential rates of obesity and overweight:

> The expression of overweight requires a certain level of food availability above which the relative contributions of genetics and environment probably vary within and across populations. High-fat diets combined with low levels of physical activity play an important role in the increase of overweight that accompanies the transition from poverty to affluence…In an affluent population of individuals with similar socio-economic values and resources, genetic factors will become relatively more important in determining which individuals will become obese (WHO, 1995: 317).

This allows the authors to mention the '"thrifty genotype" hypothesis' - that 'populations exposed to inadequate or fluctuating food supplies are genetically selected for a high level of efficiency in caloric utilization or fat storage…[which] may lead to an increase in the prevalence of overweight and non-insulin-dependent diabetes mellitus' (*ibid.*) - as one example of such genetic factors (see section 9.5).

In the 1990s, the debate about the use of BMI to measure body fat transformed into a narrower argument about the extent to which different populations are characterised by differing body proportions. The BMI classification, and its cut-off points, was used in both the later WHO technical reports, demonstrating how it had become, in some senses, a globally standard definition. However, the fact that there were still ongoing discussions about the appropriateness of this definition for different population groups suggests that this process of standardising the definition was partial and incomplete.

> Body mass index appears to be a good indicator of the deposition of excess energy as fat in adult white men and women living in Europe and North America. It is probably less appropriate in other population who differ in body build and body proportions'(WHO, 1995: 327).

BMI had become generally accepted as an appropriate measure of body fat for Caucasian populations, but discussions were beginning about how to devise appropriate ethnically specific definitions and classifications for other populations. Despite the centrality of BMI to the anthropometry of overweight, this debate was mentioned only in this quote and a brief discussion of the differential risks of cardio-vascular disease and diabetes amongst different overweight populations (WHO, 1995: 328). It was, however, another way in which obesity and overweight were not yet fully global phenomena (see section 7.3).

This acceptance of a BMI-based definition meant that the authors of TR894 could cite the earlier report, give the same cut-off points and, therefore, spend less time arguing for a particular definition and measure of obesity.[100] They also acknowledged some of the criticisms of BMI, and stressed its use as a population rather than individual measure to forestall others.

> BMI can be considered to provide the most useful, albeit crude, population-level measure of obesity. The robust nature of the measurements and widespread inclusion of weights and heights in clinical and population health surveys mean that a more selective measure of adiposity, such as skinfold thickness measurements, provides additional rather than primary information. BMI can be used to estimate the prevalence of obesity within a population and the risks associated with it, but does not, however, account for the wide variation in the nature of obesity between different individuals and populations. (WHO, 2000: 9).

Waist circumference and WHR (waist to hip ratio), were discussed but there were also problems with developing standard cut-off points due to significant differences between populations:

> For instance, abdominal fatness has been shown to be less strongly associated with risk factors for CVD and non-insulin dependent diabetes (NIDDM) in black women than in white women…Also people of South Asian (Bangladeshi, Indian and Pakistani) descent living in urban societies have a higher prevalence of many of the complications of obesity than other ethnic groups…These complications are associated with abdominal fat distributions that is markedly higher for a given level of BMI than in Europeans. Finally, although women have almost the same absolute risk of coronary heart disease (CHD) as men at the same WHR…they show increases in relative risk of CHD at lower waist circumferences than men. Thus, there is a need to develop sex-specific waist circumference cut-off points appropriate for different populations (WHO, 2000: 10).

Because of such arguments, these authors stressed that the BMI cut-off points given were specific to Caucasian populations (WHO, 2000: 11). Despite ongoing sociological debates as to its validity (Epstein, 2007, Fausto-Sterling, 2004), race and ethnicity continued to operate as variables in this area of biomedical research. Amongst the 'non-genetic' factors in the development of overweight and obesity were sex - women have, on average, a higher percentage of body fat - and ethnicity:

[100] The fact that this report used the terminology overweight and obesity, rather than just overweight like TR854, may reflect an increasing consensus around the use of BMI or it may be due to the differing composition of the expert committees that produced the two reports.

> Ethnic groups in many industrialised countries appear to be especially susceptible to the development of obesity and its complications. Evidence suggests that this may be due to a genetic predisposition to obesity that only becomes more apparent when such groups are exposed to a more affluent lifestyle (WHO, 2000: 138).

Such groups included the Pima Indians of Arizona, Australian aboriginals, indigenous Hawaiians and South Asians overseas. Obesity and its complications were seen as the result of genetic susceptibilities combined with the transition from a 'traditional' to a 'more affluent and sedentary lifestyle and its accompanying diet'(WHO, 2000: 139). The example of African Americans in the US 'where the highest rates of obesity are found in the poorest communities' (*ibid.*) was also given to argue that other environmental factors may be important.

> In these populations, fat-rich, energy-dense diets are likely to be the cheapest, and reduced levels of activity stem from unemployment. Other factors associated with poverty may also be involved (*ibid.*).

Only the African American population in the US was described as poor, and its high rates of obesity related to this poverty. It would have been equally valid to describe many of the communities listed above – especially the Pima Indians in Arizona, Australian aboriginals and indigenous Hawaiians – as poor. This leads to the question of why the prevalence of obesity in these communities was not explicitly linked to high levels of poverty in the same way (see section 9.5).

7.5 Relative risk and the expanded list of health consequences

As I described in chapter 3, the individual paradigm contained a list of health consequences that was relatively short, usually consisting of heart disease, diabetes, mechanical problems (joint pain and breathing difficulties) and psychological problems (see section 3.4). In the 1990s this list became extended and standardised using information from Framingham (see section 2.5), and the American Cancer Society studies (see section 3.4). This new data not only increased the number of conditions that were seen to be associated with obesity, but was also used in the development of a wider understanding of the health consequences of overweight and

obesity. In contrast to earlier, largely clinical, knowledge, it consisted of statistics of relative risk, derived from large population studies, which were combined with existing knowledge to produce a new understanding of wider metabolic effects of obesity than had been previously recognised, and the greater damage to health, particularly over the long term.

When this new understanding was first developing, authors typically explained these relationships at greater length and outlined models accounting for these effects. Thus, Peter Kopelman discussed the hypertensive consequences of obesity using Framingham data to illustrate the relationship between the two conditions: 'greater degrees of obesity were associated with progressively higher levels of blood pressure and hypertensive cardiovascular disease' (Kopelman, 1984: 623). The causes of hypertension in these individuals were not known but

> The alterations in endocrine function, which accompany weight gain, may contribute to an increase in blood pressure and there appears to be a relationship between plasma insulin and catecholamine[101] concentrations, fat cell size and the development of hypertension (Kopelman, 1984: 630).

Wide ranging endocrinological changes were now seen to result from obesity which increased rates of hypertension, and, so, stroke and heart disease. Other authors of the time listed the conditions with which increased body weight was associated - coronary heart disease, hypertension, diabetes, gall bladder disease, arthritis, cancer, disorders of the respiratory and menstrual systems and some psychological conditions – using the 1983 RCP report (Hautvast and Deurenberg, 1987: 67). However, obesity was also seen to be associated with high blood cholesterol, reduced glucose tolerance and high blood pressure, but it was not clear whether these were caused by obesity or its cause (Hautvast and Deurenberg, 1987: 67-8), and demonstrating this uncertainty, these conditions were described as confounding factors (see section 6.5). Writing in the same period, Garrow provided a detailed discussion of the direction of causality in these associations:

> This survey [the Seven Countries Study] showed that overweight men were more likely to have heart attacks, but that when age, smoking, blood pressure

[101] This is another name for a group of hormones such as adrenaline, noradrenaline and dopamine that are released by the adrenal glands in response to stress.

> and serum cholesterol were taken into account, relative weight did not significantly predict coronary heart disease among men aged 40-59 years. This finding may be interpreted to mean that weight is irrelevant to coronary heart disease, or alternatively that it increases the risk of death from coronary heart disease by contributing to hypertension and hypercholesterolaemia (Garrow, 1988: 11).

Evidence from feeding studies done on prisoners (see section 6.4) was inconclusive but data from Framingham demonstrated that obesity, even if not associated with high blood pressure and high cholesterol, was not 'benign'; in fact, it was a 'significant and independent predictor of disease, especially among women' since it was the third most reliable indicator of heightened risk of cardiovascular disease for women, after age and blood pressure (Garrow, 1988: 13). However, these relationships were not linear: the highest values of blood cholesterol and coronary atheroschlerosis were not found amongst the heaviest patients (*ibid.*). Other conditions associated with obesity and overweight were diabetes, gallbladder disease, impaired reproductive function (including menstrual irregularity, ovarian failure and polycystic ovary syndrome), cancers of the rectum, colon and prostate in men and the gallbladder, endometrium, cervix, ovary and breast in women[102], mechanical problems such as osteoarthritis in joints, and social and psychological disadvantages (see below) (Garrow, 1988: 13 - 15).

Garrow's list of the conditions associated with excess body weight was widely reproduced in the next ten years. In 1994, West gives the same list of diseases as Garrow[103] which are associated with obesity (West, 1994: 13 - 14) and the 1995 Health of the Nation report discusses the topic briefly using the 1983 RCP report, Garrow's 1988 textbook and an American report on diet and health. The authors argued that

> There is a large body of epidemiological evidence that links obesity to increased mortality and increased risk of chronic diseases such as cardiovascular disease, certain cancers, diabetes, gallbladder disease, hypertension and a range of bone joint and skin disorders…Risk of disease increases with BMI throughout the range of BMI, but is particularly marked at high BMI (DoH, 1995: 3).

[102] The first ACS study is cited for these conditions.
[103] Again, the authors cite the first ACS study.

As there is a significant overlap in the evidence used by two of these publications, it is arguable how large this body of epidemiological actually was**,** but these authors no longer had to make a case for this link, they could merely state it.

Similarly, in the SIGN report, the health complications of weight gain were listed as diabetes; raised blood pressure; stroke; hyperlipidaemia (and low levels of HDL cholesterol); coronary heart disease; gallstones; cancers (post-menopausal breast, endometrial, ovarian, gallbladder and colon cancers); breathlessness, respiratory disease and sleep apnoea; menstrual abnormalities, pregnancy complications including perinatal mortality and gestational diabetes; musculo-skeletal disorders and osteoarthritis (back joint and foot conditions); stress incontinence; psychological problems (social isolation, low self-esteem, binge eating and night eating), and disability (SIGN, 1996: 10-11). The list is long and daunting, but derived largely from Garrow's 1988 textbook, and included 'reduced employment prospects' and 'early retirement' which are important, but not health, consequences. Such blurring of the medical and the social gives the impression of constructing an impressive list partly for rhetorical purposes. But, for the first time, these authors discussed the hazards of intentional weight loss - gallbladder disease, the risk of which increases with increasing weight loss, and decreased bone density (SIGN, 1996: 14). Apart from Garrow's acknowledgement that weight loss does not reduce the incidence of gallstones (Garrow, 1988: 19), this is the first reference, within mainstream obesity discourse, I have found to the negative health consequences of losing weight.

In the BNF report, the complications of obesity were given as non-insulin-dependent diabetes mellitus (NIDDM), coronary heart disease, cancer, osteoarthritis, gallstones, sleep apnoea, reproductive disorders, complications of pregnancy, psychological disorders and social penalties (BNF, 1999: 7-12). Evidence for the link between diabetes and obesity came from studies of the metabolic consequences of overfeeding both humans and rhesus monkeys. In the human studies, the volunteers increased their body weight by roughly 20% in 6 months:

> There was a significant increase in fasting insulin, glucose and triaglyceride, cholesterol and amino acids, along with a decrease in oral and intravenous glucose tolerance. These are all changes of the type that characterise the non-insulin-dependent diabetic. The subjects did not become frankly diabetic but

> their BMI at maximum weight was only about 28. The overfeeding was then stopped, they lost weight and the insulin sensitivity reverted to normal (BNF, 1999: 7).

In rhesus monkeys the development of obesity and insulin resistance was followed by NIDDM. There was also epidemiological evidence for the link between obesity and diabetes:

> In epidemiological studies, the prevalence of diabetes increases with increasing severity of obesity, with increasing duration of obesity and with increasing age. Analysis of the National Health and Nutrition Examination Study (NHANES) data shows that, for each kilogram increase in weight of the population, the risk of diabetes increases by 4.5% (BNF, 1999: 7-8).

However, the association between obesity and diabetes 'underestimates the contribution of obesity to the incidence of diabetes' (BNF, 1999: 8) because individuals often lose significant amounts of weight when they develop overt diabetes.

The evidence for the link between coronary heart disease – 'the main cause of excess mortality among obese people' - and obesity was epidemiological. However, in this case, the report's authors had to construct an argument incorporating the findings of the Seven Countries study, which had shown that obesity was not an independent risk factor for CHD when age, cigarette smoking, blood pressure and serum cholesterol were already accounted for (in a calculation to predict heart attacks in men aged 40-59). To do this, they argued that

> This paradoxical solution arises because obesity itself is strongly related to hypertension and stroke, particularly in young people…Obese people also have an adverse blood lipid profile, and therefore, in Key's multiple regression equation, part of the effect of obesity has already been 'explained' by the hypertension and cholesterol (BNF, 1999: 9).

The data for increased cancer risk, once again came from the first ACS study. It showed that the mortality rate for cancer among men who were 40% overweight was 1.33 and the mortality rate for women at the same level of overweight was 1.55 (*ibid.*). The difference in increased risk between men and women was not discussed, but the increased risks included post-menopausal breast cancer in women as well as

cancers of the endometrium, uterus, cervix, ovary and gall-bladder in women and those of the colon, rectum and prostate in men (*ibid.*). Overweight and obesity were also seen to have negative effects on the female reproductive system: 'Significant associations are seen in reproductive endocrinology between excess body fat and ovulatory dysfunction, hyperandrogenism and hormone sensitive carcinomas' (BNF, 1999: 10). Excess body weight was also seen to increase the risk of complications in pregnancy: 'even moderate degrees of obesity are associated with an increased risk of hypertension, toxaemia, gestational diabetes, urinary tract infections and fetal macrosomia' moreover, 'perinatal death is three times more common in obese than in thin women' (*ibid.*). The effect of obesity and overweight on the male reproductive system was also discussed, and the authors stated that decreased levels of testosterone were found in massively obese men (*ibid.*). These arguments also illustrate an increased understanding of the endocrinological effects of overweight and obesity.

In the two WHO technical reports (TR854 and 894), evidence for the negative health consequences of obesity and overweight was discussed in a new way that focused more on the increased relative risk of associated conditions rather than on establishing plausible biological mechanisms. In 1995, the authors of TR854 discussed the health consequences of overweight under the headings of the major conditions with which it was understood to be associated – heart disease, stroke, hypertension, NIDDM, gall bladder disease, osteoarthritis and cancer (WHO, 1995: 323 - 7)[104] - by summarising the available evidence. For diabetes this evidence was particularly strong:

> During a 8-year follow-up of 113 861 women in the USA, aged 30 – 55 years, the risk of developing NIDDM increased with increasing BMI…Compared with women with a BMI below 22, risk was increased 20-fold for women with a BMI between 29 and 31, and more than 60-fold for those with a BMI above 35. Within the total cohort, 90% of diagnoses of NIDDM were attributable to a BMI greater than 22 (WHO, 1995: 324).

In the case of gallbladder disease, the relative risks quoted also showed substantial increases: data from the American Nurses Health Study demonstrated that women with a BMI of above 32 had a six fold increase in their risk, compared with women

[104] They list other associated disorders such as varicose veins, infertility, psychosocial problems, hiatus hernia, sleep apnoea and 'important social and economic disadvantage' (WHO, 1995: 326-7) without any further discussion.

with a BMI of less than 20 (WHO, 1995: 325).[105] However for other conditions the evidence seems more equivocal. In the case of hypertension research linked weight loss to decreased systolic and diastolic blood pressure, but body fat distribution was also seen to be related to hypertension, independently of BMI (WHO, 1995: 324), which undermines the earlier association. For coronary heart disease the evidence also seems mixed. The most direct statement of the association between heart disease and overweight was that:

> The relationship between BMI and CHD has usually been found to be linear, but the level of risk is modified by ethnicity, age, sex, and smoking habits. Elsewhere, it has been calculated that about 40% of the incidence of CHD was attributable to a BMI of above 21 and was therefore potentially preventable (WHO, 1995: 323).

Framingham data showed that a 10% reduction in bodyweight corresponded to a 20% reduction in the risk of developing heart disease (Ashley and Kannel, 1974), but studies following intentional weight loss had not been able to demonstrate this reduction in risk (WHO, 1995: 323), which is unexpected given the strength of the relationship between overweight and coronary heart disease that was being asserted here.

For specific cancers the results described were also equivocal as a review of studies of the relation between weight and six types of cancer showed only one definite increase in risk for endometrial cancer, a probable increase in risk for post-menopausal breast cancer, and that the 'relationships between overweight and cancer of the colon, rectum, ovaries, and prostate are uncertain; reported associations are inconsistent between and within sexes and across populations' (WHO, 1995: 326).

Some of this evidence even seems distinctly unhelpful to the arguments the authors were trying to make: 'Despite the clear relationship between overweight and hypertension… it has been concluded that overweight is not one of the major risk factors for stroke… although three prospective studies…have shown that abdominal fatness may be associated with increased risks for stroke independently of BMI'

[105] But in order to assess the importance of such increases, it would be necessary to know the sizes of the absolute risks involved.

(WHO, 1995: 323). The overall finding was that abdominal fatness was an independent risk factor, which undermines a wider case about the negative health consequence of overweight and obesity *per se*. This description of equivocal or non-supporting evidence was partly a demonstration of transparency and impartiality: the report's authors were publicly weighing all the available evidence, not just the results that supported their case.

Five years later, in TR894, the list of negative health consequences, derived from Framingham and the ACS surveys, remained the same. However, the evidence for these increased risks was discussed in more detail as the authors devoted an entire chapter of the report to this topic. They reproduced the now standard list of the major health consequences – diabetes, heart disease, hypertension, gallbladder disease, psychosocial problems and certain cancers (WHO, 2000: 39) - but for the first time, they divided them into different categories based on increase in relative risk - see table 7.5 (below).

Greatly-increased (relative risk greater than 3)	**Moderately increased (relative risk 2-3)**	**Slightly increased (relative risk 1-2)**
NIDDM	CHD	Cancer (breast cancer in post-menopausal women, endometrial cancer, colon cancer)
Gallbladder disease	Hypertension	Reproductive hormone abnormalities
Dyslipidaemia	Osteoarthritis (knees)	Polycystic ovary syndrome
Insulin resistance	Hyperuricaemia and gout	Impaired fertility
Breathlessness		Low back pain due to obesity
Sleep apnoea		Increased risk of anaesthetic complications
		Fetal defects associated with maternal obesity

Table 7.5 (WHO, 2000: 43)

Many of the new conditions now included in the broader understanding of the negative health consequences, such as cancers, impaired fertility and anaesthetic complications, showed only slight increases in relative risk. This list was reproduced in subsequent policy documents (Health Committee, 2004, Branca et al., 2007b), but this classification was removed, and these conditions were listed as if they were all equally likely. The authors themselves acknowledged that some of these risks were more serious than other, arguing that the 'more life-threatening chronic health

problems' could be classified into four different types – cardiovascular conditions, those associated with insulin resistance, certain cancers and gallbladder disease (WHO, 2000: 43), but this qualification was often also omitted when the information was reproduced in later documents.

For cardiovascular disease and hypertension, the authors argued that

> Obesity predisposes an individual to a number of cardiovascular risk factors including hypertension, raised cholesterol and impaired glucose tolerance. However, longer-term prospective data now suggest that obesity is also important as an independent risk factor for CHD-related morbidity and mortality…The Framingham Heart Study ranked body weight as the third most important predictor of CHD among males, after age and dyslipidaemia…Similarly, in women, a large scale prospective study in the USA[106] found a positive correlation between BMI and the risk of developing CHD (WHO, 2000: 47).

Data from Framingham and the Nurses Health Study was still crucial for obesity researchers constructing arguments about the negative health consequences of overweight and obesity. Without large scale prospective data demonstrating these associations, they could not have made these arguments convincingly.

For hypertension, the authors used data from NHANES II to argue that the prevalence of hypertension in overweight adults was nearly three times that for the non-overweight, and moreover that

> The risk in those aged 20-44 years is 5.6 times greater than for those aged 45-74...which in turn is twice as high as that for non-overweight adults. The risk of developing hypertension increases with the duration of obesity, especially in women, and weight reduction leads to a fall in blood pressure (*ibid.*).

The reasons for the association between increased weight and hypertension was 'not clear' but the suggestion was that it might be related to higher levels of insulin in the blood increasing the amount of sodium retained by the kidneys (WHO, 2000: 48), an example of increasing understanding of the metabolic effects of obesity discussed above.

[106] This refers to the Nurses Health Study.

The specific cancers discussed in TR894 were the same as those discussed in TR854 and the evidence from the American Cancer Study was still cited. Increased risks of endometrial, ovarian, cervical, prostate and post-menopausal breast cancer among the overweight and obese were thought to be 'a direct consequence of hormonal changes' resulting in part from 'excess' abdominal fat (WHO, 2000: 48). However it was difficult to separate out the effects of body size, fat distribution and weight in assessing relative risks for these cancers:

> In addition to overall obesity, intra-abdominal fat distribution and adult weight gain have been independently associated with an increased risk of breast cancer. For example, it has been reported that an increase in intra-abdominal fat accumulation increases the risk of postmenopausal breast cancer, independently of relative weight and particularly when there is a family history of the disease. Furthermore, weight gain during adulthood has consistently been associated with increased risk of breast cancer, even in cohort studies that showed no association between baseline relative weight and subsequent risk of breast cancer (WHO, 2000: 48 - 9).

Data from two prospective studies was used to make the striking claims that 'about 64% of male and 74% of female cases of NIDDM could theoretically have been prevented if no one had a BMI over 25' (WHO, 2000: 50). However, the detail of the evidence provided seems more equivocal, as intra-abdominal fat accumulation 'in some studies, has been an even stronger predictor of NIDDM than overall fatness' and the prevalence of NIDDM was two to four times higher in the least physically active individuals, irrespective of body mass (*ibid.*). Both these pieces of evidence call into question the precision of the authors' earlier claims.

Obesity and overweight was now seen as having important effects on a wide range of hormones because fat had become understood as a metabolically active substance:

> Recent research has shown the adipocytes (fat cells) are more than just fat depots. They also function as endocrine cells, producing many locally and distantly acting hormones and as target cells for a great many hormones. Altered hormonal patterns have been observed in obese patients, especially those with intra-abdominal fat accumulation (WHO, 2000: 51).

The endocrine disturbances associated with intra-abdominal fat accumulation were held to lead to conditions such as insulin resistance and polycystic ovary syndrome,

but the main metabolic consequence of overweight and obesity was seen to be dyslipidaemia, where blood levels of triglycerides and LDL cholesterol are raised and those of HDL cholesterol are lowered: 'This metabolic profile is most often seen in obese patients with a high accumulation of intra-abdominal fat and has consistently been related to an increased risk of CHD' (WHO, 2000: 53). These two types of disturbances - the metabolic and the endocrinological - were often found in combination with other heart disease risk factors, such as high blood pressure and abdominal obesity. In this period, the new diagnosis of 'metabolic syndrome' was beginning to be applied in such situations.[107] There was no authoritative international definition of this condition yet but epidemiological evidence confirmed that it 'occurs commonly in a wide variety of ethnic groups including Caucasoids, Afro-Americans, Mexican Americans, Asian Indians and Chinese, Australian Aborigines, Polynesians and Micronesians' (WHO, 2000: 54). This was another new way of framing high rates of chronic disease in particular population groups (see section 7.4).

A final set of health problems associated with excess body weight was labelled 'psychological' or 'social'. For the authors of the BNF report, four different studies were available, some of which showed no significant relationship between depression, anxiety and obesity. However, the severely obese participants in the Swedish Obese Subjects (SOS) study

> showed very poor ratings for mental well-being and anxiety and depression than the reference population. The scores on psychometric scales were as bad as, or worse than, those of patients with chronic pain, generalised malignant melanoma or tetraplegia after neck injury (BNF, 1999: 11).

This was a significant level of psychological distress, demonstrating that severe obesity was more hazardous to mental health than other degrees of overweight, but this finding was merely cited and not discussed further. In the case of 'social penalties', the authors stated

> There is compelling evidence that our society discriminates against fat people. This is particularly damaging to the psychological well-being of obese children, who are believed by their peers at school to be lazy, dirty, stupid, ugly, liars and cheats…Social discrimination continues into adult life. Sonne-

[107] This was also known in this period as syndrome X or the insulin resistance syndrome.

> Holm and Sorensen showed that for a given parental social class, intelligence and education, obese people achieved less than non-obese. Overweight in adolescence is associated with less social success in later life (BNF, 1999: 12).

In total five different studies were cited to provide evidence for this point, but no further argument was made. This sort of evidence would have allowed discussion of the high levels of psychological distress found in the SOS study, by linking it to the discrimination and prejudice experienced by severely obese individuals. But this was not done, presumably as the authors felt it was out with their area of expertise (see chapter 9).

The authors of TR894 also considered psychological problems, including body shape dissatisfaction and eating disorders, as well as those resulting from dealing with prejudice and discrimination. Such psychological problems were categorised differently from the other health consequences:

> It is important to note that the mechanisms leading to impaired psychological health are different from those underlying physical illness. The psychosocial problems associated with obesity are not the inevitable consequences of obesity but rather of the culture-bound values by which people view body fat and "unhealthy" and "ugly" (WHO, 2000: 56).

Whilst explicitly arguing against the 'negative stereotypes and attitudes of health professionals' (*ibid.*), these authors did not discuss such issues further. Researchers critical of conventional understandings of obesity contend that biomedical research and practice is a key site of discrimination against the overweight and obese which legitimates such attitudes and practices (Bacon, 2008, Campos, 2004, Gaesser, 2002). Official acknowledgement of the stigmatisation of these individuals required careful expression in order to forestall such analyses or accusations of further stigmatisation.

7.6 Dissenting accounts of the health effects of overweight and obesity

Within these reports there was no disagreement about the negative health effects of obesity and overweight – it can be described as a generally accepted consensus. The findings of Framingham and the American Cancer Study were accepted and

incorporated into an explanation of the effects of overweight and obesity that understood it as having profoundly negative effects on the individual metabolism.

Again, Ernberger and Haskew were the most significant internal dissenting voices that I have been able to locate. Not only did they dispute the mainstream interpretation of the evidence about the negative consequences of overweight and obesity, but, more radically, they were prepared to argue that there are health benefits to both overweight and obesity:

> The beneficial effects of moderate adiposity on longevity and the relatively modest hazard associated with extreme adiposity present a paradox when considered alongside the associations between obesity and a variety of diseases. This has led other researchers to speculate that there may be health benefits to adiposity [here they give three references] (Ernsberger and Haskew, 1987: 13).

They argued that greater body weight was associated with decreased mortality rates for cancer, hypertension and cardiovascular disease (see section 4.7), but also that it was associated with a lower incidence of respiratory disease, infectious diseases (notably tuberculosis), bone disease, anaemia and type 1 diabetes, as well as a more favourable prognosis for NIDDM, hyperlipidaemia, hypertension and rheumatoid arthritis (Ernsberger and Haskew, 1987: 18). They cited results from specific studies as evidence for each of these conditions, but I will quote only their argument for cardiovascular disease, since this is such a classic and canonical example.

> Fat people have been shown repeatedly to exhibit elevations in literally every coronary risk factor known: total cholesterol, ratio of high-density to low-density cholesterol, uric acid, triglycerides, glucose and insulin…[however] adiposity is not related to the diseases that these risk factors are purported to predict. The incidence and extent of atheroschlerosis in coronary and other vascular beds is entirely unrelated to body fatness as demonstrated in autopsy studies (Ernsberger and Haskew, 1987: 26).

Furthermore, they also suggested that the positive effects of obesity may be underestimated in certain conditions because studies do not classify individuals according to their fat distribution (see section 4.4). According to their argument, 'nearly all "obesity–related" diseases are associated with accumulation of fat in the upper body' and so 'individuals with predominantly lower body fat may benefit from

the protective effects of adiposity but escape the onus of "obesity-related" disease' (Ernsberger and Haskew, 1987: 24). However, this not to say that adiposity was beneficial to health:

> Even though fatness appears to be protective in some disorders, the overall impact of significant adiposity (BMI of approximately 30 or greater) is undeniably negative. However, the severity of the associated risk is probably less than generally assumed…Furthermore, it is no longer appropriate to consider obesity as a disease if it has benefits as well as hazards (*ibid.*).

In this framing, the negative health effects associated with overweight and obesity were caused by a cyclical pattern of weight loss and regain. Evidence showed that obese animals did not suffer from hypertension or other cardiovascular 'abnormality' which led to the question

> Why then is obesity associated with high blood pressure and heart disease in humans, alone among the animal species? The answer may be that humans are the only animals that diet to lose weight. When dogs, swine, rats or mice are repeatedly deprived until they lose 20 percent or more of their body weight, then are allowed to regain the weight, they develop high blood pressure, undergo damage to their blood vessels and develop heart disease similar to that seen in fat humans… However, animal studies fail to duplicate the major hazards of human obesity, cardiovascular disease, unless the animals are made to lose and gain weight repeatedly (Ernsberger and Haskew, 1987: 38).

Additional human and animal evidence was cited to support this argument, including studies of the effects of the Siege of Leningrad, and of 200 fat men on hospital-run total fasts (Ernsberger and Haskew, 1987: 39). Ernsberger and Haskew were knitting together laboratory feeding studies on rats with clinical data and large scale study data, in the same way as other authors, to provide an alternative narrative of the effects of starvation on human and animal physiology.

However, another dissenting voice was Ancel Keys, who set up the Seven Countries study (see sections 2.5 and 4.3). He argued that insurance company statistics and data produced by the first ACS cancer prevention study were seriously flawed:

> This is another example of the dangerous idea that serious defects in the quality of data can be disregarded if the numbers of persons is very large…The Cancer Society questionnaire, distributed by local non-

> professional volunteers, asked the age, sex, height, weight and smoking habits. For six years Cancer Society volunteers tried to find out who had died and what was reported as the cause of death. No measurements or medical examinations were involved. This approach leaves some uncertainties: Are the answers accurate? Are the subjects representative of the population? Are their results reliable? (Keys, 1980a: 18).

Because of these flaws, he argued that such data did support the conventional analysis that overweight increases the risk of heart disease or early death. In fact he used 'more sophisticated' analyses of data from the Seven Countries Study to argue that 'it is bad to overweight, worse to be underweight' and 'the best prospect of avoiding early death is to be somewhat above the average in relative weight' (Keys, 1980a: 20). The professional interests of a public health scientist like Keys, who pioneered the use of statistics in health research, could easily lead to attempts to devalue the data collection methods used by the insurance industry and voluntary bodies such as the ACS. However, it was an important critique of a body of data fundamental to the development of obesity science, and the article was reprinted in the British Medical Journal in 1986. But such criticism, from a major figure in the field of chronic disease epidemiology, appeared to have little effect on contemporary discussions.

7.7 Conclusion

The understanding of obesity and overweight, and chronic disease more broadly, in WHO reports changed significantly between the 1970s and the 1990s. The concept of diseases of affluence was largely abandoned, and it was argued that increasing rates of these conditions were the result of processes of industrialisation and modernisation (see section 5.5 and chapter 8). This breaking of the links between chronic disease and affluence meant that even relatively poor populations could be seen as at risk, and was a precursor to ideas of the 'double burden of disease' developed by researchers associated with Harvard School of Public Health and the WHO (see chapter 8). It was also consonant with the new framing of increasing rates of obesity and overweight as a global phenomenon developed in WHO publications from the mid 1990s.

The negative health effects of overweight and obesity were one of the most important elements of the arguments of obesity science. In this period, descriptions of the consequences shifted from earlier discussions focusing on heart disease and

mechanical side effects, to a much longer list of metabolic and endocrinological conditions that drew on more detailed understandings of the physiological processes associated with excess weight gain. This increased list was crucially dependent on data from large scale epidemiological studies such as Framingham. Such understandings were being linked to genetic explanations in an attempt to understand the very high rates of excess body weight, and chronic diseases, occurring in populations such as the Pima Indians of Arizona or the Samoans.

CHAPTER 8: THE WHO REPORT OF 2000 AND THE ENVIRONMENTAL PARADIGM OF OBESITY

8.1 Introduction

This chapter continues my analysis of the two WHO technical reports, TR854 and TR894, although it focuses mostly on the latter. It builds on the previous chapter's description of the way increasing rates of obesity were understood as a global phenomenon to describe how the move into the WHO led to an expanded understanding of its causes. Mobilising the concerns of international development central to the work of UN organisations, these gave increasing weight to the effects of industrialisation and modernisation on average body weights. This chapter also develops chapter five's discussion of the limitations of treatment by describing the growing importance of preventive measures in these reports. I describe the increasing attention given to the costs of obesity and overweight to healthcare systems and some of the new data from health economics and related research that was available to the authors of TR894. I give some details of one of these programmes – the Global Burden of Disease project – in order to explain the importance of its work to changing understandings of chronic disease, and ideas of obesity as a global public health problem. Finally, I give a brief summary of the new understandings of obesity and overweight – the environmental paradigm – that I have described in this and the previous chapter.

8.2 A renewed focus on the effects of modernisation and industrialisation

By the late 1990s obesity science researchers routinely referred to broader, more social causes of excess body weight (see section 5.4). These were seen to derive from the significant social changes that had occurred first in developed and then in developing countries since the Second World War. For example, the authors of TR854 contrasted affluent societies, where overweight was associated with low socioeconomic status and education level, to poorer countries where the converse was true. They argued that there had been a shift in European patterns of male overweight in the last forty years (see section 6.3). Studies carried out from the 1950s to the

1970s showed a positive relationship between obesity and socioeconomic status, whereas those carried out in the 1980s and 1990s show an inverse association: for women the majority of studies have shown an inverse relation throughout this period (WHO, 1995: 318). In less affluent countries:

> Overweight may be seen as a visible indicator of wealth and status in countries where food is scarce. Brazil is an example of a country in which there is a clear positive association between socioeconomic status…and BMI…It has been suggested that this association is mediated by the fat content of the diet…

And furthermore,

> In some traditional societies, there are pressures on women both to gain weight and remain overweight during reproductive life. An example of this is the custom of 'fattening huts' for elite pubescent girls in certain communities in West Africa. Such practices reflect cultural perceptions and values related to overweight (WHO, 1995: 319).

This brief discussion of the socio-cultural determinants of overweight consisted of one page of a thirty page chapter. The behavioural determinants were discussed with similar brevity in this report. They included smoking which was initially associated with lower BMI but since the 1980s had become linked to other 'unfavourable' habits such as high levels of consumption of alcohol and saturated fat and therefore higher BMIs. There was seen to be a 'clustering' of health damaging habits amongst particular groups in the population:

> in populations in which there is a growing health awareness and an increasing proportion of people who stop smoking, the remaining smokers are those whose lifestyles carry significant health risks (WHO, 1995: 320).

Despite a significant body of high profile research in this field (e.g. Townsend and Davidson, 1982), no mention was made of in this report of the link between such health behaviours and socioeconomic status.

Five years later in TR894, the causes of obesity were considered first in terms of diet and physical activity: 'high-fat energy-dense diets and sedentary lifestyles are the two factors most strongly associated with increased prevalence of obesity worldwide' (WHO, 2000: 108). Evidence from laboratory studies and clinical studies was cited as

demonstrating that increased amounts of fat in the diet and increased energy intake were both strongly associated with excess body weight. Because dietary fat has a higher energy density than other macronutrients, individuals eating a high fat diet often experienced 'passive overconsumption' (see section 5.3):

> The body does compensate for the overconsumption of energy from high-fat foods to some extent, but the fat-induced appetite control signals are thought to be too weak, or too delayed, to prevent the rapid intake of the energy from a fatty meal (*ibid.*).

Eating fatty food was seen as overwhelming the immediate regulatory mechanisms, and control of intake depended on longer term mechanisms that responded much better to underfeeding than overfeeding. The body's capacity for fat storage was thought to be virtually unlimited, and so when fat balance was disrupted by changes in energy balance, it was re-established by a change in body fat mass (WHO, 2000: 109). Palatability promoted overeating and fat, especially combined with sugar, was particularly palatable: 'Fat appears to be the key macronutrient that undermines the body's weight regulatory systems since it is very poorly regulated' (WHO, 2000: 110).

In the case of physical activity, cross-sectional data had shown that increasing BMI was associated with decreasing levels of physical activity, but, according to the authors, it still had not been possible to establish causality. However, this caveat was quickly ignored to argue that other research demonstrated the role of 'decreased physical activity and/or increased sedentary behaviour' in both weight gain and the development of obesity (WHO, 2000: 112). For the first time, the authors of TR894 provided a detailed analysis of the contribution of physical activity to total energy expenditure, and outlined three components of physical activity – occupational work, household and other chores, leisure-time physical activity (exercise and sport). Physical inactivity or sedentary behaviour was also defined much more carefully as including participation in physically passive behaviours such as watching TV, working on a computer, driving, talking on the phone and eating (WHO, 2000: 113).

Another reflection of increased research in this area was the new concept of physical activity level (PAL), a numerical measure to 'express daily energy expenditure as a

multiple of BMR' (WHO, 2000: 114). Individuals could now be classified into different categories based on their average levels of physical activity – a sedentary lifestyle had a PAL value of 1.4, 'limited activity' 1.55 – 1.6 and 'physically active' greater than 1.75 (WHO, 2000: 114). However, this narrow numerical range hid a significant difference in activity levels since to move from the limited activity to the active category required a daily extra hour of moderate activity[108] or half an hour of vigorous activity (*ibid.*). To reduce the possibility of becoming overweight, individuals should be advised to sustain a PAL of 1.75 or above throughout their adult life (WHO, 2000: 118). However, the authors acknowledged that in industrialised societies, PALS were 'drifting downwards' and that:

> People who make extensive and increasing use of motorized transport, automated work and sedentary pursuits, may find it difficult to attain PAL levels at or above 1.75 simply by increasing activity during "leisure time"…Increasing a PAL of 1.58 to one of 1.76 requires approximately 1 hour and 40 minutes of extra walking (at 4km/h) per day…As these activity requirements are additional to a 24-minute period of active leisure…already required for a PAL of 1.58, it follows that urban sedentary populations are likely to attain a PAL of 1.75 or more only if supported by vigorous national policies that encourage physical activity (*ibid.*).

Arguments like this led to a shift from advocating increased leisure time 'exercise' to promoting 'physical activity' as an integral part of everyday life (e.g. Foresight, 2007). Dietary composition and physical activity levels were also seen to interact as regular physical activity led to an increased capacity to burn fat rather than carbohydrate:

> moderate physical exertion allows free-living volunteers to consume ab libitum a 40% fat diet without storing excess fat, whereas the same individuals, when sedentary are in positive fat and energy balance and thus have a greater risk of becoming overweight and obese with time. If, however, they are offered a 20% fat diet, they remain in balance even when sedentary (WHO, 2000: 116).

This interaction meant that individuals with high levels of physical activity can maintain their weight on a high fat diet, whereas those who were sedentary needed to prevent weight gain by minimising their fat intake. The authors assumed that people

[108] Moderate activity is defined as brisk walking, cycling (12km/h) or gardening and vigorous activity as running, football rugby or hockey (WHO, 2000: 114).

in developed countries were mostly sedentary and so needed to maintain a fat intake of 30% or less (*ibid.*). Little data was available for developing countries, and the comparisons that had been made showed few differences from more affluent countries. Nevertheless, the authors were prepared to argue that:

> it is reasonable to conclude that people in less developed countries who spend a considerable portion of their time in finding food for their next meal and on personal chores are expending more energy in work and physical activity for a given body size than those in more developed countries (WHO, 2000: 115).

Common-sense assumptions that increasing average body weights were a direct result of industrialisation and economic development seem to have overwhelmed the limited data that did exist.

Another body of theoretical material analyses the genetics of obesity and overweight:

> Epidemiological, genetic and molecular studies of populations all over the world suggest that some people are more susceptible than others to becoming overweight and obese, and that such susceptible individuals exist in countries differing widely in lifestyle and environmental conditions (WHO, 2000: 133).

In the case of genetic susceptibility, it did not seem to be that single or multiple gene effects cause overweight and obesity, rather that 'the genes involved in weight gain increase the risk or susceptibility of an individual to the development of obesity when exposed to an adverse environment' (WHO, 2000: 134). Obesity was therefore understood as multi-factorial and polygenic i.e. the result of the interaction of many genes and their environment (see section 5.5).

Estimates of the heritability of obesity vary widely: the heritability of BMI was thought 'likely to be in the range of 25-40%', the heritability of fat distribution to be 50%, and the heritability in the amount of abdominal fat to be 50 to 60% (WHO, 2000: 134 -5). It is not clear what such a range of numerical values adds to the understanding of obesity, especially as the authors argued that there was a lack of data concerning the level of risk of developing obesity for relatives of the overweight and obesity, despite the knowledge that obesity tended to run in families (*ibid.*). Various mechanisms were suggested for the operation of this genetic susceptibility including

low resting metabolic rate (studies of the Pima Indians have shown RMR clusters within families, and that those with lower RMR have greater risks of weight gain); low fat free mass (which was a risk factor for weight gain since it depressed the level of RMR); and poor appetite control which the authors link to research into the role of leptin as a satiety factor (*ibid.*). Other factors that were seen to promote weight gain included smoking cessation, excess alcohol intake, drug treatment, genetic and endocrinological disorders, major reductions in activity levels (due to chronic illness or disabling injuries) and 'changes in social and environmental circumstances' that include marriage, the birth of a child or a new job (WHO, 2000: 139 - 42). This last heading was a catch-all category which was too broad to have any significant explanatory power, but resulted from the exclusion of sociological arguments from such accounts (see chapter 9).

As policy writing on the public health problem of obesity developed in the late 1990s, (see above) understandings of the causes of obesity became more detailed and significantly wider in scope. The authors of TR894 discussed these models under the heading of 'environmental and societal influences' that focused on the wider environmental causes, especially those deriving from processes of modernisation. When compared to the headings in TR854's label of 'socio-cultural' and 'behavioural' determinants of overweight, this is evidence of a shift to a less individualistic and more environmental understanding of the causes of increasing average body weights.

Environmental and societal influences were explicitly linked to changes in social structures resulting from modernisation and industrialisation. Early in a discussion of the regulation of body weight, the authors argued that:

> In traditional societies, where people tend to be more physically active, and provided food supplies are not limited, few adults are either underweight or overweight despite the interaction of seasonal cycles of work, individual susceptibilities to obesity for physiological or genetic reasons, and the wide range of varying physical demands within a society (WHO, 2000: 105).

This distinction between 'modern' and 'traditional' societies played an important explanatory role throughout this report. The section on environmental influences

contains nine separate references to traditional cultures, diets, lifestyles, foods and values as well as extended discussions of the effects of societal change (WHO, 2000: 119-33).

The authors also framed obesity as a major social problem, by placing it alongside other long-standing policy concerns such as unemployment, poor housing and 'broken' families:

> The trend towards industrialization and an economy based on trade within a global market in most of the developing countries has brought about a number of improvements in the standard of living and in the services available to the population. However, it has also had various negative consequences; these have led, directly and indirectly, to deleterious nutritional and physical activity patterns that contribute to the development of obesity. Changing societal structures resulting form this economic transition have given rise to new problems associated with unemployment, overcrowding and family and community breakdown (WHO, 2000: 118-9).

Industrial food production was seen, by these authors, to have led to improved food availability but 'it has not necessarily solved the problem of undernutrition in many poorer countries, nor has it improved the nutritional quality of the diets of the affluent', while motorised transport and mechanisation have lessened the physical burden for many individuals but 'leisure time dominated by television viewing and other physically inactive pastimes has increased' (WHO, 2000: 120). Other changes discussed included modernisation, the transition to market economies, increasing urbanisation, changes in the role of women, changes in labour markets and the globalisation of world markets (WHO, 2000: 120-4). The transition to market economies had led to increased food imports and increased consumption of processed foods; urbanisation to reduced levels of physical activity and increased consumption of protein and fat; rising numbers of women in paid employment to the demand for labour saving devices and convenience foods; changes in labour markets to an increasing proportion of populations working in sedentary occupations; and globalisation to increasing competition between multinational companies to sell processed foods. The underlying assumption was that these changes had resulted in negative health consequences, but no evidence was produced to substantiate this argument. Of these factors, modernisation was seen as most important, and given most explanatory power:

> Most adults who still have a 'traditional' lifestyle appear to gain little or no weight with age. Anthropometric studies have reported an absence of obesity in the few remaining hunter-gatherer populations of the world, since energy expenditure is generally high and food supplies are scarce in certain periods of the year…For the majority of the world's population, however, the process of 'modernization' has had a profound effect on the environment and on lifestyles over the last 50-60 years (WHO, 2000: 120).

Obesity was labelled as 'the first of the so-called "diseases of civilisation" to emerge' (*ibid.*). More broadly, the negative health effects of modernisation were labelled 'New World syndrome':

> Obesity can be seen as the first wave of a defined cluster of NCDs [non-communicable diseases] now observed in both developed and developing countries. This has been called the 'New World syndrome' and is already creating an enormous socioeconomic and public health burden in poorer countries. High rates of obesity, NIDDM, hypertension, dyslipidaemia and CVD, coupled with cigarette smoking and alcohol abuse, are closely associated with the modernization/acculturation process and increasing affluence. The New World syndrome is responsible for disproportionately high levels of morbidity and mortality in the newly industrialised countries, including Eastern Europe, as well as among the ethnic minorities and the disadvantaged in developed countries. …Thus, while obesity is viewed by health professionals from a medical perspective, it also needs to be recognized as a symptom of a much larger global social problem (WHO, 2000: 122).

New World Syndrome was related to ideas of the double burden of disease (see section 8.6) and appears to have been developed in the mid 1990s. However, the term was not widely adopted and does not seem to be currently used.[109] The shift in dietary composition and levels of physical activity was also labelled 'the nutrition transition' (Drewnoski and Popkin, 1997). This characterised such changes as inevitable, a stage through which all societies must pass. Unlike the New World Syndrome this concept is still being used and developed (Popkin and Doak, 1998, Popkin, 2004, Popkin and Mendez, 2007).

> Modern societies seem to be converging on a diet high in saturated fats, sugar and refined foods and low in fiber – often termed the "Western diet" – and on lifestyles characterized by lower levels of physical activity. These developments…are reflected in nutritional outcomes such as changes in

[109] A search on Medline found three articles containing the term, all dating from the mid-1990s.

> average stature, body composition, and morbidity patterns (Popkin and Mendez, 2007: 68).

As in the previous WHO report, the effects of modernisation were seen to lead to a changing relationship between rates of obesity and socioeconomic status. More information was now available to demonstrate how high socioeconomic status was negatively correlated with obesity in developed countries, and 'as the less developed countries attain higher levels of affluence, the positive relationship between socioeconomic status and obesity is slowly replaced by the negative correlation seen in developed countries' (WHO, 2000: 124). In poor countries thinness was still associated with lack of sufficient food and the performance of physically demanding manual work and so 'thin adults are considered poor, and overweight and obesity are a sign of affluence' (WHO, 2000: 125). By contrast in richer countries there was an inverse relationship between obesity and socioeconomic status, meaning that it was associated with poverty rather than affluence. Because few people were short of food and the amount of manual labour performed had decreased significantly, members of lower socioeconomic groups were not, on average, more physically active or short of food than more affluent individuals. But there was evidence that poorer families were less physically active than affluent ones and the poorest households ate cheaper and more energy-dense processed foods rather than more expensive and nutritious food such as fruit, vegetables and whole grains (*ibid.*). The inverse link between education and average body weight found in industrialised countries (WHO, 2000: 126) was seen to reinforce this relationship. Social research was beginning to be incorporated, in order to provide explanations for class differences in rates of obesity and overweight.

The last major cause of obesity and overweight discussed in this report was 'cultural influences'. The authors admitted that these were not well defined or understood, but then went on to argue that

> Cultural factors are among the strongest determinants of food choice. They include peer group pressures, social conventions, religious practices, the status assigned to different foods, the influence of other members of the household and individual lifestyles…Human beings value food for much more than its nutrient content, and it is used to express relationships between people as well

> as in celebrating religious festivities, weddings and other important social occasions (WHO, 2000: 127).

The vagueness and generality of this discussion demonstrates the limited understanding of 'the social' mobilised in such writing. In particular, culture was often understood as a barrier to modernisation, so its influence led to a rejection of recreational physical activity and a valuing of large body size as a sign of prosperity and health.[110] The discussion concluded with a brief outline of changing attitudes to body shape and weight in industrialised countries and the link between the increasing denigration of fatness and rising incidence of eating disorders and the use of 'unhealthy weight control practices' such as diuretics and self-induced vomiting (WHO, 2000: 128). These problems were forecast to increase in developing countries as they adopted the 'values and ideals' of industrialised countries.

8.3 Continuing problems with treatment

There were no new treatments for overweight and obesity discussed in TR894: weight loss diets, physical activity, behavioural therapy, pharmacological treatment and surgery remained the available options, as they had been in previous reports (see section 5. 5). However, the ineffectiveness of dieting was becoming more widely accepted, leading to an increased emphasis on obesity and overweight as life-long chronic conditions that can be managed, but rarely cured.

The authors of TR894 covered the topic from a clinical perspective to aid the development of appropriate treatment strategies, making a distinction between the different techniques used for different aspects of 'weight management': weight maintenance and the prevention of weigh gain require 'healthier eating' and a 'more active lifestyle', whereas weight loss requires a temporary negative energy balance through decreased consumption and increased energy expenditure (WHO, 2000: 206). A BMI-based classification of individual patients to decide on appropriate management strategies was also given. A partial outline of this scheme is given in table 8.1.

[110] The latter includes another mention of African 'fattening huts' for pre-pubescent girls (WHO, 2000: 127) which is consistent with an old fashioned and colonial understanding of culture as a barrier to modernisation.

BMI	Overall health risk	Management strategies
18.5 – 24.9	average	Healthy diet and advice on preventing weight gain
25 – 29.9	increased	Weight maintenance, healthy diet, exercise
30 – 34.9	moderate	Goal of 5-10% weight loss
35 – 39.9	severe	Use full therapy (diet, exercise, behaviour therapy) to achieve > 10% weight loss
BMI ≥ 40	very severe	Refer to specialist for separate management and consideration of surgery if treatment fails. Aim for 20-30% weight reduction

Table 8.1 (adapted from WHO, 2000: 207)

A successful weight management programme should include components of personal support, dietary assessment and advice, modification of physical activity patterns and behavioural advice (WHO, 2000: 208). Regular monitoring was described as crucial, as part of a long-term responsibility for healthcare practitioners: 'it should not cease when patients have reached agreed goals but should form part of continuing care' (*ibid.*). Obesity was seen here as a chronic condition requiring long-term, maybe even lifelong, medical supervision.

However, once again there was an argument against returning to an 'ideal' body weight:

> [This approach] has for too long been considered by the medical profession to be both a possible and a mandatory target for obese people. This misconception has been transmitted to the public, and has been reinforced by the promotion by the mass media of slenderness as the ideal body image. As a result, there is now considerable pressure on the overweight individual to return to his/her ideal, often at the lower end of the normal (18.5-25) BMI range (WHO, 2000: 201).

This was the most explicit critique of past medical practice in this report. This changed approach derived from the fact that weight gain was now seen as a health risk independent of BMI, important health benefits could follow from weight loss of 5 or 10 kg (see section 5.5), repeated failure to sustain weight loss might result in further weight gain and limiting weight gain was important to long term health

(WHO, 2000: 201 - 2). The dangers of repeated dieting and the health benefits of weight stability appear to have become more widely recognised.

As in earlier reports, the authors acknowledged that calorie-restricted diets were not a very effective treatment for obesity:

> Dietary restriction represents the most conventional "treatment" for overweight and obesity. It usually induces weight loss in the short term, but its poor long-term effectiveness, especially when used in isolation is widely recognised…Diets based on healthy eating principles, including the individualized modest energy-deficit and the *ad libitum* low-fat diet appear to have better long-term outcomes (WHO, 2000: 211).

Acceptance of the low success rate of short-term dietary restriction led to the idea of longer-term modification of eating habits as a necessary treatment for overweight and obesity. The two approaches mentioned in the above quote – the individualised modest energy-deficit diet and the *ad libitum* low fat diet – were still staples of contemporary weight loss advice. Low fat diets were initially used for their effects on cardiovascular risk factors, but it was found that patients managed to lose weight on them: one study found a reduction of 10% in dietary fat produced an average 5 kilo weight loss in obese patients (WHO, 2000: 212). Other possible dietary treatments were severe/moderate energy-deficit diets of 1000 to 1200 kcal/day which can produce significant short-term weight loss, but could lead to insufficient levels of certain nutrients and weight regain in the longer term, and very low calorie diets (VLCDs) which could cause rapid weight loss but 'do not seem particularly conducive to long-term weight maintenance' and so should only be used for rapid short-term weight loss in obese patients for reasons such as preparation for surgery (WHO, 2000: 213).

The combination of diet and exercise was seen as more effective than either method alone for promoting fat loss, and the necessary approach was very similar to that recommend in the 1996 SIGN report (see section 5.6).

> Physical activity strategies should aim at encouraging higher levels of low-intensity activity and reducing the amount of leisure time spent in sedentary pursuits. The main aim is to convert inactive children and adults to a pattern of "active" living (WHO, 2000: 214).

'Appropriate' levels of physical activity could be achieved by increasing activities such as walking or cycling by up to three hours daily, or by undertaking 45 to 60 minute sessions of fitness training three times a week (WHO, 2000: 215). Because of the perceived benefits of 'modest' exercise, and its acceptability to patients, walking was recommended as a way of getting overweight and obese patients to increase their physical activity levels (*ibid.*). Physical activity was also seen as important in the prevention of age-related weight gain (WHO, 2000: 320).

As in all the recent reports, behaviour modification was seen as an essential element of obesity treatment (WHO, 2000: 215). The core features of such a programme were self-monitoring, stimulus control, an emphasis on improved nutrition ('rigid dieting is discouraged in favour of balanced and flexible food choices'), cognitive restructuring, improved interpersonal skills and relapse prevention (WHO, 2000: 216). However, the initial enthusiasm about behaviour modification evident in the early reports (see section 5.5) has now faded:

> Behavioural treatment has been more intensively researched, and its effects more thoroughly documented, than any other obesity intervention. It is effective in changing behaviour in the short term and consistently produces significant weight loss in patients with mild to moderate obesity. In the long term, however, results are not encouraging, virtually all adult patients returning to their pre-treatment baseline within 5 years (*ibid.*).

This failure was seen as requiring further research into ways of making such methods more effective, and as an argument for the treatment of obesity as a lifelong endeavour. Just as the patient category was widened to include all those 'at risk' of obesity or the 'pre-obese' (see section 4.4) so the treatment became not a matter of a few months, or years, but of a lifetime.

Drug treatment was described as 'controversial, largely because of failure to understand how it should be used' (WHO, 2000: 217). However,

> Due to the paucity of data, no particular strategy or drug can yet be recommended for routine use. However, the availability of new evidence of long-term efficacy and safety of several drugs currently awaiting approval is likely to change the situation (*ibid.*).

In spite of the regularity with which anti-obesity drugs have been taken off the market due to unacceptable side effects (Ernsberger and Haskew, 1987), such optimism about exciting new drug treatments has been regularly re-stated. Drugs should be used in the treatment of patients with a BMI of ≥30 in conjunction with diet, exercise and behavioural regimes, as part of long-term treatment programme, under medical supervision and continued only if the patient has managed a weight loss of at least 10% (WHO, 2000: 217-8).[111] These principles repeated a well established orthodoxy that had been present throughout obesity science (see section 5.5). The four available weight management drugs were ephedrine and caffeine, tetrahydrolipstatin, phentermine and sibutramine (WHO, 2000: 219). The controversy over the safety of fenfluramine and dexfenfluramine was mentioned (WHO, 2000: 218), but phentermine (see section 5.5) was still listed as an appropriate drug to prescribe. Despite the side effects listed for each drug,[112] the issue of long-term usage of such substances was addressed only in a relatively bland paragraph stating that

> While the clinical tolerance of most drugs appears to be acceptable, their long-term use raises some safety concerns…As with drugs prescribed for long term treatment in other chronic diseases (e.g. hypertension, NIDDM), the risk associated with long-term drug use for weight management must be weighed against the potential benefits for each individual (WHO, 2000: 221).

This argument relies on giving obesity the status of a chronic disease like high blood pressure or diabetes. If it is not framed in this way then the hazards of drug treatment might be seen as unacceptable by both doctors and patients. If critics such as Keys and Ernsberger and Haskew were correct and only significant obesity was dangerous to health, then the side effects and risks of long term drug treatment for excess weight could not have been equated with relatively successful drug treatments for chronic conditions such as hypertension or diabetes.

The final type of treatment discussed was gastric surgery:

[111] The authors admitted that this last criterion has been described as 'unrealistic in most cases' but described it as a current UK guideline.
[112] These include abdominal pain, flatulence, insomnia, irritability, agitation, tension, anxiety, nausea, constipation, dizziness and increased blood pressure (WHO 2000: 219-20).

> Surgery is now considered to be the most effective way of reducing weight, and maintaining weight loss, in severely obese (BMI>35) and very severely obese (BMI>40) subjects. On the basis of cost/kg of weight lost, surgical treatment has been estimated, after 4 years, to be less expensive than any other treatment (*ibid.*).

Given the low success rates for other treatments (see above), it is not surprising that surgery was seen as the most effective treatment for these patients. Whether its relatively low cost outweighs the significant risks to patients was an issue that was not directly addressed here. Vertical-banded gastroplasty and Roux-en Y gastric bypass, were considered 'effective and safe' after studies that followed patients for more than 15 years (WHO, 2000: 221-2). Such treatment was suitable for patients with a BMI greater than 40,[113] who should only be considered for such treatment when other treatment have not worked, and should be 'well informed and motivated with acceptable operative risks' (*ibid.*). According to the authors, the Swedish Obese Subjects study showed that weight loss of 30 to 40 kg usually occurs in the two years after surgery, and

> surgical treatment produced remission of NIDDM in 68% of obese patients and of hypertension in 43%. For those who did not have risk factors at baseline, a 30-kg weight loss was associated with a 14-fold risk reduction for NIDDM, and 3-4-fold risk reductions with respect to the development of hypertension and cardiovascular risk factors (WHO, 2000: 222).

This list of improvements is impressive, but many of them describe reductions in risk factors rather than diseases themselves. However, the risks of such surgery were also significant and included micronutrient deficiencies, neuropathy, post-operative complications, 'dumping syndrome' and post-operative depression (WHO, 2000: 223). The authors argued that most of these complications could be treated with behavioural therapy, and, without citing any supporting evidence, that 'Operative mortality in experienced centre is a fraction of the mortality observed in unoperated patients' (*ibid.*). Developments in surgical techniques, combined with the perceived failure of other treatment methods, was leading to a situation where surgery was seen as the most effective treatment option for obesity, if not (yet) overweight.

8.4 The growing importance of prevention in the 1980s and 1990s

[113] Or ones with a BMI of 35 plus 'high-risk, life-threatening comorbid conditions' (WHO, 2000: 222).

The ineffectiveness of available treatments combined with projection of rapidly rising rates of obesity and overweight, led to a stress on prevention. As excess body weight was seen as difficult to treat once developed and affecting increasing numbers of the population, so prevention became a logical focus for public health policy. This topic was not written about regularly in early obesity science, but by the 1980s some authors were discussing it. Philip James stressed the need for prevention, as well as treatment:

> A primary care physician in Britain can expect at least a third of his adult patients to be overweight and this constitutes an extraordinary workload if he decides to undertake treatment individually. Therefore the problem is often ignored. Yet recognizing that an individual has Grade I obesity could trigger a whole strategy of simple management which would prevent a worsening of the condition, highlight problems of lifestyle which might need to be corrected and alert the physician to the need for preventive action with other members of the family (James, 1984: 636).

A 1987 British textbook (Bender and Brookes, 1987) contained an entire section on prevention – one of the earliest textbooks to do so – which included chapters on obesity as a population issue by epidemiologist Geoffrey Rose (see section 7.2), the role of exercise, Norwegian nutrition policy and the North Karelia project. Rose argued that the problem of obesity was an 'adverse shift of the whole risk factor distribution' and therefore potential solutions must 'correct the underlying causes of this shift' which were low average energy expenditure, changes in the availability and nature of food and social attitudes' (Rose, 1987: 181). His recommendations were that overweight should be prevented from occurring in childhood and early adulthood, that individuals should maintain a 'healthy' weight until retirement by increasing their energy expenditure, and that weight loss should be encouraged only in those with other risk factors such as hypertension, a family history of heart disease or diabetes or the severely overweight (those in the top 20% of the weight distribution) (Rose, 1987: 186).

The chapter on Norwegian nutrition policy summarises the country's setting of dietary goals to reduce the average amount of fat in the population's diet and increase

the amount of starchy foods and fibre, using various types of subsidies. The author argued that

> The consumption of cereals, vegetables, fruit, fish and low-fat milk has increased. Furthermore, there has been a reduction in the intake of margarine and whole milk. However, there has been no change in consumption of potatoes, meat and sugar…Concomitant with the changes in the national diet there has been a reduction in deaths from coronary heart diseases and related disorders…It is tempting to believe that the Norwegian Nutrition and Food Policy may be one of the reasons for this decline in heart diseases (Norum, 1987: 209).

This sort of government policy was possible only in a country like Norway where the government sets the prices for a significant number of food stuffs and there was some consensus about its level of involvement in food production, manufacture and retailing.

The final chapter discussed the North Karelia Project as a practical example of a community-based health education programme (see section 2.5).[114] Using Rose's ideas the authors argued that:

> The central aim is to influence the risk factor levels of the community through comprehensive intervention, using the available community structures and naturally occurring interactions. This approach will be more effective in reducing the community disease rates than would a restricted, even intense intervention among high-risk people alone, because most cases of disease come not from the relatively few people with high risk factor levels but from the large majority of the population having moderately elevated levels, but usually several risk factors. Risk-related behaviour is deeply embedded in ways of life and in the social and even physical environments. Thus, to influence permanently the health-related lifestyles of large numbers of people must involve a general process of social change (Puska et al., 1987: 211).

This is the logic for population wide prevention of overweight and obesity, rather than prevention at a purely individual level. It also acknowledged, if only in passing, the ways in which habits of eating and physical activity were socially embedded.

[114] Despite the fact that it was set up as a preventive programme aimed at cardiovascular disease in general, rather than as an obesity prevention programme, North Karelia is commonly cited as the only successful example of an obesity prevention programme.

The 1995 Health of the Nation report contains the next extended discussion of prevention: as part of a 'population approach' to obesity, the majority of its recommendations were preventive measures. These recommendations targeted key organisations. For example, the NHS should improve nutrition training for health professionals and develop schemes to prescribe exercise; local authorities should increase provision of safe cycle routes and footpaths and make facilities for physical activities more accessible to those on low incomes; schools should develop programmes for safe walking and cycling to school and devote more teaching time to PE; and the food industry should develop educational resources to explain the idea of energy balance (DoH, 1995: 21). Whilst these were worthwhile activities, all were framed as voluntary and none addressed socio-economic structural issues around diet and physical activity.

The authors of the SIGN report argued that prevention was necessary to decrease the increasing burden on ill-health that rising rates of obesity will generate:

> In most countries the rate of obesity is doubling every 5-10 years. This means that in a Scottish practice of 10,000 perhaps 80 extra patients will become obese every year unless steps are taken to prevent this (SIGN, 1996: 27).

A preventive approach was recommended despite the fact that there was no evidence of what measures should be used to prevent obesity and no formal trials of obesity prevention had yet been carried out (*ibid.*). The author was limited to discussing healthy eating advice, and the desirability of providing weight management advice to those in situations, such as enforced immobility, steroid therapy and smoking cessation, that led to weight gain.

The authors of the BNF report also began their discussion of the topic by stating that, despite the simplicity and obviousness of the solutions to obesity, there was very little evidence of the effectiveness of health education behaviours in promoting such behaviours (BNF, 1999: 139). The traditional health education approach, used in the 1992 UK Health of the Nation White Paper, was not seen to be working:

> This met with a resounding lack of success. The public received and understood the message, but the prevalence of obesity continued to increase (BNF, 1999: 141).

The other example of a population wide prevention programme discussed was the Minnesota Heart Health Programme, which, as its title suggests, was designed to reduce cardiovascular risk rather than obesity per se. This programme also showed no effect on the average BMI of participants (*ibid.*). Alternatives to these approaches mentioned in the BNF report were individual incentives such as financial sanctions for overweight and obesity or an 'ecological' approach which seeks to alter the wider environment 'to promote lower energy intake and greater physical activity' (BNF, 1999: 144). Referring to the SIGN guidelines, the authors argued that healthcare professionals had begun to address the issue of prevention, but that,

> At present, opinion formers in the media see obesity as mainly a social and cosmetic problem, not a serious public health hazard…Progress on prevention will not be made until obese people are seen to have a physical disability, for which they need help from both health-carers and from society in general (*ibid.*).

The understanding of obesity as a physical disability does not seem to have become widely accepted, perhaps due to the persistence of moralistic ideas that it results from excessive consumption due to poor self-control.

8.5 Prevention in WHO Technical Report 894

The increasing stress on prevention in obesity science, which began in the 1990s, developed out of projects such as North Karelia, but also fed back into WHO reports. By the publication of TR894, obesity and overweight was being understood as a global problem (see section 7.3) concerning the health of populations. This, combined with acknowledgement of the limitations of treatments, meant that prevention techniques became seen as important strategies, and were discussed in more detail. The problems with current individual approaches were that

> The benefit of nutritional knowledge *per se* appears to be limited. Surveys indicate that, although some people know what constitutes a healthy diet, they prefer in practice to consume a relatively unhealthy one…Obesity rates

> continue to climb, despite the increased frequency of dieting among obese people, suggesting that knowledge and frequent attempts to slim are insufficient for successful weight control. However, without these widespread attempts to control body weight, the prevalence of obesity in industrialized countries might be much higher (WHO, 2000: 126).

In a skilful rhetorical turn, the problem of widespread lack of success in dieting became fresh evidence for the importance of the issue as a public health problem. This led to arguments for preventative approaches that were framed in the context of increasing obesity rates and newly identified environmental causes (see section 8.2). Prevention became seen as an integral element of health professionals' management of overweight and obese patients:

> Until recently, obesity prevention and obesity management were perceived as two distinct processes, the former being aimed at preventing weight gain and the latter concerned with weight loss. Management was seen as the role of the clinician, whereas prevention was considered to be the domain of health promotion or the public health departments. However, it is now realized that obesity management covers a whole range of long-term strategies ranging from prevention, through weight maintenance and the management of obesity comorbidities, to weight loss…The individual strategies are interdependent, so that truly effective obesity management must address all of them in a coordinated manner (WHO, 2000: 156).

This co-ordinated approach could be seen as a group of clinical medical researchers trying to extend the use of their concepts and methods into areas of public health medicine. In the area of diet, prevention could be seen as the concern of several other professional groups including those working in health promotion, primary care and public health. Such fragmentation allowed new groups, like the obesity science coalition, to also claim ownership of the issue. At the same time the aims of obesity prevention were expanding beyond preventing normal-weight individuals becoming obese, to preventing the development of overweight in these individuals, preventing the overweight from becoming obese, and preventing weight regain in those who had lost weight (WHO, 2000: 158 -9). Such interventions were also seen to operate at several different levels - universal or public health prevention directed at the whole community, selective prevention directed at high-risk individuals and groups, and targeted prevention directed at those with existing weight problems (WHO, 2000: 160). This multi-level perspective increased the scope of such measures and, therefore, their encroachment into the public health domain.

The authors admitted that there had been little research on the effectiveness of such strategies and increasing prevalences 'cast doubt on whether it is even possible to prevent excessive gains in body weight in the long term' (WHO, 2000: 157). This lack of research and doubt about effectiveness led to detailed discussion of a 'public health approach' to overweight and obesity:

> A public health approach to obesity concentrates on the weight status of the populations as a whole, in contrast to interventions that deal exclusively with factors influencing the body fatness of individuals. In many developed and developing countries, underprivileged minority groups have to bear a disproportionately heavy burden of higher than average levels of obesity. Thus, in efforts to remove inequalities of health status as one of the main aims of public health, it is necessary to consider the causes that make particular groups more vulnerable to weight gain (WHO, 2000: 174).

This statement contains one of the few mentions of health inequalities in this report where public health was seen to be able to 'remove' health inequalities. This optimism seems profoundly at odds with the earlier description of the environmental causes of overweight and obesity.

One of the arguments for such a population-based approach comes from the relationship found between the average BMI of a population and increases in the prevalence of obesity in that population

> [Geoffrey] Rose[115] found that a 4.66% increase in the prevalence of obesity for every single increase in the population's average BMI above 23, resulting in a strong correlation between the average adult BMI of a population and the proportion of adults with obesity…In the United Kingdom between 1980 and 1993, the mean BMI increased from 24.3 to 25.9 for men and from 23.9 to 25.7 for women. Over the same period, the rates of overweight increased by one-third, whereas those of obesity doubled. This implies that further increases in mean BMI are likely to result in even more dramatic rises in the rate of obesity (WHO, 2000: 178).

This argument led to the concept of optimum population BMI: to minimise the numbers of both underweight and obese adults an average BMI of 23 was thought to be optimum, whereas to minimise only the numbers of overweight an average BMI of

[115] Rose is a British epidemiologist (see section 7.2).

21 was thought to be optimum (WHO, 2000: 178-9). Giving such figures seems futile in the face of earlier descriptions of relentless increases in average bodyweights caused by widespread environmental change.

However, the social differentiated nature of obesity rates provided indirect evidence 'that there are environmental conditions as well as genetic factors that can protect populations' (WHO, 2000: 157-8). This was a strangely circular argument that used the evidence for the public health problem of obesity to also argue for potential preventive solutions. Data from American and Finnish[116] populations was given as evidence that individuals of higher social class showed smaller weight gains than those of lower social classes and that these lower weight gains might be levelling off; a parallel was drawn between obesity and other epidemics of chronic disease, such as heart disease, which were 'abating' in countries where preventive measures had been implemented (WHO, 2000: 158). However, far from showing that such measures were effective, such figures might just have illustrated an exacerbation of existing health inequalities, as affluent groups were more able to follow preventive health advice and their mortality and morbidity rates were declining accordingly. Obesity and overweight were in the process of becoming even more strongly associated with poverty and disadvantage.

The kinds of public health interventions envisaged by the WHO report were either those that aimed to educate and improve the skills of individuals, or those that aimed to modify the environmental causes of obesity. Interventions of the first kind had not generally been very successful:

> Communities are generally well aware of the problems associated with obesity, and many individuals are actively attempting to control their weight. Participation rates in these kinds of programmes are usually high, and many succeed in reducing their weight in the short term. Nevertheless, there is generally little impact on the overall average BMI of the community and a negligible effect on obesity prevalence, so that preventive strategies are obviously of great importance (WHO, 2000: 180).

The second kind of intervention was, therefore, seen as potentially more 'effective', but, 'there have not been any well evaluated and properly organized public health

[116] The Finnish figures are derived from the North Karelia study.

programmes aimed at the population-level management or prevention of obesity' (WHO, 2000: 183). Singapore was the only country that had attempted to tackle rising obesity rates by means of a national healthy lifestyle programme targeting particular population groups.

> The Trim and Fit programme was launched in 1992 and is aimed at all children in Singapore. It combines progressive nutrition changes in school catering, and nutrition education together with regular physical activity in schools…Recent results indicate that the number of children successfully completing the fitness tests annually is increasing, and that obesity rates fell from 14.3% in 1992 to 10.9% in 1995 for primary students, from 14.1% to 10.9% for secondary students, and from 10.8% to 6.1% for junior college students…However, it should be noted that this decline in obesity rates may have been somewhat exaggerated because of the new weight-for-height norms introduced by the Ministry of Health in 1993 (WHO, 2000: 184).

Singapore is a small city state with an authoritarian government that was willing to use legislation to alter the personal behaviour of its citizens. Presumably, this was an important factor in the programme's success and makes it unlikely that the results could be replicated in other countries. This was the only national intervention that the authors could cite and the rest of the discussion used examples of heart disease prevention programmes[117] which included reduction of BMI as one of their outcomes. These all showed reductions in CHD risk factors in treatment groups, but only one showed any effect on obesity rates. In fact, in one

> BMI showed a strong secular increase despite such innovative weight-control programmes as adult education classes, a workplace weight-control programme, weight loss by correspondence course and a weight-gain-prevention programme (WHO, 2000: 185).

The North Karelia Project was repeatedly cited as a successful anti-obesity programme, yet 'Despite remarkable reductions in CHD risk factors, which were still declining in 1992…the average BMI and the level of obesity remained similar throughout the project' (WHO, 2000: 185 - 6).

[117] These included the Stanford Three Communities Project, the Stanford Five Cities Study, the Minnesota Heart Health Programme and the North Karelia Project.

Various reasons were suggested for this lack of success including a focus on heart disease risk rather than obesity, the failure to reach a sufficiently large proportion of the population and trying to target too many behaviour changes at once. But the one reason that showed the most congruence with the causes outlined in other parts of the documents was that:

> Powerful societal and environmental obesity-promoting factors have developed rapidly in many societies in the last few decades, and the intervention programmes have not been strong enough or sufficiently well coordinated to overcome them (WHO, 2000: 186).

Such arguments make it difficult to see how obesity prevention programmes based solely on changing individual behaviour could ever be successful. Conceding this point, the authors argued instead for programmes that 'reduce the exposure of the population to obesity-promoting agents by concentrating on environmental factors such as transportation, urban design, advertising and food pricing that promote the availability of high-fat, energy-dense diets and physical inactivity' (WHO, 2000: 190).

Population level interventions were seen as a 'shared responsibility' that should 'involve the active participation of governments, the food industry/trade, the media and consumers' (WHO, 2000: 167). However, the effects of government activities were described as contradictory: in principle, governments' regulatory activities should contribute to the health of the population. However,

> Modernisation and the competing demands of economic development and health have sometimes created a situation where actions by governments have contributed to a decrease in physical activity and an increase in the intake of energy-dense food, contrary to their own health guidelines (WHO, 2000: 128).

Recognition of this contradiction did not to lead to any discussion of the problems inherent in calling for government action to provide public health solutions (see table 8.2 below). The effect of the food industry could also, at best, be labelled contradictory, but this issue was also not addressed. Instead the authors described how technological advances increased food availability, but in carefully neutral language then argued that:

> Advances in food technology have also contributed to the consumption of diets increasingly dependent on processed foods…food characteristics are often manipulated to such an extent that it is difficult for individuals to associate visual, textural or taste cues with the energy content of meals. This is especially important given the increasing trend towards prepackaged foods and the concomitant decline in the use of natural and basic foods in food preparation in the home. Consumers are losing control over the preparation of the foods that they eat, and food composition is increasingly being placed in the hands of manufacturers (WHO, 2000: 131).

Very little evidence was produced for this claim – just one reference to a report on trends in consumer food choice – and its appeal to nostalgia was also part of the link made, again without any evidence, between rising obesity rates, decreasing consumption of home prepared foods and increasing consumption of take-away foods (WHO, 2000: 132). A stress on the role of consumer choice also blunted the impact of a discussion of the role of marketing and advertising in the selling of highly processed foods (WHO, 2000: 133).

Such considerations led to the list of possible strategies reproduced in Table 8.2. A substantial proportion of the entries relate to consumer education, presumably because it was one the most politically palatable options for an organisation like the WHO to recommend in a consensus report.

Area for action	**Example of possible strategies**
Urban design and Transportation policies	Create pedestrian zones in city centre Construct safe walkways and cycle paths Introduce incentive schemes to encourage uses of car parks in the outskirts of cities in conjunction with city public transport (e.g. park and ride Provide affordable facilities for securing bicycles in cities and public areas Improve public transport (e.g. frequency and reliability of services) Install traffic-calming measures to increase safety of children walking and playing in streets Allocate resources to build and manage community recreation centres Modify building design to encourage use of stairs
Laws and regulations	Improve labelling of food products Limit and regulate advertising to children
Economic incentives	Introduce subsidies for producers of low-energy-dense food (especially fruit and vegetables) Reduce car tax for those who take public transport to work during the week Provide tax breaks for companies that provide exercise and changing facilities for employees
School curricula	Provide adequate sport and activity areas and facilities, including changing and showering areas Ensure allocation of sufficient curriculum time to physical activity Ensure training in practical food skills for all children
Food and catering	Develop nutritional standards and guidelines for institutional food services and catering (e.g. school meals and workplace catering)

Promotion and education	Promote from an early age a knowledge of food and nutrition, food preparation, and healthy diets and lifestyles through curricula for schoolchildren, teachers, health professional and agricultural extensions personnel Limit television viewing by children Use the media to promote positive behaviour change (e.g. through television series) Educate the public, especially in areas where food is purchased, on appropriate behaviour change to reduce the risk of weight gain Educate the public on the need for collective action to change the environment into one that promotes rather than inhibits exercise and healthy dietary habits Educate the public about important factors in the development of obesity so that the victimization of the obese is reduced
Family food production	Encourage use of land in towns and cities for "family" growing of vegetables, legumes and other nutrient-rich crops

Table 8.2 (WHO, 2000: 193 - 4)

The discussion ended with another call for structural change addressed to developing and newly industrialised countries that, again, calls for action going well beyond the list given above.

> As in developed countries, obesity in the developing and newly industrialized countries will not be prevented simply by telling individuals and communities to change their diet and exercise behaviours. What is needed is a radical improvement in the social, cultural and economic environment through the combined efforts of government, the food industry, the media, communities and individuals (WHO, 2000: 193 - 4).

This strikingly depoliticised view of potential solutions recurs throughout much subsequent policy writing in this area (e.g. Foresight, 2007). It contrasted with the much more mundane and pragmatic nature of the measures advocated, such as those outlined in table 8.2. These two approaches co-existed in such documents without their contradictions causing problems because of their different functions (see chapter 9).

8.6 Estimating the costs of obesity and the growth of health economics

The argument that obesity and overweight gives rise to substantial costs to health care systems had a dual function within obesity science writing. Firstly, it established obesity and overweight as a serious health problem, legitimating the expertise of researchers and clinicians who work in this area. Secondly, it formed an important

part of the economic argument for both prevention and treatment. Figures estimating the large costs of obesity and overweight have been a notable feature of the obesity epidemic discourse of the last ten years (WHO, 2000, Health Committee, 2004, Foresight, 2007). Previously, they had been included only in the reports published by the Organisation for Health Economics, presumably due to its specialist interest in the topic (OHE, 1969, West, 1994).

However, marked differences in the ways the two OHE reports described these costs demonstrate that there had been significant developments in methods of analysing and forecasting the economic impact of chronic diseases. This growth occurred as part of the growth of the academic discipline of health economics. A study of the field, carried out in the late 1980s, argued that 'there was no *community* of health economists in Britain before 1970; even though there were undoubtedly several academic economists at that time who were professionally interested in health' (Ashmore et al., 1989: 5). The authors argued that an examination of academic discourse, textbooks, courses and research centres and professional organisations and conferences showed that by the period of their study 'an organised community has come into existence' (*ibid.*). The existence of such a community is further demonstrated by the increasing availability of economic analyses of healthcare expenditure and their growing sophistication.

In contrast to detailed modern accounts of the enormous expenses incurred by treating obesity, the author of the 1969 OHE report used a very simple breakdown and described the costs of obesity to the NHS as relatively insignificant (see section 3.4.6). Twenty five years later, when the second OHE report was published, much more detailed figures were available. The direct costs to the NHS were estimated to be £29.3 million (West, 1994: 38). This figure was calculated from estimates of the costs of GP practice consultations (which make up nearly half of this figure), in- and out-patient hospital treatments, prescriptions for appetite suppressants and treatment by dieticians (West, 1994: 38-9). However, West went on to argue that:

> The additional costs to the National Health Service for treating conditions triggered by obesity will, however, be much larger and would include a proportion of the cost involved in post-operative care of surgical patients,

> treating heart disease, stroke, diabetes, some cancers, osteoarthritis and hypertension among others (West, 1994: 39).

The negative health consequences of overweight and obesity (see section 7. 5) meant that the costs of overweight and obesity were much higher than these direct costs. Calculations of cost for various conditions associated with overweight and obesity– heart attack, stroke, NIDDM, arthritis and hypertension – and percentages due to obesity were used to estimate the 'obesity cost' which ranged from £100 million for diabetes to £7.75 million for heart attacks (West, 1994: 41). Approximately 10% of the overall cost arising from these conditions was due to overweight and obesity, but this varied from 5% for stroke to 80% for diabetes (*ibid.).* In total the annual costs of obesity to the NHS in the late 1980s were estimated to be £165 million (West, 1994: 40-1). These were not the only costs that could be attributed to obesity and overweight – British spending on meal replacements and diet magazines, only a couple of the available products, amounted to £80 million and £5 million (West, 1994: 41).

West used as a model for his calculations an article published by Graham Colditz in 1992 (Colditz, 1992). In this article, Colditz estimated the total costs of obesity to the US economy in 1986 to be between $39 and $56 billion dollars. He linked obesity to an increased risk of diabetes, hypertension, CVD, gallbladder disease and certain cancers to calculate these figures, and included both the direct costs to the healthcare system and the indirect costs to the American economy such as loss of output and productivity due to morbidity and mortality resulting from overweight and obesity. This initial calculation produced the figure of $39 million, but West argued that this 'may be an underestimate since several cancers have been omitted as have musculoskeletal disorders such as osteoarthritis' and if these were included 'the total costs of obesity amount to 7.8 per cent (or $56.3 billion) of the total costs of illness in the US' (West, 1994: 39-40). These percentages were derived using disease-specific estimates of American healthcare costs and then assigning a percentage of causality to overweight and obesity on the basis of epidemiological studies such as the Nurse Health Study. The prevalence of diabetes is closely associated with increasing bodyweight and so 57% of the healthcare costs due to diabetes were attributed to obesity, whereas the figure for gall balder disease was 30%, for hypertension 77%, for

CVD 19%, for breast cancer and colon cancer combined 2.5% (Colditz, 1992: 504S - 506S). The 'geological' layering of assumptions involved in the production of such estimates makes them vulnerable to gaps and inaccuracies in the original data and means that they contained larger elements of uncertainty than were usually acknowledge when they were quoted in different contexts (MacKenzie, 1990).

By the publication of TR 894, there was a well developed analysis of the costs of obesity that was given an entire chapter of this report. This illustrates the continuing growth of the relatively new discipline of health economics and the development of new techniques for analysing the costs of healthcare, particular those associated with specific conditions. Using a rhetorical technique that was very characteristic of these reports, the authors begin this chapter by stating that

> To date, there have been only a few attempts to quantify the economic burden of obesity-related morbidity and mortality. This is in marked contrast to smoking and alcohol consumption, where a large number of international studies have been undertaken to determine the magnitude of the economic burden that they impose on the community (WHO, 2000: 78).

This statement contains the familiar device of proclaiming the paucity of evidence whilst using it to make firm arguments. It also continues the comparison between overweight and obesity and alcohol consumption and smoking that I have discussed earlier (see sections 5.5 and 6.5). In the context of economic costs, this comparison was particularly important because it framed excess body weight as a problem of self-discipline and therefore an illegitimate reason for increased healthcare expenditure (Petersen and Lupton, 1996).

Total costs attributed to obesity in developed countries began to increase sharply: in TR894, the authors estimated them at between 2% to 7% of total healthcare costs (WHO, 2000: 84). These percentages translated into estimated direct costs of 464 million dollars (Australia 1989/90), 1 000 million gilders (Netherlands 1981-9) 12 000 million francs (France 1992) and 45 8000 million dollars (USA 1994) (WHO, 2000: 83). The American costs derived from the 1992 Colditz study (see above) and a revised estimate published in 1994 that increased the number of obesity-associated diseases included in the analysis. Despite this expansion the authors still argued that

this estimate 'should still be considered conservative' (WHO, 2000: 86). The first Colditz study was replicated to produce the estimates for the Australian healthcare system, whereas the Finnish, French and Dutch studies used different methods.

> The scope and methodology of the various studies vary considerably in terms of the diseases costed, the definition of obesity, the cost categories used and the epidemiological assumptions as to the relationship between obesity and disease (WHO, 2000: 83 - 84).

Reliable comparisons between different countries were difficult to make, but such caveats, along with considerations of the uncertainties inherent in producing such estimates, were included as these figures became reproduced in different contexts. Like lists of health consequences (see section 7.5) such figures were stripped of their uncertainties and caveats as they moved from one context to another by being repeatedly cited in research articles and policy documents. This parallels processes undergone by other forms of scientific knowledge which are described in the image of the certainty trough (MacKenzie, 1990), where those who are nearest to and furthest from the knowledge production processes are least certain about its accuracy, whereas those in the middle, who are disseminating the information, are most certain of its accuracy.

The growth in health economics is shown by the number of new techniques that were now used to analyse the costs of obesity. The authors of TR894 focused most of their discussion on the cost of illness study but also referred to the disability-adjusted year (DALY) in their attempt to quantify these costs (see section 8.6). However, the DALY had not at that point been used to estimate the costs of obesity[118] – and, again, the authors used the parallel with smoking to argue for further research to be done in this area:

> Obesity and overweight, in the same way as tobacco use, contribute to several NCDs. Thus, the total DALY loss attributable to obesity and overweight would represent the attributable fraction of the total loss of DALYs due to NCDs associated with excess body weight. A number of estimates of the attributable fraction associated with tobacco use have been made, thus facilitating national and regional comparisons. Efforts should therefore be

[118] The Global Burden of Disease analysis (see section87.6) considered malnutrition, hypertension and physical inactivity as causes of disease but not obesity or overweight (Murray and Lopez, 1996: 28).

made to generate similar estimates of the attributable fraction associated with obesity and overweight (WHO, 2000: 83).

The main technique that was discussed in TR894 is the 'cost-of-illness' studies. Such studies calculate the direct cost to the community (healthcare costs of obesity and related diseases), intangible costs (cost to the individual in terms of health and quality of life) and the indirect costs (welfare and economic benefits lost by other members of society) using measures such as PAF (population attributable fraction) or DALY (WHO, 2000: 78 - 9). The PAF was defined as 'the proportion of total events (e.g. death or morbidity) in a population that could be prevented if a particular risk factor e.g. obesity could be eliminated' or more simply the 'preventable proportion' (WHO, 2000: 81-2). It was calculated using the prevalence of a risk factor such as obesity in a population and the relative risk of the incidence of a particular associated disease in an obese person, compared to that of a non-obese person, and its accuracy was highly dependent on estimates of both the prevalence of the risk factor and the relative risk of the disease in particular populations. This means that it is vulnerable to the same criticisms as assertions of the health consequences effects of overweight and obesity (see section 7.5). In yet another plea for further research, the authors also argued for a systematic review or meta-analysis 'to provide a clearer understanding of the relationships found in such studies between excess weight and the diseases' (WHO, 2000: 82).

Cost of illness techniques were seen to have two major limitations. The first was that 'intangible costs and many of the direct costs of disease management and prevention, especially those incurred outside the formal health care system, tend to be ignored' (WHO, 2000: 78). Although studies of the broader economic costs of overweight and obesity were cited (WHO, 2000: 86- 87), there were only four of them,[119] mostly from Scandinavian countries, and it was not clear whether their results could be generalised. The second was that such studies can be 'misused' since diseases with high costs should not necessarily be given high priority, because of ineffective existing treatments or insufficient impact on overall mortality and morbidity: dental disease incurs high costs but low morbidity and mortality compared to youth suicide which incurs low health care cost but a high burden in terms of mortality and

[119] This included the Swedish Obese Subjects study.

reduction in quality of life (WHO, 2000: 81). Such judgements about the burden of disease and quality of life could not be reduced to the financial costs and benefits of particular treatments.

The report's authors admitted that there had not been any comparable studies on the economic impact of obesity and overweight in developing countries, so they cited studies by the WHO and the World Bank analysing the 'increasing burden associated with the rapidly emerging adult NCDs.' (WHO, 2000: 88)

> In developing countries, about 50% of deaths in 1990 were caused by NCDs, but by 2020 that proportion is expected to rise to almost 77%...In contrast in developed countries 87% of deaths in 1990 were from NCDs, and the proportion is expected to rise only slightly – to 90% - by 2020 (*ibid.*).

These figures come from the Global Burden of Disease study and the World Health Report of 1997. But the fact that these were the only figures the authors could quote demonstrates another facet of the omission of developing countries (and, therefore, a large percentage of the world's population) from this discourse.

The authors of TR894 also gave preliminary analyses of the costs and benefits of different obesity treatments. Very little research had been done on this topic apart from the Swedish Obese Subjects study which compared a group of obese individuals who had undergone weight loss surgery with a control group who did not. The results showed that the prevalence of diabetes decreased by 68% in the group that had surgery, and there was a four fold reduction in the risk of developing hypertension and raised blood cholesterol (WHO, 2000: 89). Applying the reduction of diabetes prevalence 'would decrease the total cost of obesity in [France] by approximately 3%, while in the USA costs could be reduced by almost 20%' (WHO, 2000: 90). These figures were highly provisional as they consisted of aggregates of estimates and assumptions piled on top of each other. More prosaically, they did not include the costs of surgical treatment, or subsequent medical care, which given the risks of such surgery might have been more substantial that the authors allowed.

The economic evaluation of obesity prevention and treatment was also an under-studied area and the authors could only summarise the results of two studies of

interventions for the prevention and treatment of diabetes in Australia and of the cost-effectiveness of different weight loss treatments in the Boston area (WHO, 2000: 91 - 95), but neither of them provided useful information for assessing the financial benefits of obesity prevention programmes. Again, there had been no studies done in developing countries:

> No analyses have been made of the economic costs of obesity treatment in developing countries. However, other analyses of the costs of health interventions show that prevention is more cost-effective than treatment once a disease is diagnosed (WHO, 2000: 95).

The cost of the clinical services required to treat chronic diseases was seen to be entirely beyond the resources of low income developing countries, and a major cost for the healthcare systems of middle income countries, therefore 'it would appear to be more cost-effective for money spent on obesity and other NCDs to be used for prevention rather than for expensive treatments during the advanced stage of disease.' (WHO, 2000: 96). A final point echoes earlier writing on the 'epidemic' of coronary heart disease (see section 2.2).

> In developing countries where NCD epidemics are emerging or accelerating, a large proportion of NCD deaths occur in the productive middle years of life, at ages much younger than those seen in developed countries. The health burdens attributable to excess weight gain in societies in transition are likely to be huge because of the absolute numbers at risk, the large reduction in life expectancy and the fact that that the problem affects, in particular, individuals with a key role in promoting economic development (WHO, 2000: 97).

Despite a lack of evidence this rhetoric argument still managed to construct excess body weight as a serious economic problem for developing countries.

8.7 The Global Burden of Disease Project

A contemporary research programme whose results were incorporated into obesity science by the authors of WHO reports was the Global Burden of Disease (GBD) project, begun in 1990 by the Harvard School of Public Health, WHO and the World Bank (Murray and Lopez 1996). It would have been politically expedient to use GBD results in a WHO technical report, since they came out of the organisation's

collaboration with two very prestigious organisations. Two important concepts deriving from GBD research were used by the authors of TR894. The first of these was the 'double burden of disease' that was forecast to occur in developing countries. This came from figures showing rates of non-communicable disease increasing in poorer countries:

> The next two decades will see dramatic changes in the health needs of the world's populations. In the developing regions where four-fifths of the planet's people live, noncommunicable diseases such as depression and heart disease are fast replacing the traditional enemies such as infectious diseases and malnutrition as the leading causes of disability and death. By the year 2020, noncommunicable diseases are expected to account for seven out of every ten deaths in the developing regions, compared with less than half today (Murray and Lopez 1996: 1)

According to these authors, increasing rates of NCDs now co-existed with continuing high rates of infectious disease to create levels of disease and mortality beyond the resources of poor countries' health care systems (see section 8.6). This idea of the double burden of disease was important to the authors of TR894 because it could be used in accounts of obesity as a global public health problem, not just a condition of rich industrialised countries. BMI distribution in adult populations was thought to vary according to the country's stage of development, and rising incomes led to a population shift where overweight replaced thinness. In the early stages of development, there would be an increase in the numbers of affluent people with high BMIs, whereas poor people would mostly still be undernourished: 'in countries in the early stages of [this] transition, overweight can co-exist with underweight, so that the burden of disease may be doubled' (WHO, 2000: 31). It was another important means of breaking the association between non-communicable diseases and affluence, and was also used in discussions of the causes of obesity (see section 8.3).

The second of these concepts was the DALY (disability-adjusted life years) measure. The DALY was devised as a measure to estimate the health burden resulting from a particular condition, by calculating the years lost due to increased mortality and the decrease in quality of life due to serious illness. Intended to be an 'internationally standardized' version of the QUALY, it was developed as part of the GBD project in order to 'capture the impact of both premature death and disability in a single

measure' (Murray and Lopez 1996: 6). Such a measure was useful for the authors of TR894, as it could be used to estimate more precisely the costs of the conditions associated with obesity that were not fully captured in mortality figures. As chronic diseases develop over many years and often have disabling symptoms, including the effects of morbidity as well as mortality allowed for a more complete estimate of costs to healthcare systems and society as a whole.

8.8 The environmental paradigm of obesity in TR894

The shifts in understandings of obesity and overweight that I have outlined in the previous four chapters are best considered as combining to form a new paradigm (Kuhn, 1970). However, this new paradigm did not replace older understandings of obesity, instead the approaches co-existed. The authors of TR894 still located their understanding of the causes of overweight and obesity within the framework of energy balance:

> In simple terms, obesity is a consequence of an energy imbalance – energy intake exceeds energy expenditure over a considerable period. Many complex and diverse factors can give rise to a positive energy balance, but it is the interaction between a number of these factors, rather than the influence of any single factor that is thought to be responsible. In contrast to the widely held perception among the public and parts of the scientific and medical community, it is clear that obesity is not just a result of overindulgence in highly palatable foods, or of a lack of physical activity (WHO, 2000: 101).

Multiple and incompletely understood physiological processes were seen to control the regulation of body weight, but these processes could be 'overwhelmed' by a positive energy balance arising from a combination of increased energy intake and decreased energy expenditure. In particular, the body was seen to have stronger defences against under-nutrition and weight loss than against over-nutrition and weight gain, and a small but consistently positive chronic energy balance was again seen as capable of producing large increases in body weight (WHO, 2000: 105). This was all rooted in the earlier understandings I have labelled the individual paradigm of obesity.

Between 1970 and 1995, understandings of obesity expanded and shifted focus, and by the publication of TR894 in 2000, this new approach could be considered a separate paradigm, that I have labelled the environmental paradigm. Although these two approaches co-existed, partly because they operate on different levels, I believe they are best described as different paradigms because the new approach used new sources of data, developed new classification schemes, provided new causal explanations and suggested new solutions. The new sources of data were results from epidemiological studies such as Framingham and the American Cancer Study that were incorporated into obesity science to produce a list of negative health consequences of overweight. New techniques in health economics had provided an increasing amount of statistical data on the costs of chronic diseases, and so the costs to healthcare systems had become an important element of the argument that obesity was a major public health problem. Bray and Garrow developed the new classification system of BMI cut-off points that were now used as the standard definition of obesity and overweight, despite ongoing debate about their relevance to different populations. New causal explanations focused on the environmental causes of obesity and overweight, stressing the increased availability of energy dense foods and decreased energy demands of daily living resulting from processes of modernisation and industrialisation. Individual treatment options remained the same, but the long-term failure of these measures, especially weight loss diets, was increasingly acknowledged. Combined with rising prevalences, this led to increasing formulation of preventive measures that were quite conservative despite the rhetoric of radical structural change.

In this way, researchers framing public health policy recommendations for the British government and the WHO transformed excess body weight from an individual condition caused by over-eating and treated by weight-loss diets into a global public health problem caused by changes in the food supply, patterns of work and physical environments, that required significant structural changes in order to reduce the potentially huge costs to healthcare systems.

8.9 Conclusion

By the beginning of the twenty first century, the causes of obesity and overweight were being framed in obesity science as environmental ones, resulting from processes of industrialisation and modernisation. The availability of energy-dense processed food, mechanisation in the workplace, sedentary leisure activities and motorised transport were the factors usually highlighted. Unlike the authors of TR854, the authors of TR894 did not directly refer to the 'thrifty gene' hypothesis. However, the importance they give to the distinction between modern and traditional lifestyles, combined with their focus on particular populations (see section 7.4) is consonant with such accounts (see chapter 9).

The authors of TR854 and TR894 also did not use the term 'obesogenic environment' (Swinburn et al., 1999) like the authors of the contemporary BNF report (see section 5.4), but such a concept is consonant with their analysis, and would subsequently be widely used in obesity science (WHO Regional Office for Europe, 2006, Grant et al., 2007, Foresight, 2007) and popular writing on the topic (Tsichlia and Johnstone, 2010). The more radical preventive measures discussed by the authors of TR894 – regulating advertising to children, nutritional standards for institutions, improved food labelling, encouraging physical activities and providing facilities for pedestrians and cyclists - were subsequently also widely discussed in the popular press. In the UK, they have also become part of a mainstream discussion of policy responses to the public health problem of obesity and overweight (see for example Foresight, 2007).

The increasing amount of data on the direct and indirect costs of conditions such as heart disease and diabetes (and to a more limited extent obesity) illustrates the growth in healthcare bureaucracies and the increasing range of statistical information that was collected and calculated by governments and biomedical researchers. Other authors have given historical accounts of these developments in the 18th and 19th century (Desrosieres, 1998, Hacking, 2006a) but the continuing expansion of the discipline of health economics demonstrates the further development of such techniques in the second half of the twentieth century.

CHAPTER 9: INDIVIDUAL AND SOCIO-POLITICAL EXPLANATIONS OF EXCESS BODY WEIGHT

9.1 Introduction

In the 1990s, the obesity science coalition expanded its sphere of influence markedly. Obesity and overweight became (temporarily) incorporated into central UK government health target setting in the early 1990s (DoH, 1992: 49). Concern about obesity as a public health problem had moved up the governmental hierarchy to be considered by the Department of Health as a whole (DoH, 1995) (see section 3.5), rather than by one of its expert committees, as in 1977. The coalition's activities then extended further onto the global stage as members contributed to two WHO technical reports in the late 1990s. The first, TR854 on anthropometry, included a chapter on overweight (WHO, 1995), but the second, TR894, was entirely on obesity, labelling it as a global epidemic (WHO, 2000). This was produced under the chairmanship of Philip James. As I have argued previously, TR894 was widely referred to in both the popular and the medical press, and an important resource for legitimating ideas of obesity as a global public health problem (Fletcher, 2007). The authority of the WHO made this report an easily cited piece of evidence for writers in the popular press. But this phenomenon was also found in the scientific and medical press. Referring to the existence of an 'obesity epidemic' and citing TR894 became an easy way of demonstrating the importance and relevance of many different kinds of research. It became a bandwagon in the mainstream sense, as well as in the narrower STS sense (Fujimura, 1988).

Despite its high profile, this report remains something of an oddity. Since its foundation the WHO had prioritised treating epidemic disease and building health capacity in poor countries (see section 9.4). Initially, this led to a focus on malaria, tuberculosis, and venereal diseases and on promoting prevention through work on maternal and child health, environmental sanitation, and nutrition (Staples, 2006: 138). In the 1990s, the organisation's declared priorities were fighting malaria, HIV/AIDS and tuberculosis and the regulation of tobacco marketing (Lee, 2009: 84-5, 94). The WHO had begun work on the global control of cardiovascular diseases in the 1970s (Litsios, 2008: 221) and since the 1960s, occasional technical reports had been

produced on coronary heart disease, and then also on chronic diseases (WHO, 1982, WHO, 1986, WHO, 1990). The WHO's European regional office had worked in the area of cardiovascular disease since the 1960s and developed a long-term programme of research into CHD and its risk factors[120] (Kaprio, 1991: 35-6). But these precursor activities were irregular and limited to one of the WHO's six regions: they do not fully explain why TR894 was produced at this time. From the mid 1990s the WHO had also been heavily criticised within the medical community as over-bureaucratic and inefficient, and its Director-General at that time, Hiroshi Nakajima, was unpopular and widely seen as ineffective (Anon, 1997, Godlee, 1994, Godlee, 1997). Moreover, as some rich donor countries had withheld or frozen their budget contributions, the organisation had been attempting to economise and improve its efficiency (MacGregor, 1994, MacGregor, 1996, MacGregor, 1999). A report into the global epidemic of obesity seems to be oddly disconnected from the rest of the WHO's work at the time and its declared priorities.

One of the factors that might have made the production of this report possible was its relevance to debates about future increases in health costs that would result from rising rates of chronic disease (see section 8.5). Questions of cost form a significant portion of TR894 (see section 8.4), and they are prioritised by appearing in one of the earlier chapters. Rising concern about rates of chronic disease combined with increasing forecasting of future demands on resources by healthcare systems increased debates about the rationing of healthcare from the 1970s onwards. Such debates had long been central to the discourse of preventive health (Lupton, 1995, Petersen and Lupton, 1996). In the UK in the 1980s and early 1990s, these took place in the context of widespread reductions in government health spending due to a combination of neo-liberal policies and economic recession (Berridge, 1999a: 97, Baggott, 2000:50-51). However, the British researchers who were most active in the public health coalition around obesity were mostly consultants in large teaching hospitals and researchers in universities or government funded research institutions. They were, thus, insulated from the worst of these funding reductions (Berridge, 1999a, Webster, 2002), and had the resources to develop the institutional networks required for such a coalition to function successfully.

[120] The North Karelia Project (see section 2.5) was part of this research programme.

9.2 Public health expertise and boundary-work in obesity science

As I have described above, the literature on obesity, and especially the WHO literature on the global epidemic, articulates shifts towards an environmental paradigm from the 1980s. The causes of obesity and overweight were increasingly framed as the undesirable effects of contemporary patterns of eating, working and travelling (see section 8.2). These effects were first thought to be occurring in industrialised countries, but later in poorer countries as well (see section 8.6). There were two aspects of these changes – changes in dietary composition and changes in physical activity levels. Rising average incomes had led to increasing consumption of protein, fat and sugar (see section 8.2). As part of a shift away from under-nutrition these changes do not seem inherently negative to health, but they were thought to be accompanied by a shift away from diets based on high levels of unrefined carbohydrates, and towards more energy dense diets containing much higher levels of processed food (see sections 5.3, 5.4 and 8.2). Declining levels of physical activity resulted from economic development: increasing mechanisation, industrialisation and urbanisation meant that employment became less physically demanding, sedentary leisure activities grew in popularity, and individuals relied more on motorised transport (see sections 5.4 and 8.2). Arguments about changing patterns of food consumption were based on FAO national food balance sheets – statistics of aggregate production and consumption (Drewnoski and Popkin, 1997). However claims about changing levels of physical activity levels were less well documented, and it has been argued that the existing evidence does not clearly demonstrate such a decrease (Gard and Wright, 2005: 119-24).

Obesity science researchers labelled visible signs of contemporary urban life - fast food, cars and television and computer games - as important causes of ill-health, often in the absence of concrete evidence. This was a new version of the 'diseases of civilisation' argument (Rosenberg, 1998) which appears to have expanded, filling the gaps in existing data (see section 5.4 and 8.2). Moreover, in TR894, there is a sense in which of 'traditional lifestyles', and even poverty, were being framed as healthier than affluence. By necessitating physically demanding work, a less processed diet and transport by foot or bicycle, a lack of resources meant that individuals avoided the chronic diseases associated with 'affluent' lifestyles (WHO, 2000: 120, 123-4, 126).

This is a crude over-simplification of these authors' arguments, but, by not explicitly acknowledging the health benefits of 'affluent' lifestyles, such accounts represented the health costs and benefits of changes deriving from industrialisation and modernisation in a very partial fashion.

These changes in the patterns of daily life were pervasive and highly interconnected. To reverse the effects in line with the analyses presented in these reports would have required changes on an enormous scale - for example, the restructuring of agriculture and the food processing and manufacturing industries. Moreover, the commercial interests involved - such as the motor and oil industries - form a significant portion of the global economy. These factors made it unlikely that such a programme would be carried out by contemporary governments, even those of rich industrialised countries. In particular, economic growth is often seen as a greater political priority than population health, even in affluent countries (Baggott, 2000: 56). Such (unexpressed) considerations led to an important gap in the rhetoric of obesity science which was characterised by an expansive description of the possible socio-economic causes of excess body weight, in contrast to a relatively conservative range of potential solutions.

Only in the area of prevention were some of the more wide ranging causes addressed and then their political implications were blunted by being largely framed in the health education language of individual choice. Although increased regulation of the food industry or provision of financial incentives for weight loss were sometimes suggested, most proposals involve advising, encouraging or educating consumers, medical professionals and the general public (see section 8.3). This gap is the product of a crucial form of boundary-work (see section 1.6.2) undertaken by members of the obesity science coalition who understood that it was beyond their authority to call directly for such wide ranging social change. Individual behaviour, especially in the area of diet and physical exercise, has long been seen as seen as coming under the authority of doctors (Shapin, 2003). A focus on solutions based on individual education and voluntary codes of practice was, therefore, a form of political self-limitation to ensure that these researchers would not damage their credibility by

overstepping the boundaries of their acknowledged expertise.[121] The professional interests of epidemiologists and others working in this area may have been to improve population health by expanding their sphere of influence, but even in the contest of modern preventive healthcare, there were political limits involved in the framing of policy.

9.3 The effects of global health inequalities: poverty as the missing factor

There are problems within the causal explanations of obesity and overweight provided by the environmental paradigm, and approaches from health inequalities research may be able to provide a fuller explanation of the ill-health of the exemplary populations of obesity science. The discourse of modernisation (see section 8.2) and its negative effects on the health of populations has lacunae, and there appears to be an unhelpful idealisation of the traditional taking place in this writing. Some critics of the dominant explanation of obesity have argued that poverty and social disadvantage offer an alternative and perhaps more satisfactory account of the obesity suffered by exemplary cases of obese populations including Pima Indians and Australian aborigines. The authors I discuss below do not extend this explanation to obesity in rich countries, but other researchers have as part of their wider critique of obesity science (Gard and Wright, 2005: 124-5).

Genetic differences were often discussed as possible explanation of the high rates of diabetes and obesity in such populations (see section 7.4). By describing the high rates of overweight and obesity in the Pima Indians or Australian aborigines as due to a genetic mismatch between their hunter-gatherer past and their modern present, such theories wipe out a history of displacement and poverty that have contributed equally to their community's poor health: 'diabetes has been figured as the process aboriginal people have paid for civilization rather than the penalty exacted by colonization' (Fee, 2006: 2994). In a discussion of high rates of chronic disease amongst Australian aboriginals, McDermott argued that

[121] Such boundary-work also partially explains the neglect of 'social' explanations in obesity science (see section 6.6).

> If the…"epidemiological" narrative of diabetes in Aborigines is expanded to include social history, early life experiences of malnutrition, poverty and illness followed by later experiences of welfarism, poverty, physical inactivity and obesity, explanations other than genetic ones might emerge (McDermott, 1998: 1193).

Modern genetic explanations are a narrowing of James McNeel's original concept of the 'thrifty genotype' which saw the ill-health resulting from a western lifestyle as a universal phenomenon (McDermott, 1998, Fee, 2006). This narrowing has led to a focus on research into the genetic determinants of chronic disease and a neglect of research into understanding the social determinants of ill-health (McDermott, 1998: 1194). By incorporating the work of Barker (2007) into the effects of maternal deprivation on the adult health of children, McDermott showed that an account of the health of these groups could be constructed that accorded with contemporary epidemiological consensus and acknowledged their history of colonisation, displacement and poverty (*ibid.*). At the risk of over-simplifying complex histories, many of the groups listed above, and routinely cited in the global health literature as examples of under-developed populations who nonetheless suffer very high levels of obesity, can be described as having experienced colonisation and, therefore, their collective histories will show some of the same features as those of Australian aborigines.

Instead of naturalising health inequalities through genetic explanations, other accounts describe the social inequalities that produce them. Such inequalities are a feature of many populations with high rates of obesity and overweight. Exemplary populations in obesity science included Pacific Islanders, the Pima Indians, Black women in the US and urban South Africa, and Australian aboriginals (see section 7.4). One feature that many of these exemplary populations shared was relative poverty. They shared experiences of displacement, high levels of economic dependency and financial insecurity. For example, the Pima have been displaced from their land and live on reservations in South Arizona, unable to farm the desert land they irrigated: decades of medical research have been conducted on them that appear to have brought very little benefit to their community (Gaesser, 2002: 172). Samoans and other Pacific Islanders are geographically and economically marginal: their economies are often dependent on a public sector funded by development aid and much of their food is

imported (McMurray and Smith, 2001, discussing the case of the Marshall Islands). For Samoans and Marshallese, this has resulted in diets based on highly processed nutritionally poor foods items such as tea, white rice, instant noodles and sugared drinks, and low grade fatty meats such as turkey tails and lamb flaps (Cheung, 2010: 2207). Black women are one of the poorest socioeconomic groups in America, and this is reflected in their health status.

The links between poverty and poor diet have been part of British health inequalities research since the Black Report (Townsend and Davidson, 1982, see also section 1.2.1). In the 1990s this topic began to be written about more frequently (Leather, 1996, see also Dowler et al., 2007). Nutritionists contributed to this discussion, including Philip James who co-authored an article for the BMJ on the topic, with Suzi Leather from the Food Poverty Network. They argued that

> The diet of the lower socioeconomic groups provides cheap energy from foods such as meat products, full cream milk, fats, sugars, preserves, potatoes, and cereals but has little intake of vegetables, fruit, and wholewheat bread. This type of diet is lower in essential nutrients such as calcium, iron, magnesium, folate, and vitamin C than that of the higher socioeconomic groups (James et al., 1997: 1545).

Such a diet of high fat and high sugar processed foods was precisely the kind of diet that was supposed to lead to increasing rates of obesity and overweight (see section 8.2). Given that these arguments were readily available to obesity science researchers, and even produced by one of the coalition's major participants, it is necessary to explain why they were not drawn upon to account for the causes of excess bodyweight. One important reason is that they were politically contentious and another that they did not fit with the ideas of individual responsibility embedded in chronic disease epidemiology (see section 2.5)

In an alternative framing, these differences in average body weights amongst populations were the result of global health inequalities. The highest rates were found in groups that were living in relative poverty in rich industrialised countries or in poorer regions that were dependent on rich countries. Members of such groups ate diets high in processed food because they were cheaper, readily available or high status because of being 'western'. They may have led more sedentary lifestyles due to

mechanisation and motorised transport, but individuals often worked long hours in bad conditions and did not have the time, space or cultural permission for leisure activities. These were still relatively poor populations who did not have the resources or the political clout to accrue the health benefits that other, more affluent groups or countries could derive from economic development.

9.4 The social and the biomedical: competing models of health and disease within the WHO

The contested boundary between the 'medical' and the 'political' that demanded political self-limitation on behalf on obesity science authors has been a pervasive feature of modern public health policymaking. In the context of the WHO it is illustrated by a tension between biomedical and social models of health that has existed from the founding of the organisation. Biomedical models explain the causes of ill-health in more individualised terms and seek technical solutions (Blaxter, 2010: 12-16). In contrast social models focus on the link between ill-health and social structures, and seek socio-political change to improve health (Blaxter, 2010: 16-19). These different models contain a different understanding of the boundary between the 'social' and the 'medical' and therefore differing assessments of the legitimate authority of the medical profession.

Like other UN bodies, including the World Bank and the FAO, the WHO was founded on the basis of a post-war optimism about possibilities of improving the lives of individuals and populations (Staples, 2006). The work of these organisations derived from a founding narrative of progress and economic development, a reaction to the devastation of the Second World War. In the case of the WHO, this optimism is illustrated by its famous definition of health as 'a state of complete, physical, mental and social well-being and not merely the absence of disease and infirmity' (Siddiqi, 1995: 226). Such an expansive definition derived from the ideals of a social model of health. The public health experts responsible for setting up the WHO saw it as a universal membership organisation that would act collaboratively to address a wide range of health needs. This was a vision of social medicine guided by ideas of humanitarianism and social equity (Lee, 2009: 16-7). However, Lee argues that, from

its founding, there was another narrower and more biomedical conception of the organisation's role (Lee, 2009: 46-7).

This competing vision, framed by the political context of the Cold War and functionalist political theories, was a more circumscribed understanding of its role in promoting international health co-operation. It involved the WHO focusing on the prevention of disease, especially epidemic infectious diseases, and the provision of technical advice to poorer countries. Two factors helped to promote this approach. The first of these was recent developments in medical knowledge and practice that seemed to hold the promise of significant improvements in health through more effective treatments, and, sometimes, the complete elimination of diseases (Lee, 2009: 46, Siddiqi, 1995: 195). The second was the collective professional identity of WHO representatives and staff, as members of a well-connected international medical community:

> When these internationally minded doctors came together within the World Health Organization, they combined the scientifically based authority of their profession with a commitment to apolitical internationalism in order to garner a global authority that facilitated the often unquestioning acceptance of their recommendations and ensured that countries clamoured for the organization's advice and assistance (Staples, 2006: 137).

Walt also describes how this deployment of professional expertise meant that in the 1950s and 1960s 'WHO was stable and pragmatic, largely disease oriented and dominated by medical professionals' (Walt, 1994: 137).

Despite the wider ideals embedded in its definition of health, the biomedical model was dominant in the early history of the WHO (see section 1.2.1). It enabled the organisation to operate across national boundaries and in varying political contexts: a narrower technical approach helped to avoid accusations of compromising the authority of national governments. An early focus on technical activities, such as the revision of international sanitary regulations, allowed the organisation to maintain a low profile in its early years, but 'support for tackling the social factors influencing health and disease was never far from the surface' (Lee, 2009: 18). However, for the next 20 years, the most important and prestigious of the WHO's activities was the Malaria Eradication Programme (Siddiqi, 1995). This was the epitome of a 'vertical'

programme that used the newly developed technology of DDT insecticidal sprays and a globally standard approach to eliminate one infectious disease endemic in poor countries.

By the 1980s the Malaria Eradication Programme was considered to have failed (Lee, 2009: 49) and criticisms of the social power of the medical profession and the biomedical model of disease had become more mainstream (Berridge, 1999a: 56-7). Such a climate enabled the WHO to broadened its activities and focus on the social as well as the individual determinants of health (Lee, 2009: 71-2). It switched from a vertical model of tackling individual diseases to a 'horizontal' model of primary healthcare that aimed to meet the healthcare needs of poor populations (Siddiqi, 1995: 196). Major initiatives of this period included Health For All, which came directly out of the primary healthcare movement, and attempts to strengthen global regulation of the marketing of baby milk, pharmaceuticals and tobacco (Lee, 2009: 71-98). This change resulted from a shift towards the priorities of developing countries (Siddiqi, 1995: 209), and the increasing influence of campaigning NGOs and other civil society organisations (Walt, 1994: 140, Lee, 2009: 87-95). It was so comprehensive that, writing in 1993, Siddiqi could argue that primary health care had become the 'dominant philosophy' within the WHO (Siddiqi, 1995: 196). Tackling the structural causes of ill-health involved political controversy as the WHO attempted to curb the activities, and profits, of the tobacco, pharmaceutical and food industries.

Taking on such powerful commercial interests led to criticism from the US and a long-term budgetary freeze by major donor countries, which has been described as part of a 'sustained effort to keep the WHO focused on biomedicine' (Lee, 2009: 98). This was combined with sustained accusations of politicisation (Siddiqi, 1995) and the relative unpopularity of later Director-Generals, compared to the charismatic Brock Chisholm and Halfdan Mahler (Lee, 2009: 15-16, 74-5). By the 1990s, the biomedical model was once again dominant, partly due to renewed focus on individual diseases such as TB and HIV/AIDS by rich donor countries (Lee, 2009: 84). These countries were increasingly funding disease-specific special projects, which were taking up an expanding proportion of the organisation's budget, giving them greater influence over the WHO's priority areas (Lee, 2009: 39, 85). In this way,

the rich countries managed to re-orient the organisation's agenda, and re-instate a narrower model of its activities (Lee, 2009: 98).

The WHO founding model of health, as including social well-being, was closely related to social medicine which was influential in Britain in the 1940s and 1950s (Porter, 1997a). Porter describes how social medicine in Britain in the 1940s aimed to become 'a medicine of society for society', and that 'its mission was to facilitate progressive human social and biological evolution' (Porter, 1997a: 99). Social medicine lost much of its influence in the UK after the 1960s (see section 1.2.1), and research from the social sciences began to be applied to medical topics as part of the developing sub-discipline of medical sociology, rather than in the main body of medicine (Porter, 1997b). The social model seems to have remained part of the mainstream within the WHO for longer (see above), and this may be because much of the organisation's work is in the area of public health. Because it studies population health, public health contains more of the 'social' than other medical specialities, both in terms of its understanding of the role of structural factors in health, and in its history of political engagement. However, the boundary between the 'biomedical' and the 'social' shifted regularly in post-war chronic disease epidemiology, as new forms of behaviour became to be seen as the concern of public health (see section 3.2 for one example). This continuing tension between wider socially engaged models of health and narrower more technical and biomedical ones was demonstrated in the 1990s by the gap between expansive understandings of the causes of excess body weight and much narrower recommendations for treatment and prevention.

9.5 Making an American and European health problem global?

Within this changing political orientation, concern with chronic diseases was inserted into the WHO agenda in the 1980s by developed countries, and projected onto developing countries as these diseases cease to be described as 'diseases of affluence'. By the technical report of 2000, obesity had become one of the 'chronic diseases' that was projected onto poor countries (in the absence of much evidence) at that time.

The WHO is a global health body which has historically carried out much of its work in poor, less developed countries. Because of lack of 'backstage' information (see

section 1.8.4), my arguments about WHO internal processes contain an element of speculation. However, in this context, a report on a global health problem presumably would have carried more weight inside the organisation than one that focused on a condition found mostly in rich countries and regions, especially if the condition was normally understood to be the result of over-consumption and individual lifestyle. Such considerations lead to the framing of obesity and overweight as a worldwide problem: the 'everyone, everywhere' approach (Gard and Wright, 2005: 17). This framing also fitted well with the development of new categories of 'at risk' individuals and populations that takes place within risk factor approaches to chronic disease (Petersen and Lupton, 1996: 18-22). However, to construct an argument for obesity and overweight as a global health problem required significant rhetorical work. Some of this had already been accomplished in other areas. The labelling of chronic diseases such as heart disease, stroke, diabetes and cancer shifted between the 1960s and the 1980s. In the 1960s and 1970s Morris described them as 'modern epidemics' (see section 2.3). In the 1980s they became a focus of attention as 'diseases of affluence' (Hardy and Tansey, 2006: 427-8). By the 1990s, WHO reports contained arguments for the name to change from diseases of affluence to 'chronic diseases'[122] (WHO, 1990: 10). This shift made it possible to argue that poor populations and countries could experience high rates of such conditions. Such framings were most fully articulated in ideas of the 'double burden of disease' developed by researchers from the Global Burden of Disease project (see section 8.6). As I outlined above, this work was heavily cited within TR894, as it provided an authoritative source that argued chronic disease was a global health problem.

According to the prevalence data cited by the authors of TR894, increasing rates of excess body weight were found in European and North and South America populations, and some other smaller groups such as Pacific Islanders, middle class Indians and urban Black South African women (see section 7.3). However, it did not seem to be an issue for the majority of the population of Africa and Asia, who account for approximately two thirds of the world's population. This may have been partly due to a lack of information: as the authors pointed out several times, good quality

[122] In this report such diseases were described as being caused by an 'affluent diet' (WHO, 1990: 10-14), so the idea of affluence was still being used, but gradually this usage fades as the components of such a diet become cheaper and its highly processed or industrial nature became seen as more important.

data was available only for Europe and North America (WHO, 2000: 16-30). The description of increasing rates of excess body weight as an urgent public health problem followed closely by calls for more research has been a consistent feature of obesity science. The certainty that obesity was an important public health problem had been juxtaposed with recurrent descriptions of lack of data since the 1960s.[123]

Modelling and forecasting were important in the arguments used by the authors of TR894 to frame obesity and overweight as a global health problem. In this report such techniques were used in two important ways. Firstly, in the absence of representative data, populations were modelled on the basis of what data did exist. In some cases this included generalising from obviously unrepresentative populations: Thai government officials represented their entire country; data from only six countries (including sub-populations such as urban black South Africans and Mauritians) represented the entire population of continental Africa (see section 7.3). These were obvious attempts to compensate for missing data. A more subtle form of modelling occurred when this data was presented as if increases in the past represent the only possible future pattern – it was taken for granted by these authors that average body weights not only have been rising, but would keep on rising, in all regions and populations. The possibility that increases in average body weight might begin to level off was not even considered.[124] Combined with estimates of the costs of obesity and overweight produced by health economics (see section 8.5), this extrapolation produced a vision of inexorably increasing average body weights and spiralling health care costs that was at odds with other contemporary health data for industrialised countries, showing rising life expectancies and decreasing mortality rates (Wilkinson, 1996: 29-30).

It can be argued that the data cited in TR 894 did not demonstrate that obesity and overweight was a global problem. Data did not exist for a large proportion of the world's population, and the limited data that did exist for much of African, China and other parts of South East Asia showed very low rates of excess body weight (see

[123] The acceptability of such framings may be linked to traditions within public health of raising concerns in situations of incomplete information and, therefore, high uncertainty.

[124] There is some evidence that average body weights among Canadian and American children are beginning to plateau (Gard, 2008).

section 7.3). Instead the data included in these reports demonstrated that obesity was a problem for European and American populations, and, especially for particular sub-populations. The sub-populations that were worst affected were those in industrialised economies who were marginal for reasons such as poverty, ethnicity or geography. Pacific islanders, Pima Indians, some aboriginal Australians, Black populations in the US and south-east Asian populations in the UK showed very high rates of excess body weight, often accompanied by high rates of chronic diseases such as diabetes (see section 9.5). Outside of Europe, North America and these specific populations, average body weights either remained low or they were increasing, but by relatively small amounts given the restrictiveness of the BMI-based definitions.

9.6 Conclusion

Obesity science's heavy reliance on ideas of modernisation in its explanations posed problems for how to explain obesity in under-developed populations, such as Australian aborigines, in a way that agreed with how it was explained in developed countries. Attributing it to poverty, both in developed countries and globally, would be politically sensitive, particularly in view of the corporate pressure on WHO to draw back from a political understanding of public health problems. In this respect, it was fortuitous that obesity had been placed on the WHO agenda not by public health experts but by biomedical researchers and clinical practitioners, who were disinclined to propose 'social' causes. Instead, they were able to adapt a model already developing in their own work. The dominant model in developed countries was still the individual model, though in view of the failure of medical interventions obesity science authors had started to acknowledge some 'environmental' causes. This account was expanded to include a wider social or environmental account of the causes of obesity globally, which was broadly acceptable within the WHO's remit, but which did not make reference to poverty.

Framing obesity and overweight as a global epidemic was a useful rhetorical tool for members of the obesity science coalition as it validated their work. The attribution of that epidemic to modernisation, and to the kinds of behaviour associated with modernity, was useful to governments and institutions like the WHO, as it had the effect of de-politicising the health consequences of poverty. It acted as a pressure

valve, allowing discussions that had developed from earlier concerns about high rates of heart disease to develop without disturbing the relationship between expert scientists and their government, or the WHO and its member governments. The use of modernisation as an explanation allowed for discussion of current population health issues and future forecasted problems to continue without directly referring to contentious political issues such as health inequalities, poverty or the rights of marginalised social group and ex-colonies. Although structural issues were raised in discourses about the causes and prevention of excess body weight in this period, they had little influence on the recommendations of such reports. Even when calls for reform were made, for example, in agricultural subsidies or the food processing industry, were made they were framed within a discourse of individual responsibility so that their political impact was largely blunted.

An alternative reading of material contained in TR894 would still see excess body weight as resulting from the processes of industrialisation and modernisation, but it would acknowledge that these effects vary between populations and groups within populations. It would acknowledge that economic development has brought health benefits, reflected in increased life expectancy, but that these benefits have been unequally distributed, with affluent populations and groups within populations continuing to benefit disproportionately from such development. Crucially, this more nuanced reading would accept that although body weight may be partially under individual control, it is also the result of structural factors such as changes in food production and retailing, in work patterns and in urban transport. Such changes have been encouraged by governments of many political persuasions since they are produced by, and, in turn, produce economic growth. In addition, a productionist model of agriculture (Lang and Heasman, 2004) using mechanisation and petroleum derived fertilizers and pesticides to increase productivity had been combined with agricultural subsidies in order to keep food prices low in Europe and North America. This subsidised and industrialised production of certain crops, combined with innovations in food manufacturing, has led to diets with high levels of fat sugar and salt. Decreasing average levels of physical activity can be linked to mechanisation and motorisation in workplaces and public transport. These are also processes that have been subsidised in many different ways by governments, from grants to specific industries to road building.

The environmental paradigm of obesity partially addresses such socio-political factors and potentially offers an enormous number of potential solutions such as reforming the entire food system, from agriculture through to processing and retailing, or redesigning cities and towns to encourage (or compel) individuals to walk and cycle rather than drive or take public transport. However, such solutions are definitely 'social' rather than 'medical', and political self-limitation meant that these authors reverted back to ideas of individual responsibility when proposing solutions to the problem of excess body weight.

CHAPTER 10 CONCLUSION

10.1 Introduction

In the preceding chapters I have described how medical concern about obesity developed out of the post-war growth and re-orientation of British and American public health towards chronic rather than infectious disease. I showed how research into the causes of heart disease led to the development of the large scale prospective epidemiological study and the risk factor approach to chronic diseases. I outlined the development of a public health coalition around obesity and of a policy-orientated body of expert knowledge on the causes and treatment of excess body weight that I have labelled obesity science. I summarised the initial model of obesity - the individual paradigm - that was developed by British and American researchers in the 1970s and early 1980s. Through an examination of the development of the body mass index and of the role of the energy balance model in obesity science, I showed how their model of obesity continued to develop in the 1980s and 1990s.

Next I shifted focus to describe the activities of an overlapping and international network of WHO expert scientist in the area of chronic disease and diet. I showed how members of the British obesity coalition became active in this arena, and how, due to the incorporation of new large scale epidemiological data and the move to a global forum, the model of obesity developed in this period. Lastly I analysed a WHO technical report on the topic of obesity that shows the development of an entirely new model of obesity – the environmental paradigm. I argue that there are problems with this model because it is not clear that obesity is a global health problem, and because processes of political self-limitation meant that the causes of increasing rate of obesity were attributed to the effects of industrialisation and modernity, rather than more politically contentious explanations such as poverty.

10.2 Policy activity on obesity 2000 to the present

The obesity band wagon not only continued rolling after 2000, but also gained greater momentum. The global epidemic became a high profile issue that was widely written about in the popular and scientific press. This in turn appeared to generate more

policy activity as governments and international organisations needed to be seen to be addressing such a pressing problem. In Britain, the issue was part of high level political discussion. The National Audit Office produced a report on the topic in 2001, and in 2002 organised the Joining Forces to Tackle Obesity conference. The House of Commons Health Committee held an inquiry into obesity in 2003/4 and produced a substantial report with 69 recommendations that, despite its 'optimistic' framing, stressed the role of social norms in the development of obesity and seemed sceptical about current health promotion-based interventions (Health Committee, 2004). Obesity was once again included in the government health targets. 'Reducing obesity and improving diet and nutrition' was one of the 'overarching priorities' of the Choosing Health initiative (DoH, 2004: 4), as was increasing exercise. The DoH also promised 'a new cross-government campaign to raise awareness of the health risks of obesity and the steps people can take through diet and exercise to prevent obesity' (DoH, 2004: 5) as well as a 'definitive' NICE guideline on the prevention, identification, management and treatment of obesity (DoH, 2004: 15). Obesity was also discussed in the 2007 King's Fund report into NHS funding (Wanless et al., 2007) and used as a case study in the Nuffield Council on Bioethics report on ethical issues in public health (Nuffield Council, 2007).This policy attention culminated in the launching of the government Office for Science's Foresight project Tackling Obesities: Future Choices. This report argues that 'By 2050 Britain could be a mostly obese society' (Foresight, 2007: 6) and further develops the environmental paradigm to provide an account of the key 'drivers' of obesity and an 'obesity system map' of impressive complexity, illustrating the multiple and interacting causes that have led to the development of 'passive obesity'.

In the international arena, a joint FAO/WHO technical report on diet, nutrition and the prevention of chronic disease was published in 2003 (WHO, 2003). Prominent members of the international obesity coalition sat on the committee, including Philip James125 and Boyd Swinburn,126 while temporary advisors included Anna Ferro-Luzzi and Peka Puska. This report continued to stress the increasing burden of chronic disease affecting developing countries, resulting from industrialisation, modernisation and globalisation. Obesity was also one of the conditions it considered in detail along

[125] He was listed as chairman of the International Obesity Taskforce (IOTF).
[126] Swinburn and a colleague coined the term 'obesogenic environment' (see section 6.4).

with diabetes, cancer and cardiovascular disease. In 2004, the WHO also launched a global strategy on diet, physical activity and health at the World Health Assembly (WHO, 2004). The role of unhealthy diets as one of two major causes of increasing rates of non-communicable disease has been translated into a policy concern with obesity in the reports produced by this programme. It remains active: a global ministerial conference on healthy lifestyles and non-communicable disease control will take place in Moscow in April 2011. There were major initiatives taking place at the European level as well. The European Union launched its own Platform on Diet, Physical Activity and Health in 2005 in co-operation with the WHO and the IOTF (EC, 2005), and a year later, in Istanbul the WHO Europe office held the European Ministerial Conference on Counteracting Obesity to address 'the growing challenge posed by the epidemic of obesity to health, economies and development' (WHO, 2006, see also Branca et al., 2007a). The policy momentum has died down since this period of frantic activity, but the issue still resurfaces regularly in the popular press, when relevant research results or public health data are released.

10.3 The creation of policy knowledge

In this section, I want to examine what my case study can say about the creation of public health policy knowledge. In particular I want to address three interrelated and overlapping questions. Firstly what does the development of obesity science tell us about the relationship between policy and epidemiology? Secondly what does it say about the role of public health coalitions? Thirdly, what does it add to sociological accounts of the new public health?

The relationship between epidemiology and policy is complex partly due to the unusual character of epidemiological science. It is a large and relatively heterogeneous body of knowledge – as well as biomedicine it incorporates elements of non-human biology, geography, health economics, psychology and sociology. Understanding patterns of health in human populations also involves analysing complex and ill-defined systems.[127] These factors may explain why, since the

[127] I think there are several parallels between epidemiology and environmental science in this respect. This may be one reason why, in the last few years, comparisons have been made in the press and policy literature between the obesity epidemic and climate change (Foresight, 2007).

beginning of the twentieth century, there have been recurring internal doubts about epidemiology's status as a science (Amsterdamska, 2005). Since the nineteenth century, British epidemiology has involved both policy-oriented and policy-engaged research, to use contemporary terminology. But individuals acting as expert scientists, on behalf of governments or of international organisations such as the WHO, have to be careful not to exceed their professional authority when making policy recommendations. Epidemiology contains such a broad range of accounts of the causes of health problems that multiple forms of professional expertise can legitimately be claimed in areas such as the relationship between nutrition and chronic disease. In addition, such accounts could readily be mobilised to support proposals for solutions to health problems, ranging from providing behavioural advice to individuals at the micro-level to recommendations for wholesale social change at the macro-level.

I have outlined above the effects of a political self-limitation whereby the recommendations of expert scientists in the area of diet and health are largely restricted to initiatives aimed at improving individual's food choice or level of physical activity (see section 9.2). I have also described how the causes of excess body weight in particular populations are seen to derive from the effects of modernity, rather than inequality or poverty (see section 9.3). In these kinds of ways, expert scientists in this area limit what they say according to *what is politically acceptable or expedient*. This self-limiting has important implications not just for overt policy recommendations, but also for the content of obesity science more generally – including what kinds of approaches, findings and arguments are incorporated into that literature and presented as authoritative. Insofar as epidemiology is a policy science, then, it is powerfully informed by the expectations and expediencies of policy, as much as by the original scientific research on which it draws.

My research demonstrates that public health coalitions are a normal and productive element of biomedical research. Lobbying for funding for research or treatment programmes is a normal professional activity, and so is attempting to influence the formation of public health policy and raise public awareness of particular health conditions. Coalitions form as a normal aspect of the academic research process when individuals and groups perceive their work as having a common concern with

important health issues. As well as a means of sharing information, these coalitions also develop out of the sense of responsibility that often propels individuals to work in public health and related research areas. The work of such coalitions is motivated by a desire to improve society, and this accounts for some of the sense of campaigning that their activities generate.

However, in the UK, the formation of such policy collations was influenced by the NHS re-organisation in 1974 that severed the links between public health and local authorities (see section 1.2.1). Throughout the period of my research, the resulting institutional weakness of public health meant that clinical medicine researchers and practitioners could claim expertise in areas that previously would have been the domain of public health professionals. British health policy has typically been developed by a relatively closed community of invited experts and, given the relative prestige of clinical medicine, this facilitated such a takeover. Public health coalitions appear to have developed around many of the major risk factors for heart disease, including smoking, hypertension and physical activity. The coalition that developed around excess body weight has been very successful but I think in one sense it can be described as opportunistic: from the late 1960s, nutrition researchers, whose research into malnutrition had suddenly become much less fashionable were looking for interesting new research opportunities and formed alliances with research working on heart disease and diabetes so that they could participate in the new area of chronic disease epidemiology. British public health science went through a significant reorientation when, following the undermining of the old public health institutions, clinicians effectively got together to colonise the public health policy space. And this had important implications for the way that public health policy science conceived of health problems, including obesity and chronic disease.

Finally, my analysis of changes in the epidemiological and public health understanding of obesity provides an empirically rich account of the emergence of one expression of 'new public health'. This supplements the work of authors such as Petersen and Lupton who analyse and criticise the growth of individualism in contemporary public health and its focus on the individual management of lifestyle to prevent chronic diseases. Whilst I agree with many of their criticisms of contemporary public health – particularly the stress on individual rather than

structural causes of ill-health, and the responsibility that is put on individuals to maintain their own health – the lack of empirical detail in their account explaining how the new public health came to replace older forms of public health is problematic. The historical and professional contexts of the development of this knowledge is omitted which gives their account a slightly detached and faintly conspiratorial character. This is compounded by the fact that individual and group motivation to adopt these new ideas and practices remains unaddressed throughout. My research, however, provides a historical and institutional context for such changes. It shows how such changes are the result of the shifting disciplinary constitution of public health following the dismantling of environmentally-oriented institutions of public health in the UK, the colonisation of public health by clinicians and biomedical research scientists, and the exportation of that model into a WHO that was under pressure from wealthy donor countries to identify public health problems and solutions relevant to those countries.

10.4 Boundary-work and policy science in public health nutrition

Thomas Gieryn's (1983, 1995, 1999) concept of boundary work argues that researchers rhetorically define and re-define the boundaries of science in order to present their knowledge claims as credible, reliable and trustworthy (see section 1.6). These boundaries are context-specific, he argues, and thus highly contingent and mutable, but they are constructed in order to maintain the epistemic authority of both the individual making the claim, and their discipline, by demonstrating the knowledge as well-founded and relevant to the specific context. In the preceding account of the development of obesity science, I have outlined the ways in which researchers define and redefine boundaries between public health nutrition and other kinds of knowledge in order to ensure that the claims that they make could be seen as legitimate and authoritative. In chapter 6, I described how a focus on individual behaviour led to the exclusion of social and structural factors as explanations of eating behaviour. This means that the explanations of obesity science drew on a relatively narrow understanding of health (similar to the biomedical model discussed in section 9.4), where the boundaries between the social and the biomedical were considered to be quite firm. This narrower model was a central feature of the risk factor analysis that

came out of the Framingham study (see section 2.5) and, therefore, was a readily available and prestigious approach for these researchers to use.

In chapter 8, I outlined how the failure of earlier models to fully explain increasing rates of excess bodyweight, led to the incorporation of wider environmental causes into a new model of obesity and overweight. Factors such as the widespread availability of highly processed foods or decreasing requirements for energy expenditure came to be seen as key causes of increasing average bodyweights. This model involved a much wider understanding of the causes of health and disease more akin to the social model of health (see section 9.4), and would seem to imply that wide-ranging, large-scale changes and economically damaging changes would be required to halt or reverse this trend. However, as I argued in section 9.2, the authors of these reports did not call for such changes. Instead they recommended more politically palatable changes relying on individual choice or voluntary codes of practice, such as improved food labelling, increased regulation of advertising for products targeted at children, nutritional standards and guidelines for institutional catering, increased education in nutrition and in food preparation, and promotion of exercise and physical activity.[128] A narrower focus on individual causes allowed obesity scientists to frame recommendations that largely relied on accepted health promotion techniques in order to persuade individuals to change their behaviour, or opportunistically incorporated facilities developed for other purposes such as road safety or physical education in schools. This gap between an expansive set of causes and a narrow and conservative set of solutions was a key example of the boundary-work undertaken by obesity science researchers. It meant that their recommendations largely focused on the area of personal behaviour, traditionally an area in which medical expertise was considered authoritative, rather than exceeding the accepted boundaries of their authority by advocating large-scale social and economic change.

Having illustrated that boundary-work has played a key role in the development of obesity science, in this concluding discussion I will reflect upon the significance of this phenomenon in greater depth. In doing so, I will also describe how this thesis has

[128] More intrusive measures such as the creation of urban pedestrian walkways and cycle paths or subsidies for fruit and vegetable growers were also listed but these were a minority of the measures and were still relatively conservative compared to the potentially wide-ranging interventions suggested by their analysis of the causes of excess body weight.(WHO, 2000: 269).

developed Gieryn's concept of boundary-work and why this is a necessary and important move in the analysis of obesity science.

The material that I outlined in section 1.3 demonstrates that such processes have been present throughout the history of nutrition science. Since the late nineteenth, when they contributed to debates about the link between poverty and poor nutrition or minimum wage levels, nutrition researchers have had to work in an area that is characterised by shifting boundaries between the political and the technical. This has meant that their recommendations have often been controversial, and that individual expert scientists have had to engage in ongoing sophisticated boundary-work to maintain the distinction between nutritional and political judgements, and maintain their credibility by demonstrating their scientific credentials or lack of commercial funding. For example, the controversy about the protein levels necessary for health accompanied the beginnings of nutrition science in the late nineteenth century and recurred until the 1950s. It was linked to arguments about minimum wage levels, since animal protein was an expensive and desirable component of poor peoples' diet, but also ideas about appropriate eating. In this period, several generations of nutrition researchers tried, and often failed, to persuade the public to accept novel pulse-based proteins as inexpensive substitutes for meat. This socially conservative form of activism saw the role of nutrition as advising individuals on 'intelligent' food choices rather than linking improved nutrition to increased wage levels: nutrition science could be used to advise individuals on how to choose correctly in order to obtain the maximum nutrients for the minimum cost, but, according to these accounts, it could not be used to argue that wage levels were insufficient to obtain adequate levels of nutrients. The latter was deemed to be a matter of political economy – to be decided by employer and legislators - and so outwith the legitimate authority of nutrition researchers.

The diet-heart disease hypothesis of Ancel Keys and others (see section 2.5) led to a protracted controversy around the health effects of consuming foods such as butter, eggs, milk and meat. This controversy (mostly) abated despite the absence of definitive scientific proof, and in spite of the lobbying of influential food producing groups, such as the dairy and egg industries (Garrety, 1997). Other researchers in the field as well as representatives of particular food producer groups persistently

questioned the scientific evidence linking high rates of cholesterol consumption to increased risk of heart disease and labelled low-fat diets as 'food faddism', that is non-scientific and based on irrational fears (Garrety, 1997: 751). As Garrety points out, because of the wide dissemination and practical relevance of nutrition science 'the boundary between "food fads" and orthodox nutrition is particularly difficult to maintain. Considerable energy has been – and is – devoted to keeping fad diets under control' (Garrety, 1997: 750-1). Because of their reliance on weight loss diets as a treatment, obesity science researchers when working in an official capacity also have to be careful to maintain the boundary between orthodox nutrition and fad diets.[129] They have needed to give details of the biochemical research findings underlying their dietary recommendations and outline the clinical results demonstrating their effectiveness in order to differentiate them from wide a range of alternative recommendations. These can be based on particular kinds of food (raw foods, whole foods or commercial weight loss products), recommend abstention from specific foods (vegetarian, vegan or fruitarian) or advocate a particular balance of foodstuff (high-protein or macrobiotic). As many of these unorthodox diets also use nutritional science to claim legitimacy, researchers have often had to spend time rebutting the claims made on behalf of these 'fad' diets as well making very detailed evidence for the legitimacy of their own recommendations.

Thus obesity science researchers produced their policy advice in the knowledge of previous controversies about expert dietary advice and, in particular, of the role played by commercially powerful interests within the food and agriculture sectors in inciting and sustaining such disputes. Such commercial interests often promoted a narrower model of health (in this case body size or physical fitness) that focused on the role of individual responsibility rather than wider factors such as food availability or advertising. Individual researchers needed to be able to successfully engage in various kinds of boundary work in order to maintain their authority and avoid their advice being discredited by being described unscientific. Examples of such boundary-work included drawing on particular kinds of research results – often biochemical and physiological - in order to substantiate their arguments; emphasising their credentials

[129] However several of my participants did manage to translate their nutritional expertise into best-selling diet books, including Alan Howard and Ian MacLean-Baird who developed the Cambridge Diet (now the Cambridge Weight Plan).

as members of state-funded research centres and as governments scientists recommending relatively uncontroversial dietary treatments and policy initiatives (see above); and largely distancing themselves from campaigning groups or commercial interests that might be seen as 'contaminating' their findings.

By making use of Gieryn's concept of boundary-work in my research in this fashion, I have extended his argument into a new empirical area. When describing boundary-work Gieryn (1983, 1999) used examples from the history of the physical and biological sciences, whereas my empirical material concerns the scientific knowledge created in the making of public health policy.

In conclusion, I want to return to Sheila Jasanoff's ideas about the distinctive nature of policy science. As I outlined above (see section 1.6), Jasanoff (1990: 227) describes policy science as a hybrid entity composed of many elements assembled in response to the demands of answering key policy questions, but also as an important influence on subsequent policy**.** I argue that one of the important characteristics of policy science is that it necessarily requires more boundary-work than other forms of science. There are two main reasons for this. Firstly, policy science does not appear to be like other kinds of science or like the accepted model of scientific knowledge creation: it does not take place in a clinic or a laboratory, is based on the results of several different kinds of research and makes broad claims about the nature of contemporary society**.** This means that extra work has to be done to make policy science appear more scientific. Such activities may include stressing the expert credentials of participants, reference to previous reports in the same area, an invocation of moral languages or reference to rules of thumb such as, in environmental contexts, the precautionary principle or guidelines for sustainability.

Secondly policy science often relates to contested scientific/technical arenas which are also inhabited by other powerful interest groups who contest the scientific legitimacy of specific claims and the individuals experts making them: examples of this in public health nutrition case include food producers contesting specific nutrition claims and producing counter-evidence about nutritional properties of particular foods**,** such as meat or diary products, or of particular patterns of consumption, such as low fat or whole-food diets. Nutrition researchers have often had the problem of

producing authoritative advice whilst negotiating the interests of powerful food producers. However, this need for increased levels of boundary-work – including providing evidence of elite scientific status, conventional political activity and acceptable interactions with commercial interests - is not unique to public health nutrition, as in many areas of environmental policy science claims made by researchers are countered using similar methods.

I have argued that the nature of policy science means that it will require more boundary-work (than other kinds of science) and some authors (Weinberg, 1972, Jasanoff, 1990, Wynne, 1996) state that such boundary-work will often be based on extra-scientific moral arguments, which can, in principle always be challenged by alternative interpretations, which in turn leads to further boundary-work. Examples of such extra-scientific moral arguments, or meta-scientific judgements, include the precautionary principle in environmental policy and ideas about the un-healthiness of certain body sizes in obesity science. These kinds of arguments are a routine feature of science, but are used mostly frequently in contentious situations when evidence is limited as they function to 'fill in the gaps' of particular arguments. But because disputes involving such judgements cannot be settled by recourse to more scientific knowledge – they cannot be settled at the level at which they are being conducted - they are prone to recur regularly, as examples outlined in section 1.3 from the history of nutrition clearly demonstrate.

I have demonstrated how boundary-work was pervasive in the attempt to provide governments with credible public health recommendations in the area of diet and nutrition and argued that this is because it is inevitably part of the creation of policy science. However, my case study demonstrates that the increased levels of boundary-work performed in this area of policy science have led to problems in the ways in which research scientists participated in the making of recent public health policy on diet and nutrition. Pressure to maintain expert authority in this highly contestable area of science has led to attempts to erect firm boundaries around what counts as expert knowledge in a way that has deleted important social factors. Following Wynne (1993), I argue that this situation could be mitigated if obesity scientists were given the political space in which to reflect on this problem i.e. the rhetorical strategies of boundary-work that they have to use in order to maintain their authority as experts.

Such reflection might allow them to realise and acknowledge the ways in which their own institutional processes lead to the deletion of important kinds of explanations and sources of evidence, which in turn might make it possible to redefine the boundaries of what counts as expert or authoritative public health policy knowledge and extend the parameters of discussion to consider structural factors more fully. This might involve incorporating existing national and international research into health inequalities into their explanations of increasing average bodyweight. It could also involve contacting patients and other advocacy groups in order to gain alternative perspectives into the causes of excess body weight and the effect of current treatments. Finally, it might involve examining the link between overweight and ill-health and analysing how robust these links, and at what point the increased risks of conditions such as diabetes and hypertension are more significant than the risks of current treatments (see section 7.6)..

These pressures have been exacerbated by the tradition of basing participation in policymaking on professional achievement and history of public service that has led to a socially unrepresentative group of elite scientists developing British public health policy in this area. Due to the post-war weakness of British public health (Lewis, 1986, Berridge, 1999a, Berridge, 2007a) these elite scientists have also largely been inclined towards a narrow model of health that focused on individual behaviour as a cause of excess bodyweight (see sections 6.6 and 9.2). Moreover, the UK approach of private deliberation leading to the publication of consensus reports (see section 1.3) – in contrast to the more open American system (Jasanoff, 2005: 262) - has also accentuated the lack of wider public participation in the development of this body of knowledge.

This pattern of private deliberation amongst elite researchers leading to the production of consensus reports appears also to have been a feature of the production of WHO technical reports. Despite the availability within the organisation of alternative, more social, models of health (see section 9.4), this mode of knowledge production, combined with the active participation of British and American nutrition researchers and external political pressures on the WHO, led to partial accounts of the causes of excess body weight that omitted important potential causes such as poverty and social inequality. As in the examples I discussed above, in this case, the legitimate use of

scientific evidence from nutrition research results (and other biomedical research results) is restricted to explanations of increasing average body weights largely framed terms of individual behaviour. Explanations framed in terms of wider social and economic change risk being described as 'political' and therefore not scientifically legitimate. The authority of such partial explanations has been reinforced by their congruence with culturally pervasive and highly moralised 'common sense' accounts of modernity, health and bodyweight (Gard and Wright, 2005). These accounts provide explanations of excess body weight that place undue emphasis on factors such as irrational food choice or inappropriate leisure activities, as opposed to more structural factors such as restricted food choice, due to low income, or changes in work practices resulting from widespread mechanisation.

In the case of medical knowledge, the social processes of creating new policy science have also been different from those in other areas of science, partly due to the historical durability of medical authority in areas of health and individual conduct. As accredited and government appointed medical experts, research scientists who are members of consultations and expert committees have multiple and overlapping forms of expertise upon which they can draw - including those of a medical practitioner, a research scientist, a government scientists and as a member of the 'great and the good' (Jasanoff, 1990: 229, Jasanoff, 2005: 268). This can be described as an 'excess' of authority upon which to draw, and by virtue of their position they are given the authority to make very wide ranging judgements about the proper conduct of individuals and the organisation of society that go well beyond their specific research expertise (Jasanoff, 1990: 229, 237, see also Wynne, 1996). This is not an abuse of their role, since the form of embodied expertise practiced in the British political system is based on the exercise of such judgement (see section 1.6.2). Recruitment of the 'great and the good' on the basis of professional and personal standing legitimates the fact that these individuals and bodies are often asked to make political judgements that go beyond the available science.[130]

[130] In my account I have emphasised the difference between policy science and other kinds of science (see section 15). However, policy science has remained an analysts' rather than an actors' category. It is therefore an empirical question requiring further research to establish how expert scientists understand their participation in such committees, other work involved in the development of public health policy and the knowledge created during such processes. In particular it would be useful to establish whether there is an actors' category that maps onto the analyst's one of 'policy science'?

As I have explained earlier (see section (see sections 6.4 and 9.5), there are also important gaps in contemporary biomedical knowledge about the causes and long-term health effects of excess bodyweight in both individuals and populations. In this way, obesity science fits Funkowitz and Ravetz's (1993) definition of post-normal science. However, their call for an 'extended peer community' to review such science seems to be warranted not by the uncertainties of these bodies of knowledge, but by the social justice issues deriving from the limited democratic participation in the making of public health policy in the British political system. Precisely because many of these judgements rely on extra-scientific and often moral arguments, they should be assessed by a more socially inclusive body of participants to avoid the stigmatisation of particular individuals and social groups.

In these respects the construction of current ideas about the obesity epidemic can itself be seen as the outcome of the political process – driven by the work of well-intentioned research scientists and medical practitioners concerned to improve the health of the population, but unwittingly and implicitly shaped by political forces that have led to the writing-out of overtly politically sensitive solutions from public health policy.

10.5 Conclusion

Dominant accounts of present-day public health are framed in a broad historical narrative that emphasises the importance of a number of 'transitions', all of which serve to underline the idea that we live in a time of both peculiarly good health and of specific forms of illness that can be attributed to 'modernity', itself understood rather vaguely as a condition of economic and material plenty that is quite distinct from the constitution of earlier 'traditional' societies. Obesity science has added to this narrative the further concept of a 'nutritional transition', which brings both an increase in the amount of food available, but also a shift from healthy traditional diets and patterns of activity to more harmful lifestyles and forms of consumption.

The aim of my thesis has been to show how the story of this transition was constructed through the work of a specific coalition of scientists, working within a particular social and economic context of policy science. And in so doing, I have sought to open up the possibility that other, more political accounts of obesity and chronic disease might be seen as equally or more valid and appropriate to address the health problems that confront the modern world.

APPENDIX 1

Principal primary sources

DATE	TITLE	AUTHOR
1969	Obesity: Medical and Scientific Aspects: Proceeding of the First Symposiums of the Obesity Association	Edited by I.M. Baird and A Howard
1969	Obesity and Disease	Office of Health Economics
1969	Obesity and its Management	Denis Craddock
1977	Research on Obesity	Department of Health and Social Security/Medical Research Council
1978	Obesity and its Management (3rd edition)	Denis Craddock
1978	Energy Balance and Man	John Garrow
1982	The Prevention of Coronary Heart Disease	WHO (technical report 678)
1984	Obesity (Clinics in Endocrinology and Metabolism volume 13 no 3)	W P T James
1985	Body Weight Control	Edited by AE Bender and LJ Brookes
1986	Community prevention and control of cardiovascular diseases	WHO (technical report 732)
1988	Obesity and Related Disease	John S Garrow
1988	Healthy Nutrition: Preventing nutrition-related diseases in Europe	WPT James with A Ferro-Luzzi, B Isaksson and WB Szostak (WHO Regional Publications, European Series no 24)
1990	Diet, nutrition and the prevention of chronic diseases	WHO (technical report 797)
1994	Obesity	Richard West for the OHE
1995	Obesity: Reversing the Increasing Problem of Obesity in England	Department of Health
1995	Physical Status: The Use and Interpretation of Anthropometry	WHO (technical report 854)
1996	Obesity in Scotland: Integrating Prevention with Weight Management	Scottish Intercollegiate Guidelines Network (SIGN)
1999	Obesity	British Nutrition Foundation
2000	Obesity: Preventing and Managing the Global Epidemic	WHO (technical report 894)

APPENDIX 2

DATE	Title and author/ organisation	Definition (e.g MLC tables or BMI)	Prevalence (low/stable or high/increasing)	Health consequences (short/long list)	Causes (individual or environment)	Treatment or prevention	Health-care costs (high/ low)
1969	*Obesity: Medical and Scientific Aspects: Proceeding of the First Symposiums of the Obesity Association* (eds I.M. Baird & A. Howard)	Ponderal index + MLC weight tables /relative weight	Not known	Diabetes, coronary artery disease, hypertension, osteoarthritis	Individual	Treatment	Not discussed
1969	*Obesity and Disease* (Office of Health Economics)	Relative /ideal weight	20% overweight, 5% obese	Diabetes, possibly CHD, osteoarthritis	Individual	Treatment	Low costs largely to individuals
1969	*Obesity and its Management* (Denis Craddock)	Relative /ideal weight	50% overweight	Coronary artery disease, hypertension, cardio-respiratory disorders, diabetes	Individual (with brief discussion of social factors)	Treatment	Not discussed
1977	*Research on Obesity* (Department of Health and Social Security/Medical Research Council)	MLC weight tables/ relative weight plus W/H^2	Small increase in prevalence since 1943 surveys	Diabetes, possibly CHD, hypertension, osteoarthritis	Individual (but also related to social class)	Discusses prevention but focus is on treatment	Not discussed
1978	*Obesity and its Management* (3rd edition) (Denis Craddock)	MLC weight tables/ relative weight	50% of men and women overweight	CVD, respiratory insufficiency, diabetes, gallstones, liver damage, osteoarthritis	Individual (with brief discussion of social and environmental factors)	Treatment	Not discussed
1978	*Energy Balance and Man* (John Garrow)	MLC weight tables/ relative weight	Not discussed	Not discussed	Individual	Discusses prevention but focus is on treatment	Not discussed
1983	*Obesity* (Royal College of Physicians)	MLC weight tables/ relative weight	31% of men and 27% of women overweight 5.4% of men and 6.4 % of women obese	CHD, hypertension, diabetes, gallbladder disease, cancer, breathing difficulties	Individual (with brief discussion of social factors)	Treatment	Not discussed
1984	*Obesity* (Clinics in Endocrinology	BMI + Garrow's cut-off	Not discussed	CHD, diabetes, joint disorders, hypertension,	Individual	Treatment	Not discussed

	and Metabolism vol 13 no.3) (W Philip James)	points		Respiratory disorders			
1985	*Body Weight Control* (eds AE Bender and LJ Brookes)	MLC weight tables /relative weight plus ref to BMI	24.2 % of men and 27.1% of women overweight (from NHANES II)	CVD, diabetes, hyper-tension, gout, hyperinsulinaemia, raised blood cholesterol, locomotor and psychiatric disease	Individual (also related to ethnicity)	Treatment	Not discussed
1988	*Obesity and Related Disease* (John Garrow)	BMI + Garrow's cut-off points	Men: 34 % overweight, 6% obese Women: 24% overweight, 8% obese	Heart disease, diabetes, hypertension, gall- bladder disease, respiratory insufficiency, osteoarthritis	Individual (brief discussion of social factors)	Treatment (plus brief discussion of prevention)	Not discussed
1994	*Obesity* (Richard West for the Office of Health Economics)	BMI + Garrow's cut-off points	Men: overweight 40%, obese, 13% Women: overweight, 29%, obese, 15%	CHD, stroke, cancer, diabetes, hypertension, hypercholesterolaemia, gall bladder disease, osteo-arthritis, gout, ovulatory failure, menstrual irregularities and PCOS**	Individual (brief discussion of socio-economic factors)	Treatment (plus brief discussion of prevention)	First details of costs to healthcare services: £29 million in UK, $39 billion in US
1995	*Obesity: Reversing the Increasing Problem of Obesity in England* (Department of Health)	BMI + Garrow's cut-off points	UK Men: obese, 13% UK Women: obese, 15% US Men: obese 31% US Women: obese: 35%	CVD, certain cancers, gall bladder disease, hypertension, and a range of bone skin and joint disorders	Individual (but discusses relation to social class and ethnicity)	Gives equal weight to treatment and prevention	Not discussed
1996	*Obesity in Scotland: Integrating Prevention with Weight Management* (Scottish Intercollegiate Guidelines Network)	BMI + Garrow's cut-off points	Scottish men: obese, 14%, overweight, 44% Scottish women: obese, 17% and overweight, 32%	Diabetes, hypertension, stroke, hyperlidiaemia, CHD, gallstones, cancers, breathlessness, respiratory disease and sleep apnoea, menstrual abnormalities, pregnancy complications, musculo-skeletal disorders and arthritis, stress	Individual	Gives roughly equal weight to treatment and prevention	Not discussed

				incontinence			
1999	*Obesity* (British Nutrition Foundation)	BMI + Garrow's cut-off points	Between 1980 and 1996 prevalence in UK has more than doubled from 6 to 16% in men and 8 to 18% in women	Diabetes, coronary heart disease, cancer, osteoarthritis, gallstones, sleep apnoea, reproductive disorders, pregnancy complications, psychological disorders	Individual	Treatment (plus one chapter on prevention)	Not discussed
2000	*Obesity: Preventing and Managing the Global Epidemic* (WHO Technical Report 894)	BMI + Garrow's cut-off points (with discussion about problems of applying to different populations)	Prevalence of overweight and obesity is increasing globally at an alarming rate	**Greatly increased risk**: diabetes, gallbladder disease, dyslididaemia, insulin resistance, breathlessness, sleep apnoea **Moderately increased risk**: CHD, hypertension, osteoarthritis, hyperuricaemia and gout **Slightly increased risk**: some cancers, reproductive hormone abnormalities, PCOS, impaired fertility, low back pain, anaesthetic complications, foetal defects (due to maternal obesity)	Individual and environment	Gives equal weight to treatment and prevention	Contains detailed discussion of economic costs in developed and developing countries

* MLC is an abbreviation for Metropolitan Life Company (see section 3.4.1 for a discussion of their ideal weight tables)
** PCOS is an abbreviation for polycystic ovary syndrome

APPENDIX 3

Fletcher, Isabel (2010) Will the entire population be overweight by 2230? Common sense, scientific consensus and the 'obesity epidemic' in Mathar, Thomas and Jansen, Yvonne J.F.M (eds.) (2010) *Health Promotion and Prevention Programmes in Practice: How Patients' health Practices and Rationalised, Reconceptualised and Reorganised,* transcript Verlap, Bielefield

Introduction

Large body size has historically been interpreted in many ways – as evidence of the sin of gluttony, as a sign of prosperity and, in places where tuberculosis or HIV/AIDS are common, as a sign of health. However, in Western Europe and the United States, large body size is currently defined as 'overweight' or 'obesity'. Obesity (and to a lesser extent overweight), takes large body size out of the category of an individual attribute or a moral failing and defines it as a medical problem. In this chapter I will argue that, in the last ten to fifteen years, obesity has been redefined as a global public health problem. As obesity is, by definition a medical problem (Sobal, 1995: 70-1), this redefinition is not an example of medicalisation, rather what I describe in this chapter is a shift in medical understandings of obesity.

"Obesity is a chronic disease, prevalent in both developed and developing countries, and affecting children as well as adults. Indeed it is now so common that is it is replacing the more traditional public health concerns, including under-nutrition and infectious disease, as one of the most significant contributors to ill health." (WHO, 2000: 1)

This quote, from a widely publicised World Health Organisation (WHO) technical report on obesity, illustrates a major change in understanding of obesity. Obesity is now considered as a major public health problem that affects populations in both rich and poor countries worldwide. In this chapter I aim to analyse when and how this change in understanding took place.

This framing of obesity as a major public health problem sees prevalences of overweight and obesity as continuing to increase, and leading to significant increases in population ill-health that can not be afforded by under resourced health care systems. The following quote, from a letter to *The Lancet* (one of the most prestigious medical journals in the UK) written by two American obesity researchers and published in July 1995, demonstrates the ways in which such ideas were being regularly discussed in the medical press:

"Obesity continues to rise in industrialised nations, an apparent unstoppable side-effect of modernisation. The American public spends $33 billion per year in weight-control efforts,

without any apparent success at population level. Based on prevalence studies from 1960 to 1991, we calculate that by the year 2230, 100% of adults in the USA will be overweight, as defined by a body mass index of more than 27.8 for men and 27.3 for women [...]

The immense research effort into obesity has not yet culminated in effective help for the overweight. 34 894 publications related to obesity have accumulated since 1966, with 4785 related to treatment. Even so, there is still no non-surgical treatment that reliably produces lasting weight loss." (Foreyt and Goodrick, 1995: 134)

The image of the entire American population as overweight is striking, but seems fundamentally unlikely since even in populations with very high levels of obesity, such as the Samoans, some individuals remain slender. Such inflated and hysterical imagery is very typical of the rhetoric that surrounds the 'obesity epidemic'; and for those reasons makes a good title for this chapter. At this early stage of my research, I cannot explain why this shift in understanding of obesity occurred or analyse the entire 'obesity epidemic 'phenomenon, but in this chapter I hope to outline one of its important elements, namely changes in scientific and policy understandings of obesity. These changes are, of course, intertwined with other factors that include the increasing costs of healthcare in affluent countries and the economic interests of both pharmaceutical companies and the food industry. Other important factors more internal to obesity science are changes in research funding and individual career paths.

This chapter will start with a brief description of my research methods, and discuss the resources from Science and Technology Studies and the history of medicine I use in analysing this empirical material. Once I have given a brief outline of the immediate context of the period of my research, I will then provide a brief overview of the history of obesity science, highlighting the changing understanding of obesity found in my material. I will go on to argue that this changing understanding can be best described as the development of a new paradigm of obesity which I will summarise. In particular I will use the work of Thomas Kuhn and Sheila Jasanoff to argue that this involves the development of a new regulatory science or advisory science paradigm. Finally, I shall conclude by examining some of the broader implications of these findings: how these changes relate to larger scale developments within biomedicine.

Outline of research approach and methods

Until very recently my research has been largely document based. Because of my interest in the term 'obesity epidemic' I began to research the history of its use in the popular and scientific press. I rapidly tracked it back to the WHO technical report 894, referred to above, titled *Obesity: Preventing and Managing the Global Epidemic* which was published in 2000. This was an important focus of my early research. Section 3 of this chapter, addressing the history of obesity science, is based on the background reading that I also undertook at this period in my research.

I then analysed a series of British reports on the topic of obesity beginning with a 1969 report by the Office of Health Economics (OHE, 1969), a pharmaceutical industry funded think-tank.

This was my starting point because, as far as I am aware, it was the first modern report on the topic. The series finishes in 2000 with the WHO technical report and includes six other documents: in total two of the eight reports were produced by the Office of Health Economics, three by UK government departments or ad-hoc committees, two by professional organisations and one by the World Health Organisation[131][132]. The material for section 4 of this chapter on obesity paradigms is based on these documents. My analysis does not extend beyond the WHO technical report of 2000 because the main elements of what I have labelled as the 'environmental' or 'population' paradigm of obesity are all present in this report.

These eight documents were selected for two reasons. The first was availability; these are most of the reports that were produced on the topic during this period[133]. The second reason was the involvement of Professor Philip James who was part of the group producing five of these eight reports. Philip James, currently head of the International Obesity Taskforce, has been involved in the production of government reports on the problem of obesity since the 1970s, and was a key figure in the production of the WHO technical report on the subject. He has adopted an almost campaigning role to successfully argue that obesity should be understood as a major public health problem, and so it seemed important to analyse the reports with which he was involved. Finally, I am aware that this is a restrictive sample, which limits the claims that I can make about my findings. However, given that my study involves the development of a new shared understanding of obesity, and alternative constructions of the relationship between bodyweight and health do not form part of these discourses, this small sample is sufficient to illustrate the changes I will discuss.

In order to provide a context for my understanding of these changes, I also tracked the coverage of obesity, the WHO technical report, and the WHO itself in the UK medical press (*The Lancet* and the *British Medical Journal*) between 1993 and 2002. In the last six months, I have also begun to interview a variety of practitioners and academics who write about obesity. Interviewing is supplementing my documentary research in interesting ways, and allowing me to discuss some preliminary understanding of my topic with public health practitioners and other researchers.

Theoretical framework

My research aims to apply the conceptual tools of Science and Technology Studies (STS) to public health, nutritional science, and specifically ideas about the relationship between body weight and health. These are areas that have not been well covered by STS scholars, who, when analysing the biological sciences, have concentrated on recent developments in the life

[131] I anticipate that this list will eventually end with the UK government Foresight report *Obesities: Future Choices* published in 2007

[132] The organisations are the Royal College of Physicians and the Scottish Intercollegiate Guidelines Network

[133] Obviously I have restricted my sample to those published in English, but I have also chosen to focus on the British and European political contexts. I have not yet been able to get access to one report by the British Nutrition Foundation, published in 1997

sciences e.g. genomics, reproductive technologies, stem cell research and synthetic biology. One major exception is Garrety who writes about the development of a consensus around the relationship between cholesterol and heart disease (Garrety, 1997). It seems obvious to apply STS to nutritional science as it is a very significant example of the role(s) that science plays in everyday lives. Writers, such as Gard and Wright (2005), whom I discuss below, have also taken an implicitly STS approach to the obesity epidemic, without labelling themselves as working in this field.

This is also a study of the formation of consensus. One of the things that surprised me when I started this research was how little controversy the idea of an 'obesity epidemic' attracted. There are people and groups who dispute both the link between obesity and ill-health, the existence of increases in average body weight and the fact that this constitutes a global public health problem (Campos, 2004, Gard and Wright, 2005, Oliver, 2006) but these accounts have remained marginal throughout the period of this study. Therefore, my research is mostly studying the formation of a consensus or common sense, which is an approach that can be traced back to Thomas Kuhn (Kuhn, 1970) and his ideas of the role of paradigms in normal science. Kuhn's concept of paradigm provides a way of understanding science in terms of constellations of concepts, theories and practices; large elements of which are taken for granted and unchallenged. This captures the way in which obesity has been discussed since the 1960s, and allows me to analyse sets of understandings and practices that are shared amongst relatively large and diverse groups of research scientists, clinical practitioners and policymakers. Kuhn's ideas and the relevance of his approach remain contested within contemporary STS. However, the scale and complexity inherent in the concept of paradigms is particularly useful in the context of my research, unlike more localised concepts such as 'epistemic communities' (Knorr-Cetina, 1999).

What is being analysed in this chapter is not just the science of obesity, rather it is a hybrid science-policy paradigm in which obesity as a public health problem and its possible solutions are being co-produced (Jasanoff, 2005). This is because scientific knowledge about obesity is a form of biomedical knowledge that has also developed in areas other than the laboratory or the clinic. Apart from epidemiological studies - which I will discuss later - much of this knowledge is assembled in official reports and other policy documents. It is an example of the kind of advisory science about which Sheila Jasanoff has written extensively (Jasanoff, 2005, Jasanoff, 1990). Steven Hilgartner (2000) has also written about this kind of science in his study of controversies about three National Academy of Science reports on the links between diet and health, but as far as I am aware nobody has written about the science of diet and health in the context of the UK[134].

[134] Another approach would be to examine this as a case of the relationship between field science (epidemiological studies) and laboratory science (biochemical and physiological research on animals and humans) in a similar manner to Robert Kohler's analysis of the modern history of biology (Kohler, 2005).

Technologies of quantification and enumeration are an essential element of this advisory science. Within STS there is also a body of writing that addresses the growth of these practices. Deriving from the work of Michel Foucault, Nikolas Rose discusses the increasing use of measurement of individuals and populations by governments (Rose, 2007). Porter also provides a series of case studies demonstrating the growth of quantification as a social technology (Porter, 1995); Yvonne Jansen discusses this point in more detail in this book. These studies overlap with the work of Rothstein that I discuss below. Of particular relevance to the study of obesity is work on the development of new standards and forms of classification of human bodies (Bowker and Leigh Star, 1999, Hacking, 1999). Hacking is the only one of these writers to explicitly discuss obesity, when he uses it as an example of a "kind of people" (Hacking, 2006b), but obesity is not the central focus of his research.

Methodologically, I have also adopted Elizabeth Fee and Daniel Fox's approach of contemporary history, developed in their writing about AIDS as a chronic disease (Fee and Fox, 1992). Fox and Fee argue that such an approach allows for an emphasis on the social construction of diseases, scepticism about ideas of progress, and an avoidance of presentism which they define as using analogies from the present to interpret earlier and still not well understood events (see Fee and Fox, 1992: 9). This theoretical approach fits very well with the basic approach of social constructivist STS.

Because there are strong historical elements to this approach I am also able to use material from the history of medicine to supplement these theoretical resources. Within the history of medicine, several authors have written on changing ideas about body size and fat (Sobal, 1999a, Schwartz, 1986, Stearns, 2002). These authors provide useful historical context for my research since they illustrate changing ideas about the relationship between excess weight and ill-health, and the development of research into the underlying physiological processes involved in nutrition and weight control. Jeffery Sobal (1995) also argues that from the late nineteenth century until the 1970s obesity was increasingly medicalised and in the last twenty to thirty years a process of de-medicalisation has occurred as political demands for the acceptance or rights of fat people have become more accepted. [135] In contrast, other authors conclude that despite the existence of alternatives to the medical framing obesity – for example social justice and market choice framings (Kwan, 2009a) - the medical framing remains dominant (see also Saguy and Almeling, 2008).

The history of public health research in the early 20th century is also starting to be written (Mendelsohn, 1998, Amsterdamska, 2001, Amsterdamska, 2005). William Rothstein (2003) has also written on this period in the history of public health, focusing part of his discussion on cardiovascular disease and describing the development of the concept of risk factors. His account is another important part of the historical context to my research because much of the science I am studying comes out of the post second world war anxiety about rates of

[135] I see no evidence of similar successful campaigning in the UK, but would also question his assumption that obesity can be de-medicalised, given the history of the term as a medical synonym for 'fat'

cardiovascular disease (Rothstein, 2003: 192-209). However, his broader analysis of the development of statistical analysis in modern medicine, which he describes as an 'incomplete revolution' is also very important for understanding the use of risk factors in classifying populations and explaining clinical and individual experiences of disease. This analysis is complemented by the work of Robert Aronowitz (Aronowitz, 1998) who outlines the history of the 'risk factor approach' to CHD and the central role that the Framingham Heart Study played in the development of this new way of understanding heart disease. He links this change to changes in economic circumstances, research practices, professional organisation and disease classification that took place in the US in the 1950s and 1960s (Aronowitz, 1998: 113).

In a similar fashion to heart disease in the early part of the twentieth century, obesity has also been labelled as an epidemic. It is important to investigate the consequences that flow from labelling such chronic non-infectious conditions as 'epidemics'. Rosenberg discusses the socially contingent nature of defining a disease as an epidemic, as opposed to endemic. His argument is that this process ultimately depends on the level of disease expected to exist within a particular population; therefore plague was epidemic among the white colonising population of India in the nineteenth century, but endemic amongst the indigenous Indian population (Rosenberg, 1992, Rosenberg, 1998). The "expected" level of disease in a population is obviously an important topic for sociological analysis, and assessments of increasing levels of overweight and obesity are a crucial element of my research material.[136]

A History of Obesity Science

Historical overview of biomedical research into cardiovascular disease

Before outlining my empirical findings, I am going to give a brief overview of modern biomedical research into cardio-vascular disease (CVD), since this is the context from which research into obesity largely developed. Diseases such as heart disease, stroke, diabetes and cancer have often been known as non-communicable diseases (NCDs) to differentiate them from infectious diseases such as cholera, typhoid and tuberculosis. Infectious diseases were the major cause of mortality and morbidity in the UK, Europe and the USA in the 19th and early 20th centuries. Some authors argue that a combination of public health measures, such as improved sanitation, and new drugs, including sulphanilamide and penicillin, led to declining mortality from infectious diseases and increasing rates of non-communicable or 'degenerative' diseases. This process has been labelled the 'epidemiological transition' and some authors argue that it resulted in dramatic increases in life expectancy in industrialised

[136] In the context of stem cell research, Nik Brown and Mike Michael (Brown and Michael, 2003) have written about the role of expectations and hype in the development of medical technologies, however imagined profits from novel applications of biomedical technologies are different from projected levels of morbidity and mortality in a population

countries (McMurray and Smith, 2001: 9).[137] The phenomenon of increasing prosperity been seen to lead to higher rates of NCDs, links debates around obesity to an ancient strand of thought about 'diseases of civilisation' where the unhealthiness of modern lifestyles are compared to the superior lifestyles of earlier generations (Rosenberg, 1998).

In the early twentieth century, the development of mortality statistics by American insurance companies showed a statistical link between body weight and mortality, particular from heart disease. As rates of infectious disease fell, there was widespread medical concern about high rates of heart disease amongst certain populations (Rothstein, 2003). In the 1950s this situation was described as an 'epidemic' of heart disease, that particularly affected middle aged men. Because the precise causes of this epidemic were unknown a major research effort was seen as necessary, particularly in America, but also in Europe. Coronary heart disease is a multi-factorial condition that develops over several decades, and researchers could not identify specific aetiological factors, as for infectious diseases (Rothstein, 2003: 5). Such a research programme was extensive, and therefore expensive. Clinical research was undertaken in order to understand the physiological mechanisms underlying heart disease and other non-communicable diseases. At the same time, large scale studies such as the Framingham Heart Study were also set up in order to determine accurate prevalences of CHD and identify factors that would predict it (Aronowitz, 1998). As it continued Framingham became a study of the development of cardiovascular disease in a previously healthy population, and an attempt to understand its relationship to specific physiological and behavioural factors such as age, gender, smoking, diet and physical activity (Rothstein, 2003: 279-85).[138]

Although deaths from heart disease have begun to decline in rich countries (see Rothstein, 2003: 343) there is still a profound concern about the impact of these 'epidemics' upon the life expectancy of individuals and populations. In the period of my research a discourse of the 'double burden of disease' has also developed (WHO, 2000). This describes how poor countries face the combined problem of high rates of infectious disease with increasing rates of non-communicable diseases due to increasing industrialisation and urbanisation.

A concern for obesity derives very readily from biomedical research into CVD. It is initially understood as an important risk factor for chronic diseases such as heart disease, stroke and

[137] The framing of changing patterns of health and illness as an 'epidemiological transition' and the role of medicine in these changes is hugely contested. Critics such as McKeown (1979) argue that improved sanitation and nutrition (from increased standards of living are responsible, rather than new drug treatments or preventive practices such as inoculation.

[138] Although not mentioned in these reports, another important line of research developed into the personality traits and behaviours that were seen to result in increased vulnerability to CHD, the stereotypically competitive and deadline orientated individual labelled 'Type A man' (Aronowitz, 1998, Riska, 2000).

diabetes. However, as the focus on obesity increases, it becomes treated more often as if it were a disease in its own right (Aronowitz, 1998: 116).[139]

I have developed a timeline of the important events relevant to my research. As part of this process I have also tried to divide this period into different phases (or maybe strata) in order to understand the changes that have been taking place. An edited version of this timeline is presented below. It is divided into four phases as an ad-hoc way of making sense of a large body of empirical material – the changes that make up the shift to a new paradigm of obesity occur throughout the entire timeline, at different points for different aspects of that paradigm (as discussed in section 4)

First phase late 1940s to 1969: obesity as a risk factor for coronary heart disease

In this period the first big epidemiological studies like the Framingham Heart Study and the Seven Countries Study begin (1948 and 1958) and the results of the Build and Blood Pressure Study (1959) are published. These studies are often jointly funded by US university public health departments and international organisations such as the WHO. The actuarial tables of the Metropolitan Life Company are the standard reference for adult weights. In the statistical data quoted from these tables, body sizes are classified on the basis of percentage of average weights - >110% of average weight considered overweight and >120% obese: frame sizes are included in height and weight tables.

There is a geographical movement of information from US to the UK and this American data is absorbed into existing British research activities. Using the framework developed by the Framingham researchers (Aronowitz, 1998), obesity is usually described as a risk factor for other conditions, notably heart disease, and individual solutions of appropriate diet are recommended, (there is a worry that exercise is counterproductive since it increases appetite). The research summarised in the reports of the period is asking whether all obesity is the same, and whether it is an inevitable part of aging. It is also investigating whether the obese are different from the non-obese in significant ways (other than weight), such as metabolism.

Second phase 1970s to mid 1990s: the growth of obesity science

The WHO MONICA (Multinational MONItoring of trends and determinants in CArdiovascular disease) project starts in 1979. This becomes an important source of data about increasing rates of obesity in European populations. Occasional reports are produced exclusively on obesity in the UK (DHSS/MRC, 1976, RCP, 1983); this increasing focus shows how obesity is making the transition from being understood as a risk factor to being treated as

[139] These same processes can be seen to have occurred, to a lesser extent, with high blood pressure and high blood cholesterol levels

a disease in its own right. BMI (body mass index)[140] is starting to be used to classify body sizes, the standard definition (BMI > 25 is overweight, and > 30 is obese) is proposed in 1973 by George Bray, at an international obesity conference, and gradually becomes widely adopted: frame sizes become less important and, by the end of this period, obsolete.

As part of these processes the list of the health consequences of obesity is becoming standardised, (i.e. developing a routine form that was repeatedly cited) in part due to the results coming out of the Framingham Heart Study and the second American Cancer Society study. The focus in the reports during this period is still on individual solutions such as appropriate diet, but there is more stress on ideas of healthy eating, and discussions of the limited role of exercise in weight loss are starting to be included. Research cited is still questioning whether the obese are different from the non-obese in significant ways (other than weight).

Third phase 1995 to 2002: obesity becomes a global public health problem

During this period there is an increase in activity around the topic of obesity – the Scottish Intercollegiate Guidelines Network (SIGN) report is published in 1995, the International Obesity Taskforce (IOTF) is founded in 1996, the National Obesity Foundation (NOF) is founded in 2000. A major series of papers containing results from the WHO MONICA study are published in 2000. The WHO consultation takes place in 1997, and Technical Report 894 is published in 2000; subsequently the phrase 'obesity epidemic' is frequently used in the popular and scientific press.[141] In this report, and other WHO publications, the increasing prevalences of obesity become seen as a global problem, supported by ideas of the 'double burden of disease' affecting poor countries (Murray and Lopez 1996: 9). Towards the end of this period the focus of concern shifts increasingly onto the problem of increasing rates of childhood obesity.

Obesity becomes described as a major public health problem that will reduce life expectancy and increase healthcare costs significantly (figures are often given to support these arguments). Solutions discussed in these reports and in both the scientific and popular press include individual ones of better diet and increased activity levels but also broader based ones of restricting advertising and sales of certain kinds of foods. Research continues into the physiology of appetite and maintaining body weight, but it is now generally accepted that the majority of the overweight are the same as the rest of the population, except that at, some point, they have over-consumed. However, at the same time there is a developing understanding that some populations seem more vulnerable to obesity and the accompanying health problems than others. The standardised BMI classification has been widely accepted

[140] The formula for body mass index is W/H^2. It was developed by the French statistician Adolphe Quetelet in the early 19th century and initially known as the Quetelet Index. In the reports I discuss, it is first known as W/H^2 and then BMI. I have kept to the name used by each report's authors.
[141] Once this usages is established obesity then becomes regularly described as 'pandemic' (Manson et al., 2004) or using a new term 'globesity'

and is used by the WHO, but there is also the beginning of discussions about whether they should be different for some groups e.g. S.E. Asians. This shows the movement between a global classification system and the development of specific classifications for different population groups that incorporate the metabolic differences resulting from varying levels of maternal nutrition and neo-natal development.

Fourth phase 2003 – present: policy responses to the problem of obesity

The number of official reports about the public health problem of obesity increases again, including the report of the UK government Foresight scoping project *Tackling Obesities: Future Choices* which is published in 2007. European initiatives including the Platform on Diet Health and Activity DIoGENES (Diet, Obesity and genetics, which is a multi-centre research project) and the PORGROW project, which investigates potential policy responses to the public health problem of obesity, are launched.

The important role of sedentary lifestyles in leading to weight gain is stressed – for children blame is put on too much time spent watching TV or playing computer games. Obesity starts to be widely and routinely described as an environmental, rather than solely an individual, problem - the phrase 'obesogenic environment' (Egger and Swinburn, 1997) becomes widely used and accepted (parallels start to be drawn with global warming). Environmental solutions such as redesigning urban space in order to promote physical activity begin to be discussed alongside traditional individual solutions. Increased policy discussion is not reflected in concrete responses – perhaps because the problem is widely acknowledged to be complex and multi-faceted. The secondary literature analysing this new understanding of obesity also begins to grow.

Interestingly, despite the recent spread of genetic explanations for disease, they have not become common in this area of biomedicine. Although there are a few specific genetic conditions that give rise to obesity in a minority of individuals, the search for a genetic cause for obesity in the majority of the population has been unsuccessful. In fact supporters of the idea of an obesogenic environment argue that the fact that dramatic changes in the prevalence of obesity have taken place in the last 20 years shows that the causes cannot be genetic (WHO, 2000: 101).

Surgical treatments have developed enormously in this period, but have not led to significant changes in understandings of obesity, or potential treatments for the majority of patients. Throughout this period research into the basic physiological processes involved in appetite, digestion and regulation of body weight, as well as more focused research looking for potential therapies has continued. There have been periods of excitement, most notably after the discovery of leptin in the mid 1990s (Shell, 2003) and a growing list of drugs that have been prescribed for weight loss and then been withdrawn due to adverse health effects. However, these developments also do not seem to have profoundly changed the ways that obesity is currently understood.

The two paradigms of obesity

These phases cover changes in the measurement of obesity and the subsequent classification of bodies; in understandings of the causes and health consequences of obesity; in research undertaken about obesity and related chronic diseases; and in the potential policy responses to it. As I have accumulated this material I have become increasingly aware of the links between these developments, and argue that they are best thought of in terms of the development of a new paradigm of obesity. The older individual understanding of obesity still retains some explanatory power in particular situations, but it has been supplemented and, in some cases largely eclipsed, by a broader environmental and population based understanding of obesity. Because they operate at different levels of explanation (i.e. the individual and the population) these two different understandings of obesity are not mutually incompatible; mostly they co-exist with limited areas of overlap. They aren't exemplary achievements (Kuhn, 1970), but they do contain different conceptions of the problem of obesity, different sources of information about it and lead to different sets of practices around it, so I think the concept of paradigm is an appropriate one to use in this context.

The individual and the population/environmental paradigms of obesity

In the older documents I have analysed, obesity is understood as an individual health problem, but from the 1980s onwards a different understanding of obesity develops in which it has become a population issue, and a major public health problem: 'weight gain and obesity are posing a growing threat to health in counties all over the world' (WHO, 2000: 1).

In the rest of this section I shall describe six areas of significant difference between these two advisory science paradigms of obesity. Table 1 (below) summarises this discussion by comparing the older (pre-1990s) individual paradigm of obesity, and the new population/environmental (post-1990s) paradigm.

Individual paradigm	Environmental/ population paradigm
Incidence/prevalence of obesity	
Low and stable prevalence so an individual health problem	High and increasing incidence so a population /public health problem
Measurement and classification of bodies	
Measured by weight and frame size	Measured by BMI
Accepted consequences of obesity to health	
Knowledge of health risks derived from case histories and insurance company tables	Knowledge of health risks comes from large scale epidemiological studies
Assumed causes of obesity	
Due to overeating and lack of exercise	Due to inappropriate diet and sedentary lifestyle
Treatment/prevention	
Corrected by dietary restrictions	Solutions involve changing food supply and restructuring urban environment
Cost of obesity	
Cost of obesity limited and mostly born by individuals	Cost of obesity becomes significant problem for national healthcare systems

Table 1: The two paradigms of obesity

1. Incidence/prevalence data

The individual paradigm of obesity was developed in a context where the prevalence of obesity was usually understood to be relatively low and, therefore, it could be seen as an entirely individual problem. In the earlier reports, small scale studies from the UK are usually cited and prevalence of 'clinical obesity' is estimated at 20% in one study (OHE, 1969: 5). After information had started to be collected in the UK, one report gives prevalences that use BMI to define 6% of men and 8% of women as obese and 34% of men and 24% of women as overweight (RCP, 1983: 21).

However, rising incidences of obesity were one of the major reasons for the change in understandings of obesity: if the majority of a population are (or will become) overweight or obese, it makes no sense to understand it as an entirely individual condition. In 1994 the second Office of Health Economics report argues that in the UK, 'over half the male population and just under half the female population are overweight to a clinically undesirable degree' (West, 1994: 5). The 1995 Department of Health report gives figures of

13% of men and 16% of women as obese, and a total of half the adult population as overweight or obese (DoH, 1995: 2)

In the WHO technical report the figures for prevalence come from the WHO MONICA project and European national surveys and NHANES (for the US). Information for other parts of the world is repeatedly described as inadequate. However, the available information was described as showing high or increasing rates of obesity for all areas of the world. In particular, to compare with the figures above, the prevalence of obesity for England is given as 15% for men and 16.5% for women; for China it is given as 2% for men and 1% for women; for the US it is 20% for men and 25% for women, and for urban Western Samoa it is given as 58% for women and 77% for men (WHO, 2000: 22). Rates of overweight are no longer discussed.

2. Definitions of obesity and classifications of bodies

The individual paradigm of obesity uses height and weight table with ranges of ideal weight to define individuals: 'The commonest method is to weigh an individual and check the weight against average weight tables. Almost all such tables are based on US data collected by 26 large life insurance companies over a period of twenty years, and involving the observation of nearly five million insured people' (OHE, 1969: 4). Relative weight is used as a way of classifying the overweight and obese. The standard used is the Metropolitan Life Insurance Company reference tables: 'A cut-off point often used for separating obese from non-obese is a relative weight 120 per cent of the "desirable"' (DHSS/MRC, 1976: 4). A later report introduces the classification of 110 – 119% of ideal weight as overweight, and greater than 120% as obese (RCP, 1983: 4). The Metropolitan Life Insurance Company tables also use frame sizes to give a range of ideal weights[142]. This is a measure of obesity that is relative to the average weight in a given population.

The environmental paradigm of obesity uses BMI[143] to classify individuals and populations. This is a measure that does not depend on average weights; it is in some sense an absolute standard. The weight/height^2 calculation is first mentioned in the Department of Health and Social Security (DHSS)/Medical Research Council (MRC) 1976 report as an alternative to relative weight, but not actually used throughout that report, although Bray's classification of desirable weight, overweight and obese[144] is also mentioned. In the Office of Health Economics report of 1994 and the 1996 Scottish Intercollegiate Guidelines Network (SIGN, 1996), Bray's classification is presented as standard, and by the time of the WHO technical report these processes of standardisation are complete, since it has become the global standard; although the authors also acknowledge that the cut-off points may need to vary between populations, due to different susceptibilities to the metabolic consequences of obesity (WHO, 2000: 8).

[142] For a 5ft 9in man these range from 136 – 145 lbs for a small framed individual to 151-170 lbs for a large framed individual; for a 5ft 5in woman the comparable ranges are 111-19lbs and 125-142lbs (OHE, 1969: 5).
[143] BMI is defined as W/H^2 i.e. weight divided by height squared
[144] A BMI of 20-24.9 is considered a desirable weight; a of BMI 25-29.9 as overweight: a BMI of 30-40 as obese and a BMI >40 as severely or morbidly obese (WHO, 2000: 9).

3. The assumed causes of obesity

On an individual level, the causes of obesity have been understood, since Hippocrates and Galen, to be overeating and a sedentary lifestyle (Gard and Wright, 2005: 70-1). In the earlier reports it is seen to be caused by consuming too many calories – too much food - but also too much of the wrong type of food i.e. a diet high in fat and sugar (OHE, 1969: 11). It is also seen to be caused by too little exercise. However, studying the association between low levels of physical activity and obesity is complicated by the problem of 'whether this is a primary condition which induces obesity or a secondary response to the high energy cost of moving' (DHSS/MRC, 1976: 30).

A key element of the environmental paradigm of obesity is that the causes of obesity are environmental or structural rather than solely individual.[145] The first mention of environmental causes of obesity comes in the Royal College of Physicians' report: 'British and American epidemiological studies are consistent with environmental factors being important in determining the body weight of both children and adults' (RCP, 1983: 24). However, the argument is not further developed in this report. The WHO technical report argues that the rising proportion of fat and increasing energy density of diets combined with reduced levels of physical activity are contributing to rises in average body weights of populations. The authors extend this argument to state that 'The global obesity problem can be viewed as a consequence of the massive social, economic and cultural problems now facing developing and newly industrialised countries, as well as ethnic minorities and the disadvantaged in developed countries' (WHO, 2000: 102).

4. Describing the health effects of obesity

For individuals some of the health effects of obesity have been part of medical knowledge since antiquity, but statistical knowledge of relative risk is incorporated in to the Metropolitan Life Insurance Company tables so that they listed 'desirable' weights for men and women of different heights based on 'weights associated with lowest mortality' (OHE, 1969: 4). The Build and Blood Pressure Study (Society of Actuaries, 1959) was also cited to show that amongst men more than 10% overweight excess mortality was found to be approximately 20% and amongst men more than 20% overweight it was almost one third (OHE, 1969: 15, DHSS/MRC, 1976: 21). Health consequences listed included mechanical i.e. effects on joints, obstruction of breathing and varicose veins and metabolic i.e. diabetes and raised cholesterol leading to heart disease.

Beginning with the Royal College of Physicians Report in 1983, the list of health consequences of obesity starts to becomes standardised, using the data from Framingham and the American Cancer Society studies. The list includes coronary heart disease, hypertension, diabetes, gall

[145] These authors acknowledge that the incidence of obesity is inversely related to socio-economic class, however, the link between environmental stresses and the development of insulin resistance that would allow a connection to be made with work on the social determinants of health (Marmot and Wilkinson, 1999) is not made in these documents

bladder disease, arthritis, certain kinds of cancers and psychological problems (RCP, 1983: 9). By the time of the WHO technical report, the authors can argue that most of these consequences are 'well documented', supported by a number of studies or the association between obesity and the risk of developing a condition has been 'repeatedly observed' (WHO, 2000: 47). Thus a consensus has been created about the wide ranging and negative effects of obesity on mortality and morbidity.

5. Treatment and prevention

The individual to the environmental paradigm contain different ideas about the appropriate treatment of obesity. In the new paradigm, there is a partial acceptance of the ineffectiveness of dieting; the subject of physical activity is also regularly discussed, as its role in the prevention of weight gain became seen as important. Initially the treatments discussed are calorie restricted diets of various kinds, sometimes combined with medication such as amphetamines, 'to help the patient adhere to a particular diet' (OHE, 1969: 24)[146]. In these earlier reports, there was also a worry that exercise would increase appetite: 'whilst both activity and resultant heat loss owing to muscular inefficiency dissipate body energy, they also lead to an increase in both caloric requirements and appetite' (OHE, 1969: 22)

The beginnings of a shift to broader environmental causes of obesity is shown in the conclusion of one early report which discusses measures that the government and food manufacturers could take 'to promote change in the national diet' (RCP, 1983: 50) such as breeding animals with lower body fat, promoting the use of low fat spreads and developing low fat, low sugar processed foods. The 1995 Department of Health report is the first to argue for a population approach to the problem of obesity (DoH, 1995). It discusses trends in food consumption, physical activity and environmental temperature as factors that might explain increases in average BMI, and also lists steps to be taken by the NHS, local authorities, schools, workplaces and industry in order to increase activity levels (DoH, 1995: 10). The WHO technical report discusses prevention and management strategies that focus on 'elements of the social, cultural, political, physical and structural environment that affect the weight status of the community or population at large' (WHO, 2000: 154).

6. Costs to healthcare services

Initially, obesity is described as making up a small proportion of GPs consultations and not ordinarily being the sole cause of a patients visit. The cost of obesity is estimated at £3.5 million, made up of £3 million on prescribed drugs and the rest on hospital-bed days (OHE, 1969: 25).[147] The second Office of Health Economics report gives the direct costs of obesity to the NHS as £29.35 million, made up of costs from general practice (the largest element), in-patient treatment, out-patient treatment pharmaceutical service and treatment by dieticians (West, 1994: 39).

[146] The most widely prescribed drugs in the 1960s were amphetamines; in the 1980s fenfluramine and phentermine were often used (RCP, 1983: 76)
[147] This figure is also described as small compared to the much larger sums spent by individuals on diet foods and slimming products.

By contrast, the WHO technical report quotes a range of international studies of both the direct and indirect costs of obesity estimating that they amount to 2 to 7% of total health care costs or 12 000 million francs in France for 1992 and $56 300 million in the US for 1986. The report' authors argue that these figures 'clearly indicate that obesity represents one of the largest items of expenditure in national health care budgets' (WHO, 2000: 79).

Discussion

In the sections above, I have outlined six changes that have occurred in scientific understandings of obesity – in its prevalence and incidence, its measurement, its causes, its health effects, its treatment and its cost to healthcare systems. I have argued that these changes when combined amount to a new understanding of obesity which I have labelled an advisory science paradigm shift. I am not arguing that they amount to a scientific revolution, as Kuhn (1970) would understand it, since the two paradigms of obesity continue to co-exist, and the changes I describe are incremental ones that have occurred with each new report or other enactment of the paradigm. However, other writers have accepted that this is one way in which paradigm shifts can occur (Barnes, 1982: 86-7).

The science of obesity contained within these reports is based on physiological and epidemiological research, but goes beyond it by constructing an argument for obesity as a national, and then a global, health problem. The conclusions of these reports are a synthesis of many different research studies, created by the work of ad hoc committees, government departments and international organisations such as the WHO. Hence, these processes create a new entity of advisory science or 'policy-relevant science' (Jasanoff, 1990). Using such a perspective to discuss the 'new' science contained in these reports brings together the work of Thomas Kuhn with that of Sheila Jasanoff (ibid. and see also Jasanoff, 2005) to understand the processes involved in the creation and dissemination of advisory or policy science paradigms. It involves considering particular reports as enactments of such paradigms and understanding the development of such hybrid knowledge/policy practices.

Tentative explanations for such changes include the growth in obesity science (deriving from the overall growth in biomedical research in this period) and the increasing amount of health statistics collected about individuals and about health care services. Another important element is the role played by particular entrepreneurial scientists, such as Professor Philip James, who adopt a dual role as research scientists who also serve on governments committees and compile the reports I have analysed. But these factors do not explain the impressive success of such initiatives.

Recent social science writing about health and illness argues that in the last twenty or thirty years population-level knowledge about health risks has been used to identify and target 'at-risk' individuals as part of wider attempts to convince them of their personal responsibility for their future health (Rose, 2007). In other worlds, this body of literature depicts a focus on the health of populations as another means of individualising responsibility for health. However,

my research raises questions about this line of argument. This is because the environmental paradigm of obesity, which constructs obesity as a population-level health issue, does not target individual health behaviour, but rather emphasises interventions that are aimed at populations e.g. structural factors such as changes in food production and manufacturing and changes in the built environment and transportation systems. This suggests that biomedical understandings of the relationship between individual and population health are more complex than our current social science theories allows.

Conclusion

In this chapter, I have summarised important developments in recent obesity science. I have outlined how it developed out of a more generalised concern with rising rates of NCDs amongst populations of industrialised countries experiencing increased prosperity after World War 2. This concern led to a major research effort that attempted to understand the causes, and prevention, of conditions such as heart disease, diabetes, cancer and stroke. Clinical and laboratory research into NCDs was supplemented by large scale epidemiological studies, such as the Framingham Heart Study, in order to identify the most significant risk factors for such diseases. One of the effects of such research was to identify obesity as one important risk factor, and intensify a process of medicalisation that had begun with the publication of the Metropolitan Life height and weight tables in the earlier part of the century. Obesity made the transition from a personal characteristic to an important risk factor for several important chronic medical conditions.

In the second part of this chapter I argued that in the last twenty years there has been an important change in the way that obesity is understood, both medically and as a public health problem requiring the development of new policy solutions. As I have outlined above this new understanding, which I have labelled the environmental/population paradigm, encompasses new information about the prevalence of obesity, about its causes, its health effects, its treatment and its costs to healthcare systems and society at large.

It seems obvious that obesity is now understood as a global epidemic, because of the rising prevalences that have been documented in communities in Europe and North America. However, I would argue that although average body weights may be increasing in certain populations, this bald fact is insufficient to explain the development of a consensus around the idea that rapidly increasing rates of obesity have to be tackled as a matter of urgency before they decrease average life expectancies and bankrupt healthcare systems. This understanding has become commonsense, partly due to decades of research and lobbying of governments, and international organisations such the WHO, by a group of committed obesity scientists who saw it as a major public health problem decades before the current high levels of concern. In making this argument I am not trying to put forward a conspiratorial understanding of such developments. All branches of biomedical research have to lobby for funding and attention from policymakers. Nor am I trying to impugn the motives of the

individuals who have adopted this campaigning approach to the problem of obesity. Public health research and policymaking is usually undertaken by people who see themselves as tackling important public health problems. [148]

My research is different from many of those who have already written on obesity, not only because it uses the theoretical tools of STS to analyse the science behind ideas of the epidemic, but also because it does this in a systematic and symmetrical (Bloor, 1976) fashion that is not part of a critique of the construction of obesity as a public health problem. Such approaches are important (Fee and Fox, 1992) because they can unpick the development of common sense ideas about what issues should be the focus of public health and preventive medicine. They, therefore, have value as a way of analysing claims made by different individuals, and groups, about the urgent nature of particular health problems, including the ones with which I began this chapter.

[148] This sense of purpose exists despite knowledge of the harmful side effects of certain treatments for overweight and obesity (pharmaceutical, surgical and dietary) and is seen by activists and other critics of the obesity epidemic to promote discrimination against individuals of larger body size.

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