BOVINE HERPESVIRUS 1 INFECTION IN NORFOLK: EPIDEMIOLOGY AND ELIMINATION

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DECLARATION

I hereby declare that:-

a. I composed this thesis myself.

b. The work is all my own except where specifically mentioned in the acknowledgements or in the main text.

c. This thesis has not been submitted in candidature for any other degree, diploma or professional qualification.

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The substance of Chapter 5 was presented at a meeting of the Society for Veterinary Epidemiology and Preventive Medicine at Edinburgh on 3.4.92 and details are included in the published proceedings (Pritchard 1992).

ABSTRACT

The epidemiology of bovine herpesvirus 1 (BHV1) infection in Norfolk was studied by scrutinizing records of submissions to Norwich Veterinary Investigation Centre and undertaking a cross-sectional analysis of serological findings from breeding herds in a disease monitoring scheme. Longitudinal studies examined the feasibility of eliminating BHV1 infection from individual herds by a test-and-cull programme and of maintaining seronegative status by employing security measures including serological screening of replacements. Alternative strategies were adopted in two heavily infected herds. A review of the appropriate literature and a description of the Norfolk cattle industry are also presented.

Thirteen incidents of BHV1 fetopathy and 83 outbreaks of systemic infectious bovine rhinotracheitis (IBR), including 13 clinically mild low morbidity recrudescences in dairy herds, were confirmed between 1980 and 1989. These affected about 11 per cent of dairy herds and five per cent of suckler herds. Outbreaks occurred mainly during the winter months in rearing/fattening herds but throughout the year amongst adult breeding cattle. BHV1 antibodies were present in 510 (26.7 per cent) of 1908 sera and 122 (44.5 per cent) of 274 herds tested. There were no significant differences in antibody prevalence between herd types or between systemic illness and fetopathy submission categories but fewer sera from apparently healthy cattle were seropositive ($P<.001$). Antibody prevalence increased significantly with age after three years.

BHV1 antibodies were detected in the sera of 639 (15.1 per cent) cattle from 4219 aged at least two years in 56 scheme herds with no history of clinical IBR or IBR vaccination; almost 50 per cent of cattle aged 10 years or more were seropositive. Antibody prevalence amongst purchased cattle was much greater than in homebred cattle ($P<.001$ in dairy herds, $P<.05$ in suckler herds). Reactors were present in 40 of the 56 herds: in seven herds more than 40 per cent of adult cattle were seropositive. In these high prevalence (HP) herds most cows seroconverted to BHV1 within two years of first calving whereas there was virtually no evidence of active infection in the 33 low prevalence (LP) herds. Maternally derived antibodies waned by about six months and young stock in infected herds subsequently remained seronegative provided they were kept apart from the cows and external sources of infection.

BHV1 infection was readily eliminated from LP herds; serological freedom was retained during surveillance periods of up to four years by strict adherence to disease security rules. The repeated use of inactivated BHV1 vaccine to reduce virus shedding appeared to prevent further spread within an HP suckler herd; combined with partial segregation it was also used in the successful phased removal of reactors from an HP dairy herd within 30 months.

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DEDICATION

This thesis is dedicated to the memory of my father,

Eric Dudley Pritchard.

LIST OF ABBREVIATIONS

ADAS	Agricultural Development and Advisory Service
ADV	Aujeszky's disease virus
AI	Artificial insemination
BHV1	Bovine herpesvirus 1
BSE	Bovine spongiform encephalopathy
BVDV	Bovine virus diarrhoea virus
CHS	Cattle Health Scheme
CI	Confidence interval
CVL	Central Veterinary Laboratory
D	Dairy
EBL	Enzootic bovine leukosis
EC	European community
ELISA	Enzyme-linked immunosorbent assay
ET	Embryo transfer
FAT	Fluorescent antibody test
HP	High prevalence
IBR	Infectious bovine rhinotracheitis
IHAT	Indirect haemagglutination test
IPB	Infectious pustular balanoposthitis
IPV	Infectious pustular vulvovaginitis
IR	Inconclusive reactor
LP	Low prevalence
MAFF	Ministry of Agriculture, Fisheries and Food
MDA	Maternally derived antibody
OD	Optical density
PI3	Parainfluenza type 3

RH	Relative humidity
S	Suckler
SD	Standard deviation
SE	Standard error
SNT	Serum neutralisation test
SVS	State Veterinary Service
VIC	Veterinary Investigation Centre
VIDA II	Veterinary Investigation Diagnosis Analysis II
VIS	Veterinary Investigation Service
VTM	Virus transport medium

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PREFACE

Bovine herpesvirus 1 (BHV1) is responsible for a broad spectrum of disease manifestations in cattle, the most important being infectious bovine rhinotracheitis (IBR) and infectious pustular vulvovaginitis (IPV)/infectious pustular balanoposthitis (IPB). The latter genital tract conditions were not encountered during these investigations and are alluded to only briefly in the text. To avoid the inconsistencies with nomenclature that are apparent in much of the literature, the term "BHV1" has been used throughout when referring to the causal virus and "IBR" (or IPV/IPB) when describing the clinical disease.

In recent years there has been growing interest in the control and elimination of BHV1 infection, particularly in Europe, because of its significance to international trade in live cattle, semen and embryos in addition to the potentially serious economic effects of clinical disease outbreaks. Despite its importance, there remains a dearth of information about the epidemiology of this common endemic infection - detailed observational studies have been particularly lacking.

The work presented in this thesis utilizes available field data to examine the natural history of BHV1 infection in the numerically small, but none the less important, cattle population of Norfolk during the decade spanning its introduction into the county to the first steps in its elimination.

CHAPTER 1: REVIEW OF THE LITERATURE

BHV1 infection has been the subject of several fairly comprehensive published reviews, notably those by Gibbs and Rweyemamu (1977), Kahrs (1977), Yates (1982), Wyler and others (1989) and Straub (1990a). In keeping with the general theme of this dissertation the present review concentrates mainly on those aspects of BHV1 infection that relate to its clinical features, epidemiology, control and eradication.

HISTORY AND OCCURRENCE

The respiratory form of BHV1 infection was first observed in beef feedlots in Colorado, USA in 1950 (Miller 1955) and subsequently described as an apparently new upper respiratory disease of dairy cattle in California in 1953 (Schroeder and Moys 1954). The clinical condition was formerly named as "infectious bovine rhinotracheitis" by McKercher and others (1955). The causal agent, initially isolated in tissue culture by Madin and others (1956) and identified as a herpesvirus by Armstrong and others (1961) was the first virus (Mohanty 1978) shown definitely to cause respiratory infection in cattle.

Gillespie and others (1959) found that the virus causing IBR was serologically indistinguishable from that causing a mild transmissible venereal disease of cattle recognised in Central Europe and North America since the 19th century. This genital tract

condition is now generally known as "infectious pustular vulvovaginitis" (Kendrick and others 1958); the analagous clinical condition in bulls is usually termed "infectious pustular balanoposthitis" (Studdert and others 1964).

Following its emergence in the USA, IBR was soon identified elsewhere including: New Zealand (Webster and Manktelow 1959), Germany (Grunder 1960), Canada (Studdert and others 1961), Britain (Darbyshire and others 1962) and Australia (Snowdon 1964). This disease was uncommon and economically insignificant in Britain during the 1960s and early 1970s (Wiseman 1988) compared with IPB and IPV (Huck and others 1971, Collings and others 1972, Deas and Johnston 1973) which carried the serious potential risk of being transmitted in semen from artificial insemination (AI) studs. However, with the introduction of strict control measures in AI centres (Lucas 1986) IPB/IPV was rarely seen in Britain after the 1970s (Edwards 1983, Edwards 1988).

In contrast to the decline of IPB/IPV, there was a sharp increase in the incidence and severity of IBR in Britain after 1976 (Edwards 1988): outbreaks in the north east of Scotland were clinically indistinguishable from those first described in the USA feedlots during the 1950s (Wiseman and others 1978, Cuthbertson 1979). Reports of similar outbreaks followed from the rest of Britain (Anon 1979) and were also described in continental Europe where a virulent field strain causing severe IBR had been seen since the early 1970s (Straub 1978, Wiseman and others 1979). BHV1 now has a worldwide

distribution and is endemic amongst most European countries although Norway, Finland and Sweden are almost free from infection and it has been virtually eradicated from Denmark and Switzerland (Ackerman and others 1990a).

AETIOLOGY

BHV1 is classified as an alphaherpes virus, a subfamily that also includes herpes simplex virus and Aujeszky's disease virus (ADV). These all grow rapidly, are highly lytic and produce acute disease followed by life-long latent infection (Studdert 1989). The BHV1 genome contains double-stranded DNA; the icosahedral nucleocapsid consists of 160 capsomeres surrounded by a lipid-containing envelope to form somewhat pleomorphic virions 150 - 200 nm diameter (Gibbs and Rweyemamu 1977, Wyler and others 1989). The virus penetrates the host cell by fusion with the plasma membrane and replicates in the nucleus.

BHV1 is stable for up to a month at $4^{o}C$ and over a wide pH range (6.0 - 9.0) but is inactivated within 21 minutes at $56^{o}C$ (Griffin and others 1958) and very susceptible to lipid solvents and common disinfectants including 0.5 per cent sodium hydroxide, one per cent phenol derivatives and one per cent quaternary ammonium bases (Straub 1990a). It is stable in extended semen for several days at room temperature and can survive five freeze-thaw cycles without a

significant drop in titre (Drew and others 1987). It is potentially capable of contaminating virus-free semen when stored in the same container (Krpata 1982).

Elazhary and Darbyshire (1979) examined the aerosol stability of BHV1 suspended in calf nasal secretions. They found that it survived in the atmosphere for at least three hours giving it the potential for airborne transmission, particularly under intensive housing conditions; low temperature ($6^{o}C$) and a high relative humidity (RH) of 90 per cent were most favourable, with a lower RH required with increasing temperature. According to Straub (1990a) BHV1 can survive in the environment for 30 days in winter and inside buildings for six to 13 days (winter) and five to nine days in spring.

BHV1 isolates all exhibit the same serotype in cross neutralisation tests, regardless of whether they are derived from IBR or IPV cases; vaccine and virulent strains are similarly antigenically indistinguishable (Homan and Easterday 1981). However, the use of restriction endonuclease analysis and selective reactivity of monoclonal antibodies and viral protein patterns (Misra and others 1983, Metzler and others 1985) has demonstrated five virus subtypes: 1, 2a, 2b, 3a, 3b. Subtype 1 is generally considered to be "IBR-like" and subtype 2 "IPV-like" but in their review, Wyler and others (1989) concluded that such references largely reflected evolutionary history and noted that it was still not possible to establish a strict correlation between clinical origin of isolates and their molecular subtype.

Edwards and others (1990) classified British isolates from the 1960s as type 2b but found predominantly type 1 (resembling the prototype USA Colorado strain) and occasional type 2b from 1977 onwards; types 2a and 3 were not detected; only type 2b was found in Northern Ireland. The close similarity between isolates from severe IBR outbreaks in Europe in the 1970s and those in the USA in the 1950s led Wiseman (1984), Nettleton (1986) and Edwards and others (1990) to conclude that a virulent strain of BHV1 (subtype 1) was introduced into Europe and the United Kingdom during the 1970s with imports of infected cattle, particularly Holsteins (Msolla and others 1981) from North America.

CLINICAL AND PATHOLOGICAL FEATURES

The effects of BHV1 infection can vary from mild or inapparent illness to severe and fatal disease (Kahrs 1977). This wide diversity of clinical outcome appears to be determined multifactorially by virus strain, route of exposure, immunological status, age susceptibility and environmental factors (Kahrs 1977, Blood and Radostits 1989a).

IBR is economically the most important clinical entity (Straub 1990a). The incubation period for field cases is probably as long as a week (Wyler and others 1989); all ages and breeds of cattle are susceptible but natural disease occurs mainly in animals over six months old (Blood and Radostits 1989a, Straub 1990a). Clinical descriptions of severe disease from the USA in the 1950s (Schroeder

and Moys 1954, Miller 1955) are essentially the same as those in Britain during the late 1970s (Wiseman and others 1978, Cuthbertson and Wood 1979, Wiseman and others 1980). There is pyrexia (up to 42°C), increased respiratory rate, persistent harsh cough, anorexia, depression and a marked fall in milk production by milking cows. A clear bilateral nasal discharge becomes thick and mucopurulent within a few days of onset; conjunctivitis with profuse lacrimation is usually present. The acute phase lasts for between five and 10 days after which animals recover, although there may be loss of condition. Mortality is usually low or negligible but morbidity is invariably high: more than 90 per cent of cattle were affected in 10 of the 15 outbreaks described by Wiseman and others (1980). Secondary bacterial infection, particularly with Pasteurella sp. or Mycoplasma bovis (Allan and others 1980, Yates 1982) can result in pneumonia although this may not be apparent clinically (Wiseman and others 1980). The main pathological features of severe IBR include necrotising rhinitis, pharyngitis and laryngotracheobronchitis with extensive pseudomembrane formation (Allan and others 1980).

Clinically milder outbreaks of IBR have been described in the USA (Abinanti and Plumer 1961), Britain (Dawson and others 1962) and Ireland (Timoney and O'Connor 1971). These were characterised by high morbidity and intense, generally bilateral conjunctivitis with profuse lacrimation; congestion of the nasal mucosa and a serous to mucopurulent nasal discharge were noted by Dawson and others (1962) but respiratory signs were not reported by the other authors.

Whilst IBR is well recognised as a disease of fattening beef cattle (Wiseman 1980, Ross and others 1983) some authors including Peters (1987) have suggested that IBR plays a significant role in multifactorial enzootic pneumonia of younger calves (Blood and Radostits 1989b). There is clearly synergism between BHV1 and Pasteurella haemolytica in the shipping fever complex of adult cattle and feedlot calves (Yates 1982) and Briggs and others (1988) showed that BHV1 infection can alter blood neutrophil function in calves and thereby predispose to secondary bacterial pneumonia. However, Stott and others (1980) found no evidence of BHV1 infection in their survey of respiratory disease on a calf rearing unit in the south of England in the 1970s and Verhoeff and Nieuwstadt (1984) reported only minimal involvement by BHV1 in respiratory disease in calves on dairy farms in Holland compared with respiratory syncytial virus and parainfluenza type 3 (PI3) infections. The general view as expressed by Stott and others (1987) and Kiorpes and others (1988) is that BHV1 is probably not an important cause of calf enzootic pneumonia.

There are few reports in the literature of IBR in young calves (Obi and others 1981) but a systemic form of the disease can occur in neonatal calves infected in utero during late gestation or shortly after birth (Kahrs 1977). Ross and others (1983) described an outbreak in three to four-day-old calves in which pathological changes in the alimentary tract, including ulcerative oesophageal lesions, were present in addition to respiratory tract disease. In a similar incident of fatal systemic infection in very young calves,

Higgins and Edwards (1986) also found ulcerative lesions in the upper alimentary tract as well as focal necrotic liver lesions identical to those described in aborted fetuses by Kendrick (1973). Grieg and others (1981) described marked alimentary tract involvement and ulcerative lesions with diarrhoea in calves with concurrent IBR and bovine virus diarrhoea virus (BVDV) infection. Working with experimentally infected calves, Peter and others (1966) identified BHV1 virus in ulcerative alimentary tract lesions resembling those seen with BVDV infection.

There are numerous reports in the literature of reproductive failure associated with BHV1 infection. According to Wyler and others (1989) up to 25 per cent of pregnant cattle can abort following an outbreak of IBR and Gaines (1989) claimed that an explosive short-term decrease in pregnancy rates can occur in herds immunologically naive to BHV1. Owen and others (1964) and Kendrick (1973) demonstrated that fetal death, with widespead focal necrotic lesions, can follow maternal infection with BHV1 and subsequent viraemia, after a lag phase of up to three months. Abortions may occur following either clinical or non-clinical infection (Kahrs 1977). Experimental infection with field strains of BHV1 or modified live virus IBR vaccines can cause inflammatory lesions in the corpora lutea and ovary leading to reduced progesterone levels, lowered conception rates, early embryonic death and abortion (Miller and Van Der Maaten 1985, Van Der Maaten and others 1985, Miller and Van Der Maaten 1986,

Smith and others 1990, Chiang and others 1990). Miller and others (1991) reported that BHV1 isolates from aborted fetuses belonged to subtypes 1 or 2a but not 2b.

In Britain, Wiseman and others (1980) observed that abortion occurred during or after IBR outbreaks in four of 15 herds and Stubbings and Cameron (1981) reported 15 abortions from a herd of 160 cows. Laboratory surveys in Scotland (Nettleton and others 1981) and Northern England (Murray 1990) suggested that up to about 12 per cent of abortions may have been caused by BHV1 infection. However, a similar study by Lucas and others (1986) at the Central Veterinary Laboratory (CVL), Weybridge showed only minimal involvement of BHV1 in abortion. A more extensive investigation by Edwards (1988) similarly indicated that reproductive failure was only a minor consequence of BHV1 infection in British herds.

As mentioned earlier, some strains of BHV1 can cause the genital tract conditions of IPV and IPB. The clinical features of these were described in detail by Kendrick and others (1958) and Studdert and others (1964). Infection is transmitted venereally causing a painful inflammation by three days after mating; small pustules are present on the mucosal surface of the oedematous vulva or on the penis or prepuce. Temporary infertility can occur due to loss of libido and pain (Gibbs and Rweyemamu 1977). Secondary bacterial infection is uncommon and clinical recovery usually ensues within 10 - 14 days. Concurrent respiratory and genital tract disease has been described (Collings and others 1972) but appears to be very rare.

An encephalitic form of BHV1 infection, first described in calves in Australia by French (1962) has been tentatively attributed to subtype 3 virus (Wyler and others 1989) although Studdert (1990) designated it "bovine encephalitis herpesvirus". The clinical condition, which is manifested by nervous signs including circling, blindness and opisthotonos (George 1991) has not been reported in Britain although Higgins and Edwards (1986) found histological evidence of non-suppurative encephalitis associated with BHV1 infection in a suckler calf. Other diverse and unusual clinical syndromes associated with BHV1 infection (Gibbs and Rweyemamu 1977) appear to be of little significance in Britain.

DIAGNOSIS

Depending on severity, IBR may need to be clinically differentiated from conditions such as infectious bovine keratoconjunctivitis, pneumonic pasteurellosis, bovine malignant catarrh, mucosal disease, calf diphtheria, allergic rhinitis and rinderpest (Gibbs and Rweyemamu 1977, Kahrs 1977, Blood and Radostits 1989a). According to Kahrs (1977), the presence of coalescing white necrotic pustules on respiratory or ocular mucosa are the best aid to clinical diagnosis.

Cowdry type A inclusions (Jubb and others 1985) were described by Crandell and others (1959) in the epithelial cells of the upper respiratory tract of experimentally infected calves but because their

presence is transitory they are generally considered to be of very limited diagnostic value (Wyler and others 1989).

The isolation of virus in primary cell culture is the preferred method of detecting BHV1 in the laboratory (Gibbs and Rweyemamu 1977, Edwards and others 1983) and can readily be achieved by using nasal or ocular cotton wool swabs collected into virus transport medium (VTM) from early clinical cases (Nettleton and others 1983, Nettleton 1986). BHV1 induces a focal cytopathic effect within a few days of inoculation although virus recovery can sometimes take up to 14 days (Nettleton 1986). Other methods of virus antigen detection have been developed to permit more rapid diagnosis. These include immunofluorescence techniques (Terpstra 1979, Nettleton and others 1983), enzyme-linked immunosorbent assay (ELISA) (Edwards and others 1983), reverse passive haemagglutination (Edwards and Gitao 1987) and immunoperoxidase staining using monoclonal antibodies (Rodriquez and others 1989); a polymerase chain reaction test is also available (Belak and others 1988).

Natural BHV1 infection or vaccination with modified live virus vaccines activates humoral and cell-mediated components of the immune system (Kahrs 1977). The latter plays the dominant protective role via the T cell response, whereas direct humoral antibody mediated neutralisation is only functional in cases of reinfection or reexcretion after reactivation of latent virus (described later) (Aguilar-Setien and others 1980, Wyler and others 1989, Straub 1990a). Despite this the humoral response has traditionally served

as the indicator of prior infection. The serum neutralisation test (SNT) using various techniques and standardisation criteria is the most commonly performed method of antibody detection. Neutralising antibody (IgM and IgG) to BHV1 can be detected in the serum at about eight days after natural infection (Gibbs and Rweyemamu 1977) and persists for at least five and a half years (Chow 1972).

Various other serological tests have been developed to overcome problems with the SNT, namely its slowness, dependence on subcultures and interference by non-antibody neutralising factors in some sera (Lyaku and others 1990). The ELISA and a 24-hour SNT method have proved equally sensitive and superior (Edwards and others 1986) to the standard one-hour SNT (Frerichs and others 1982) and to an indirect haemagglutination test (IHAT) (Kirby and others 1974). The ELISA is now used routinely by many diagnostic laboratories and being sensitive, rapid and economic it is ideal for large-scale surveys and has also been used to test bulk milk samples in eradication programmes (Wyler and others 1989, Gibson and Edwards 1991). A system of standardisation has been proposed by Lyaku and others (1990) to overcome problems associated with widely varying techniques which have hindered the adoption of ELISA for international trade purposes.

Colostrally acquired antibody, which is mainly IgG1, can persist until six or seven months in the serum of calves that suck immune mothers (Kahrs 1977, Bitsch 1984) and offers some (but not absolute) protection against severe disease (Aguilar-Setien and others 1980,

Pastoret and others 1984). Maternally derived antibody (MDA) can be distinguished from actively produced antibody by an intradermal test (Forschner 1988, Brown and others 1990, Jungblut and others 1991). This test measures cell mediated immunity via the delayed-type hypersensitivity reaction (Brown and others 1990) in animals that have experienced BHV1 infection and provides a potential means of rapidly evaluating infected herds in the field. However, repeated intradermal injections of BHV1 antigen can induce a serological response in seronegative cattle (Thiry and others 1992).

TREATMENT AND VACCINATION

The use of broad-spectrum antibiotics, in addition to nursing care and supportive therapy (Kahrs 1977) is invariably recommended to control secondary bacterial infection during IBR outbreaks (Blood and Radostits 1989a). Wiseman (1984) advocated antibiotic therapy for up to 12 days, until the temperature of affected animals came down to below 103°C.F

BHV1 vaccines with either live modified or inactivated virus and either mono or polyvalent, have been in commercial use in many countries for the past 30 years. In Britain, Wiseman and others (1979) expressed concern over the limited options then available to veterinary practitioners faced with severe outbreaks of IBR. Two modified live virus vaccines: "Tracherine" (Smith Kline Animal Health Limited) and "Nasalgen" (Wellcome Foundation Limited) were subsequently licensed for use in Britain (Frerichs and others 1982).

These vaccines were developed for intranasal administration (McKercher and Crenshaw 1971) and were free of the abortifacient properties of other live IBR vaccines. They proved to be superior (Frerichs and others 1982) to the inactivated adjuvanted polyvalent vaccine "Pneumovac Plus" (C-Vet Limited) that had previously been the only biological product available in Britain for protection against IBR. The immunogenicity of the latter vaccine was later enhanced (Straub and Mawhinney 1988). Inactivated vaccines are considered to be only suited for prophylactic purposes (Straub 1990a); their use to control virus shedding by latently infected cattle is described later. In contrast, the live vaccines effectively prevent further spread and reduce losses when used during an outbreak (Wiseman 1984, Peters 1987, Van Donkersgoed and Babiuk 1991) by the prompt production of interferon and secretory antibodies (IgA) on mucosal surfaces (Wyler and others 1989, Straub 1990a).

PATHOGENESIS AND LATENCY

BHV1 enters the body via the mucous membranes of the upper respiratory tract or conjunctiva (or genital tract with IPV/IPB); oral infection has not been confirmed (Straub 1990a). After experimental nasal infection the virus can be isolated from various tissues including respiratory tract, eye, brain and intestines (McKercher and others 1963). Simultaneous infection of genital and respiratory tracts is very uncommon (Gibbs and Rweyemamu 1977, Straub 1990a). Viraemia has rarely been reported and its role has not been critically evaluated (Yates 1982, Straub 1990a): it is

probably weak and transient because of the small number of infected leukocytes in the circulation (Peter and others 1966) but it provides the apparent explanation for fetal or systemic neonatal infection (Pastoret and others 1984).

After entering the neural cell at the nerve endings by fusion, naked nucleocapsids are probably transported within the axon by retrograde flow mainly to the trigeminal ganglia (respiratory tract infection) or sacral ganglia (genital tract infection) where they establish latent infection (Narita and others 1981, Ackermann and others 1982). Wyler and others (1989) defined latency as "the silent persistence of viruses in the body not detectable by conventional virological procedures, with subsequent intermittent periods of excretion". The ability of all BHV1 strains, including attenuated live virus vaccines (Kahrs 1977, Yates 1982, Nettleton and others 1984) to establish latent infection was first evident from experimental studies by McKercher and others (1963) and Snowdon (1965). In view of this phenomenon, it is generally accepted (Kahrs 1977) that any animal with measurable humoral antibody is a virus carrier and potential source of infection for susceptible herd mates, probably for life (Bitsch 1973, Bitsch 1984) - a concept that forms the basis of eradication programmes for BHV1 (Ackermann and others 1990a). Furthermore, as pointed out by Kennedy (1973) and Nettleton (1986) it also means that recovery of virus does not constitute proof of primary infection.

REACTIVATION OF LATENT INFECTION

The demonstration by Sheffey and Rodman (1973) that experimentally infected, clinically recovered cattle reexcreted BHV1 after several days administration of high doses of corticosteroid (40 mgs dexamethasone) was a major advance in the understanding of BHV1 latency. It provided scope for numerous further experimental reactivation studies in which reexcretion from the respiratory or genital tracts was demonstrated following natural infection or live virus vaccination (Darcel and Darwood 1975, Nettleton and Sharp 1980, Pastoret and others 1984).

The amount of virus excreted at reactivation is much less than during primary infection but appears to have unaltered biological and molecular properties (Rodriquez and others 1984, Pastoret and others 1984) although there may be some genome variability (Whetstone and others 1989). Narita and others (1981) described degenerative changes in the trigeminal ganglia following each of two dexamethasone-induced recrudescences three months apart and concluded that cattle can excrete virus several times during their lives. Miller and others (1987) similarly used dexamethasone to demonstrate the presence of BHV1 in the ovary of latently infected cattle thereby indicating that embryonic death could develop following reactivation.

Reactivation of latent virus may cause reexposure to antigen leading to an increase in humoral antibody, which partly explains the long-term persistence of BHV1 titres in infected cattle (Bitsch 1973,

Kahrs 1977). Pastoret and others (1984) suggested that sufficiently immunised animals can control their rate of virus reexcretion and dissemination and that reactivation may serve as a booster to the immune system. Despite this, Straub (1990a) noted that corticosteroid treatment of latently infected cattle did not necessarily produce an increase in SNT titre. The mechanism of dexamethasone-induced BHV1 reactivation is not known. It appears that immunosuppression itself is not directly responsible (Davies and Carmichael 1973) and that dexamethasone may have a direct effect on virus-neuron interaction (Rock and others 1992).

Naturally occurring events associated with viral reactivation are poorly understood although it is generally accepted that it occurs as the result of increases in endogenous corticosteroids following stress factors such as transport, parturition, intercurrent disease, dehorning, castration and overcrowding (Nettleton and Sharp 1980, Guy and Potgieter 1985, Rock and others 1992). Thiry and others (1987) stimulated reactivation experimentally using transportation as did Msolla and others (1983) by challenging latently infected bullocks with lungworm larvae; Mensik and others (1986) achieved the same effect with PI3 virus. Espinasse and others (1983) reactivated latent BHV1 using intraruminal 3-methylindole, shown previously by Carlson and others (1975) to be the causal agent of fog fever. Edwards and Roeder (1983) failed to reactivate latent BHV1 in calves with experimental immunosuppression by ruminant pestiviruses. Parturition was shown by Thiry and others (1985) to induce BHV1 reactivation but they considered it was a rare event and suggested

that the stress of labour rather than the physiological status itself was responsible (Thiry and others 1984).

There are reports in the literature (Gibbs and Rweyemamu 1977) of clinical recrudescence at the time of reactivation although this is not usually as severe as expected with primary infection (Kahrs 1977) and is generally considered to be insignificant (Straub 1990a), albeit subject to specific immune status (Pastoret and others 1984, Ludwig 1984). However, a watery or mucopurulent nasal discharge, conjunctivitis and ulceration of the nares and laryngeal mucosa have been described following corticosteroid-induced reactivation (Davies and Carmichael 1973, Davies and Duncan 1974, Narita and others 1981) and Msolla and others (1983) described typical signs and lesions of IBR in latently infected bullocks subjected to a heavy challenge with lungworm larvae. Observations from the field (Hyland and others 1973, Van Nieuwstadt and Verhoeff 1983, Corkish and Richards 1983) have revealed mild apparently recrudescent clinical disease in closed endemically infected breeding herds, the sources being attributable to reactivation of latent infection (Kahrs 1977). Donkersgoed and Babiuk (1991) similarly remarked that IBR seen in feedlot calves that had been on the unit for some time was the result of reactivation and transmission of virus from latently infected pen mates.

TRANSMISSION AND CIRCULATION

Many authors have observed that IBR outbreaks are usually preceded by the recent introduction of new cattle into a herd (Gibbs and

Rweyemamu 1977, Wiseman and others 1980, Blood and Radostits 1989a). Karge (1989) considered that BHV1 is mainly transmitted from herd to herd and imported into disease-free countries by latently infected cattle - a view shared by most other authors. Cattle that have become infected without showing clinical signs and which are still shedding virus (up to 10 - 16 days following experimental infection according to Straub 1991) and those with mild clinical signs that are attributed to factors such as transportation stress, are other potential sources of infection (Straub 1990a), as are calves born alive after surviving late gestational infection (Pospisil and others 1979). Spread of infection from live virus vaccinates to susceptible close contacts has also been reported (McKercher and Crenshaw 1971, Zygraich and others 1974) although it may be fairly limited in extent (Baker and others 1989).

Non-bovine mammalian species such as rabbits (Lupton and others 1980), skunks and hamsters (Straub 1990a) can be experimentally infected with BHV1. Serum neutralising antibodies to BHV1 have been found in a variety of ruminant species, although there is considerable cross-reactivity with other antigenically related herpesviruses including those of cervidae and caprines (Nixon and others 1988). The virus has also been isolated from the soft shelled tick Ornithodorus coriaceus (Taylor and others 1982). The role of these various species as carriers or reservoir hosts is unclear although Straub (1990b) listed direct or indirect contact with virus-shedding wild animals as a possible source of BHV1 infection for cattle. Collings and others (1972) speculated about the

possibility of BHV1 being transmitted by the stable fly (Stomoxys calcitrans) but Johnson and others (1991) found no experimental evidence to indicate that it occurred with the face fly (Musca autumnalis). Whilst aerosol transmission is undoubtedly important over short distances, particularly under intensive housing conditions (Elazhary and Darbyshire 1979, Yates 1982) there are no reports of the long distance airborne spread seen with ADV (Gloster and others 1984).

It has long been accepted that semen from bulls actively or latently infected with BHV1 provides an important potential source of infection either through natural service (Huck and others 1971) or via AI (Drew and others 1987). This led to the adoption of strict control measures on AI studs (Lucas 1986). Despite such precautions, BHV1 was still introduced into a herd in Switzerland by imported semen (Kupferschmied and others 1986). Straub (1990a) examined the possible role of embryo-transfer (ET) in the transmission of BHV1 and concluded that no danger existed: although the virus adheres to the zona pellucida it is readily removed by washing and trypsin treatment (Singh and others 1983, Stringfellow and others 1990).

It has been proposed (Gibbs and Rweyemamu 1977) that virus perpetuation results from an interplay between short cycles of acute disease with high levels of virus excretion and the prolonged reexcretion of reactivated latent virus. Calf rearing and fattening units appear to play an important part in the maintenance of BHV1 by virtue of the regular introduction of young susceptible calves from

heterogenous herds of origin and extensive opportunities for stress induced reactivation and close contact. The onset of new cases in susceptible populations can be gradual, particularly in large herds being continually replenished and where cow-to-cow contact is minimal (Kahrs 1977). Ferris and others (1964) observed that new cases continued to occur for 10 to 20 days in herds of less than 100 cattle kept in close contact. In what appears to be the only detailed long-term study of BHV1 circulation in infected herds reported in the literature, Van Nieuwstadt and Verhoeff (1983) found no serological evidence of virus circulation for at least three years in eight of 19 infected dairy herds in Holland.

ANTIBODY PREVALENCE SURVEYS

Numerous surveys have been carried out to examine the prevalence of BHV1 antibody as an indicator of natural exposure or vaccination in the cattle population. The findings from 35 such surveys performed worldwide since 1975 were summarised by Straub (1990a): 14 of the 15 largest surveys (>1000 sera) showed antibody prevalences of between about one and 37 per cent.

In Britain, Dawson and Darbyshire (1964) examined 2000 sera collected from abattoirs and suspected cases of mucosal disease and found 2.1 per cent contained BHV1 antibodies. They recorded serological evidence of infection in 16 of 42 counties in England and Wales. In 1974, Kirby and others (1978) found that 6.8 per cent of 2,368 sera submitted to Reading Veterinary Investigation Centre (VIC) for

brucellosis testing from herds in Buckinghamshire, Berkshire and Oxfordshire, were seropositive for BHV1. Subsequent surveys in Britain demonstrated increasing antibody prevalences: Peters and Perry (1983) found 11.4 per cent reactors in young pedigree beef bulls held at two Meat and Livestock Commission bull performance testing centres between 1979 and 1981; Msolla and others (1981) recorded a seroprevalence of 12.1 per cent from a large survey of dairy herds in Scotland.

Edwards (1988) undertook a retrospective analysis of serological data held at the CVL for the period 1970 to 1986. He reported that BHV1 seroprevalence amongst healthy cattle tested for export or entry into AI centres increased from less than five per cent in the early 1970s to 10 - 12 per cent by the mid-1980s; the proportion of seropositive diagnostic submissions increased from 9.1 per cent in 1969 - 1974 to 34.7 per cent for 1984 - 1986. In a more recent survey, Hogg (1992) found a seroprevalence of 32.4 per cent amongst cattle aged four to 12 months in 503 herds in Britain with a history of respiratory disease.

Detailed information on age-specific prevalence patterns for BHV1 infection is generally lacking from the literature. However, Kirby and others (1978) noted a higher proportion of seropositive cattle in older age groups (≥five years) compared with those aged two years or less and Forschner (1988) also commented that seroprevalence increased with age. Msolla and others (1981) found more reactors

amongst dairy cows than heifers in Strathclyde but not in the Grampian region of Scotland.

Forschner (1988) noted that fewer than 20 per cent of animals were infected in more than 70 per cent of diseased herds in Germany. In their serological study in Spain, Espuna and others (1988) found that seroprevalence exceeded 30 per cent in 87 per cent of infected herds although 46 per cent of herds contained no reactors. Edwards (1988) reported that 48 per cent of herds in England and Wales contained at least one seroreactor.

CONTROL AND ERADICATION

Within the European community (EC) IBR is classified in Group 3 (except on AI and ET units where it is subject to mandatory control as a Group 2 disease) (Prince 1990). Category 3 comprises endemic diseases intended to be controlled by voluntary attestation programmes which member states should aim to harmonise with each other (Gibson 1991). Although potent vaccines are available to prevent economic losses from IBR, their widespread adoption in countries such as the USA has caused problems with the export of live cattle, semen and embryos to parts of the world demanding BHV1-seronegative status (Wyler and others 1989, Straub 1990b). Despite potential drawbacks suggested by Kahrs (1977), elimination of BHV1 infection from herds or regions presents a viable alternative to vaccination (Bradley 1985, Blood and Radostits 1989a). Bitsch (1985) commented that attempts to eliminate BHV1 should have a good chance

of success if infected animals can be separated from uninfected cattle and precautions taken to prevent indirect transmission.

There are a few detailed reports in the literature of successful schemes for eliminating BHV1 infection from individual herds. Bitsch (1984) and Bradley (1985), both working with heavily infected beef suckler herds, built-up uninfected populations by rearing seronegative calves separately from reactors. Corkish (1988) similarly divided an infected suckler herd into seropositive and seronegative groups: purchases were required to be seronegative for BHV1 when tested after a month in isolation, before being allowed into the negative group. Bradley (1985) observed that calves born to cows previously infected with virulent BHV1 became seronegative following maternal passive antibody decay and if kept isolated could preserve their eligibility for entry into AI studs or for export. Ackermann and others (1990b) failed to stop BHV1 transmission by preventing direct contact between seropositive and seronegative groups of calves on a large fattening unit although they subsequently eliminated the infection when separation was improved (including the use of a plastic curtain) and stricter hygiene measures were adopted to prevent indirect contact.

In addition to control methods based on segregation of reactors and testing of replacements, there has been much interest in the last five or six years, particularly in Germany, in the use of inactivated vaccines to boost the immunity of latently infected carriers to reduce their viral excretion and thereby prevent spread to uninfected

herd members. According to Forschner (1988) virus shedding is suppressed if humoral antibody titres can be maintained at a high level by repeated vaccination (usually at six monthly intervals). This approach is considered to be of particular value in herds where slaughter of all reactors would be prohibitively expensive (Wittkowski 1990).

In the initial trials in a large herd of bulls, Meyer and others (1985) reported increased serological titres for four to five months after vaccination and a significantly reduced duration of virus excretion following immunosuppressive stress. In subsequent field trials in Germany performed over more than three years, Forschner and others (1987) claimed that BHV1 infection ceased to spread in 36 of 38 herds following primary immunisation and repeated revaccination of reactors. Gossger (1988) reported similar findings in a smaller study over an 18 month period and questioned the optimum time interval between revaccinations in relation to cost. Although the strategic use of inactivated BHV1 vaccine is now widely adopted in Germany (particularly Bavaria), Gibson and Edwards (1991) found very little scientific evidence to confirm that vaccination actually suppressed virus shedding and recent experiments by Straub (1991) have shown no correlation between humoral antibody titres and virus shedding which casts doubt on conclusions from earlier studies.

In Switzerland (Ackermann and others 1990c) and Denmark (Report 1985), IBR has been made compulsorily notifiable and successful eradication programmes based on slaughter of seropositive animals,

with compensation, have resulted in the virtual elimination of BHV1 infection from their national herds (Ackermann and others 1990c, Edwards 1991). Elsewhere in Europe, BHV1 infection is variously managed using combined vaccination/eradication programmes depending on disease significance and governmental strategy (Wyler and others 1989). Straub (1991) made a distinction between "eradication" (freedom from BHV1 in the whole cattle population of a country or state without using vaccine) and "sanitation" (elimination of BHV1 from a herd or group of organised breeders using vaccine if appropriate).

Well developed voluntary programmes in both France and Germany rely on an initial bulk milk ELISA test to detect infected dairy herds, combined with individual blood testing to identify seroreactors (Gibson and Edwards 1991). Inactivated vaccine, as discussed above, is used when reactors are retained: in Germany, reactors only are vaccinated but in France the entire age cohort containing reactors is vaccinated. In these countries herds containing only a few seropositive cattle can achieve BHV1-negative status following a series of herd blood tests, with removal of reactors, combined with control measures to prevent reinfection. A similar scheme (Agricultural Development and Advisory Service [ADAS] 1987) was introduced into Britain in 1987 (Penton 1987) and is featured in this study.

CHAPTER 2: THE STUDY AREA AND ITS CATTLE POPULATION

The underlying reference population applicable throughout this dissertation comprised cattle herds maintained in the county of Norfolk, England, within the period 1980 - 1991. In view of its numerically small size, in an area dominated by arable farming, this account of cattle production in Norfolk was considered essential to allow the findings to be seen in context with the rest of the British cattle industry.

GENERAL CONSIDERATIONS

Norfolk (Fig 2.1) is bounded by the Wash and the Cambridgeshire Fens to the west and the North Sea to the north and east; it is separated from Suffolk to the south by the River Waveney (Fig 2.2). The adjoining counties of Norfolk and Suffolk constitute the region of Britain known as East Anglia.

Although basically flat, the Norfolk landscape gently undulates up to 300 feet above sea level. It is a county of low average annual rainfall and extremes of ambient temperature; the topography, climatic conditions and abundance of high quality agricultural land have favoured arable production (Fig 2.3) for centuries (Ministry of Agriculture, Fisheries and Food [MAFF] 1981a).

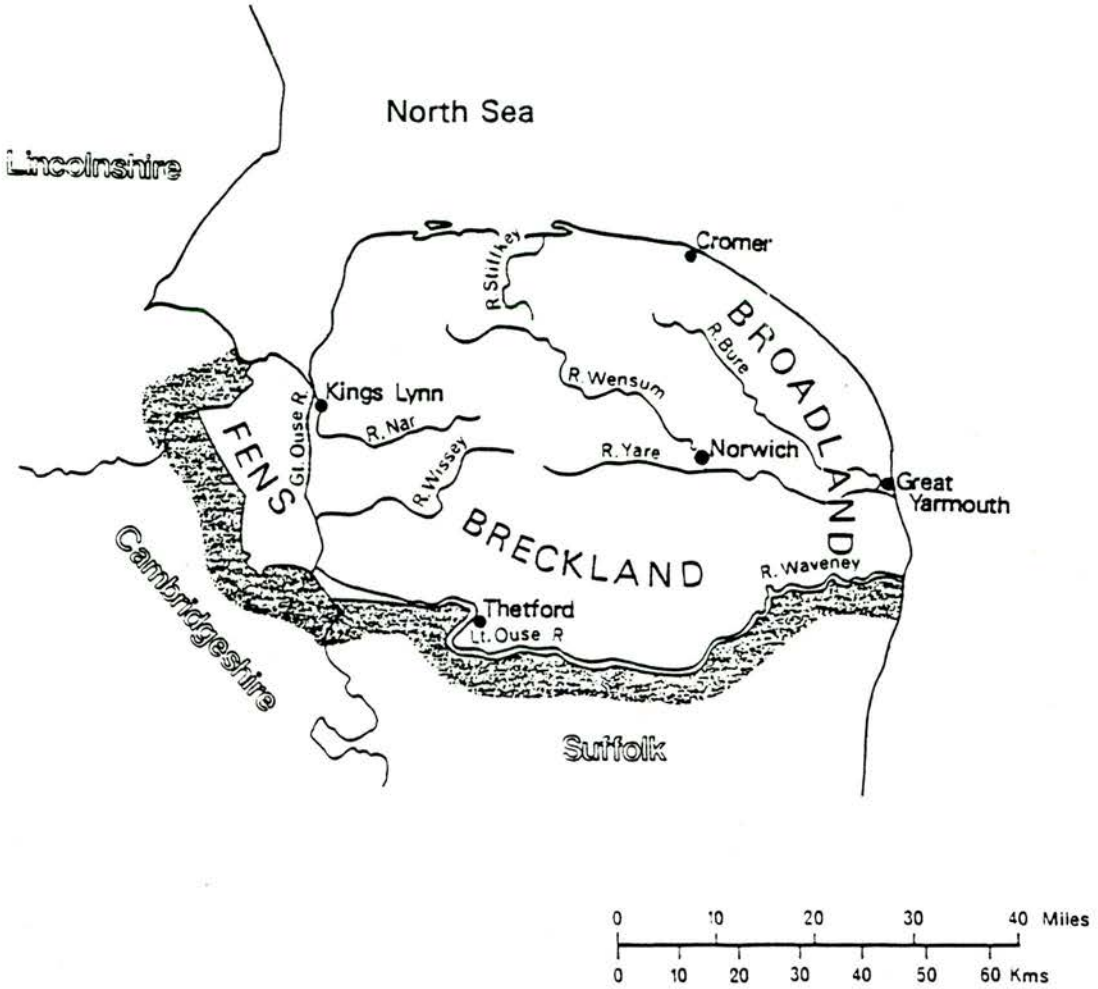

Fig 2.1: Map of Norfolk showing county boundaries, principal rivers, towns and sub-regions.

(Adapted from MAFF 1981a)

Poultry and pigs dominate livestock farming: over 30 per cent of turkey production and eight per cent of pig production in England and Wales is based in Norfolk (MAFF 1990a). Although the sheep population is less than 0.5 per cent of the total for England and Wales (MAFF 1990a), numbers continue to increase and there are now several very large flocks in the Breckland area (Fig 2.1).

Fig 2.2 : Aerial photograph of the Waveney valley grazing area of Norfolk

(By courtesy of the Aerial Photography Unit, MAFF Cambridge)

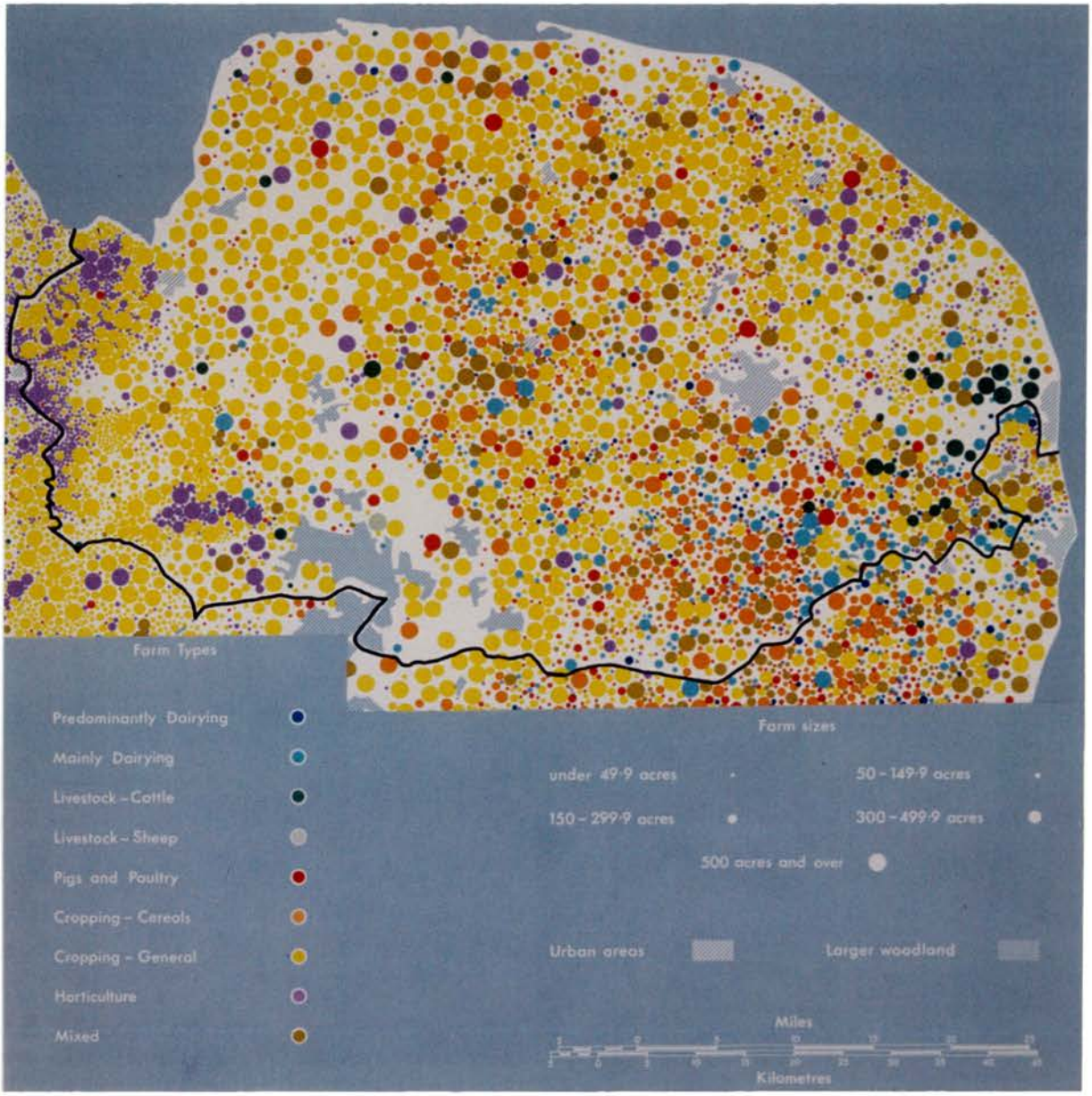

Fig 2.3 : Distribution of main agricultural enterprises in Norfolk

(By courtesy of ADAS, Norwich)

Fig 2.4 : Cattle grazing marshland near Great Yarmouth, Norfolk

SEASONAL MARSHLAND GRAZING

Seasonal marshland grazing (Fig 2.4), mainly centred around Broadland (Fig 2.1), is an integral part of traditional cattle farming practice in the region. Broadland is a popular holiday area which extends into north-east Suffolk; it contains tidal rivers, river valleys and broads (shallow lakes originating from 15th century peat diggings). Most of the farmed area of Broadland is permanent pasture maintained at, or below, sea level by a drainage system which pumps water up from a series of dykes into the rivers.

Marshland graziers can be broadly divided into two categories (Report 1987). The first of these, the owner-occupiers, usually with dairy herds, are mainly located up the river valleys towards the source. Their marshes tend to be more secluded and have less potential contact with other herds than elsewhere. Further down the valleys, most grassland is rented as annual grazing lets of between about 10 and 30 acres, traditionally sold by annual public auction in the spring and available for use only until the end of October of each year.

Although cattle are separated by dykes, several metres wide, for many herds the marshes represent a significant potential source for acquiring new infections (Pritchard and others 1989). About 400 different (mainly local) cattle producers (including dealers) regularly used rented marshes during the 1980's to supplement home

grazing, particularly for replacement dairy heifers, dry cows and cross-bred beef calves (G A Pritchard, personal communication). There was an increasing trend to introduce sheep onto the marshes as the decade progressed.

CATTLE PRODUCTION

Despite its relatively small size, the cattle industry in Norfolk is important and highly efficient. In June 1989, the total cattle population of Norfolk was 112,615 which represented 1.3 per cent of the total number of cattle in England and Wales (MAFF 1990a). This compared with 150,250 cattle in June 1980, which was 1.6 per cent of the England and Wales total (MAFF 1981b).

Dairy herds

The dairy industry in Norfolk has steadily declined since the 1960s accelerated by beef conversion schemes, the introduction of milk quota and increased cereal production. In 1989, the number of dairy producers registered under the Milk and Dairies (General) Regulations 1959, fell to 249 herds compared with 311 in 1985 and 409 in 1980. Despite this decline, dairying remained the major grass utilising enterprise in Norfolk during the 1980s, based mainly on unploughable pasture in low lying areas such as the Waveney valley of south Norfolk (Fig 2.2).

Whilst the total number of dairy cows in the county fell during the decade, herd structure did not change significantly (Table 2.1). About 20 per cent of herds contained less than 10 cows (mainly house-cows) and over 50 per cent of herds contained more than 50 cows. The average size of registered dairy herds in 1989 was about 100 cows. The majority of dairy herds were in long established large mixed enterprises; the remainder were mostly small family-owned farms. Holstein/Friesian was by far the predominant breed; most herds claimed pedigree status.

TABLE 2.1 : Dairy cow population trends in Norfolk 1980 - 89

Year	Herd size (no of cows)				Total no of cows	% E&W total
	All	≥10	≥50	≥100		
1989						
No herds	313	250	178	84	22,777	0.97
% total	100	79.8	56.9	26.8		
1985						
No herds	404	313	218	102	27,437	1.06
% total	100	77.4	54.0	25.2		
1980						
No herds	523	417	282	121	34,275	1.28
% total	100	79.7	53.9	23.1		

% E&W total : Percentage of total cattle population of England and Wales.

(Adapted from Agricultural Census returns: MAFF 1981b, 1986, 1990a)

Breeding was invariably by AI, often supplemented with a "sweeper" bull; the more progressive herds utilised ET to increase the genetic potential of their best cows. Herds calved mainly during the autumn. Winter housing was generally in strawed yards although many herds also used cubicles. Supplementary and winter feeding was based mainly on arable by-products, particularly sugar beet tops, brewers grain and straw, fed in conjunction with grass silage.

There was generally little or no contact between the adult milking herd and calves reared as breeding replacements. After removal from their dams soon after birth, calves were usually housed separately from the main herd during the winter and spent two years on marsh and, or, sheltered upland grazing before returning to their parent herds to calve at between two and three years of age.

Disease security precautions adopted on most dairy farms were minimal although large blocks of arable land (Fig 2.2) invariably separated herds and prevented direct contact (apart from at communal marshland grazing).

Suckler herds

Total beef suckler cow numbers remained fairly static during the 1980s (Table 2.2) but there was a slight upturn towards the end of the decade attributable to the introduction of the Environmentally Sensitive Areas (The Broads) Designation Order 1986, which offered financial incentives to encourage traditional livestock grazing on

the marshes, and the introduction of the High Mowthorpe system of suckled calf production (ADAS 1988) which proved to be well suited to the rough grazing areas of Norfolk (G Thwaites, personal communication).

TABLE 2.2 : Suckler cow population trends in Norfolk 1980 - 89

Year	Herd size (no of cows)				Total no of cows	% E&W total
	All	≥10	≥50	≥100		
1989						
No herds	695	322	79	Not listed*	13,918	1.72
% total	100	46.3	11.4			
1985						
No herds	695	303	70	20	13,235	1.93
% total	100	43.6	10.1	2.9		
1980						
No herds	757	337	91	23	14,983	1.96
% total	100	44.5	12.0	3.0		

* For disclosure reasons

% E & W total : Percentage of total cattle population of England and Wales.

(Adapted from Agricultural Census returns: MAFF 1981b, 1986, 1990a)

Suckler herds were largely established on farms with some permanent grassland, and arable by-products and home grown fodder available for winter feeding. A variety of different husbandry and management

practices were adopted and many herds were small part-time enterprises. The preferred period for calving was between November and April but some herds calved virtually all the year round.

Most herds were solely commercial, based on Hereford X Friesian or Limousin X Friesian cows, with Charolais, Limousin or Simmental bulls. These produced cross-bred calves for finishing on cereals. An estimated 20 per cent of suckler herds were either entirely pedigree or included a small pedigree herd managed alongside the commercial herd. Although a few indigenous breeds were represented, the majority of pedigree herds were made up of continental breeds. There was increasing interest during the decade in the importation of more exotic breeds such as Piedmontese or Salers; owners frequently used ET to capitalise on the high prices available for the calves.

Precautions against the introduction of disease in suckler herds were invariably poor although they were improved by the presence of large expanses of arable land and long approach roads. Disease security in commercial suckler herds was particularly bad because they usually replaced some 20 - 25 per cent of cows each year with heifers obtained from a variety of sources of unknown disease status (Pritchard and Bardsley 1990). In pedigree herds using ET, recipients were often purchased from markets, thereby further increasing the risk of disease introduction.

Calf rearing and fattening herds

Most surplus calves born to local dairy and suckler herds remained in the county. Some breeders reared their own surplus calves to 10 - 14 weeks of age for subsequent sale to barley beef units; others fattened them through to finishing weight.

Several farming companies and cooperatives ran contract rearing and fattening schemes. These collected calves for local redistribution or obtained calves (via dealers) from outside the area, particularly from the West Country. The numbers of calf rearing units in Norfolk fluctuated widely depending on cost of concentrate feed, the export market and viability of alternative enterprises. Precise information on the size of the industry was not available from census returns. Most rearing units averaged about 50-60 calf places and reared three batches of calves a year, often as a part-time business using a wide variety of husbandry and feeding systems.

Because of concern regarding infectious diseases, such as salmonellosis, hygiene measures were often better than in breeding herds. However, since more than 60 per cent of calves came from outside the county (E Randall, personal communication), calf rearers remained continually at risk of introducing new disease.

CHAPTER 3: CLINICAL DISEASE OUTBREAKS

IBR was first convincingly recorded in Norfolk during the winter of 1979 when four separate outbreaks were confirmed by Norwich VIC amongst recent imports of fattening cattle into the county from store markets. This was during a period of increased disease surveillance by the Veterinary Investigation Service (VIS) and heightened awareness by veterinary practitioners and cattle producers. The subsequent occurrence and main clinical and epidemiological features of confirmed outbreaks of IBR in Norfolk during the 1980s are reviewed in this chapter.

MATERIALS AND METHODS

Sources of data

During the period covered by this study the author was engaged as a veterinary investigation officer at Norwich VIC with a particular responsibility for ruminant diseases. Norwich VIC was established in 1958 as part of a national network of veterinary laboratories operated by the State Veterinary Service (SVS). Amongst its activities, the Centre provided a diagnostic and advisory service for practising veterinary surgeons in Norfolk and maintained local surveillance of disease trends in farmed livestock with particular reference to new and emerging conditions. The data used for the studies described in this chapter were obtained from several sources as detailed below.

Laboratory diagnostic submissions

Using standard laboratory questionnaire forms (Appendix 3.1), veterinary practitioners submitted a variety of specimens from cattle herds in Norfolk to the VIC for diagnostic tests for IBR. In addition to the identity of the affected herd, these submission forms included details of the age and clinical history of the stock involved.

On-farm investigations

Some IBR incidents, particularly those of an unusual or complex nature, were investigated on the farm by the author in conjunction with the owner's veterinary surgeon. Specimens collected at these farm visits were submitted to the VIC for appropriate examinations. Detailed reports were prepared.

Telephone consultations

Veterinary practitioners frequently sought telephone consultations to discuss IBR outbreaks. Details were usually recorded and these provided an important, albeit anecdotal source of intelligence data concerning disease trends and the efficacy of treatment and control measures.

Statutory abortion investigations

Under the Brucellosis (England and Wales) Order 1981 (as amended), cattle owners were required to notify the Divisional Veterinary Officer of all abortions that took place less than 271 days after insemination. Specimens from the aborted cow, including a clotted blood sample, were submitted to the VIC by the farmer's veterinary surgeon to test for Brucella abortus, accompanied by a report form (Appendix 3.2) which provided identification details, age and breed. Practitioners frequently requested further tests, including serological examinations for BHV1, to be undertaken as part of the differential diagnosis to establish the cause of the abortion.

Farm-file records

Detailed records of disease history were maintained in individual herd files kept at the VIC. These included records of telephone consultations and results of all diagnostic tests, including statutory abortion submissions and formed a convenient source of data for measuring the occurrence of IBR and the serological status of individual herds.

Monthly Disease Reports

An internal report was prepared at the VIC giving brief clinical and epidemiological details relating to the main diagnoses achieved each month. These reports, which also provided the basic information for

the account of VIS activities featured in the Veterinary Record, contributed a useful recorded summary of local disease trends during the study period.

Veterinary Investigation Diagnosis Analysis II

Specimens received at VICs provided the basis of a computerised diagnostic data bank known as Veterinary Investigation Diagnosis Analysis II (VIDA II) (Hall and others 1980). IBR was not specifically recorded by VIDA II until 1980 when confirmed incidents were listed either under group 1 "Systemic diseases of cattle and those not readily classified organically" (code 213) or under group 9 "Diseases of the reproductive and mammary system of cattle" (code 039) which included fetopathies attributed to BHV1 infection. Repeat diagnoses on further specimens from the same incident were excluded.

Laboratory diagnosis

Techniques employed

Diagnostic procedures used during these studies followed standard methods adopted by the VIS (MAFF 1984). It was outwith the scope of this dissertation to provide a detailed account of virological techniques employed in the laboratory to confirm BHV1 infection: these procedures will therefore be described in outline only.

Virus isolation was undertaken routinely at Norwich VIC between 1980 and 1986, using primary bovine monolayer culture (kidney or testis) supplied by the Virology Department of the CVL. After 1986, as part of a rationalisation programme, samples were sent to other VICs for virus isolation. A fluorescent antibody test (FAT) developed at the CVL (Edwards and others 1983) was used at Norwich VIC after 1980 to detect BHV1 antigen in nasal, naso-pharyngeal or conjunctival swabs or lung tissue. The detection of Cowdry type A intranuclear inclusion bodies in bronchial or alveolar epithelium (Jubb and others 1985) was occasionally used for disease confirmation until 1982 but this histological approach was discontinued once virus isolation and FAT techniques were fully established in routine use.

Sera from clotted blood samples were dispatched to other VICs or the CVL to test for BHV1 antibodies. Between 1980 and 1982, most sera were examined with the IHAT (Kirby and others 1974). This test was subsequently discontinued by the VIS in favour of the one hour serum neutralisation test (SNT) (Frerichs and others 1982) which was used routinely for diagnostic submissions until 1985. The more sensitive 24 hour SNT was occasionally used as a decisive test for samples with an apparently low antibody level (Edwards and others 1986). After 1985, the SNT was largely superseded for routine diagnostic use by a simpler ELISA technique (Edwards and others 1986). In accordance with standard guidelines used in the VIS, antibody titres for BHV1 of $\geq$1/8 with the IHAT were considered "positive" as were titres of $\geq$1/2 with the SNT and optical densities (ODs) of $\geq$0.1 with ELISA.

Specimen collection

The choice of diagnostic material rested with veterinary practitioners. They were requested to provide fresh specimens (ocular, nasal or naso-pharyngeal swabs, viscera or carcases) from recent cases for virological examinations or to submit "paired" blood samples (taken at the acute and convalescent stage of infection, two to four weeks apart) if serological diagnosis was required. For the FAT they were advised preferably to use naso-pharyngeal swabs - either long guarded "brush" swabs (Thomas and Stott 1975) supplied by the CVL or human laryngeal swabs (Medical Wire Company) for sampling, depending on the age of animal, and to submit the swabs to the VIC in VTM (Tryptose phosphate broth with antibiotics) if possible.

Criteria for laboratory confirmation

Apart from the occasional use of histology for laboratory confirmation in 1980/81, a diagnosis of IBR was reached only if BHV1 was isolated in tissue culture or detected by FAT in specimens submitted from suspected clinical cases, or if seroconversion (a change in titre from negative to positive) or a significant increase in titre (four-fold increase in SNT or IHAT titre or an increase in OD of $\geq$0.2 with the ELISA) was demonstrated with paired samples. BHV1 fetopathy was confirmed by virus isolation in primary cell cultures using pooled fresh fetal tissue or with paired serology from aborting dams (Pritchard 1990). A positive antibody titre or OD

value for BHV1 was taken to indicate either exposure to field infection, vaccination or, in the case of calves up to about six or seven months old (Kahrs 1977, Bitsch 1984) the possible presence of MDA. High titres for BHV1 can be maintained for long periods of time (Chow 1972, Bitsch 1984): single serological titres were therefore not used for disease diagnosis because they did not provide any reliable information concerning the timing of the incident. Single-sample serology was however, widely used for antibody screening purposes, as described in subsequent chapters.

Extraction, collation and analysis of data

The terms "incident" and "outbreak" were used synonymously to describe an identified occurrence of disease involving one or more animals (Thrusfield 1986a). Details of confirmed IBR incidents in Norfolk between January 1980 and December 1989 were obtained from a systematic manual search of farm-file archives, monthly reports and records of on-farm investigations and telephone consultations. The main clinical and epidemiological features of each incident were recorded using a card-index system.

Using the approach adopted by VIDA II, outbreaks were broadly classified as either systemic disease or fetopathy. Systemic outbreaks in breeding herds were further subdivided on the basis of clinical history (supplemented if necessary by further consultation with the veterinary surgeon involved) into either epidemic disease with high morbidity in fully susceptible herds or clinically mild low

morbidity recrudescences (Gibbs and Rweyemamu 1977, Kahrs 1977).

The findings were tabulated according to date of incident (month/year of commencement or of laboratory submission if not known), herd identity, herd type (dairy, beef suckler, or "fattener" ie calf rearing/fattening unit), age of affected stock and clinical definition of the incident (epidemic, recrudescence, fetopathy). Monthly disease occurrence was compared with VIDA II data (MAFF 1990b) supplied by the Epidemiology Unit, CVL. Reasons for any discrepancies between archival data and VIDA II were noted where possible. Laboratory methods used to confirm IBR outbreaks were recorded by year to show trends in diagnostic methodology.

RESULTS

Laboratory diagnosis

Although practitioners were advised on the suitability of specimens for IBR diagnosis, the quality of material submitted to the laboratory depended on numerous factors: the client, the stage and severity of outbreaks, potential economic losses, expertise and experience of the practitioner, availability of sampling equipment and distance from the laboratory. Submissions came from more than 300 different herds and varied from a single nasal swab or clotted blood sample to several pieces of fresh or decomposing viscera or entire carcases. Some swabs had clearly been left for several days to dry out in the veterinary surgeon's car; others were almost

completely lacking in mucosal epithelial cells because of poor sampling technique.

Eighty-three systemic IBR incidents were recorded in Norfolk between 1980 and 1989. Most were confirmed either by paired serology (39.8 per cent) or the FAT alone (32.4 per cent). Table 3.1 shows the clear trend away from virus isolation in tissue culture to the FAT as the method of choice for virus detection in systemic outbreaks. All thirteen fetopathy incidents were confirmed by paired serology; virus isolation was attempted on viscera from more than 100 fetuses without success.

TABLE 3.1 : Methods of laboratory confirmation of systemic incidents

Year	No of Outbreaks	Method of confirmation				
		Virus Isolation Only	FAT + Virus Isolation	FAT only	Paired Serology	Histology*
1980	8	4	0	0	3	1
1981	12	2	5	0	3	2
1982	3	1	0	2	0	0
1983	5	2	1	1	1	0
1984	8	0	3	3	2	0
1985	11	1	1	6	3	0
1986	7	0	0	4	3	0
1987	7	0	0	3	4	0
1988	16	0	0	6	10	0
1989	6	0	0	2	4	0
Totals	83	10	10	27	33	3
%	100.0	12.1	12.1	32.4	39.8	3.6

*Discontinued after 1982

Serological diagnosis was requested mainly for abortion investigations and systemic outbreaks (particularly after 1985) in

which there was no significant respiratory component (eg during suspected recrudescences) to merit the collection of nasal or ocular swabs, and to ascertain whether BHV1 infection was involved in multifactorial calf pneumonia outbreaks. Second (convalescent) blood samples were received from less than 10 per cent of incidents for which serological diagnosis was sought.

Disease occurrence

Outbreaks of systemic IBR were confirmed every year, with peaks in 1981, 1985 and 1988; the lowest incidence (three outbreaks) was in 1982. As shown in Table 3.2, there was a fairly close match between the VIC archival data and VIDA II. When it occurred, over-recording on VIDA II could usually be attributed either to inadvertently counting rediagnoses as new incidents or basing diagnoses on inadequate criteria. Under-recording was associated mainly with a failure to register all confirmed incidents or not recording them until several weeks after their commencement (in which case they usually appeared in the next month's entry). In two of the 13 confirmed fetopathy incidents (Table 3.3) abortions occurred during systemic outbreaks. The under-recording of fetopathies by VIDA II when compared with the farm-file records, was almost entirely due to inadequate recording and failure correctly to match up results from paired serology.

As shown in Table 3.4, most outbreaks (44.6 per cent) occurred in dairy herds, closely followed by fattening herds (41.0 per cent). In

TABLE 3.2 : Confirmed systemic IBR incidents according to year and month of occurrence and comparison with VIDA II

Year	Month J	F	M	A	M	J	J	A	S	O	N	D	Totals
1980	2(2)	0(0)	1(0)	0(0)	1(1)	0(0)	0(0)	0(0)	1(1)	0(0)	0(0)	3(1)	8(5)
1981	0(1)	2(1)	0(2)	1(1)	0(0)	0(0)	1(0)	1(0)	1(2)	2(0)	2(2)	2(2)	12(11)
1982	1(1)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	1(1)	1(1)	3(3)
1983	1(0)	2(2)	0(0)	0(0)	0(0)	0(0)	1(0)	0(0)	0(0)	0(0)	0(0)	1(0)	5(2)
1984	2(2)	0(1)	0(0)	1(0)	0(1)	1(1)	1(0)	1(0)	1(0)	0(0)	1(0)	0(1)	8(6)
1985	2(2)	1(1)	0(1)	0(0)	0(0)	0(0)	0(0)	1(0)	0(0)	2(3)	2(1)	3(4)	11(12)
1986	2(1)	0(0)	0(0)	0(0)	0(3)	1(0)	1(1)	1(0)	0(0)	1(0)	1(1)	0(0)	7(6)
1987	1(1)	1(1)	1(3)	0(0)	1(2)	0(0)	1(3)	0(0)	0(0)	0(0)	0(0)	2(2)	7(12)
1988	1(1)	2(2)	2(0)	1(0)	1(0)	1(0)	0(0)	1(1)	1(0)	1(1)	1(1)	4(4)	16(10)
1989	1(1)	1(0)	1(0)	1(3)	0(0)	0(0)	0(0)	0(0)	0(0)	1(2)	1(1)	0(0)	6(7)
Totals	**13(12)**	**9(8)**	**5(6)**	**4(4)**	**3(7)**	**3(1)**	**5(4)**	**5(1)**	**4(3)**	**7(6)**	**9(7)**	**16(15)**	**83(74)**

( ) VIDA II data

TABLE 3.3 : Confirmed IBR fetopathy incidents according to year and month of occurrence and comparison with VIDA II

Year	Month J	F	M	A	M	J	J	A	S	O	N	D	Totals
1980	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
1981	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
1982	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
1983	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
1984	0(0)	0(0)	0(0)	0(0)	1(0)	0(0)	0(0)	*1(0)	0(0)	0(0)	0(0)	0(0)	2(0)
1985	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	1(0)	0(0)	1(0)
1986	1(0)	1(1)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	1(0)	0(0)	3(1)
1987	0(0)	1(0)	0(0)	0(0)	*1(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	2(0)
1988	0(1)	1(0)	0(0)	0(0)	0(0)	0(0)	0(1)	0(0)	1(0)	0(0)	1(0)	2(2)	5(4)
1989	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
Totals	**1(1)**	**3(1)**	**0(0)**	**0(0)**	**2(0)**	**0(0)**	**0(1)**	**1(0)**	**1(0)**	**0(0)**	**3(0)**	**2(2)**	**13(5)**

( ) VIDA II data *Confirmed during confirmed systemic incident

TABLE 3.4 : Annual occurrence of confirmed systemic and fetopathy incidents according to type of affected herd

Herd type / Year	Dairy	Suckler	Fattener	Totals
1980	3(0)	1(0)	4	8(0)
1981	5(0)	3(0)	4	12(0)
1982	0(0)	0(0)	3	3(0)
1983	1(0)	2(0)	2	5(0)
1984	5(2)*	1(0)	2	8(2)
1985	7(1)	0(0)	4	11(1)
1986	3(1)	1(2)	3	7(3)
1987	2(2)*	1(0)	4	7(2)
1988	9(3)	2(2)	5	16(5)
1989	2(0)	1(0)	3	6(0)
Totals	**37(9)**	**12(4)**	**34**	**83(13)**
%	44.6	14.4	41.0	100

( ) Number of fetopathy incidents
* Fetopathy incident confirmed during confirmed systemic incident
% Percentage of all systemic incidents

TABLE 3.5 : Monthly trends for all incidents according to herd type

Herd Type	Month of commencement J	F	M	A	M	J	J	A	S	O	N	D	Totals
Dairy	4(0)	2(2)	3(0)	3(0)	2(2)	2(0)	1(0)	5(1)	2(1)	2(0)	2(2)	9(1)	37(9)
Suckler	2(1)	3(1)	0(0)	0(0)	1(0)	0(0)	3(0)	0(0)	1(0)	1(0)	1(1)	0(1)	12(4)
Fattener	7	4	2	1	0	1	1	0	1	4	6	7	34
Totals	**13(1)**	**9(3)**	**5(0)**	**4(0)**	**3(2)**	**3(0)**	**5(0)**	**5(1)**	**4(1)**	**7(0)**	**9(3)**	**16(2)**	**83(13)**

( ) Number of fetopathy incidents recorded in addition to systemic outbreaks shown

dairy herds, systemic IBR was confirmed every year except 1982. Nine of the 13 recorded fetopathies were in dairy herds; none were confirmed before 1984. There was a regular pattern of disease occurrence in fattening herds with between two and five outbreaks confirmed each year. Excluding rediagnoses of separate incidents in subsequent years, new outbreaks of clinical IBR (including fetopathy) were confirmed in 35 different dairy herds, 14 suckler herds and on 30 fattening units during the study period.

Table 3.5 shows the monthly occurrence of all confirmed systemic incidents and fetopathies according to herd type. Outbreaks on fattening units occurred almost entirely during the late autumn and winter, between the months of October and March, whereas outbreaks in breeding herds (amongst adult cattle) were seen throughout the year. Overall, 40 (48.2 per cent) of the 83 systemic outbreaks were in adult cattle compared with 22 (26.5 per cent) amongst calves aged up to six months (these included six incidents in breeding herds). Ten outbreaks (12.0 per cent), all in fattening herds, occurred amongst cattle aged six to 12 months and 11 (13.3 per cent) involved cattle aged between one and two years. Seven of the latter outbreaks were in fattening cattle.

Clinical and epidemiological features

Most outbreaks in fattening herds were associated with a history of recent purchases, particularly from markets. A few outbreaks on larger units appeared to result from immunologically naive calves

becoming infected from direct or indirect (aerosol) contact with older calves that had recovered from clinical disease and were sharing the same airspace. The clinical features of typical IBR outbreaks in beef fattening cattle have been described extensively in the literature and will not be considered in any detail here. They varied in severity from mild conjunctivitis (Fig 3.1) and watery nasal discharge to severe fatal disease with necrotising tracheitis (Fig 3.2). After 1984, practitioners sometimes commented that clinical features suggestive of mild IBR were present during multifactorial calf pneumonia outbreaks but it was rarely possible to substantiate these claims to any extent. Coexistent infection with BVDV was confirmed in IBR outbreaks on two fattening units.

Epidemic outbreaks in breeding herds generally had a clear history of the recent introduction of purchased replacement breeding stock - usually from outside East Anglia. In some incidents, the added-animals themselves exhibited clinical signs soon after their arrival, but more usually, outbreaks commenced about two or three weeks later amongst in-contact cattle in the resident herd. In breeding herds the disease followed the typical course described in the literature: the case history of one such outbreak investigated by the author is presented as an addendum to this chapter. Considerably fewer outbreaks were confirmed in suckler herds than dairy herds (Table 3.6) and they tended to be of less economic significance. Six outbreaks in breeding herds were apparently restricted to calves but insufficient information was available to establish the degree of contact with the main herds. Even during severe epidemic outbreaks,

Fig 3.1: Calf with conjunctivitis due to IBR

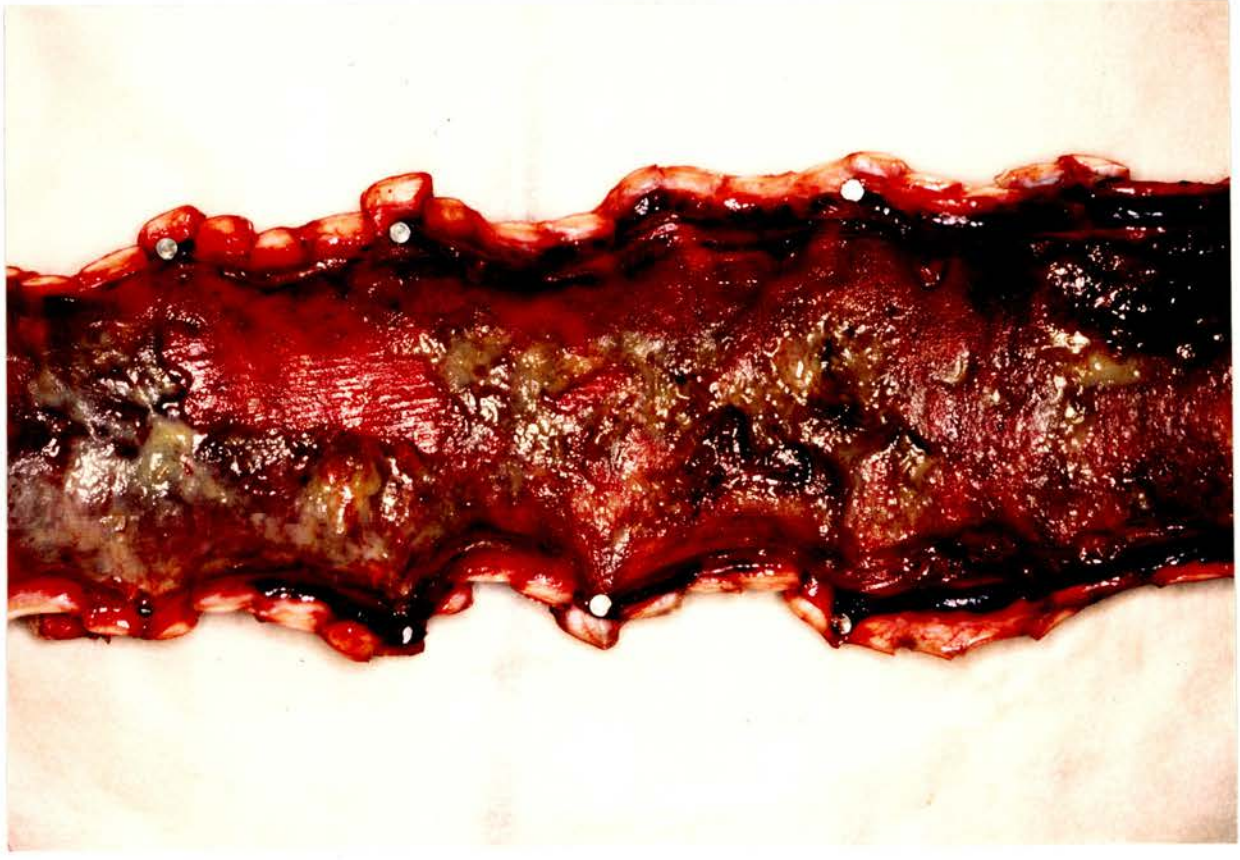

Fig 3.2: Necrotising tracheitis with thick diphtheritic exudate due to IBR

there were no confirmed accounts of the disease spreading from the milking herd into the calf house but there were several reports from the field of IBR spreading to calves grazing alongside infected suckler cows. Four outbreaks occurred amongst maiden or bulling heifers which were being kept on grazing completely separate from the adult herd. Concurrent parasitic bronchitis was confirmed during IBR outbreaks in three dairy herds and one suckler herd.

TABLE 3.6 : Types of IBR outbreaks in breeding herds

Type of outbreak	Number of outbreaks Dairy herds	Suckler herds	All herds	%
Epidemic disease in adult cattle	17	9	26	41.9
Epidemic disease in heifers*	2	2	4	6.4
Epidemic disease in calves**	5	1	6	9.7
Recrudescence in adult cattle	13	0	13	21.0
Fetopathy	9	4	13	21.0
Totals	**46**	**16**	**62**	**100**

* Aged 12 - 24 months ** ≤six months old

Thirteen (35.1 per cent) of the 37 systemic outbreaks in dairy herds were classified as recrudescences (Table 3.6). These invariably occurred in herds known to have a high prevalence of seropositive cattle based on farm-file data (see Chapter 4) or a past history of IBR. They generally involved less than five per cent of cattle in

the herd - often only one or two younger cows were affected. Clinical signs were usually mild and comprised slight pyrexia, inappetence and a fall in milk yield for two or three days; a slight watery oculonasal discharge was often present. Unlike epidemic outbreaks, recrudescences were not usually preceded by the purchase of breeding replacements althougn homebred heifers entering endemically infected adult herds sometimes developed mild clinical signs. Recrudescence was not confirmed in suckler herds.

All fetopathies involved single abortions apart from incidents in two herds in which two and three abortions were confirmed during periods of about two weeks. Abortions were occasionally reported by practitioners during suspected epidemic outbreaks but, except in two incidents, all confirmed fetopathies occurred in herds with no history of overt clinical disease, although there was usually evidence to suggest that they were endemicaly infected. One of the two exceptions was in a dairy herd in which two cows aborted two weeks after the commencement of a typical epidemic outbreak; the other incident occurred during recrudescence amongst a group of second and third parity cows.

DISCUSSION

Despite the well known shortcomings (Martin and others 1987a) of using laboratory data, particularly the biased voluntary submissions to VICs (Thrusfield 1988), to monitor disease occurrence, it was probable that submissions to Norwich VIC for IBR diagnosis provided

an accurate reflection of the situation in the field. The introduction of improved diagnostic tests and efficacious vaccines during the early 1980s motivated veterinary practitioners to seek laboratory confirmation for suspected outbreaks. Virtually all diagnostic testing for IBR in England was undertaken within the VIS/CVL (Anon 1979) which further improved the effectiveness of disease surveillance.

Although the sources of information were essentially the same, the data collected from Norwich VIC archives provided a more accurate and comprehensive account of disease occurrence than VIDA II. Not only were original reports re-examined and diagnostic criteria standardised but the type of herd involved and the form of the outbreak were also considered. Despite these refinements, the close correlation with VIDA II data was reassuring in view of the numerous opportunities for operator error and potential for diagnostic inconsistencies inherent in the recording system. By using the 1985 census data for breeding herds with more than 10 animals (Table 2.1) as a guide to mean population size during the study period, it was evident that about 11 per cent of dairy herds and five per cent of suckler herds in Norfolk experienced confirmed IBR during the 10 years under investigation.

As pointed out by Nettleton (1986), the submission of inadequate material to the laboratory adversely affects the diagnostic success rate for IBR. This undoubtedly reduced the number of confirmed outbreaks recorded during these studies although probably not to any

great extent, because practitioners often subsequently submitted further, more suitable samples, from the same incident. Virus isolation in cell culture is the most sensitive method of diagnosing BHV1 infection but it suffers inherent drawbacks, including a possible delay of up to 14 days before achieving a diagnosis (Nettleton 1986). With veterinary practitioners constantly under pressure from clients seeking a rapid diagnosis, it was not surprising that the FAT evolved as the main method of confirming IBR outbreaks. Kahrs (1977) and Nettleton (1986) both advised that seroconversion to BHV1 should be demonstrated in addition to virus detection, in order to distinguish between primary infection and virus reactivation. Such an approach was impractical in most field situations and despite the number of outbreaks confirmed by serology, practitioners were generally reluctant to submit paired blood samples. Although not used for disease diagnosis, testing single serum samples from convalescent animals often proved useful in excluding BHV1 infection from the differential diagnosis, particularly with fetopathies.

The FAT was not used to diagnose BHV1 abortions because of its low efficacy with fetal tissue (Lucas and others 1986). Similarly, fetal fluid serology was not used to diagnose BHV1 abortion because it is generally considered (Kendrick 1973, Lucas and others 1986) that the virus causes fetal death before antibody can be produced. Despite this, Murray (1990) used the latter method to apparently confirm BHV1 abortion in fetuses from Cumbria examined between 1984 and 1986. Focal necrotic lesions, which are reported to occur in cases of BHV1

infection (Owen and others 1964) were not seen in any of the fetuses examined. Virus isolation in tissue culture is potentially the best method of confirming BHV1 abortion (Stubbings and Cameron 1981), but this proved completely unsuccessful during these studies. Because of the delay between BHV1 infection and abortion (Pritchard 1990), seroconversion has almost invariably occurred before paired blood samples can be collected. Consequently, a rise in antibody titre or seroconversion are rarely demonstrable. Despite these problems, paired serology was the only method whereby BHV1 fetopathy was confirmed during these studies although the possibility that the stress of abortion reactived latent virus (Thiry and others 1985) leading to a coincidental increase in antibody titre could not be discounted. The actual number of recorded BHV1 abortions undoubtedly underestimated the field situation because of difficulties with diagnosis. However, there was no noticeable increase in the number of statutory abortion submissions from recently infected herds and no evidence from anecdotal sources to suggest that BHV1 was a significant cause of abortion in Norfolk herds. These observations supported the conclusions of other authors in Britain, including Edwards (1988).

The term "recrudescence" has been used somewhat loosely in the literature to describe recurrent infection associated with reactivation and excretion of latent virus (Gibbs and Rweyemamu 1977, Kahrs 1977, Narita and others 1981, Straub 1991) although Russell and Edington (1986) reserved its use for excretion associated with fresh lesions, as with herpes "cold sores" in man. Naturally occurring IBR

recrudescence has received little attention in the past and most authors appear to be of the opinion that it is clinically insignificant. These present studies would dispute that view and suggest that not only can recrudescence cause some economic loss, particularly by reducing milk yield, but it is also a sure indication of continuing virus circulation and active infection. The significance of this in relation to programmes for the elimination of BHV1 from infected herds is discussed later in this thesis.

It was not possible to determine conclusively whether recrudescences at a herd level occurred predominantly amongst animals experiencing virus reactivation or amongst naive in-contacts, but the fact that second and third parity cows appeared to be the most commonly affected group suggested that the latter was usually the case. This was borne out by findings detailed in Chapter 5. The clinical recognition of recrudescence was somewhat haphazard, being particularly dependent on alert stockmanship, especially if only a few animals were involved in the outbreak. The number of recrudescences recorded here was inevitably a gross underestimate of the extent of recrudescence in the field.

Whereas epidemic IBR outbreaks were almost invariably associated with the recent introduction of new stock, there were no such obvious external sources of infection with outbreaks classified as recrudescences. This observation, also made by Kahrs (1977) supported the hypothesis that these incidents were endogenous in origin. Significantly, herds experiencing recrudescence were

invariably known or suspected to be carrying a heavy weight of endemic infection.

Although IBR was prevalent amongst calves on fattening and rearing units, relatively few outbreaks were confirmed in young calves in breeding herds. Wiseman and others (1980) similarly noted that IBR did not appear to spread significantly from the adult cows to calves on the same farm. Wiseman and others (1980) also commented that cattle kept outside experienced clinically less severe IBR outbreaks than housed cattle. This could explain why fewer outbreaks were reported in suckler herds than in dairy herds. Furthermore, mild outbreaks, particularly recrudescences, were more likely to pass unnoticed in suckler herds because herdsmen had less contact with their cattle. Nevertheless, the low incidence of IBR recorded in suckler herds was still surprising because they ran a greater risk of introducing infection with purchased replacement breeding stock (Pritchard and Bardsley 1990) than dairy herds, which mainly bred their own replacements.

The present findings generally supported the observations of Wiseman and others (1980) and Edwards (1988) regarding the occurrence of most IBR outbreaks during the winter housing period. However, it was clear from these studies that systemic outbreaks in adult breeding cattle occurred throughout the year whereas outbreaks in calves on rearing and fattening units invariably occurred during the winter. The winter pattern for calf outbreaks was probably due to the same causes as those predisposing to enzootic calf pneumonia including

cold, dampness and poor ventilation (Blood and Radostits 1989b). There was no clear explanation for the all-year-round occurrence of outbreaks in adult cattle but it may have been associated with a tendency to introduce purchased (ie possibly infected) replacement breeding heifers during the summer months.

Whilst IBR outbreaks occurred sporadically in Norfolk throughout the 1980s, there were no reports of the genital forms of BHV1 infection (IPV/IPB). After a period of great concern for the cattle breeding industry during the early 1970s, these conditions now appear to have been virtually, if not completely, eliminated from Britain (Edwards 1983, Edwards 1988, Edwards, personal communication) with the introduction of strict control measures at AI Centres (Lucas 1986).

ADDENDUM : CASE HISTORY OF AN IBR OUTBREAK IN A DAIRY HERD

Herd details

This was an established 100-cow family-run dairy herd in an isolated location. Cows were winter-housed in strawed yards on a conventional ration of silage, sugar-beet pulp and concentrates. Breeding was by artificial insemination to calve July - September; a "sweeper" bull was used on the heifers. Male calves were sold soon after birth; female calves were retained as heifer replacements, initially kept in the calf house adjacent to the cows but moved to a separate farm after weaning where they remained until returning to the main herd to calve at about two years of age. The herd had been virtually closed until 12 cows purchased from a dispersal sale in the Midlands were delivered by a dealer in three separate loads between 20/10/84 and 6/11/84.

History

On 8/12/84, five purchased cattle developed pyrexia of 103 - 107°F (39.5 - 42.0°C), inappetence, a watery ocular-nasal discharge and showed a fall in milk yield (Table 3.7). The affected cows were injected with broad-spectrum antibiotic by the farmer but by the next day four homebred cows were also affected. By 11/12/84, a total of 27 cows from the 80 cows then in the milking herd were showing similar clinical signs. Eighteen cows were considered by the owner's veterinary surgeon to be sufficiently ill to merit a three day course

of antibiotic therapy. There were no fatalities or abortions during or after the outbreak and clinical signs were not seen amongst any cattle apart from those in the milking herd.

Diagnosis

To confirm the outbreak, three conjunctival swabs and three naso-pharyngeal "brush" swabs were taken from recently affected cows and put into VTM. At the VIC, a good positive result for IBR was obtained with the FAT carried out on the naso-pharyngeal swabs but there were insufficent cells from the conjunctival swabs to provide a definitive result. The FAT results were subsequently confirmed by virus isolation in tissue culture.

Vaccination

The entire milking herd (including clinical cases) was vaccinated with "Tracherine" (Smithkline Animal Health Ltd) on 13/12/84. No fresh cases were seen in the herd within three days after vaccination and milk yield soon returned to normal (Table 3.7). Heifers that joined the milking herd in July 1985 were vaccinated but there was no subsequent revaccination of the milking herd.

Economic losses

The cost of the outbreak was summarised by the farmer as: £650 for veterinary expenses (including vaccination), £150 for milk discarded

due to antibiotic usage (1000 litres @ 15p/litre) and £510 for loss in potential milk yield due to illness (3400 litres @ 15p/l). The total cost of the outbreak was therefore estimated at £1310 (approximately £16 per lactating cow).

Table 3.7: Effect of IBR outbreak on herd milk yield

Date (Dec 1984)	Milk yield (litres)		
	Expected	Actual	Shortfall
2 - 8	11,500	11,457	43
9 - 15	12,000	8,373*	3,627
16 - 22	12,700	11,978	722
23 - 29	13,500	13,493	7
Totals (2 - 29)	49,700	45,301	4,399

* Including milk discarded due to antibiotic treatment

Recrudescence

After the initial IBR outbreak had resolved, sporadic recrudescences with pyrexia, milk drop and mild respiratory signs, were confirmed by paired serology between 1986 and 1988. These mainly affected a few second and third parity cows and suggested that BHV1 was still circulating in the herd. This was further confirmed by serological tests in 1988, after which a programme was set up to eliminate the infection (see D24, Chapter 6).

CHAPTER 4 : SERO-EPIDEMIOLOGICAL OBSERVATIONS

Despite numerous serological surveys, there is a lack of information in the literature about the distribution of BHV1 infection in different types of herd and age-specific prevalence patterns. The studies described in this chapter utilise the results of tests for BHV1 antibodies performed on sera submitted to Norwich VIC to examine these aspects in some detail and to provide general background information on the BHV1 serological status of Norfolk herds.

MATERIALS AND METHODS

Extraction and collation of data

Details of all sera from cattle in Norfolk herds tested for BHV1 antibodies (by IHAT, SNT or ELISA) between 1980 and 1989 were obtained by manually searching farm-file records as described in the previous chapter. The serological results were recorded as negative or positive; they included findings from statutory brucellosis abortion submissions (for which differential diagnosis had been sought) and those from systemically diseased or apparently diseased cattle. Where paired sera were submitted, only the results from the second samples were included. The results of tests on healthy cattle examined for disease screening purposes were also recorded. Single laboratory submissions of more than 20 sera from the same herd were excluded as were the results of tests carried out specifically for Cattle Health Scheme membership (see Chapter 5).

The information provided by veterinary practitioners on the appropriate laboratory submission forms (Appendices 3.1, 3.2) was used to extract the following data : year of submission, herd identity and type, age (if given), number of sera tested, number of positive sera (reactors) and submission category (reason for testing). The latter were classified as either: fetopathy investigation (including infertility), diagnosis of systemic illness (i.e. respiratory disease, milk-drop or other non-specific, usually febrile illness) or health screening tests undertaken prior to sale, purchase or export. Other criteria including seropositive thresholds were as described in the previous chapter.

Analysis of data

The number and proportion of sera containing BHV1 antibodies were calculated according to year, herd, age and submission category. The results were tabulated to show annual trends and combined to provide period prevalence. Individual herds were classified as infected if one or more sera were positive, based on the assumption that most cattle with humoral antibodies were latently infected and therefore potential sources of infection to other members of the herd (Kahrs 1977).

The proportion of possibly uninfected breeding herds was estimated (Cannon and Roe 1986) from the period prevalence data in those herds for which 10 or more sera had been tested without detecting a

reactor, assuming average (1985) herd sizes listed in Tables 2.1 and 2.2 and an expected 25 per cent of positives. Such herds in which at least 50 per cent of sera tested were positive were identified as likely to be heavily infected.

Statistical tests were calculated at the five per cent level of significance unless stated otherwise. Differences in antibody prevalence between submission categories were compared using the chi-square test (Dunn 1967). The 95 per cent confidence intervals (CI) for the proportion of potentially uninfected, infected, and heavily infected breeding herds and for the prevalence of reactors according to year, submission category, herd type and age, were calculated for sample size n and estimated prevalence P using the formula (Snedecor and Cochran 1967):

$$P \pm 1.96 \sqrt{\frac{P\,(1-P)}{n} + \frac{1}{2n}}$$

RESULTS

Annual trends and submission categories

The wide annual variation in the number of sera submitted for testing (Table 4.1) appeared mainly to reflect changes in the disease surveillance and charging policies of the VIS. During the period 1980 - 1982, sera submitted for disease diagnosis were not normally examined unless a second sample was subsequently received. About 50 per cent of sera examined were from statutory abortion investigations. Most sera from cases of systemic illness tested

Table 4.1 : Prevalence of BHV1 antibodies according to year and submission category

Year	Reason for testing: Systemic illness No	+	%	Fetopathy No	+	%	Health screen No	+	%	Totals No	+	%	95% CI
1980	15	14	93.3	4	2	50.0	5	0	0.0	24	16	66.7	45.7-87.6
1981	45	25	55.6	25	1	4.0	NT	NT	NT	70	26	37.1	25.1-49.2
1982	58	19	32.8	24	2	8.3	7	3	42.9	89	24	27.0	17.2-36.7
1983	18	4	22.2	63	10	15.9	NT	NT	NT	81	14	17.3	8.4-26.1
1984	63	22	34.9	103	36	34.9	42	4	9.5	208	62	29.8	23.4-36.3
1985	40	6	15.0	24	9	37.5	31	0	0.0	95	15	15.8	7.9-23.6
1986	69	14	20.3	207	72	34.8	16	0	0.0	292	86	29.5	24.0-34.8
1987	31	10	32.3	98	25	25.5	45	4	8.9	174	39	22.4	15.9-28.9
1988	68	26	38.2	192	54	28.1	88	17	19.3	348	97	27.9	23.0-32.7
1989	130	35	26.9	244	66	27.1	153	30	19.6	527	131	24.9	21.1-28.6
Totals	537	175	32.6	984	277	28.2	387	58	15.0	1908	510	26.7	
95% C I	28.5 - 36.6			25.3 - 31.0			11.3 - 18.7			24.7 - 28.7			

No Number of sera tested
+ % Number and percentage of sera containing BHV1 antibodies
95% C I 95 per cent confidence intervals
NT No sera tested

between 1980 and 1985 related to respiratory disease. After 1985, tests were more frequently carried out as part of investigations into causes of milk-drop or non-specific febrile illness, as these features of recrudescent BHV1 infection became more widely appreciated. There was an increased interest by farmers in disease screening tests in 1988 and 1989 following the introduction of the Cattle Health Scheme (see Chapter 5).

A total of 1908 sera from 274 herds (158 dairy, 89 suckler 27 fattener) were tested for BHV1 antibodies: 510 (26.7 per cent) were positive. Of the 537 sera from systemically affected animals, 175 (32.6 per cent) were positive; this was not significantly different to the proportion of positives (28.2 per cent) from fetopathies. A significantly smaller proportion of sera (15.0 per cent) from healthy cattle contained BHV1 antibodies than either of the two diseased categories (chi-square = 37.6, $P<.001$). None of the 76 sera (from eight different herds) tested for export and included in Table 4.1 were positive although the majority of export blood samples were sent direct to the CVL and results were not all available to the author.

Type of herd

The overall proportion of sera containing BHV1 antibodies was greatest in fattening herds (Table 4.2) but there were no statistically significant differences in antibody prevalence between the three herd types.

Table 4.2 : Prevalence of BHV1 antibodies according to year and herd type

Year	Dairy			Type of Suckler Herd			Fattening		
	No	+	%	No	+	%	No	+	%
1980	16	12	75.0	NT	NT	NT	8	4	50.0
1981	61	22	36.1	NT	NT	NT	9	4	44.4
1982	47	14	29.8	27	8	29.6	15	2	13.3
1983	65	13	20.0	9	0	0.0	7	1	14.3
1984	186	44	23.7	20	16	80.0	2	2	100.0
1985	67	12	17.9	22	2	9.1	6	1	16.7
1986	224	68	30.3	50	17	34.0	18	1	5.6
1987	122	23	18.9	39	17	43.6	13	9	69.2
1988	278	70	25.2	53	20	37.7	17	7	41.2
1989	388	102	26.3	129	27	20.9	10	2	20.0
Totals	1454	380	26.1	349	97	27.8	105	33	31.4
95% C I	23.8 - 28.4			23.0 - 32.6			22.1 - 40.8		

No Number of sera tested
NT No sera tested
+ % Number and percentage of sera containing BHV1 antibodies
95% C I 95 per cent confidence intervals

The 1454 dairy herd sera contributed more than 75 per cent of all samples examined; one or more reactors were found in 76 (48.1 per cent) of these 158 herds. This provided 95 per cent CI of 40.0 to 56.2 per cent for the minimum proportion of infected dairy herds in the sample population. No reactors were detected in 13 (26.5 per cent) of 49 different dairy herds from which at least 10 sera were submitted. This gave 95 per cent CI of 13.1 to 39.9 per cent for the proportion of potentially uninfected herds. Similarly, in 17 (34.7 per cent) of the 49 herds (95 per cent CI 20.3 to 49.1 per cent) at

least half the sera tested were positive, suggesting that these were heavily infected herds. These included herds with recrudescent outbreaks as mentioned in the previous chapter.

Reactors were found in 31 (34.8 per cent) of the 89 different suckler herds (95 per cent CI 24.4 to 43.5 per cent) and 15 (55.6 per cent) of the 27 fattening units (95 per cent CI 36.8 to 74.4 per cent), neither of which was significantly different to the proportion of infected dairy herds. No reactors were found in four of 11 different suckler herds contributing 10 or more sera; at least half the sera tested were positive in five herds.

Age-related effects

Ages were available for 1208 (83.1 per cent) of the sera from dairy herds, 314 (90.0 per cent) of sera from suckler herds and for all fattening herd sera. There were no significant differences in the proportion of reactors in sera from cattle for which age was provided compared with sera from all cattle tested.

Table 4.3 shows the age-related prevalence of BHV1 antibodies by herd type using the combined data from 1980 - 1989. The proportion of positive sera from calves aged up to six months in dairy herds was less than in either fattening or suckler herds. However, since the samples were derived from a relatively small number of herds, statistical comparisons were inappropriate. Antibody prevalence fell to its lowest level at between six and 12 months of age in all three herd types but subsequently increased in yearlings. The mean

Table 4.3 : Age-specific prevalence of BHV1 antibodies according to herd type

Age in Years	Dairy No	Dairy +	Dairy %	Suckler No	Suckler +	Suckler %	Fattening No	Fattening +	Fattening %	Totals No	Totals +	Totals %	95% CI
0 - ½	74	10	13.5	43	15	34.9	73	28	38.4	190	53	27.9	21.3-34.5
>½ <1	17	1	5.9	36	0	0.0	27	4	14.8	80	5	6.2	0.1-12.2
1	205	30	14.6	76	27	35.5	5	1	20.0	286	58	20.3	15.4-25.1
2	121	23	19.0	6	3	50.0				127	26	20.5	13.1-27.9
3	161	26	16.2	34	12	35.3				195	38	19.5	13.7-25.3
4	170	48	28.2	27	3	11.1	N O N E			197	51	25.9	19.5-32.3
5	157	47	29.9	28	7	25.0				185	54	29.2	22.4-36.0
6	120	39	32.5	28	8	28.6				148	47	31.8	23.9-39.6
7	68	28	41.5	10	3	30.0	T E S T E D			78	31	39.7	28.2-51.2
8	55	16	29.1	12	2	16.7				67	18	26.9	15.5-38.2
9	26	6	23.1	6	3	50.0				32	9	28.1	11.0-45.3
≥10	34	13	38.2	8	3	37.5				42	16	38.1	22.2-54.0
Totals	1208	287	23.8	314	86	27.4	105	33	31.4	1627	406	25.0	
95% C I	21.3 - 26.2			22.3 - 32.5			22.1 - 40.8			22.8 - 27.1			

No Number of sera tested with age provided
+ % Number and percentage of sera containing BHV1 antibodies
95% C I 95 per cent confidence intervals

seroprevalence then remained virtually unchanged (at about 20 per cent) until three years, after which it increased with age to peak at nearly 40 per cent in seven-year-old cattle. As shown by the 95 per cent CI given in Table 4.3 antibody prevalence in seven-year-old cattle was significantly greater than in those aged six months to three years.

DISCUSSION

Although inevitably derived from a biased sample the serological data analysed here represented a sizeable proportion of the Norfolk cattle population - the 170 dairy herds comprised 54.3 per cent of the 313 herds containing at least 10 cows as listed in the 1985 agricultural census returns (Table 2.1). In their survey, Dawson and Darbyshire (1964) found no evidence of BHV1 infection in Norfolk but the number of sera tested was not stated. The prevalence (29.7 per cent) of BHV1 antibodies in the 1521 sera (Table 4.1) submitted for disease diagnosis (ie excluding screening tests on healthy cattle) between 1980 and 1989 was slightly, although probably not significantly, less than the 34.7 per cent recorded by Edwards (1988) in a much larger survey of diagnostic sera tested at the CVL from herds in England and Wales.

The findings demonstrated that BHV1 infection was more widespread in Norfolk than suggested by the number of confirmed outbreaks of IBR described in the previous chapter. Brenner and others (1989) similarly remarked on the disparity between BHV1 seroprevalence from

surveys and reports of clinical cases of IBR. The introduction of the ELISA test after 1985 improved diagnostic sensitivity over the standard SNT and IHAT tests (Edwards and others 1986) but it seemed unlikely that any resultant increase in the number of reactors detected from low titre sera significantly affected the findings reported here.

Period prevalence is generally of limited epidemiological value as a measure of disease occurrence because it fails to distinguish between new and old cases (Thrusfield 1986b). However, because BHV1 titres persist and seropositive animals constitute a continuing source of infection (Kahrs 1977), period prevalence was still considered to provide a useful guide to assessing the proportion of herds and individual cattle in the study population that had experienced BHV1 infection and were therefore still potentially infective.

The significantly lower proportion of reactors amongst healthy cattle supported the findings of Peters and Perry (1983) and Edwards (1988). It should be noted, however, that these surveys of apparently healthy cattle mainly involved testing sera from young bulls or heifers prior to export or AI. Such cattle would be expected to show a lower antibody prevalence than apparently diseased cattle of various ages because they were in a consistently younger age group. The age-related effect of BHV1 infection questions the validity of using such sources of data as the basis of estimates of antibody prevalence amongst clinically normal cattle. The most reliable estimate of antibody prevalence in breeding herds, particularly dairy herds, came

from the abortion sera. These indicated that between 25.3 and 31.0 per cent of adult cattle in Norfolk were seropositive for BHV1. Unlike systemically diseased animals, aborting cows represented a clearly defined population of mature cattle; furthermore, since abortion was not a significant sequel to BHV1 infection (Chapter 3), their sera provided a relatively unbiased sample of the normal cattle population.

Edwards (1988) reported that 48 per cent of herds in England and Wales tested between 1984 and 1986 contained one or more reactors. This was very similar to the total of 122 (44.5 per cent) from 274 different herds (including fattening units) in Norfolk which contained at least one reactor. However, Edwards (1988) classified each batch of sera submitted as a different herd which probably underestimated the true proportion of infected herds. Also, more significantly, his findings were based on herds examined during a three year period instead of the 10 years of the present study.

The prevalence of BHV1 antibodies in sera from fattening calves was greater (although not significantly) than in dairy herds but attempts to draw any conclusions from this were hampered by the different population turnover rates and sources of origin of calves in these two types of herds, the small number of fattening sera tested and the effects of MDA. Nevertheless, since most calves on fattening units came from outwith the county, the higher antibody prevalence amongst this group further suggested that BHV1 infection was more prevalent outside Norfolk than in indigenous herds.

The low antibody prevalence in cattle aged between six and 12 months in all three herd types supported the findings described in the previous chapter which showed that the incidence of clinical disease was also fairly low amongst this age group. The fall in antibody prevalence was also likely to be associated with the decline in MDA after about six months of age. The subsequent increase in prevalence of BHV1 antibodies amongst yearling cattle (mainly breeding heifers reared apart from the main herd) suggested that some of these became infected before rejoining their parent herds for calving. This may have occurred whilst grazing communal marshland but it also possibly reflected the serological status of heifers purchased as breeding replacements from outside the area.

The age-related prevalence data for older cattle was derived mainly from individual cows and represented a large number of herds surveyed. It showed a statistically significant increase in the proportion of cattle with BHV1 antibodies between three and seven years of age. Since most herds calved at between two and three-years-old the findings suggested a constant probability of acquiring infection after calving with increasing age. This was consistent with horizontal spread of an endemic infection (Martin and others 1987b) and supported reports by other authors (Kirby and others 1978, Msolla and others 1981, Forschner 1988) that BHV1 seroprevalence increased with age.

CHAPTER 5: EPIDEMIOLOGICAL STUDIES IN CATTLE HEALTH SCHEME HERDS

The concept of BHV1 latency dictates that after recovering from acute infection, cattle probably remain a potential source of virus for life. Although many studies have shown that reactivation and shedding of latent virus can be induced experimentally, the extent to which this occurs naturally under field conditions, leading to new infection, is not known. The cross-sectional studies described here utilise the results of whole-herd serological screening tests undertaken in breeding herds in Norfolk to examine the epidemiology of subclinical BHV1 infection with particular reference to the transmission of virus from serological reactors to uninfected herd members.

MATERIALS AND METHODS

Cattle Health Scheme herds

In April 1987, the SVS introduced the Cattle Health Scheme (CHS) for Great Britain (Penton 1987, ADAS 1987). This superseded the Enzootic Bovine Leukosis (EBL) Attested Herd Scheme (Roberts and Bushnell 1982) and also offered an IBR monitoring programme which gave farmers the opportunity to carry out serological testing for BHV1 and to eliminate the infection from their herds where appropriate.

Sixty-three breeding herds in Norfolk joined the CHS to become EBL attested during the four year period between March 1987 and

March 1991. A standard questionnaire (Appendix 5.1) was completed at the initial visit to each farm, at which the author was accompanied by the owner's veterinary surgeon. Details recorded included: herd size (number of cattle aged two years or more), herd type (dairy or beef suckler), breed, status (pedigree or non-pedigree/commercial), replacement policy (main sources and numbers purchased during the previous 10 years), general management practices and disease history.

Owners were encouraged to have some, or all, of their cattle examined for serological evidence of BHV1 infection when they were tested for EBL (by their veterinary surgeon or by a MAFF Animal Health Officer), provided the herd had no recent clinical history of IBR (or IPV/IPB) or IBR vaccination (which would have made it virtually impossible to interpret serological findings). Seven herds were thereby excluded from the investigations: in three the owners declined to undertake any serological testing for BHV1 and four had a history of IBR and had been vaccinated during the previous two years. One dairy herd that had experienced epidemic IBR in 1981 and been vaccinated with "Tracherine" (SmithKline Animal Health Limited) was included because there was no history of revaccination or clinical recrudescence. None of the remaining 55 herds had any apparent history of IBR although 25 had contributed serological data, including some seropositive samples, to the analysis for 1980 - 89 presented in the previous chapter.

The 23 dairy herds in the study population had all been established for at least 20 years and were of pedigree status (20 herds were

Holstein/Friesian and three were Ayrshire). Most of the 33 suckler herds had been assembled during the previous 10 years and all but one contained solely or predominantly pedigree cattle. The main suckler breeds represented were: Simmental (10 herds), Charolais (four herds), Blonde d'Aquitaine (four herds), Limousin (three herds), Salers (two herds), and one herd each of: Aberdeen Angus, South Devon, Bazardais, Longhorn, British White, Murray Grey, Highland, Belgian Blue, Shorthorn and Hereford. The systems of husbandry and management were essentially as described in Chapter 2, but being high quality pedigree herds, the sale of breeding stock and regular exhibition at agricultural shows featured prominently in most enterprises.

Serological testing

All cattle, including calves, were blood sampled in 22 herds (10 dairy, 12 suckler): the actual number of calves sampled in these herds depended on the time of year of the test in relation to the calving pattern. Adult cattle only (≥two years) were tested in 31 herds (10 dairy, 21 suckler) and in four dairy herds testing was limited to a sufficient number of adult cattle to detect a 10 per cent level of reactors with a 95 per cent confidence (Cannon and Roe 1982).

An ELISA technique (Edwards and others 1986) performed at Penrith VIC, was used to examine CHS sera for BHV1 antibodies. When the scheme was first introduced, cattle were deemed seropositive

("reactors") at OD values of ≥0.1 using a serum dilution of 1/50, but an inconclusive reactor (IR) category (OD 0.10 - 0.19), which required resampling, was subsequently introduced by the SVS in April 1989 to cope with some inconsistent results. In 1990, the 24 hour SNT (Edwards and others 1986) was routinely adopted at the CVL as the decisive test for IRs that could not be clarified by ELISA: SNT titres of ≥1/2 were classed as positive. Reagent concentrations for use by the VIS for CHS tests were altered in October 1989 resulting in a change in the OD range for IRs to 0.15 - 0.25, although the sensitivity of the ELISA test remained the same.

Collation and analysis of data

The OD values for each animal tested were expressed as either seropositive or seronegative and recorded with their ear tag or freeze brand number, sex, age, source (homebred or purchased) and any individual pre-purchase vaccination history (if known).

Herds were coded D (Dairy) or S (Suckler) and numbered sequentially according to the date of the blood test. The serological results were tabulated and also displayed graphically to show the distribution of BHV1 antibodies according to herd identity, herd type (D or S), age and whether cattle were homebred or purchased. Herds were classified according to antibody prevalence and the apparent rate of seroconversion amongst susceptible cattle.

Results were analysed using standard statistical methods (Dunn 1967); mean values, standard deviations (SD) and standard errors (SE) were calculated where appropriate. Differences in antibody prevalence between dairy and suckler herds were compared using the chi-square test. Correlation coefficients (r) were used to examine the association between the number of reactors and: herd size, number of cattle purchased in the previous 10 years and number of purchased cattle currently in the herd. The association between reactors and purchase was further examined by fitting a generalised linear model (binomial error, logit link) with Genstat 5 (1987), the linear predictor consisting of terms herd and homebred/purchase.

After the test results were known, herd managers were contacted by telephone to discuss the significance of the findings. In herds with a low seroprevalence, details of the movement history (eg communal marsh grazing, attendance at agricultural shows and temporary residence in other herds) of individual reactors and the length of time they had been in the herd were recorded. Where seropositive calves up to nine month old were known to have sucked seropositive dams, they were assumed to have acquired MDA. This was slightly later than the seven months duration of MDA suggested by Bitsch (1984) but was based on experiences gained during the studies in CHS herds.

RESULTS

Antibody prevalence according to type and size of herd

Sera from a total of 4,219 cattle (3,201 dairy, 1,018 suckler) aged >two years were examined for BHV1 antibodies: 639 (15.1 per cent) were positive. Herd sizes and the number and proportion of reactors in dairy and suckler herds are shown in Tables 5.1 and 5.2. The proportion (14.6 per cent) of reactors in dairy cattle was not significantly different to the proportion (16.8 per cent) of reactors amongst sucklers (chi-square = 2.84, $p>0.5$). There was a significant correlation ($r=0.05$, $p<.05$) between the number of reactors and herd size in suckler herds but not in dairy herds.

There were no reactors in five (21.7 per cent) of the 23 dairy herds and 11 (33.3 per cent) of the 33 suckler herds (chi-square = 0.90, $p>.05$). Of the remaining dairy herds, 14 held 20 per cent or fewer reactors but the other four (D3, D16, D20, D21) each contained more than 60 per cent reactors. Fifteen suckler herds contained 20 per cent or fewer reactors; only one suckler herd (S27) had a seroprevalence of more than 60 per cent but in two others (S19, S20) it exceeded 40 per cent. The frequency distribution for the proportion of reactors in all herds is summarised in Fig 5.1.

Table 5.1 : Prevalence of BHV1 reactors in dairy herds

Herd	Month/Year of test	Cattle aged ≥2 years No	+	%
D1	3/87	227	0	0
D2	3/87	*60 (95)	5	8.3
D3	4/87	136	97	71.3
D4	5/87	*26 (136)	5	19.2
D5	10/87	254	14	5.5
D6	10/87	189	0	0
D7	1/88	133	18	13.5
D8	4/88	154	6	3.9
D9	7/88	123	6	4.9
D10	11/88	114	0	0
D11	5/89	117	1	0.9
D12	11/89	*30 (161)	0	0
■D13	12/89	117	12	10.3
D14	1/90	140	2	1.4
D15	3/90	176	7	4.0
D16	3/90	*28 (200)	25	89.3
D17	3/90	260	19	7.3
D18	5/90	121	7	5.8
D19	9/90	52	0	0
D20	2/91	134	84	62.6
D21	2/91	147	92	62.5
D22	2/91	196	39	19.9
D23	3/91	267	29	10.9
Totals	3/87 - 3/91	3201	468	14.6

No Number of cattle tested for BHV1 antibodies
\+ Number of reactors
% Percentage of reactors
* Statistical screening only
(total number of cattle ≥2 years in herd)
■ IBR in 1981

Table 5.2 : Prevalence of BHV1 reactors in suckler herds

Herd	Month/Year of test	Cattle aged ≥2 years No	+	%
S1	7/87	24	2	8.3
S2	2/88	10	0	0
S3	2/88	18	1	5.5
S4	2/88	90	3	3.3
S5	3/88	56	6	10.7
S6	4/88	7	1	14.3
S7	11/88	16	1	6.3
S8	12/88	60	4	6.7
S9	1/89	20	0	0
S10	2/89	29	4	13.8
S11	5/89	25	0	0
S12	6/89	56	11	19.6
S13	7/89	25	2	8.3
S14	7/89	4	0	0
S15	7/89	5	0	0
S16	11/89	9	0	0
S17	12/89	27	6	22.2
S18	1/90	86	4	4.7
S19	2/90	76	42	55.3
S20	4/90	125	55	44.0
S21	5/90	18	0	0
S22	6/90	6	0	0
S23	9/90	10	3	30.0
S24	10/90	25	0	0
S25	11/90	12	0	0
S26	11/90	8	2	25.0
S27	11/90	16	10	62.5
S28	12/90	40	0	0
S29	12/90	13	5	38.5
S30	12/90	10	2	20.0
S31	12/90	22	2	9.1
S32	1/91	35	4	11.4
S33	1/91	35	1	2.9
Totals	7/87-1/91	1018	171	16.8

No Number of cattle tested for BHV1 antibodies
\+ Number of reactors
% Percentage of reactors

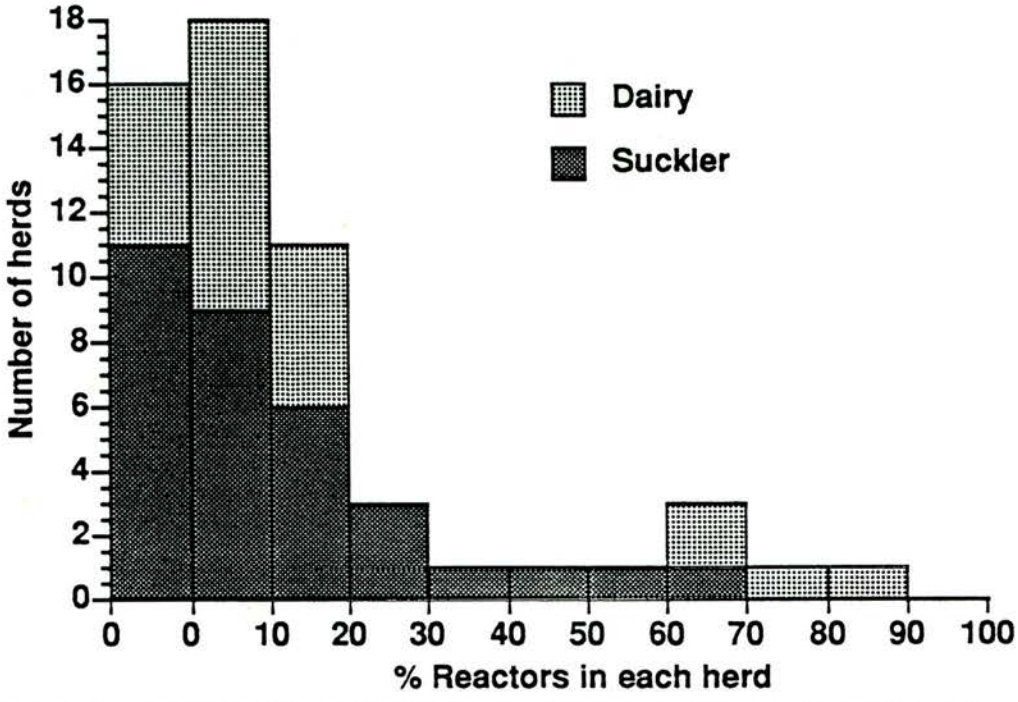

Fig 5.1: Frequency distribution of BHV1 reactors in CHS herds

Antibody prevalence according to source of origin

Breeding replacements were purchased from a wide geographical area, mainly outside Norfolk, extending from Scotland to the West Country and continental Europe. Only three herds (D1,D6,D9) had been completely closed during the previous 10 years (Tables 5.3, 5.4), with the mean number of cattle purchased being 17.1 ± SD 17.5 in dairy herds and 22.7 ± SD 22.5 in suckler herds. The mean number of purchased cattle in dairy herds at the time of the test (7.2 ± SD 8.4) was less than half that in suckler herds (15.9 ± SD 20.1) but the latter included several recently established herds (notably S27, which had been formed a year previously from cattle purchased exclusively from S19).

Table 5.3 : Prevalence of BHV1 reactors in dairy herds according to source

Herd	Purchased Cattle			Homebred Cattle	
	No in 10 years	No in herd	No of Reactors	No in Herd	No of Reactors
D1	0	0	0	227	0
*D2	25	5	3	55	2
D3	24	16	15	120	82
*D4	30	2	1	24	4
D5	2	2	1	252	13
D6	0	0	0	189	0
D7	60	34	7	99	11
D8	58	20	6	134	0
D9	0	0	0	123	6
D10	21	14	0	100	0
D11	10	10	1	107	0
*D12	6	4	0	26	0
D13	8	8	4	100	8
D14	8	8	1	132	1
D15	40	16	5	160	2
*D16	32	9	9	19	16
D17	2	1	1	259	18
D18	10	2	1	119	6
D19	10	0	0	52	0
D20	3	1	0	133	84
D21	25	4	4	143	88
D22	10	9	5	187	34
D23	9	0	0	267	29
Total	393	165	64	3036	404

* Statistical screening only

No in 10 years: Total number of cattle purchased in the previous 10 years

No in herd : Total number of purchased cattle in the herd and included in the test

Significant correlations were noted in suckler herds, but not in dairy herds, between the number of reactors and the number of cattle purchased in the previous 10 years ($r = 0.42$ $p<.05$) and between the number of reactors and the number of purchased cattle in the herd at the time of the test ($r = 0.63$, $p<.01$). In dairy herds, the mean

Table 5.4 : Prevalence of BHV1 reactors in suckler herds according to source

Herd	Purchased Cattle			Homebred Cattle	
	No in 10 years	No in herd	No of Reactors	No in Herd	No of Reactors
S1	20	12	2	12	0
S2	3	1	0	9	0
S3	18	3	1	15	0
S4	8	3	2	87	1
S5	30	23	4	33	2
S6	5	5	1	2	0
S7	36	6	1	10	0
S8	25	18	4	42	0
S9	6	6	0	14	0
S10	27	27	4	2	0
S11	8	4	0	21	0
S12	54	54	10	2	1
S13	25	25	2	0	0
S14	4	4	0	0	0
S15	3	3	0	2	0
S16	4	4	0	5	0
S17	24	12	6	15	0
S18	94	86	4	0	0
S19	65	47	21	29	21
S20	45	68	26	57	29
S21	18	18	0	0	0
S22	12	6	0	0	0
S23	4	4	2	6	1
S24	6	1	0	24	0
S25	4	3	0	9	0
S26	8	8	2	0	0
S27	16	16	10	0	0
S28	30	9	0	31	0
S29	13	13	5	0	0
S30	4	4	2	6	0
S31	70	4	2	18	0
S32	10	8	4	27	0
S33	50	20	0	15	1
Totals	749	525	115	493	56

No in 10 years: Total number of cattle purchased in the previous 10 years

No in herd : Total number of purchased cattle in the herd and included in the test

percentage of purchased reactors (41.3 ± SE 3.2) was significantly greater than that of homebred reactors (17.6 ± SE 0.7) ($p<.001$). In suckler herds this difference was less marked (13.9 ± SE 1.5, 9.9 ± SE 1.4 respectively) but still statistically significant ($p<.05$).

In two dairy herds (D8, D11) and 14 suckler herds (S1, S3, S6, S7, S8, S10, S13, S17, S26, S27, S29, S30, S31, S32) all reactors had been purchased; some comprised the original foundation stock, still in the herd after seven (S30) or eight (S6) years. Many of the purchased reactors in suckler herds, but not in dairy herds, were ET recipients known to have been vaccinated before introduction. Details of the vaccination history and sources of individual reactors are listed according to age in Tables 5.5 and 5.6.

Antibody prevalence according to age

All reactors detected amongst calves up to six months old (Tables 5.5 and 5.6) and the single reactor aged nine months in D5 had received colostrum from known seropositive dams, with the exception of the reactor in S9 which was from a dam of unknown BHV1 status having been purchased from a local market at two months of age. Single reactors were present amongst cattle less than two years old in three herds (D10, S9, S11) in which all older cattle were seronegative. Those in S9 and S11 were both purchases and that in D10 was a homebred heifer known to have broken in with a group of cattle of unknown disease status whilst grazing communal marshland about a month before the test.

Table 5.5 : Relationship between age and source of BHV1 reactors in infected dairy herds

Herd	Age of reactors in years																																	
	0-½		>½-<1		1		2		3		4		5		6		7		8		9		10		11		12		13		14		15		16	
	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P
D2															2	0			0	1	0	2														
D3					1	0			5	0	10	0	31	0	8	14	9	0	6	0	3	0	4	1	1	0	2	0	3	0						
D4									1	0					1	0	1	1					1	0												
D5			1	0									1	0			1	0	1	0	8	0	1	0	1	0			0	1						
D7											1	0	0	2					1	0	6	1	1	0	1	1	0	2			1	1				
D8															0	1	0	5																		
D9											1	0							4	0							1	0								
D10					1	0																														
D11											0	1																								
D13	2	0							0	2			0	1			2	1					6*	0												
D14																					0	1									1	0				
D15													1	1			0	3	0	1	1	0														
D16									12	1	1	2	0	2	0	1	1	1	0	1	0	1	1	0	1	0										
D17															1	0			3	0	2	0			8	0	3	0			1	1				
D18																	1	0			2	0			1	1	2	0								
D20									2	0	12	0	16	0	21	0	17	0	7	0	3	0	3	0	1	0					2	0				
D21									1	1	37	1	13	0	10	0	4	0	4	1	4	0	6	0	4	0	3	1							2	0
D22											1	0	3	0	3	1	9	4	6	0	4	0	4	0			1	0					2	0	1	0
D23	9	0									1	0	4	0	3	0	5	0	7	0	5	0	3	0			1	0								
Totals	11	0	1	0	2	0	0	0	21	4	64	4	69	6	49	17	50	15	39	4	38	5	30	1	18	2	13	3	3	1	5	2	2	0	3	0

H Homebred P Purchased * Vaccinated during IBR outbreak eight years previously

Table 5.6 : Relationship between age and source of BHV1 reactors in infected suckler herds

Herd	Age of reactors in years																																	
	0-½		>½-<1		1		2		3		4		5		6		7		8		9		10		11		12		13		14		15		16	
	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P	H	P
S1	2	0																	0	1					0	1									
S3											0	1*																							
S4													1	0											0	2									
S5					0	7*	0	3*			1	0	0	1*			1	0																	
S6	1	0																					0	1											
S7															0	1																			
S8																					0	1	0	3											
S9	0	1																																	
S10					2	0	0	3*	0	1																									
S11					0	1*																													
S12	1	0							0	1	0	2*	0	1*	1	0			0	1			0	1	0	2	0	1	0	1					
S13											0	1											0	1											
S17											0	1*					0	1*	0	1*	0	1*			0	1*							0	1*	
S18									0	1			0	3																					
S19							8	1	10	2	1	2	1	0	0	2	1	4	0	2	0	2	0	2	0	1					0	3			
S20					18	0	3	0	4	2	4	6	1	1	1	3	6	11	2	1	3	0	2	2	1	0	1	0	1	0					
S23															0	1					0	1					1	0							
S26													0	2																					
S27	1	0			2	0	0	4	0	3			0	2									0	1											
S29							0	4					0	1																					
S30																							0	1									0	1	
S31									0	2																									
S32																							0	2					0	1			0	1	
S33									1	0																									
Totals	5	1	0	0	22	8	11	15	15	12	6	13	3	11	2	7	8	16	2	6	3	5	2	14	1	7	2	1	1	2	0	3	0	3	

H Homebred P Purchased * Known or suspected vaccinates when purchased

Table 5.7 : Age-specific prevalence of BHV1 antibodies in dairy herds

Herd	Age in years																					
	0 - ½		>½ - <1		1		2		3		4		5		6		7		8		9		≥10	
	No	+	No	+	No	+	No	+	No	+	No	+	No	+	No	+	No	+	No	+	No	+	No	+
D1	4	0	32	0	30	0	70	0	38	0	34	0	27	0	13	0	15	0	10	0	8	0	12	0
#D2*									17	0	5	0	12	0	6	2	6	0	8	1	6	2		
■D3					70	1	4	0	23	5	23	10	32	31	23	22	9	9	6	6	3	3	13	11
#D4*							6	0	5	1	1	0	3	0	2	1	4	2	1	0	3	0	1	1
#D5			1	1	1	0	71	0	42	0	40	0	40	1	27	0	16	1	5	1	8	8	5	3
D6	19	0	28	0	68	0	13	0	48	0	32	0	30	0	28	0	16	0	9	0	3	0	10	0
#D7							24	0	17	0	21	1	27	2	21	0	4	0	2	1	8	7	9	7
#D8					25	0	30	0	20	0	14	0	22	0	32	1	28	5	6	0			2	0
#D9	7	0			46	0	17	0	20	0	18	1	15	0	16	0	9	0	13	4	4	0	11	1
D10	23	0	6	0	13	1	18	0	17	0	26	0	7	0	8	0	10	0	8	0	6	0	14	0
#D11	12	0	9	0	32	0	21	0	32	0	22	1	6	0	9	0	6	0	2	0	9	0	10	0
D12*									10	0	3	0	5	0	1	0	4	0	2	0	3	0	2	0
#D13	21	2			13	0	19	0	24	2	19	0	19	1	15	0	11	3	3	0	1	0	6	6
#D14	6	0			8	0	21	0	26	0	21	0	16	0	16	0	11	0	13	0	7	1	9	1
#D15							24	0	17	0	21	0	37	2	21	0	27	3	18	1	8	1	3	0
■D16*					2	0	2	0	13	13	3	3	2	2	1	1	3	2	1	1	1	1	2	2
#D17							54	0	24	0	39	0	41	0	37	1	12	0	21	3	12	2	20	13
#D18							7	0	28	0	24	0	22	0	14	0	7	1	2	0	10	2	7	4
D19	17	0	6	0	9	0	10	0	5	0	10	0	6	0	5	0	11	0	2	0	2	0	1	0
■D20					6	0	12	0	21	2	19	12	22	16	26	21	18	17	7	7	3	3	6	6
■D21							9	0	29	2	39	38	17	13	17	10	4	4	6	5	7	4	19	16
#D22					4	0	55	0	23	0	23	1	19	3	26	4	22	13	14	6	4	4	8	8
#D23	65	9	41	0	56	0	64	0	53	0	40	1	30	4	26	3	28	5	12	7	9	5	5	4
Totals	174	11	123	1	383	2	551	0	555	25	499	68	457	75	390	66	281	65	171	43	125	43	175	83
% Reactors	6.3		0.8		0.5		0		4.5		13.6		16.4		16.9		23.1		25.1		34.4		47.4	

* Statistical screening only ■ High prevalence herd # Low prevalence herd

Table 5.8a : Age-specific prevalence of BHV1 antibodies in suckler herds

Herd	Age in years																					
	0 - ½		>½ - <1		1		2		3		4		5		6		7		8		9		≥10	
	No	+	No	+	No	+	No	+	No	+	No	+	No	+	No	+	No	+	No	+	No	+	No	+
#S1	23	2	4	0	16	0	3	0	3	0	4	0	4	0	5	0			1	1	2	0	2	1
S2					15	0	5	0	1	0	3	0			1	0								
#S3									3	0	5	1	2	0	1	0	3	0	1	0			3	0
#S4	42	0	10	0	34	0	29	0	18	0	13	0	5	1	9	0	4	0	3	0	4	0	5	2
#S5			4	0	18	7	11	3	13	1	6	0	10	1	4	0	6	1	4	0			2	0
#S6	2	1			2	0	3	0	1	0	1	0					1	0					1	1
#S7	3	0	12	0			4	0	2	0	3	0	1	0	2	1	3	0	1	0				
#S8	2	0	14	0	22	0	22	0	10	0	9	0	6	0	8	0	1	0			1	1	3	3
S9	5	1			15	0	3	0	3	0	3	0	4	0	1	0	1	0	2	0	1	0	2	0
#S10					9	2	25	3	3	1													1	0
S11	2	0	13	0	3	1	4	0	4	0	4	0	3	0	3	0	2	0	2	0	1	0	2	0
#S12	11	1	9	0	23	0	11	0	10	1	5	2	6	1	3	1	2	0	2	1	2	0	15	5
#S13	21	2	3	0					19	0	4	1	1	0									1	1
S14	3	0							3	0			1	0										
S15					1	0	2	0							3	0								
S16	2	0	7	0	1	0	2	0	2	0	1	0	1	0			1	0	1	0	1	0		
#S17	6	0	12	0	7	0	4	0	6	0	3	1	4	0	1	0	2	1	1	1	1	1	5	2
#S18							23	0	2	1	31	0	30	3										

...continued/

Table 5.8b : Age-specific prevalence of BHV1 antibodies in suckler herds cont'd

Herd	Age in years																							
	0 - ½		>½ - <1		1		2		3		4		5		6		7		8		9		≥10	
	No	+	No	+	No	+	No	+	No	+	No	+	No	+	No	+	No	+	No	+	No	+	No	+
■S19					1	0	10	9	16	12	10	3	6	1	9	2	6	5	6	2	6	2	7	6
■S20					29	18	32	3	23	6	16	10	5	2	13	4	20	17	3	3	4	3	9	7
S21							18	0																
S22			6	0											6	0								
#S23							2	0	1	0			1	0	1	1					2	1	3	1
S24							7	0	8	0	1	0	4	0	1	0	2	0			1	0	1	0
S25			4	0			2	0	4	0			4	0			1	0					1	0
#S26	5	0			5	0	2	0					6	2										
■S27	5	1	4	0	4	2	6	4	4	3	2	0	2	2					1	0			1	1
S28							4	0	16	0	11	0	3	0	1	0	2	0			1	0	2	0
#S29							6	4	1	0	2	0	2	1	2	0								
#S30			1	0			3	0			1	0	1	0	1	0			2	0			2	2
#S31							13	0	9	2														
#S32					7	0	8	0	7	0	5	0	1	0	3	0	1	0	1	0	2	0	7	4
#S33							2	0	5	0	5	0	6	0	3	0	6	1			3	0	5	0
Totals	132	8	97	0	212	30	266	26	197	27	140	18	119	14	81	9	64	25	31	8	32	8	80	36
% Reactors	6.1		0		14.2		9.8		13.7		12.2		11.8		11.1		39.1		25.8		25.0		45.0	

■ High prevalence herd

Low prevalence herd

As shown in Tables 5.7 and 5.8 the mean antibody prevalence in dairy and suckler herds calculated from pooled data declined from about six per cent in calves aged up to six months to virtually nil (only one reactor from 220 calves) by 12 months. Seroprevalence amongst yearlings, two-year-olds and three-year-olds was markedly lower in dairy herds than in suckler herds but since most of the latter reactors were limited to a few herds (particularly S19), statistical comparisons between herd types were considered inappropriate. From four years of age, mean seroprevalence increased at a similar rate in both types of herd and by 10 years nearly 47 per cent of all cattle were reactors, including all eight (in herds D21,D22,S17,S30 and S32) that were 15 years or older (Tables 5.5 and 5.6).

Seroconversion patterns

As illustrated by the pooled data presented in Figs 5.2 and 5.3 the age-specific prevalence for individual herds revealed two distinct patterns of seroconversion which corresponded to the main antibody prevalence groupings noted earlier. In the four dairy herds (D3, D16, D20, D21) with a high prevalence (HP) of BHV1 antibodies (>60 per cent) seroconversion appeared to commence amongst heifers within two or three months after they joined the main herd for calving; uncalved heifers on separate grazing were all seronegative apart from the single yearling reactor in D3 which appeared to have acquired infection through contact with a yearling steer from another herd. By four years of age, 75 per cent of cattle in these herds were reactors.

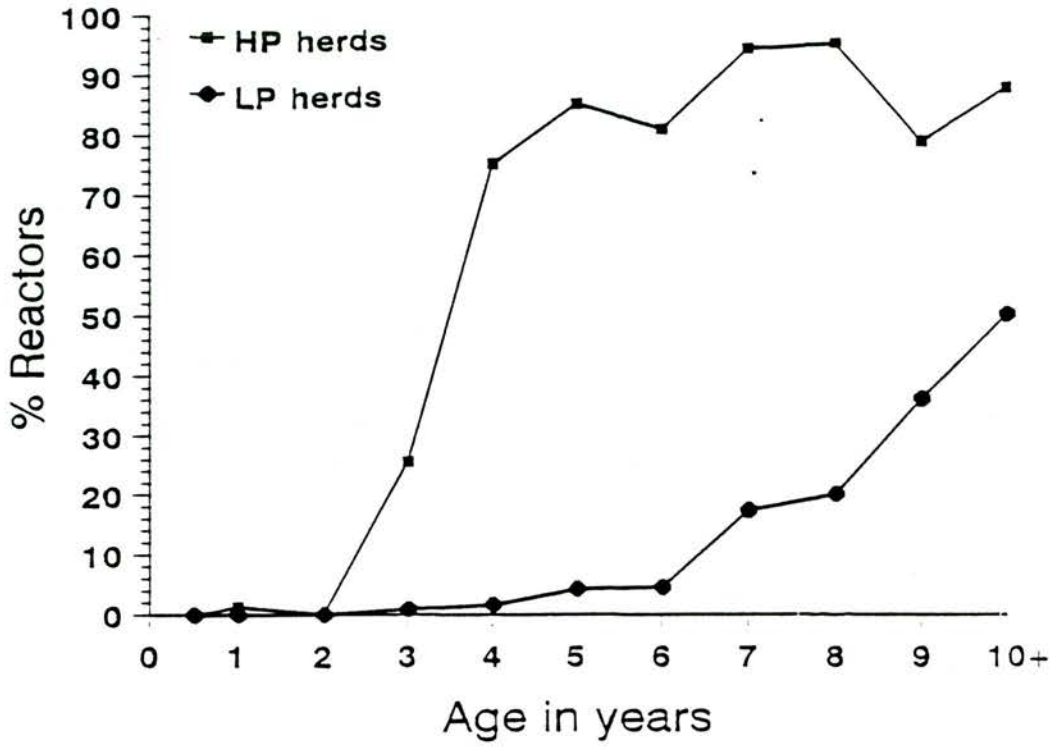

Fig. 5.2 - Seroconversion patterns in dairy herds according to antibody prevalence category

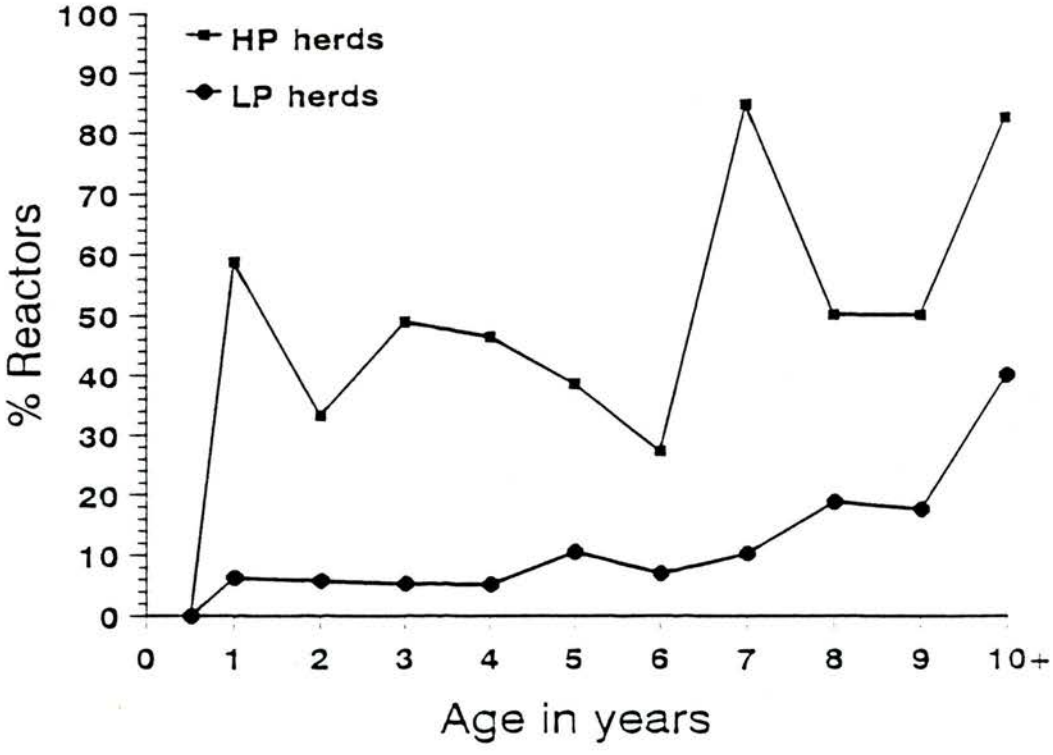

Fig. 5.3 - Seroconversion patterns in suckler herds according to antibody prevalence category

In the 14 dairy herds with a low prevalence (LP) of BHV1 antibodies (<20 per cent) there was no such evidence of active infection amongst heifers after calving and less than five per cent of cows in their second or third parity were seropositive. These included purchased cattle that may have seroconverted before entry into their new herds. The proportion of reactors in LP dairy herds rose sharply after six years of age (Fig 5.2); seroprevalence amongst cattle aged nine years or more was higher than average in D5, D7, D22 and D23 (Table 5.7).

Only three suckler herds (S19, S20, S27) showed marked increases in seroprevalence indicative of extensive virus circulation. These were also herds identified previously as having a high prevalence of BHV1 antibodies (>40 per cent). Instead of commencing amongst recently calved heifers, seroconversion in the HP suckler herds appeared to start at between one and two years of age and to continue at a lower rate after calving than in HP dairy herds. The age-specific prevalence pattern in LP suckler herds (Fig 5.3) was virtually identical to that recorded in LP dairy herds (Fig 5.2).

DISCUSSION

The 56 herds included in this study represented about nine per cent of all dairy and suckler herds containing 10 or more cows listed in the 1989 agricultural census for Norfolk (MAFF 1990) (Chapter 2). The extent of BHV1 infection in this sample population was essentially unknown before investigations commenced: apart from D13 which had experienced an epidemic outbreak eight years previously,

none of the herds had any clinical history of IBR. Significantly, the prevalence of BHV1 antibodies (15.1 per cent) was virtually identical to that in sera from apparently healthy cattle featured in the analysis for 1980 - 1989 described in the previous chapter. Furthermore, if the four (large) herds with a recent history of IBR/vaccination had been included in these studies instead of being excluded at the outset, overall seroprevalence, and the proportion of heavily infected herds, would have been close to previous estimates.

The large proportion of herds with a low prevalence of BHV1 antibodies appeared similar to the situation in Germany (Forschner 1988) and Spain (Espuna and others 1988). The latter authors lent further support to the present studies by also finding that herds tended to be either heavily or lightly infected, with few having intermediate seroprevalence.

The significant correlation between herd size and the number of reactors in suckler herds appeared mainly to reflect a tendency for larger suckler herds to contain more purchased cattle. On average, suckler herds contained more than twice as many purchased cattle as dairy herds. The four largest dairy herds (D1, D5, D17, D23) had purchased a total of only 13 replacements during the previous 10 years.

The strong association between reactor status and purchase supported the generally accepted view (Gibbs and Rweyemamu 1977, Pastoret and others 1984) that BHV1 is most frequently introduced by the addition

of latently infected carriers to susceptible herds. Remarkably, however, purchased adult cattle - including many that had been in the herd for several years - were the only reactors detected in 16 of the 40 infected herds, suggesting that no virus transmission had occurred following their introduction. Similarly, in LP herds in which some young homebred cattle were also reactors, anecdotal information often suggested sources of infection other than lateral spread from seropositive purchased animals. In S4, for example, the only homebred reactor was a bull that had been loaned to a herd in the south of England for four months before returning to Norfolk. In D13, six of the eight homebred reactors had been in the herd during the IBR outbreak recorded in 1981. No satisfactory explanation was found for the exclusively homebred reactors in D9 and S33 but it was assumed that they had acquired infection in unrecorded incidents, similar to that in D10, in which a heifer broke in with a group of other cattle.

Two of the homebred reactors in D18 were specifically identified as having attended several agricultural shows. However, several other large herds had exhibited cattle at six or seven major shows each year for the previous 15 years without any evidence of them acquiring infection from this source. Although detailed records were rarely available to make any systematic risk assessment, the present findings suggested that agricultural shows and similar livestock events did not pose any major threat, particularly when there was

only a short period of close contact with other cattle. Further observations regarding show attendance are included in the next chapter.

The extent to which virus transmission occurred within infected herds was clearly linked to their seroprevalence. There was a marked difference between LP and HP herds in the rate of seroconversion amongst naive cattle, as illustrated by their contrasting age-specific prevalence patterns. The antibody prevalence recorded amongst adult cattle in HP herds was partly influenced by the timing of the herd blood test in relation to the calving season - proportionally fewer two-year-olds were likely to have seroconverted if they were uncalved or had calved only recently when tested and had not had time to encounter infection. The less well-defined distinction between LP and HP status in suckler herds (compared with dairy herds) was mainly the result of a high proportion of purchased reactors being present in very small herds.

Overall, field observations suggested that, in LP herds, the risk of virus being transmitted from seropositive cattle (including vaccinates) to susceptible in-contacts was quite small. Although reexcretion of latent virus is undoubtedly of potential epidemiological importance, its role under natural conditions, in the absence of experimental provocation, appears to have been overestimated. Van Nieuwstadt and Verhoeff (1983) similarly found no evidence of virus transmission for at least three years in eight of 20 dairy herds in Holland and Baker and others (1989) detected only a

very low rate of transmission of live intranasally administered vaccinal virus to co-mingled steers. Pastoret and others (1984) commented that only about 60 per cent of infected cattle shed virus after dexamethasone treatment which suggested that not all seropositive animals are latent carriers.

Widespread virus circulation, manifested by a high rate of seroconversion, was clearly evident in the seven herds with the highest antibody prevalence. Presumably, these herds were initially infected via purchased replacements or similar sources. Unlike the situation in LP herds, the virus then appeared to have spread rapidly to susceptible cattle, amongst which it was maintained sufficiently, presumably by regular reactivation and reexcretion, to infect each year's input of heifers. There were no discernible differences between HP and LP herds in environmental or intercurrent disease stress factors such as lungworm infestation (Msolla and others 1983) likely to cause viral reactivation.

Edwards and others (1991) found that calves infected with BHV1 subtype 1 excreted more virus than those infected with subtype 2b strains and it appeared likely (S Edwards, personal communcation) that the apparent divergence in virus behaviour between LP and HP herds was associated with different strains of virus, possibly beyond the sensitivity of simple restriction endonuclease fingerprinting techniques. This hypothesis was supported by the observation that herd S27, which was formed entirely from cattle in S19, had assumed the seroconversion pattern of its parent herd, as was evident by the

presence of two homebred yearling reactors. A less likely explanation for the contrasting rates of seroconversion was that one or more particularly heavy shedders of virus were present in HP herds but not in LP herds.

Corkish (1988) suggested that calves in suckler herds were more likely to acquire infection from their dams than those in dairy herds which were removed soon after birth. There was, however, no serological evidence from any of the herds in these studies to indicate that active infection had occurred amongst calves aged less than a year. MDA invariably disappeared by about six months and as noted by Bradley (1985) these calves did not seroconvert until after joining the cow herd as recently calved heifers. Although sera were examined from calves in only one (S27) of the three HP suckler herds, consistently seronegative findings from calves in this age range in LP suckler herds suggested that the likelihood of them acquiring infection through sucking seropositive dams was small.

Seroconversion occurred amongst 12 - 24 month old heifers in HP suckler herds but not in the HP dairy herds. This difference was attributed to the management practice commonly adopted in suckler herds, whereby maiden heifers were housed close to adult cattle during the winter. Similar findings, including seroconversion to BHV1 amongst yearlings, were noted in suckler herds in the north-west of Scotland (N. G. Brookes, personal communication). In dairy herds, replacement heifers were almost invariably maintained as completely separate populations, on marsh or upland grazing, until

they joined the milking herd after calving. Hence, provided they did not acquire infection from outside sources, uncalved heifers from HP dairy herds were seronegative on entering the milking herd. Forschner (1988) similarly commented that the proportion of reactors amongst young cattle was lower on farms where they were kept apart from the adult herd. By comparing serological status with date of calving it appeared that most seroconversion in HP herds occurred two or three months after calving, coinciding with winter housing.

The increase in seroprevalence with age supported findings described in Chapter 4. In LP herds the likelihood of cattle being reactors did not increase until after six years whereas most cattle in HP herds had seroconverted by this time. Regardless of prevalence category almost 50 per cent of all cattle in infected herds, aged 10 years or more, were seropositive. The preponderance of reactors amongst older animals suggested that the LP herds (particularly D5, D7, D22 and D23) had been more heavily infected in the past, but with regular routine culling, infection was gradually working its way out of these herds. Provided it is not reintroduced, self-elimination of BHV1 from LP herds appears to be inevitable in view of the lack of spread to susceptible herd members. Van Nieuwstadt and Verhoeff (1983) similarly suggested that BHV1 might disappear in the long-term from herds in which no recent virus circulation had occurred. Conversely, BHV1 infection was likely to be maintained indefinitely in HP herds unless there was a change in virus behaviour or in some

factor not apparent from these studies. There was no obvious explanation to account for the presence of eight seronegative cows from amongst the 57 aged 10 years or more in the seven HP herds.

Because of the high rate of virus circulation detected in HP herds, further enquiries were made to check whether there had been any clinical history that could possibly be attributed to BHV1 infection. These revealed that in August 1989, four recently calved cows in D21 experienced pyrexia and milk drop; a further similar case had occurred in October 1990. No BHV1 antibodies had been detected in diagnostic blood samples taken from these cows at the acute stage of illness but when they were eventually retested (for the CHS) in February 1991 all five were found to have seroconverted to BHV1. Although not conclusive because of the long time interval between testing, this evidence suggested that the herd may have experienced clinical recrudescence of the type identified in Chapter 3.

Further evidence linking recrudescence and HP status, also in the absence of a history of epidemic IBR, subsequently emerged from D3. At the time of the herd test in 1987 there was no clinical history of BHV1 infection but several typical IBR recrudescences were subsequently confirmed by paired serology amongst second and third parity cows between 1989 and 1991. These findings in D21 and D3 supported observations made in Chapter 3 regarding the occurrence of recrudescence in heavily infected herds.

CHAPTER 6: ELIMINATION OF BHV1 INFECTION

Besides economic benefits attributable to the absence of clinical disease, BHV1-free serological status confers potential advantages on herd owners when selling breeding stock, semen or embryos, particularly for export. The introduction of the CHS in 1987 increased awareness of the health status of cattle herds in Britain and promoted interest in the elimination of BHV1 infection - already underway in some other countries of the EC. The longitudinal studies presented here examine the progress of those herds featured in the previous chapter that subsequently undertook the CHS programme to achieve and maintain freedom from BHV1 infection. The results in two HP herds that adopted different strategies are also described.

MATERIALS AND METHODS

Serological testing

The laboratory criteria used to determine whether cattle were BHV1 reactors were as described in the previous chapter.

CHS testing programme

Procedures

Herds in which no BHV1 reactors were detected amongst cattle aged ≥two years at the first blood test undertook a second qualifying test

of all animals (including calves) at least four months later. After two consecutive clear blood tests, herds were registered as having achieved "IBR monitored negative" status (ADAS 1987) and were then subject to annual periodic tests of all adult cattle.

If reactors were disclosed at the first or subsequent tests and the owner wished to proceed with the scheme, these cattle were usually retested immediately to eliminate misidentification or laboratory error; if the result was confirmed they were either culled or put into isolation. A repeat test (either of all cattle in the herd or of those aged two or more years) was performed at least a month after reactors had been segregated. Provided this test was negative, these herds reverted to the testing programme outlined for herds with no reactors at the first test.

In addition to the blood testing programme, owners agreed to comply with scheme rules (ADAS 1987) which included restrictions on the use of semen from BHV1-seropositive bulls and the strict maintenance of farm boundaries to keep scheme cattle at least three metres apart from cattle of unknown disease status. An "added-animals" procedure was adopted for purchased replacement stock and after exhibiting at agricultural shows: this required cattle to be seronegative for BHV1 when blood tested after at least a month in isolation, before being allowed to join or rejoin the herd.

Participating herds and data recording

Four dairy herds (D1, D6, D10, D19) and 10 suckler herds (S2, S9, S11, S14, S15, S16, S21, S22, S24, S28) with no reactors aged two or more years at the first test proceeded with the CHS programme to reach IBR monitored negative status. The single reactors detected amongst cattle aged less than two years (see Chapter 5) in three of these herds (D10, S9, S11) were removed at least four months before the second tests were undertaken. Nine LP dairy herds (D5, D8, D9, D11, D14, D15, D17, D18, D23) and 12 LP suckler herds (S1, S4, S6, S7, S8, S10, S12, S13, S17, S18, S32, S33) undertook further testing after removal of reactors.

Prospective studies were undertaken in the above 35 herds until August 1991: serological results were recorded and tabulated according to date (month/year) of the tests, the number of cattle tested and the number of reactors. If new reactors were identified, herd managers were consulted to determine the probable source of infection and steps were taken to limit its spread; additional blood testing was undertaken as appropriate. Attendance at agricultural shows was recorded and results of post-show serological screening tests were noted.

High prevalence dairy herd

Background details

This CHS herd (designated D24) was located in East Suffolk and experienced the outbreak of epidemic IBR described in the addendum to Chapter 3 (q.v. for details of management and husbandry practices). In June 1988, when the herd was first screened for BHV1 antibodies, 31 (75.6 per cent) of 41 randomly selected cattle at least two years old proved to be seropositive. All cattle aged a year or more were subsequently tested in December 1988: 106 (84.8 per cent) of the 125 milking cows then in the herd were reactors but all the younger cattle, including the 10 uncalved two-year-olds, were negative (Table 6.1). Of 10 cows that were seronegative in June 1988 and retested in December 1988, four (all aged between two-and-a-half and four years) had seroconverted to BHV1, confirming recent virus circulation and HP status.

Elimination strategy

A plan was devised whereby the uncalved heifers and the 19 seronegative cows identified in December 1988 were to provide the basis for a new BHV1-free milking herd to be established within three years, without the need for any seronegative breeding replacements to be purchased.

Table 6.1: Age-specific prevalence of BHV1 antibodies in D24 before commencement of eradication programme

Age (years)	Cattle tested No	+	%
1	23	0	0
2	20	10#	50.0
3	26	19	73.1
4	25	23	92.0
5	17	17)	
6	8	8)	
7	7	7)	100*
8	6	6)	
9	8	8)	
≥10	8	8)	

No Number of cattle tested
\+ Number of reactors
\# All recently calved
% Percentage of reactors
* All vaccinated in 1984/85

Between January 1989 and June 1991, a non-infected group, initially comprising the 19 seronegative cows, was managed separately from the infected group (initially comprising 106 cows). Recently calved homebred heifers were introduced into the negative group and infected cows were gradually culled when convenient. Instead of allowing heifers born in 1987 to calve as two-year-olds in 1989, service was delayed in order to calve most as three-year-olds in 1990; most 1988-born heifers also calved in 1990, but as two-year-olds. These management changes were adopted to allow as many seronegative cattle as possible to be introduced simultaneously, having first reduced the size of the infected group. Heifers were served exclusively by a BHV1-seronegative bull and kept strictly apart from the main herd until they calved. No cattle were exhibited at agricultural shows or sales.

The seronegative cows were milked first and had no direct contact with the reactors either in the milking parlour or the collecting yard; separate equipment and utensils were used for infected and uninfected groups as far as was practicable. The two groups were grazed separately and had no direct contact whatsoever between the months of April and September. There was insufficient accommodation to house them totally apart during the winter but contact was minimised by dividing up the strawed yard (Fig 6.1). In 1989 and 1990, the small negative group occupied section A; in 1991 the expanded negative group went into sections B, C and D and the remaining reactors moved to section A.

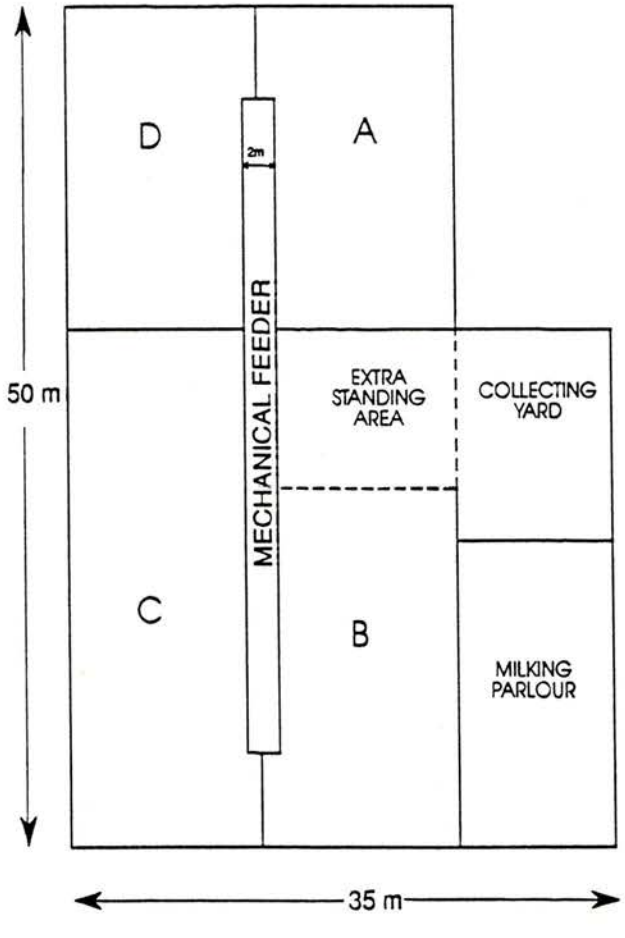

Fig 6.1 : Layout of buildings used to house cattle in D24

Some head-to-head contact was still possible between cattle in sections A and B through unenclosed metal gates which were moved to provide an extra standing area during milking; a mechanical feeder, about two metres wide, separated most of sections A and D.

In addition to segregation, the reactors were vaccinated with "Pneumovac Plus" (C-Vet Limited) in an attempt to reduce virus shedding during winter housing by following the approach adopted in Germany (Meyer and other 1985, Forschner 1988, Wittkowski 1990) as discussed in the literature review. This inactivated (multivalent) BHV1 vaccine had been used for this purpose in Germany until fairly recently (G D Bell, personal communcation). Seropositive cattle received two 2ml doses of the vaccine, intramuscularly in January and February 1989 (about four weeks apart); those still in the infected group were revaccinated in November 1989 and September 1990. The seronegative group was blood tested every two to six months to monitor progress: any new reactors were moved into the positive group and vaccinated. Results were tabulated to show the date (month/year) of each blood test, the number of cattle in infected and uninfected groups and the number of new reactors.

High prevalence suckler herd

Background details

This herd (listed as S20 in the previous chapter) comprised about 100 adult cattle and 150 followers in April 1990. The cows were managed

in four separate groups each containing at least one bull; calving mainly took place in the months of December and January. Some of the female calves were retained as breeding replacements to calve at two to two-and-a-half-years of age; the remaining calves were fattened intensively. Uncalved heifers were grazed separately from the cows but all age groups, including fatteners, were housed together during the winter.

Fifteen (55.6 per cent) of 27 randomly selected cows were seropositive for BHV1 at an initial part-herd screening test in November 1989. A subsequent herd test in April 1990, of all 154 cattle aged at least nine months (ie to avoid MDA), revealed that 73 (47.4 per cent) were seropositive, as were 55 (44.0 per cent) of the 125 aged two or more years; the full age-specific prevalence pattern was shown in the previous chapter (Table 5.8). One of the 12 cows (a three-year-old) that were seronegative in November 1989, had seroconverted by April 1990, thereby confirming recent virus circulation in the herd.

Elimination strategy

The plan was to attempt to reduce virus circulation - particularly the high rate of seroconversion amongst heifers - for at least a year and then to embark on a formal test-and-cull programme. The management system did not readily lend itself to group segregation and it was therefore proposed to rely entirely on vaccination (as described for D24) to reduce or stabilise virus shedding. No

additional procedures were adopted apart from ensuring that no other BHV1-seropositive cattle entered the herd. For ease of management and to reduce costs, testing for BHV1 to monitor progress was undertaken to coincide with the qualifying and periodic tests for the EBL Attested Herd Scheme (Chapter 5).

All reactors at least nine months old identified in April 1990 were vaccinated with "Pneumovac Plus" in May 1990 and revaccinated four to six weeks later. All previous non-reactors and other cattle that had by then reached nine months of age were tested in September 1990: any new reactors were vaccinated as above. All reactors first identified in April 1990 received a booster vaccination in October or November 1990 before calving. Non-reactors and previously untested cattle aged at least nine months were examined for BHV1 antibodies in May 1991. The numbers of new reactors detected at each blood test were recorded and the results tabulated according to age and date of test.

RESULTS

Seronegative herds

As shown in Table 6.2, a reactor was found in only one of 32 second or subsequent tests (involving a total of 3138 sera) performed over a period extending to almost four years in the 14 herds that were uninfected at the first test. The reactor (in S11) was a three-year-old heifer that had been seronegative when tested on three

Table 6.2: Subsequent tests for BHV1 antibodies in CHS herds containing no adult reactors at the first test

Herd	1st Test			2nd Test			3rd Test			4th Test			5th Test		
	Date	No	+	Date	No	+	Date	No	+	Date	No	+	Date	No	+
D1	3/87	227	0	1/88	373	0	12/88	207	0	1/90	176	0	1/91	190	0
D6	10/87	189	0	2/88	303	0	2/89	311	0	2/90	309	0	2/91	215	0
D10	11/88	114	0*	4/89	172	0	12/89	136	0	3/91	104	0			
D19	9/90	52	0	2/91	84	0		-			-			-	
S2	2/88	10	0	6/88	22	0	7/89	15	0	4/90	25	0	6/91	13	0
S9	1/89	41	0*	6/89	42	0	1/90	21	0	1/91	49	0		-	
S11	5/89	25	0*	10/89	37	0	1/90	57	0	1/91	24	1#		-	
S14	7/89	4	0	11/89	7	0	10/90	5	0		-			-	
S15	7/89	5	0	1/90	7	0	1/91	7	0		-			-	
S16	11/89	9	0	3/90	26	0	8/91	15	0		-			-	
S21	5/90	18	0	9/90	18	0		-			-			-	
S22	6/90	6	0	2/91	15	0		-			-			-	
S24	10/90	25	0	3/91	52	0		-			-			-	
S28	12/90	40	0	4/91	101	0		-			-			-	

Date Month/Year
No Number of cattle tested
+ Number of reactors

* Single reactor <2 years old detected at first test
\# Suspected vaccinate

previous occasions and was one of four suspected vaccinates imported from continental Europe two years previously. One other member of this group was seropositive when first tested as a yearling (Table 5.6) and had since been culled.

Low prevalence herds

Herd managers were often reluctant to sell large numbers of reactors at once and frequently opted for their phased removal over several months or longer (ie 19 months in D9 and 11 months in D17) and made no special efforts to keep them segregated until a month before the second test.

BHV1 antibodies were found in five herds (eight cattle) from 45 second and subsequent tests, (involving a total of 5,506 sera), undertaken after the removal of the initial reactors. In four of the herds (D17, S10, S12, S33) new reactors were detected at the repeat test (Table 6.3) performed after removal of reactors from the first test. In S8, reactors were detected after at least 19 months freedom from infection (Table 6.3); details of this incident are given in the next section.

The three reactors in D17 (cows aged three, six and eight years) were identified 11 months after the initial test. Two of these reactors had calved during the previous four months: for two weeks before parturition, and on the day of parturition, they had been in close proximity to known reactors (awaiting culling) that calved at the

Table 6.3: Subsequent tests for BHV1 antibodies after removal of reactors from infected CHS herds

Herd	1st Test#			2nd Test■			3rd Test			4th Test			5th Test		
	Date	No	+	Date	No	+	Date	No	+	Date	No	+	Date	No	+
D5	10/87	254	14	2/88	252	0	2/89	417	0	2/90	231	0	1/91	272	0
D8	4/88	154	6	8/88	191	0	1/89	229	0	8/90	133	0		-	
D9	7/88	123	6	2/90	118	0	2/91	200	0		-			-	
D11	5/89	117	1	7/89	116	0	11/89	280	0	11/90	127	0		-	
D14	1/90	140	2	5/90	146	0	1/91	296	0		-			-	
D15	3/90	176	7	6/90	161	0	6/91	230	0		-			-	
D17	3/90	260	19	2/91	254	3※		-			-			-	
D18	5/90	121	7	9/90	102	0		-			-			-	
D23	3/91	267	29	7/91	206	0		-							
S1	7/87	24	2	10/87	31	0	4/88	60	0	4/89	42	0	4/90	36	0
S4	2/88	90	3	7/88	75	0	3/89	174	0	3/90	102	0	3/91	101	0
S6	4/88	7	1	1/91	10	0		-			-			-	
S7	11/88	16	1	5/89	38	0	12/89	30	0	2/91	20	0		-	
S8	12/88	60	4	4/89	117	0	8/89	118	0	11/90	65	2*		-	
S10	2/89	29	4	10/89	37	1※	11/90	17	0		-			-	
S12	6/89	56	11	11/89	74	1※	12/90	37	0	4/91	85	0		-	
S13	7/87	25	2	2/90	23	0		-			-			-	
S17	12/89	27	6	4/90	25	0	11/90	78	0		-			-	
S18	1/90	86	4	1/91	85	0		-			-			-	
S32	1/91	35	4	2/91	31	0		-			-			-	
S33	1/91	35	1	5/91	34	1※		-			-			-	

Date Month/Year
No Number of cattle tested
\+ Number of reactors
* Part-herd breakdown (described in text)
※ See text for details
■ Undertaken at least one month after removal of reactors
\# See Tables 5.1 and 5.2 (Chapter 5)

same time. The calves born to the new reactors were seronegative for BHV1 when tested at two and four months of age. In S12, a four-year-old cow seroconverted during the five month period between tests. Unlike in D17, reactors from the first test had been moved to a separate farm as soon as they were identified leaving no opportunity for further contact with seronegative cattle.

In S10, further enquiries indicated that the reactor may have been misidentified when first tested and was probably not a new case. A five-year-old cow in S33 which gave consistently inconclusive results with the ELISA test but which was marginally seropositive with the 24 hour SNT (titre 1/2) was eventually classified as a reactor even though it appeared very unlikely that it was actually infected.

Reinfection of S8

During the summer of 1990 most of the cattle (58 cows) in this herd went out to marshland grazing, apart from a group of six cows with calves at foot, and a two-year-old bull, which remained on upland pasture close to the home farm. In August 1990 an apparently healthy yearling steer of unknown BHV1 status, from a neighbouring farm, broke through double fencing to gain access to the separate group of 13 cattle. The period of time during which direct contact occurred was unknown but it was probably only a few hours. The group remained clinically healthy and the owner decided, without consultation, that the risk of introducing BHV1 had been minimal and chose to ignore the incident apart from improving the fencing.

The main herd returned from marsh grazing in November 1990 and was subdivided into groups B, C and D and housed in a well ventilated purpose-built cattle yard (Fig 6.2). The 13 cattle (group A) returned to the farm buildings on 22/11/90. The herd remained in separate groups during the winter: group A was separated from the cows in group B by a metal gate which permitted head-to-head contact but was kept apart from groups C and D by a three-metre wide feeding passage.

On 29/11/90 all 65 cattle aged two or more years were blood sampled for the annual periodic blood test for BHV1. Group A was put through the race and crush separately from the rest of the herd and there was

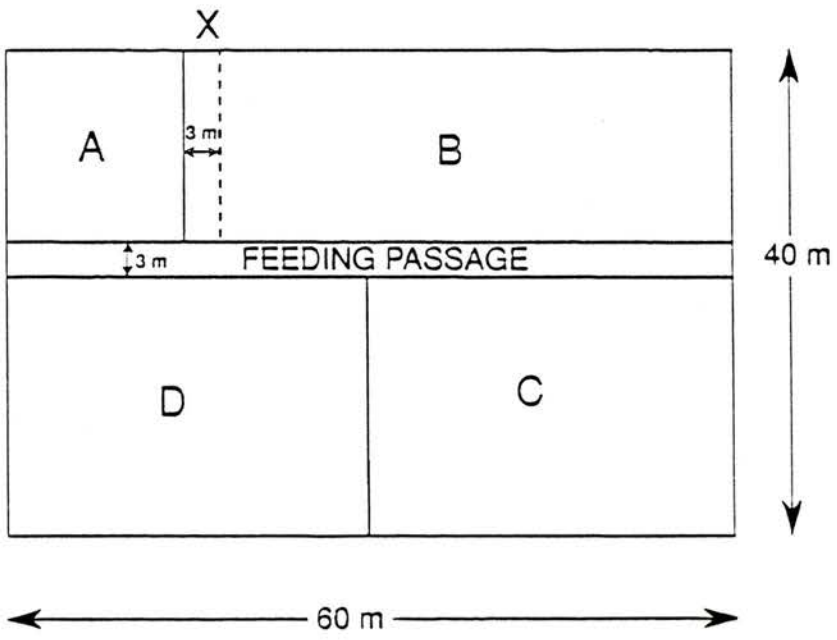

Fig 6.2 : Layout of buildings used to house cattle in S8

no direct contact with the other groups. The serological results (Table 6.4) showed that the bull and cow 1 (both in Group A), were BHV1-positive but the rest of the herd was seronegative. The reactors were isolated as soon as the results were known and the rest of Group A (including the calves) was further separated from the adjacent Group B by installing a three metre barrier of metal gates (shown as X in Fig 6.2); no other suitable isolation facilities were available to house this number of cattle.

The remaining 11 cattle in Group A were retested on 10/12/90, revealing a further reactor - the 10-month-old calf of cow 1. This was removed and the cattle still left in Group A, and all of Group B, were retested on 17/1/91. No reactors were found amongst Group B, but a further reactor, a four-month-old calf (calf 2) was detected in Group A. This calf was removed and the entire herd of 126 cows and calves was retested on 27/3/91 revealing two further reactors, both in Group A - a recently calved cow (cow 3) and her 10-week-old calf. Another reactor (cow 4) was detected in Group D, which was housed in a yard located on the opposite side of the feeding passage. The three new reactors were removed on 30/3/91 and the entire herd retested on 2/5/91: no further reactors were detected. The (isolated) calf of cow 4 (born 16/12/90) was seronegative for BHV1. None of the cattle that seroconverted showed any clinical signs of IBR.

Table 6.4 : New BHV1 reactors detected in blood tests performed after reinfection of S8

Date of Test	Group * A	B	C	D	Identity of new reactors	Date reactors removed
29/11/90	2	0	0	0	Cow 1 Bull	5/12/90
10/12/90	1	NT	NT	NT	Calf of cow 1	16/12/90
17/ 1/91	1	0	NT	NT	Calf 2	27/ 1/91
27/ 3/91	2	0	0	1	Cow 3 Calf of cow 3 Cow 4	30/ 3/91
2/ 5/91	0	0	0	0	-	-

* See Fig 6.2 NT Not tested

Attendance at Agricultural Shows

Twenty-seven (77.1 per cent) of the 35 herds undergoing the CHS programme exhibited regularly at up to seven major agricultural shows each year during the surveillance period: no new reactors were detected in the show groups in any of these herds.

High prevalence dairy herd

Most reactors were culled during 1990 and 1991 to coincide with the main influx of heifers; a few remaining reactors were sold shortly before the final test in June 1991 which confirmed that BHV1 infection had been eliminated within two-and-a-half years. A total of three new reactors were detected (Table 6.5) during this period and were put into the positive group. In addition, an IR detected 2/89 was initially moved into the infected group where it remained for several months before rejoining the uninfected cows after proving to be seronegative with the 24 hour SNT.

Table 6.5: Elimination of BHV1 infection from D24

Date of test (Month/year)	Number of cattle Positive group*	Negative group	New reactors
12/88	106	19	0
2/89	103	19	0
4/89	102	19	1
8/89	100	26	0
12/89	91	27	0
6/90	91	30	2
9/90	63	74	0
4/91	30	88	0
6/91	0	86	0

* Not retested after 12/88

Table 6.6: Stabilisation of BHV1 infection in S20

Year of Birth	Age in years at 4/90	Non-reactors at 4/90	Non-reactors from 4/90 retested 9/90	New reactors detected 9/90	Non-reactors from 9/90 retested 5/91	New reactors detected 5/91
1990	<1	NT	17*	0	26**	0
1989	1	11	11	1	8	0
1988	2	29	28	0	28	0
1987	3	17	16	7	9	0
1986	4	6	6	1	5	0
1985	5	3	3	2	0	0
1984	6	9	9	1	4	0
1983	7	3	2	0	1	0
1982	8	0	0	0	0	0
1981	9	1	1	0	0	0
≤1980	≥10	2	2	1	0	0
Totals		81	78	13	81	0

* Not previously tested (aged <9 months at 4/90)
** Including 19 not previously tested (aged <9 months at 9/90)
NT None tested

Reactors vaccinated 5/90 and 6/90; revaccinated 10/90 or 11/90

High prevalence suckler herd

Antibody prevalence amongst cattle aged at least nine months declined from 73 (47.4 per cent) of 154 animals tested 4/90 to 65 (43.1 per cent) of 151 tested 9/90 and 54 (39.1 per cent) of 138 tested 5/91. Table 6.6 shows the number of new reactors detected according to age cohort; some cattle were sold as the result of management policies unrelated to BHV1 status and could not be retested. Of the 81 non-reactors from 4/90, 78 were retested 9/90 and 13 (16.7 per cent), including seven three-year-olds, had seroconverted. None of the 17 calves that were less than nine months old and therefore not previously tested, were positive at the 9/90 test but one born in February 1989 and inadvertently omitted from the 4/90 test, was found to have seroconverted.

At the final test carried out 5/91, there was no evidence of new infection amongst the 62 cattle that were negative at all previous tests or amongst the 19 that were too young to be tested earlier. Seroconversion was not detected in any cattle born after April 1989.

DISCUSSION

The relative ease with which BHV1 was eradicated from LP herds using a simple test-and-removal approach supported previous observations concerning the virtual absence of virus circulation in these herds. Excluding the apparently misidentified reactor in S10, and the

anomalous IR in S33, virus transmission had occurred between qualifying tests in just two herds (S12, D17). Both of these initially contained quite large numbers of reactors which inevitably increased the potential for naive animals to be exposed to infection. However, spread of virus was not detected in other herds such as D5 or D23 which contained similarly large numbers of reactors at the first test.

The adoption of a phased programme for the removal of reactors from LP herds generally appeared to be justifiable since it enabled producers to sell when cull cow prices were favourable or after reactors had calved. In D17, however, it appeared that this somewhat casual approach to eradication was responsible for spread of virus to three other cows. In this herd, the findings suggested that parturition provided the stress factor (Thiry 1985) that precipitated viral reexcretion and transmission by known reactors still in the herd: had infection been transmitted more than about two weeks before parturition then the calves born to the reactors would almost certainly have been seropositive for BHV1 through MDA. The evidence from D17 suggested that the small risk of virus transmission by retained reactors in LP herds could be reduced by segregating them at calving.

Elimination of BHV1 infection from HP herds required a more aggressive approach than that adopted in LP herds: a simple test-and-cull programme was not feasible because of the high rate of virus transmission. However, test-and-removal at short intervals was

used to successfully eliminate rapidly spreading infection from S8. This herd originally contained four reactors - all foundation herd members - when first tested in December 1988. The apparent lack of virus transmission to other cattle in the herd from these initial reactors contrasted markedly with the rapid spread to a total of seven cattle (about one new case per month) after the virus (presumably a different strain) was reintroduced two years later following a breach in herd security. It seemed probable that if virus transmission had been left unchecked this herd would soon have reached HP status.

In S20, vaccination alone proved surprisingly successful in halting the spread of virus. Preliminary serological findings in this herd had indicated that virus circulation amongst yearlings born in 1989 was superimposed on the typical HP herd pattern of seroconversion after first calving. No significant changes in management practice were adopted in this herd between 1989 and 1990, yet with the commencement of the vaccination programme no new reactors were found amongst cattle born after April 1989. This evidence suggested that virus ceased circulating after the third dose of vaccine, which supported similar claims by Forschner (1988). Wittkowski (1990) noted a three to five per cent quota of new reactors in heavily infected herds in Bavaria following a similar vaccination-based eradication programme even though the overall proportion of seropositive cattle declined by nine or 10 per cent annually. Unfortunately S20 was dispersed later in 1991 and the period of the trial could not be extended.

It was difficult to assess whether the elimination of BHV1 infection from D24 was achieved by separation alone or whether vaccination played a significant part. On-farm circumstances unfortunately dictated that separation between infected and uninfected groups was not complete: reliance was therefore placed on vaccination to reduce virus shedding and transmission during the critical winter housing period although the time period (nine to 10 months) before revaccination was greater than the six months advised by Forschner (1988). In the event, the detection of three new reactors was not unacceptable. After initial failure when relying on separation alone, Ackerman and others (1990b) subsequently eliminated BHV1 from a calf fattening unit when additional strict hygiene precautions - far more stringent than those adopted here - were introduced.

The inevitable constraints imposed on field trials performed under farm conditions meant that it was not feasible to set up control groups for the strategies adopted in D24 and S20. However, the previously established histories of extensive active infection in these herds provided some basis for comparison. Whilst the results were encouraging, it must be accepted that the studies were not exhaustive and further similar longer-term investigations, including assessments of the effect of repeated vaccination on humoral antibody titres, are needed to verify the findings.

Reactor misidentification and inconclusive test results were subjects frequently raised by farmers. It was inevitable that

misidentification occurred occasionally in blood tests involving large numbers of animals sampled under farm conditions: similar problems were noted by Wittkowski (1990). Fortunately, owners invariably agreed to have reactors retested as a safeguard against potentially costly mistakes, several of which were averted by such action. Regarding the question of IRs, a simple cut-off point with the ELISA test was originally envisaged to separate reactors from non-reactors. However, it soon became clear that although the ELISA test performed consistently well for herd screening purposes it tended to lack specificity at low antibody titre. Although an IR category requiring resampling was subsequently introduced, it was not until 1990, when the 24 hour SNT was routinely used for IRs that the main difficulties were overcome. A few cattle (<0.2 per cent) were consistently IRs with ELISA and although most of these subsequently proved to be seronegative with the 24 hour SNT, owners usually decided to cull them because of their potential nuisance value in eradication programmes. Animals showing fluctuating inconclusive results with ELISA often had a history of vaccination or suspected vaccination.

Once BHV1 had been eliminated, herds remained free of infection during the follow-up period despite regular attendance at agricultural shows or sales where cattle were often housed close to others of unknown BHV1 status for several days. These observations further indicated that reactivation and reexcretion of BHV1 is an uncommon event, even in the apparently stressful environment of the show ring. However, if a virus strain of the type that reinfected S8

was in circulation then rapid spread and widespread seroconversion could be envisaged.

The one month isolation and testing routine used for added-animals proved to be a successful precautionary measure for keeping infection out of seronegative herds. It was used not only for show animals and purchased replacements but also for calves born to reactors. In the latter situation, where adequate isolation facilities were available (preferably on separate premises) owners often continued to breed from seropositive cows, subsequently introducing their calves into the negative herd after they had been tested seronegative for BHV1 at least a month after MDA had disappeared - usually by about six months. One herd (S4) retained a seropositive bull on a separate farm and used it on successive groups of seronegative heifers for three seasons. The heifers were removed from the bull and tested for BHV1 antibodies after a month's isolation: none of them seroconverted despite being in close contact with the bull for periods of up to three months.

CHAPTER 7: GENERAL DISCUSSION AND MAIN CONCLUSIONS

Norfolk, with its numerically small cattle population, widely dispersed within a predominantly arable landscape, provided an ideal area in which to undertake this form of study. Two main sources of information were available to the author: heterogeneous diagnostic submissions to Norwich VIC and data from CHS herds. Each related to essentially different, yet sizeable sections of the target population and when considered together provided a comprehensive basis for epidemiological analysis.

Unlike experimental investigations performed under controlled conditions, observational field studies can suffer from numerous constraints on data collection (Thrusfield 1988) but apart from inevitable sample bias no significant problems were encountered here. Because of regional differences in stock concentrations, husbandry methods and disease prevalence, attempts to extrapolate the present findings to herds elsewhere in Britain should be undertaken guardedly. However, it was reassuring to note that the main conclusions were supported by similar observations made to the author by veterinary colleagues elsewhere including general practitioners and those in the SVS responsible for CHS herds in other parts of Britain.

Although IBR failed to emerge as a serious threat to cattle herds in Norfolk during the 1980s, outbreaks occurred sporadically and by 1989 clinical disease had been confirmed (mainly by FAT or paired

serology) in about 11 per cent of dairy herds and five per cent of suckler herds in the county. In rearing/fattening herds the disease was seen mainly during the winter months but outbreaks in adult breeding cattle occurred throughout the year.

Retrospective analysis of laboratory serological submissions indicated that BHV1 antibodies were widely distributed in Norfolk herds. The best estimate (based on abortion serology) suggested that about 28 per cent of adult cattle were seropositive; 44 per cent of herds contained at least one reactor. The overall findings, including crude comparisons with seroprevalence in CHS herds in other regions (L A S Gibson, personal communication) suggested that cattle in Norfolk had a slightly lower prevalence of BHV1 infection than those in Britain as a whole. It should be appreciated, however, that the seroprevalence (15.1 per cent) amongst cattle in the 56 CHS herds featured here was artificially low because four large herds with a recent clinical history of IBR and vaccination were excluded from the study population at the outset.

The analysis of archival data highlighted the importance of recrudescence as a recognisable clinical entity in endemically infected herds. In the literature the term "recrudescence" has been used mainly to describe reactivation and reexcretion of infection by latently infected carriers rather than resurgence of disease amongst immunologically naive in-contacts, which appeared to account for most incidents recorded here. The significance of clinical recrudescence has been virtually ignored by previous authors yet in dairy herds it

accounted for almost as many confirmed IBR outbreaks as did epidemic disease: significantly, VIDA II fails to distinguish between epidemic and recrudescent disease outbreaks. In addition to causing economic loss, recrudescence is an outward demonstration of virus circulation in affected herds - a factor of major importance when considering programmes to eliminate infection. Observations from CHS herds supported the hypothesis that overt recrudescence is essentially a feature of herds with a high antibody prevalence. Despite sporadic recrudescences, most BHV1 infections in these herds appeared to be subclinical although anecdotal evidence suggested that some HP herds had experienced previously unrecognised morbidity associated with the spread of virus amongst naive young cows.

The widely differing rates of virus transmission in HP and LP herds were probably attributable to infection with different strains of virus but it was beyond the scope of these studies to investigate this aspect further. Individual herd histories showed that HP status sometimes followed epidemic IBR outbreaks but this was not an inevitable sequel: some HP herds had no history of epidemic disease. In all four HP dairy herds antibody prevalence amongst adult cattle exceeded 60 per cent, whereas it was somewhat less in two of the three HP suckler herds. This probably reflected management and husbandry factors instead of inherently different patterns of virus activity. The relatively small average herd size and higher proportion of purchased animals accounted for the wider seroprevalence range in LP suckler herds compared with LP dairy herds. Regardless of herd type, virus circulation is likely to be

negligible if seroprevalence amongst adult cattle is less than 20 per cent whereas extensive active infection is almost certainly present if it exceeds 60 per cent. In herds of intermediate prevalence the seroconversion pattern should be examined in relation to herd size, management practices and origin of reactors before defining status.

Previous authors have commented that IBR outbreaks are often preceded by the introduction of added-animals. Similar observations were made with outbreaks in Norfolk as noted in Chapter 3. However, clinical disease outbreaks suprisingly failed to follow the introduction of presumably latently infected carriers into susceptible CHS herds. This supported the contention that reactivation and reexcretion of latent virus is a relatively uncommon event under natural conditions although it could also be attributable to low virulence (including vaccinal) strains of virus.

The association between reactor status and purchase appeared partly to reflect a higher antibody prevalence in herds located outside Norfolk from which most purchases were derived. The subsequent lack of spread from purchased reactors made them appear all the more conspicuous in herds that otherwise contained few, if any, seropositive cattle.

A small number of homebred reactors were present amongst second and third parity cows in LP herds. These were probably the result of outside contacts with infected cattle or virus transmission from older (often purchased) reactors in the same herd. Where there were

substantial numbers of homebred reactors in this age range (ie two-and-a-half to five years) it was invariably an indication of HP status. With some 50 to 75 per cent of young cows in HP herds likely to have seroconverted, only a few (ideally homebred animals) would need to be sampled for BHV1 antibody screening to ascertain the seroconversion pattern of individual herds. This approach would provide a low-cost alternative to the initial whole or part-herd screening tests for BHV1 infection currently used in the CHS. Once the BHV1 status of herds has been established, owners can be advised on appropriate methods to control or eliminate the infection.

It is widely recognised that semen from infected bulls is a potential source of BHV1 infection, either through natural service or AI. With the introduction of strict measures to control IPV/IPB, most bulls now held at AI studs in Britain are seronegative for BHV1 and semen from the few seropositive bulls is routinely examined for the presence of virus before use. It was not possible from these studies to assess the possible role of semen in the transmission of BHV1 in Norfolk herds during the 1980s: virtually all herds used AI and even if semen transmission had occurred despite precautions, as in the incident reported by Kupferschmied and others (1986), this route of spread was almost certainly of minimal significance compared with the effects of purchased reactors and other direct contacts with infected cattle.

BHV1 also has the potential to be passively carried by embryos during ET activities but in his review, Straub (1990a) concluded that this

did not pose any significant risk and there was no circumstantial evidence from these present studies to suggest that the virus had been introduced by this means. ET recipients were frequently seropositive through vaccination but as with other seropositive dams their calves were invariably negative for BHV1 antibody by about six months of age.

Although not specifically examined here, there was no circumstantial evidence to suggest that long-distance airborne spread occurred with BHV1 as has been demonstrated with ADV, which is also an alphaherpesvirus (Gloster and others 1984). The possibility of infection being acquired through contact with other ruminants (Straub 1990b) was similarly undetermined although it appeared to be unlikely. Nixon and others (1988) suggested that testing deer or goats was unnecessary for BHV1 control programmes.

It was evident both from the analysis of diagnostic data and from CHS serological findings that antibody prevalence was at its lowest amongst cattle aged between six and 12 months (ie after MDA had declined). This supported clinical observations presented in Chapter 3 regarding the relatively low incidence of disease amongst this age group. Antibody prevalence subsequently increased with age after first calving, coinciding with heifers returning to their parent herds from separate grazings. Although antibody prevalence increased at contrasting rates in the CHS LP and HP herds, by

10 years of age nearly 50 per cent of all cattle tested were reactors, regardless of the antibody prevalence category of the herds involved.

Clearly, the longer that susceptible cattle remain in infected herds the more likely they are to acquire infection and seroconvert. However, in LP herds, seroprevalence generally remained low until about six years of age before rising sharply. This suggested that BHV1 infection may previously have been more prevalent in these herds but, unlike in HP herds, it had subsequently failed to maintain itself. Provided infection is not reintroduced, LP herds with their intrinsically slow rate of virus spread, might be expected to eventually achieve seronegative status of their own accord merely by the routine culling of older (and mainly seropositive) cattle.

It is common management practice in dairy herds, but to a lesser extent in suckler herds, for replacement heifers to be reared completely separately from the adult herd until after first calving. The disadvantage of this approach for disease control is that it fails to naturally immunise young cattle against infections endemic in the adult herd. The present observations indicated that the arrival of naive heifers into HP herds sometimes provoked recrudescent outbreaks of IBR: analogous findings were noted by Hathaway and Little (1983) amongst second parity cows in dairy herds endemically infected with Leptospira interrogans serovar hardjo. Despite this drawback, rearing heifers apart from the adult herd permits the establishment of a population of seronegative breeding

cattle of the main age range for national and international trading purposes, despite high levels of residual infection being present in the main herd. This explained why some owners had successfully exported young BHV1-seronegative cattle without realising that their adult herds contained numerous reactors.

The apparent absence of virus transmission in most infected herds suggested that the excessive provocation afforded by the high doses of corticosteroid used in experimental reactivation studies may not be relevant to the field situation. Observations in one CHS herd (D17) supported the findings of Thiry and others (1985) regarding the role of parturition as a stimulus for virus reexcretion. Reactors retained pending culling from herds undergoing programmes to eliminate BHV1 infection should therefore be calved in isolation. It would also be advisable for them not to receive corticosteroid therapy, even though dose rates used in veterinary practice are somewhat less than those used experimentally.

In LP herds, BHV1 infection was readily eliminated by following a simple test-and-cull approach, phased if necessary over a period of months or even years. By adhering to CHS rules governing added animals and contact with cattle of unknown BHV1 status, herds subsequently remained free of infection during the follow-up period, despite continued attendance at agricultural shows. However, the introduction of rapidly spreading infection in one IBR monitored negative herd sounded a note of caution regarding the possibly disastrous consequences of failing to maintain adequate farm

boundaries and other disease security precautions. Further longer-term studies are needed to establish whether herds can remain free of infection beyond the three to four years examined here.

The high rate of seroconversion amongst second and third parity cows in HP herds presented a major obstacle to removing infection. In these herds, strongly motivated owners could adopt programmes based on separation and, or vaccination, similar to those described here. They could even consider depopulating and restocking. However, in most HP herds these were not economically viable options. Furthermore, provided the young stock and the adult herd were kept apart, owners could still confidently expect to maintain the enhanced sale potential of seronegative heifers or bulls once MDA had waned. In herds in which recrudescence was a problem, heifers could be vaccinated intranasally with a modified live virus vaccine before entering the main herd.

The strategic use of inactivated vaccines to reduce virus shedding by latently infected carriers currently forms an integral part of voluntary control schemes for BHV1 infection in Germany. It is not clear from the German literature whether the apparently successful use of inactivated vaccine to stabilise virus transmission applies equally to herds in the high prevalence category and to those containing only a few reactors (ie LP herds). As demonstrated in these present studies, the latter herds would be expected to exhibit only minimal virus shedding even without vaccination. Despite the widespread promotion of these vaccination programmes there is

little published scientific evidence to support their efficacy - a conclusion also reached by Gibson and Edwards (1991). Furthermore, the crucial concept that virus excretion is reduced if humoral antibody titres remain high (Meyer and others 1985, Forschner 1988) has recently been challenged by Straub (1991a). Also, in a recent small study at the CVL, "Pneumovac Plus" failed to reduce the titre of virus shed from experimentally infected calves following the administration of dexamethasone (S Edwards, personal communication). Despite these reservations, the use of inactivated vaccine in the present studies seemed to be remarkably successful. Although the contribution made by partial separation to the programme adopted in D24 could not readily be separated from the effects of vaccination, it appeared that vaccination alone had halted virus transmission in S19.

Straub (1991a) has proposed a rather complicated control scheme for BHV1 using either inactivated or live vaccines in a schedule based on herd size and antibody prevalence rate. In the same publication the author claims, without apparent scientific proof, that virus shedding ceases after three doses of live intranasal vaccine have been administered. Many questions concerning this whole topic remain unanswered and there is clearly a need for further investigations - preferably controlled field trials - to fully evaluate the effects of vaccination on virus shedding by latently infected carriers.

It is noteworthy that some of the previously unreported epidemiological findings from these present studies bear marked

similarity to published work on ADV infection of pigs. This closely related virus has been the subject of eradication and control schemes in several European countries, including Britain (Watson 1986) similar to those adopted for BHV1. ADV can also establish latent infection resulting in clinical recrudescence several years later (Basinger 1979) affecting up to about five per cent of adult pigs (E N Wood, personal communication). Offspring of seropositive sows can be raised as seronegative replacement stock (Thawley and others 1980) as demonstrated here with BHV1.

Several recent field studies with ADV infection in the USA have revealed further close parallels with the epidemiology of BHV1 infection as observed in Norfolk herds. Annelli and others (1991) found that ADV infection failed to spread in 23 of 27 herds containing single reactors and they attributed this to low virulence strains of virus rather than environmental factors. Similarly, Morrison and others (1991) noted that 42 per cent of quarantined herds contained less than 20 per cent of adult reactors. Claiming this as a major new finding they concluded that ADV infection stops spreading in most herds after the initial epidemic. Duffy and others (1991) noted a positive association between the spread of ADV and the housing of gilts in the same building as sows, which supports comparable observations with BHV1 infection in HP suckler herds.

In Europe, interest in BHV1 infection is likely to increase during the next few years. The removal of trade barriers and harmonisation of animal health controls within the EC after 1992 will highlight

regional differences in patterns of disease, including those caused by BHV1. Recent EC directives have emphasised the importance of BHV1-seronegative status for international trade in embryos and semen. Compulsory eradication programmes for BHV1 are already underway in Denmark and Switzerland; in Germany and France cattle producers have been encouraged to join voluntary eradication schemes.

The introduction of the CHS belatedly awakened cattle producers in Britain to the potential benefits of voluntary improvements in health status and herd security - aspects of disease control that have been well appreciated by other livestock sectors, particularly pigs, for some years. Enthusiasm was partly dampened by export restrictions imposed following the emergence of bovine spongiform encephalopathy (BSE) (Wells and others 1987) but when export restrictions are eventually lifted, the industry can be expected to show renewed interest in eliminating BHV1 infection. Organisers of major agricultural shows may even decide to follow the lead set by "Tier und Technic '89, in Frankfurt, Germany, by regulating the BHV1 status of cattle being exhibited.

After BSE and EBL, BHV1 infection is a possible choice for a national eradication programme in Britain during the next decade. Whether this occurs will depend on political and trade pressures. It is hoped that the studies described in this dissertation have gone some way towards improving the understanding of the epidemiology of BHV1 infection and will provide a useful basis for discussions concerning its eradication at herd or national level.

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APPENDIX 3.1 : LABORATORY SUBMISSION FORM

Ministry of Agriculture, Fisheries and Food
VETERINARY INVESTIGATION SERVICE

ADAS

V.I.Centre Reference
Date of receipt _____

1. Name and full farm address of owner _____

Date collected _____

2. Name and full postal address of veterinary surgery (to which report will be sent)

3. For the attention of _____
Previous reference (if applicable) _____

4. Examination required

5. Type of specimen _____

6. Species/Breed _____

7. Identification _____ 8. Age _____ 9. Sex _____

10. State MAFF Health Scheme (if relevant) e.g. Pig/Poultry Health Scheme. _____

11. Please give case history e.g. clinical signs/treatment/PM findings etc.

FLOCK/HERD DETAILS

12. No. of losses _____ 13. Date losses commenced _____ 14. No. in group at risk _____

15. No. affected _____ 16. Date purchased _____ 17. Total stock _____

18. Flock/Herd treatment (including vaccinations/anthelmintics)

19. Housing/Husbandry

20. Feeding

21. Other information

APPENDIX 3.2 : ABORTION REPORT FORM

Ministry of Agriculture, Fisheries and Food
Scottish Office Agriculture and Fisheries Department
Welsh Office Agriculture Department

Please use BLACK BALL POINT PEN

Brucellosis: Report on Investigation of Abortion or Calving

1. (a) Name and address of owner

Postcode Telephone No.

(b) Address where herd is kept (if different from above)

Postcode

2. Ear No. of Dam
3. Breed of Dam
4. Vaccination Status of Dam (tick appropriate box)
   None ☐ S19 ☐ Not known ☐
5. Date of abortion / calving
6. Place of abortion / calving (tick appropriate box)
   isolation box ☐ stall ☐ yard ☐ pasture ☐ elsewhere ☐
7. BS 27: has been served ☐ has not been served ☐
8. Is Dam:
   (a) Homebred ☐ Purchased ☐ Had previous normal calving in this herd ☐
   (b) IR ☐ Dangerous contact ☐ Tracing ☐
   (c) If so, is it in isolation? YES ☐ NO ☐
9. Visit No. 1 ☐ 2 ☐ 3 or more ☐

Report to be sent to Animal Health Office

10. CPHH No.
11. Duration of pregnancy
    Under 150 days ☐
    150 — 179 days ☐
    180 — 209 days ☐
    210 — 239 days ☐
    240 — 269 days ☐
    270 days & over ☐
    Unknown ☐
12. Calves born: All live ☐ Any dead or foetus not found ☐
13. Date of sampling
14. Age of Dam (in years)

Note: Samples submitted will be examined for evidence of Brucella abortus infection. If additional examinations for other pathogens are required please submit a VIO 16 form with this form giving all relevant information including the tests required. These additional tests will normally attract a charge.

Signed LVI / VO

Date

Practice address

Postcode Telephone No.

Laboratory Results

Comments	Sample (Delete if not sent)	Sample Reference No.	Lab. Ref. No. / Date Received
	Blood		RBPT — + — ☐ SAT (Titre) — + 0 — ☐ CFT (Titre) — + 0 — ☐ Other Tests
	Milk / Colostrum		Summary Culture (+ or —) ☐
	Vaginal Swab		
	Placenta / Stomach contents		

Signed

Date

To be completed in Animal Health Office

15. Is this a normal calving in an infected herd? YES ☐ NO ☐
16. Is this enquiry associated with tracing of a DC? YES ☐ NO ☐
17. Action required

18. Test result: Pass ☐ Fail ☐ Inconclusive ☐
19. Officer sampling: LVI ☐ MAFF Officer ☐

Signed DVO / VO Date

BS 7 (Revised 1989)

3&8 / MAR 91 / PR0288 / 200812

APPENDIX 5.1: CATTLE HEALTH SCHEME QUESTIONNAIRE
(Adapted from MAFF form CH3: details not applicable to this study have been omitted)

1. Date of visit

2. CPH Number

3. Owner's name, address and telephone number

4. Address of Scheme (where *different/additional to 3)

5. Name, address and telephone number of farm manager or agent (if different to 3 above)

6. OS map reference number of Scheme premises

7. Type of herd: Dairy = D, Beef Suckler = S, Mixed = O

8. Main breeds

9. Address of other herds in the same ownership

10. Name, address and telephone number of owner's veterinary surgeon

11. Herd details:

Pedigree Non-pedigree

Dairy | Mixed | Beef Suckler | Fattening | Heifer rearer | Dealer | Other

Flying herd | Self contained herd | Open herd with purchased stock

12. If purchased give details of sources and total number of cattle purchased in previous 10 years.

13. Method of mating cows:

Own bull | Hired/loaned bull | AI | ET

14. Details of any movement/contact between this herd and any other herd in different ownership

15. Agricultural show attendance - approximate number attended each year

16. Stock numbers:

Bulls
Cows and heifers (over 24 months)
Heifers (between 12-24 months)
Young stock (other than steers)
Steers

Totals

17. Details of premises:

(a) Isolation of the herd - are the boundaries satisfactory with a minimum separation of 3m between scheme and non designated stock?

(b) Describe isolation premises and state number of cattle that can be accommodated and whether the premises can be cleansed and disinfected

(c) Are isolation facilities adequate for IBR control?

18. Is there more than one herd in the same ownership?
If yes, give details of contact between the herds

19. Details of grazing policy:

    Minimal grazing Seasonal grazing Total grazing

    (a) Temporary grazing

    (b) Communal grazing

20. Details of visitors and vehicles - describe routine precautions

21. If AI is used is the owner aware of the restrictions on the use of semen from seropositive bulls?

22. Has IBR ever been diagnosed in the herd previously?

23. Has IBR vaccine been used in the herd?

24. Any other relevant information or remarks