

Objective measurement of imitation problems in autism

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Abstract

Imitation is a complex behaviour used to allow faster learning of skills, including pivotal social cognitive processes such as language and gesture. Difficulties in imitating others have been broadly found within Autistic Spectrum Disorders (ASD) populations. This paper discusses two possible theories explaining these deficits: self-other mapping theory, whereby imitation deficits in ASD have been proposed to restrict the ability to map relationships between social representations of others and themselves; and self-other comparison theory, whereby the individual must distinguish similarities and differences between themselves and the other, which are then related to emotional and contextual differences learnt through experience in order to provide emotional context.

Whilst imitation difficulties have been widely reported, the recordings of such difficulties have been done subjectively. This paper, however, approaches this well-known phenomenon objectively through the use of a clinical-kinematics assessment tool (C-Kat). Furthermore, this paper discusses different ways to precisely describe the imitative act to be copied and the performance of the imitator.

This paper aimed to objectively investigate whether, when compared to typically developing peers, an imitative deficit was present in ASD adolescents (ASD $n = 16$; TD $n = 24$). Secondly, it aimed to determine whether such a deficit existed only for bodily imitation. Results showed a clear group difference and suggested a developmental delay in imitation ability within ASD rather than a deficit. Furthermore, results suggested a specific ASD difficulty in bodily imitation. However, following comparison of imitation stimuli and measures, the possibility is discussed that these results may be due to focusing on elements other than the critical movement information within the action to be imitated.

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1. Introduction

Within the last decade, there has been an exponential increase in Autism research (Tager-Flusberg, 2007), with the neuropsychological developmental disorder now being understood to be part of a broader phenotype (Folstein et al., 1999) with sub-syndromal symptoms found across populations (Fombonne, 1999). Despite numerous attempts, it is perhaps due to the disorder's complex dimensional clinical picture that no underlying mechanism has been found to account for the variety of symptoms and the range of their expressions (Williams, Whiten, Suddendorf & Perrett, 2001; Tager-Flusberg, 2007). Nevertheless, the Autism Spectrum Disorders (ASD) are consistently characterized by specific developmental impairments and deficits in social interaction and interpersonal communication (Kanner, 1943). Whilst the Theory of Mind hypothesis by Simon Baron-Cohen, Leslie and Frith (1985) provides a cognitive explanation for these social and communicative symptoms of ASD (Tager-Flusberg, 2007); a ToM deficit appears to be unsatisfactory as a primary root of ASD, because whilst ToM (as tested by Baron-Cohen et al., 1985) generally becomes stable at age 4, ASD can be apparent at an earlier age (Tager-Flusberg, 2001). Furthermore, the universality and specificity of a ToM deficit in ASD has been queried due to evidence of children with ASD passing ToM tasks whilst children with other disorders fail them, for example non-signing deaf children, (Peterson, Wellman & Liu, 2005; Tager-Flusberg, 2007). Additionally, ASD can also be characterized by social and non-social abnormalities not explained by the ToM hypothesis, for example delayed and deviant language development and repetitive behaviours (Tager-Flusberg, 2001). Therefore another route of investigation is required, to which Rogers and Williams (2006) note that there is strong evidence supporting the preponderance of the difficulties ASD individuals have in coordinating themselves not only psychosocially, but also at a physical, bodily level – particularly in imitation.

Imitation is a complex behaviour illustrated across many species at all ages and levels of intelligence, not only guiding behaviour but also providing a faster route to learning skills which would otherwise require time consuming trial-and-error learning (Bekkering, Wohlschläger & Grattis, 2000). Such skills include pivotal social cognitive processes such as language and gesture, which form foundations to future developmental accomplishments (Hepburn & Stone, 2006). Previous studies have

shown that childrens' levels of imitation ability can be predicted by abilities of language (measured by verbal intelligence) and social cognitive skills (Charman et al, 2003; Charman, 2006; Williams, Whiten & Singh, 2004). Therefore a direct relationship between imitative ability and social and cognitive development has been proposed (Rogers & Williams, 2006). With infants aged as young as 12-21 days illustrating the ability to imitate diverse facial and manual gestures (Meltzoff & Moore, 1977), this evidence indicates that the direct matching of visually perceived input and motor output starts to develop immediately and is at a vital stage during infancy (Hepburn & Stone, 2006). This early commencement of skill development has stimulated discussion regarding the possibility of imitation being an early indicator of ASD compared to ToM – the development of which reportedly starts much later than that of imitation in typically developing children (TD), at 7 months when infants start to understand the attention of others (Baron-Cohen, 1991).

There is a long history of research regarding imitation ability within ASD, beginning with the first suggestion of a relationship almost 60 years ago (Ritvo & Provence, 1953). However, the details of such a connection were not further reviewed until 1991 when Roger and Pennington outlined the possibility of early abnormal stages of social communicative development within ASD stemming from early motor imitation deficits. They suggested that hindering automatic mimicking of emotion would affect the development of awareness of others as individual, separate beings. This would then have consequences for the development of joint attention, pretend play and communication (both verbal and nonverbal) – elements necessary to establish a functioning ToM (Williams et al., 2001; Rogers & Williams, 2006). In order to imitate, the individual must create an action plan drawing on the perception of another's action, thus requiring the imitator to see the task from the other person's perspective. Therefore imitation can be considered as a precursor to ToM comparable as a form of mental simulation or 'simulation theory of mind' (Carruthers & Smith, 1996). This theory suggests that in order to 'read' another's mind, children put themselves in another's shoes and simulate the mental processes likely to be operated by the other – a mental process similar to that involved with imitation (Whiten, 1997).

Literature consistently shows that those with ASD display difficulty imitating others (Smith & Bryson, 1994; Rogers & Williams, 2006). Evidence ranges from young

ASD children struggling to learn how to brush their teeth by another's example (Rogers, 1999), to paradigms utilizing more robust methodologies where most group differences are found to be at high levels of significance despite testing only 10-20 individuals per group (Williams et al., 2004). With methodologies becoming continuously more rigorous, Williams and colleagues (2001) presented evidence that the magnitude of imitative deficit within ASD could be at least as great, if not greater than that of ToM as, remarkably, ASD imitation deficits have consistently been found across studies differing markedly in the manner the imitation was produced and measured (Hepburn & Stone, 2006).

It is surprising that until recently there has been little interest regarding the relationship between imitation and ASD, especially when the role of immediate and deferred imitation in child development has proved vital to the research of multiple key psychology theories, such as those of Bandura, Piaget and Skinner. Almost ironically, the lack of impromptu imitative interaction with others by ASD individuals is often camouflaged by their characteristic gross lack of appropriate social engagement (Rogers & Williams, 2006). Charman and colleagues (1998) suggested that the acuteness of this imitative deficit could in fact correlate with the severity of ASD. However, an alternative view provided by Hepburn and Stone (2006) suggests that whilst there is a similar pattern of imitation performance across ages for both ASD and TD children, the performance of the ASD children is at levels lower than that of age matched TD children. Nonetheless, there is a gap in the research regarding age-related performance patterns, as there are very few studies of ASD imitative abilities in adolescents and adults (Hepburn & Stone, 2006). From the few investigations available (e.g. Hobson & Lee, 1999; Rogers, Bennetto, McEvoy & Pennington, 1996; Stone, Ousley & Littleford, 1997), it appears that ASD imitative deficits remain throughout adolescence. However, the nature of the deficit changes with an increasing ability to complete simple imitations, whilst the inability to imitate complex or sequential actions becomes more evident due to less practiced motor skills caused by a social interaction deficit now more apparent with age (Tantam, 1991; Hepburn & Stone, 2006).

Theories of imitation problems in ASD

Self-other mapping theory and its expansion

A multitude of theories have been proposed regarding the imitation differences between ASD and TD individuals. Based upon the widely recognised difficulties ASD children exhibit with symbolic play and language development, a problem in creating and adjusting representations of the target action has been put forward (Rogers & Williams, 2006). However, investigations of symbolic and motor representational problems have found little evidence of deficits specific to ASD, suggesting that another element must be involved – perhaps a link between these representations and the self.

By building upon Stern's (1985) model of interpersonal development (Stern, 1998), Roger and Pennington (1991) were the first to theorise how imitative deficits in ASD could restrict the ability to map relationships between social representations of others and themselves. Roger and Pennington further hypothesised that such a representational deficit had a domino effect on the development of social and communicative skills such as shared attention and imaginative play.

With the discovery of mirror neurons (MN) – neurons which activate whilst both watching and enacting actions performed by another (Gallese, Fadiga, Fogassi & Rizzolatti, 1996) – Williams and colleagues (2001) re-examined Roger and Pennington's (1991) theory. The now expanded theory suggests that MN provide the neural mechanism underlying the deficit in mapping between representations of the self and others. A further step taken with this extension of the self-other mapping theory was to propose that instead of using an imitation deficit as an alternative causation of ASD from the ToM theory (Baron-Cohen et al., 1985); Williams et al. likened the imitation deficiency to a simulation deficit. Therefore, as following Gallese and Goldman's (1998) theory of simulation, ASD individuals struggle to imagine themselves in another's place as they fail to relate their observations of others' behaviours to their own neural codings linked to memories of similar behaviours (Williams et al., 2004).

Self-other comparison theory

Reviews of imitative ability in ASD research (e.g. Williams et al., 2004) have highlighted that ASD individuals often complete simple imitative tasks successfully and, rather than disengaging from the task, make errors on more complex tasks. For example, individuals with ASD produced more errors when asked to imitate meaningless gestures (Rogers et al., 1996), or when recreating an irregular action with common objects (Smith & Bryson, 1994). Consequently, it has been proposed that ASD children have a specific problem imitating the *style* of the modeled action (Hobson & Lee, 1999). Perra and colleagues (2008) furthered this train of thought by describing an *adverbial* imitation deficit, whereupon ASD individuals display a specific shortfall in perspective-taking and co-ordinating representations of another onto themselves. As a result, the overall action is successfully completed whilst the method by which the task was completed (the *how* e.g. the speed or the force of the action) is incorrectly imitated. Therefore, these proposals suggest that the basic ability to map from oneself to another is present in ASD individuals, but does not function to the same level as TD individuals, especially for sequences of actions (Williams et al., 2001). Therefore, an essential component for successful imitation is in the detection of similarities and differences between oneself and another – elements which ASD individuals struggle with, especially for complex actions (Williams, 2008; Pennington, Rogers & Williams, 2006).

In social situations, comparing oneself to others is a rudimentary skill (Williams, 2008). Therefore, it is plausible that self-other comparison is required for the development of a ToM through imitation learning. This modified theory proposes that whilst imitating another, the individual distinguishes similarities and differences between themselves and the other, which are then related to emotional and contextual differences learnt through experience. This in turn allows the individual to grasp how the emotional and contextual memories impact upon the different forms of action (Williams, 2008). Therefore, continually comparing oneself with others could not only allow an understanding of how individuals differ from each other, but also how differing behaviour can stem from different motives, thereby allowing the formation of secondary representations required for a ToM (Williams, 2008).

Different forms of imitation

Typically within psychological research, imitation has not routinely been approached as a variably distributed ability within the population; instead it has been typically considered as a universal developmental process occurring in stages. For example, Piaget's classical research considered imitation an ability achieved in the final stages of sensorimotor development, a maturation achieved through successful cognitive representations (Flavell, Miller & Miller, 2002). As illustrated by the infantile studies by Meltzoff and colleagues (e.g. Meltzoff & Moore, 1977), a more modern approach to imitation is that of a landmark ability, similar to that of walking in that it is a skill individuals achieve at a basic level early in life in order to learn more effectively from the world. Before the time of Piaget's work, Thorndike (1898, cited in Thorndike, 2000) clearly stated that imitation should be treated as a dichotomous variable and so labeled as an ability which is either present or not.

In recent years, imitation has become a popular subject in a wide range of specialties ranging from robotics to primatology to comparative and developmental psychology (Dautenhahn & Nehaniv, 2002; Whiten, 2006; Want & Harris, 2002). These diverse fields conducting imitation research conclude that there are a variety of imitation skills requiring different cognitive mechanisms which must be highlighted when analysing imitative ability (Uzgorsis, 1999). Want and Harris (2002) consider that the taxonomy of imitative behaviours used by comparative psychologists should be used within the developmental research of ASD. Variations in the mechanisms used to copy actions may be of particular relevance for ASD (Whiten, 2006), as due to varying group memberships and various imitative tasks currently being examined, combining and effectively comparing results across studies is impractical (Subiaul, 2010).

Whilst a single term of 'imitation' may be used in lay terms to describe any matching behaviour, comparative psychology facilitates four categories of differing matching behaviours – mimicry, gestural/body level imitation, emulation and object movement re-enactment (OMR) (Whiten, 2006). The first, mimicry, refers to the release of a previously learnt behaviour as the result of a common stimulus. An example would be emotion contagion, whereby perception of an emotional behaviour stimulates that same emotional behaviour to be displayed, yet no social learning takes place

(Halfield, Caccioppo & Rapson, 1994). Secondly, gestural or body-level imitation simply describes the intentional replication of an observed action that could be novel to the observer and could involve (though not necessarily) new social learning. Essentially, this form of imitation involves performing an action by seeing how it is done (Whiten & Ham, 1992). Meanwhile the third category, emulation, is used to catalogue the completion of a task by achieving the same end state as the modeled task, but not necessarily through the same particular behaviours. Tomasello (1998) proposes that during emulation, rather than studying the behaviour, the imitators learn about the environment. Therefore instead of copying the action (imitation), the effects of the action are copied (emulation). Such behaviours were noted by Wood (1989) when children not only used imitative actions to impersonate others, but also attempted to achieve the same objective, often by the impersonator using behaviours similar, yet differing from those of the model. Finally, the fourth category commonly used by comparative psychologists, known as OMR, is used to describe a finer distinction of emulation when the movements of the objects within the task are analyzed more than the actions of the model (Whiten, 2006). A clear example of this was shown by capuchin monkeys when shown how to remove bolts from a device. Despite watching a model *poking* the bolts out of the device, some of the monkeys used an alternative removal technique by *pulling* the bolts out (Custance, Whiten & Fredman, 1999). Rogers and Williams (2006) note an important methodological point regarding studying OMR is that in order to identify such a categorization of imitation, sensitive scoring is required to record the imitation with precision, for instance: the directionality of the movement, the action timing and the orientation of the space used.

Remarkably, despite multiple propositions as to the processes behind the development and working of imitation, for the most part imitation is still approached with an all-or-nothing technique whereby imitation is regarded as a dichotomous variable (Jones, 2009). The majority of psychological studies still use a rather basic method of scoring the imitation via the subjective grading of performance (e.g. 0, 1, 2 or 3) on a scoring system based on a number of variations of the act to be imitated. Furthermore, these scales are then discussed in terms of being able to imitate or not rather than the quality of the imitation (McGuigan, Makinson & Whiten, 2010). Therefore limitations are encountered when comparing groups of children showing

fine differences in imitative ability. In order to capture any problems and to investigate whether specific types of imitation are more strongly affected within ASD, the definition of the imitation the purpose for which it is being measured are vital (Rogers & Williams, 2006; Smith et al., 2006). Therefore, each of the four categories described above can be further defined by dichotomies such as whether the imitation involved an object; whether it was meaningful (i.e. was symbolic of familiar); whether it were immediate or deferred; spontaneous or elicited and finally whether it was structured or 'freestyle' (Williams et al., 2004). Whilst some scoring systems have become more refined, and more attention has been paid to the quality and description of the imitation, these scoring techniques are still subjective, therefore affecting conclusions drawn from the data, especially when considering small changes in ability (Smith, Lowe-Pearce & Nichols, 2006). Thus, regardless of how well defined the act to be copied is, the original and imitated actions must be compared objectively in order to successfully summarize results across and within studies.

This study aims to investigate whether ASD adolescents have difficulty in imitating. Furthermore, this study investigates whether if there is such a deficit, it is specific to bodily imitation. In order to assess whether there is a more specific imitation deficit, a 'ghost control' is used whereby movement end point re-enactment is assessed, as described by Heyes, Jaldow, Nokes and Dawson (1994). Within this methodology, one set of stimuli shows a model conducting an action with an object and another stimuli set shows the object conducting the same actions without the aid of the model. This methodology has been more typically used within animal research, but it has recently become more popular in developmental psychology after the discovery that young children's reenactment of a covertly motorized object is the same as when the equivalent actions were performed by an actor (Huang, Heyes & Charman, 2002).

To precisely define the actions to be imitated: immediately after viewing the modeled action, the participants were be prompted to copy a structured symbolic action by using a pen to draw on a tablet PC's screen. A tablet PC is used to take advantage of a clinical-kinematics assessment tool - CKAT, which allows complex movements to be captured by measuring precise hand movements on a unique system integrating both a kinematics recording device and the visual display (Culmer, Levesley, Mon-Williams & Williams, 2009). This produces a detailed objective scoring system for

both the original and copied actions allowing accurate comparisons to be made regarding the quality of the participants' imitation. The system is 'low-tech' and easily portable so that it can be used in any setting, making it very appropriate for work with children with ASD, who may be better suited to testing in familiar settings such as their schools. Furthermore, by simply using a digital equivalent of 'pen-and-paper', CKAT is able to detect subtle movement differences including variation of the kinematics' temporal and spatial qualities, as well as frequency and smoothness (see Culmer et al., 2009 for more details regarding these measures).

It is hypothesised that this study will find imitative ability to be worse for ASD adolescents compared to their TD peers. Secondly, it is hypothesised that this ASD deficit will be worse for bodily imitation. Furthermore, it is expected that results will show positive relationships between imitation ability and age, intelligence (more specifically verbal intelligence) and general motor abilities, as well as a negative relationship between imitation ability and ASD severity. As a series of actions are being examined (see methodology for full details), instead of considering an absolute error between the imitator and the model, the accuracy of how the imitator alters their movements according to how the model's movements change will be examined. In order to produce an objective measure of this imitation fidelity, the strength of how much the imitator and model's actions correlate with each other will be calculated.

2. Method

Participants

Ethical approval was received from the North of Scotland Research Ethics Committee. A total of 17 ASD children (all males) were recruited from clinics at the Royal Aberdeen Children's Hospital and the MICAS Unit at Dyce Academy, Aberdeen, and a total of 26 neurotypical developing (TD) children (all males) were recruited on an opportunity sample basis within the Aberdeenshire area. All children were aged 11-17 (ASD: $M = 14.19$, $SD = 2.17$; TD: $M = 14.00$, $SD = 2.12$), the break down of participants ages within each group can be found in Appendix 8.4.1. One participant from each group withdrew from the study.

Materials

All computer tasks were created prior to the experiment using Clinical-Kinematics Assessment Tool (CKAT v6.3) (Culmer et al., 2009) and presented on a touch-screen laptop (laptop A: 1.8 GHz Intel(R) Core™2 Duo CPU T5670 Toshiba tablet with a 12-inch screen set at a resolution of 1280 by 800, WXGA). The participant responded to stimuli presented on the tablet's screen by moving a stylus across the tablet surface. The signal generated by the stylus movement was then processed to generate highly accurate information regarding the participant's movement e.g. acceleration and smoothness of movement. Therefore the participant was able to draw on the screen just as they would with pen and paper, but also providing an objective record of the movement. External video stimuli were presented through Microsoft PowerPoint on a 2.6GHz Intel Core 2 Duo Mac OS X 10.6.7 laptop with a 15-inch screen set at a resolution of 1440 by 900 (WXGA+) - laptop B.

Design

Imitation

The aim of this task was to derive an objective measure of imitation fidelity by making a direct comparison between the kinematic recordings of an actor executing actions on CKAT and those of an imitator attempting to perform the same actions by observing the actor. In addition, we sought to obtain a measure of object movement re-enactment (OMR) ability by displaying the path taken by the movement end-point rather than the actor. Therefore, there were two sets of stimuli, each consisting of a series of video-clips played to the participant through Microsoft PowerPoint on laptop B, with written instructions asking them to pay attention to the shape, size and speed of the stimuli (see Appendix 8.1.1 for full wording of instructions). Emphasis was drawn to these aspects of the stimuli as the participant was also verbally asked to carefully watch and copy the shape, size and speed of the stimuli. After watching a single clip, the participant was prompted to reproduce the movements they had watched by drawing with the stylus on laptop A's screen. The first set showed an actor drawing shapes on laptop A whilst the second set showed laptop A and a still actor, with a dot tracing out the same movement end-points. Within each set, the following trials were completed: 3 shapes (circle, triangle and square); 3 sizes of

shape (small, medium and big); and 3 speeds of drawing (slow, medium and fast) – making a total of 27 trials within each stimuli set, and a grand total of 54 trials.

Construction of stimuli

For the imitation stimuli, an actor was video-recorded as he drew the stimuli free-hand on the laptop. Kinematics of all these actions were recorded using CKAT. The kinematics for each segment of the stimuli were plotted to ensure that through natural variation there was a normal distribution of speeds and path lengths (e.g. checking that for ‘medium speed’ the actor had drawn faster than when drawing a ‘slow speed’, yet slower than when drawing a ‘fast speed’). For the movement end-point re-enactment (OMR) stimuli set, the kinematics of the actor’s actions were replayed in a MATLAB run script producing video-clips of a moving dot (diameter 1cm) following the same path as the actor with exactly the same patterns of speed change and error.

The MATLAB script allowed the movement end-point re-enactment video clips to be produced immediately after the original movements were recorded within CKAT, therefore allowing both sets of stimuli to be filmed in the same sitting, with only the camera angle being changed between stimuli sets. In the action stimuli the camera angle was deliberately set so that participants could not see the content of the laptop screen; however in the movement end point re-enactment stimuli, the camera angle was raised in order to provide a clearer view of the laptop’s screen and therefore of the moving dot (see Figure 1). When filming, the actor performed with a neutral expression on his face and continuously kept his gaze on the tablet’s screen.

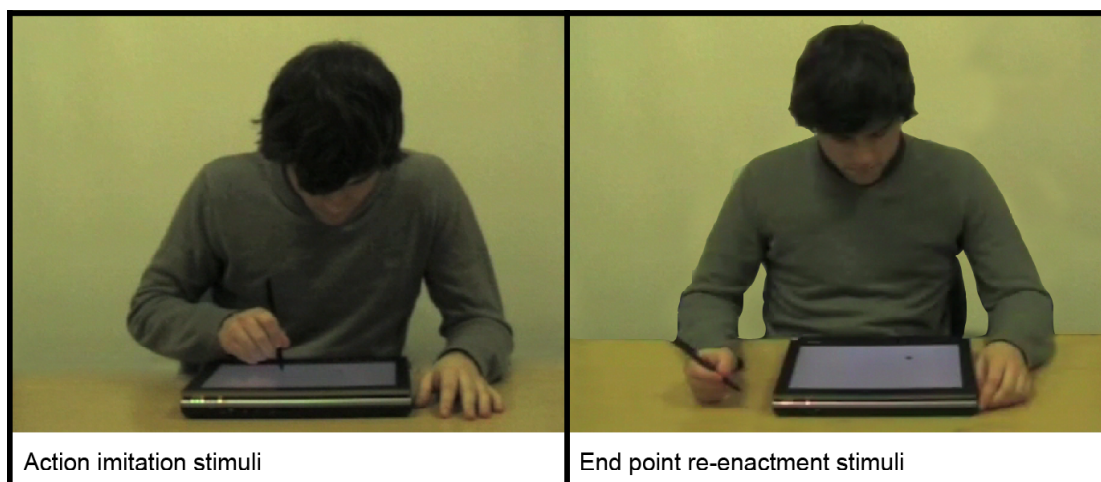


Figure 1: illustrating the different camera angles for each stimuli set

Two pseudorandomised versions of each stimuli set (DotA, DotB and ActorA, ActorB) were created to vary the presentation of the stimuli content – the shapes, sizes and speeds. These four different presentation possibilities were also used to counterbalance the presentation order of the stimuli sets: DotA/ActorA, DotA/ActorB, DotB/ActorA, and DotB/ActorB.

Whilst the participant drew their response on laptop A's screen, CKAT continuously measured path length, task duration and smoothness of movement. This study only presents data from two of the parameters – path length, providing information on the form and size of the object drawn, and task duration, describing the time taken to complete the action. These two measures were then compared to those of the actor's original movements in order to assess the fidelity of the participant's imitation across the series of trials.

Control motor tasks

A further 3 tasks were used to assess other non-imitative aspects of motor control (see Appendix 8.1.2 for full wording of instructions for these tasks).

A) Dynamic dot tracking task (Figure 2A)

Part *i* (Figure 2A*i*): Participants were required to keep the stylus on a single 1cm diameter dot which moved in a figure-of-8 spatial path once the stylus had been placed upon it - varying as $\sin(\text{time})$ (i.e. the dot sped up, reached peak speed halfway from left to right, slowed to a stop; reversed and repeated it's movement - creating a figure-of-8 lying on its side). Over 9 trials the frequency at which the dot moved increased parametrically: the first 3 trials were 'slow' with a x-frequency of $\sim.79\text{Hz}$ and y-frequency of $\sim.39\text{Hz}$; the second 3 trials 'medium' with x-frequency $\sim 1.57\text{Hz}$ and y-frequency $\sim.79\text{Hz}$; and the final 3 trials 'fast' at $\sim 3.14\text{Hz}$ x-frequency and ~ 1.56 y-frequency. Within this task, the speed of the participant's stylus following the dot was measured at each speed, allowing an average speed to be taken.

Part *ii* (Figure 2A*ii*): The 9 trials from part *i* were repeated but with a guide illustrating the path that the dot would follow, as described to the participant by further illustrated instructions.

B) Pentagram: a goal-directed action task (Figure 2B)

This simple task displayed a green dot (diameter 0.7cm) – the goal, to which the participant was asked to draw to as fast and as accurately as possible with the stylus. Once they reached this goal, another goal was displayed in a different location. In total there were five different goal locations – the five points of a pentagram. Each location was displayed 16 times, thereby creating a total of 16 pentagram trials. Measures included peak speed, distance time, movement time and path length.

C) Tracing task (Figure 2C)

Within this task, participants were asked to trace between the ‘tramlines’ (0.25 cm wide) outlining two random shapes. Each shape was displayed three times, totaling 6 trials for this task. The main measures of this task were accuracy and speed of the movement in order to evaluate the participant’s trade-off between accuracy and speed. In order to illustrate time constraint, a box outlined the first section of the shape’s tramlines. After five seconds, the box moved onto the next section of the tramlines and continued in this method until the end of the shape was reached (see Figure 2Ci and Figure 2Cii).

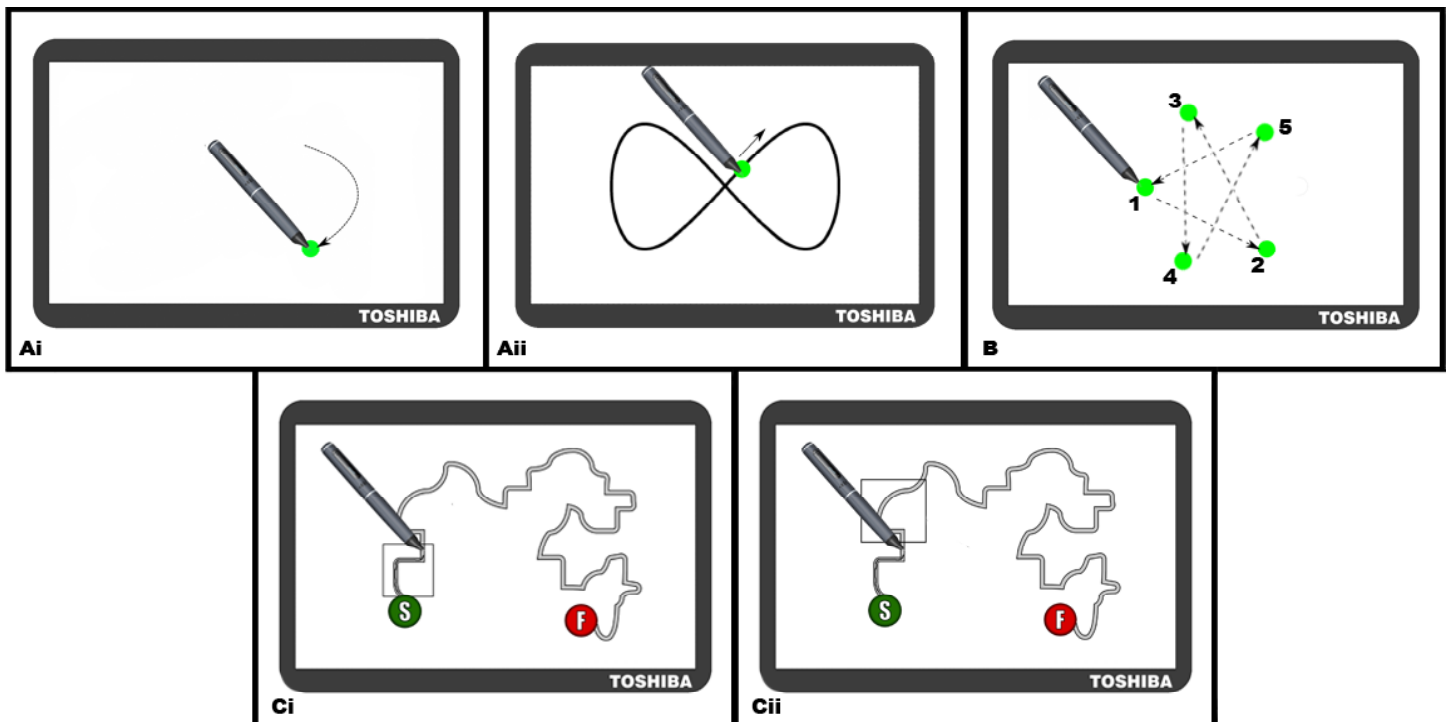


Figure 2: illustrating the motor control tasks

Questionnaires

A) Social Responsiveness Scale (SRS, 2005)

This questionnaire was employed to provide a measure of autism severity as studies have shown it to correlate well with severity measures on the ADI-R (e.g. Constantino et al., 2003). In order to assess the degree to which the participant expressed the autism phenotype, which the scale assumes to be normally distributed within the normal population, this 65-item rating scale focuses on the child's reciprocal, cognitive, expressive and motivational aspects of social behaviour along with their autistic preoccupations. Questions came under five headings: Social Awareness, Social Motivation, Social Cognition, Social Communication and Autistic Mannerisms. A T-score of ≥ 76 was labeled as 'severe ASD'; a T-score of 60-75 was labeled as 'mild/moderate ASD'; and a T-score of < 59 was labeled as 'normal'.

B) The Developmental Coordination Disorder Questionnaire 2001 (DCDQ, 2007)

The aim of this parent report measure was to help identify children with Developmental Coordination Disorder (DCD). A total of 15 rated items asked parents to compare their child's motor abilities to their peers on three subscales – Control during Movement, Fine Motor and Handwriting, and General Coordination. Participants with a total score of ≤ 57 were labeled as suspected DCD (Wilson et al., 2009).

Procedure

Prior to testing, parents were asked to complete the two questionnaires (the SRS and DCDQ) on behalf of their child. During the testing session, laptop A was placed in a landscape position approximately 4cm in front of the participant. The control motor tasks (dynamic dot tracking, pentagram and tracing) were conducted in approximately 10 minutes, with each task commencing with simple on-screen instructions and illustrations (see Appendix 8.1.1 for full wording of instructions).

The control motor tasks were followed by the imitation task, where laptop B was placed behind laptop A, putting laptop B's screen approximately 52cm away from

the participant. The visual stimuli for the imitation task were then presented on Microsoft PowerPoint on laptop B. When prompted by the PowerPoint presentation slide, participants imitated the action they had just watched by drawing on laptop A's screen with a stylus (see Figure 3 for PowerPoint ordering; see Appendix 8.1.2 for full wording of instructions).

When the participant completed the imitated movement, the PowerPoint was manually furthered to the following slide and trial video. The total imitation task lasted approximately 10 minutes.

After the computer tasks were completed, the four subtests (Vocabulary, Block Design, Similarities and Matrix Reasoning) of the Wechsler Abbreviated Scale of Intelligence (WASI-IV, Psychological Corporation, 1999) were administered within the same sitting, totaling testing time at roughly 1 hour. Testing was usually conducted in a quiet classroom, but in some instances the participants were tested in a quiet room at home.

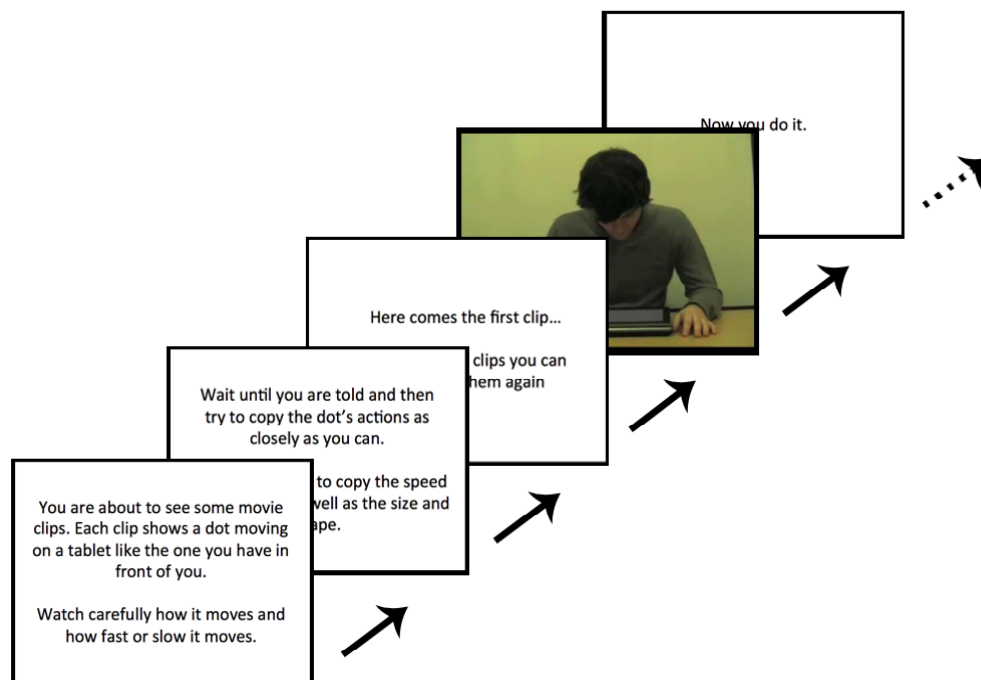


Figure 3: illustrating the ordering of the imitation task PowerPoint's

3. Results

In order to measure the degree of statistical dependence between the imitator's action and that of the model, the correlation between each imitator and the model was calculated. This calculation was completed for two parameters of the imitation – path length, describing the form and shape of the movement; and trial duration, the time taken to complete the action. The correlation slopes were firstly considered by representational change via the strength of the correlation to investigate the general effects of the imitation tasks, and secondly by bias to explore more specific effects of the stimuli conditions.

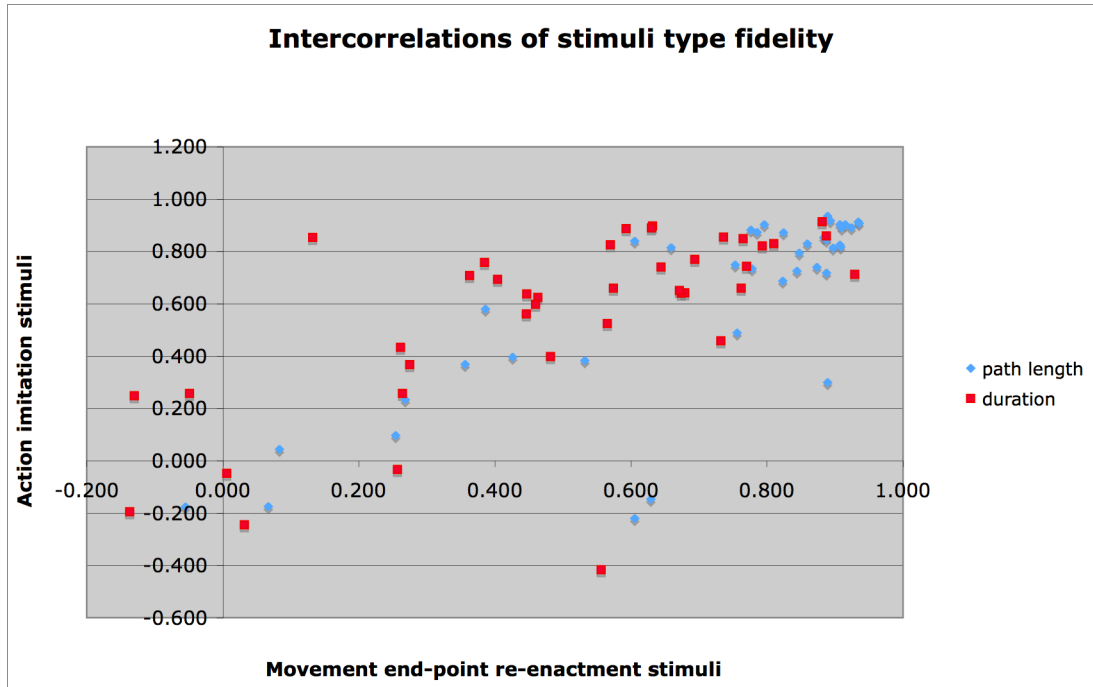
Examination of representational change - fidelity

Group differences

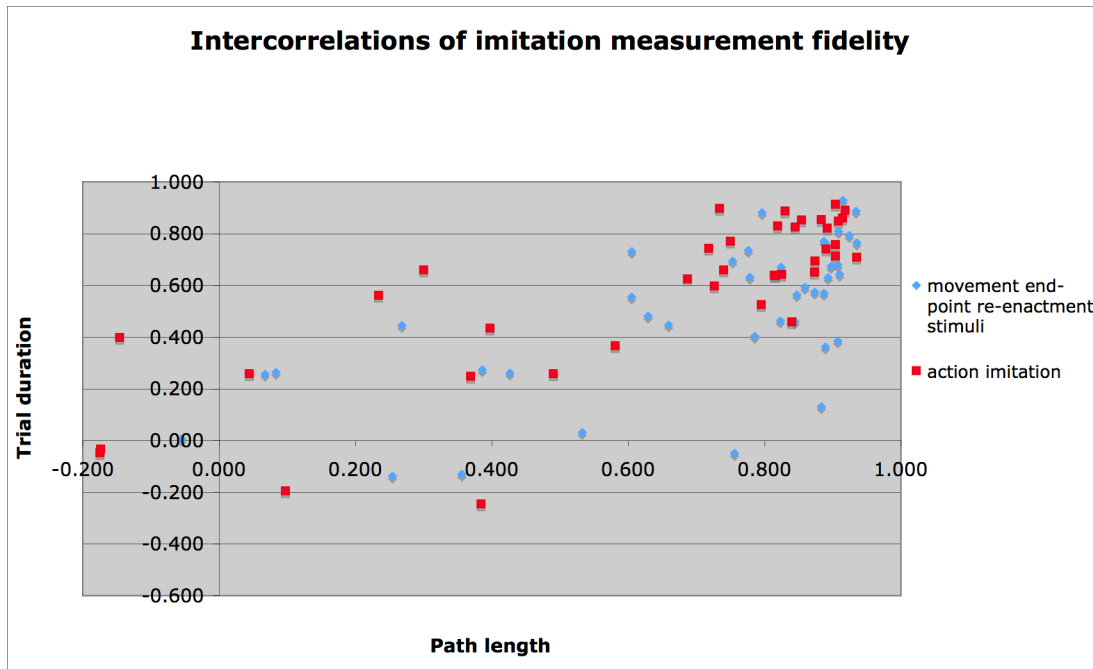
Imitation fidelity was calculated in order to provide the representational change between stimuli by examining the strength of correlation between the model and each imitator for path length and duration of imitation for both sets of stimuli (action imitation and movement end-point re-enactment). These four correlation strength measures were found to be highly intercorrelated (see graphs 1 & 2), therefore justifying the measures to be collapsed across the stimuli type (action imitation and movement end-point re-enactment) and imitation measures (path length and duration) to provide a mean measure of imitation fidelity. According to the Kolmogorov-Smirnov test this mean measure followed normal distributions and so parametric measures were facilitated for analysis. An independent t-test showed that TD imitators had significantly better imitation fidelity ($M = .70$, $SD = .19$) than ASD imitators ($M = .44$, $SD = .32$), $t(38) = 3.26$, $p = .002$, $d = 1.03$.

Mean fidelity values (x) were found to follow a dichotomous distribution with values of either less than or equal to .402 or more than or equal to .640; therefore each participant was labeled as either a 'poor' ($x \leq .402$) or a 'good' ($x \geq .640$) imitator. A 2-tailed 2X2 Fisher's exact test demonstrated the difference between labeling to be statistically significant $p = .005$ (Joose, 2011). However, the labeling did not totally separate by group, with ASD participants making up 75% of 'poor' imitators and 25% of 'good' imitators; however the 'poor' ASD imitators mean age was 13.44 ($n = 9$, $SD = .16$) compared to the mean age of the 'good' ASD imitators being 15.14 ($n = 7$, $SD = .21$) (see table 1). Furthermore, the three TD participants classified as 'poor'

imitators were amongst the youngest of the control group: aged 11, 11 and 12 (see Appendix 8.4.1). Additionally, the SRS score for ‘poor’ imitators ($n = 12$, $M = 91$, $SD = 56.85$) was significantly higher than that of the ‘good’ imitators ($n = 28$, $M = 34.11$, $SD = 39.46$), $t(38) = 3.65$, $p = .001$, $d = 1.18$ (see graph 3).



Graph 1: showing the intercorrelation of both sets of stimuli (action imitation and movement end-point re-enactment)



Graph 2: showing the intercorrelation of both sets of imitation measurement (path length and time duration)

Imitation quality	Group	N	Mean imitation fidelity	Age	SRS scores		
Poor	ASD	9	-0.068	14	115		
			0.005	15	127		
			0.131	16	69		
			0.162	11	111		
			0.176	11	165		
			0.211	12	173		
			0.341	13	111		
			0.364	14	101		
			0.380	15	92		
	TD	3	0.029	11	16		
			0.378	11	9		
			0.402	12	3		
	Good	ASD	7	0.650	11	162	
				0.712	17	65	
				0.724	17	84	
0.760				13	92		
0.763				15	71		
0.813				16	75		
0.874				17	108		
TD				21	0.640	16	8
					0.653	15	13
					0.657	13	16
		0.659	13		13		
		0.681	12		4		
		0.683	11		35		
		0.689	14		6		
		0.739	16		15		
		0.742	12		13		
		0.755	17		19		
		0.756	15		10		
		0.780	17		14		
		0.782	11		23		
0.792		15	4				
0.796		14	2				
0.833		15	7				
0.842	15	39					
0.858	13	7					
0.864	17	9					
0.865	16	31					

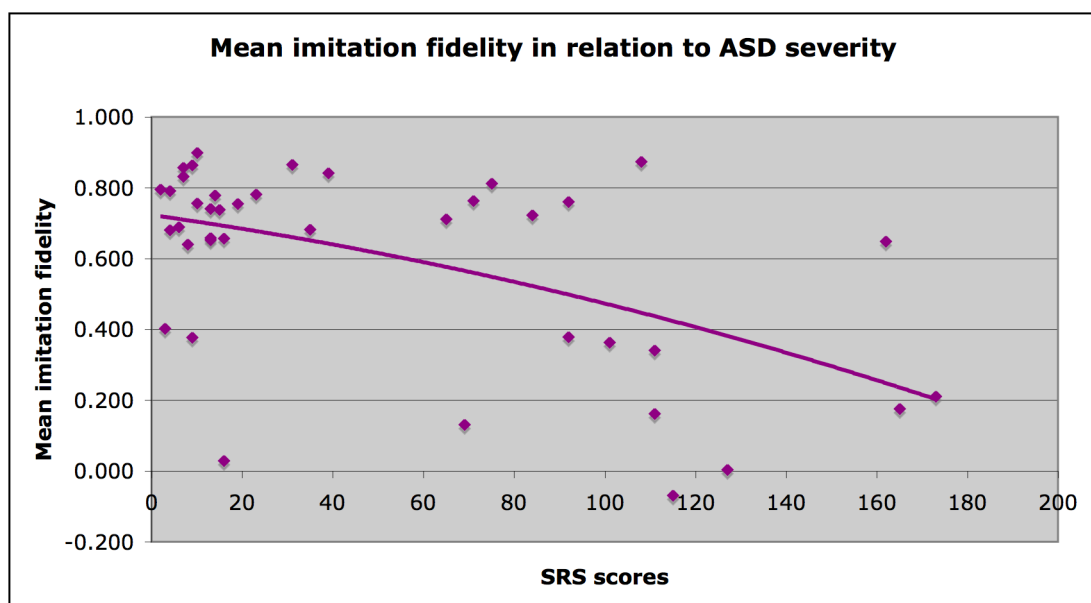
Table 1: breakdown of imitation quality labeling by group, along with individual's mean imitation fidelity, age and SRS scores. Ordered by mean imitation fidelity.

Individual differences

In order to explore the individual differences, the fidelity strength measurements for each stimuli set (action imitation and movement end-point re-enactment) and measurement type (path length and duration) were used. As discussed previously, these measures were found to be highly intercorrelated and so were collapsed into a mean measure of fidelity strength. According to the Kolmogorov-Smirnov test this mean measure followed normal distributions and so parametric measures were facilitated for analysis for both groups.

TD

Age was found to have a significantly strong positive correlation with imitation fidelity strength, Spearman's $r(24) = .55, p = .005$. A positive correlation was also found between imitation fidelity and verbal IQ (similarities subtest raw score). However, no significant correlation was found between SRS and imitation fidelity. When compared with the motor control measures no significant correlations were found between the mean speed for the tracking task (background and no-background) or accuracy from the tracing task; but imitation fidelity strength was found to be significantly negatively correlated with the distance time measure of the pentagram task, $r(24) = -.59, p = .002$.



Graph 3: showing a decline in mean imitation fidelity whilst ASD severity increases

ASD

Comparatively, within the ASD group no significant correlations were found between imitation fidelity strength and age, SRS scores, verbal IQ or any motor control measures.

Examination of bias*Comparison of stimuli*

In order to investigate if each group of ‘good’ and ‘poor’ imitators performed differently for each stimuli type (action imitation and movement end-point re-enactment), the bias of correlation was investigated between each imitator and the model for both path length and duration of imitation. A paired-samples t-test indicated that ‘poor’ imitators of both the ASD and TD groups showed no significant difference in their bias to copy the action imitation and movement end-point re-enactment stimuli sets. However, within the ‘good’ imitators, the ASD group showed a significantly higher imitative path length bias for the movement end-point re-enactment stimuli ($M = .83$, $SD = .13$) than that of the action imitation ($M = 1.03$, $SD = .19$), $t(6) = 2.47$, $p = .049$, $d = .093$. Comparatively, duration bias for action imitation stimuli ($M = .97$, $SD = .24$) was significantly higher than that of the movement end-point stimuli ($M = .66$, $SD = .18$) for the ASD group, $t(6) = 4.47$, $p = .004$, $d = 1.69$.

However, a different pattern was found for the ‘good’ TD imitators. Whilst the duration bias was also significantly higher for the action imitation stimuli ($M = .81$, $SD = .24$) than the movement end-point stimuli ($M = .64$, $SD = .29$), $t(20) = 2.27$, $p = .035$, $d = .50$; a significant difference was not found for path length bias (action imitation: $M = .96$, $SD = .21$; end-point re-enactment: $M = .88$, $SD = .13$), $t(20) = 1.71$, $p = .102$, $d = .37$.

Comparison of measures

Bias of correlation between each imitator and the model was also used to determine if the groups performed differently for the different elements of the imitation – path length and duration. An independent-samples t-test indicated that the action imitation path length bias was significantly higher for the TD group ($M = .79$, $SD = .33$) than the ASD group ($M = .53$, $SD = .53$), $t(22.81) = 2.28$, $p = .033$, $d = .79$. However the

TD group action imitation duration bias was not significantly higher ($M = .74$, $SD = .30$) than that of the ASD group ($M = .48$, $SD = .55$), $t(21.07) = 1.75$, $p = .095$, $d = .62$. Levene's test indicated unequal variance for both path length ($F = 7.07$, $p = .011$) and duration ($F = 9.76$, $p = .003$) measures for the movement end-point stimuli, degrees of freedom were adjusted accordingly.

The same pattern was found for the movement end-point stimuli: firstly the path length bias was found to be significantly higher for the TD group ($M = .81$, $SD = .24$) than the ASD group ($M = .61$, $SD = .31$), $t(38) = 2.32$, $p = .026$, $d = .73$. However, the duration bias for the TD group ($M = .59$; $SD = .32$) was not significantly higher than that of the ASD group ($M = .39$, $SD = .35$), $t(38) = 1.84$, $p = .074$, $d = .59$.

4. Discussion

The data clearly shows a large group difference in imitation fidelity, with the TD group performing more accurately. Further analysis of labeling the participants as 'good' and 'poor' imitators significantly split the imitators by ability. This also split them largely by group with most of the poor imitators having ASD and most of the good imitators being typical participants. However, the split was not complete and the youngest typical participants fell into the 'poor' imitators group, whilst 7 of the ASD participants were 'good' imitators. These were the older participants. The dichotomous distribution of performance on this task suggests dependence upon a single cognitive capacity. With the three of the youngest of the control group being labeled as 'poor' imitators, and the oldest of the ASD group being 'good' imitators, the data suggests that such a cognitive capacity comes into play at some point in early adolescence, but that its emergence is delayed in people with ASD. This provides support to previous findings that an imitation deficit remains in adolescents with ASD (e.g. Hobson & Lee, 1999; Rogers et al., 1996; Stone et al., 1997). However a similar gulf in imitation fidelity to that of the TD group was found within the ASD group. Therefore a developmental delay in imitation ability within ASD is suggested, furthering the proposal of Hepburn and Stone (2006) that a similar pattern of imitation performance is found in both ASD and TD adolescent populations. However the ASD performance is at levels lower than their TD peers. Therefore, this proposal of a delay in imitative abilities echoes the proposal of a delay in the development of ToM

in ASD rather than a total deficit as described by Baron-Cohen (1989). Furthermore, differing ranges of SRS scores within the ASD ‘poor’ and ‘good’ imitating groups provides support for the assertion of Charman and colleagues (1998) that as ASD severity increases, the acuteness of imitation decreases.

With the data still split by the labels ‘good’ and ‘poor’ imitator, ‘poor’ imitators from both the ASD and TD groups were no better at movement end-point re-enactment (OMR) compared to action imitation. However, within the ‘good’ TD imitators, for the duration measure of imitation, action imitation was better than OMR performance. Similarly, the ‘good’ ASD imitators were also significantly better at action imitation compared to OMR for the duration measure.

However the reverse was the case for the path length measure. Whilst ‘poor’ ASD and TD imitators were no better at OMR compared to action imitation, those ASD adolescents classified as ‘good’ imitators were better at OMR than action imitation. ‘Good’ TD imitators showed no difference for action imitation compared to OMR in imitating path length. Therefore, even in older subjects who were capable imitators, group-specific differences emerged according to relative performance on ‘true’ imitation tasks compared to those using a ‘ghost’ control, but which depended upon the movement parameter being copied. All capable imitators performed better on the ‘true’ imitation task compared to the ‘ghost control’ for the duration measure, suggesting that action-perception may somehow enhance time or speed estimation. Meanwhile, for the path-length measure, only the capable imitators with ASD showed a difference between OMR and action imitation, suggesting that for them, action-perception made the task more difficult rather than easier. One explanation could lie in the differential attentional demands of the OMR and action imitation tasks.

Following these results and the proposal of an adverbial imitation deficit in ASD by Perra and colleagues (2008), a comparison of the bias strength of each imitation measure (path length and duration) was conducted in order to assess whether the participant groups conducted the tasks in different ways. Interestingly, whilst there was no significant difference between groups for the duration imitation measure from either stimuli set; the TD group were significantly less biased in imitating the path length from both the action imitation and movement end-point imitation stimuli sets.

Compiling these results suggests that there is a specific deficit in ASD in following the sequence of multiple elements of action imitation, as successfully monitoring the sequence of movements of the actor would have led to a smaller relative magnitude of difference due to successful copying of the form and size of the shape – an ability which the TD adolescents effectively facilitated. Tantam (1991) and Hepburn and Stone (2006) propose that an inability to imitate complex sequential actions within ASD may be due to less practiced motor skills caused by a social interaction deficit made more apparent by age. Alternatively, it could be due to problems with attentional flexibility and/or perceived biological motion often found in ASD (Ozonoff, Strayer, McMahon & Filloux, 1994; Rogers & Williams, 2006; Freitag et al., 2008). However, the ‘good’ ASD imitators were able to successfully pick up information regarding the duration of the movement from the action stimuli, more so than the object-only stimuli. The same result was found within the ‘good’ TD group, and TD adolescents were no better than the ASD adolescents in imitating duration. With the screen being visible within the movement end-point re-enactment set, it could be that it was easily possible to follow the form and size of the shape being drawn, thus allowing the ASD adolescents to succeed in copying path length from this stimuli set, whilst struggling to derive such information from the action-only perception. Yet, the TD group were still more effective in imitating the path length from the OMR task and even more so for the imitation task, possibly because they were able to consider and effectively process the sequence of multiple elements from the action imitation stimuli to provide more information than the movement end-point stimuli could provide. Further studies could investigate this marginal stimulus effect by monitoring the gaze of the participant whilst watching the imitation stimuli as further to ASD adolescents having difficulties with making multiple shifts in visual attention (Ozonoff et al, 1994), they may also have unusual attention foci during imitation tasks. Results of eye tracking during imitation by Klin, Jones, Schltz, Volkmar and Cohen (2002) suggest that even with attentive ASD participants, visual attention was not always focused on the critical stimulus and so relevant aspects of the movement required for accurate imitation were not perceived or processed.

Alternatively, these results could reflect ASD adolescents struggling to organize motor actions as a chain of motor acts. As previously discussed, it has been proposed

that ASD individuals fail to relate their observations of others' behaviours to their own neural codings linked to memories of similar behaviours due to their MN system (Williams et al., 2004). Fabbri-Destro, Cattaneo, Boria and Rizzolatti (2009) further this theory by proposing that this malfunction within the mirror system could in fact be due to a lack of ability in organizing the chained actions on which the mirror understanding of others' intention is based. Both TD adults and children have been found to plan their visually determined action globally rather than as a collection of independent steps. However some studies investigating this theory suggest that ASD children program single independent motor acts as they are incapable of translating their motor intention into a global chain of actions. Therefore it follows that if ASD children are unable to successfully structure an organization of global movements to reach a goal as illustrated by a model, they will be impaired in understanding the actions of the model (Fabbri-Destro et al., 2009). An example study describes how when asked to tell the experimenter what action the model was completing and why, ASD children were able to recognize the motor act, but failed more frequently than their TD peers in understanding the model's intention behind the action (Boria et al., 2008). The movement end-point stimuli were shown as a single dot moving continuously on the screen. Therefore these stimuli were showing simple actions which could be easily translated into simple movements in order to successfully copy them. However, the action imitation stimuli were much more complex, showing a model drawing on a screen which cannot be clearly seen. Hence, following the theory of Fabbri-Destro et al. (2009), the imitator must translate the overall stimuli into an organized chain of motor acts in order to understand what is being drawn – a circle, square or a triangle i.e. easily identified shapes. Therefore, it could be that whilst TD adolescents were able to interpret the chain of motor acts performed by the model into which shape was being drawn, the ASD adolescents were unable to do so, thus leading to their poorer performance in path length.

Individual differences were examined to see if they influenced the strength of imitation fidelity. Results quite clearly showed that imitation fidelity increased with age in TD adolescents, but not with ASD adolescents. Furthermore, TD adolescents also increased their imitation fidelity in relation to their verbal IQ whilst ASD adolescents did not, therefore replicating previous findings (e.g. Sigman & Ungerer, 1984; Royeurs, Van Oost & Bothuyne, 1998). Finally, imitation ability correlated

with motor control skills (more specifically with the pentagram task) only in TD adolescents. Further individual analysis could be carried out on this data set regarding the direction of the imitators' movement, as not only does the individual have to possess motor capabilities in order to carry out an action plan produced by watching the model perform, the individual must first have representational skills to translate the observed behaviour into an action plan – a skill often found to be lacking in ASD (Hepburn & Stone, 2006).

5. Conclusion

Using objective measurement of imitation fidelity, this paper investigated whether an imitation deficit is present in ASD adolescents, and furthermore whether this deficit was specific to action imitation. Through exploratory analysis, results show that there is a clear group difference in imitation fidelity in children aged 11-17, with imitation performance declining as the severity of ASD increases and an increase of performance correlating with age only in TD children. Further analysis suggests that there is a jump in imitation ability in both TD and ASD children. However this leap is delayed in ASD. Therefore following the proposal of Whiten and Brown (2006) that instead of a fixed imitative deficit, there is a developmental delay of imitative ability within ASD.

Furthermore, ASD children labeled as 'good' imitators showed a specific deficit in copying those action imitation stimuli consisting of more complex actions to be imitated. However, this deficit was only found for the measure of path length as the duration of the trial was successfully imitated from the complex stimuli by the ASD group at a similar level to that of the 'good' TD imitators. Therefore it was suggested that this specific deficit may be due to problems in perceiving biological movement and focusing upon other elements of the stimuli other than the critical movement information, as the trial duration information was successfully perceived and processed. However the sequence of movements required in order to imitate the form and size of the drawn shape were not.

These results suggest that an imitation developmental delay is present in ASD. However, further research would be required with other clinical groups to investigate how specific this developmental delay is to ASD.

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