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Chapter 9

Behavioural, Biopsychosocial, and Cognitive Models of Autism Spectrum Disorders.

By

Sophie E. Lind[1] and David. M. Williams[2]

[1]Department of Psychology, City University London, Northampton Square, London, EC1V 0HB (sophie.lind.2@city.ac.uk)

[2]Department of Language and Communication Science, Psychology, City University London, Northampton Square, London, EC1V 0HB (david.williams.2@city.ac.uk)

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Introduction

Autism spectrum disorder (ASD) comprises a range of developmental disorders including autistic disorder, Asperger's syndrome, and pervasive developmental disorder not otherwise specified/atypical autism, each of which is characterised by a triad of impairments in social interaction, communication, and restricted, repetitive behaviours and interests (RRBIs) (American Psychiatric Association, 2000; World Health Organization, 1993). ASD was originally identified and described in 1943 by Kanner, who believed the disorder to be biologically based. However, in the following decades, psychosocial explanations for ASD began to gain influence. Most notably, Bettelheim (1967) attributed the development of ASD as a response to emotionally "cold" parenting. Although this theory was influential for a significant period of time, it has not received empirical support, and it is now widely agreed that ASD is a biologically based disorder. Twin studies have consistently indicated that ASD is a highly heritable disorder (e.g., Bailey et al., 1995; Folstein & Rutter, 1977; Steffenburg, Hellgren, Gillberg, Jakobsen, & Bohman, 1989). Furthermore, although molecular genetic studies have not yet established a set of necessary and sufficient genes that cause the disorder, they have begun to identify a set of genes that are reliably associated with ASD (International Molecular Genetic Study of Autism Consortium, 1998, 2001, 2005).

Thus, ASD is a behavioually defined disorder with a biological basis. The behavioural definition and the (presumably forthcoming) identification of a set of genetic or neurobiological markers for ASD represent *descriptive* models of the disorder. Morton and Frith (1995) have argued that in order to develop a *causal* model of ASD, a third level of explanation – the cognitive or psychological level – is necessary to bridge the gap between the biological and behavioural levels. In other words, in order to understand the mechanisms by which genes and

neurobiology influence behaviour, it is necessary to understand the cognitive processes that mediate that behaviour – there is arguably no direct link between biology and behaviour in ASD. For this reason, cognitive accounts are essential to our understanding of the disorder.

This chapter reviews three of the most influential accounts (as well as some of their variants/alternatives) and their respective relationships to brain function and behaviour, in the spirit of the causal modeling approach. We will begin by describing the theory of mind hypothesis of ASD – a domain-specific, "social" account – and go on to discuss the executive dysfunction and weak central coherence hypotheses – domain-general, broadly "non-social" accounts. Evidence for and against each of the theories will be considered, as well as issues of universality, specificity, and relationships to ASD diagnostic features. The chapter concludes with a section considering future directions for cognitive explanations of ASD.

The Theory of Mind Hypothesis

According to the original definition, a "theory of mind" (ToM) should be credited to any individual who attributes mental states (such as beliefs, desires, and intentions) to self and others in order to explain and predict behaviour (Premack & Woodruff, 1978). In everyday life, attributing mental states to people is a swift, frequently non-conscious activity. For this reason the term "mentalizing", which places less emphasis on the conscious, deliberate acquisition of any *theory* about the mind, is preferred to the term ToM by some researchers (e.g., Frith, 2003). From a cognitive science perspective, the computational underpinnings of ToM/mentalizing are complex. According to Leslie (1987), in the first year and a half of life, typically developing infants possess only primary representations of the world. During these early months, reality (objects, people, events, etc.) is represented purely veridically. At approximately 18 months of age, however, infants develop the capacity to form second-order representations that are

"decoupled" from reality. According to Leslie, these representations are no longer of/about the world (as primary representations are), but of/about one's (primary) representation of the world – they are representations *of* representations, or "M-representations" (Leslie & Roth, 1993). Because the infant can now represent representations (rather than just reality), they can begin to think about other people's (mental) representations and manipulate their own representations, as reflected in the act of pretence, which typically emerges at around 18 months of age. According to Leslie, M-representations are innately specified by a "theory of mind mechanism", which is damaged in ASD. With a diminished ability to form M-representations, children with ASD are limited largely to representing their own and others' behaviour, but not the mental states that underlie behaviour.

Intuitively, the ToM theory makes sense of the specific profile of everyday behavior characteristically diminished amongst people with ASD, who rarely tell jokes, understand or use sarcasm, tell lies or deceive others, or communicate important information to other people. In one way or another, each of these behaviors appears to require the recognition that it is possible to hold a mental perspective on reality, which is separate from reality itself. Likewise, two cardinal features of ASD are potentially explained in the same way; a limited propensity to initiate, or respond to, joint attention can be explained by a failure to recognize that other people hold a mental perspective that is different from one's own (Leslie & Happé, 1989). Also, the fact that children with ASD tend not to understand pretend play in others, or engage in pretend play themselves, can be explained by a limited ability to form M-representations. For example, to understand that a banana can be used as a telephone (or anything else) during pretend play arguably requires the ability to recognize the distinction between reality (that the banana is a banana) and one's own and others' pretend representations of reality (Leslie, 1987). From a

clinical perspective, then, the ToM theory of ASD parsimoniously explains many of the social and communicative difficulties that are core features of ASD.

Evidence for the ToM hypothesis

This version of the ToM hypothesis of ASD has the advantage of being very well specified and providing clear, testable predictions as to what kinds of activity and understanding children with ASD show limitations. Specific empirical evidence for the hypothesis came from a series of landmark studies, beginning in the mid-1980s, which found that children with ASD had significant difficulties with recognising that other people can hold false beliefs about the world. For example, Baron-Cohen, Leslie, and Frith (1985) gave children with and without ASD a "location change" false belief task (Wimmer & Perner, 1983) in which a scenario is presented where one character, Sally, places her marble in a basket, and then leaves the vicinity. While Sally is out of sight, another character, "naughty Anne" removes the marble from the basket and places it in a nearby box. Participants were then asked a ToM test question regarding which location Sally will look for her marble when she returns to collect it. A correct answer requires the participant to recognise that Sally will act on the basis of her belief (that the marble is in the basket), rather than reality (that the marble is in the box). Strikingly, Baron-Cohen et al. found that 80% of the children with ASD failed this question, despite the fact that, on average, they had a mental age of 5.5 years, which is 1.5 years higher than the age at which typically developing children reliably pass false belief tasks (Wellman, Cross, & Watson, 2001). The fact that participants with ASD succeeded on a number of carefully designed control tasks ensured that failure on the ToM test question was not merely the result of difficulties with memory or general logical reasoning. Children with ASD seemed to have a specific problem with recognising mental states, as the ToM theory predicted would be the case.

Baron-Cohen et al.'s (1985) study formed the cornerstone for a further two decades of research into ToM in ASD, which has consistently revealed deficits in the ability of affected individuals to explicitly understand a variety of mental states, including beliefs (Happé, 1995), intentions (Williams & Happé, 2009a), knowledge and ignorance (Lind & Bowler, 2009b), and deception (Sodian & Frith, 1992) in others, or in self (see Williams, Lind, & Happé, 2009). For example, in one experiment, Sodian and Frith asked children to keep a piece of candy safe by placing it in an opaque box. The children were then introduced to two puppets – a "nasty thieving wolf" and a "nice friendly seal". The participants' task was to prevent the wolf from getting the candy, since he would steal it. Instead, the children had to ensure that the seal found the candy so that they received a reward of two pieces of candy. In one condition, participants could achieve these goals by lying to the wolf and telling him that the box did not contain candy, but telling the seal the truth about the contents of the box. In this "deception" condition, for the child to achieve their goals they needed to *induce a false belief* in the wolf, which clearly requires a ToM. In another condition, participants could achieve their goals by physically locking the box whenever the wolf appeared, but simply leaving the box unlocked whenever the seal appeared. In this "sabotage" condition, participants only needed to understand a chain of physical causation; locking the box prevented a puppet from getting the candy, whereas leaving the box unlocked allowed the puppet to obtain the contents. Sodian and Frith found that whereas all participants, including those with ASD, performed very well in the sabotage condition, children with ASD were unique in performing poorly in the deception condition, highlighting the specificity of the mentalizing deficits in ASD.

The ToM theory has also received support from neurobiological studies, which have identified a "core ToM network" of brain regions amongst typically developing children. Areas

of the brain reliably recruited specifically for the purpose of solving ToM tasks include the medial prefrontal cortex and orbitofrontal region, anterior- and paracingulate cortices, superior temporal sulcus, and temporoparietal junction (e.g., Gallagher et al., 2000). Indeed, in a recent meta-analysis focusing on neurobiological studies of ToM in typical development, the medial prefrontal cortex was associated with ToM task performance in 37/40 studies (Carrington & Bailey, 2009). Supporting the ToM theory of ASD, several studies have now shown that this core ToM network – particularly the medial prefrontal cortex – is under-activated amongst individuals with ASD during ToM tasks (e.g., Castelli, Frith, Happe, & Frith, 2002; Kana, Keller, Cherkassky, Minshew, & Just, 2009; see also Zilbovicius et al., 2006). At a minimum, these studies confirm that ToM difficulties in ASD are *associated* consistently with specific neurobiological abnormalities. However, it is important to note that these findings do not necessarily indicate that the neural *basis* of ToM impairments in ASD has been uncovered. The brain regions that comprise the core ToM network have a protracted development amongst typically developing individuals, not reaching full maturity until late adolescence, or early adulthood (see Dumontheil, Burgess, & Blakemore, 2008). As such, it may be that these regions *become* specialized for mentalizing, rather than being "pre-wired" for such a function as Leslie's 1987 theory might predict.

Challenges to the ToM hypothesis

Despite the fact that the ToM theory of ASD has intuitive appeal and is supported by a significant body of empirical evidence from both cognitive and neurobiological studies, it has been challenged in several ways in recent years. The first challenge to the theory concerns the *universality* of the ToM impairment in ASD. Even from the earliest cognitive studies of ToM, it was clear that not all children with ASD failed traditional ToM tasks. In a meta-analysis of 13

studies of false belief understanding in ASD, Happé (1995) found participant success rates ranged from 16.6% to 60%. Across studies, 17 out of 70 (20%) children with ASD passed all false belief test questions. This compared with 20 out of 34 (58.8%) participants with moderate learning disability (MLD), who did not have ASD. Clearly, on a strong version of the ToM hypothesis, if an impairment in ToM is the underlying cognitive cause of the social-communicative features of ASD, then impairments on false belief tasks should be universal in ASD, assuming these tasks are valid measures of ToM.

Additionally, some have claimed that a further problem for the ToM hypothesis concerns the *diagnostic specificity* of the ToM impairment in ASD. Several studies have now shown that a proportion of children with other disorders manifest difficulties on traditional measures of ToM. Hence, children with congenital deafness who are born to hearing parents show diminished performance on false belief tasks (e.g., Peterson & Seigal, 1995), as do children with congenital blindness (e.g., Peterson, Peterson, & Webb, 2000), or generalized intellectual disability (Yirmiya, Erel, Shaked, & Solomonica-Levi, 1998). The fact that children with developmental disorders other than ASD show impaired ToM task performance, but generally not the social-communicative difficulties associated with ASD, presents an additional challenge to the ToM account of ASD.

Although the issues of universality and diagnostic specificity are apparent challenges to the ToM account of ASD, we are not inclined to view them as particularly persuasive ones. In our view, as highlighted above, it is essential to distinguish surface level behaviour (in this case, task performance) from the cognitive underpinnings of behaviour. The same behaviour may have different underlying cognitive causes in different disorders (Morton & Frith, 1995). In particular, several researchers have argued that, whereas children with ASD fail traditional false

belief tasks because they have diminished ToM competence as a result of a faulty underlying cognitive mechanism, children without ASD fail such tasks because of extraneous (performance) factors. In other words, children without ASD possess the basic underlying cognitive mechanism responsible for ToM, but fail to utilize this mechanism appropriately during ToM tasks. One way to test whether poor task performance has the same underlying cognitive cause across disorders is to manipulate the task of interest in systematic ways and investigate whether the manipulations affect performance in a comparable way in each disorder. If the underlying causes of ToM task success and failure are different amongst individuals with ASD than amongst children without ASD, then the issues of universality and diagnostic specificity do not provide strong challenges to the ToM account of ASD.

Adopting this approach, several studies have indicated that, in fact, different manipulations to false belief tasks affect the performance of children with different disorders in different ways. For example, Surian and Leslie (1999) found that asking children with ASD where Sally would look for her marble *first* (as opposed to merely asking where she would look) on a location change false belief task made no difference to their task performance. By contrast, this manipulation significantly increased the performance of typically developing 3-year-olds, as it also did in a study by Siegal and Beatie (1991). Conversely, Williams and Happé (2009b) found that if children with ASD were not allowed to verbalise their own false belief in an "unexpected contents" false belief task (Hogrefe, Wimmer, & Perner, 1986), then their performance on the subsequent "self test question" (which assessed awareness of own false beliefs) was significantly poorer than their performance on the "other-person test question", which assesses awareness of another person's false belief. In contrast, parallel performance across the self and other-person test questions was observed amongst both children with

intellectual disability and young typically developing children, regardless of whether or not they verbalised their own false belief. Furthermore, Peterson, Wellman and Liu (2005) found that children with ASD were unique in finding false belief tasks significantly more difficult than a task requiring the identification of a hidden emotion (e.g., in an individual who pretends not to be embarrassed, when they really are). Both typically developing and congenitally deaf children showed the opposite pattern of performance, finding it more difficult to identify a hidden emotion than a false belief.

These studies suggest that the underlying cognitive causes of false belief task *failure* amongst children with ASD are somewhat different to the causes underlying task failure amongst children without ASD. As such, the issue of diagnostic specificity of diminished ToM task performance is not such a challenge to the ToM account of ASD. Conversely, other studies suggest that *success* on traditional ToM tasks also has different causes in children with and without ASD. Several studies have observed a close connection between verbal ability and success on traditional false belief tasks. In particular, grammatical knowledge appears strongly tied to successful performance amongst children with ASD, but significantly less so (Fisher, Happé, & Dunn, 2005), or not at all (Lind & Bowler, 2009a) amongst children with intellectual disability. This has led to the claim that children with ASD employ compensatory strategies to "hack out" solutions to ToM tasks in the absence of true ToM competence (Bowler, 1992; Happé, 1995). Supporting this possibility, Senju, Southgate, White, and Frith (2009) found that adults with ASD who pass traditional, *explicit* false belief tasks nonetheless perform atypically on a measure of *implicit* ToM that requires attention to be spontaneously deployed on the basis of another person's mental state. This suggests that explicit knowledge of mental states amongst verbally able individuals with ASD has been acquired in an atypical fashion and *not* primarily

via the ToM mechanism. As such, the finding that some individuals with ASD perform well on traditional ToM tasks does not necessarily challenge the ToM account.

A third challenge to the ToM account concerns the fact that evidence for a direct link between ToM task performance and severity of clinical features in ASD is somewhat mixed. In two studies of general adaptive behavior amongst individuals with ASD, significant differences in specific aspects of social behavior were observed between those individuals who passed ToM tasks and those who did not (see Fombonne, Siddons, Achard, Frith, & Happé, 1994; Frith, Happé, & Siddons, 1994). Frith et al. interviewed parents about their children with ASD using, amongst other measures, an "Active and Interactive Sociability Scale". The Active Sociability subscale consisted of questions concerning social skills that were *not* thought to rely on mentalizing (e.g., saying "please" when requesting something; playing board games). The Interactive Sociability subscale consisted of questions concerning social skills that *were* thought to rely on mentalizing (e.g., keeping secrets; apologising for hurting another's feelings). In addition, the children with ASD, themselves, were given two standard false belief tasks. Frith et al. found that while there were no differences on the Active Sociability subscale between ToM passers and ToM failers, differences were observed on the Interactive Sociability subscale. Hence, on average, children who passed the false belief tasks were rated as showing superior interactive social skills than children who failed the false belief tasks. This supports the ToM account. However, Frith et al. also found that the real life social skills of a significant proportion of children in the study could not be distinguished by false belief task performance. Hence, a number of children who passed the false belief tasks nonetheless displayed diminished interactive social skills, suggesting that ToM is not closely related to severity of clinical features.

Frith et al.'s (1994) results were replicated and extended by Fombonne et al. (1994), providing only partial support for the ToM account. However, caution needs to be taken when drawing absolute conclusions from these studies. As highlighted above, some individuals with ASD may well hack out solutions to traditional ToM tasks, using intellectual strategies. Therefore, performance on false belief tasks may not relate to severity of social-communication deficits amongst individuals with ASD because performance on the false belief tasks may not always tap true ToM competence. It is essential that future studies of the relation between ToM and feature severity in ASD employ implicit measures of mentalizing ability, the solutions to which are not easily hacked out by explicit problem-solving strategies (e.g., Senju et al., 2009). For the ToM account of ASD to be plausible, a relation must be observed between implicit awareness of mental states on these tasks and severity of social-communication impairments in ASD.

Even if the ToM account can ultimately explain the social-communication deficits in ASD, it is less likely to explain adequately the repetitive and stereotyped behaviors and interests that characterize the disorder. Although repetitive behaviors and interests could be considered a reaction to anxiety and confusion caused by social incomprehension (caused ultimately by a ToM deficit; see Happé, Ronald, & Plomin, 2008), Turner (1999) has argued that this is unlikely to be the case. As she points out, repetitive behaviours have both an *arousing*, as well as a calming, function amongst many individuals with ASD. Thus, they are unlikely to be merely a coping strategy employed to deal with social-communication problems. As such, it seems that the ToM account may not be able to explain fully the whole range of clinical features in ASD.

Alternatives to the ToM hypothesis

Arguably, one of the most significant challenges to the ToM account of ASD is to explain those core social features of ASD that do not obviously require the high-level representation of mental states. Manifestations of primary inter-subjectivity, such as social smiling, social orienting, and the maintenance of eye contact – all of which are diminished in ASD – do not seem to require high level mentalizing. Rather, one might intuitively suppose that difficulties with spontaneous orienting to and establishing co-ordination with others are related to, or perhaps even underpin, difficulties with representing mental states in people. Indeed, Hobson (1989, 1993, 2002) has argued persuasively that difficulties with establishing and maintaining affective engagement with others *leads to*, rather than *results from*, a diminished ToM (related, but subtly different, accounts are provided by Loveland, 1991, and Klin, Jones, Schultz, & Volkmar, 2003). Hobson suggests that in typical development, young infants have a biologically-based predisposition to "identify" with the bodily-expressed stances and attitudes of others. For example, even from the earliest weeks of life, infants have been shown to coordinate their own feelings closely with those of others (Haviland & Lelwica, 1987). According to Hobson, it is through this (pre-conceptual, initially non-representational) process of being continually moved between different perspectives throughout early development that leads typically developing children to eventually *recognise* the distinction between perspective and reality (i.e., develop a ToM). According to Hobson, the diagnostic deficits in social-communication in ASD, as well as the characteristic deficits in ToM, are a consequence of an innately reduced propensity to identify with others.

Hobson's (1989, 1993, 2002) theory shares characteristics with the ToM account of ASD. It is "domain-specific", in that it places social deficits at the core of the disorder, and it emphasises children's difficulty with representing mental states as a cause of the characteristic

features of the disorder (e.g., deficits in pretend play). Arguably, however, it has the advantage of being able to account, in principal, for difficulties with primary inter-subjectivity, as well as with ToM, amongst people with ASD. Hobson's theory has not gone unchallenged by ToM theorists, however, who have argued that his theory fails to account for exactly how concepts of mind emerge from the pre-conceptual process of identification (see Leslie & Frith, 1990; Hobson, 2002, pp.255-260). Also, like the ToM account, Hobson's theory has difficulty explaining the apparently "non-social" repetitive behaviours and interests that characterize ASD. To explain these deficits, alternative cognitive accounts may be necessary.

The Executive Dysfunction Hypothesis

Although the definition of executive function is hotly debated (Jurado & Rosselli, 2007), the term is commonly used to refer to a broad range of high-level cognitive functions, primarily underpinned by the frontal lobes (Stuss et al., 2002), such as planning, cognitive flexibility/set shifting, inhibitory control, working memory, and generativity (Hill, 2004). Neuropsychological patients with specific damage to the frontal lobes show inflexibility of cognition and behavior that is reminiscent of that observed amongst individuals with ASD. This led to an early hypothesis that executive functions would be impaired in ASD and that this impairment could underpin the behavioral features of the disorder (e.g., Hughes, Russell, & Robbins, 1994; Pennington & Ozonoff, 1996).

The executive dysfunction hypothesis makes intuitive sense of the restricted, repetitive, and stereotyped patterns of behaviour in ASD. A difficulty in generating, planning, and controlling behavior would likely result in a tendency to engage in only a limited number of activities that would be repeated over and over again, with limited imagination or variation (e.g., Turner, 1999). The theory also has the potential to explain deficits in joint attention associated

with ASD. Joint attention requires the disengagement of attention from one's current focus and a shifting of attention to the object or event in the world that is the focus of another person's attention. As such, joint attention deficits in ASD might result from executive difficulties with set-shifting/cognitive flexibility (e.g., McEvoy, Rogers, & Pennington, 1993), rather than a failure to recognize mental states in others (e.g., Leslie & Happé, 1989). In a similar vein, some researchers have argued that deficits in pretend play are most parsimoniously explained by a limited ability to *generate* pretend scenarios (Jarrold, 2003), rather than an inability to *form* M-representations, as the ToM hypothesis suggests (Leslie, 1987). Some theorists have gone further than this, however, and suggested that executive dysfunction could have cascading effects that result in the full range of social-communication deficits, as well as repetitive behaviours, in ASD.

Arguably the most theoretically-developed executive dysfunction account of ASD was provided by Russell (e.g., 1996). According to Russell, the most basic executive function is the ability to monitor one's actions. Russell and Hill (2001, p.317) define action monitoring as, "the mechanisms that ensure that agents know, without self-observation, (a) for which changes in perceptual input they are responsible and (b) what they are currently engaged in doing". Therefore, effective action monitoring allows an individual to distinguish between 'self-caused' and 'world-caused' changes in experience and hence, in Russell's (1996) theory, gives rise to an experience of *agency*. This "sub-personal", non-conscious process, which is a pre-requisite for all other (higher) components of executive functioning (e.g., planning), is thought to involve generating a visual ('efference') copy of a motor intention for action and then monitoring this copy in relation to the sensory consequences of one's action (e.g., Wolpert, Ghahramani, & Jordan, 1995). According to Russell, a primary deficit in action monitoring results in a failure to

develop higher forms of executive functioning, which contributes to repetitive behaviors and interests in ASD. More importantly, in Russell's theory a deficit in action monitoring leads to a failure amongst individuals with ASD to experience their agency. As a developmental consequence, infants with ASD fail to recognize *others* as agents (with mental states), which directly causes their diagnostic social-communication impairments.

Evidence for the executive dysfunction hypothesis

Consistent with the executive dysfunction theory, several higher components of executive functioning have been shown to be diminished in ASD. For instance, planning ability is assessed most frequently using the Tower of London task (Shallice, 1982). This task involves moving beads across pegs from an initial starting position to match a particular specified end-state in as few moves as possible. Several studies have shown that individuals with ASD perform less well than matched comparison participants on this task, requiring a greater number of moves to complete the puzzle (e.g., Bennetto, Pennington, & Rogers, 1996; Ozonoff, Pennington, & Rogers, 1991; Pellicano, 2007).

Another component of executive functioning that appears reliably impaired amongst individuals with ASD is cognitive flexibility, which is traditionally assessed using the Wisconsin Card Sorting Test (WCST) (Heaton, 1981). In this test, participants are presented with four *stimulus* picture cards depicting (a) one red triangle, (b) two green stars, (c) three yellow crosses, and (4) four blue circles, respectively, as well as a deck of *response* picture cards that vary on the dimensions of number, colour, and shape. The participant is required to match the response cards to one of the stimulus cards according to one dimension specified by a particular unspoken rule (e.g., match according to colour). Although the experimenter does not explicitly state the sorting rule, she/he provides corrective feedback for each of the participant's card placements.

On the basis of this feedback, the participant must deduce the nature of the rule. Once the participant has provided 10 consecutive correct responses, unbeknownst to him/her, the experimenter changes the unspoken rule. Thus, in order to continue providing correct responses s/he must flexibly shift to the new rule (e.g., sort according to number). Individuals with ASD typically show significantly more perseverative errors on the WCST (e.g., Ambery, Russell, Perry, Morris, & Murphy, 2006; Dichter & Belger, 2007; Ozonoff, Pennington, & Rogers, 1991; Rumsey, 1985). Such findings are generally taken as evidence for impaired set shifting/cognitive flexibility in ASD. However, it is also possible that difficulties with other components of executive functioning, such as working memory and/or inhibition contribute to such perseveration.

Inhibitory control refers to "the ability to suppress the activation, processing, or expression of information that would otherwise interfere with the efficient attainment of a cognitive or behavioral goal" (Christ, Holt, White, & Green, 2007, p.1156). In ASD, it has been most commonly assessed using Stroop (Stroop, 1935) and Go/No-Go (Drewe, 1975) tasks. Standard Stroop tasks require participants to identify the color of the ink in which stimulus words are presented. In the key inhibitory control condition, the stimulus words are colour names printed in incongruent ink colours (e.g., "red" displayed in green font). In order to successfully name the colour of the ink, the participant must inhibit the automatic behavior of simply reading the word. Studies have overwhelmingly shown intact Stroop performance in ASD (e.g., Ambery, Russell, Perry, Morris, & Murphy, 2006), notably, even when participants with ASD have significantly lower IQs than typical comparison participants (e.g., Christ, Holt, White, & Green, 2007; Ozonoff & Jensen, 1999).

In Go/No-Go tasks, participants are repeatedly presented with two different stimuli (e.g., a square and a circle), one at a time, in a random order, and asked to respond to only one of them (e.g., by pressing the spacebar). Hence, they might be asked to press the spacebar when they see a square (Go trials) but not when they see a circle (No-Go trials). Hence, on the No-Go trials they must inhibit their motor response. Thus, the key variable of interest is the commission error rate on No-Go trials. The majority of studies employing well matched groups suggest unimpaired performance amongst individuals with ASD (e.g., Geurts, Begeer, & Stockmann, 2009; Happé, Booth, Charlton, & Hughes, 2006; Schmitz et al., 2006). Despite the fact that individuals with ASD have few difficulties on classic test of inhibition such as the Stroop and Go/No-Go tasks, they show impairments on tasks which require the inhibition of *prepotent responses* (Hill, 2004). For example, Hughes and Russell (1993) assessed this type of inhibition using a "detour reaching task", which involved retrieving a marble from a platform inside a box. Reaching directly for the marble caused an infrared light beam to be broken resulting in the marble dropping through a trapdoor, rendering it inaccessible. Therefore, to perform successfully on the task, the participant had to inhibit this prepotent response. Instead, they needed to either flick a switch on the side of box in order to turn off the beam of light and then directly pick up the marble, or turn a knob that caused the marble descend down a chute from which they could safely retrieve it. It was found that participants with ASD were significantly impaired on this task, finding it difficult to inhibit the urge to reach directly for the marble.

Working memory is another executive function that has been the focus of many studies of ASD. Working memory is a term used to refer to "a limited capacity system allowing the temporary storage and manipulation of information" (Baddeley, 2000, p.418). Working memory can be measured using "n-back" tasks, which involve both the short-term storage *and*

manipulation of information. Here, participants are asked to monitor a continuous sequence of stimuli (e.g., letters) and respond when the presented stimulus matches one presented n trials previously, where n is typically pre-specified as 1, 2, or 3 (Owen, McMillan, & Laird, 2005). Studies involving zero-back (where the target is the first item in the stimulus sequence), one-back, and two-back conditions have shown that individuals with ASD are unimpaired (Koshino et al., 2005; Ozonoff & Strayer, 2001; Williams, Goldstein, Carpenter, & Minshew, 2005). However, ceiling effects may have obscured possible group differences in these studies – the error rate was very low in each case. Indeed, it should be noted that the zero-, one-, and two-back conditions used in each of these experiments may have lacked the sensitivity needed to detect possible group differences in working memory - in other words the tasks were not taxing enough to reveal group differences if present. Indeed, Hockey and Geffen (2004) have argued that n-back tasks only involve a significant working memory load when n equals three or more.

Computerised box search tasks, such as the spatial working memory task from the Cambridge Neuropsychological Test Automated Battery (Cambridge Cognition, 1996), have also been used to assess working memory in ASD. In this type of task, participants must search through an array of boxes in order to retrieve hidden tokens. Importantly, they are asked to avoid opening boxes that they have already searched – tokens never appear in the same box more than once. They key dependent measure in such tasks is the number of search errors in which participants re-search an already searched box for a particular target token. The majority of studies indicate that individuals with ASD show significantly more search errors than age and IQ matched comparison individuals (Landa & Goldberg, 2005; Steele, Minshew, Luna, & Sweeney, 2007; Sinzig, Morsche, Bruning, Schmidt, & Lehmkuhl, 2008; but see Edgin & Pennington, 2005; and Happé, Booth, Charlton, & Hughes, 2006). Consistent with the suggestion made

above, in relation to methodological weaknesses in n-back studies, Steele et al. (2007) found that ASD-specific working memory impairments were load-dependent, such that difficulties only began to emerge in high load conditions. Thus, the evidence regarding working memory in ASD is equivocal – some studies find impairments, others do not. Further research using more demanding tasks will be required to resolve the debate.

It should be clear from this brief review of the literature that ASD does not involve blanket executive function deficits – some aspects appear to be impaired whilst others appear to be intact, and results are variable across studies. But what is the neural basis of those deficits that are present? Since the late 1970s, it has been hypothesized that specific abnormalities within the frontal lobes have a causal role in ASD (Damasio & Maurer, 1978) and such hypothesized abnormalities could potentially account for executive dysfunction. Indeed, a number of functional neuroimaging studies have indicated functional abnormalities within the frontal lobes of individuals with ASD during executive tasks (Di Martino et al., 2009). Unfortunately, many of these studies are not able to directly address the question of whether frontal lobe dysfunction underlies executive dysfunction in ASD. A number of investigations have demonstrated different levels and patterns of neural activity during executive task performance amongst individuals with ASD but this is alongside *intact* behavioural performance on those executive tasks (e.g., Gilbert, Bird, Brindleya, Frith, & Burgess, 2008; Kana et al., 2007; Koshino et al., 2005; Schmitz et al., 2006). Whilst such findings are interesting in their own right, they tell us little about the neural basis of executive function *impairments*. However, at least two functional imagining studies have established functional abnormalities in the presence of behavioural impairments. For example, Luna et al. (2002) found that individuals with ASD showed reduced levels of activation within the dorsolateral prefrontal cortex and posterior cingulate cortex during

a spatial working memory task. Furthermore, Just, Cherkassky, Keller, Kana, and Minshew (2007) observed similar levels of activation between participants with and without ASD but reduced functional connectivity between frontal and parietal areas during the Tower of London planning task. Findings such as these provide a plausible explanation for why some individuals with ASD experience executive dysfunction. However, in order to further clarify these issues, it will be important for imaging researchers to carefully select behavioral paradigms that are robustly found to be problematic for individuals with ASD.

One significant strength of the executive dysfunction hypothesis is that it potentially explains the RRBIs that characterize ASD. Although there have been some negative or mixed findings (e.g., Dicher et al., 2009; South, Ozonoff, & McMahon, 2007), a number of studies have reported significant correlations between executive dysfunction and level of RRBIs. For example, Lopez, Lincoln, Ozonoff, and Lai (2005) found that cognitive flexibility, working memory, and inhibition (but not planning or fluency) were significantly related to RRBIs. In another study, Boyd, McBee, Holtsclaw, Baranek, and Bodfish (2009) assessed the relationship between executive dysfunction and repetitive behaviours using the Behavior Rating Inventory of Executive Function (BRIEF) (Gioia, Isquith, Guy, & Kenworthy, 2000) and the Repetitive Behavior Scale-Revised (RBS-R) (Bodfish, Symons, & Lewis, 1999; Lam & Aman, 2007). The BRIEF is a questionnaire that yields both a composite score and two separate indices: Behavioral Regulation (subscales: inhibition, shifting attention, and emotional control) and Metacognition (subscales: initiating, working memory, planning/organizing, organization of materials, and monitoring). The RBS-R is a questionnaire that assesses various repetitive behaviours across six subscales (stereotypy, self-injurious behavior, compulsions, rituals, sameness and restricted interests). Boyd et al. found that RBS-R total score was significantly correlated with the BRIEF

Behaviour Regulation score but not BRIEF Metacognition or Composite scores. These findings suggest that the executive dysfunction theory offers a credible explanation for at least some of the clinical features of ASD.

Challenges to the executive dysfunction hypothesis

Despite the evidence discussed in support of the executive dysfunction account, several challenges have been made to the account in recent years. Firstly, although some higher-order components of executive functioning are impaired in ASD, basic action monitoring skills appear to be preserved, contrary to Russell's (1996) central claim. Williams and Happé (2009c) explored action monitoring amongst children with autism, using a task adapted from Russell and Hill (2001). In this task, participants needed to judge which one of several colored squares on a computer screen was under their intentional control (through movements of the mouse) and which 'distractor' squares were under the control of the computer. So, although all of the squares on the screen were activated when the mouse was moved, only one of the squares behaved directly in accordance with the participant's movements. The movements of the other squares were randomly generated by the computer. Ensuring that participants could not solve the task by visually comparing the movements of their hand with the movements of the squares on the screen (i.e., through self-observation), their hand was located inside a box while they moved the mouse. Therefore, to recognize the square that they were controlling, participants needed to monitor accurately an efference copy of their motor command and compare this to the visual input from the computer screen (i.e., to detect their own agency). Williams and Happé found that adolescents with ASD performed well on this task and not significantly differently from closely-matched comparison participants. Moreover, Williams (in press) confirmed that participants with ASD in Williams and Happé's study showed significantly diminished ToM.

Therefore, there was no evidence either that action monitoring was impaired in ASD, or that awareness of one's own agency resulting from action monitoring is sufficient to recognize others as agents with mental states (see also David et al., 2008; Frith & Hermelin, 1969).

Although these findings provide strong evidence against Russell's (1996) version of the executive dysfunction account of ASD, they do not necessarily show that impairments in other components of executive functioning are not a primary cognitive deficit in ASD. This latter suggestion is challenged by other findings, however. Firstly, although diminished cognitive flexibility/set-shifting, inhibition of a prepotent response, and working memory (see above) have been observed in children with ASD from around 5.5 years of age (e.g., Dawson, Meltzoff, Osterling, & Rinaldi, 1998; McEvoy, Rogers, & Pennington, 1993), studies of younger children with ASD have failed to find evidence of deficits in any of these areas. In studies by Dawson et al. (2002) and Griffith, Pennington, Wehner, and Rogers (1999), children with ASD between the ages of 3 and 5 years were tested on a variety of executive functioning tasks that are suitable for young children and on which impairments are seen in monkeys with lesions to the prefrontal cortex (e.g., Diamond & Goldman-Rakic, 1989). In particular, in contrast to studies involving older children with ASD, no differences were seen between children with and without ASD on a Spatial Reversal task (assessing cognitive flexibility and inhibition of a prepotent response), the A not B task (assessing working memory and inhibition of a prepotent response), or Boxes tasks (assessing working memory).

These findings amongst young, preschool children with ASD cast doubt on the hypothesis that impairments in (at least these components of) executive functioning represent a primary cognitive deficit in ASD. Arguably, a parsimonious interpretation is that executive dysfunction *emerges* with age in ASD and is a secondary consequence of other deficits in this

disorder. Establishing this possibility will, ultimately, require longitudinal studies of executive functioning in ASD, using a variety of measures that are sensitive to executive dysfunction.

Consideration of universality and specificity is also pertinent when evaluating the executive dysfunction hypothesis. With regard to universality, it is clear that results are mixed, with many studies obtaining null results. Even within those studies that have found executive difficulties on the group level, there is still considerable variability *within* the groups. For example, Pellicano, Maybery, Durkin, and Maley (2006) found that only 48% of children with ASD fell at least one standard deviation below the mean of the comparison group on the Tower of London planning task, and only 55% on a set-shifting task similar to the WCST. It should also be noted that executive dysfunction is certainly not specific to ASD – it is common in other disorders such as attention deficit hyperactivity disorder (ADHD) and Tourette syndrome, which involve a very different set of behavioural impairments to those seen in ASD. However, what may prove to be specific to ASD is the executive dysfunction *profile* that characterizes the disorder (Happé, Booth, Charlton, & Hughes, 2006; Ozonoff & Jensen, 1999).

The Weak Central Coherence Hypothesis

The weak central coherence hypothesis of autism was originally proposed by Frith (1989, 2003). The hypothesis draws on ideas from gestalt psychology and assumes that whilst typically developing individuals have "a built-in propensity to form coherence over as wide a range of stimuli as possible, and to generalize over as wide a range of contexts as possible" (Frith, 2003, pp.159-160), individuals with ASD have a diminution in this propensity. Metaphorically, according to Frith's original weak central coherence account, children with ASD are unable to "see the wood for the trees". Weak central coherence was originally conceptualized as a core *deficit* in extracting meaning and processing wholes. However, more recent versions of the

theory have adopted the idea that weak central coherence is a "cognitive style" rather than a cognitive deficit (Happé, 1999). According to this view, individuals can fall anywhere on the continuum of central coherence, ranging from preferential processing of parts – local bias – to preferential processing of wholes – global bias. Thus, individuals with ASD tend to show a cognitive style that falls towards the "local" end of the continuum.

Weak central coherence was originally thought to contribute to the social features of ASD and Frith (1989) hypothesised that weak central coherence may underlie ToM impairments in the disorder. However, more recent versions of the theory have retracted from this argument; instead focusing on the theory's potential to account for non-social features and perceptual anomalies (Happé & Frith, 2006). For example, the hypothesis is consistent with Kanner's (1943) early observation that individuals with autism show an "inability to experience wholes without full attention to the constituent parts" (p.246), and clinical observations that children with autism tend to be pre-occupied with parts of objects (e.g., spinning the wheels on toy cars) and show increased sensitivity to minute changes in the environment.

Evidence for the weak central coherence hypothesis

Although there are a number of contradictory findings, several sources of evidence suggest that individuals with ASD show *superior* performance when tasks depend on processing of local features but *inferior* performance when tasks depend on processing of global features, providing support for the weak central coherence hypothesis (see Happé & Frith, 2006, for a recent review). Studies of block design represent one particularly compelling source of evidence for the hypothesis. The block design test is a subtest from the Wechsler Intelligence Scales (e.g., Wechsler Intelligence Scale for Children, Wechsler, 2004) and requires participants to reproduce two-dimensional, red and white, geometric patterns using a set of three-dimensional cubes,

which have two red, two white, and two diagonally oriented half-red and half-white sides. Although there are a handful of contrary findings (e.g., Bölte, Holtman, Poustka, Scheurich, & Schmidt, 2007), it is generally accepted that individuals with ASD show superior performance on the block design test (e.g., Caron, Mottron, Berthiaume, & Dawson, 2006). According to the weak central coherence hypothesis, this performance peak is attributable to the fact that individuals with ASD process the two-dimensional patterns in a piecemeal fashion, automatically focusing on the constituent parts of the design.

Support for this interpretation was provided in a landmark study by Shah and Frith (1993). Both low- and high-functioning adolescents/young adults with autism were asked to complete two different versions of the block design task: the standard "un-segmented" version and a "pre-segmented" version in which the constituent blocks were shown spaced apart in the two-dimensional design to be copied. It was found that participants with autism showed significantly superior performance (i.e., construction times were significantly faster) only in the un-segmented version of the task. The authors argued that the pre-segmented version effectively eliminated the advantage that individuals with ASD had over typical individuals in the un-segmented version by removing the global image and highlighting the constituent parts (already apparent to participants with ASD) to the non-autistic participants. These findings have recently been replicated by Caron, Mottron, Berthiaume, and Dawson (2006) in a sample of high-functioning adults with autism. However, Kaland, Mortensen, and Smith (2007) reported no significant group differences in either segmented or un-segmented block design between high-functioning adolescents with autism/Asperger's syndrome and typical adolescents (matched on chronological age, VIQ, PIQ, and FSIQ). In fact, the ASD group was a little slower than the comparison group in each case.

Research using the Embedded Figures Test (Witkin, Oltman, Raskin, & Karp, 1971) has also been cited in support of the weak central coherence hypothesis. The task is to search for and identify a simple hidden target shape within a more complex picture (e.g., a triangle within a picture of a pram). Shah and Frith (1983) found that low-functioning children with ASD achieved significantly higher accuracy on this task than age and non-verbal mental age matched comparison children with intellectual disability (55%) and non-verbal mental age matched typically developing children. In a later study of high-functioning adults with autism or Asperger syndrome, Jolliffe and Baron-Cohen (1997) found that although all groups showed a similar level of *accuracy*, whilst typical participants took an average of 53 seconds to locate the hidden figures, participants with autism or Asperger syndrome (matched for age, VIQ, PIQ and FSIQ) took only 29 and 32 seconds, respectively.

Shah and Frith argued that individuals with ASD experience less "capture by meaning". In other words, individuals with ASD are faster or more accurate on the task because they are unhindered by the overall global meaning of the picture (e.g., the fact that it is a picture of a *pram*). Although a number of studies have replicated the finding that individuals with ASD outperform matched comparison individuals on the embedded figures test (Edgin & Pennington, 2005; Jarrold, Gilchrist, & Bender, 2005; Pellicano, Maybery, Durkin, & Maley, 2006), there have also been a number of contrary findings (Bölte, Holtman, Poustka, Scheurich, & Schmidt, 2007; Morgan, Maybery, & Durkin, 2003; Schlooz et al., 2006; Kaland, Mortensen, & Smith, 2007).

Susceptibility to visual illusions is also thought to be mediated by central coherence. Happé (1996) found that low-functioning children with autism were less likely to succumb to visual illusions such as the Müller-Lyer figures and Kanisza triangle than comparison children

(matched for age and verbal IQ). Confirming this finding, a recent study by Bölte, Holtman, Poustka, Scheurich, and Schmidt (2007) revealed that high-functioning adults with autism were less susceptible to visual illusions than typical comparison adults (matched on verbal IQ, performance IQ, and age). Other studies have failed to find evidence for decreased susceptibility to visual illusions in ASD (Hoy, Hatton, & Hare, 2004; Ropar & Mitchell, 1999; Ropar & Mitchell, 2001). However, each of the studies that obtained null results failed to include comparison groups that were closely matched on age and intellectual ability. For example, in the Hoy et al. study, the autism group was younger and less verbally able than the comparison group. Indeed, the effect sizes for the differences between the autism and comparison groups on age (Cohen's $d = 1.05$) and verbal mental age (Cohen's $d = 0.54$) were large and moderate, respectively, indicating that the groups were not closely matched. The validity of these findings is therefore questionable.

Studies of homograph reading are another key source of evidence that is consistent with the weak central coherence hypothesis. Homographs are words with one spelling but multiple pronunciations and meanings (e.g., "She had a pink *bow.*" versus "She made a deep *bow.*"). In homograph reading tasks, participants are required to read aloud sentences that include homographs (e.g., "It was *lead* in the box that made it so heavy."). In such tasks, the correct pronunciation for the homograph can only be ascertained through considering the overall context of the sentence. It has been consistently found that children (Frith & Snowling, 1983, Experiment 5), adolescents (Happé, 1997; López & Leekam, 2003), and adults (Jolliffe & Baron-Cohen, 1999, Experiment 1) with ASD are significantly more likely to mispronounce homographs, tending to adopt the most common pronunciation without taking into account the sentence context. Frith and Snowling suggest that the tendency to mispronounce homographs in

context arises from using a word-by-word reading strategy – evidence for weak central coherence.

By and large evidence from block design, embedded figures, visual illusions, and homograph reading is consistent with the idea that weak central coherence can result in either inferior or superior performance, depending upon the particular task demands. Superior block design/embedded figures test performance and decreased susceptibility to visual illusions, alongside inferior homograph reading suggests that weak central coherence operates at both the perceptual and conceptual levels.

Challenges to the weak central coherence account of ASD

As is the case for impaired ToM and executive dysfunction, weak central coherence does not appear to be universal in ASD. Teunisse, Cools, van Spaendonck, Aerts, and Berger (2001), for example, found that although weak central coherence was more common in ASD, it was not universally found – only 57% (20/35) of young adults with ASD showed this cognitive style. On the other hand, Pellicano, Mayberry, Durkin, and Maley (2006) found that 92% of children with ASD were more than one standard deviation quicker than the mean of the matched comparison children on the embedded figures test. There are also questions over the diagnostic specificity of weak central coherence to ASD. For example, in their study of visual illusions, Bölte et al. (2007) found that adults with depression or schizophrenia were less susceptible to visual illusions than typical adults and did not significantly differ from participants with ASD in terms of level of susceptibility.

Perhaps more importantly, the explanatory power of the weak central coherence hypothesis is rather limited. At best, it is likely to explain only limited aspects of the behavioural phenotype. Although few studies have empirically assessed the relationship between weak

central coherence and the clinical features of ASD, those that have generally fail to find significant relationships with either social impairments (Teunisse et al. 2001) or RRBIs (South et al. 2007). This would seem to suggest that weak central coherence should be viewed as largely an explanation of non-triad features of ASD, which affect some individuals but not others. Intriguingly, however, research employing a non-clinical sample indicates a positive relationship between weak central coherence and levels of autistic-like traits (Stewart, Watson, Allcock, & Yaqoob, 2009).

Alternative explanations for weak central coherence "effects"

One of the main limitations of the weak central coherence account is that it remains largely descriptive – no specific cognitive or neural mechanisms for local bias are posited. Alternative accounts for weak central coherence "effects" have gone some way towards specifying the underlying mechanisms responsible.

The enhanced perceptual functioning theory (Mottron & Burack, 2001; Mottron, Dawson, Soulieres, Hubert, & Burack, 2006) is the probably the most influential alternative explanation for weak central coherence "effects". According to this theory, individuals with ASD do not show deficits in global processing or a bias towards local processing (as suggested by the weak central coherence account) but, rather, show *enhanced* perceptual processing, which results in a local bias. Here, perception is broadly defined and is said to range from "feature detection up to and including pattern recognition" (Mottron, Dawson, Soulieres, Hubert, & Burack, 2006, p.28).

In terms of their neurobiological basis, Brock, Brown, Boucher, and Rippon (2002) have hypothesized that weak central coherence effects in ASD are underpinned by a deficit in temporal binding. Thus, the brain activity of individuals with ASD is characterized by

diminished temporal synchronization of neuronal activity between functionally specialized local neural networks. Brock et al. argue that individuals with ASD therefore show diminished performance on experimental tasks that require the integration of information from different functionally specialized local networks but undiminished performance on tasks that depends only upon isolated local networks. In a similar vein, Just, Cherkassky, Keller, and Minshew (2004) propose that underconnectivity – diminished coordination and communication – between brain regions underlies weak central coherence effects. Like the temporal binding hypothesis, the underconnectivity hypothesis also predicts that individuals with ASD should perform well when tasks do not require coordination of disparate cortical regions but poorly when tasks depend upon the multiple cortical regions. Data from a number of fMRI studies do seem to suggest reduced functional connectivity in ASD (Castelli, Frith, Happe, & Frith, 2002; Just, Cherkassky, Keller, & Minshew, 2004).

The Future of Psychological Theories of ASD: Multiple or Single Deficit Accounts and the Possible Fractionation of the Triad

Cognitive theories of ASD have generally attempted (with varying degrees of success) to identify a *single* primary cognitive deficit as the causal factor underlying the complete triad of behavioural impairments that characterize ASD. However, it is becoming clear that, standing alone, none of the existing cognitive theories (the most influential of which have been described above) provide a satisfactory explanation for the full range of behavioural deficits seen in ASD. For this reason, the trend towards *multiple* deficit accounts is now gaining momentum (Bishop, 1989; Boucher, 2006; Goodman, 1989; Happé, Ronald, & Plomin, 2006). Researchers have also begun to question whether the domains of the triad of impairments are separable. If they are separable, then arguably we already have adequate theoretical explanations for the individual

triad domains. For example, Happé and Ronald (2008) suggest that whilst ToM impairments may account for social and communication impairments, executive dysfunction may account for RRBIs. On the other hand, if the triad of impairments is not fractionable, it is clear that we have a lot of theory building left to do. Clearly then, the future of psychological models of ASD will critically depend on the outcome of research investigating the relations amongst the triad of impairments.

The earliest study to address this question was conducted by Wing and Gould (1979). They selected a sample of 132 children from a "psychiatric and mental retardation register", who had severe intellectual disability (mental retardation) or one or more of the following clinical features: (a) impairment of social interaction; (b) impairment of verbal and nonverbal communication; (c) repetitive, stereotyped activities. On the basis of clinical assessments, Wing and Gould established that impairments in each domain tended cluster together within individuals. For example, of the 74 children with social impairment, 55% had no speech, and 72% showed RRBIs. However, we can only draw limited conclusions from these findings, given that definitions and criteria for ASD have changed so significantly since 1979 (Happé & Ronald, 2008). Another notable disadvantage of this study is that the sample was drawn from a register of psychiatric patients – a population that is likely to show higher rates of co-morbidity (e.g., between each domain of the triad) than community samples. This sampling method may therefore artificially elevate the number of individuals in the sample who have impairments across multiple domains (Caron & Rutter, 1991).

More recent research has overcome this limitation by employing population based community samples and "bottom-up" factor analytic techniques. This type of research takes a dimensional rather than categorical approach to the ASD triad and assumes that autistic-like

traits within each of the triad domains vary continuously in the general population (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001; Constantino & Todd, 2003). Here, sub-clinical levels of autistic-like traits are referred to as the "broader autism phenotype". Using this type of approach, Constantino et al. (2000) assessed 287 school-aged typically developing children and 158 child psychiatric patients using the Social Reciprocity Scale (SRS) (now referred to as the "Social *Responsiveness* Scale"; Constantino, 2002) – a 65 item, parent/teacher-rated quantitative scale, measuring autistic-like traits. A factor analysis revealed that a single factor (rather than three factors as would be expected if the triad is fractionable) explained 70% of the variance in SRS scores.

However, studies using other measures of autistic-like traits have generally found multiple factor solutions, ranging from 2 to 8 factors (Auyeung, Baron-Cohen, Wheelwright, & Allison, 2005; see Happé & Ronald, 2008, for a review; Hoekstra, Bartels, Cath, & Boomsma, 2008). For example, Austin (2005) assessed 201 undergraduate students using the Autism-spectrum Quotient (AQ) (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001) – a 50 item self-report questionnaire – and found that a three factor model explained 28% of the variance in scores. These finding were closely replicated in a recent study of 1005 psychology undergraduate students (Hurst, Mitchell, Kimbrel, Kwapil, & Nelson-Gray, 2007). However, it should be noted that each of these studies used an oblique rotation method, which allows factors to be correlated. Neither study reported the inter-factor correlations, making it difficult to judge the degree of independence amongst the factors. A recent study by Stewart and Austin (2009) employing a sample of 536 university students, reported a four factor solution, accounting for 29% of the variance in AQ scores. These factors were labeled as follows: (1) Socialness, (2) Patterns, (3) Understanding Others/Communication, and (4) Imagination. The correlations

between each pair of factors ranged from .00 to .22, with the highest correlation being between socialness and understanding others/communication.

Clearly, there is a great deal of variation in results both within and between the different types of measure of autistic traits. Overall, these findings call into question the notion of a *triad* of domains of autistic-like impairments in the general population, with the majority of studies suggesting either fewer or a greater number of domains. What is also unclear is the degree to which these domains are correlated with one another. Studies of clinical populations provide equally variable results, with some studies suggesting a single factor (e.g., Constantino et al., 2004; Szatmari et al., 2002) and others suggesting multiple factors (e.g., Dworzynski, Happé, Bolton, & Ronald, 2009; Mandy & Skuse, 2008). One point to bear in mind when considering the results of these studies is that factor structure may be an artifact of the particular *measure* used rather than a reflection of any objectively independent causal factors.

The question of whether or not ASD involves a triad of independent impairments is yet to be definitively answered. Undoubtedly, there will be an intensive research effort to this end in the coming years. This research will have clear implications for psychological accounts of ASD, which will need to be refined according to the outcome of this research effort. Equally, a better understanding of the cognitive basis/bases of ASD could help to shape our understanding of the etiology of the disorder. For example, Bishop (e.g., 2006) has argued that one way for genetic studies to circumvent the difficulties associated with defining the behavioural phenotype is to "cut loose from conventional clinical criteria for diagnosing disorders and to focus instead on measures of underlying cognitive mechanisms. Psychology can inform genetics by clarifying what the key dimensions are for heritable phenotypes" (p.1153). By developing a finer-grained understanding of cognition in ASD, it might be possible to focus down on a set of genes that are

reliably associated with a specific cognitive endophenotype in ASD (as has been done in other disorders; e.g., SLI Consortium, 2004). Vice versa, such genetic studies may also clarify the nature of cognition in developmental disorders like ASD (e.g., Bishop et al., 1999). Either way, we believe that a complete account of ASD will require an understanding of the cognitive underpinnings of the disorder.

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