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Abstract: At the heart of debates over underlying causes of autism is the "Kanner hypothesis" that autistic deficits in social reciprocity, and a cognitive/perceptual 'style' favouring detail-oriented cognition, co-vary in autistic individuals. A separate line of work indicates these two domains are normally distributed throughout the population, with autism representing an extremity. This realisation brings the Kanner debate into the realm of normative co-variation, providing more ways to test the hypothesis, and insights into typical development; for instance, in the context of normative functioning, the Kanner hypothesis implies social costs to spatial/numerical prowess. In light of this growing body of research, we review relevant factor analytic and correlational, behavioural studies. Findings are then synthesised into three themes: an alternative triad of primary autistic trait categories - Social Interaction Deficits, Cognitive Inflexibility, and Sensory Abnormalities - that more accurately reflects the factor structure of autistic traits; continuity between clinical and non-clinical autism-spectrum trait presentation; and indications that although social and non-social autistic traits may be initially independent, Kanner-like co-variance emerges behaviourally from dynamic trait interactions over the course of development. A dynamic developmental model subsuming these patterns is offered, and its advantages demonstrated in a novel account of ritualistic behaviours: as developmentally emergent, compensatory mechanisms for interactions between cognitive inflexibility and sensory abnormalities. We conclude with the broader imperative that behavioural scientists appealing for directly and exclusively genetic links may instead benefit from a developmental framing within their own discipline.

Highlights:

- We review and synthesize evidence for the “Kanner hypothesis” of co-varying autistic social deficits and detail-oriented cognition prowess, in clinical and typically developing populations
- Evidence suggests an alternative triad of autistic trait categories – social interaction deficits, cognitive inflexibility, and sensory abnormalities – that more accurately reflects the factor structure of autistic traits
- Evience suggests continuity between clinical and non-clinical autism-spectrum trait presentation
- Evidence suggests Kanner-like covariance emerges behaviourally from dynamic trait interactions over the course of development
- A developmental dynamic interactionist model best accounts for these patterns of evidence

INTERDEPENDENCE OF AUTISTIC TRAITS WITHIN AND BEYOND THE SPECTRUM

Detail-Oriented Cognitive Style and Social Communicative Deficits, within and beyond the Autism Spectrum: Independent Traits that Grow into Developmental Interdependence

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Abstract

At the heart of debates over underlying causes of autism is the "Kanner hypothesis" that autistic deficits in social reciprocity, and a cognitive/perceptual 'style' favouring detail-oriented cognition, co-vary in autistic individuals. A separate line of work indicates these two domains are normally distributed throughout the population, with autism representing an extremity. This realisation brings the Kanner debate into the realm of *normative* co-variation, providing more ways to test the hypothesis, and insights into typical development; for instance, in the context of normative functioning, the Kanner hypothesis implies social *costs* to spatial/numerical prowess. In light of this growing body of research, we review relevant factor analytic and correlational, behavioural studies. Findings are then synthesised into three themes: an alternative triad of primary autistic trait categories – *Social Interaction Deficits*, *Cognitive Inflexibility*, and *Sensory Abnormalities* – that more accurately reflects the factor structure of autistic traits; continuity between clinical and non-clinical autism-spectrum trait presentation; and indications that although social and non-social autistic traits may be initially independent, Kanner-like co-variance emerges behaviourally from dynamic trait interactions over the course of development. A dynamic developmental model subsuming these patterns is offered, and its advantages demonstrated in a novel account of ritualistic behaviours: as developmentally emergent, compensatory mechanisms for interactions between cognitive inflexibility and sensory abnormalities. We conclude with the broader imperative that behavioural scientists appealing for directly and exclusively genetic links may instead benefit from a developmental framing within their own discipline. (242 words)

Keywords: autism spectrum conditions, diagnostic triad, broader autism phenotype, normative cognitive variation, empathising, systemising

Current consensus holds that autism represents the extreme of a spectrum subsuming certain cognitive, social, and behavioural characteristics. These characteristics are categorised behaviourally in terms of the diagnostic "triad" of social interaction deficits, communicative deficits, and restricted and repetitive behaviours and interests (American Psychiatric Association, 1994). They are described theoretically in terms of deficits in Theory of Mind (ToM) or understanding intentionality (Baron-Cohen, 1995; Baron-Cohen, Leslie, & Frith, 1985); deficits in future-oriented cognitive flexibility, or executive functioning (EF) (Ozonoff, Pennington, & Rogers, 1991); and a bias toward local over global information processing, or weak central coherence (WCC) (Frith & Happé, 1994). In the terms of normative psychology, these two groups of traits are equivalent to Trope's notions of decreased "psychological distance" and low "level of construal," respectively (see Trope & Liberman, 2010). Less extreme positions along the autism spectrum are occupied by Asperger syndrome (AS), pervasive developmental disorder not otherwise specified (PDD-NOS), and the more subtle, sub-clinical idiosyncrasies of many first-degree relatives of individuals with autism, the sort described by Kanner in his seminal work (1943). This latter, broader autism phenotype has been empirically distinguished to such a degree that elements of it are sometimes referred to as the proper-noun Broader Autism Phenotype, or BAP (Baron-Cohen & Hammer, 1997a; Dawson et al., 2002; Happé et al., 2001; Lord, Cook, Leventhal, & Amaral, 2000; Piven, 1999).

The "Kanner Hypothesis"

Researchers and clinicians have long wondered whether social (e.g., ToM deficits) and non-social (e.g., WCC) aspects of ASC are related, their severities co-varying between individuals in a manner implying a shared aetiology, and potentially a singular cause of autism. Both previous reviews of this topic (Mandy & Skuse, 2008; Happé & Ronald, 2008) argued that although the idea of social/non-social co-variance is an assumption rooted more in the history of autism than in empirical evidence, it continues to guide our search for autism's causes. According to Mandy & Skuse (2008), assumptions of social/non-social co-variance are traceable to Kanner's original case descriptions (Kanner, 1943; Eisenburg & Kanner, 1956): "it was Kanner who first proposed the association between social-communication and non-social impairments as part of an autism syndrome" (Mandy & Skuse, 2008, p. 797).Kanner (1943), and Eisenburg and Kanner (1956) make no explicit assertions or speculations about ætiological associations between "extreme aloneness" and "preoccupation with the preservation of sameness" ASC traits. To the contrary, their only explicit assertion is that there is "little likelihood that a single etiologic agent is solely responsible for the pathology in behaviour" (Eisenburg & Kanner, 1956, p. 563). Yet Mandy and Skuse's interpretation is not unreasonable, as Kanner's case descriptions do seem to portray "aloneness" and "sameness" as two sides of the same coin. For instance, Kanner argues that when people interfere with ASC individuals' "excellent, purposeful, and 'intelligent' relations with objects that do not threaten to interfere with their aloneness," they are treated as objects: "If dealing with another person becomes inevitable, then a temporary relationship is formed with the person's hand or foot as a definitely

detached object, but not with the person himself." (Kanner, 1943, p. 249). Alternatively, Happé and Ronald (2008) argue that while Kanner established social deficits and cognitive inflexibility as the two core features of autism, assumptions of social/non-social relatedness began with the implementation of the diagnostic triad (Happé and Ronald, 2008). This clinical definition of autism, the authors say, simply set in stone the interpretations of Kanner's work reflected in Mandy and Skuse's inferences. Whether the so-called "Kanner hypothesis" – that "aloneness" is the social manifestation of a need for "sameness" – began with Kanner himself, or with the clinical definition of autism, it is an assumption with generational amnesia, forgetting that Kanner's seminal work was based on cases presenting with social and non-social symptoms to begin with, to the selective exclusion of cases where social or non-social deficits were absent. Assessing the validity of evidence and causal theories based on how well they account for social and non-social symptoms simultaneously thus may be an exercise in circular logic (Happé & Ronald, 2008; Happé, Ronald, & Plomin, 2006). Nevertheless, the assumption of social/non-social relatedness has continually re-emerged in autism theories and debates since Kanner's time (for a historical review, see Happé & Ronald, 2008), most recently in searches for a common genetic ætiology for social and non-social traits.

The Kanner Hypothesis in the Normative Population

A recent line of research has extended the autism spectrum into and throughout the normative population, such that social and non-social autistic traits are normally distributed across all individuals, with social deficits and cognitive idiosyncrasies taking subclinical forms. On these dimensional axes, clinically diagnosable autism differs from normative functioning

quantitatively and by degree, rather than being qualitatively distinct from normative cognitive variation. The best known of these descriptions is Empathising-Systemising (E-S) theory, which holds that ASC is an extreme version of the human brain in terms of its neuroarchitectural "male-ness," the prototypical "male brain" being organised for superior "systemising" (Baron-Cohen, 2002; Baron-Cohen & Hammer, 1997b), or processes of observation-based rule-making reducing the world to a series of "lawful, finite, and deterministic" rules. The prototypical "female brain," by contrast, is better organised for "empathising," the ability to accurately attribute and affectively respond to others' mental states (Baron-Cohen, 2002).

The full range of these attributes, from high to low empathising and high to low systemising, constitutes the entire E-S spectrum, within which the general population is normally distributed (Baron-Cohen & Hammer, 1997b). One's position on this continuum is thought to be determined partly by levels of prenatal testosterone exposure (Baron-Cohen, Lombardo, Auyeung, Ashwin, Chakrabarti, & Knickmeyer, 2011; Baron-Cohen, Lutchmaya, Baron-Cohen, Lutchmaya, & Knickmeyer, 2004), with higher levels of prenatal exposure producing a more lateralised, systemising-oriented, "male" brain (Witelson & Nowakowski, 1991; Geschwind & Galaburda, 1987), gestational overexposure to testosterone being only one amongst many interacting factors that can bias brain and cognitive development towards an autistic outcome. Supporting this assertion is evidence of higher testosterone levels in amniocenteses of individuals presenting with autism-like empathising difficulties and systemising prowess later in development (Baron-Cohen et al., 2004), and lower than average second-to-fourth digit length ratios (a biomarker proxy measure of prenatal testosterone exposure) among ASC individuals

and their first degree relatives, individuals manifesting the BAP, and individuals with systemising prowess and sub-clinical empathising difficulties (see Valla & Ceci, 2011 for a full review).

As E-S theory was built upon – or, perhaps, to account for – the assumptions of social and non-social co-variance in clinical populations that began with Kanner, it is worth questioning the Kanner hypothesis not only in the context of clinical presentation, but in typical development, too (Carroll & Chiew, 2006; Valla et al., 2010). If the E-S factor space comprises a single axis with high empathising and low systemising at one end and low empathising and high systemising at the other, as initially described (Baron-Cohen & Hammer, 1997b), then systemising ability should carry with it *tradeoffs* in empathising, and *vice versa*. Such would be, in effect, a normative-variation extension of the Kanner hypothesis. An alternative model, implicit in subsequent work (Baron-Cohen, 2002), presents the E-S factor space as bidimensional, wherein empathising and systemising vary *independently*, an autistic, extreme male brain arising from concurrently low empathising and high systemising abilities, an extreme female brain arising from the opposite pattern. This alternative, two-dimensional model of the E-S factor space implies no tradeoffs between systemising and empathising.

This question of E-S co-variance carries substantial import for understanding normative cognition. For one, an inverse E-S co-variance pattern in the normative population (i.e. the single-axis E-S model) would mean that mathematical and spatial skills or deficits would predict such seemingly unrelated social deficits or skills as understanding intentionality. In lay terms,

testing for this type of E-S co-variance in the normative population means questioning the stereotype of spatial and/or mathematical prowess going hand-in-hand with social ineptitude.

Insofar as any normative E-S co-variance patterns may hold also at the clinical extremity, evidence for or against E-S co-variance in the normative population would also be essential to consider alongside evidence for or against the Kanner hypothesis in clinical presentation. More to the point, E-S independence in typically developing individuals would bring into question theories attempting to account for ASC with a single-cause explanation, and the broader idea of ASC arising from coincidence of partially independent but synergistic factors might begin to seem more likely (Happé, Ronald, & Plomin, 2006). Thus, whilst understanding E-S co-variance patterns can tell us important things about normative functioning, it can also inform us on the causes underlying ASC.

Specifically, social/non-social independence may be particularly pronounced in cases or populations where levels of autistic traits are subtle and where, therefore, mutually reinforcing interactions amongst these symptom domains are weak. Whilst the debate has been framed here as one of evidence for *or* against social/non-social co-variance, the distinction between co-variance on the one hand and independence between autism's social and non-social symptom domains on the other need not be binary: social and non-social symptoms may be partially dependent, synergising and reinforcing each other to some extent, but varying independently enough to give rise to a multitude of individual phenotypes. Thus independence of social and non-social symptom domains may be strongest not within but beyond the autism spectrum, in individuals with the Broader Autism Phenotype (Piven, 1999) and in the subtle levels of autistic

traits that occur throughout the general population, and – to apply Baron-Cohen's terms – may be stronger in "female brain" individuals rather than the "male brain" which, according to Baron-Cohen, autism more closely approaches (Baron-Cohen et al., 2004). Evidence regarding E-S co-variance in normative samples is in this way at least as important as clinical sample data in assessing the veracity of the Kanner hypothesis.

It is important to note that the use of an E-S theory framework for interpreting normative data is not meant to imply that E-S theory, its constructs, or its predictions are undisputed. Rather, E-S theory seems the most fully articulated example of Kanner co-variance assumptions, within and beyond the spectrum, in contemporary autism theory and research. The present review is not the first of its kind to recognise the utility of an E-S framework in this context (Mandy & Skuse, 2008). The use of E-S terminology nonetheless necessitates two key qualifications. First, the terms 'male brain' and 'female brain' are perhaps unfortunate in that a complete identification of brain type with sex and/or gender can mislead; the initial elaboration of E-S theory came with the caveat that many females would be classified as having male-type brains, and many males as having female-type brains (Baron-Cohen et al., 2004). Second, in terms of the idea of empathising 'deficits' in autism, the term 'empathising' in this case refers specifically to *cognitive* empathy, rather than *affective* empathy; the difference between the two is the difference between explicit perspective taking (Baron-Cohen, 1995) and emotional contagion.

The Present Review

The shift toward biological approaches in autism research makes it more important than ever to recognise that even the most cutting-edge genetic and neurological approaches are only as useful as the behavioural models guiding their interpretation. Those doubting the relevance of the Kanner hypothesis and behavioural model accuracy to high-tech biological approaches need only consider the diagnostic criteria changes set in place by the DSM-5. In distilling social and communicative domains into a single social-communicative factor, the DSM-5 criteria contain implicit assumptions about relationships between social and communicative domains, and distinctions between them and restricted and repetitive interests/interests; these assumptions, in turn, will determine whether or not social/non-social overlap in neural network activation and heritability estimates will be interpreted modelled factors or as statistical noise. Indeed, these diagnostic revisions necessitate a review of the body of evidence regarding ASC trait structure, and ensure that resources would not be misspent on applying biological approaches to outdated models of how autistic behaviour, cognition, and social functioning arise.

To this end, we offer the present review, the aims of which are threefold. First, we review the body of behavioural evidence relevant to the Kanner hypothesis, including studies using clinical and normative samples, and correlational and factor analytic methods. Second, we synthesise this evidence into an up-to-date, integrative, dynamic picture of autistic development, contextualised within an interactive specialisation model of neurodevelopment. In short, the proposed model posits that social and non-social ASC traits are not initially two sides of the same coin; they *become* two sides of the same coin across development, as their repeated

interactions in behaviour give rise to stable neurological networks that, in turn, further encourage their behavioural interaction. Last, we demonstrate the novelty and explanatory power of the proposed model, first by extrapolating to an in-depth, novel account of autistic behavioural stereotypies; and then in a more brief and speculative fashion, with potential developmental accounts of other traits – such as musical savantism and communicative deficits – that have tended in the past to encourage single-cause accounts of autism.

Relevant Population Characteristics and Evidence for Assessing the Validity of the Kanner Hypothesis

The normal distribution of autistic traits throughout the clinical and non-clinical population (Baron-Cohen & Hammer, 1997b) necessitates that factor structures and co-variance patterns in clinical, subclinical, and non-clinical samples be considered alongside one another. The developmental nature of autism necessitates inclusion of evidence from across age groups, as social/non-social co-variance patterns present early in development (i.e. close to the prenatal period) may differ greatly from patterns emerging later, after any comorbid social and non-social traits repeatedly interact behaviourally and cognitively.

Measures most directly relevant to assessing the Kanner hypothesis are trait inventories, and cognitive and social psychometrics. Trait inventories include clinically-oriented symptom inventories (e.g., Autism Diagnostic Inventory - Revised, or ADI-R); behavioural inventories applicable to clinical, subclinical, and non-clinical populations (*e.g.*, Social Responsiveness Scale, or SRS); and self-reported assessments of sub-clinical manifestations of autistic

symptoms, in the form of personality traits, behaviours, preferences, and tendencies used mainly with sub-clinical and normative populations [e.g., Autism Spectrum Quotient, or AQ (Baron-Cohen et al., 2001); the Systemising Quotient, or SQ (Baron-Cohen, Richler, Bisarya, Gurunathan, & Wheelwright, 2003); and the Empathising Quotient, or EQ (Baron-Cohen & Wheelwright, 2004)]. Interpretations of ASC trait structure based on the AQ, EQ, and SQ come with the caveat that these measures do not capture all ASC traits, being based on the higher-functioning end of the autism spectrum (e.g., Asperger Syndrome, BAP). The AQ, for instance, contains few high functioning analogues of the sensory abnormalities and motor stereotypies seen in clinical cases (Valla et al., 2010).

Relevant cognitive measures include those roughly categorised along systemising and empathising lines. In the systemising category are assessments of spatial skills and pattern recognition (e.g., Block Design and Matrix Reasoning subtests of the Wechsler Intelligence Scale for Children, or WISC-III) and other measures favouring cognitive inflexibilities and detail-oriented biases, such as disembedding speed on the Embedded Figures Test (EFT; Witkin, 1950), and susceptibility to visual illusions (*e.g.*, Walter, Dassonville, & Bochler, 2008). In the empathising category are assessments of facial mental state/emotion reading (Reading the Mind in the Eyes Test, or RMET; Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001), face recognition (*e.g.*, Benton face recognition test; Benton, Hamsher, Varney, & Spreen, 1983), and ToM (*e.g.*, false belief test; Wimmer & Perner, 1983).

Relevant analytic methods can be roughly separated into two complementary approaches: factor analyses of the underlying trait structure of social and non-social ASC-related traits *within*

individuals, and correlational analyses assessing co-variance of these traits *between* individuals. The latter approach allows social/non-social relationships to be tested more directly than do factor analyses, wherein social/non-social relationships are inferred from factor loadings. Factor analytic methods used in the studies reviewed below can be further divided into exploratory (EFA) and confirmatory (CFA) subtypes. CFA is a top-down, theory driven, model-fitting method that tests the accuracy of imposed factor structures, in terms of their ability to account for individual variation in a given behavioural inventory. EFA, meanwhile, exposes relationships between ASC traits in a data-driven, bottom-up manner which complements correlational studies and CFAs by making no presuppositions about factor structure. Factor analytic studies in the literature use EFA, CFA and, in some cases, EFA validated by CFA using a second, independent sample.

Below we include a table summarising the factor analytic studies reviewed herein. For each, we note the ASC trait domains that were factor-analysed; sample sizes and diagnostic category; age means and ranges; behavioural inventory used; factor rotation method applied during analysis; factor structures suggested by each analysis; the percent of variance explained by these factor structures (if EFA was used); Goodness of Model Fit statistics (if CFA was used); and the inter-factor correlations for the factor structure suggested by each analysis.

[Insert Table 1 About Here]

Review of Evidence For and Against the Kanner Hypothesis

A review of the literature finds significant evidence against the Kanner hypothesis, though the evidence in favour is not insubstantial. Three interrelated themes emerge, which together reconcile these two seemingly opposing sets of evidence for and against the Kanner hypothesis, suggesting a model in which initially independent social and non-social traits interact during development of behaviour and cognition, giving rise to emergent Kanner-like co-variance in behavioural presentation.

The three themes are as such: First, evidence suggests an alternative triad of primary, independent ASC factors – *Social Interaction Deficits*, *Cognitive Inflexibility*, *and Sensory Abnormalities* - which may more accurately reflect the underlying structure of autistic traits than the currently applied diagnostic triad. The proposed alternative better accounts for heterogeneities within two domains of the diagnostic triad – *Communicative Deficits* and *Restricted and Repetitive Behaviours and Interests* – heterogeneities that can produce spurious appearances of Kanner-like social/non-social co-variance in some studies. Second, these three alternative factors may be independently heritable, implying multi-factor causation at the biological level. Third, despite this biologically based independence which may seem to negate the Kanner assumption, Kanner-like social/non-social co-variance in *behavioural* presentation within individuals can arise and amplify during postnatal development. Three additional patterns in the data support this model: increased Kanner-like co-variance appearing with increases in trait extremity in multiple domains; greater Kanner-like co-variance in males compared to females; and continuity in this pattern of increasing behavioural co-variance across non-clinical, subclinical, and clinical populations.

Evidence Against the Kanner Hypothesis

Empirically, the *Communicative Deficits* factor of the diagnostic triad seems to consolidate incorrectly two separable types of deficits: those related to social reciprocation *per se,* and communicative-domain instances of more domain-general cognitive inflexibilities (*e.g.*, verbal stereotypies). Indeed, even inflexibilities in the domain of motor control and praxis may impact communicative measures (Belmonte et al., 2013). Factor analyses of several trait inventories converge on this fractionation between symptoms subsumed under the *Communicative Deficits* category. In a principal components analysis of the ADI-R (Georgiades et al., 2007), delays in language development loaded on a *Social Communication* factor, whereas repetitive and idiosyncratic speech loaded on an *Inflexible Language and Behaviour* factor along with non-communicative, cognitive inflexibility-related behaviours (*e.g.*, circumscribed interests, ritualistic behaviours). Although Tadevosyan-Leyfer et al. (2003) found a more granular factor structure comprising *Spoken Language*, *Social Intent*, *Sensory Aversions*, *Savant Skills*, and *Compulsions* in their EFA of the ADI-R, inter-factor correlations revealed the *Spoken Language* factor was more highly related to both *Social Intent* (0.45) and *Compulsions* (0.33) factors than the other factors were related to each other (Table 1), singling out *Spoken Language* as a partial combination of a social and a domain-general factor, and suggesting that the appearance of Spoken Language as a distinct factor might have more to do with the structure of the test instrument and the clinical diagnostic definition of the syndrome than with the underlying trait structure. Two additional factor analyses of the ADI-R each resulted in two-factor models aligning social reciprocation aspects of *Communication Deficits* with other social deficits, and

cognitive inflexibility aspects with a *Restricted and Repetitive Behaviours and Interests* factor (Snow, Lecavalier, & Houts, 2009; Frazier, Youngstrom, Kubu, Sinclair, & Rezai, 2008). These analyses were notable in terms of their large sample sizes (N = 1861, N = 1170, respectively).

Even though a factor analysis of the CARS revealed a distinct *Social Communication* factor, as was the case with the aforementioned ADI-R analysis this factor was highly correlated with both *Social Interaction* (0.529) and the non-social *Stereotypies and Sensory Abnormalities* (0.415) factors (Magyer & Pandolfi, 2009). Similarly, among Child Asperger Syndrome Test (CAST) diagnostic triad subscores, *Communication Deficits* correlated more highly with *Social Interaction Deficits* and with *Restricted and Repetitive Behaviours and Interests* (0.39, 0.42, respectively, in males; 0.26, 0.34 in females) both, than *Social Interaction Deficits* were correlated with *Restricted and Repetitive Behaviours and Interests* (0.27 in males, 0.17 in females) (Ronald et al., 2006b). Such correlational differences would be expected from the mixture of social-communicative and cognitive-inflexibility symptoms lumped into the *Communication Deficits* factor of the current diagnostic triad.

In two independent factor analyses of items comprising the *Restricted and Repetitive Behaviours and Interests* subscale of the ADI-R, an *Insistence on Sameness* component was significantly correlated with the *Communication Deficits* domain subscore of the ADI-R, but not the *Social Interaction Deficits* domain subscore (Szatmari et al., 2006; Lam et al., 2008). Another ADI-R analysis (Van Lang et al., 2006; cross-culturally validated in Boomsma et al., 2008) found that a triad of *Impaired Social Communication*, *Stereotyped Features in Behaviour and Communication*, and *Impaired Play Skills* outperformed both the DSM-IV triad and a single-

factor model. Although the third factor of our hypothesised triad is *Sensory Abnormalities* where in this investigation it was *Impaired Play Skills* (e.g., difficulties with social play and forming peer relationships), the *Communicative Deficits* factor in both cases is split between social reciprocity difficulties and cognitive inflexibility.

A retrospective-versus-current presentation longitudinal analysis of child ADI-R scores found that both verbal and non-verbal repetitive behaviours fractionated into two distinct factors depending on level of complexity: stereotyped utterances and echolalia loaded on a *Stereotypies* factor along with motor stereotypies, whereas verbal rituals and neologisms loaded on a domain-general cognitive inflexibility factor *Anxiety and Compulsions* along with restricted interests. Communicative reciprocity (*i.e.* give-and-take conversational skills), on the other hand, loaded on a *Social-Communication* factor (Kamp-Becker, Ghahreman, Smidt, & Remschmidt, 2009). On ADOS-G inventories of current (6-24y) symptom presentation, similar loadings mirrored the same distinctions between deficits of social reciprocation, and cognitive inflexibility: language ability loaded negatively on an *Interests and Compulsions* factor, and speech abnormalities and stereotyped behaviours loaded on a *Non/Verbal Behaviour* factor, whereas conversational communication skills loaded on a *Social-Communication* factor. This combination of results at different ages indicates some developmental continuity of heterogeneity in *Communicative Deficit* domain symptoms.

Matson and colleagues' (2010) factor analysis of Baby and Infant Screen for Children with Autism Traits (BISCUIT)-Part 1, an inventory for ~1-3y infants and children, reveals an even more telling developmental picture. Although an EFA revealed a three-factor solution

similar to the diagnostic triad, the vast majority of items loaded on *Socialization/Non-verbal Communication* and *Repetitive Behaviour/Restricted Interests* factors, with multiple reciprocation-related communicative behaviours loading on the former factor, and inflexibility-related communicative deficits loading on the latter factor. This fractionation further supports heterogeneity among *Communicative Deficit* behaviours, but also highlights the utility of a developmental lens in understanding how and when a heterogeneous *Communicative Deficit* factor emerges.

Cognitive inflexibilities subsumed under the *Communicative Deficits* diagnostic domain may be fed by deeper roots. Repetitive verbal behaviours (*e.g.,* verbal rituals) and verbal behaviours unrelated to cognitive flexibility (*e.g.,* presence of phrase speech) both are less variable within versus between affected sibships, whereas no such significant sibship effect exists for *Communicative Deficits* domain scores amalgamating flexibility-related and unrelated communicative skills (Silverman et al., 2002). This statistical dissociation has been interpreted as implying that these two types of "communicative" (or at least verbal) symptoms arise from independent genetic origins (Silverman et al., 2002). In a factor analysis of a new symptom inventory assessing autism-relevant endophenotypes, verbal stereotypies (*e.g.,* echolalia) loaded on a *Stereotypic Behaviour* factor along with non-verbal, motor stereotypies (Sacco et al., 2010). The same study's loading of "age at first social smile" on the cognitive inflexibility-related *Stereotypic Behaviour* factor seems, though, to violate a factorisation of social reciprocity versus cognitive flexibility-related communicative deficits; this relationship is, one could argue, the

Kanner hypothesis in its most basic form. This seemingly contradictory finding is re-interpreted in more detail below.

The just-released DSM-5 includes a realignment of social symptoms that closely resembles what has been suggested here. Specifically, the new DSM-5 combines the *Social Interaction Deficits* and *Communicative Deficits* factors of the DSM-IV diagnostic triad into a single *Social/Communicative Deficits* factor. The data reviewed here suggest that this social-communicative combination is a step in the right direction; but also that further improvement could be made by parsing out cognitive inflexibility-related aspects of communicative deficits, and placing them in the *Restricted and Repetitive Behaviours and Interests* factor. This approach is preferable to that of a recent test of the construct validity of the DSM-5 (Mandy, Charman, and Skuse, 2013). Using confirmatory factor analysis this study found the two-factor DSM-5 model to be superior in fit to the three-factor DSM-IV model; the authors improved the fit of the DSM-5 model from 'adequate' to 'good' by removing the 'problematic' (in terms of decreasing model fit) items of verbal stereotypies and imaginative and pretend play.

Assuming factorial continuity between clinical and non-clinical populations, further support for heterogeneity within the *Communicative Deficits* domain comes from studies on typically developing populations using the AQ (Austin, 2005): Of the "Big Five" personality traits (Openness to new experience; Conscientiousness; Extraversion; Agreeableness; and Neuroticism, well-established dimensions of personality), AQ Communication subscores are significantly positively correlated with Neuroticism, and negatively correlated with Extraversion, Openness, Agreeableness, and Conscientiousness. Meanwhile, AQ Social Skills subscores are

related (negatively) only to Extraversion and Agreeableness, and AQ Attention to Detail subscores are related (negatively) only to Openness and Conscientiousness. AQ Communication subscores, in other words, describe personality constructs ostensibly related to cognitive (in)flexibility (Neuroticism, Conscientiousness, and Openness) as well as traits related to willingness to engage in social interaction (Extraversion and Agreeableness). AQ Social Skills and Attention to Detail subscores, meanwhile, do not overlap in their Big Five trait loadings, yet both overlap with AQ Communication subscores (Austin, 2005). Even though this same study identified a three-factor model of the AQ with a Communication subscale distinct from Social Skills and Details and Patterns (as the diagnostic triad would predict), a pattern like that of the five-factor model arose when these three subscales were correlated against Big Five traits.

A replication of the same three-factor extraction revealed that whilst items loading on the first component came from the AQ Social Skills subscale, and items loading on the second component from the Details and Patterns subscale, the third component included item loadings from both the AQ Communication subscale and the AQ Social Skills and Details and Patterns subscales (Hurst et al., 2007). These patterns together support the idea that the *Communicative Deficits* symptom domain is an amalgam of social and non-social traits of distinct cognitive (and, perhaps, biological) origins. Finally, in each of the AQ-based factor analytic studies in which inter-factor correlation patterns were provided (Kloosterman et al., 2011; Stewart & Austin, 2009; Austin, 2009; Table 1), there were significant correlations between social interaction-related factors and communication factors, and between communication factors and inflexibility factors, but no such relationships between social interaction and inflexibility factors.

Evidence also supports the idea that the *Restricted and Repetitive Behaviours and Interests* factor of the diagnostic triad, like the *Communication Deficits* factor, conflates traits rooted in *Cognitive Inflexibility* (*e.g.*, restricted interests) and those rooted in lower-level *Sensory Abnormalities* (*e.g.*, auditory, tactile and visual hypersensitivities). Aforementioned ADI-R-based factor analyses focusing specifically on *Restricted and Repetitive Behaviours and Interests* diagnostic items all resulted in fairly clean splits between traits and behaviours concerning cognitive inflexibility, and those concerning lower-level sensory issues (Szatmari et al., 2006; Papageorgiou et al., 2008; Lam, Bodfish, & Piven, 2008).

Two additional ADI-R factor analyses within the *Restricted and Repetitive Behaviours and Interests* element of the diagnostic triad likewise found separable factors of *Repetitive Sensory Motor Actions* and *Resistance to Change* in one case, and *Repetitive Sensorimotor* and *Insistence on Sameness* in the other (Cuccaro et al., 2004, and Richler, Bishop, Kleinke, & Lord, 2007, respectively). In a previously mentioned ADI-R analysis, three components - *Repetitive Motor Behaviours*, *Insistence on Sameness*, and *Circumscribed Interests* - were extracted from items subsumed under the *Restricted and Repetitive Behaviours and Interests* domain (Lam, Bodfish, & Piven, 2008). Even though *Circumscribed Interests* was identified as its own component, individual items pertaining to *Circumscribed Interests* were the only items loading on both *Repetitive Motor Behaviours* and cognitive flexibility-related *Insistence on Sameness* factors. Items loading on *Repetitive Motor Behaviours* and *Insistence on Sameness*, in other words, were distinct in their loadings. And the aforementioned ADI-R factor analysis by Tadevosyan-Leyfer et al. (2003) found distinct factors for inflexibility-based *Savant Skills* and

Sensory Aversions, with a virtually nonexistent inter-factor correlation (Table 1). On the other hand, the *Compulsions* factor which, according to the current hypothesis, should have been be a heterogeneous mixture of inflexibility and sensory abnormalities, constituted an independent factor, with extremely low inter-factor correlations between these three factors. Finally, loadings from a factor analysis of the Childhood Autism Rating Scale (CARS) distinguished between items concerning fear, nervousness, and adaptation to change (i.e. reactions to novel situations reflecting cognitive flexibility) and those regarding sensory and motor issues related to taste, touch, smell, body use, and stereotypies (i.e. *Sensory Abnormalities*) (Magyer & Pandolfi, 2007).

The factor analyses of retrospective ADI-R and current ADOS-G inventories described above (Kamp-Becker et al., 2009) lend longitudinal support to the hypothesised heterogeneity of symptoms subsumed under the *Restricted and Repetitive Behaviours and Interests* domain (insofar as retrospective ASC trait inventories reliably reflect past trait presentation). Retrospective ADI-R items load onto 4 factors: *Social Communication*, *Anxiety and Compulsions*, *Stereotyped Behaviour*, and *Inadequate Behaviours*; current presentation (ADOS-G) items load onto 5 partially coincident factors: *Social Communication*, *Non/Verbal Behaviour*, *Hyperactivity*, *Stereotyped Behaviour*, and *Circumscribed Interests/Compulsions*. Importantly, retrospective ADI-R items concerning circumscribed interests load on the *Anxiety and Compulsions* factor, suggesting that ritualistic behaviours are adaptive attempts at regulating the anxiety-evoking unpredictability in one's environment. A subset of the *Cognitive Inflexibility/Insistence on Sameness* factors in aforementioned studies distinguishes these interests from low-level sensory abnormalities; and the ADOS-G factor *Circumscribed*

Interests/Compulsions maintains this separability from sensory abnormalities at current presentation. In the diagnostic triad, then, just as social reciprocity is distinct from cognitive flexibility within *Communicative Deficits*, sensory abnormalities are distinct from cognitive flexibility within *Restricted and Repetitive Behaviours and Interests*. These social/cognitive and sensory/cognitive distinctions pertain across retrospective (ADI-R) and current (ADOS-G) behavioural inventories.

Implicit in the distinction between *Sensory Abnormalities* and *Cognitive Inflexibilities* is the idea that abnormalities in sensory modulation are a primary, core feature of ASC, rather than secondary and largely peripheral to core ASC traits. As Rogers and Ozonoff (2005) point out, sensory issues have historically received only peripheral attention in explanatory accounts of autism, often subsumed under broader accounts such as executive functioning deficits and WCC. The reconceptualisation of sensory modulation issues as primary to ASC is gaining traction in the literature, however. Indeed, the most thorough meta-analysis to date found that sensory modulation issues consistently distinguish ASC and TD individuals across studies (Ben-Sasson et al., 2009). Sensory issues are largely separable not only from cognitive inflexibility (as argued above), but also from executive functioning (Boyd et al., 2009; Chen et al., 2009), and social interaction deficits (Watson et al., 2011), the latter observable as early as infancy (Baranek, 1999). Moreover, although the affected modalities can vary greatly between ASC individuals (perhaps explaining some of the ambivalence towards including sensory issues as a core symptom) there is some evidence that, within ASC individuals, severity of sensory modulation abnormalities highly correlates across sensory modalities (Kern et al., 2007).

Beyond the implications for specific diagnostic domain heterogeneities and the hypothesised alternative triad, the above analyses indicate more generally that social and non-social ASC traits are somewhat independent within individuals, contrary to the Kanner hypothesis. Additional findings support the more general notion of social/non-social independence. *Social Affect* and *Restricted, Repetitive Behaviours* were the two trait domains indicated by one factor analysis of the ADOS, later replicated in an independent sample (Gotham et al., 2007; 2008). Similar separability was evident as young as 18 months in a general population confirmatory factor analysis using the ADI-R (Beuker et al., 2013). Likewise, correlational evidence from autistic and typically developing child samples suggests social and non-social ASC traits are separable early in development. Pellicano, Maybery, Durkin, and Maley (2006) found false belief performance to be unrelated to EFT disembedding speed, in both their ASC and typically developing samples of 4-7 year olds. Morgan, Maybery, and Durkin (2003), meanwhile, found that weak central coherence, joint attention, and verbal ability are separable earlier in ASC and typical development (3-5 years). This finding is particularly interesting because it has been suggested that joint attention is a crucial early locus for social/non-social ASC trait interactions (Charman, 2003). For instance, joint attention is a skill in which detail-oriented cognitive biases could plausibly lead to social-communicative deficits, by making it more difficult to learn new words from a caregiver.

Social/non-social separability was also the consensus among reviewed factor analyses of the AQ in more typically developing (i.e. sub- and non-clinical) individuals, with different analyses variously revealing: a) a diagnostic triad-like, three-factor model of *Social Deficits,*

Communication Deficits, and (preference for) *Details and Patterns* (Austin, 2005, replicated in Hurst et al., 2007); b) a factor structure mirroring the five subscales originally intended by Baron-Cohen et al. during construction and validation of the AQ (*Social Skills, Communication, Restricted/Repetitive Behaviours, Imagination,* and *Attention to Detail* (Kloosterman, Keefer, Kelley, Summerfeldt, & Parker, 2011; Lau, Kelly, and Peterson, 2013); and c) a four-factor model in-between these two, including *Imagination* but without *Restricted/Repetitive Behaviours* (Stewart & Austin, 2009). Reconciling these discrepancies is a hierarchical factor structure with superordinate factors of *Social Interaction* and *Attention to Detail*, the former comprising four subfactors: *Social Skills, Attention Switching, Communication, and Imagination* (Hoekstra, Bartels, Cath, & Boomsma, 2008). The same hierarchical factor structure was replicated in another analysis (Valla et al., 2010); when analysed separately by sex, the superordinate split between social and non-social traits was particularly strong in males, compared to females. In all these analyses, social factors were distinct from non-social factors.

Similar social/non-social separability has also arisen in other E-S correlations in typically developing individuals. For instance, resistance to visual contextual illusions (framed as an epiphenomenon of autism-like, detail-oriented processing biases) and Systemising Quotient (SQ) scores have been shown to be independent of Empathising Quotient (EQ) scores (Walter, Dassonville, & Bochler, 2008), while RMET scores are in one study of adults unrelated to EFT disembedding speed, performance on a block design test, and SQ score (Carroll & Chiew, 2006). Additional studies of empathising-systemising co-variance (discussed in further detail below) have found sex-dependent patterns wherein social and non-social ASC traits are more

independent either in males compared to females (Voracek & Dressler, 2006), or females compared to males (Valla et al., 2010).

Evidence For the Kanner Hypothesis

Although the body of evidence countering the Kanner hypothesis is substantial, evidence supporting Kanner-like co-variance is not insignificant. Two analyses support a single factor model of ASC traits. Constantino and colleagues (2004) found principal components analyses of SRS and ADI-R inventories of PDD and non-PDD psychiatric cases supported a single underlying component accounting for a large proportion of individual variation in social and non-social ASC traits (35% of variation in SRS scores, 40% in ADI-R scores). Although Szatmari et al. (2006) found a two-factor solution accounting for a majority of the variance (68% in a sample of 5y+ cases, 70% in a sample of 4-6y cases) in ADI-R scores, the first of these factors comprised severity of traits on all three subscales, with the second factor accounting for variance in overall level of functioning. Sample sizes for both studies were, however, notably smaller (N = 226, and N = 129, respectively) than most ADI-R factor analyses in the literature.

In some cases, Kanner-like co-variance has arisen alongside evidence against the Kanner hypothesis. In one of the ADI-R factor analyses aforementioned, two of three components extracted from *Restricted and Repetitive Behaviours and Interests - Repetitive Motor Behaviours* and *Insistence on Sameness* - correlated significantly and positively with social interaction deficits (Lam, Bodfish, & Piven, 2008). A more recent factor analysis of ADI-R scores of a large sample of young children with ASD was posed in support of the two-factor DSM-5 model

distinguishing *Social Communication Deficits* and *Fixed Interests and Repetitive Behaviors*, despite the fact that inventory items of nodding and head shaking both loaded heavily on the former factor (Georgiades et al., 2013). Neither behaviour is ostensibly social, both are repetitive behaviours, yet the two-factor model was chosen based on the circular logic of its relative interpretability and agreement with previous findings in the literature. In the aforementioned endophenotypic analysis (Sacco et al., 2010), lower-level sensory abnormalities (e.g., decreased pain sensitivity) loaded primarily on the first of four factors (*Circadian and Sensory Dysfunction*) while more cognitively based repetitive behaviours and circumscribed interests (e.g., verbal stereotypies) loaded on the fourth factor (*Stereotypic Behaviour*). This separability of high- and low-level repetitive behaviours reinforces the distinction between *Cognitive Inflexibility* and *Sensory Abnormalities* in the hypothesised alternative triad. There is also decreased variability within versus between monozygotic twin sibships in social and communicative deficits, and restricted interests, but not sensory issues (Kolevzon et al., 2004).

Although the absence of detectable heritable co-variation does not support sensory symptoms' representing a primary factor in ASC, it does support the separability of *Sensory Abnormalities* from *Cognitive Inflexibility*. In the twin studies that teased apart genetic and phenotypic overlap of the diagnostic triad, although each of the triad domains was highly, individually heritable, phenotypic overlap between social and non-social domains was moderate but certainly greater than zero (Ronald et al., 2006a). (From the discussion of an alternative triad it could be argued that phenotypic overlap reflects the heterogeneity within diagnostic domains that the alternative triad is meant to correct; however, overlap existed even between *Social*

Interaction and *Restricted and Repetitive Behaviours and Interests*, which have no symptoms in common even in the alternative triad.) Moreover, such phenotypic overlap increased as severity of social and non-social symptoms increased (Ronald et al., 2006b) – meaning that even if slight degrees of autistic traits in the typical population may be largely independent, more pronounced degrees of the same traits begin to reinforce each other across symptom domains, culminating in the apparent syndrome of autism.

Longitudinal studies support this notion of developmental reinforcement of symptoms across domains: although severity in diagnostic triad domains at 2 years of age does not predict severity at 7 years, severity of social and communicative deficits at 3 years predicts all three diagnostic domains at 7 years (Charman et al., 2005). Additional behavioural evidence for early emergence of social/non-social autistic trait co-variance includes replicated findings that RMET scores and visual perceptual disembedding speed on the EFT are inversely related in 4-5 year old children (Jarrold et al., 2000; Pellicano, Maybery, & Durkin, 2005). The former study found this co-variance pattern in children (9 years) with ASC diagnoses as well, documenting some spectral continuity in such social/non-social co-variance. Later in development, poor ToM skills (measured via first and second order false belief tasks) are associated with both decreased susceptibility to visual illusions (in which detail-oriented processing gives one the advantage of ignoring visual contexts giving rise to such illusions) and increased scores on the Wechsler Block Design subtest, in a Kanner-like fashion, in typically developing adolescents and adults with subclinical autistic traits (Best et al., 2008).

Similar Kanner-like social/non-social co-variance patterns in the normal adult population has arisen between disembedding speed on the EFT and social deficit items of the AQ (Russell-Smith, Maybery, Bayliss, & Sng, 2012); this result contrasts with the lack of Kanner-like relationships in other measures (e.g., Carroll and Chiew, 2006). A similar association between disembedding speed and social skills arose in an earlier study, but in this case the social measure was performance-related (an early version of the RMET) (Jarrold et al., 2000). When re-analysed separately by sex, this Kanner-like co-variance remains in women but not men – a heterogeneity that might explain some of the conflicting results in mixed-sex samples. At the same time, specific patterns of sex-dependent co-variance vary from one study's set of measures to another's, with some finding co-variance in females but not males (Jarrold et al., 2000), others males but not females (Valla et al., 2010). In this latter study, men in systemising-related undergraduate major fields were characterised more by empathising deficits (measured via the RMET, the Benton Face Recognition Test, and the Social Interaction hierarchical subscale of the AQ) than by systemising skills (measured via the EFT and the AQ Details/Patterns subscale), whereas no such link between systemising field and face reading difficulties was present in females (Valla et al., 2010). Heterogeneity in sex-dependent co-variance findings is not restricted to the question of which sex exhibits the co-variance, either; it also seems to depend on the specific measures whose co-variation is being assayed. When co-variance is assayed from RMET scores and Systemising Quotient scores, males but not females exhibit not inverse (Kanner-like) but actually *positive* empathising-systemising co-variance, with RMET scores predicting systemising tendencies (Voracek & Dressler, 2006). Of these various sex-dependent outcomes, the pattern in

Valla et al. (2010) is unique in that an independent study provides developmental support for it: Kanner-like co-variance has been found in school-age boys, but not girls (Skuse et al., 2009), supporting the "extreme male brain" notion that males are closer than females to the autism spectrum (Baron-Cohen, 2002).

Reconciling the Evidence: A Developmental Dynamic Interaction Model

The mutual exclusivity between social/non-social ASC trait independence and Kanner-like co-variance, an assumption implicit in many framings of the debate, downplays and overlooks the role of development in the progression of symptoms of autism – which is, after all, a *developmental* disorder. In so doing, the assumption overlooks a potential framework for reconciling what appear to be opposing sets of evidence. Independently heritable social and non-social ASC traits may *develop* into Kanner-like patterns of co-variance through repeated, dynamic behavioural interactions, eventually synergising into the quintessentially autistic collection of characteristics that Kanner described.

It is in this developmental construction that non-social capacities can be drafted into the service of social cognitive ends, in the way that a positive correlation between figure disembedding ability and facial emotion reading has been interpreted as males applying systemising skills to empathising problems (Valla et al., 2010). (For experimental evidence of such piecemeal face processing, in ASC individuals, see Evers, Noens, Steyaert, and Wagemans, 2011). Explicit instruction of people with Asperger syndrome in solving empathising problems

using systemising skills has been described as "systemising empathy" (Golan & Baron-Cohen, 2006).

We argue that this phenomenon – the application of systemising skills within cognitive domains that are more typically, more commonly or more efficiently approached via empathising skills – need not be the subject of explicit instruction or explicit cognitive strategy, and is not restricted to people with autism-spectrum conditions. We argue, rather, that it happens during typical cognitive development as systemising and empathising skills supplement – and in some instances supplant – each other during interaction with environmental task demands. Indeed, reconsidering the reviewed evidence through this developmental lens helps clear up some seeming contradictions amongst findings within and between studies. A prime example as to how the hypothesised developmental model would manifest empirically can be found in a series of related studies on a single large twin sample (Ronald et al., 2006a,b; Dworzynski et al., 2007). First, aforementioned findings demonstrate the non-mutual-exclusivity of the two sides of the Kanner hypothesis debate, supporting Kanner-unlike, independent heritabilities of social and non-social ASC traits, but also Kanner-like, moderate correlations between domains of behavioural presentation (Ronald et al., 2006ab). Correlations between diagnostic domains of *Communicative Deficits* and both *Social Interaction Deficits* and *Restricted and Repetitive Behaviours and Interests* can be ascribed partly to the heterogeneity in the communication domain, argued above. Such *intra*-domain heterogeneity within the *Communicative Deficits* domain would give the appearance of deeper *inter*-domain links than the hypothesised possibility: that social interaction deficits are incorrectly paired with cognitive and motor

flexibility-related linguistic deficits affecting communication. Heterogeneity *within* domains cannot account for correlations *between* the distinct diagnostic domains *Social Interaction Deficits* and *Restricted and Repetitive Behaviours*, however.

The same data from this large twin sample were also reanalysed separately within subgroups of individuals who presented with extremity (top 5% cutoff) in one, two, and three symptom domains. Behavioural overlaps between symptom domains (i.e. intra-domain correlations of variation in symptom severity) were significantly greater within the group of individuals exhibiting severity in all three domains, compared to groups of individuals exhibiting top 5% severity in zero or one domain (Ronald et al., 2006b). Similar increased overlaps related to extremity in multiple domains have been found in other studies (*e.g.*, Skuse et al., 2009; Frazier et al., 2008), including a recent study comparing ASC individuals', first-degree relatives', and typically developing individuals' correlations between AQ-based indices of systemising and empathising (Grove et al., 2013). According to Baron-Cohen's "extreme male brain" notion this heightened correlation at the extreme should be doubly true of males, who are closer to the autism spectrum; and indeed, the same study found greater inter-domain behavioural overlap in males than females. Greater Kanner-like co-variance in males compared to females is a common albeit not a universal result in studies of autistic traits (Valla et al., 2010). The hypothesised developmental model may also support the conjecture that a positive correlation in males between figure disembedding and facial emotion labelling (EFT and RMET, respectively) reflects a predominantly male cognitive strategy of applying systemising skills to empathising problems (Valla et al., 2010).

The present model of developmental interaction between symptom domains is consistent with longitudinal observations of the aforementioned twin samples (Ronald et al. 2006ab), measured by the MacArthur Communicative Development Inventories at 2, 3, and 4 years, and the CAST at 8 years. Early language deficits and later *Social Interaction* and *Communication Deficits* were related primarily genetically, rather than phenotypically. Although early language deficits were *genetically* unrelated to later *Restricted and Repetitive Behaviours and Interests*, *Communication Deficits* and *Restricted and Repetitive Behaviours and Interests* domains were phenotypically related later in development. A combination of independent heritability and phenotypic dependence arising in concert as they did in these twin samples supports developmentally arising Kanner-like co-variance (even after taking into consideration the shared cognitive inflexibility component), with later *Communicative Deficits*, representing a developmentally emergent interaction between cognitive inflexibility and linguistic elements of social behaviour.

This model of *Communicative Deficits* emerging developmentally as primary social reciprocity- and motor and cognitive inflexibility-related factors interact is also reflected in the aforementioned BISCUIT-1 factor analysis (Matson et al., 2010). In the earlier developmental stage represented by this study's sample, the *Communicative Deficit* factor seems to have begun emerging, carrying a small number of inventory items, while many reciprocation-related communicative behaviours load on a *Socialization/Non-verbal Communication* factor, and verbal stereotypies load with cognitive inflexibilities and sensory issues on a *Repetitive Behaviours/Restricted Interests* factor. This study also highlights the idea, distinguishing the

current model from previous models, that a secondary deficit, though it emerges from more primary behavioural interactions, can take on a 'life of its own', its presentation becoming independent of the primary factors from which it emerges.

The proposed developmental model helps explain other seeming empirical contradictions. The longitudinal finding that communicative deficits are not predictive of any symptom domain at 2 years, but significantly predict all three diagnostic domains later in childhood (Charman et al., 2005) would be expected if inter-domain behavioural interactions increase with development. At 4-5 years verbal rituals load on the same factor as repetitive use of objects and other manifestations of cognitive inflexibility (Kamp-Becker et al., 2009). By 6-7 years, a communication-related factor emerges encompassing stereotyped phrases, eye contact, and facial expressions. The hypothesised model would explain that whereas social deficits may be largely independent of cognitive inflexibility early in development, behavioural and cognitive interactions over time produce repetitive phrases - part linguistic inflexibility, part social communicative deficit.

In another apparent contradiction, the loading of "age of first social smile" on a *Stereotypic Behaviour* factor (Sacco et al., 2010) may seem to suggest early links between *Social Interaction Deficits* and *Restricted and Repetitive Behaviours and Interests* diagnostic domains, as in single-cause accounts of autism (*e.g.*, Constantino et al., 2004). In the context of the hypothesised alternative triad and developmental model, however, one might posit that "age of first social smile" is an early example of behavioural interactions between *Social-Communicative Deficits* and stereotypy-related *Cognitive Inflexibility* factors. In case cognitive flexibility may

seem behaviourally unrelated to social smiling, consider the rapid cognitive control in play during a smiling response to a social overture, or during a decision to share an emotional response with a social partner at the same time as one is busy experiencing that response.

Applying the Proposed Developmental Model: A Novel Account of Repetitious Behaviours

In the proposed developmental model, primary, initially independent *Social Interaction Deficits, Cognitive Inflexibility,* and *Sensory Abnormalities* behaviourally interact and become related. Here we demonstrate the utility of this model by explaining the non-primary autistic trait of repetitive behaviours in terms of interactions between the latter two primary traits. To be clear, this is not meant to imply that these two primary traits interact more or less than any other pairing; to highlight this point, we follow our discussion of repetitive behaviours with a brief account of how communicative deficits would arise from interactions between *Social Interaction Deficits* and *Cognitive Inflexibility.*

In the current diagnostic triad, repetitive behaviours are considered primary symptoms, and are subsumed under the domain of *Restricted and Repetitive Behaviours and Interests*; sensory abnormalities, meanwhile, are treated as secondary symptoms. On face, repetitive behaviours are more easily categorised and accounted for using the diagnostic triad than the alternative triad, as such behaviours do not fit as clearly under any of the alternative triad's domains. Viewing the alternative triad through the lens of the proposed developmental model, however, such behavioural stereotypies can be explained as external, motor manifestations of developmentally snowballing interactions between independent primary traits of *Cognitive*

Inflexibility, and *Sensory Abnormalities* such as hypersensitivity to auditory, visual, or tactile stimuli. This sensory-cognitive interaction would explain why Chen et al. (2009) found that sensory hypersensitivities and cognitive inflexibility each predict the presence of repetitive behaviours, yet are unrelated to each other. Repetitious movements may be externalised efforts to impart predictability on a world rendered chaotic and intractable by an abnormal, cognitively inflexible, perceptually overstimulated mental environment (Belmonte, 2008). Whilst this point remains a speculative one, relationships between ritualistic behaviours and perceived uncertainty or fearfulness do exist in young, typically developing children (Evans et al., 1999).

A crucial distinction, demonstrating the advantages of the present developmental interactionist account, is that as repetitive behaviours are continually used to compensate for cognitive-sensory interaction chaos, the more likely it becomes that neural networks hard-coding such interactions will arise. Once encoded in dedicated neural networks, these behaviours could then take on a 'life of their own', arising under conditions that may not have triggered them prior to developmental snowballing. Overall level of functioning, then, may determine the executive control capacity available to inhibit externalising, overtly motor behaviours. Every individual would have some threshold beyond which cognitive-perceptual load could not be pushed without evoking externalising behaviours. High-functioning people, with high thresholds, when confronted with sensory perceptual challenges would manifest mostly internalised responses that impose conceptual 'sameness,' whereas in low-functioning individuals this drive to render perceptual organisation tractable might more often spill over into externalised, motor stereotypies that directly, mechanically impose sensory sameness. Recently, Johnson (2012)

offered a similar argument in an attempt to explain relationships between executive functioning deficits and conditions such as autism and attention deficit hyperactivity disorder. In short, Johnson argues that affected individuals with the executive capacity to compensate for their cognitive abnormalities early in development avoid the developmental snowballing that leads to clinical severity and, eventually, diagnosis.

Multiple findings support this characterisation of repetitive behaviours as emergent from an interaction of more primary sensory and cognitive symptoms, with overall level of functioning determining the internal or external manifestation of such interactions. Positive relationships between IQ and age, and restricted interests, have arisen in some cases, as well as negative relationships between IQ and age, and sensorimotor stereotypies (Szatmari et al., 2006; Papageorgiou et al., 2008). (IQ controlled for age, and age, within individuals, are taken here as correlates of overall functioning). Likewise, factor analyses of retrospective (childhood) ADI-R items in one of the aforementioned longitudinal studies (Kamp-Becker et al., 2009) reveal an *Anxiety and Compulsions* factor; if internalised anxieties arise from cognitive inflexibilities faced with overstimulating novel situations, compulsive behaviours would be their external analogues. The event-related timing of dorsolateral prefrontal cortical activation in a non-social visual attention task in boys with autism spectrum conditions correlates with ADI-R social and communicative subscores but not with the ADI-R repetitive behaviours subscore (Belmonte et al., 2010), as might be expected if repetitive behaviours were an ætiologically secondary symptom.

On the ADOS-G measure of current symptom presentation in Kamp-Becker et al.'s study, items related to anxiety loaded negatively on the ADOS-G factor *Hyperactivity*, meaning that decreased hyperactivity was related to increased anxiety. Such a relationship would be expected if internalised manifestations of cognitive inflexibility and hypersensitivity to context novelty (e.g., anxiety in novel situations) increase as externalised manifestations (e.g., hyperactivity) decrease (Kamp-Becker et al., 2009). The same investigation also found that full-scale IQ was at once unrelated to the *Social Communication* factor on both retrospective (childhood) ADI-R and current ADOS-G inventories, and significantly, negatively correlated with behavioural stereotypies at both time points. This ability-repetitive behaviours link was precisely the conclusion of another ADI-R-based study with young children (Honey et al., 2008). More tellingly, Bishop et al. (2006), using a particularly large sample (830) of young children with ASC, found that whilst nonverbal IQ was negatively related to low-level, sensory-related repetitive behaviors, it was positively related to the circumscribed interest subset of ADI-R RRBI items. Thus high IQ might help flip the switch, as it were, from externalised, behavioural manifestations of inflexibility to internalised, cognitive manifestations.

In this respect, some differences between ADI-R analyses may be complementary rather than contradictory. Where one analysis (Szatmari et al., 2008) found evidence for influence of higher cognitive functioning (as indicated by IQ and age) on ASC symptoms, in the form of a cognitively based need for sameness, others found evidence for the symptomatic manifestation of lower cognitive functioning in ASC, in the form of ritualistic behaviours (Lam et al., 2008; Kamp-Becker et al., 2009); one of the latter found this relationship both in early development

and later presentation (Kamp-Becker et al., 2009). When extracted factors from the *Restricted and Repetitive Behaviours and Interests* diagnostic domain were correlated with age, Vineland Adaptive Behaviour Scale scores (VABS), and ADI-R domain scores, *Insistence on Sameness* presentation (current and ever) was significantly, positively correlated with the ADI-R communication domain, and *Repetitive Sensory and Motor Behaviours and Interests* also were highly negatively correlated with adaptive functioning and IQ, measured via the VABS and Leiter IQ, respectively. The former result provides additional support for the proposed alternative triad in which communicative inflexibilities are grouped with the high-level repetitive behaviour of circumscribed interests, as in Georgiades et al. (2007). The latter provides more concrete support for the idea that overall level of functioning (in this case indicated by IQ) may determine, or may correlate with, the threshold between internalised, cognitive drives, and low-level repetitive behaviour of externalised, stereotypic, motor drives.

Of course, these are inferences about divergent developmental trajectories based on cross-sectional data; even the longitudinal analysis (Kamp-Becker et al., 2009) relied on retrospective ADI-R inventories for inferences about early developmental patterns. There is some more direct developmental support for this hypothesis, though; a within-subjects, repeated measures, longitudinal analysis comparing high- and low IQ (median split) groups from age 4 through age 19, with an intermediate data collection at age 13, using the ADI-R (with an added emotional responsiveness portion), ADOS, and the Vineland Adaptive Behaviour scale (VABS), as well as the Stanford-Binet IQ test (McGovern & Sigman, 2005), found that both IQ groups significantly improved in *Social Interaction Deficits* and *Restricted and Repetitive Behaviours*

and Interests domains with age, but that the high IQ group improved significantly more than the low IQ group in *Restricted and Repetitive Behaviours and Interests*, and not in *Social Interaction Deficits*. Thus, across development higher functioning carries advantages specific to reducing repetitive behaviours.

The novel account of repetitive behaviours offered here reconciles the multiple hypotheses of the origins and functioning of such behaviours offered by Turner (1999). In imparting predictability on cognitive/perceptual chaos arising at the junction of *weak central coherence*-related cognitive inflexibility, and sensory hypersensitivity, such behaviours serve as a *homeostatic mechanism* at the sensory perceptual level. If the over-arousing stimulus is another person whose movements/touch/etc. cannot be predicted, these behaviours avoid reliance on the *impaired mentalizing* that would otherwise render the other person's actions predictable. Once such behaviours take on a life of their own in interactively specialised neural networks, they become *operant behaviour*, present even in the absence of what originally brought them on. Such neural networks would also make it more difficult to inhibit these behaviours, or to extinguish them via generation of novel behaviour, both of which symptoms appear as aspects of *executive functioning* deficits.

Meanwhile, inconsistencies in relating level of functioning to internalised and externalised manifestations of 'sameness' are reconciled by the notion that overall level of functioning establishes a maximum cognitive capacity for integrative processing and abstraction, determining whether sensory inputs are internalised or externalised. As Turner notes, high-functioning people with ASC can exhibit 'low-level' motor repetition, and low-functioning

people with ASC can exhibit an internalised insistence on sameness. Such apparent inconsistencies can be accounted for by the present threshold model of overall functioning: sensory inputs hyper-arousing enough to surpass high functioning individuals' greater threshold could produce externalised motor compensation, whilst some inputs beneath even the lower threshold of low functioning individuals could produce an internalised compensatory need for sameness. These thresholds also could be expected to vary with cognitive-affective state from day to day and hour to hour.

The hypothesised relationship between cognitive inflexibilities and compensatory repetitive behaviours is well summarised in an interpretation of sibling correlations of ASC symptom severity (Spiker, Lotspeich, Dimiceli, Myers, & Risch, 2002): "while all the individuals with autism had some ritualistic or repetitive behaviours or preoccupations, with increasing cognitive ability these tend to be more symbolic and less motoric." Just as typical cognitive development elaborates iconic sensory and immediate motor capacities into symbolic concepts and sequential, goal-directed plans, atypical cognitive development elaborates a need for sameness in these concrete domains into a need for scripting in domains that typically are more abstract. Social psychology in typical individuals has demonstrated that the low "level of construal" of relatively immediate, unprocessed, unelaborated, detail-oriented, iconic perception evokes a similarly low "psychological distance" from one's own immediate perspective on objects, events, contexts, and social partners (Trope & Liberman, 2010), giving rise to a cognitive style that emphasises peripersonal rather than distant spaces, current rather than past or

future events, absolute rather than conditional plans, and egocentric rather than allocentric social perspectives – and *vice versa,* low psychological distance evokes low level of construal.

Such effects arise during short-term, situational manipulations; analogous long-term, developmental influences might be expected to be even more potent. As such, it is not difficult to conceive of how *Cognitive Inflexibilities* in higher-functioning ASC individuals might interact with, and developmentally adapt to, a basic *Sensory Abnormality* such as auditory hypersensitivity in such a way that narrow specialities such as musical savantism may emerge in the factor space between these two more primary, independently inherited traits. Or, for a less exceptional example, the *Communicative Deficits* domain of the diagnostic triad may be better understood as deficits emerging developmentally from the factor space between primary *Social Interaction Deficits* and *Cognitive Inflexibility* domains of the alternative triad: whilst the former domain impedes development of the back-and-forth conversational reciprocity, the latter domain impedes the linguistic and conceptual fluidity and flexibility needed to adapt as conversations unfold and progress in unpredictable, unscripted ways.

The Bigger Picture

The advantage of a developmental perspective may seem obvious; behavioural theorists know well that developmental models are essential for teasing apart nature and nurture. A previous review addressing the Kanner hypothesis, for instance, argues that the diagnostic triad is fractionable in terms of causal origins, with a concluding caveat that ASC traits do in any case interact developmentally (Happé & Ronald, 2008). The developmental model posed here is

more than an elaboration of such developmental caveats, though, offering an explanation for Kanner co-variance but also suggesting how secondary, compensatory phenomena such as repetitive behaviours can become stable aspects of autistic behaviour, their expression dissociated from their original coping purpose.

Specifically, the dynamic, developmentally snowballing interactionism posited here is best described within the model of "interactive specialisation" (Johnson, Halit, Grice, & Karmiloff-Smith, 2002). A typical developmental perspective would take the view that independently heritable social-communicative deficits and cognitive inflexibilities both are programmed into neural architecture and begin to co-vary as they are expressed through shared behavioural and cognitive outlets. An interactive specialisation view, on the other hand, would hold that as these traits are integrated in co-expression repeatedly across development, their integration would come to be reflected in neural networks. These altered networks, in turn, become co-conspirators in this process, encouraging further integration by serving as a path of less and less resistance.

If development occurred according to the former model, that of trait interaction without interactive specialisation, it would not be as clear how social and cognitive integration would become so deep that observations like Kanner's depict restricted interests as a concomitant of social dysfunction. Within the interactive specialisation view, however, a bio-behavioural mechanism producing social aversion may also induce an equal and opposite reaction of object preoccupation. A child with ASC may spend early years being repeatedly bombarded with the unsolicited advances of what appear to be large objects with irreproducible, inexplicable cause-

and-effect. Meanwhile, the more physically salient details of the inanimate world capture their attention: a spinning ceiling fan can be a more visually salient motion than a moving mouth (i.e. it covers more physical space, at a greater speed, and in a more predictable fashion), a car horn a more intense sound than a voice. Physiologically, autism may begin with pathologically low-entropy local neural networks; these aberrant local representations then prevent the normal activity-dependent development of long-range connections subserving brain and cognitive integration (Belmonte et al., 2004ab) via a bio-behavioural feedback loop. As the child develops, the child repeatedly retreats from the unpredictability of people to the predictability of the inanimate world, and their attention is captured by small parts of this inanimate world. As with all behavioural skills and strategies, practice makes perfect: repetition of these learnt coping behaviours can allow underused, potentially integrative networks to atrophy and local processing to hypertrophy, specialising the brain for a detail-over-context, iconic-over-symbolic, egocentric-over-allocentric mode of perception and cognition. This hypothesised developmental model, and interactive specialisation more generally, seem more equipped to explain autism as a *developmental* disorder, and to account for the Kanner-like co-variance observed to arise between initially independent traits over the course of development. This paradigm may aid behavioural scientists appealing for genetic links to behavioural outcomes, as well as cognitive neuroscientists interested in tying the proposed model to neural mechanisms.

Future Directions

The most important theoretical imperative of this review is a recognition that debates over single versus multiple causes of autism mean different things at the genetic, neurological

and behavioural levels, and that a dynamic developmental lens is essential to understanding autism across these three levels of analysis. If, for instance, a single genetic effect leads to low entropy local networks (Rubenstein & Merzenich, 2003), a single 'cause' could look multi-factorial from the outset at the neurological level, each disturbed inter-regional connection an additional neurological 'cause', with the behavioural result being initially independent effects on social and non-social behaviour. These behavioural effects would then interact developmentally according to the proposed model, leading to networks interactively specialised for the collection of behavioural traits we call autism, with social and non-social behaviours, and their neurological underpinnings, being somewhat connected, somewhat independent, and highly individualised. In a different scenario, if social and non-social ASC traits were to begin entirely independently at both genetic and neural levels (e.g. Smith & al., 2011), interactive specialisation could result in their being seemingly related behaviourally and neurologically downstream.

The wide age ranges in the reviewed studies – varying as widely as 2-46 years, frequently with maximum ages greater than 20 years, and mean ages greater than 8 years – present a problem of interpretation. For the present synthesis, inferences about developmental trajectories of social/non-social co-variance could be made only from three sources: the few longitudinal investigations that currently exist (e.g., Kamp-Becker et al., 2009); cross-sectional investigations repeating their analyses separately for younger and older sub-sections of the total sample (e.g., Szatmari et al., 2002); and meta-analytic comparisons of studies at early ages (e.g., Matson et al., 2010) to other, less restricted samples. It is only through the groundwork laid by such studies that the full importance of a developmental picture can be seen; with wide age ranges come large

sample sizes (e.g., Frazier et al., 2008). We also sympathise with the fact that every subject counts when it comes to such analyses. As such, rather than excluding older cases, we encourage investigators factor-analysing ASC traits to include developmental elements. Entering age as a co-variate in regression models, or segmenting samples cross-sectionally into age groups for separate factor analyses are two straightforward ways to add a developmental element. In the latter case, cross-sectional cuts between early ages would ideally be as thin as statistical power would allow, to capture as much early behavioural dynamism as possible.

A thorough test of the model proposed here would involve longitudinally tracking the developmental trajectory of relationships between ASC trait domains from infancy through late childhood. If the aforementioned account of repetitive behaviours is correct, then the occurrence of such behaviours early in development implies that inter-trait interactions rapidly produce secondary traits. Particular emphasis would be placed on potential loci of inter-domain interaction (e.g., joint attention), and aspects of behaviour hypothesised to be emergent manifestations of inter-trait interactions. In tracking repetitive behaviours, for instance, it would be important to note the contexts in which they initially arise (e.g., in response to loud noises), as well as when they began appearing in the absence of the initially associated context; the former would indicate the compensatory, primary trait associations of repetitive behaviours, and the latter when, developmentally, repetitive behaviours become an independently developing trait domain.

The ideal instrument for such a study would be a developmentally-minded behavioural inventory, as extensive as the ADI-R or ADOS-G but, unlike these inventories, allowing easy re-

testing at different ages that would document changes in trait severity and developmental emergence of secondary traits, including the contexts in which the inventoried behaviours arise. Constructing and validating inventories has been a major accomplishment for the field; what is needed now is the capability to apply these inventories longitudinally to tracking age-related changes in autism. To date, the closest that studies have come to this approach are current-versus-ever comparisons of the ADI-R (Kamp-Becker et al., 2009); and a combination of multiple data collection points (2, 3, and 4 years old) using the MacArthur Communicative Development inventory, and the CAST at age 8 (Ronald et al., 2006ab). However, the 'ever' component of the former is susceptible to hindsight biases and recall accuracy; and although the latter tracks early developmental changes in communication using multiple data points on the MacArthur, this inventory only measures communication traits, and changing to the CAST to inventory later childhood behaviours raises issues of inter-instrument consistency.

If the ideal instrument were available, exploratory factor analyses, treating inventory items individually, could then be compared between data collection points. The proposed model would predict that between infancy and early childhood, factor analyses would indicate primary *Sensory Abnormalities*, *Cognitive Inflexibility,* and *Social Interaction Deficits* factors. These would interact, and by late childhood communicative deficits would arise and load on the latter two factors; and secondary repetitive behaviours would emerge and load on the former two factors. By adolescence, Kanner-like behavioural interactions between factors would become more neurologically stable, resulting in emergent *Communicative Deficits* and *Repetitive Behaviours* factors, reflecting behavioural presentation that is partly independent of primary

factor associates. If such developmental comparisons were repeated for individuals from the bottom 85%, 85-95%, and top 5% of inventory scores, then the degree of Kanner co-variance should be more pronounced with increased trait extremity. *Communicative Deficits* and *Repetitive Behaviours* factors would arise earliest in the top 5% group, latest (if at all) in the bottom 85% group. Factoring out sex in such comparisons would be necessary to avoid unmodeled variance.

A productive shift for diagnosis and treatment would be to institute a new core diagnostic triad of primary behavioural symptoms – *Social Interaction Deficits*, *Cognitive Inflexibility*, and *Sensory Abnormalities* – that better reflects the factor structures found in the literature. The DSM-5 revisions to the diagnostic criteria for autism are a start. For instance, the proposed changes include moving reciprocation-related behaviours from the current *Communicative Deficits* factor into a broader social-communicative deficit category, and moving verbal inflexibilities into a broader restricted and repetitive behaviours and interests category. Further progress, then, can be made by recognising the heterogeneity of behaviours categorised as restricted and repetitive behaviours and interests, separating cognitive inflexibilities and sensory/motor dysfunction into distinct factors. The diagnostic definition of autism should also present symptoms in a hierarchical fashion, distinguishing between a primary triad of *Social Interaction Deficits*, *Cognitive Inflexibility*, and *Sensory Abnormalities* and secondary symptoms (e.g. repetitive behaviours). In this way, the presence of common secondary symptoms could be approached as an indication of co-morbid primary traits, but diagnosis would not depend on symptoms that manifest in highly individualised ways.

The theoretical insights provided by the proposed model should help inform more effective treatment, too. Emergent symptoms, though diagnostically valid, would not be ætiologically primary (Yoder & Belmonte, 2011), and early therapies addressing primary symptoms (*Cognitive Inflexibility*, *Sensory Abnormalities*) might be a more effective treatment for emergent symptoms than are therapies that attempt to confront behavioural diagnostic symptoms head-on. For instance, early non-social cognitive exercises targeted at executive control and integration could exert knock-on effects on later social cognition (Belmonte et al., 2004ab). This conjectured developmental mechanism would reinforce the case for the earliest possible intervention, and further would target such intervention at ætiologically primary rather than diagnostically determinative symptom domains. The aim of these early therapies would be to work with, rather than against, autistic brain physiology and autistic cognitive style (Chen et al., 2012), to channel development towards more integrative sensory and cognitive control.

Non-social behaviours arising from *Cognitive Inflexibilities*, for instance, might be exploited for social cognitive therapeutic ends – a "back door" route to training social cognition (Belmonte et al., 2010). Boyd, Conroy, Mancil, Nakao, and Alter (2007) is a perfect demonstration of the behavioural mechanism through which Kanner-like co-variance may arise, as well as the potential therapeutic benefits of exploiting this mechanism: Peer-initiated play interactions are briefer when the toys in question lie outside the circumscribed interests of ASC children than when play is initiated with toys relevant to circumscribed interests. When toys of interest are present, ASC children's initial social bids to peers occur faster than when less

preferred toys are present. With the right context, attention to social interaction might be bootstrapped via shared attention to special interests.

Conclusion

Biologically and behaviourally emergent aspects of autistic traits and their sub- and non-clinical manifestations can both be better understood as an independently or semi-independently inherited triad of primary factors *Social Interaction Deficits*, *Cognitive Inflexibility*, and *Sensory Abnormalities* dynamically interacting over time in cognition and behaviour. These interactions may become specialised within neural networks, turning their emergent properties into stable, secondary traits, and giving rise to the quintessentially autistic behavioural presentation first described by Kanner.

Ironically, whilst this dynamic, developmental, interactive specialisation model emerged from an effort to settle the debate around the Kanner hypothesis, such a model seems to have been Kanner's understanding of autism from the beginning. Indeed, Kanner's conceptualisation mirrors the proposed model not only in the general sense of dynamic development ["Arguments that counterpose 'hereditary' versus 'environmental' as antithetical terms are fundamentally in error. Operationally defined, they are interpenetrating concepts." (Eisenburg & Kanner, 1956, p. 563); "The dualistic view implicit in a rigid distinction between 'organic' and 'functional' is no longer tenable." (p. 564); "Present knowledge leads to the inference that innate as well as experiential factors conjoin to produce the clinical picture."(p. 564)], but also in the specific, prescient sense of interactive specialisation ["It is equally important to recognise that originally

psychogenic forces must by their enduring action transform the physiological substrate, as the conditioned reflex so clearly demonstrates." (p. 564)].

Coming full circle, we might say that the origin of the Kanner hypothesis is not in Kanner's seminal works (Mandy & Skuse, 2008), or the diagnostic criteria that gave Kanner's "aloneness-and-sameness" definition its veneer of diagnostic inviolability (Happé & Ronald, 2008), but in the way in which the role of development has been downplayed in our interpretations of Kanner. It is only in the absence of development that Kanner's distinction between "aloneness" and "sameness" seems at odds with the two-sides-of-the-same-coin portrayal in his behavioural observations. Decoding Kanner's descriptions in this dichotomous fashion construes all ASC symptoms as primary and heritable, and reduces the question of aetiology to one of nature versus nature, single- versus multi-factor heritability. Moving forward, we must go beyond simply acknowledging development as a side note to genetic factors, and consider the possibility that the behavioural products of dynamic development are the tail that wags the biological dog in autism.

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Table

Table 1. Summary of reviewed factor analytic studies, including which ASC trait domains were analyzed; sample sizes, by diagnostic cate sample age mean and range; the behavioral inventory used; the factor rotation method applied prior to interpretation; the factor structure suggested by each analysis; the percentage of variance explained by each exploratory factor analysis; the Goodness of Model Fit statistic each confirmatory factor analysis; and the correlations (Pearson's *r*) between the factors suggested by each analysis.

Study	Traits	Sample	Age *M* (*SD*) Range	Inventory	Factor Rotation Method	Factors	% Variance Explained (EFA)	Goodness of Model Fit (CFA)	Inter-fa Correlat
Beuker et al., 2013	All	11,332 ASC, PDD, TD	1.55 (0.55)	Ad hoc, from multiple inventories	N/A	Social Interaction Communication Stereotyped and Rigid Patterns of Behaviour	N/A	RMSEA = 0.018	SI-C: 0.46* SI-SRPB: 0. C-SRPB: 0.
Matson et al., 2010	All	405 ASC, PDD	2.26 (0.4) 1.26-3.08	BISCUIT-Part 1	Oblique (Promax)	Socialization/Non-verbal Communication Repetitive Behaviour/Restricted Interests	33.2%	N/A	N/A
Georgiades et al., 2013	All	391 ASC	3.19 (0.68)	ADI-R	Orthogonal (Varimax)	Communication Social Communicative Deficits Fixed Interests and Repetitive Behaviours	32%	N/A	N/A
Magyer & Pandolfi, 2009	All	164 ASC, PDD	3.61 (1.65) 1.6-6.8	CARS	Orthogonal (Varimax, Quartimax), Oblique (Promax, Direct Oblimin)	Social Interaction Social Communication Emotional Regulation Stereotypies and Sensory Abnormalities	57.16% (Orthogonal) 41.67% (Oblique)	N/A	SI-SC: 0.52 SI-S&SA: 0. SI-ER: 0.13 SC-S&SA: 0 SC-ER: 0.18 S&SA-ER: 0
Georgiades et al., 2007	All	209 ASC, PDD	5.46 (2.25) 2.3-40.2	ADI-R	Orthogonal (Varimax)	Social Communication Inflexible Language and Behavior Repetitive Sensory and Motor Behavior	50%	RSMEA = 0.08	N/A

Szatmari et al., 2002	All	129 ASC, PDD	5.5(0.9) 4-6y 11.6(5.8) 5-N/A	ADI-R	Orthogonal (Varimax)	Symptom Severity	70.3%	N/A	N/A
						Overall Level of Functioning	67.7%		
Gotham et al., 2007	All	1139 ASC, PDD	5.64 (2.2) 1.2-15.3	ADOS	Oblique (Promax)	Social Affect	N/A	RMSEA = 0.04-0.07	0.38-0.59
						Restricted and Repetitive Behaviors			
Gotham et al., 2008	All	1259 ASC, PDD	6.2 (2.12) N/A	ADOS	Oblique (Promax)	Social Affect	N/A	RMSEA = 0.05-0.08	0.34-0.57 (a 0.12-0.35 (A
						Restricted and Repetitive Behaviors			
Snow et al., 2009	All	1861 ASC, PDD	8.32 (3.16) 4-18	ADI-R	Oblique (Quartimin)	Social Communication	N/A	RMSEA = 0.026 (Non-verbal), 0.068 (Verbal)	N/A
						Restricted/Repetitive Behaviors			
Frazier et al., 2012	All	14774: 8911 ASC, 5863 Sibs	SRS: 8.4 (3.99); SCQ: 7.53 (4.37)	SRS, SCQ	N/A	Social Communication	N/A	N/A	0.36-0.81 (a 0.53-0.97 (s
						Autism Mannerisms			
Frazier et al., 2008	All	1170 ASC, PDD	9 (4.88) 2-46	ADI-R	Oblique (Promax)		N/A	RMSEA = 0.07	N/A
						Social Communication			
						Restricted/Repetitive Behaviors			

Mandy et al., 2013	All	708: 488 ASC, 220 BAP	9.5 2.4-21.1	ADI-R	N/A	Social Communication Restricted/Repetitive Behaviors	N/A	RMSEA = 0.079 (DSM-5), 0.061 (DSM-5 modified), 0.054 (DSM-5 with sensory abnormalities)	N/A
Boomsma et al., 2008	All	263 ASC, PDD	11 (5) 4-24	ADI-R	N/A	Play Skills Social Communication Stereotypies in Communication and Behavior	N/A	RMSEA = 0.05 (Current), 0.07 (Ever)	N/A
Van Lang et al., 2006	All	255: 125 TD, 130 ASC, PDD	11.25 (3.92) 4-20	ADI-R	N/A	Play Skills Social Communication Stereotypies in Communication and Behavior	N/A	RMSEA = 0.03	PS-SC: 0.48 PS-SCB: 0.3 SC-SCB: 0.4

Study	**Traits**	**Sample**	**Age *M* (*SD*) Range**	**Inventory**	**Factor Rotation Method**	**Factors**	**% Variance Explained (EFA)**	**Goodness of Model Fit (CFA)**	**Inter-fa Correlat**
Kamp-Becker et	All	140: 35 TD,	12.04 (4.59)	ADI-R	N/A	Social Communication	N/A	N/A	N/A

al., 2009		105 ASC, PDD	6-24			Inadequate Behaviors		
						Anxiety and Compulsions		
						Stereotyped Behavior		
					Oblique (Varclus)	Spoken Language	41%	N/A
						Social Intent		
Tadevosyan -Leyfer et al., 2003	All	292 ASD	15.58 (6.21) 2-47	ADI-R		Compulsions		
						Developmental Milestones		
						Savant Skills		
						Sensory Aversions		
Constantino et al., 2004	All	226 ASD, PDD, TD	4-18y	ADI-R	Orthogonal (Varimax)	Single factor	35%	N/A
					Oblique (Promax)	Social Skills	29%	N/A
Hurst et al., 2007	All	1005 TD	19.36 (3.89) 17-55	AQ		Communication		
						Attention to Detail		
Valla et al., 2010	All	144 TD	20.2 (1.86) 18-27	AQ	Orthogonal (Varimax)	Social Interaction	64%	N/A
						Preference for Details/Patterns		
Kloosterman et al., 2011	All	522 TD	21 (5.15)	AQ	Oblique (Promax)	Social Skills	45%	RMSEA = 0.053

SL-SI: 0.45*
SL-C: 0.33*
SL-DM: 0.17
SL-SS: 0.02
SL-SA: -0.0
SI-C: 0.25*
SI-DM: 0.38
SI-SS: 0.04
SI-SA: 0.06
C-DM: 0.07
C-SS: 0.04
C-SA: 0.06
DM-SS: -0.1
DM-SA: 0.0
SS-SA: 0.09
N/A
N/A
N/A
N/A
SS-C: 0.22*
SS-RRB: 0.

Stewart & Austin, 2009	All	536 TD	24.3 (10.5)	AQ	Orthogonal (Varimax)	Communication Imagination Attention to Detail Restricted and Repetitive Behaviors Social Skills Communication Imagination Attention to Detail	29%	N/A	SS-I: 0.12 SS-AD: -0.0 C-RRB: 0.3 C-I: 0.26* C-AD: -0.35 RRB-I: 0.00 RRB-AD: 0. I-AD: -0.19 SS-C: 0.16* SS-I: 0.22** SS-AD: 0.07 C-I: -0.04 C-AD: 0.00 I-AD: 0.18**
Hoekstra et al., 2008	All	1374: 1338 TD, 15 ASC, 15 OCD, 15 SAD	24.65 (4.32)	AQ	Oblique (Promax)	Social Interaction Preference for Details/Patterns	N/A	N/A	0.19
Austin, 2005	All	337 TD	34.22 (7.23)	AQ	Oblique (Promax)	Social Skills Communication Attention to Detail	28%	N/A	SS-C: 0.20
Grove et al., 2013	All	1034: 363 ASC, 439 Parents, 232 TD	ASC: 36(11) Parents: 42(8) TD: 33(10)	AQ	N/A	Empathising Systemising	N/A	RMSEA = 0.00 (all three groups, individually)	ASC: -0.62 Parents: -0.5 TD: -0.22
Lau et al., 2013	All	455: 141 ASC, 314 TD	40.6 (7.8 ASC, 8.8 TD) 19-71y	AQ	Oblique	Sociability Social Cognition Narrow Focus	48.1%	RMSEA = 0.001 (ASD), 0.07 (TD)	S-SC: 0.759 S-NF: 0.751 S-IP: 0.631* S-RC: 0.710 SC-NF: 0.66 SC-IP: 0.58

						Interest in Patterns			SC-RC: 0.72 NF-IP: 0.753 NF-RC: 0.69 IP-RC: 0.62
						Resistance to Change			
Study	**Traits**	**Sample**	**Age *M* (*SD*) Range**	**Inventory**	**Factor Rotation Method**	**Factors**	**% Variance Explained (EFA)**	**Goodness of Model Fit (CFA)**	**Inter-fa Correlat**
Richler et al., 2007	RRBI	279: 65 TD, 165 ASC, PDD	2.23 (0.45)	ADI-R	Orthogonal (Varimax)	Insistence on Sameness	N/A	N/A	N/A
						Repetitive Sensory and Motor Behavior			
Papageorgiou et al., 2008	RRBI	153 ASC, PDD	5.96 (3.3) 1.6-19	ADI-R	Orthogonal (Varimax)	Insistence on Sameness	52%	N/A	N/A
						Repetitive Sensorimotor Behaviors, Interests			
Szatmari et al., 2006	RRBI	339 ASC, PDD	8.4 (5.51)	ADI-R	Orthogonal (Varimax)	Insistence on Sameness	36% (current), 33% (ever)	N/A	N/A
						Repetitive Sensory and Motor Behavior			
Lam et al., 2008	RRBI	361 ASC, PDD	9.02 (6.15) 1.6-29	ADI-R	Orthogonal (Varimax)	Insistence on Sameness	52%	N/A	IS-CI: 0.146 IS-RMB: 0.0 CI-RMB: -0.
						Circumscribed Interests			
						Repetitive Motor Behavior			
Cuccaro et al., 2003	RRBI	207 ASC, PDD	9.06 (4.57) 2.4-21.2	ADI-R	Oblique (Promax)	Resistance to Change	32%	N/A	0.08
						Repetitive Sensory and Motor Behavior			

RRBI = Restricted and Repetitive Behaviors and Interests; TD = Typically Developing; ASC = Autism Spectrum Conditions; PDD = Pervasive Developmental Disorders; BAP = Broader Autism Phenotype; CARS = Childhood Autism Rating Scale; ADI-R = Autism Diagnostic Interview – Revised; ADOS = Autism Diagnostic Observation Schedule; SRS = Social Responsiveness Scale; SCQ = Social Communication Questionnaire; AQ = Autism Spectrum Quotient; EFA = Exploratory Factor Analysis; CFA = Confirmatory

Factor Analysis; RMSEA = Root Mean Squared Error of Approximation (< .05 = close fit; < .08 = good fit; > 0.1 = poor fit), Abbreviations in inter-factor correlation column correspond to the factor domains (listed in Factor column) specific to that study; e.g., the correlation between the Social Interaction and Social Communication factors found by Magyer & Pandolfi (2009) was $r = 0.529$. For inter-factor correlations, * < .05, ** < 0.01, *** < 0.001.

***Detailed Response to Reviewers**

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27 August 2013

Professor Mark Howe
Associate Editor, *Developmental Review*
Department of Psychology
University of Lancaster

Dear Professor Howe,

We wish to thank you and the reviewers for giving our manuscript another close reading. We do apologise for the length of time taken for this revision, which is a consequence of the second author's chance of primary institutions.

Below we detail our efforts to address the points raised by each of the reviewers, in some cases by refutation, in others by incorporation. In the latter instances, we tried to be as prudent as possible, adding length to the manuscript only when absolutely necessary. Reviewers' points are emboldened, and our responses provided after each point.

In addition, at your request we have done another literature search to ensure any new findings of relevance are incorporated. Our search revealed seven additional works relevant to our manuscript; whilst each adds depth and nuance to the themes we cover, none required substantial reworkings of our manuscript to incorporate. The factor analytic studies among these have also been added to the table summarizing the reviewed works.

With thanks,

Jeffrey M. Valla
Matthew K. Belmonte

Reviewer A

Page 3: "They are described theoretically in terms of (1) deficits in Theory of Mind (ToM) or understanding intentionality (Baron-Cohen, 1995; Baron-Cohen, Leslie, & Frith, 1985) and in future-oriented cognitive flexibility, or executive functioning (EF) (Ozonoff, Pennington, & Rogers, 1991)"

Why are these two very different cognitive functions grouped together?

This was a mistake on our part; we did not intend to group ToM and executive functioning. This has been corrected as such:

"They are described theoretically in terms of deficits in Theory of Mind (ToM) or understanding intentionality (Baron-Cohen, 1995; Baron-Cohen, Leslie, & Frith, 1985); deficits in future-oriented cognitive flexibility, or executive functioning (EF) (Ozonoff, Pennington, & Rogers, 1991); and a bias toward local over global information processing, or weak central coherence (WCC) (Frith & Happé, 1994)."

Page 4: ", the 'Kanner hypothesis' - that, in Kanner's parlance, a preference for "aloneness" is the flipside of a related need for "sameness" –"
Did Kanner actually argue that these are 2 sides of the same coin? I'm not in favour of putting words into others' mouths, especially when they are no longer alive and able to refute these ascriptions.

This is an excellent point, and returning to Kanner's seminal works for a closer reading gave us a better understanding of the origins of the Kanner hypothesis; helped us better contextualize the arguments of the present review in terms of how they improve upon previous reviews; and provided a way to bring the manuscript full circle and tighten the overall narrative. Please see pages 4-5, and 44-45 for these revisions.

Page 6: "Supporting this assertion is evidence of higher testosterone levels in amniocenteses of individuals presenting with autism-like empathising difficulties and systemising prowess later in development (Baron-Cohen et al., 2004),"
The additional more up to date citation for this sentence would be the Plos-Biology 2011 article by Baron-Cohen and colleagues.

Citation added to page 6.

Page 9: "In fact, challenging E-S theory is in a sense the purpose of this review, though the interest here lies more narrowly in challenging the Kanner assumption of co-variance between the "E" and the "S," rather than evaluating E-S theory in general." This sentence seems both redundant and inaccurate, since the article has many primary purposes, none of which are about challenging the E-S theory. (From this reviewer's perspective, the primary purposes include outlining a developmental model that starts with cognitive overstimulation and reduced ToM and snowballs into cognitive inflexibility and repetitive behavior; and conducting various factoranalytic approaches to autistic traits). Moreoever, given that the E-S theory is

usually summarized as two orthogonal dimensions but where there may be a weak inverse correlation, this doesn't seem at odds with anything the authors are proposing.

We have revised our discussion of E-S theory (pages 5-9) to better reflect its role in the review. To address concerns by this and other reviewers about the overall purposes and specific aims of our review, and how the review is structured to address these, we have also added a section (pages 10-11) in which we lay out the overall purposes, specific aims, and structure of the review in an explicit fashion.

Page 13: "In the dialectic that follows, we begin by reviewing.."
I don't think the term 'dialectic' is appropriate for a review article.

This statement is not included in the new section laying out the purposes, aims, and structure of the review (pages 10-11).

Page 17: "Cognitive inflexibilities subsumed under the Communicative Deficits diagnostic domain may be fed by deeper roots."
Why lump these two quite separable aspects of behavior together?

One of the factor analytic themes we discuss throughout the review is the fact that communicative deficits currently covers a heterogenous mix of linguistic inflexibilities and social interaction deficits. Because of this heterogeneity, cognitive inflexibility and communicative deficits are not as separable as this reviewer suggests.

Page 28: "the bailiwick of.."
Will the average reader know what this word means?

Changed to the following (page 30):

"It is in this developmental construction that non-social capacities can be drafted into the service of social cognitive ends…"

Page 33: "Repetitious movements may be externalised efforts to impart predictability on a world rendered chaotic and intractable by an abnormal, cognitively inflexible, perceptually overstimulated mental environment (Belmonte, 2008)."
The authors should acknowledge this is speculative, not empirical.

We have added the following (page 35):

"Whilst this point remains a speculative one, relationships between ritualistic behaviours and perceived uncertainty or fearfulness do exist in young, typically developing children (Evans et al., 1999)."

Page 39: "behavioural theorists are well inured to the idea"
This phrasing seems a little informal.

Changed to the following (page 42):

"The advantage of a developmental perspective may seem obvious; behavioural theorists know well that developmental models."

Page 42: "varying as widely as 2-46y,"
Y should be spelt out as 'years'

See page 45.

Reviewer B

The authors have responded to the call to link this work to current changes in DSM-V criteria. However, while I felt the level of detail in this section was sufficient when we came to it (on page 45), we do need at least some mention of these DSM revisions much earlier on. There is lots of talk of the 'current diagnostic triad' and even 'the diagnostic triad' but most readers will look at this knowing that these criteria are set to change.

This is an excellent point, and briefly discussing the DSM-5 changes helped us better contextualize the present review in the newly added section laying out the purposes, aims, and structure (page 10-11).

I welcome the additional detail in Table 1. However, I felt that the table should be properly 'introduced' in the text, perhaps on page 12. A related point is that I couldn't find a caption for this table in the files I was sent, and so there really is a lack of explanation as to what it represents/includes.

See page 13 for added introduction the table. See table for caption and key.

If the authors are going to review correlational studies between social and non-social traits (e.g., p. 26), they should probably include more of the work from Murray Maybery's group in this area. Suzanna Russell-Smith has published a number of papers that may be relevant (particularly Russell-Smith et al., 2012, but see also Morgan et al., 2003; Pellicano et al., 2006). A recent paper by Johnson (2012) might be useful in supporting the

claim (bottom of p. 33) that "Overall level of functioning, then, may determine the executive control capacity available to inhibit externalising, overtly motor behaviours", and some of the subsequent discussion of the moderating effects of IQ.

See pages 28, 24, 24, and 36, respectively, for added discussions and citations of these articles.

I buy most of the arguments presented in the paper, but found some of the suggestions on the bottom of page 40 too speculative, (e.g.,) that spinning fans may be more visually salient than moving mouths. More predictable perhaps, but does that necessarily make them more salient?

We have clarified our assertion about the relative saliency of our spinning fan example as such (page 43):

"Meanwhile, the more physically salient details of the inanimate world capture their attention: a spinning ceiling fan can be a more visually salient motion than a moving mouth (i.e. it covers more physical space, at a greater speed, and in a more predictable fashion), a car horn a more intense sound than a voice."

Page numbers are needed for the Kanner quotes on page 4 and the Spiker et al. quotes on page 38.
p. 25 line 7 – is there a missing space after the full stop?
p. 42, line 4 – were to begin entirely independently ?

Both typos have been corrected; as a result of other editing, the former is now on page 27, the latter on page 44).

Reviewer C
This paper examines the veracity of the so-called "Kanner hypothesis", that is, that individual differences in social communication and repetitive and restricted behaviours covary both in people diagnosed with an autism spectrum condition. The authors have done a good job responding to my and the other reviewers' comments.

Upon reading the manuscript afresh, however, I found it very difficult to work out precisely what the authors are trying to achieve. The initial paragraph begins by alerting the reader how the core features of autism have been interpreted thus far. But no mention is made regarding the goals of the current review. To ensure that their review has the greatest impact, I suggest that they include a brief paragraph at the beginning (after the initial paragraph on page 3), which clearly outlines for the reader both the aim of this review and precisely how the argument will progress (including the types of evidence analysed).

A fresh reading of our manuscript left us with the same criticism. We hope that Reviewer C will find the revised manuscript greatly improved in this respect, given the aforementioned revision to the framing of the Kanner hypothesis, and the aforementioned addition of a section laying out the purposes, aims, and structure of the review explicitly.

Also, I felt that the authors often conflate behavioural and cognitive characteristics. I certainly don't see these as one and the same and it seemed, at least in the first paragraph that the authors agreed. For example, they state that the behavioural characteristics are "described theoretically in terms of (1) deficits in theory of mind … and executive functioning … and also (2) a bias toward local over global information processing" (p. 3).

But in the subsequent paragraph, they seem to refer to cognitive "traits" and behaviours as interchangeable. For example, they state that Kanner "framed autistic social and detail-oriented cognitive bias as two sides of the same coin" (p. 4). Yet, Kanner did not describe autism in this way. Instead, he stated that there are "two specific symptoms that always have to be present in order to justify the diagnosis. They are extreme aloneness from practically the beginning of life and what I call the desire for the preservation of sameness". Kanner, therefore, did not frame autism in terms of underlying cognitive atypicalities, as the authors suggest; this is the authors' and others' subsequent interpretation (within a cognitive framework) of Kanner's initial description. The phrasing of this particular paragraph should therefore be amended.

These issues raised by Reviewer C have been addressed in the process of handling other reviewers' criticisms, especially in overhauling our treatment of Kanner's seminal works and how they have been interpreted. These changes can be found in the introduction (pages 4-5), and the conclusion (pages 49-50)

I suspect that the authors might disagree with me on this point because they don't include cognition in their levels of explanation on page 42; they only refer to understanding autism at the genetic, neurobiological and behavioural levels. But many researchers would agree that understanding autism at the cognitive level of explanation is just as important as knowledge at the neurobiological or genetic levels and should be considered distinct from behaviour. I therefore urge the authors to consider distinguishing cognition and behavior throughout the manuscript, including in the title (I prefer the running head) and abstract (also see para 2, page 26 for another example).

Whilst we understand Reviewer C's concerns about our using the term "behaviour" broadly to encompass social and cognitive aspects of ASC, the context in which the present review takes place necessitates this usage for reasons best demonstrated by the factor analytic studies comprising the majority of the evidence that we review. The inventories used in these studies clearly register cognitive and social attributes, in addition to ASC traits that are behavioural in the most literal sense (e.g., motor stereotypies). But these measures are still considered behavioural inventories because even internal, cognitive aspects of ASC, such as a need for sameness, are still being inferred from external behaviors (e.g., stacking; preoccupation with inanimate objects; restricted interest in predictable physical phenomena). Within the context of these inventories, we do distinguish between literal behavioural attributes, and the social and cognitive traits being inferred from external behaviour. When placing the present review within the broader context of autism research, though, it seems to make more sense to follow others' prior practice of grouping together studies that infer social, cognitive, and motor ASC traits from external presentation.

Note that DSM-V should be DSM-5.

We have corrected this discrepancy throughout the manuscript.