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# 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

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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.

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## Introduction

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, Briskman, & Frith, 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 ([Happé & Ronald, 2008](#); [Mandy & Skuse, 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 and Skuse \(2008\)](#), assumptions of social/non-social co-variance are traceable to Kanner’s original case descriptions ([Eisenberg & Kanner, 1956](#); [Kanner, 1943](#)): “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 [Eisenberg and Kanner \(1956\)](#) make no explicit assertions or speculations about aetiological 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” ([Eisenberg & 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 & Ronald, 2008](#)). This clinical definition

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