**Title: The coherence of autism**

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**Lay abstract:** There is a growing body of opinion that the syndrome of autism can be separated into subsets of clinical features that only sometimes occur together. Often affected individuals’ restricted social engagement is accompanied by atypical patterns of communication and stereotyped activities, but this is not always the case. Different genetic predispositions might apply to different parts of the clinical picture. Moreover, each subset of atypicalities might need explanation in terms of its own, distinctive psychological theory. The present paper offers a critique of this position. It argues for the alternative view, that autism is a coherent syndrome in which principal features of the disorder stand in intimate developmental relation with each other. Although we shall need different levels of explanation if we are to understand autism, this does not mean we should abandon integrative theories. One such theoretical approach acknowledges diverse causes of autism, but posits that the syndrome develops through a final common pathway involving disruption in engagements between affected individuals and other people. This disruption interferes with a developing child’s experience of a world that is shared with others. Studies of congenitally blind children offer support for this view of autism, and suggest that a source of coherence in the syndrome is restriction in certain forms of perceptually dependent social experience.

**Title: Exploring the ‘fractionation’ of autism at the cognitive level**

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**Lay abstract:** Autism is diagnosed on the basis of difficulties within the social domain, such as communication problems and poor social skills, and difficulties within the non-social domain, such as restricted and repetitive behaviour and interests, and sensory abnormalities. Psychological theories have been proposed to explain these symptoms of autism. For example, people with autism tend to have a difficulty in understanding their own and other peoples’ thoughts, otherwise known as a poor ‘theory of mind’. They also show difficulties in ‘executive function’, i.e. they have difficulty planning and monitoring their actions. In addition, people with autism are often good at noticing and remembering details, sometimes at the expense of ‘the big picture’, otherwise known as ‘weak central coherence’. However, it seems unlikely that any one of these theories can explain both the social and non-social symptoms of autism. Recently, it has been proposed that the core symptoms of autism are ‘fractionable’; the social and non-social symptoms of autism are suggested to have distinct causes at the genetic, neural and psychological or cognitive levels. For example, some individuals have social difficulties without repetitive behaviour. The present paper reviews the evidence that cognitive functions are fractionable in autism. First, we discuss whether performance on tests of theory of mind, executive function and weak coherence are strongly related in autism. We also review research on the relationship between performance on these tests and everyday behaviour or symptoms. Finally, we suggest that a multiple cognitive account of autism can help us better understand the varied symptoms of autism.

**Title: Confirmatory factor analytic structure and measurement invariance of quantitative autistic traits measured by the Social Responsiveness Scale-2**

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**Lay abstract:** The Social Responsiveness Scale (SRS) is a questionnaire measuring the severity of autistic traits – which are largely inherited – as they occur in everyday settings (as rated by either parents or teachers). In this study, we analysed a large number of SRS reports (n = 9635) involving children and adults with a wide range of levels of symptom severity. The data were analysed using a statistical technique called ‘factor analysis’, which involves studying the ways in which items ‘travel together’ in nature, revealing small numbers of core
factors that represent key biological variables captured by the instrument. Using this approach, we found that the data collected from the SRS could be parsed into two factors: (1) social communication impairments, and (2) restricted, repetitive behaviours, as has been operationalized in DSM-5. We were also able to further differentiate between three sub-factors encompassing social communication impairment (emotion recognition, social avoidance and interpersonal relatedness) and two sub-factors encompassing restricted, repetitive behaviour (insistence on sameness and repetitive mannerisms), which may have biological relevance and/or represent intervention targets for specific sub sets of patients with ASD. While the large sample size of this study allowed statistical differentiation of these factors, correlational analysis revealed that they remain closely associated with one another in nature. A priority for future research is to understand the biology of the co-emergence of these symptoms.

**Title:** Investigating the cross-cultural validity of DSM-5 autism spectrum disorder: Evidence from Finnish and UK samples

**Authors:** Mandy, Charman, Puura and Skuse

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**Lay abstract:** With the recent publication of DSM-5, the official definition of autism spectrum disorder (ASD) has been revised. This was based on research from North America and the UK. ASD is an increasingly global diagnosis, and it is unclear how well its new definition generalises beyond North America and the UK: perhaps ASD’s manifestation varies depending on the cultural and linguistic context in which it presents. To explore this possibility, we compared how well the DSM-5 account of ASD described the autistic symptoms of young people from Finland (n = 240) and the UK (n = 708). Our sample included participants with both clinically severe ASD and milder, sub-clinical autistic traits. We used a statistical technique called confirmatory factor analysis to examine whether the way in which autistic symptoms clustered together was consistent with the DSM-5 description of ASD; and to test whether their pattern of clustering was the same in Finish and UK participants. For participants with ASD, the DSM-5 model worked equally well in Finland and the UK. Among those with sub-clinical difficulties, it fitted well in the UK but poorly in Finland. This suggests that the DSM-5 symptom model generalises well to Finnish people with ASD, but not to those with sub-clinical autistic traits: cross-cultural variability may be greatest for milder autistic characteristics. We encourage researchers with data from other cultures to emulate our approach, to map any cultural variability in the manifestation of ASD and the broader autism phenotype (BAP). This would be especially valuable given the on-going revision of the *International Classification of Diseases (ICD)*, the most global of the diagnostic manuals.