

Are dimensional approaches to autism appropriate? Letter to the Editor Journal of Autism and Developmental Disorders by Francesca Happé and Uta Frith, September 2021

We are grateful for the careful thought that Chown and Leatherland have given to one part of our article in the Journal of Child Psychology and Psychiatry looking back over thirty years of autism research. They highlight a topic of much current debate: whether autism lies on a continuum with non-autism or ‘neurotypicality’. This is part of a wider discussion of whether neurodevelopmental and other conditions should be conceptualised dimensionally or categorically.

In our, necessarily brief, consideration of this question in our paper, we used the Causal Modelling framework (Morton & Frith, 1995) to clearly separate three levels of explanation: biological, cognitive and behavioural. At the biological level – not discussed by Chown and Leatherland – the same genetic factors appear to influence diagnosed autism and variation in traits relevant to autism in the general population (ref), supporting a dimensional approach. Indeed, the ‘broad autism phenotype’ was coined to recognise that relatives of diagnosed autistic people show more traits relevant to autism than is typical of the general population. Our reading of the biological research to date is that the techniques available do not, as yet, offer reliable evidence of qualitative differences in brain structure (Carmon et al. 2020), or well replicated qualitative differences in brain function in autism. So, although it may prove true, on the basis of the current evidence it is hard to support Chown and Leatherland’s assertion that, ‘Autistic individuals share a neurological type’.

At the cognitive level, as noted in Chown and Leatherland’s footnote, we suggest there are qualitative differences (in automatic mentalising) and quantitative differences (in central coherence) between autism and non-autism. In this respect we are in full agreement with Chown and Leatherland that what defines autism is a different way of processing the world. Indeed, we have spent more than 30 years proposing and testing cognitive theories of autism, and in some cases arguing strongly for the importance of understanding cognition against a tide of predominantly biological or behavioural autism research (e.g., Frith, 2012). It is ironic, then, that Chown and Leatherland consider that we take too behavioural a view of autism.

The third level of explanation, that of behaviour, is tricky because – as Chown and Leatherland quote from our paper – the same behaviour can have different causes and different cognitive underpinnings. For this reason, one might be dubious about (typically questionnaire) measures of ‘autistic traits’ and whether these – which show a dimensional distribution of such behavioural traits in the general population, with no discontinuity with diagnosed autism – measure something qualitatively akin to autism. The interesting question to ask is, what would convince one either way? As cognitive psychologists we would be convinced if those qualitatively and quantitatively distinct cognitive characteristics of autism show a strong association with behavioural (typically questionnaire) autism trait measures (for an interesting recent example see Coll et al, 2020). Biological researchers may be more persuaded by genetic or neuroimaging evidence for the validity of autism trait measures.

Unfortunately, the behavioural level is where the diagnosis of autism – as of many other conditions (e.g., ADHD) – currently rests. We wish that really good cognitive tests were able to reliably identify autism in the clinic at the individual level. Some of lab-designed tests are used in diagnostic clinics to add information about processing style and strengths and weaknesses, but no clinician would currently rely solely on cognitive tests. Instead, autism is currently a behavioural diagnosis. Relying on current observed behaviour and past developmental history is imperfect, but all the clinician has to work on. Good clinicians will try to see a child in varied settings (e.g. school playground), and will ask an adult how they experience different contexts and what it costs them to cope socially. The need for careful consideration of the limitations of behavioural diagnosis has been brought into greater focus through the recent work on compensation (Livingston & Happé, 2017) and camouflaging, referred to in our paper and Chown and Leatherland’s letter.

In summary, the question, ‘Is autism qualitatively or quantitatively distinct from non-autism?’ confirms our belief that we need to distinguish the biological, cognitive and behavioural levels of description, explanation and empirical research. There can, therefore, be no single answer. Our reading of the research literature to date would suggest that biologically and behaviourally it has not proved possible to pinpoint reliably any qualitative distinction, but at the cognitive level there are both quantitative and qualitative differences in how autistic people process and experience the world. It would be wonderful if future research could develop neurocognitive tests sensitive enough to be reliably used in diagnosis; we would welcome the day when autism can be said to be a neurocognitive (not purely behavioural) *diagnosis*, as we certainly believe that autism is best understood and characterised at the cognitive level.

References

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