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‘Autism’ or ‘Autism Spectrum Disorder’: Does either represent a Natural Kind of Psychological Disorder?

Abstract

In DSM-5, “autism spectrum disorder” (ASD) is a new diagnostic category effectively replacing the previous category of “autism”. In this paper I question whether either effectively represents a psychological natural kind with significant scientific and explanatory value.

Despite the new categorisation, “ASD” and “autism” are effectively synonymous, and current understandings of ASD are based largely on previous research focussed specifically on autism. However, there has been no stable consensus over the past 40 years about what autism actually is. No biological explanation has been discovered, and no single psychological theory can account for the heterogeneity of autistic symptoms. A recent large-scale population-based study failed to identify a unifying cognitive account of the variety of symptoms of autism.

In the philosophy of science literature, there are widely accepted accounts of natural kinds which emphasise their role in scientific explanations and induction. These claim that natural kinds can typically be identified by clusters of properties which are held together by causal processes and which reflect the causal structure of the world in terms of their explanatory and predictive value. However, the concept of ASD fails to indicate any causal explanation and has very limited discriminant and predictive validity. Consequently ASD, as a diagnosis, cannot plausibly be seen as a psychological natural kind, since it does not appear to function as a powerful explanatory concept in science. Psychologists involved in autism diagnostic services should try to explain more clearly what it is that they believe they are diagnosing.

The concept of autism has been with us since Leo Kanner first identified and named it in the 1940s. However, its defining characteristics have altered dramatically since then, and particularly so from the 1980s onwards. The assumption that it represents a distinct kind of diagnosable disorder with significant scientific and explanatory value has persisted throughout this period. In particular, this view now underpins the thinking of clinical and educational psychologists who actively participate in autism diagnosis teams, despite (or perhaps because of) the fact that such an assumption has remained largely unexamined. However, the view of autism as a predominantly scientific category has been challenged by, among others, the philosopher Ian Hacking (2015) who has argued that our current conception of autism has been shaped substantially more by advocacy and activism than by science.

In this paper, I examine the question of whether autism can constitute a natural kind with explanatory significance in science. I address this question with particular reference to whether the various symptoms of autism can be explained by any unifying psychological deficiency which might constitute a psychological natural

kind. I start by noting the various changes in its conception introduced in DSM-5 (American Psychiatric Association, 2013). Next I consider the main theories about hypothesised psychological deficits presumed to underlie autistic symptomatology. I discuss one recent and particularly authoritative study of a large number of young people with an autism diagnosis which demonstrates very strongly that no single cognitive deficit can account for the symptoms leading to the diagnosis. This, along with previous research, casts doubt on the idea that autism can be regarded as a psychological natural kind definable by a single psychological feature.

I then discuss the function of natural kinds as explanatory concepts in science, with reference to two closely related theories of natural kinds proposed by the philosophers, Richard Boyd and Muhammad Ali Khalidi. I describe the key features of these theories, in particular the requirement that natural kinds should in some way reflect the causal structure of the world such that they can ground reliable explanations and inductions. I explain that these accounts have general applicability across all the sciences and that natural kinds in science have explanatory and inductive significance. I then argue that the category of autism has little scientific value in this sense and I conclude that, on current evidence, autism does not represent a psychological natural kind.

Changes in diagnostic criteria

In DSM-IV (American Psychiatric Association, 1994), autism was identified with the “triad of impairments” which were: (1) impairments in social interaction, (2) impairments in language and communication, and (3) restricted or repetitive behaviours and interests (RRBIs). In DSM-5 this has now been superseded by a new category of “autism spectrum disorder” (ASD). To meet the criteria for a diagnosis the individual concerned needs to display two types of impairment: (1) persistent deficits in social communication and interaction, and (2) RRBIs (as in DSM-IV). This in effect amounts to the current definition of ASD, albeit in brief form. Some symptoms need to be present (but not necessarily evident) during the child’s early development and the symptoms must cause clinically significant impairment in the individual’s functioning. The new category of autism spectrum disorder contrasts with the previous categorisation in DSM-IV, which listed five sub-categories in what was then termed “Pervasive Developmental Disorders” (PDD) – these were: autism, Asperger syndrome, Rett syndrome, childhood disintegrative disorder (CDD), and

pervasive developmental disorder (not otherwise specified) (PDD-NOS). Whilst “autism” has now been omitted as a formal category, it nevertheless remains in regular use as short-hand for the new category of ASD, with which it appears virtually synonymous. There were various reasons for excluding the other sub-categories from DSM-5, some of which can be briefly summarised here. Rett syndrome is a rare genetic disorder predominantly affecting girls and is in effect a distinct medical disorder. CDD is also a very rare condition, and there is significant uncertainty as to whether it actually constitutes a distinct disorder. PDD-NOS refers to cases where the individual displays some autistic traits which are typically mild and do not amount to an unambiguous diagnosis of autism. Some of these would now be covered by the separate category of “social communication disorder” in DSM-5.

However, the most notable exclusion from DSM-5 is Asperger syndrome. The criteria in DSM-IV failed to distinguish it clearly from autistic disorder, and particularly from high-functioning autism, and there has been wide variation in how it has been applied in practice (Happé, 2011). What has persisted however is the concept of an “autism spectrum”, such that this concept, rather than “autism” simpliciter, now constitutes the named disorder in DSM-5. Typically however, the two terms are used interchangeably, as I shall do here.

In addition, DSM-5 has introduced a new category of “social-communication disorder”. The criterion for this is essentially the same as the first criterion for ASD, namely a persistent deficit in social cognition and interaction skills. Someone who displays such a deficit without any restricted or repetitive behaviours and interests (the second criterion for an ASD diagnosis) cannot be diagnosed with ASD. The only available diagnosis for such individuals is social-communication disorder.

Due to lack of space, I cannot review all the various changes that have occurred to the diagnostic criteria for autism since Kanner’s time (but see Verhoeff, 2013, for a detailed account of how these changes reflect some fundamental shifts in the conceptualisation of autism). Nevertheless, the quite major changes from DSM-IV to DSM-5 would suggest that autism is not a condition that has yet achieved much stability in its conceptualisation.

Psychological theories of autism

A number of theories have been proposed about an underlying psychological deficit which can account for the presenting symptoms of the condition. Such a unifying

deficit, if found, might be associated with some kind of neurological abnormality which would explain the nature of the disorder. However, no theory so far advanced has been able to do this and several autism researchers are now asking whether any such explanation is even possible (e.g. Happé, Ronald & Plomin, 2006). Typically, the psychological theories put forward have tended to focus on hypothesised cognitive deficiencies in people with an autism diagnosis. These include a “theory of mind” deficit (an impaired ability to understand the mental states of others), weak central coherence (difficulty in integrating detailed information into larger meaningful wholes), and executive function deficit (an inability to plan and co-ordinate actions to achieve intended goals). Although other psychological deficits have also been hypothesised, these three have generated the most interest for researchers. However, the large number of research studies which have investigated the association between these cognitive features and children with an autism diagnosis have failed to produce any consistent and replicable findings which might explain all the symptoms of autism (Brunsdon & Happé, 2014).

This is clearly demonstrated by a recent population-based study which is one of the largest of its kind (Brunsdon et al., 2015). Recruiting adolescents with an ASD diagnosis from the UK Twins Early Development Study, the researchers, based at the Institute of Psychiatry in London, studied 181 young people with ASD, 73 non-ASD co-twins and 160 normally developing controls. All of the ASD group were diagnosed using “gold-standard” instruments, specifically the Autism Diagnostic Inventory-Revised and the Autism Diagnostic Observation Schedule. All participants were assessed on a range of tests designed to test for a theory of mind deficit (ToM), weak central coherence (CC), and executive function deficit (EF). All the tests used were taken from previous studies in which the test procedures had been established. The results of this study failed to demonstrate a clear and consistent pattern of cognitive deficits in the ASD sample. Only a small number demonstrated deficits in a single area (5% in ToM; 8% in CC; and 6% in EF). Others had deficits covering at least two areas, and the largest group (32%) had deficits in all three areas. The authors argue that this therefore represents a characteristic pattern in ASD adolescents. Nevertheless, it is clear that more than two thirds of the sample did not show this pattern. Moreover, it seems from the reported percentages showing the various cognitive deficits that 9% of the ASD sample failed to demonstrate any deficit in any of the areas examined, but this apparent result is not discussed by the authors. It was

also noted that corresponding cognitive deficits occurred in the non-ASD co-twins and in the controls, though in smaller numbers. The combination of all three deficits (ToM + CC + EF) was found in 11% of the non-ASD co-twins and in 6% of the controls, despite their lacking a diagnosis. Thus, when tested using operationalized measures of the three hypothesised cognitive deficits in one of the largest high-quality empirical studies yet conducted on this subject, no consistent and unifying account of the psychological underpinning of ASD symptoms emerged.

The results of this study offer further evidence for what is now termed the “heterogeneity” of autism or the “fractionable autism triad” (Happé & Ronald, 2008). In their paper, Happé and Ronald summarise evidence that the three domains of autism (as defined in DSM-IV) appear to have independent causes at the genetic, neural and cognitive levels – what they describe as “fractionable” causes. They go on to say:

“The suggestion that the different aspects of the ASD triad have fractionable causes... is sometimes taken as an attack on the validity of the diagnosis of autism... However, it is quite compatible to assert... that the resulting mix has a special quality, distinct prognosis and response to intervention, and is therefore worthy of a distinct diagnostic label” (2008, p299).

Despite this, they do not say anything more about what this “special quality” actually is, and in view of the substantial changes in the defining characteristics of autism over many years this is clearly very difficult to do. There is also a question of whether autism does actually have a distinct prognosis in the sense of distinguishing it from other conditions (I return to this question below). Consequently, the heterogeneity of autism, coupled with the lack of any consistent and replicated evidence for a unifying cognitive account of the symptoms of the condition (or explication of its “special quality”), raise significant doubts about whether autism or ASD can represent a natural kind in psychology. I therefore now examine this issue in the context of natural kinds in science.

Is autism a natural kind?

Before discussing autism here, it is first necessary to say a little about natural kinds in science. Progress in science typically depends upon the identification of kinds of things that are presumed to exist in nature. The successful identification of such kinds enables the relevant phenomena to be organised into meaningful schemes of

classification. Natural kinds can provide the basis for explanations of salient phenomena and support reliable inductions and predictions about future phenomena in which the kinds may feature. These kinds are classes of objects, individuals or entities which share some set of causally linked properties which can account for their explanatory power. The paradigmatic examples of natural kinds are the chemical elements, such as sodium and chlorine. Many of the chemical properties of the individual elements are explicable in terms of their atomic number and structure. Biological species are also frequently cited as examples of natural kinds.

There are various accounts of natural kinds in the philosophy of science literature. Those that are applicable to biology, medical science, and other special sciences (including psychology) involve the notion of a clustering of relevant properties which together define the kind, without it being necessary for all such properties being present in every instance of the kind. A particularly influential account is given by Richard Boyd (1991, 1999) who emphasises that natural kinds must accommodate to the causal structure of the world and that the cluster of properties in the kind are held together by sets of “homeostatic causal mechanisms”. A similar, but more general, account of natural kinds as property clusters in causal networks is provided by Muhammad Ali Khalidi (2013) in which the defining properties of the kind are linked by causal processes in some manner. What these accounts have in common is their emphasis on causal processes as integral to the identification of natural kinds. As such, they provide an account of kinds which is applicable across all the sciences and which reflects the central epistemic endeavour in science generally – i.e. to uncover the explanatory causal processes for salient phenomena. This contrasts with rather weaker accounts of natural kinds (e.g. Chakravartty, 2007; Dupré, 1993) which accept clusters of properties as defining the kind without the additional criterion of causal linkages amongst these properties. However, we normally expect natural kinds to have significant explanatory value in science and to reflect the causal processes underpinning their explanatory function.

Many physical diseases can be regarded as natural kinds in virtue of their explanatory and predictive functions (e.g. Dragulinescu, 2010; Williams, 2011). This applies to infectious diseases as well as more complex diseases caused by, for example, autoimmune disorders and genetic abnormalities. In such cases, the disease kinds can be understood as clusters of characteristic signs and symptoms linked by complex causal chains, consistent with the accounts given by Boyd and Khalidi.

Specifically, the identification of a diagnosable disease is important for understanding the causal processes underlying the patient's symptoms and for grounding predictions about the likely prognosis. Another way of putting this is to say that medical scientists generally expect disease categories to have good discriminant and predictive validity.

How do these considerations relate to autism? As currently understood, autism looks like a term that covers a range of psychological features and as such it could be seen as a property cluster kind. However, if it is to constitute a natural kind on either Boyd's or Khalidi's account, the properties (symptoms) by which it is defined must be shown to be causally linked in some way and it must also be able to ground inductions and predictions about its consequences (e.g. about effective treatments, if any, and prognosis). In all these respects, autism fails as a diagnostic category with explanatory value.

First, a diagnosis of autism conveys no information about the aetiology of the condition in the particular individual receiving the diagnosis. More generally, there is no known causal association amongst the range of symptoms, the conjunction of which are said to constitute autism.

Second, the diagnosis carries no implications for specific interventions, except where dedicated services for people with an autism diagnosis (e.g. in schools or the community) have been made available following decisions by service commissioners. In such cases, the implications for intervention arise because of stipulative local policies, rather than from research studies demonstrating effective and empirically validated treatments with that particular group. In the case of people with autism and intellectual disabilities, there are no appropriate and effective interventions which are not equally effective for other people with intellectual disabilities (Bromley et al., 2012; Collins, 2016). In such cases, the choice of intervention should be based on a detailed analysis of the individual's specific abilities and needs, rather than on an apparently irrelevant diagnosis.

Third, the prognostic implications of an autism diagnosis are rather unclear, with highly variable outcomes in adulthood for those diagnosed in childhood (Helt et al., 2008; Levy & Perry, 2011; Magiati et al., 2014). Whilst those children receiving the diagnosis can be expected to have difficulties with social skills and independent functioning in adult life, it is not clear how much these result from autism per se rather than from intellectual disability. Of course, many people with an autism diagnosis do not have an intellectual disability, but in these cases the adult outcomes

appear to be rather more variable, with some individuals able to live independent and productive lives and form intimate relationships. Reviews of research in this area tend to agree that early childhood IQ and language level are important predictors of outcome in higher functioning individuals (Helt et al., 2008; Levy & Perry, 2011; Magiati et al., 2014). Generally studies of long term outcome depend upon retrospective data regarding diagnoses made in childhood and may therefore depend upon initial assessments of variable quality. However, a recently published prospective study, in which participants were assessed at ages 2, 3, and 19, again found that IQ scores in early childhood strongly predict later outcomes for children with an ASD diagnosis (Anderson et al., 2014). The authors also report that, by the age of 19, 9% of youths no longer had a clinical diagnosis and an additional 28%, whilst retaining features of ASD, nevertheless had much improved social and cognitive functioning.

Interpreting the findings from outcome studies, which have very variable methodologies and sample sizes, is inevitably difficult and consequently it might be argued that a diagnosis of autism does have significant predictive value. However, it remains the case that disentangling the predictive validity of an ASD diagnosis from that of intellectual disability is very difficult in many cases. Moreover, because the category of “social-communication disorder” was only introduced in DSM-5, there are no long-term outcome studies comparing the predictive validity of this category with ASD, but it is not immediately clear why these should differ in any substantial way.

In summary, autism does not seem to be a concept which strongly reflects the causal structure of the world due to its lack of explanatory significance and its very limited predictive validity.

Conclusions

The accounts of natural kinds given by Boyd and Khalidi, which emphasise the role of causal processes in identifying kinds in science, are comprehensive accounts applicable across all the sciences. In particular, they accord well with many disease concepts in medical science, including infectious, autoimmune, and neurological diseases amongst others, where we need to know about causal processes. However, on current evidence, the concept of ASD does not look like any kind of distinct and diagnosable disease and it does not appear to represent a natural kind of psychological

disorder. Whilst future research might change this picture, there is currently no known genetic or neurological explanation for it, despite huge international research efforts over many years, and it has so far proved impossible to describe in any clear terms what it is that people with an ASD diagnosis share, other than the diagnosis itself. The concept itself does not appear to do any explanatory work and offers little by way of clear predictions about the prognosis for those diagnosed beyond what can be predicted from an individual's intellectual and language levels and specific social-cognitive deficits. This is the case regardless of whether we are referring to "autism" or "ASD".

Given the heterogeneity of autism and its poor scientific value, it surely falls to those psychologists involved in autism diagnostic services to try to make clear what exactly they think they are diagnosing. Since any intervention plan for an individual with identified social-cognitive impairments needs to be based upon their specific profile of strengths and needs, a truly individualised formulation and treatment plan could presumably dispense with such an apparently empty diagnosis.

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