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How Should Applied Psychologists Conceptualise an Autism Spectrum Disorder Diagnosis and its Predictive Validity?

Abstract

Aim: Many educational and clinical psychologists are involved in autism diagnosis services, yet there is no clarity about the nature of the disorder underlying what they believe they are diagnosing. This paper therefore aims to examine some typical assumptions about the implications of an autism spectrum disorder (ASD) diagnosis.

Rationale: This conceptual review questions whether ASD represents a distinct kind of disorder, in view of the substantial changes in definitions of autism over the past 50 years. This is addressed from the perspective of philosophy of science by considering whether ASD constitutes a natural kind with explanatory and predictive value.

Findings: Research studies have failed to identify any distinct cognitive abnormality which uniquely characterises autism. There are no interventions specifically for people with an ASD diagnosis which, when available, are not also effective for those with other conditions. Outcomes for those with the diagnosis may be more varied than hitherto supposed and may depend on other variables apart from the diagnosis itself.

Limitations: It is impossible to cover all relevant research for a fully comprehensive overview of the field in a paper of this length. However, most of the research and review papers cited are of recent date.

Conclusions: It is difficult to see ASD as a distinct natural kind of psychological disorder. The outlook for those diagnosed is often hard to predict. Psychologists therefore need to be cautious about the manner in which they communicate diagnoses to avoid overly gloomy prognostications.

Key words: Autism, ASD, Diagnosis, Natural kinds, Validity

Working under pressure in busy autism diagnosis services, professionals such as educational and clinical psychologists may have little time to reflect on the nature of the process in which they are involved. Specifically, they may fail to reflect on the enormous power they exercise over the children and young people they diagnose. The nature of this power is evident in the consequences for access to additional support

services reserved for those children with an autism spectrum disorder (ASD) diagnosis. Professional power is also evident in the labelling effects of a diagnosis: it has a long term impact on the way in which those concerned are perceived by others and indeed on their own narratives about themselves. This is apparent also in its capacity to generate strong reactions of shock, confusion and grief in parents when they are informed by professionals of their child's diagnosis – see Nadesan (2005) and Runswick-Cole (2016) for accounts of their own personal reactions on receiving such news.

This might seem an unremarkable situation if there were a clear understanding about the nature of the disorder being identified. However, despite decades of research, no such stable understanding about the nature of autism has yet been reached. Indeed such consensus as does exist centres around the heterogeneity of autistic features and symptoms at the genetic, neurological, and cognitive levels (e.g. Brunsdon & Happé, 2014; Constantino & Charman, 2016; Rutter, 2013). Despite this state of affairs, there is a temptation for applied psychologists and other professionals to take for granted the validity of the condition they believe they are identifying when they diagnose a child with autism. They frequently appear to be guided by the assumption that autism represents some distinct kind of disorder characterised by an underlying abnormality which has predictive validity and is shared by all those with the diagnosis, despite the large overt variability in symptoms displayed by those affected. In other words, the assumption appears to be that autism is a natural kind of psychological disorder, something more than just a label, which can be confidently identified through the use of current diagnostic procedures.

In this conceptual review, I argue that such an assumption can reasonably be questioned. I first briefly review recent changes in diagnostic criteria for autism, now categorised as autism spectrum disorder in DSM-5 (American Psychiatric Association, 2013). Such changes may be expected to cast doubt on whether autism is a sufficiently stable concept to reflect a natural kind of disorder. I then discuss how natural kinds are typically understood by philosophers as having explanatory and predictive importance in science. In view of the fact that autism is treated in diagnostic manuals as a disorder of some kind, however broadly understood, I discuss how natural kinds can be understood in medical science. Applying these considerations to the case of autism, I argue that it fails to constitute a natural kind of medical or psychological disorder, and that this is equally the case for the latest

category of ASD in DSM-5. Neither the concept of autism nor that of ASD provide any explanatory account of the particular configuration of symptoms in any given individual, they give little indication of appropriate therapeutic interventions beyond what can be derived from a personalised formulation, and the outcome for those with the diagnosis is less deterministic than has hitherto been supposed. I conclude by suggesting that the indeterminacy concerning an ASD diagnosis needs to be reflected in the manner in which it is communicated to families.

Changes in diagnostic criteria

One of the more remarkable features of autism concerns the significant shifts in its conceptualisation and diagnostic criteria since Kanner's (1943) paper first described the condition. The most recent change occurred in 2013 when DSM-5 replaced DSM-IV (American Psychiatric Association, 1994). In the latter, autistic disorder was classified under the broader heading of pervasive developmental disorders (PDD) and identified with the "triad of impairments": i.e. (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 been replaced by the new category of "autism spectrum disorder" (ASD), which is now specified by 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. In addition, DSM-5 has introduced a new category of "social-communication disorder" (SCD). The single criterion for this is essentially the same as the first criterion for ASD – i.e. a persistent deficit in social cognition and interaction skills. Someone who displays such a deficit without any RRBIs would be diagnosed with SCD rather than ASD.

A significant change in DSM-5 has been the exclusion of Asperger syndrome. The criteria in DSM-IV failed to distinguish it sufficiently clearly from autistic disorder and particularly from high-functioning autism (Happé , 2011). What has persisted however is the concept of an "autism spectrum", and this concept, rather than "autism" simpliciter, now constitutes the named disorder in DSM-5. Nevertheless, these terms are frequently used interchangeably – to "have autism" is to "be on the autism spectrum".

These changes are the latest in a series of discontinuities in the way in which autism has been conceptualised over the past 70 years, which tend to be obscured by

the predominant belief in autism as an essentially stable entity (Verhoeff, 2013). These can be understood as occurring in three broad phases according to Verhoeff. The first of these, initiated by Kanner in 1943, treated autism as a disorder characterised by extreme aloneness and insistence on sameness. The term "autism" was coined in the early 1900s by the Swiss psychiatrist Eugen Bleuler to describe one of the primary symptoms of schizophrenia (Kanner, 1973). Kanner adopted this term, conceiving of autism as a form of childhood schizophrenia of early onset. In this phase, it was regarded as a distinct, albeit rare, psychiatric condition falling within the remit of psychiatry. The second, beginning in the 1960s and influenced by increasingly sophisticated empirical investigations, conceptualised autism more in terms of cognitive and perceptual abnormalities than as an affective disorder. The third, beginning around 1980 and coinciding with the changes introduced by DSM-III (American Psychiatric Association, 1980), shifted the principal emphasis to abnormalities of social cognition and communication. The previous association with childhood schizophrenia was abandoned (Frith, 2003) and the notion of a spectrum of autistic disability introduced. Nevertheless, the assumption that a biomedical explanation for the condition would ultimately emerge has continued to underpin the conception of autism as a distinct diagnosable disorder.

Rates of autism diagnosis began to increase rapidly as the boundaries of the diagnosis widened with the publication of DSM-III and subsequent revisions. Corresponding with this broadening of boundaries, there has been a dramatic increase in the prevalence of autism. Thus, Frith (2012) notes that the prevalence has risen from 0.04 to 1 per cent of the population over the past 50 years and one recent study puts the prevalence as high as 2.64 per cent (Kim et al., 2011).

Taken together, these successive changes raise the question of whether the latest conceptualisation of ASD in DSM-5 will be any more stable than preceding ones in earlier editions of the DSM.

Natural kinds in science

Progress in science normally requires the identification of categories which represent the kinds of entities presumed to exist in the world. This is sometimes described as "carving nature at its joints". To the extent that such categories succeed in indicating explanations and grounding predictions, they can typically be understood as representing natural kinds (Bird, 1998). In normal circumstances, of course, scientists

do not discuss their theorising in the terminology of natural kinds. However, the value of such an analysis is that it can clarify the extent to which the central concepts in a scientific discipline are actually doing explanatory work (Khalidi, 2013).

Paradigmatic examples of natural kinds include the chemical elements (e.g. hydrogen, gold) and biological species (e.g. tigers, ring-tailed lemurs). These are kinds which have some degree of stability and the power to ground scientific laws and reliable predictions. There is an extensive literature in philosophy of science detailing different accounts of natural kinds and their explanatory function. The simplest, 'essentialist', account (e.g. Ellis, 2001) holds that natural kinds are defined by an essential property as a necessary and sufficient condition for membership of the kind – e.g. the atomic number of a chemical element is its essence and explains many of its causal properties. However, a more complex account is needed for those sciences, particularly biological and biomedical sciences, where it is generally not possible to identify a single defining essence present in all members of a kind. Biological species, for example, are the product of evolutionary processes which of necessity depend upon variation in the genetic and other features of species members. In order to accommodate such kinds, alternative accounts explain natural kinds as characterised by clusters of properties, not all of which need be present in any specific member, but which collectively delineate the kind. Typically such accounts emphasise that the clusters of properties in natural kinds are linked by causal mechanisms which account for the role of the kind in scientific explanations (e.g. Boyd, 1991, 1999; Khalidi, 2013). What these accounts emphasise is that natural kinds should have explanatory and predictive value in science and reflect the causal structure of the world. Khalidi (2013) develops this further by demonstrating how natural kinds have causal effects in a large range of sciences, including medical and psychiatric science.

Several philosophers have described how medical diseases can be seen as natural kinds in virtue of their explanatory and predictive function. For example, Lange (2007) explains this as follows:

> …a diagnosis is intended to explain the patient's signs and symptoms. Therefore, a disease category must have 'validity'…which means that the disease must be a natural kind rather than an arbitrary category (2007, p.266, italics in original).

Lange illustrates this using the example of phenylketonuria (PKU), a genetic disease characterised by a deficiency in a specific enzyme necessary for metabolizing

phenylalanine (an amino acid). The symptoms of PKU are explained by the clustering of properties represented by the enzyme deficiency and the presence of these properties allows for reliable predictions about the appropriate dietary restrictions needed to avoid lifelong disabilities in the child concerned. Other examples of natural disease kinds have been described in the literature, including Graves disease (Dragulinescu, 2010) and rheumatoid arthritis (Williams, 2011). In both these cases, the symptoms are linked within complex causal networks which indicate the aetiology of the disease and enable predictions about its course.

What such examples demonstrate is that, despite the fact that diseases do not necessarily have a unique defining essence, they can be understood as causally linked clusters of symptoms produced by distinct aetiological processes. Therefore, if autism does represent a natural kind of disorder, we might expect it to constitute a natural kind on a similar basis, given the heterogeneity of its presenting symptoms. That is, we would expect there to be some set of causal relationships linking its different features and the concept itself would have significant explanatory and predictive value.

Is autism a natural kind?

Whilst one might argue that autism does not constitute a medical disease as generally understood, it is nevertheless seen as a disorder of some kind, something which can only be diagnosed by a professionally constituted child development team or mental health service. Indeed, it is this particular conception of diagnosis, without which autism cannot be identified in an individual, which is inextricably associated with medical and healthcare practice in general (Rosenberg, 2002). Moreover, the continuing search for genetic and neurological causes of the condition shows that a biological account is still being sought. This seems to be motivated by the enduring belief that autism constitutes a neuropsychological natural kind, an objective entity that will eventually be identified following further biomedical investigations (Verhoeff, 2012). Typically in medical science, diagnoses are expected to point to a causal explanation of the patient's symptoms when contextualised by a broader theoretical framework which accounts for the functions of the relevant causal mechanisms (Maung, 2017). However, no clarity about either a causal explanation or a general theoretical framework for autism has yet been attained. When the diagnosis fails to point to any such explanation and when its predictive value is limited, there

must be doubts about whether it represents a natural kind. I therefore discuss the prospects for a cognitive explanation of autism, before examining its predictive value for treatment interventions and longer-term outcomes.

Possibility of a cognitive explanation

A common view of autism is that it must be characterised by a distinctive cognitive abnormality or profile which is capable of explaining the very varied symptoms in different individuals (e.g. Frith, 2012). However, extensive research efforts have so far failed to reliably support any unifying cognitive account which encompasses everyone with the diagnosis and discriminates autistic from non-autistic individuals.

Various 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 and confirm its status as a natural kind. 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 et al., 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 findings which might explain all the symptoms of autism. Instead, as Brunsdon and Happé (2014) discuss in a recent review of many studies in this area, the picture is one of a heterogeneous collection of different cognitive abnormalities, typically with low correlations amongst them and with differing consequences for behavioural abnormalities.

This was demonstrated recently by a large population-based empirical study by Brunsdon et al. (2015). Recruiting adolescents with an ASD diagnosis from the UK Twins Early Development Study, the researchers studied 181 young people with ASD, comparing them with 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 atypical cognitive performance in a single domain only (5% in ToM; 8% in CC; and 6% in EF). Others had atypicalities covering at least two domains, and the largest group (32%) showed atypicalities in all three domains. The authors argue that this can therefore be seen as a characteristic pattern in adolescents with ASD. Nevertheless, over two thirds of the sample did not show this pattern. Moreover, no deficit in any of the areas examined was found in 9 per cent of the ASD sample, but this is not discussed further 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 per cent of the non-ASD co-twins and in 6 per cent of the controls, despite their lacking a diagnosis. Only 14 per cent of the control group showed no measurable abnormality. Thus, when tested using operationalized measures of the three hypothesised cognitive deficits in one of the largest empirical studies yet conducted on this subject, no single unifying account of the cognitive underpinning of ASD symptoms emerged. At the same time similar deficits were observed in some of the co-twins and controls, despite their lack of an ASD diagnosis.

It would seem therefore that studies of cognitive abnormalities in children with the diagnosis have not so far consistently supported a view of autism as a natural kind of disorder with a unifying cognitive explanation. Despite this, one might argue that autism still has significant predictive value. I therefore now discuss what predictive value might be attached to the concept of autism regarding its implications for treatment interventions and outcomes.

Treatment interventions

An ASD diagnosis, on its own, has no specific treatment implications distinct from those which might also be beneficial for other children. 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). For example, Collins describes several treatment packages, including the Treatment and Education of Autistic and Related Communication Handicapped Children programme (TEACCH), the Picture Exchange Communication System (PECS), and Applied Behaviour Analysis (ABA). All of these, however, are equally applicable to other people with intellectual disabilities. Moreover, ABA is based upon the use of functional analysis in planning interventions for challenging behaviour, and such interventions have no specific applicability to people with an ASD diagnosis. In such cases, Collins argues, the choice of intervention needs to be based on a detailed analysis of the individual's specific abilities and needs, rather than on a diagnosis which appears to add nothing to the treatment plan.

Howlin et al. (2009) seem to reflect this view in their review of early intensive behavioural interventions (EIBI) for young children, although they stop short of saying that the diagnosis is irrelevant to treatment planning. They conclude that such interventions work well for some, but not all, children with ASD, and attribute this variable response to the heterogeneity of cognitive and behavioural profiles covered by the diagnosis. They argue, therefore, that treatment needs to be based on the particular skills and deficit profile of the individual child. Similar conclusions are also reached by Reichow and Wolery (2009) in their synthesis of results of published EIBI studies.

There is general agreement in the literature that EIBI programmes are helpful for children with ASD. Thus, Reichow (2012) in a survey of five meta-analyses of EIBI studies concluded that many children, though not all, showed improvement in IQ scores and, to a lesser extent, in adaptive behaviour following intervention. Typically however, these programmes are explicitly intended for young children with an ASD diagnosis and may not be offered to those with other forms of developmental disability. Therefore, it is not clear why EIBI programmes would not be equally effective for other children in need of developmental support if these were offered.

Reflecting on the variability of response to treatment, Constantino and Charman (2016) comment that further discoveries about the aetiology of autism will likely lead to more personalised therapies, rather than standardised packages, in the future. It is unclear therefore what role the diagnosis itself, as opposed to individual behavioural

profiles, should play in treatment planning, other than to provide access to interventions which service commissioners and providers reserve for those with a diagnosis.

Outcomes

Autism has conventionally been regarded as a lifelong condition. Where individuals have been noted to improve their social functioning to the point that they no longer meet criteria for a diagnosis, this might typically be explained in terms of autistic symptoms becoming "masked" or the individual having gained "compensatory skills". By the use of such ad hoc explanations, the view of autism as necessarily persisting for life could be maintained. Some support for this view comes from prospective studies of children and reveal significant stability of the diagnosis during childhood (Constantino & Charman, 2016) – e.g. Lord et al. (2006) followed up children first diagnosed with autism at 2 years of age and found nearly all of these retained the diagnosis at 9 years.

However, despite this stability of diagnosis in childhood, the longer-term prognostic implications of an autism diagnosis are rather unclear, with quite variable outcomes in adulthood for those diagnosed in childhood (Helt et al., 2008; Levy & Perry, 2011; Magiati et al., 2014). For example, Helt et al. (2008) in their review of outcome studies report that between 3% and 25% of children appear to lose their ASD diagnosis by adulthood. Whilst many of 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 as distinct from other learning difficulties or 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). One exception was a study by Helles et al. (2015) of 100 males with a diagnosis of Asperger syndrome for whom the outcome was not predicted by early IQ and language levels. Even so, there was an increase over time in the numbers no longer meeting criteria for a diagnosis. Psychiatric co-morbidity also appears to be a predictor of poorer long-term outcomes, as indicated by a recent study of 50 adult

males given a diagnosis of Asperger syndrome in childhood and followed up for periods of between 13 and 26 years (Gillberg et al., 2016). The results showed that those individuals who no longer met the criteria for the diagnosis in adulthood were also generally free of psychiatric symptoms.

Generally, studies of long term outcome have been based on retrospective data regarding diagnoses made in childhood and may therefore depend upon initial assessments of uncertain and 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. The outcome which was more reliably predicted at 19 years was intellectual disability, which was predicted by IQ scores at the earlier ages.

Interpreting the findings from outcome studies, which have very variable methodologies and sample sizes, tends to be difficult. Furthermore, space limitations prevent a comprehensive review of all the literature in this area and consequently it might still be argued that an ASD diagnosis does have some predictive value. However, it seems clear from the recently published empirical and review papers cited here that distinguishing the predictive and discriminative validity of an ASD diagnosis from that of other conditions is difficult in many cases. Moreover, because the category of social-communication disorder (SCD) was only introduced in DSM-5, there are no long-term outcome studies comparing the predictive validity of this category with ASD, and indeed little is known in general about how SCD might differ in its effects from ASD (Swineford et al., 2014). However, it is not immediately clear why long-term outcomes for SCD should differ in any substantial way, given that the main difference between it and ASD is the absence of RRBI as a diagnostic criterion.

To summarise, neither autism nor ASD appear to have much value as explanatory concepts in science. They do not explain the pattern of symptoms in individual cases, since the aetiology is unknown in most cases. In addition, the predictive and discriminative validity of ASD is very limited, since decisions about treatment and predictions about outcome are determined at least as much by other factors concerning the individual as by the diagnosis itself. Moreover, the fact that autism has undergone several significant changes in its conceptualisation since its

introduction by Kanner suggests that there is no reason to assume that its current description in DSM-5 will endure any longer than previous versions. Indeed, some autism researchers now expect the diagnosis of autism to undergo further evolution in response to new research (e.g. Constantino & Charman, 2016; Volkmar & McPartland, 2016). It is difficult therefore to see how autism or ASD can be conceptualised as a natural kind of psychological disorder.

Conclusions

The question considered here is whether autism is best understood as a qualitatively distinct categorical disorder or a broader cluster of social-cognitive abnormalities indicating only dimensional differences between people with the diagnosis and those without it. However, despite the recent DSM revisions, many autism researchers still appear to regard autism as something qualitatively distinct. Thus, Frith (2012) states that there is something different about the autistic mind, whilst acknowledging that the enormous amount of research over the last 30 years has failed to clarify what this might be. In contrast, Volkmar and McPartland now conjecture: 'It might be time to move beyond diagnostic categories based at the syndrome level towards new, more descriptive, and broader conceptualisations of social-learning differences' (2016, p.238). This suggests a far from settled position, not just about the nature and aetiology of the condition, but also about whether it constitutes a distinct disorder at all.

The implications of this for psychologists involved in autism diagnosis services ought to be a matter of concern. It is difficult to avoid the assumption, when making a diagnostic judgment regarding whether the child concerned has autism, that there is some distinct entity which the child 'has'. However if, as argued above, autism is not a natural kind of psychological disorder, there must be real doubt that it corresponds with any kind of distinct entity. There can be no obvious reason to suppose that one individual with the diagnosis necessarily has the same kind of disorder as another with the same diagnosis, nor that the implications for intervention and longer term outlook are similar. In other words, one can reasonably ask whether a diagnosis of autism constitutes anything more than just a label.

It may be very difficult for psychologists currently to escape the demands of contributing to autism diagnosis teams, because of irresistible pressures from service commissioners and the public. Nevertheless, this should not prevent them thinking

critically about what kind of entity they think they are diagnosing. Such a critical approach can influence the manner in which a diagnosis is communicated to children and parents, so as to avoid implying a gloomy prognosis that might accompany the label and instead aim for a nuanced analysis of the various other factors that are at least as important in influencing the longer-term outlook. As Constantino and Charman state: '...over-prognostication carries with it the potential to do real harm, and predictions about any child's life prospects are best kept open, with an appropriate emphasis on what is possible, and honest recognition of the limits of what is known' (2016, p.288). As noted, the experience for parents of receiving their child's diagnosis can be very traumatic for them. The indeterminacy of an ASD diagnosis, regarding both the nature of the condition and its longer-term implications, ought therefore to be reflected in the manner in which the diagnosis is communicated to those concerned.

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