

The coherence of autism

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Autism
2014, Vol 18(1) 6–16
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sagepub.co.uk/journalsPermissions.nav
DOI: 10.1177/1362361313497538
aut.sagepub.com



Abstract

There is a growing body of opinion that we should view autism as fractionable into different, largely independent sets of clinical features. The alternative view is that autism is a coherent syndrome in which principal features of the disorder stand in intimate developmental relationship with each other. Studies of congenitally blind children offer support for the latter position and suggest that a source of coherence in autism is restriction in certain forms of perceptually dependent social experience.

Keywords

Autism, blindness, coherence, fractionation, identification, intersubjectivity, syndrome

In some quarters, it is becoming accepted wisdom that autism is not coherent as a syndrome. Instead of comprising a close-knit grouping of clinical features that regularly co-occur, as many had thought, autism is ‘fractionable’ into relatively independent components. Sure enough, these components sometimes appear together, and when they do, affected individuals satisfy current diagnostic criteria for autism or autism spectrum disorder (ASD). But so, too, they often appear in isolation from one another. Evidence from family and twin studies might be taken to suggest that the components are distinct in heritability.

The alternative view is that far from being independent, the clinical features of autism are deeply interconnected. The connections do not have to be of one kind – for instance, two clinical features might reflect a common underlying dysfunction, or one might express a compensatory adjustment in relation to the other – nor do they need to be so strong that the presence of one clinical feature implies that related features always co-occur. The argument for coherence rests on whether (a) the prevalence and (b) the qualities of any given form of behaviour may be influenced by pathogenic and pathoplastic factors that are either shared with, or arise directly from, other features of the syndrome.

The coherence view does not entail that one accepts a crude ‘thing’ version of autism. It is commonplace to forget that autism is neither more nor less than a syndrome, that is, a constellation of clinical features that tend to appear together (Kanner, 1943). For example, it is easy to fall into the trap of thinking that autism *is* a ‘central’ neurological disorder (Perez-Pereira and Conti-Ramsden, 2005) or ‘*is based on a disorder of information processing*’ (Brambling, 2011: 1596, my italics). Such a stance would need to be

justified by empirical evidence that central brain-based or information-processing dysfunction plays a causative role in *all* cases of autism, and/or would imply that those with the syndrome who fail to fulfil certain criteria for neurological or information-processing dysfunction should not qualify for the diagnosis. One aim of this article is to give reasons why it would be inappropriate to redefine autism according to such criteria, even if they are applicable to many cases, just as it would be misguided to reconceptualise autism as a loose grouping of clinical features. Coherence in the clinically defined syndrome points to otherwise obscure continuities as well as discontinuities in typical as well as atypical mental development. It prompts us to explain how and why these *particular* features cluster together (to the extent that they do) and are mutually constituted (to the extent that they are).

What is to count as ‘Coherent’?

What is to count as ‘coherent’? Consider the issue of consistency in the clinical picture. One option is to take the position that in order for a syndrome to be coherent as a syndrome, all the features that are said to characterise the syndrome need to be present in all or nearly all individuals who are to receive the diagnosis. Yet this is a very strict criterion. For example, in borderline personality disorder,

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another psychiatric syndrome, individuals qualify for the diagnosis if they manifest a sufficient number of overlapping clinical features that are diverse in quality. When present, each of these features seems to be related to (and arguably, indirect expressions of) a central problem in the person's intense and conflictual interpersonal relations. So, even though a given feature may not be present in any given individual with borderline personality disorder, there is something to be explained in accounting for its presence (when it *is* present) alongside other elements of the syndrome. It belongs in the family of features, as it were, even if it is not always present in a given family picture.

There is not a single way to construe coherence. From a diagnostic viewpoint, there are grounds for suggesting that the syndrome of autism is not sufficiently consistent in presentation to *require* that an individual shows repetitive and ritualistic behaviour (RRB; Mandy and Skuse, 2008). The danger would be that without further justification, this species of claim about incoherence might morph into another. Once the frame of reference alters – for instance, to a concern with the pathogenesis of RRBs when they *do* occur as part of the clinical picture – then one should not presume this is independent of other features of autism which might, for instance, predispose to or alter the expression of RRBs. A syndrome can be fractionable in one respect, yet coherent in another.

What is to count as 'Explanation'?

One of the arguments for dividing up subgroupings of clinical features pivots on whether separate explanatory accounts are required for each. It is in this context that Happé et al. (2006: 1218) suggest that it is 'time to give up on a single explanation for autism'.

One meaning of 'explain' provided by the Oxford English Dictionary is 'to unfold; to make plain or intelligible'. What would count as a single explanation for autism is far from clear. This might well be one that invokes *different* explanatory levels, with different causative factors on each level, while remaining an integrated whole. We should not demand nor expect that a given level of explanation should encompass everything about autism, nor that a single explanation should be simple.

Consider an example from the psychological realm that is relevant for subsequent parts of this article. Wing and Wing (1971) suggested that autism might result from multiple perceptual and cognitive deficits. Such deficits would need not only explanation at lower levels (and Wing, 1969, studied a variety of perceptual handicaps with different causes), but also feed into an account of how those deficits lead to higher levels of dysfunction – perhaps, say, interfering with the development of executive function, or impoverishing the perception of people's non-verbal communication. Then the question would arise, how far do *these* factors explain the clinical picture, or do we need

further levels of explanation before the overall picture is 'unfolded' and has become intelligible?

A further complexity is that sometimes, but not always, different *levels* of explanation implicate different *kinds* of explanation, for instance, genetic, biochemical, neurological or psychological. In tracing the developmental psychopathology of autism, we may need to shift *between* (say) neurologically and psychologically based accounts. At one stage in a child's development, a given pattern of clinical features might reflect the several effects of a particular form of brain dysfunction, whereas at different stage, some part of the clinical profile (*and* some features of atypical brain activity) might be attributable to psychological impairments arising from restrictions in experience due to earlier perceptual, cognitive or social-affective deficits. We cannot presume which factors are in the driving seat nor which kind of explanation is needed to account for associations or dissociations among clinical features.

Finally, there are several criteria that may be applied when judging whether an explanation is basic to understanding autism. This depends on the question: 'Basic for what?' One view is that basic impairments are universally present in, and specific to, a condition. The picture is complicated insofar as a basic psychological impairment may have a range of different (even more basic?) causes. From a complementary perspective, a factor that is not basic for autism as a whole may be necessary for providing an adequate account of some essential part(s) of the condition.

Of course, we are concerned with how much a given account explains. For instance, to how many children with autism does it apply, how many of the clinical features does it encompass and how far does it provide coherent links between lower-order causes/levels (whether genetic, biochemical, neurological or psychological in kind) and superordinate levels of explanation? Beyond this, there is the further question of whether a particular account is *necessary* to make the syndrome intelligible. Some levels of explanation may prove useful for a while, but become redundant when a better theory becomes available. Others are likely to prove indispensable.

The upshot is that even within a single theoretical account, there might be basic, coherence-inducing abnormalities at different levels of explanation. Special importance would attach to any abnormality that gives overall coherence to autism as a syndrome.

The case for radical incoherence

Background

Prompted by Wing's (1969) study of different groups of handicapped children including those with language disorders and severe perceptual abnormalities, Wing and Wing (1971) concluded that children with autism appear to suffer from multiple impairments that vary in severity, and postulated that 'a combination of language, perceptual, motor,

and autonomic impairments underlies autistic behavior' (p. 256). They stressed that this combination could have single or multiple aetiology, that isolated fragments of the full clinical picture frequently occur, and that there is a need for 'detailed and systematic observation of behavior of autistic children' (p. 256). Goodman (1989: 409) suggested that 'autism may involve multiple functional deficits due to multiple coexistent neurological deficits', perhaps involving deficits in language skills, on the one hand, and in social relatedness, on the other. In more recent times, leading proponents of the 'fractionable' view of autism have been Happé, Ronald, and colleagues, and from this point, I shall focus on these researchers' comprehensive approach to the topic.

An article by Happé et al. (2006) begins with an abstract in which the authors write: 'We argue that there will be no single (genetic or cognitive) cause for the diverse symptoms defining autism ... At the cognitive level, too, attempts at a single explanation for the symptoms of autism have failed' (p. 1218). Later on, they state that 'it is time to give up on the search for a monolithic cause or explanation for the three core aspects of autism, at the genetic, neural and cognitive levels' (p. 1219).

It is difficult to see why the same or even similar considerations should apply to accounts that might posit a single (e.g. psychological) cause, on the one hand, and a single explanation, on the other (also Goodman, 1989; Wing and Wing, 1971). To repeat, it is perfectly plausible that one could offer a single explanation with multiple levels and alternative kinds of causation. Any account of pathogenesis is going to describe either one or several trajectories in which at one stage what is caused, at the next developmental stage becomes a cause of further repercussions. The crux is whether there are aetiological and/or pathogenic mechanisms that give coherence to autism as a syndrome.

The principal points made by Happé et al. (2006), and elaborated further by Happé and Ronald (2008), may be grouped under three headings, as follows.

Epidemiology and genetics

The evidence. Although there is a complex and somewhat conflicting body of epidemiological evidence (well reviewed by Happé and Ronald, 2008), here I shall focus on an investigation summarised by Happé et al. (2006). These researchers report a population-based study of what they call 'autistic-like traits' among over 3000 twin pairs assessed between the ages of 7 and 9 years. They write, 'We can ask, for example, whether a child joins in playing games with other children easily, can keep a two-way conversation going or likes to do things over and over again in the same way all the time' (and see Ronald et al., 2005, for further examples of questionnaire items comprising descriptions of children, such as 'considerate of other people's feelings', 'gets on better with adults than with other children', and 'is afraid in social situations'). The respondents were teachers

and parents, and each item (10 on a social scale and 6 on a non-social scale) was rated 'not true', 'somewhat true' and 'certainly true'.

The authors (Happé et al., 2006: 1218) summarised their principal results as follows:

The distribution of such traits supports a smooth continuum (at least on the behavioural level) between individuals meeting diagnostic criteria for ASD and individuals in the general population.

Of the modest-to-low correlations between autistic-like behavioural traits in the three core areas, they wrote,

Somewhat to our surprise, even social and communication impairments, which are often seen as almost indistinguishable in real life and have been suggested to result from a single cognitive deficit, were only modestly related (with correlations (r) in the range of 0.2 to 0.4).

Indeed, this is surprising, given that from a conceptual as well as empirical standpoint, the interface between social interaction and communication is so porous. In addition, they report that 'a considerable number of children showed isolated difficulties in only one area of the autistic triad' (p. 1218).

A second line of evidence concerns family studies of individuals with ASD, and again a quotation from Happé et al. (2006) serves as a succinct summary:

Family and twin studies have shown that it is not only autism itself that is heritable, but that relatives show increased rates of the 'broader autism phenotype', which refers to subclinical manifestations of all or part of the triad of autistic features. Importantly, some relatives show only isolated traits, for example communication difficulties without social impairment or rigidity. This suggests that the genes that contribute to autism segregate among relatives and have distinct influences on the different parts of the phenotype. (p. 1219)

How far do above lines of evidence warrant the conclusion that autism is fractionable?

Methodological considerations. In the case of the twin studies by Happé and colleagues, a critical question is this: What do the responses of parents or teachers to this kind of questionnaire mean? Is it justified to consider the measure to be one of 'autistic-like traits', or, as Ronald et al. (2005: 446) state when referring to high scores on the 10 items of the social scale, 'autistic behaviours'? Imagine you were responding to the questions cited above, with reference to a 7- to 9-year-old in your own family. For instance, suppose you considered it to be 'certainly true' that he/she is afraid in social situations' – would you be capturing and conveying something that relates to autism *at all*, when the child might have anxieties that differ not only in quality from those experienced by children with autism, but also in their

underlying causes? There are many, many reasons why a parent or teacher might classify a child in one or several of the categories that are supposed to correspond with putative ‘autistic-like traits’.

In particular, given additional concerns about the modest internal consistency and reliability of the measures within the domains studied in this research – concluding that X does not correlate with Y is hazardous if X does not correlate well with itself, for example (see critique by Mandy and Skuse, 2008) – it is questionable we learn much about autism from such data. The fact that there is not a bimodal distribution in the numbers that emerge may be telling us as much about the non-specificity of the measures as about the phenomena they are supposed to measure. The potential (albeit limited) strengths of this methodology in yielding conclusions about the *severity* of reported impairments – and the authors stress that isolated difficulties ‘appeared to be at a level of *severity* comparable to that found in children with diagnosed ASD in our sample’ (p. 1218 of Happé et al., 2006, author’s italics) – is offset by its insensitivity to the *qualities* of the children’s clinical features.

When we turn to consider the broader phenotype in relatives of probands with autism, that is, relatives among whom potentially separate expressions of a propensity to autism may be discerned, the ‘autistic-like’ question crops up in a different way. But first, it is important to acknowledge that the findings here may challenge an extreme version of the coherence view. If, say, language impairments appear in relatives of probands with a diagnosis of ASD and these relatives show no other signs of psychological disorder, then clearly *these* language impairments are unlikely to result from other features of autism-related disorder.

There are two issues to be settled before this conclusion is justified. First, do the observed forms of impairment in each domain come *close enough* to those seen in autism, or do seeming similarities belie essential differences? Second, is it securely established that no other autism-related atypicalities are present in the relatives? If they were present in some form that eluded measurement, then clearly the independence of features would be open to question.

If doubts over these matters are dispelled, then how far should one accept the evidence as suggesting that features of autism fractionate? Answer: some distance, but by no means the whole way. At most, one could conclude that in some families, there may be independent genetic *contributions* to different features of the syndrome. This leaves considerable scope for additional pathogenic and pathoplastic influences to operate, contributing cause(s) of and giving shape to features of the syndrome as these occur together in probands. For example, mutual *interactions* among potentially separable communicative and cognitive/linguistic disabilities might influence both the qualities and degrees of (say) pragmatic language impairment and/or repetitive behaviour among individuals with the full syndrome. If this

were the case, then such interactive effects operating over the course of the children’s early development would constitute a potent source of coherence. In addition, it would remain to establish how far one should generalise any conclusions to all cases of autism, in some of whom (as I shall discuss later) similar forms of impairment in language, social relations and repetitive behaviour appear to arise from causes very different to the genetic factors posited to underlie deficits identified as fractionable.

Cognitive approaches

Happé et al. (2006) highlight what they see as ‘a failure to find a single cognitive account for the three core features of autism’ (p. 1219). Happé and Ronald (2008) refer to the ‘satisfactory working theories ... for the various different aspects of autism’, namely, those that posit a specific ‘theory of mind’ deficit, executive dysfunction and weak central coherence (p. 297).

It is open to question quite how satisfactory these theories are. Importantly, for the present article, theory of mind theorising has been criticised for neglect of the role of embodied/interactional relatedness in characterising the foundations for mental state understanding and communication (e.g. Hobson, 1990, 1991). Accounts of limitations in central coherence or executive dysfunction are helpful from a descriptive viewpoint, but without detailed specification of the source(s) and structure of these atypicalities as they apply to autism, one might question how far they render the pathogenesis of the syndrome intelligible in terms of processes that we understand better than the phenomena we are seeking to explain.

These matters become important in the light of the claim that the three satisfactory theoretical accounts map on to the three sets of dissociable features in autism. If an alternative *kind* of account of autism – one overlooked by investigators espousing fractionation – were to prove even more satisfactory, then this might be congruent with a quite different pattern of associations and dissociations among clinical features.

Neuroscience

Happé et al. (2006) take the view that neuroimaging studies offer support for the fractionable nature of autism. For instance, they note that imaging in healthy and ASD adult volunteers suggests that social cognition relies upon a specific network of brain regions including the medial frontal cortex, temporoparietal junction, superior temporal sulcus and temporal poles, whereas rigid and repetitive behaviour has been linked to caudate abnormality in ASD.

A single but critical consideration influences the weight to be accorded to current neurofunctional evidence in this context. Of course, in typically developing individuals well past infancy, different neural systems subservise different

domains of psychological functioning. Of course, too, such systems may be differentially affected among persons with autism. The problem is that as yet, little is known about the *development* of such specialised systems, and even less about their atypical development.

Brains develop in relation to the experience of the people whose brains they are (e.g. Karmiloff-Smith, 1998). There is good reason to think that from early in life, the experience of children with autism is severely atypical. It follows that whatever correlations between neural and psychological function or dysfunction may be uncovered among children, adolescents or adults with autism, we cannot know in which direction the causative arrows point. Has atypical *experience* at Time 1 (perhaps from dysfunction of brain system A, or perhaps from a variety of causes) led to atypical development in brain systems B and C at Time 2, or are primary dysfunctions in systems A, B and C responsible for the atypicalities we see at Time 2, or is some other set of interactions involved?

For instance, there is evidence that executive dysfunction is relatively late in onset among children with autism (e.g. Griffith et al., 1999). It is entirely plausible that this set of impairments (which incidentally, do *not* assume a profile characteristic of those cases on the basis of which the concept of 'executive dysfunction' was founded) might stem from disruption in other psychological functions and *result in* atypical functioning in those areas of the brain known to mediate planning, working memory and so on.

Therefore, atypicalities in brain function may reflect, rather than underlie, atypicalities in psychological functioning at any given point in time. Of course, the reverse may also be the case. In a developmental disorder of early onset, such as autism, we cannot decide the issue. Therefore, as presently constituted, research findings from neurofunctional studies appear to count neither for nor against a fractionation view of autism.

The case against radical incoherence

Epidemiology

As Happé and colleagues acknowledge, an important epidemiological study suggesting that autism *is* a syndrome was conducted by Wing and Gould (1979) in the former London borough of Camberwell. These researchers screened 35,000 children under the age of 15 years for the presence of at least one of the following items, regardless of level of intelligence: (a) absence or impairment of social interaction, especially with peers; (b) absence or impairment of the development of verbal and non-verbal language; and (c) repetitive, stereotyped activities of any kind. The one additional group included was that of ambulant children with severe intellectual disability, whatever their

pattern of behaviour and impairments. These criteria resulted in the selection of 132 children. Professional workers or parents involved with the children were interviewed with a structured schedule to assess the children's behavioural skills, and the children themselves were observed in the classroom or at home.

The overall prevalence of impairments in reciprocal social interaction was 21.2 per 10,000 of the population. All the children with social impairments had repetitive stereotyped behaviour, and almost all had absence or abnormalities of language and symbolic activities. This led Wing to talk about the 'autistic continuum' of cases that manifest the triad of impairments in social interaction, communication and imagination, usually associated with a repetitive, stereotyped pattern of activities.

In a respectful critique of this study, Happé and Ronald (2008) point out that Wing and Gould used a narrow definition of autism. The sample was selected from a psychiatric and mental retardation register, and so might have been skewed towards children with more severe and/or co-morbid conditions. What is true of children with limited intellectual capacities may not be true of more able children with autism (Mandy and Skuse, 2008). Still one needs to account for the coherence of the syndrome as revealed by a study notable for the thoroughness with which qualities of the children's impairments were assessed. So far, results from factor analytic studies (reviewed by Happé and Ronald, 2008; Mandy and Skuse, 2008) have not been decisive in this respect.

Clinical and theoretical coherence

There are no *a priori* principles for dividing up the psychological domain for the purposes of explaining typical or atypical development. As Goodman (1989) and Wing and Wing (1971) indicated, there is much to be gained from examining in detail whether the phenomenology and clinical features of autism, both within the syndrome and between autism and plausibly related conditions, appear to bear a close mutual relation. Such examination may uncover connections among features that had seemed disconnected. Having said this, the dangers of misjudging whether and in which respects features are similar – the spectre of phenomimicry (Bishop, 2010; Williams et al., 2008) – attends this process every step of the way.

In part, empirical studies of coherence are informed by, as well as informing, theoretical perspectives. I now turn to a theoretical approach that accounts for substantial coherence in the phenomena of autism in terms of the developmental implications of restricted *interpersonal* relations (note: this is *not* a psychogenic theory). Subsequently, I shall illustrate the potential value of this approach for interpreting findings from studies of congenitally blind children.

The hypothesis is as follows. A 'final common pathway' accounts for substantial coherence in the presentation of

autism. This final common pathway is psychological in kind (rather than genetic, biochemical, neurological, etc.). More than this, it needs to be characterised in terms that are intersubjective rather than individual because it concerns breakdown in a *system* of self-in-relation-to-other. If one considers causative factors in the *individual* children affected, then these causes are several in nature. Yet they converge in causing a distinctive, shared social-developmental handicap with a coherent set of developmental sequelae.

If this approach is valid, then one would expect *both* substantial homogeneity *and* underlying heterogeneity among people with autism. The homogeneity arises from the shared social-developmental handicap – in summary, limited engagement with other people’s engagement with a shared world – and its developmental implications. Much of the heterogeneity arises from additional, direct expressions of the *various* aetiological and pathogenic sources of the social impairment. Shortly, I shall illustrate this principle of similarity-with-difference among children with autism through the special case of those who are congenitally blind.

There is an additional source of heterogeneity among individual children with autism. As in the case of borderline personality disorder cited earlier, only some of a family of clinical features may be expressed in any one individual. For instance, some children with autism appear to be more adept than others in finding ways to compensate for limited role-taking abilities in their verbal communication. One implication for assessing coherence is that one needs to step back from an exclusive focus on the individual, and consider whether a *set* of theoretically interlinked clinical features cluster together and characterise a *group* of affected individuals, even when given individuals might differ in which particular subset of those features are manifest. If this seems paradoxical as an argument for coherence (one might ask, ‘How can a set of clinical features be coherent when one or more of those features might be missing?’), consider how the separable but coherently related symptoms of diabetes in the eye, heart or nervous system may or may not afflict a given individual with the disease. Importantly, however, that individual is vulnerable to the full set of physical complications, some of which may exist in subclinical form.

According to intersubjectivity theory and its more specific variant, Identification Theory (e.g. Hobson, 1993, 2002, 2007), among individuals with autism, there *is* a common underlying structure to atypicalities in non-verbal and affective communication, linguistic functioning, imitation, self-consciousness and other aspects of symbolic and social-cognitive functioning, including Theory of Mind. Shared developmental underpinnings in *specific* forms of restricted interpersonal experience mean that affected individuals are vulnerable to this coherent set of abnormalities. For instance, not only deficits in pragmatic language and

dialogue but also ‘concrete’ modes of thinking among individuals with autism appear to be part and parcel of limitations in individuals’ ability to register, engage with and adjust to another person’s perspective (e.g. Charney, 1981; Hobson, 2012; Hobson et al., 2012). From a developmental perspective, such role-taking appears to reflect something early in onset, namely a propensity to identify with another person’s bodily-expressed attitudes towards a shared, visually specified world. If movements in mental stance generated in the context of non-verbal communication *between* people can become movements in mental stance within an individual’s own mind – and if this is bound up with the achievement of self-reflective awareness and creative symbolic functioning, as Mead (1934) and Werner and Kaplan (1963/1984) suggested – then deficits in basic forms of interpersonal engagement in autism may have far-reaching cognitive-developmental implications (Hobson et al., 2006).

This Identification Theory is a ‘single’ theory of autism in two important senses, embodied in two related claims about what is basic to autism. The first claim is that any account of autism will need to include a level of explanation that invokes breakdown in the intersubjective system of self-in-relation-to-other. If one fails to include such a level, then important features of autism will go unexplained and/or will appear to be independent of one another. The second claim is that impairments in intersubjective engagement that include limitations in co-reference towards a shared world are universal to *all* cases of autism, early in onset, and constitute a major causative and/or shaping influence on the emergence of a range of other features of the disorder (also, for example, Mundy et al., 2009).

This theory is *not* ‘single’ in the sense that it explains everything about autism. As already argued, no single dysfunction *could* encompass everything relevant to causation. Diverse lower-order explanations, either domain-general or domain-specific, might account for the ways in which a child’s capacity for intersubjective engagement can be impaired. The approach allows for – indeed, anticipates that there are – *multiple* primary deficits across different children, at lower levels of psychological explanation. As I shall illustrate through the case of blind children, alternative psychological causes may operate from one case to another, and/or from one developmental stage to another. The causes may differ in kind (e.g. some might be neurological), and they will have their own distinctive effects *in addition* to those that prevent or disrupt critical forms of social experience. Importantly, therefore, the theoretical approach specifies what leads to coherence in the syndrome of autism, while at the same time indicating where one should expect heterogeneity in aetiology and pathogenesis.

Like most theories, this one has strengths and weaknesses. For instance, when it comes to explaining limitations in self-regulation and executive functioning (Hobson and Hobson, 2011), it relies heavily on the theoretical position of Vygotsky

(1978), who considered that the interiorisation of interpersonal functioning ‘applies equally to voluntary attention, to logical memory, and to the formation of concepts’ (pp. 56–57). As yet, there is little direct evidence for the suggestion that impairments in intersubjective engagement are responsible for weakness in top-down modulation of psychological function in autism, although there are intriguing pointers in this respect (Williams et al., 2012). So, too, there is limited evidence to support the idea that repetitive behaviour and/or insistence of sameness (Richler et al., 2010) arise from the absence of socially derived movements in thought and feeling, together with defensive channelling of motivation and interest according to what the individual can control and manage (also Baron-Cohen, 1989; Rodgers et al., 2012).

Most important for the present purposes, however, is that if one conceives of the final common pathway to autism as disorder in *interpersonal* relations, then this radically alters one’s perspective not only on mechanisms of causation but also on coherence among clinical features of the syndrome and sources of heterogeneity across affected individuals.

Atypical autism

Suppose there are circumstances in which children present with the syndrome of autism, but in which some of the usual causal factors are replaced by new ones? Such atypical forms of autism might help us to gain fresh purchase on sources of coherence and/or incoherence in the disorder. Once again, one would need to determine whether the atypical form of autism is similar enough to more typical forms of autism to justify comparisons and contrasts, especially given that the conditions predisposing to autism are going to distinguish atypical from typical cases.

Here, I shall focus on the case of autism among congenitally blind children. For the present purposes, the critical issues are as follows. First, does profound lack of vision from birth put a child at risk for developing the syndrome of autism? Second, do we find coherence in autism among blind children – and if so, why?

Early reports from clinicians working with blind children (e.g. Fraiberg and Adelson, 1977; Wills, 1979a, 1979b) have been complemented by more recent studies in suggesting that these children manifest a range of psychological abnormalities that resemble those seen in sighted children with autism. This is the case in relation to social-communicative functioning (Curson, 1979; Rowland, 1983; Urwin, 1983), creative symbolic play and language (Andersen et al., 1984; Bishop et al., 2005; Dunlea, 1989; Hobson and Bishop, 2003; Preisler, 1993; Rogers and Puchalski, 1984), stereotypies, mannerisms and ritualistic behaviour (Chess, 1971; Wills, 1979a, 1979b), and uneven profiles of cognitive abilities, including difficulties with abstract thinking (Tillman, 1967; Wills, 1981; also Elonen and Cain, 1964; Green and Schecter, 1957). Either the

whole or parts of the syndrome of autism have been reported for visually impaired children with specific medical diagnoses, for example, retrolental fibroplasia (Keeler, 1958; also Chase, 1972), maternal rubella (Chess, 1971) and Leber’s amaurosis (Rogers and Newhart-Larson, 1989).

Brown et al. (1997) conducted systematic comparisons between congenitally blind children and sighted children with autism. These investigators selected 24 children from six schools for the blind on the basis that they were aged between 3 and 9 years and had total or near-total blindness from birth and an absence of manifest neurological impairment. In all, 10 out of the 24 congenitally blind children satisfied *Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Text Revision (DSM-III-R)* criteria for autism (also Fraiberg, 1977). The diagnosis was not restricted to any particular medical diagnosis. The sex distribution in the overall sample was 11 males and 13 females, and among the 10 children diagnosed with autism there were 4 males and 6 females.

It seemed unlikely that the findings reflected ‘comorbidity’ between two potentially separable conditions, blindness on the one hand and autism on the other. Not only was the sex distribution atypical, but clinical features such as echolalia, poor imaginative play and stereotyped body movements were distributed across the blind children, with *and* without the full syndrome of autism. Blind children *without* autism have also been reported to have limitations in Theory of Mind reasoning (McAlpine and Moore, 1995; Minter et al., 1998; Pring et al., 1998). It appears that lack of vision can lead to restricted social role-taking across a number of functional domains. This restriction explains the coherence of the constellation of clinical features that find most vivid and comprehensive – but not exclusive – expression among children with the full syndrome of autism. Elements of the constellation sometimes appear in *relative* isolation, but insofar as they may represent alternative modes of presentation of the self-same role-taking difficulties and that arguably, the children are vulnerable to the full set of related features, this should *not* be taken as evidence for the fractionation hypothesis.

There remained a critical question: Are the clinical features seen in blind children with the full syndrome of autism more or less like those of sighted children with autism in the *qualities* of clinical presentation? Hobson et al. (1999) constituted a new group of closely matched sighted children with autism, to compare with nine of the blind children with autism. All the children in each group satisfied *DSM-III-R* criteria for autism. There was substantial overlap, but also subtle differences, on systematic observational measures. For instance, there was a relatively high proportion of blind children rated as having postural oddities and motor stereotypies, abnormal personal pronoun usage, and immediate echolalia, but a relatively low proportion of the blind were abnormal in the variety and depth of affect and modulation of affect.

Further evidence that (a) the source of the blind children's autism was atypical and (b) the syndrome was coherent came from an 8-year follow-up study of these same children (Hobson and Lee, 2010). Overall, 8 out of the 9 blind children now failed to meet formal diagnostic criteria for autism. However, the blind participants still displayed a range of clinical features such as echolalia, difficulties with personal pronouns, lack of awareness of the existence/feelings of others, poor imaginative play, and stereotyped body movements and/or restricted patterns of interest. There was substantial coherence to the clinical picture even when its seriousness had diminished. Marked impairments in specific forms of social relatedness, language, and restricted and stereotyped forms of behaviour were prevalent among affected children.

Why should this be? There are at least two parts to this question. First, why should children who (probably) are *not* genetically predisposed to develop the various features of autism, *nor* (probably) afflicted by sufficiently specific primary neurological impairment, develop a clinical picture so close to that of autism in sighted children? Second, among those congenitally blind children who present with only parts of the clinical picture, whether because they are not so impaired from the outset or because they have partially 'recovered' from autism, why do supposedly disparate features still seem to cohere in the sense that they all find expression within the group as a whole?

The studies of congenitally blind children by Hobson and colleagues were prompted by a set of hypotheses generated from intersubjectivity/Identification Theory already described. The investigators considered sighted children with autism to have a weak propensity to identify with others' attitudes for a range of constitutional ('biological') reasons. They reasoned that *because they lack sight*, those with congenital blindness also suffer severe impediments to joint attention, social referencing and other forms of mental co-orientation with others vis-à-vis a visually specified world. Congenitally blind children, too, are at a disadvantage in grasping how objects and events can be construed differently by different individuals. Blind children's atypicalities in language, thought and play reflect this difficulty, for instance through their diminished appreciation of speech roles (e.g. confusions between 'I' and 'you') and novel person-anchored meanings attributed to the materials of play.

The obstacles to sharing and taking new perspectives *through* other people are not insurmountable, as the relatively typical development of many blind children attests. Although blind children who develop autism may well be subject to additional constitutional and/or environmental disadvantages, it is plausible that many of their seemingly diverse clinical features are expressions of developmental sequelae to perceptually ground *intersubjective* handicaps. Restriction in experience of person-person-world co-ordination of perspectives is profoundly important for the development of blind and sighted children alike. In each

group, this level of explanation accounts for substantial coherence in the clinical picture of autism.

This returns us to the question of whether we should consider autism among blind children *as* autism, or instead describe the children as 'autistic-like' or showing 'quasi-autistic features' (Frith, 2003; Rutter et al., 1999). The problem with the latter formulation is that it leads us away from the diagnostic criteria for autism towards some other unspecified notion of 'true' autism. Sure enough, there are differences between congenitally blind children with autism and sighted children with autism. This is precisely what one would expect if lack of vision is a risk factor peculiar to the former group. But is there a principled reason for arguing that, if their condition fulfils current diagnostic criteria, their autism is not true autism? Perhaps, instead, one needs to see that there is an open question over the degree of heterogeneity among *all* children who present with the syndrome. This is a matter that has special importance when considering the potential for influencing affected individuals' developmental trajectories.

There is a related hazard attached to calling isolated or co-occurring clinical features 'autistic-like', whether these occur among blind or other children. This stance implies that autism is *the* reference point for understanding. A more balanced position would be this: insofar as certain clinical features are truly *common* to many congenitally blind children (as well as some children who have suffered especially severe privation: e.g. Rutter et al., 2007, also Livermore-Hardy et al., 2013) and those we classify as having autism, then these features should be considered in relation to pathogenic factors shared in the emergence and expression of the features. The latter perspective respects coherence among clinical features – a coherence that reflects underlying pathogenesis, that is, what is common in how the features *develop* in mutual relation to one another – while allowing variability among different individuals in aetiology, in preliminary phases of their developmental trajectory, in which features they manifest, and in how their natural history unfolds. After all, at follow-up, the blind children studied by Hobson and Lee (2010) showed some, but only some, of the clinical features of autism they had shown 8 years previously.

In summary, the evidence strongly suggests that there is more than surface similarity between clinical features of autism among sighted and congenitally blind children (also Hobson, 2005). The differences in some details of the clinical picture, the (probable) differences in sex ratio and the differences in natural history suggest that blindness per se may be acting as a risk factor. Although a matter requiring further study, it seems that other potential predisposing (e.g. genetic) factors probably play a lesser role in the genesis of the syndrome.

And yet, autism among congenitally blind children has coherence. It is not just that the qualities of nearly all the features of the syndrome are similar to those seen among

sighted children with autism. It is also that the *range* of features – social, communicative and repetitive and ritualistic – are represented. This coherence is in keeping with what had been anticipated on the basis of a theory focussed on the developmental implications of disruptions to the children's *interpersonal functioning* – a single but multi-level theory.

Conclusion

There is no simple answer to the question: Is the syndrome of autism coherent or fractionable? Yet this is a question well worth asking, whether one is concerned with conducting research (and wishing to constitute meaningful groups that have relevant kinds of within-group homogeneity), establishing diagnostic criteria, assessing genetic or other biological contributions to causation, developing psychological theories, tracing the natural history of affected children, or implementing intervention programmes. A lot is at stake. For instance, the fractionation view could encourage interventions that address fragments of the clinical picture, whereas the coherence view might lead one to focus instead on remediating a final common pathway (perhaps what happens between affected children and others) that leads to the syndrome being constituted as it is.

Therefore, we need to be clear in discriminating among the various ways in which autism may be fractionable on the one hand, and coherent on the other. Within the framework of genetics, for example, the evidence from the broader autism phenotype suggests that there is some fractionation of genetic risk for different features of the disorder. On the other hand, there are grounds for positing psychological factors that bear upon the development and clinical presentation of the *set* of atypicalities characteristic of autism. It is too soon to reject the idea that causative psychological factors shared among different clinical features, or mutual influences that give shape to the clinical phenomena, bring coherence to the picture of autism.

Acknowledgements

I give sincere thanks to Dr David Williams for his encouragement and detailed advice. I am grateful to the Center for Advanced Study in the Behavioral Sciences, Stanford, and the Tavistock and Portman NHS Foundation Trust, London, for supporting a sabbatical during which the article was written.

Funding

This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

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