Models of autism
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Abstract

There is a high degree of agreement about the core features of autism. On closer examination, however, this agreement is much less solidly based than it appears.

One key problematic issue is the assumption underpinning much of the autism literature that autism is a single syndrome, in which an underlying cause or causes results in a number of co-occurring symptoms.

Another possibility is that the term ‘autism’ is being applied to several quite different conditions, which may occur in isolation or which may happen to co-occur in the same individual, either coincidentally or with one being the consequence of another. This option has received comparatively little attention, but it appears to be a better fit with the observed phenomena. It also allows for clearer insights into the causes, diagnosis and possible treatment of the conditions involved.

One issue which appears to have been marginalised in the syndrome model is that of sensory processing problems. Although these are clearly important in autism, and have been lucidly described and discussed by Bogdashina and others, this work does not feature prominently in the autism literature.

I conclude that the syndrome model of autism is in need of radical re-assessment, and that there is a strong initial case for more research into the separate-conditions model. I also conclude that the role of sensory processing deserves considerably more attention.

Introduction

At first sight, some aspects of autism appear to be well understood, with general consensus about its nature and diagnosis, even if the details of its causes and most appropriate treatments are more obscure. On closer examination, however, this apparent consensus is problematic, raising questions about whether the cause and treatments are being sought in the wrong place. This article describes a detailed re-examination of some of the main concepts relating to autism. Its primary finding is that some key concepts are based on very shaky foundations; as a way of improving this situation, it suggests other, more solidly grounded, explanations derived from other fields.

Background

The term *autism* was coined by Eugen Bleuler, an influential figure in psychiatry at the turn of the 20th century. ‘Autism’ described the self-absorbed, detached behaviour of patients with schizophrenia - another term which Bleuler gave to the world. The concept of autism was borrowed by both Leo Kanner and Hans Asperger when, independently, they produced papers in the early 1940s describing what looked like a newly-discovered personality disorder in children. Autism is a condition about which there is still, after sixty years of research, a great deal of debate. Although there is widespread agreement about the core symptoms – impairments of social interaction, of two-way social communication and of imaginative activities, together with stereotyped behaviour – in practice these concepts are so open to subjective interpretation that the consensus is much less solidly based than first appears. We’re not clear about the boundaries of the condition – who has it and who doesn’t – or exactly what its symptoms are.
The criteria of The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition of the American Psychiatric Association (DSM-IV) are clearly designed to be used only by specialists. There’s no exhaustive list of specific characteristics, and the core features of autistic disorders - impairment of social interaction, social communication and imagination, with stereotyped behaviour - are described in such a way that a diagnosis could be carried out only by experienced practitioners who have already seen a significant number of people with an autistic disorder.

Unfortunately, relying on human judgement means that diagnosis is open to the errors and biases common to the way all human beings categorise information and make decisions - more so, given the uncertainty about the nature and causes of autistic disorders. In order to clarify the reasons for the inclusion of the defining symptoms, I decided to look at the diagnostic process itself, or, more accurately, the categorisation and mental modelling processes used by the experts making the diagnoses.

Method

Some areas of research quickly yield results because researchers happen to have the most appropriate theoretical model of the problem and because good data are readily available; some problems take years of painstaking data collection before they can be solved; other areas remain unclear for a long time, often until researchers suddenly find a new way of looking at the problem. The Knowledge Modelling Group at Keele have been developing a systematic approach to the analysis of human error and bias in research, which they call Verifier. Essentially, Verifier examines the process, rather than the content, of research, to try to find bottlenecks which may have occurred because researchers have become ‘stuck’ with one particular conceptual model, or because research questions may have been overlooked, or alternative interpretations of the data may have been missed and so on.

Verifier consists of three main stages. The first involves obtaining a detailed understanding of the problem, of methods previously used to tackle it, and of previous findings. The next stage involves identifying points at which errors are particularly likely to have arisen, based on the well-established literature about human errors and biases. The third stage involves a detailed examination of those points to see whether errors actually have arisen, using methods such as formal logic, argumentation and category theory to investigate whether there are errors in the reasoning, use of evidence or theoretical frameworks being used.

I used the Verifier approach to follow the development of the model of autism from the original papers written by Leo Kanner and Hans Asperger, through to the formulation of the DSM-IV diagnostic criteria. In this article I present an overview of the way the current model of autism has evolved. In other articles I plan to look in more detail at specific aspects of its development, such as the diagnostic processes used by Kanner and Asperger and subsequent researchers.

Terminology  

Autism was originally viewed as a personality disorder, or a form of mental illness – an implication being that people suffering from it may recover. It’s become increasingly apparent that many characteristics of autism are inherited and persistent, and autism is now commonly viewed as the result of abnormal brain structure or function - depending, of course on what you consider ‘normal’ brain structure or function to be. Although I feel the medical model is not the most accurate one for describing autism I do use words associated with it like ‘clinician’, ‘patient’ and ‘symptoms’ only because they tend to characterise the relationship between many people with autism, their behaviour and the experts observing them. There are also differences of opinion in the literature as to whether people with autism should be referred to as ‘people with autism’ (the ‘people first’ approach) or ‘autistic people’ or ‘autistics’. I use the terms ‘autistic’ or ‘autism’ to refer to the diagnostic category to which some people have been allocated, but I’ve largely ignored this debate, for reasons which will,
I trust, become clear. There is also debate about whether autism is a disability or not. I think the answer is that sometimes it is, and sometimes it isn’t, for reasons which will become apparent, but I do talk about ‘impairments’ and ‘problems’ and ‘difficulties’ because the characteristics of autism often are problematic, if not for the autistic person, for the society in which the autistic person has to live.

Findings

Early models of autism

Sigmund Freud died in 1939, four years before the publication of Kanner’s paper, and had left a legacy of enormous influence on psychiatry. One of the first psychiatrists to embrace Freud’s ideas, if guardedly, was Eugen Bleuler, whose seminal book on schizophrenia had been published in 1911, and with whose work Kanner and Asperger were clearly familiar, since they used his terminology. Freud’s influence can be seen in Bleuler’s model of schizophrenia – proposing that a set of apparently diverse symptoms could originate from a few, not immediately obvious, causes (Bentall, 2003).

Syndrome syndrome The syndrome model of mental illness was a popular one in central Europe between the mid-nineteenth and mid-twentieth centuries. Kraepelin, Bleuler, Freud, Gerstmann, Kanner and Asperger each identified in their patients groups of symptoms of mental problems which frequently co-occurred, and which were attributed to an underlying (and often speculative) cause.

One example of potential problems with the concept of syndrome is Gerstmann’s syndrome. In his book The Shattered Mind, Howard Gardner (of Multiple Intelligences fame) outlines the history of Gerstmann’s syndrome (Gardner, 1974). Josef Gerstmann first noted this phenomenon in 1924. His patient, who had suffered a stroke, was unable to name or recognise the fingers on either of her hands (finger agnosia). Gerstmann noticed that other patients with this problem had additional symptoms; they confused left and right, could not write (agraphia) and couldn’t perform mathematical operations (acalculia). Gerstmann felt sure the symptoms were linked in some way, and that he had discovered a new syndrome. There are of course, a number of ways in which the symptoms observed by Gerstmann could be related. For example,

- the four characteristics may have a single common cause
- the characteristics may be sequentially linked – a problem with one may lead to problems with all, or some, of the others
- the four symptoms may be co-occurring by chance

Gardner’s analysis of Gerstmann’s syndrome doesn’t venture any conclusions as to whether or not the symptoms are interdependent (ie whether this syndrome really exists), but does point out that some of the observations made by Gerstmann and his followers are less than meticulous in places (they were working from the assumption that the syndrome did exist and were not always scrupulous about noting absences of symptoms) and that Gerstmann became very protective of his syndrome when other practitioners showed signs of scepticism. Gerstmann does not appear to have given serious consideration to alternative causal models for the cluster of symptoms he had observed.

Claims of this sort are particularly susceptible to problems with a classic type of judgmental error known as confirmation bias (Ross & Anderson, 1982), where the individual tends, usually without realising it, to notice and remember examples which confirm their belief, and to overlook and forget examples which do not agree with it. This bias affects experts as well as lay people, and is one of the reasons for the growth of evidence-based practice in medicine. It is particularly likely to be a problem in the case of syndromes, which by their very definition involve multiple features which may not always occur in every patient. The implications for the concept of autism as a syndrome are obvious, and will be examined in more detail later in this article.
In 1944, Hans Asperger, who was at the time working at a children’s clinic in Vienna, published a paper describing the characteristics exhibited by some of the boys referred to him (Asperger, 1944). He called the condition he observed autistic psychopathy and its symptoms were remarkably similar to those which had been described as autistic disturbances of affective contact in 1943 by Leo Kanner (Kanner, 1943) working in the USA, although, at that time, Asperger was not aware of Kanner’s work. One Freudian concept which Kanner adopted was the idea of an underlying drive giving rise to a variety of superficially unrelated behaviours. He cites other researchers who suggested that children taken into care, or who were obese, ate large amounts of food to fulfil their unmet need for affect. Kanner’s children, on the other hand, rejected food - and noises and social contact. It was clear to him that the children did not want intrusion of any kind, suggesting a disruption of the need for affect at a fundamental level. It is also clear that Kanner felt that a diverse range of behaviours had their origins in the children’s ‘all-powerful need for being left undisturbed’.

Asperger’s findings were not widely cited at first, but have since been referred to more frequently, the cluster of characteristics he described becoming known as Asperger’s Syndrome or Asperger Syndrome (AS). In 1991, Uta Frith published her translation into English of Asperger’s 1944 paper - together with a selection of other papers from experts working with individuals with autism – in the book she edited entitled Autism and Asperger Syndrome (Frith, 1991). AS and Kanner’s autism are now widely considered to be part of the same condition, often referred to as autism disorder (AD) or autism spectrum disorder (ASD). AS is sometimes described as high functioning autism (HFA) since individuals with this as a diagnosis often show only slight impairments, and frequently have a high IQ.

In the discussion section of his paper, Kanner outlines some ‘essential common characteristics’ of the syndrome he is describing and concludes that what links the varied characteristics is a ‘disturbance of affective contact’. Asperger’s list of common features amongst his autistic patients is much longer than Kanner’s, and he notes that ‘not every case has every feature’. He suggests that the problems result from a lack of ‘harmony between affect and intellect’, and ‘drives and instincts are often severely disturbed.’ This sounds very like Bleuler’s concept of schizophrenia, in which he felt that the different functions of personality had been separated.

Kanner and Asperger had suggested models of autism which were, at the time, both reasonable and plausible; reasonable because they were using a widely accepted theoretical framework for their models and plausible because the models neatly explained the wide diversity of symptoms they had observed in the children. (I will return to the issue of plausibility later.) Both Kanner and Asperger had observed autistic-like symptoms in the families of the children they had studied and this finding was supported by later researchers. They recognised that autism had some inherited component, but because the symptoms of autism were so diverse, and varied so much between children, it was difficult to see any patterns which could suggest possible biological causes. This issue was addressed by Lorna Wing, an influential figure in the field of autism research, who took the model of autism a stage further.

Further developments of the model

Lorna Wing’s analysis For some time, there had been a debate as to whether Kanner and Asperger had described the same syndrome. Wing undertook a detailed comparison of Kanner’s and Asperger’s original papers and their later writings. She identifies ten ‘striking similarities’ between the children described by the two authors (Wing, 1991):

- excess of males over females
- social isolation
- problems with verbal communication
• impaired non-verbal communication
• lack of flexible imaginative play
• dislike of change, stereotyped behaviour
• hyper/hypo sensitivity
• clumsiness and dexterity
• aggressiveness, restlessness
• outstanding cognitive skills

Wing concludes that a) Kanner and Asperger have each identified a real syndrome and b) that they have identified the same syndrome, albeit with minor differences. Kanner and Asperger based their descriptions of the features of their syndrome on observations of a large number of patients, selecting a smaller number to describe in detail. The chose the case studies because the children, between them, showed symptoms typical of the syndrome. However, none of the children showed all the characteristics. Given the number and diversity of symptoms, a systematic study of a larger population was clearly necessary for patterns of co-occurrence to become apparent. This is exactly what Lorna Wing provided. She and Judith Gould looked at children with social interaction problems in Camberwell, in south London (Wing & Gould, 1978). They found that three impairments frequently co-occurred in the children – those of social interaction, two-way social communication and imaginative activities. These features are now commonly known as the triad of impairments. The implication drawn from these findings is that if certain people have problems with imagination they will be unable to see the world from a perspective other than their own. Hence appropriate interaction or two-way communication with other human beings will be extremely difficult for them.

Of course, imaginative activities is a rather broad concept and needed refinement before its role in social interaction could be studied. This led to the next step in the current model of autism, the concept of Theory of Mind (ToM) (Leslie, 1987; cited in Frith, 1991). Theory of Mind is not, as may first appear, a philosophical speculation about the possible constituent parts of a human being. The idea is that individuals have a concept, or theory, of ‘mind’ - the perceptions, concepts, feelings, memories, that are unique to an individual; that other people have their own perception of the world, which is different from one’s own. So if people don’t have ToM, they can’t perceive the world from someone else’s perspective, which is obviously going to impact on their ability to interact socially or to communicate effectively.

Problem space

The field of artificial intelligence uses the concept of problem space. Problem-solving initially involves a process of reducing the problem space – frequently by the systematic elimination of false apparent solutions. If the cause of genetically determined impairment of social interaction is the problem, Kanner and Asperger reduced the problem space in that they identified children who showed certain kinds of behaviour resulting from difficulty with social interaction. Wing narrowed it further by identifying three frequently co-occurring impairments within such a group of children. It was reduced even more by the concept of the Theory of Mind. But has the Theory of Mind concept narrowed the problem space enough to throw light on possible causes of ASD?

Theory of Mind research

A popular way of finding out whether or not someone has ToM has been the false-belief test. To pass a false-belief test you need the ‘understanding that different people can have different thoughts about the same situation’ (Baron-Cohen, 2000). To pass a first-order false-belief test you need to understand what a person other than yourself is thinking about a situation. To pass a second-order false-belief test you need to understand what a person other than yourself is thinking about what a third person is thinking about a situation. The classic first-order false-belief test is the ‘Sally-Anne’ test, or a variant of it. In a typical Sally-Anne test, the following scenario is described or depicted. Sally and Anne are playing together. Sally leaves the room. Whilst she is gone, Anne moves Sally’s doll/ball/marble from the place she left it and hides it in a box/under a cushion/behind another object. Sally then comes back into the room. The child being tested is asked ‘Where will Sally
look for her doll/ball/marble?’ Children with ToM say she will look for it where she left it. Children without ToM will say she will look for it where Anne hid it, the location it has been moved to, since they are unable to see the world from Sally’s point of view.

There have been some ambiguous results from work with false-belief tests. ‘Normal’ children pass the test at different ages. Some hearing-impaired children have problems with first-order tests. Some children diagnosed with ASD can pass first-order false-belief tests but not second-order ones. Some children fail the test when it’s a verbal one, but pass it when it’s presented pictorially, or as a model, or as a real-life enactment. In Understanding Other Minds Simon Baron-Cohen identifies no less than twenty different approaches by researchers to Theory of Mind tests. Clearly, the constructs Theory of Mind or understanding that different people can have different thoughts about the same situation are not quite specific enough, if there are so many ways in which they can be interpreted by researchers and if results are so varied.

Another important issue is that children could fail false-belief tests for a variety of reasons, such as a delay in general development, or language problems making it difficult for them to understand the test in verbal form, or difficulty with auditory processing, or problems with working memory meaning that they could not handle information about different scenarios simultaneously. There is no doubt that children who fail false-belief tests have some sort of problem with the tests compared to children who pass them, but it is by no means clear whether the problem relates to Theory of Mind, or to the test’s ability to assess what it is supposed to assess. If a child is unable to understand what they are supposed to do on the test, whether because of hearing problems, language problems, or reading problems then the results from that test will not provide trustworthy information about that child.

The spectrum model

Kanner and Asperger had identified a group of individuals with a range of unusual behavioural characteristics. The characteristics tended to co-occur, so it was not unreasonable to assume that they were linked in some way. Their key features were an abnormal response to affect and a dislike of intrusion, causing severe impairments in social interaction. Lorna Wing later found in a similar group of children that three characteristics, impairments of social interaction, social communication and imaginative activity, co-occurred most frequently. Because of the variation between individuals due to genetic differences and environmental factors, the symptoms of autism could vary in their nature, severity and distribution. So people with autism could show very different specific impairments but still have the same diagnosis. This variation has given rise to the description of autism as a spectrum disorder. The concept of a spectrum disorder is plausible because it neatly accommodates the wide variation in symptoms. However, the model has some serious limitations.

Discussion

Two problems with the spectrum model

‘A dash of autism’ One problem is that characteristics of people with autism spectrum disorders (ASD) also appear in people who don’t have an ASD diagnosis, and who wouldn’t qualify for one either. Kanner and Asperger both identified autistic-type characteristics in other members of the families of the children they saw, Christopher Gillberg’s family study of ASD people shows autistic characteristics cropping up throughout families (Gillberg, 1991) and the literature is replete with references to the focussed, absent-minded tendencies of scientists. In the introductory chapter of her book, Autism and Asperger Syndrome, Uta Frith comments; ‘Like Asperger, I too would sometimes like to claim a dash of autism for myself. A dash of autism is not a bad way to characterise the apparent detachment and unworldliness of the scientist who is obsessed with one seemingly all-important problem and temporarily forgets the time of day, not to mention family and friends.’ Digby Tantam
(1991) contributes a chapter to Frith’s book, about adults with Asperger syndrome (AS). In his table 5.3 he displays results from a study comparing typical ASD symptoms between two groups of people, those with an AS diagnosis and those without. Although the symptoms occur in a much larger proportion of the AS group than the non-AS group, a significant percentage of the non-AS group still showed AS symptoms (circumscribed, unusual interests 43%, impaired non-verbal expression 38%, pragmatic abnormalities 14%, semantic or syntactic abnormalities 7%, clumsiness 36%). The participants in the study had been selected because they had been identified by psychiatrists as eccentric and socially isolated, so this finding isn’t surprising, but it raises important questions. If autistic-type symptoms are so widespread in the ‘normal’ population, what distinguishes those ‘normal’ people from those with ASD? Is it that people with ASD have more of the symptoms, or that the symptoms they have are more severe, or is it that many people have a mild form of ASD and have simply not been diagnosed? Unless researchers studying a specific behaviour are very careful in the selection of autistic people they work with, they could be looking at people with very different behavioural profiles. They would need to ensure that their ‘autistic’ group showed the particular feature they were researching, and, because single autistic features are not uncommon in the normal population, that the particular feature was absent in the ‘normal’ group.

Range of characteristics  Another problem involves the wide range of the characteristics which are distinguishing features of ASDs. Any defining description of a behavioural disorder has to accomplish two tasks; it has to be general enough to encompass the variety of behavioural characteristics of which it is made up, and it has to be specific enough to distinguish that disorder from other disorders. An example of a broad diagnostic criterion from another domain may illustrate the point. Walking into town one day, you are surprised to pass no less than three people displaying a marked limp. Their gait appears very similar – the word ‘limp’ identifies the commonality between the three limpers and also distinguishes the way they are walking from, say, shuffling or hobbling. However, what you don’t know is what is causing the limps. As it happens, one limp is the result of blister on the heel, another limper is recovering from a sprained ankle and the third sustained a leg wound in combat many years earlier. Note that presenting symptoms are almost identical, the underlying symptoms very different, in both their nature and degree of severity, and the causes of the underlying symptoms different again. The interventions needed for the woman with the blister on her heel are different from those needed to improve mobility for the man with the sprained ankle. The wounded soldier has probably learned to live with his old war wound. It’s relatively easy, of course, to identify the causes of limping. One can usually ask the patient, or examine their lower limbs. Ascertaining the causes of impaired social interaction in people with communication difficulties is more challenging. But different interventions tailored to specific needs will be needed to support people with ASD. The lack of specificity in the definition of autistic spectrum disorders has significant implications for both support interventions for people with ASD and for the direction of research.

Implications

Support  At one level, the lack of specificity in the DSM-IV doesn’t matter too much; when, for example, the DSM criteria are used to decide whether someone suffers sufficient impairments through ASD to warrant additional support which requires public funding. At this level, the particular characteristics of autism experienced by the person in question are not of vital importance – it is often sufficient for them to have been confirmed as having autism or Asperger’s syndrome. And if someone is described as having impairment in social interaction, social communication and imaginative activities, that description distinguishes them from someone with, say, a visual impairment, or with mobility difficulties. However, at another level, the particular characteristics experienced by an individual do become important – at the point where they need practical support or treatment, for example. People who are closely involved with autism research may be acutely aware of the wide variety of symptoms and causes of ASD, but that’s not the picture you get from reading the popular literature on the topic, or even from talking to people who have to deal with children with ASD. It's
all too common to find a teacher who thinks that because a child continually flaps their hands or refuses to look them in the eye, they must have a fundamental problem with social interaction. They may indeed have a social interaction problem, but it may not be helped by providing them with a ‘playground buddy’, as we shall see.

Directions for research The DSM’s list of characteristics reflects the predominant model of ASD. And the predominant model, derived from Kanner and Asperger, in turn derived from Freud and Bleuler, assumes that the characteristics of ASD are linked and possibly share the same underlying cause. The broad Freudian concept of drive has largely been replaced by an assumption that the underlying cause of autism is a specific biological one (Frith, 1991). If a problem with neurological development had arisen early in the life of an embryo, it could account for the pervasive nature of the symptoms of autism. The variation in type and intensity of symptoms could be accounted for by variations in the genetic makeup and experience of individuals. This is one direction which could be taken by research. Another productive way of searching for the underlying cause of autism, is to look at what might be causing a particular characteristic of ASD. Much of this research is taking place at a neurological level, so we need to examine the approaches adopted by cognitive neuroscientists.

The way brains work I – modular processing

Evidence has been around for many years which suggests that specific parts of the brain have specific information-processing roles. The frequency of military conflict in Europe during the 18th and 19th centuries provided physicians with many opportunities for investigating brain function. We have known for well over a century, for example, that people with damage to a particular location in the left hemisphere of their brain, known as Broca’s area, have difficulty finding the words they need, and in saying them, even though it is clear they still have a good understanding of language. Patients with damage in the nearby Wernicke’s area can speak fluently, but their speech can appear meaningless and unconnected. In the 1950s, Hubel and Weisel’s study of the visual cortex of the cat showed that information-processing took place at a cellular level, with small clusters of cells responding to one kind of visual stimulus and other clusters responding to another kind. Since then, with vastly improved techniques of detecting the location of activity in the brain, we now have a much clearer picture of which parts of the brain are involved in which information-processing tasks.

A relatively simple task, such as detecting a vertical black line against a white background, involves a small part of the visual cortex; a complex task such as reading, which is made up of many sub-tasks, will involve several parts of the brain, notably the visual and linguistic and motor areas. A dysfunction of just one, small, specific processing component involved in the complex task of reading could lead to serious reading difficulties. To take an improbable example, if a child had difficulty detecting vertical lines, their perception of letter shapes would be very different from that of normal readers. Some letters would be easily readable because they have a unique configuration of horizontal or curved lines, but others would be easily confused. Both c and d would appear as c and c; b and p as the mirror image of c, and so on. The child could possibly learn to read by compensating for some of the confusions, but would make reading errors which would appear very puzzling unless one understood what was causing them. Social interaction embraces a much more complex set of tasks than reading, involving sensory, motor, spatial, linguistic, emotional and meta-cognitive processes; it could take only one abnormally functioning processing unit in one of these domains to impair social interaction.

Given that specific areas of the brain are dedicated to responding to specific, finely-differentiated types of information, and given the potential for variation in individuals due to their unique genetic make-up, and the potential for damage due to the complexity of embryonic neurological development or environmental experience, what’s surprising is not that there are large numbers of people who show a wide variety of differences in information-processing, as in autism, but that anyone could be described as ‘normal’ at all. (I plan to return to the concept of normality in a later article.)
Shape of the model

I began to wonder if it wasn’t the content of the model of ASD which was giving rise to inconclusive results in research (not enough data or not the right data), but the structure of the model itself. Although both the content of the model and its structure have been modified (imaginative play has been added to the list of significant symptoms, and the Triad of Impairments and Theory of Mind concepts have narrowed the problem space) the basic shape, or template, of the model is still the one proposed by Kanner and Asperger; an underlying cause or causes leading to impairment of social interaction which in turn gives rise to numerous varied behavioural characteristics.

I noted earlier that Kanner’s model of a disturbance in affect was plausible because it fitted the wide range and variation of symptoms in autism. However, good fit is not the only factor to be considered when assessing a theoretic model, since many models could be constructed which fit the data well - an infinite number, in fact (Kaplan & Kaplan 2003). To take some extreme examples, one could construct well-fitting models explaining the symptoms of autism in terms of alien abduction or possession by evil spirits. (And if this seems far-fetched, recall the cases in the not too distant past, where social workers have taken children into care because of their suspected involvement in inter-generational institutionalised satanic ritual abuse.) The spectrum model does indeed fit the symptoms, but that’s because it has been designed to do so. We’ve seen that the basic structure of Kanner’s model looked like this;

![Diagram of model](image)

I wondered what would happen if the causality between social interaction and symptoms were to be rearranged;

![Diagram of rearranged model](image)

Figure 1: Two possible causal relationships in ASDs

So, if genetic variation or environmental damage had produced a change to normal functioning in a particular part, or parts, of the brain, this could manifest itself in unusual behaviours which in turn could cause problems with social interaction. In other words, the unusual behaviours could lead to problems with or avoidance of social interaction, rather than the individual having an underlying impairment in any aspect of social interaction per se.

Red rash disease  To illustrate, I want to show what would happen if a unitary cause model were used to explain a number of conditions with similar symptom, for which we know the causes. One morning, you wake to find your child has a high temperature and a skin rash. You consult the doctor. The doctor examines the child. “Hmm…” says the doctor, “a clear case of red rash disease.” “What’s that?” you ask. “Oh”, replies the doctor, “red rash disease is very common in young children. It’s what we call a spectrum disorder, since the symptoms vary between individuals. The rash can vary in appearance, and can occur on various parts of the body. Sometimes it’s accompanied by a fever, or headaches, or nausea. In severe cases, it can cause brain damage. And some forms are very persistent; the rash lasts for years, and is made worse by the child eating certain foods.” “But what causes it doctor?” “We don’t know. It could be a virus, or a bacterium, or an allergic reaction. Not to worry, I’m sure young Polly will soon feel better.” Since we now know the causes of chicken pox,
german measles, measles, scarlet fever, eczema, and other once common childhood diseases, a doctor supplying a diagnosis of ‘red rash disease’ would nowadays be treated with scepticism to put it mildly. But anyone who did not have access to information about the causes of the illnesses could easily see them as different manifestations of the same underlying condition.

*Sensory information processing* I now want to show how different underlying causes can lead to similar presenting symptoms by looking at sensory information processing. Sensory information processing is what happens to sensory information in the brain, as distinct from what happens to it in the sense organs. Differences in sensory processing are easily overlooked unless you are familiar with the behaviours they can produce. There are a number of ways in which sensory processing can be impaired. Some of the most frequent include problems with the following: *modulation of sensory information, integration* of information from different sensory channels, *figure-ground discrimination* and *integration of parts* into wholes (Bogdashina, 2003).

A child whose modulation of auditory information is weak could be hyper- or hypo-sensitive to auditory stimuli or both – they could hear everyday sounds as fluctuating in loudness. If auditory figure-ground discrimination is difficult the child cannot separate out one sound to focus on against background noise. A not uncommon visual processing problem is in integrating the parts of a figure, or an object, or a face, into a whole. People whose integration of sensory information from different sensory channels is too low often report having to shut down one sensory channel in order to concentrate on another. People whose integration of sensory integration is too high, are described as *synaesthetic*, a phenomenon which I will return to later. One of the difficulties in detecting sensory processing impairments in a child is that if the child is concentrating on stimuli from a single sensory channel, they often perform very well. It’s in situations where more than one sensory channel is bringing in large amounts of information that problems arise. So a hearing test or sight test could show the child as having adequate hearing or eyesight, but in the classroom the child appears not to hear, or to be able to see what’s on the board, because they are having to ignore either their auditory or visual channels to reduce confusion.

One of the striking features of Kanner’s description of the children in his case studies is the number of behavioural characteristics which could be explained by sensory information processing difficulties. For example, seven of the eleven children in Kanner’s case studies had at one time been suspected of being deaf or hard of hearing. Given the severity of the difficulties experienced by the children, it’s probably safe to assume that their hearing had been tested and found to be normal. However, several of Kanner’s children did things like making repetitive noises, or covered their ears when other children were angry with them, or were fearful of noisy machinery or had unusual voice pitch or intonation, or difficulty with phonics, all of which suggest auditory problems. Kanner’s reasoning seems to be that if the children were showing signs of hearing abnormality, but their hearing had been shown to be normal, then their apparent auditory difficulties must have a deeper ‘psychological’ cause.

Kanner recorded other unusual sensory responses. Some of the children loved to watch spinning objects; others were afraid of moving things. Several children had unusual tactile responses such as putting food in their hair, or an intense dislike of dirt. Many of the children had had problems with feeding when younger; some children put all kinds of objects in their mouths, or had strong food preferences. Kanner attributes the feeding problems to a dislike of intrusion, but an alternative explanation is that the children had digestive problems or unusual gustatory responses.

Sensory processing problems keep cropping up in the autism literature. Hyper- or hyposensitivity to sensory stimuli is not uncommon in autistic people, but is generally viewed as a condition which can co-occur with autism, rather than as a central characteristic, which is why it is not mentioned in the DSM-IV or the ICD-10 (International Classification of Diseases, tenth edition). Carol Stock Kranowitz describes many symptoms of sensory processing impairments in her book *The Out-of-Synch Child*.
(Kranowitz, 1998). Much of the content of her book is derived from the occupational therapy literature. Occupational therapists are familiar with problems in sensory processing, and with the difficulties some people have in integrating information from different sensory channels. In addition to the five senses of smell, touch, taste, sight and hearing, children can have problems with the vestibular sense (balance) or proprioceptor sense (information from parts of the body, such as muscles), which can severely affect their co-ordination and motor control. Autism researchers frequently note that people with ASD have problems with the flexibility of responses required in social interaction. Flexibility requires fast responses and efficient sensory processing. Any sensory difficulties could make normal social interaction very difficult. A child with multiple sensory difficulties could find normal social interaction impossible.

Sensory processing problems may also contribute to the second strand of DSM-IV criteria, social communication, particularly if the sensory channel involved is the auditory one. Any problems with auditory processing put language acquisition at risk. And of course, language acquisition is a complex process involving many brain areas other than those dealing with sensory input. The variety of linguistic problems observed by Kanner alone, is highly suggestive of multiple causes. The third strand of the criteria, stereotypical behaviour, groups together ‘restricted and repetitive’ interests, rituals, motor movements and bizarrely, ‘persistent preoccupation with parts of objects’. Again, sensory processing problems could account for all of these. If your sensory input is unreliable, sticking with the same routine, or the same arrangement of furniture, or exploring the same domain of interest, is infinitely safer than branching out into uncharted territory. Autistics give the name stimming to repetitive motor movements, such as flapping the hands, or peeping through the fingers, or rocking, because they have the effect of regulating sensory input by stimulating or damping down the relevant sensory channel as required. ‘Persistent preoccupation with parts of objects’ could be caused by difficulties in producing a gestalt or complete picture of the features of an object, or in some cases, of faces.

So if the three strands of behaviours which define autistic spectrum disorders can arise from many different causes, why has a unitary model of autism persisted? Why are people with widely divergent profiles of impairments all classified as autistic? We’ve looked at the how the similar characteristics of ASDs can arise from diverse abnormalities of brain function - autism from the inside, as it were. Now we need to look at another aspect of brain function, to find out why the model of autism has developed as it has - autism from the outside. Next I want to consider default strategies in information processing.

**How the brain works II – default strategies**

The human brain processes vast amounts of information simultaneously – about the body it is located in, about the environment, about past, present and future scenarios. To deal with each piece of information separately, from scratch, would mean that the owner of the brain would be in a state of perpetual confusion due to information overload. So brains which have developed ways to reduce the amount of information being processed at any one time have made it much more likely that their owners would survive to reproduce. Gradually, human brains have evolved default strategies, often structurally embedded (or hard wired) which ensure that certain types of information are dealt with automatically (and usually subconsciously) freeing up the parts of the brain which are needed to solve problems or make decisions. These default strategies include compiled skills - where repeated rehearsals of certain clusters of behaviours lead to the cluster becoming automatic (such as reading or driving), the formation and maintenance of mental causal schemata - in which we arrange information about a group of entities in a pattern based on our perception of the way the entities are related, such as developing a theory or belief, and a tendency toward conformity of belief to match the beliefs of others in the social group to which we belong.

It’s easy to see how default strategies could improve our chances of survival if we were living the precarious existence of a hunter-gatherer. Compiled skills would mean fast responses when hunting;
causal schemata mean we can learn to recognise dangerous situations; persistence of schemata means we can recognise dangerous situations in changing circumstances (‘everyone who has eaten these berries has died – I mustn’t eat these berries even though I am very hungry’); and conformity to group norms enhances the coherence and stability of the social group. Of course, not everyone uses these default strategies to the same extent: high-performing athletes have excellent motor skill compilation - which some of us could never match; some people find it easy to switch between belief systems - other people stubbornly stick to their original viewpoint in the face of overwhelming evidence that they are wrong; some of us are easily swayed by group opinion - others are always rebels.

Also, although cognitive default strategies are advantageous most of the time, they are not advantageous all of the time. In some circumstances they can be more of a hindrance than a help. Compiled skills really get in the way when you are trying to drive a courtesy car, and your brain thinks you are driving your own car which you left at the garage; the persistence of simplistic negative schemata (stereotypes) causes enormous social problems; and most of us suffer at some time from unwelcome social pressure. I want to examine the development of models of autism with these cognitive default strategies in mind. First, let’s take a look at some of the properties of human cognitive models, or schemata.

Causal relationships  Every student of the biological sciences has had drummed into them at some time the mantra ‘correlation does not imply causality’. The fact that A and B usually occur together does not mean that A causes B or vice versa. A and B may both be caused by C, where C can be a simple entity or a complex causal chain. Or, both A and B can have separate, unrelated causes. However, phenomena which frequently co-occur are often causally linked, so attributing a causal relationship to frequently co-occurring phenomena is a useful cognitive default strategy in everyday life; it removes the need for us to examine causal relationships each time we observe the same phenomena co-occurring. But where many phenomena are involved, causal relationships (if any) are more likely to be complex than simple. So a default strategy which assumes a simple causal relationship can seriously interfere with the requirement, in some circumstances, to examine all possible causal explanations. I should emphasise that default strategies, by their nature, are largely subconscious. They are a product of the way our brains operate, and not of laziness or stupidity. So avoiding using a cognitive default strategy requires conscious effort.

Persistence of schemata  Most of us have seen those gestalt images which flip instantly from looking like one thing to looking like another; the young woman who transforms into an old woman’s face, the cube which is coming out of, then going into, the page, the vase which changes into two faces in profile. Even when you know that the picture can look like something else, and you know what that something is, it still requires effort to flip between the two images. Our mental models behave in a similar way. Once you’ve got a model in your head, even if you are aware that the entities in your model can be arranged in a different way, it’s difficult to rearrange them, and when you have rearranged them, it’s difficult to return to the first configuration. The holding of one model inhibits access to another, because of the neurophysiology of cognition; when you are concentrating on something, the brain actively reduces activity in alternative neural pathways, presumably to reduce the risk of confusion and to channel resources to the most urgent task. This is one of the reasons that creative thought can be so difficult – the process of generating one new idea will simultaneously weaken neural links to some other related ideas.

People can persist in holding onto belief-structures even in the face of overwhelming contradictory evidence (Ross & Anderson, 1982). This is a major problem in research; researchers who have no vested interest in propagating a specific theory in their field, and who are very keen to study all aspects of the topic they are investigating, still find it very difficult to look at the problem from a completely different angle. However, one factor which can generally be relied upon to change our beliefs, is what people around us are thinking. Many of us find it difficult to maintain a schema if we are in a minority in holding that mental model, particularly if we are constantly exposed to the
opposing viewpoint. There isn’t space here to go into detail about the mechanisms of persistence of schemata, but it’s possibly because neural pathways which are used frequently, or have been used recently, are more easily activated than those which are used infrequently, or have not been used for some time.

So how does this apply to the model of autism? I want to focus on Kanner’s model since it is the one which has been most influential in the development of current ideas about autism. Kanner’s thesis that the behavioural characteristics he had observed were linked in some way, and that their causal relationship could be due to disturbances of affective contact, was not unreasonable at the time he was writing. It was one possible explanation for the phenomenon of co-occurring behaviours which he had observed. But there were other possible explanations (such as unusual sensory responses) which Kanner overlooked or did not develop. With his professional background, Kanner would have been aware of the correlation-causality issue, but he still appears to have made the assumption that because the behaviours co-occurred, they must be causally related in some way. Of course, a concept like disturbances of affective contact neatly explained all the strange behaviours he had witnessed, even though the precise nature and mechanism of the disturbance was largely speculative.

I suspect Kanner chose his model for the following reasons. Firstly, he notes that the characteristics of the children’s behaviour were remarkably similar to those Bleuler had termed as autistic when describing schizophrenia. Kanner was familiar with Bleuler’s model of schizophrenia and the Freudian idea of drives. Primed by this familiarity, Bleuler’s model readily sprang to mind when Kanner was searching for an explanation for the symptoms shown by his sample of children. Kanner would have referred to Bleulerian and Freudian concepts frequently in his daily clinical practice, so the salience of the model would have been repeatedly reinforced. In addition, Freudian ideas were very much in vogue in the 1940s so Kanner would have been surrounded by other researchers also reinforcing the kind of model he was proposing. This schematic priming and reinforcement would also have inhibited the development of any other models for autism.

Although Kanner’s model has been modified by later researchers (the disturbances of affective contact explanation has developed into the Triad of Impairment, and impairment of Theory of Mind posited as a cause for the triad) it still retains the same essential template - the assumption that a very diverse range of behaviours has a single underlying cause. Little serious consideration appears to have been given to the possibility of other causal relationships between the characteristics of autism. I believe there are two primary reasons for this. The first is that researchers subsequent to Kanner will have gone through a similar process to Kanner with regard to their own schemata of ASDs. They would have been introduced to the concept of autism through unitary models, either by a historical description starting with Kanner and Asperger, or through a diagnostic description starting with something like the DSM -IV. There would therefore have been a priming effect in the construction of their own models. Researchers would have needed to rehearse the model repeatedly to become familiar with it, leading to reinforcement of the unitary model and inhibition of alternatives, and everything they read and everyone they talked to would have confirmed that the unitary model is the correct one.

There is one other factor that could strengthen the conviction that the unitary model explains the condition – observing the behaviour of people with autism. Even though people with autism show a wide variation in their behavioural impairments, the kinds of behaviours they exhibit are readily identifiable. Even as a novice, every time I see someone diagnosed with ASD or read a case study, I experience the ‘Aha!’ of recognition, as, presumably, do the clinicians undertaking the diagnoses, since it seems they show a high level of agreement as to their diagnostic conclusions. This recognition appears to confirm the commonality of the symptoms and the veracity of the unitary model. But people with autism have very different profiles of impairment, so why do their behaviours look so similar?
Levels of categorisation  I think the apparent similarity of behaviours is a consequence of the level of categorisation of the characteristics that we are observing. Imagine a set of behaviours which contains all human abnormalities (for want of a better word). Within that set are subsets of behaviours such as impairments in social interaction and social communication, stereotypical behaviour, mobility difficulties, impaired vision, criminal behaviour, delusions, mood disorders etc. Within each of those subsets there are yet more subsets, and so on until we arrive at a level of specificity below which we cannot divide the classification of behaviour further without it becoming meaningless. So within the set of criminal behaviours we could have, amongst others, a subset of fraud and a subset of robbery. Clearly someone who has made a career out of hitting old ladies over the head and running off with their handbags is very different to someone who has indulged in sophisticated financial dealings at the margins of legality, yet for many purposes, they are both classed as ‘criminals’. And someone who is feeding a heroin habit, or to someone who gets a kick out of intimidating elderly women. Just as there are different levels of categorisation for criminal behaviour, there are different levels of categorisation for social interaction.

Social interaction is a set of behaviours which includes a huge number of entities - Michael Argyle’s well-known book on the subject runs to over 250 pages. So the possible forms impairment of social interaction can take are very numerous indeed. The DSM-IV looks for ‘marked’ impairments, which narrows it down slightly (but not clearly) and gives some examples, such as ‘eye gaze’. But the examples themselves are also sets of phenomena. Eye gaze looks, at first glance (no pun intended) like a fairly simple behaviour. People either meet your gaze or they don’t. But there could be several reasons for someone not using eye gaze appropriately. They could have abnormalities of the eye muscles or retina, meaning that they use peripheral vision (a feature observed by Asperger). Or the problems may be at a sensory processing level. Autistic people themselves report a variety of reasons for not looking directly at someone else’s eyes; for instance, they experience it as a tactile stimulus; it’s painful; it’s overwhelming; the other person’s eyes don’t keep still; they find processing visual information such as eye contact interferes with listening to what the other person is saying (Bogdashina, 2003). Indeed ‘normal’ children have been shown to avert their gaze when answering a question which requires concentration (Doherty-Sneddon, 2004). Experiencing eye contact as a tactile stimulus is worth examining more closely. Synaesthesia – a response to a stimulus in one sensory channel also occurring in part of the brain which usually deals with another - is not uncommon in the normal population, particularly in children. It can involve ‘smelling’ sounds or ‘seeing’ musical notes as colours. It could equally easily involve ‘feeling’ visual stimuli such as eye contact. (A useful source of information about synaesthesia is Jamie Ward’s website, www.psychol.ucl.ac.uk/jamie.ward/synaesthesia.htm.) Another point worth noting is that eyes are a very salient visual stimulus in many phyla of the animal kingdom, in brains much more ancient, in evolutionary terms, than those of humans, so it’s unlikely that eye gaze variations arise solely as a result of abnormalities in human social interaction systems. Note that none of the first-hand accounts includes, as a reason for avoidance of eye gaze, a lack of interest in interacting or communicating with the other person, just difficulty with the visual processing of the eye contact stimulus.

We’ve seen that the member of the set of behaviours social interaction, which we call eye gaze, is in itself a set with a number of members, or components, some of which include differences in processing visual information. Social communication is another hugely complex set involving wide variations in speech and language; social imagination is also a complex construct which would take us into the realms of visual imagery, dreams, memory and so on. I suggest that the reason we experience recognition of the commonality of the symptoms of autism is that we are categorising behaviours at a relatively undifferentiated level; we are matching broad subsets of human dysfunctions, much as we did when noticing our three passers-by who limped. But the broad subsets each contain a large number of entities, so that two people diagnosed with ASD at the social interaction level may have quite different impairments when diagnosed at the eye gaze level and even more differences if diagnosed at the very highly differentiated, components of eye gaze level.
**The DSM triad**

It could be argued that it’s the co-occurrence of all three strands of behavioural impairments - interaction, communication and stereotypy - which distinguish ASDs from other disorders. How likely is this co-occurrence, unless there is a single underlying cause?

If each of the three strands of behaviours is defined at a very general level, each of the strands can involve a huge number of information processing units, and connections between units, in the brain. If only one of the processing units or connections is functioning abnormally, and that unit or connection happens to be involved in both social interaction and social communication, *and* its dysfunction produces what is described as a stereotypical behaviour, then we have someone who is diagnosed with an ASD. And specific-function processing units are used in a wide variety of complex tasks. If a group of cells in the visual cortex responds to vertical lines, it does so whether the lines are tree trunks, parts of letters, features in paintings, or notes on a musical stave, so it can be involved in a wide range of behavioural or cognitive domains. Given the complexity of processing involved in each of the behavioural strands of ASD, it is quite likely that there are a number of processing unit or connection dysfunctions which could be involved in producing behaviours in all three diagnostic strands. So in any given individual, there may be only one dysfunctional processing unit giving rise to impairments on all three diagnostic criteria, but this could also occur as a result of the dysfunction of a different processing unit in another individual. Both would receive a diagnosis of ASD, even though the underlying single cause for their behavioural abnormalities was different.

In other words, impairments in social interaction can look pretty similar regardless of the cause. You may show impaired social interaction because you don’t perceive other people as people (but as objects); or because you can’t imagine someone else’s point-of-view; or because you can’t see faces as faces, but as collections of disconnected facial features; or because you can’t identify facial expressions; or because you can’t identify emotions (in yourself or others); or because you have problems with language; or auditory problems; or peripheral vision, so you can’t make direct eye contact; or because you are aware that for some reason other children find you irritating and don’t like you and you can’t face any more rejection. Whatever the reason, if you can’t manage social interaction effectively, you are likely to avoid people, avoid conversation, avoid eye contact, and behave inappropriately in social situations (Figure 2).

Perhaps here I should make it clear what I am *not* saying. I am not saying that people diagnosed as being on the autistic spectrum do not have anything in common; they clearly do – it’s what we call impairment in social interaction and social communication. But those impairments in social interaction and social communication can have a wide variety of causes. Secondly, I am not saying that there cannot be a single underlying cause for those impairments – there may well be, but even when we locate it, that still leaves a lot of people diagnosed with ASD needing support with their specific range of problematic behavioural characteristics. Thirdly, I am not suggesting that ASDs are all caused by sensory information processing problems; that’s only one possible explanation for social interaction problems in some people. Fourthly, I am not suggesting that problems with social interaction *per se* do not exist; some individuals may not be able to perceive other people as people, or may have no Theory of Mind, but the results from the false-belief research suggest that there may be a variety of causes for impairments directly involved in the ability to interact socially. Lastly I am not saying that people diagnosed with ASD do not have a problem - conventions of interaction and communication are very salient to human social groups and anyone who can’t conform to those norms is in danger of social disapproval at best and ostracisation at worst, and will probably need support to enable them to interact with others effectively.
underlying problem | behaviour | DSM criteria met
--- | --- | ---
Difficulty simultaneously processing auditory & visual information | Avoids eye contact when another person is speaking, to concentrate on auditory input | Marked impairment of social interaction

**person 1**
- Experiences visual interference when monitoring their own speech; avoids using gesture
- Chooses activities which involve only visual or only auditory processing

**person 2**
- Has difficulty integrating parts into wholes
- Problems recognising faces and emotions; avoids looking at facial features
- Chooses activities with clear sequential processes; avoids anything ‘intuitive’

**person 3**
- Difficulty distinguishing between emotions
- Difficulty modulating information in several sensory channels
- Insensitive to feelings of others’ puts hands over ears or over eyes
- Avoids conversation and social situations
- Puts hands over ears or over eyes, flaps hands, frequently spinning or rocking

Marked impairment in social interaction
Marked impairment in social communication
Repetitive stereotypical behaviours

**Figure 2: Some possible alternative causes for diagnoses of autism spectrum disorder**

**Mosaic model**

What I am suggesting is that there are other options for models which explain the characteristics of the people who we currently identify as having ASDs. One is a model which, rather than positing a speculative cause leading in unspecified ways to many different symptoms, starts from a highly differentiated identification of the symptoms themselves, and looks at possible causes for the particular cluster of symptoms shown by the individual. If the person’s behavioural and cognitive
profile were described accurately and extensively enough, any common features of the symptoms could point to the underlying cause, or causes. For example, if a child spent a lot of time in the classroom under a desk with their hands over their ears, was afraid of noisy machinery, had speech problems and had difficulty learning to read, one would be tempted to investigate in more detail the way he or she processed auditory information. Or if a child’s social interaction was characterised by difficulty responding to other children appropriately, not seeming to care if they hurt or upset someone else, and the child was unable to describe how they themselves felt, it might be worth exploring their emotional awareness or their ability to monitor their own behaviour.

If avoidance of eye contact, and the possible reasons for it, were to be represented in a systematic format, this would make it easier for the diagnostician to test for each possible cause in turn, and also to identify potential treatments for the most likely causes. The table below makes no attempt to be exhaustive, but illustrates the approach.

<table>
<thead>
<tr>
<th>Possible cause</th>
<th>Possible physical area involved</th>
</tr>
</thead>
<tbody>
<tr>
<td>poor control of eye muscles</td>
<td>eye</td>
</tr>
<tr>
<td>poor foveal vision</td>
<td>eye</td>
</tr>
<tr>
<td>synaesthesia (identify other sensory channels involved)</td>
<td>visual/auditory/tactile/olfactory/gustatory processing areas</td>
</tr>
<tr>
<td>visual hyper-sensitivity</td>
<td>visual cortex</td>
</tr>
<tr>
<td>auditory hypo-sensitivity</td>
<td>auditory cortex</td>
</tr>
<tr>
<td>Integration of sensory stimuli</td>
<td>cerebellum</td>
</tr>
<tr>
<td>vestibular inefficiency (difficulty processing moving visual stimuli)</td>
<td>cerebellum</td>
</tr>
<tr>
<td>difficulty forming facial gestalt</td>
<td>fusiform gyrus</td>
</tr>
<tr>
<td>difficulty recognising faces</td>
<td>fusiform gyrus/prefrontal cortex</td>
</tr>
<tr>
<td>difficulty discriminating between facial expressions</td>
<td>fusiform gyrus</td>
</tr>
<tr>
<td>difficulty identifying facial expressions</td>
<td>fusiform gyrus/amygdala</td>
</tr>
<tr>
<td>poor emotional awareness</td>
<td>amygdala</td>
</tr>
<tr>
<td>difficulty identifying emotions</td>
<td>amygdala</td>
</tr>
<tr>
<td>lack of awareness of other people as other people</td>
<td>temporal cortex/prefrontal cortex</td>
</tr>
<tr>
<td>desire to avoid social interaction</td>
<td>prefrontal cortex</td>
</tr>
</tbody>
</table>

Table: Suggested analytical format for locating underlying cause of behavioural impairment

The condition as more than the sum of its parts The idea of analysing symptoms rather than assuming an underlying cause is not new by any means. Olga Bogdashina (2003) in Sensory Perceptual Issues in Autism and Asperger Syndrome looks systematically at sensory processing and its potential to explain many of the symptoms of autism. She has also devised an elegant way of recording and displaying individual sensory profiles. Her assessment instrument clusters behaviours according to the sensory channel which is most likely to cause them and according to the kind of processing which is occurring within that channel. From this analysis one can obtain an overview of the individual’s sensory difficulties which could assist practitioners in predicting what other problems are likely to be encountered (Bogdashina, 2003; pp. 184-199). The use of a similar approach could, of course, inform diagnosis of other common behavioural conditions in children, such as ADHD (Attention Deficit Hyperactivity Disorder) or ODD (Oppositional Defiant Disorder). Indeed, there is often a considerable overlap between several developmental conditions. Deidre Lovecky, working with gifted children with various learning difficulties (Lovecky, 2004), struggles to classify their conditions concisely and has to resort to a mix and match approach with standard assessment instruments in order to accommodate the mix of symptoms in some children. And Richard Bentall
(2003), examining the way symptoms are categorised in "Madness Explained," raises some serious questions about the classification system for mental illness.

**Miracle cures**  The mosaic approach could also offer some insights into the reasons why there's so much controversy over the 'miracle cures' claimed for some developmental or behavioural conditions. Fish oils, changes in diet, balancing exercises, and multisensory phonics have all been shown to produce dramatic improvements in conditions such as ADHD, ODD, dyslexia and dysgraphia. The evidence for the outcomes of these interventions has also been robustly criticised as being 'unscientific', or contradictory evidence has been produced to show that the interventions have no effect whatsoever on children with the relevant condition. If the conditions are perceived as unitary, with the same underlying cause in all children diagnosed, when in fact the conditions diagnosed may be the result of a variety of underlying problems, then the contradictory evidence starts to make sense. If a child has poor myelination of their cerebral neurones, information processing may be slowed down and fish oils may well produce a dramatic improvement in cognitive ability. If a child cannot hear the difference between some sounds used in speech, they will certainly experience difficulty matching those phonemes with the relevant graphemes when they start to read and multisensory phonics exercises could help them develop multiple associative connections in making phoneme-grapheme matches. If another child has difficulty with the spatial features of visual stimuli, and can't form letters clearly or write in a straight line, they could benefit from exercises which develop their ability to locate their bodies in space. In other words, if there are different causes for children being diagnosed with a particular developmental condition, then they are going to respond to different treatments, and a treatment which does not address the underlying cause of a child's difficulty will not improve it. That does not mean that the same treatment will not lead to a dramatic improvement for another child.

Returning to ASDs, starting from a fine-grained analysis of the symptoms and looking at the pattern they form, could, of course, result in many people no longer being classified as on the autistic spectrum. There may well still be 'true autistics', those who have problems processing only information directly related to interactions with people, but even then, it is likely that a range of neurological processing differences will eventually emerge as causes for these problems.

**Conclusion**

ASD is generally viewed as a single syndrome with many different symptoms. There is a large amount of evidence which appears to be consistent with this model.

However, there is also a large amount of evidence which does not fit well with this model for various reasons. When the original, canonical descriptions by Asperger and Kanner are analysed in detail, there are clear variations in what is being reported. There are also reported features of autism attributable to sensory processing problems which do not fit comfortably with the single-syndrome model. These symptoms fit better with a multi-condition model. In this model, there are several separate conditions, one or more of which may happen to occur within the same individual, just as a single individual may happen to have a broken leg and flu at the same time. If someone happens to have more than one of these conditions simultaneously – for instance, sensory integration problems and problems with understanding other people's behaviour – then these conditions are likely to interact with each other and lead to more complex difficulties.

The multi-condition model makes it possible to focus more precisely and more systematically on specific symptoms, which offers the prospect of precisely defined diagnosis and treatment. For instance, a prominent feature in many patients is the presence of hyper- and hyposensitivity to sensory stimuli. Both these conditions can be improved by fairly simple interventions, such as giving a child a quiet place to rest or by providing stimulating activities. This model also makes it easier to establish which symptoms in a given case are likely to be the causes of a problem, and which symptoms are likely to be secondary effects.
Perceiving autism as a unitary condition, with many different symptoms, could be the result of the persistence of a schema developed in the 19th century which was then plausible, but which may now need to be changed in the light of new understandings of how perception and cognition work. The unitary condition model can also be counterproductive in terms of treatment for the individual concerned and in terms of directing research. There may be a single underlying cause for the symptoms we describe as autistic – but we cannot assume that there is, and the evidence, particularly the accounts from people with autism, suggests that whatever the underlying cause or causes, the unique behavioural profile of the individual is critical in determining what support or interventions they require. I feel it is time for a major re-appraisal of the spectrum disorder model of autism, and for the development of a much more highly differentiated analysis of the characteristics we currently include under the umbrella of this condition.

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