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The Cheat with the Ace of Diamonds, *by Georges de la Tour: an autistic child would be mystified by people "talking with their eyes"*

The origins of autism

A striking new theory of autism suggests that the disorder arises from an in-built abnormality in a child's brain. As a result, the thoughts and feelings of other people remain mysterious. This is why autistic people find social interactions difficult to cope with

John Morton

HOLLYWOOD'S portrayal of autism in the recent film *Rain Man* has drawn the public's attention to this mysterious disorder. Controversy over the origins and nature of autism has flourished since the syndrome was first recognised in 1943. For instance, some influential theorists have argued that it stems from "bad mothering" in the first months of a child's life. Recent evidence suggests that this picture is radically mistaken: autism develops because of a particular and peculiar "cognitive deficit". This deficit results in an inability to attribute certain kinds of thoughts and emotions to other people. Without this ability, we would never build up rich emotional relations with other people, and would remain about as social as cats.

In terms of social relations, autistic children are sometimes described as treating objects and people alike, although in fact their behaviour can vary from complete withdrawal—the autistic symptom of popular mythology—to repetitive pestering of an often totally inappropriate kind. The manifestation of the social oddness changes with development. In particular, the more able individuals who can be severely withdrawn in early childhood tend to become either merely passive or "odd" in their social behaviour from about the age of five onwards.

Problems in communication arise in both verbal and nonverbal behaviour. The children learn syntax and phonology appropriate to their mental age but they are usually slower to learn other aspects of language. Autistic children have particular problems with the pragmatic aspects of language. They are very literal, both in their use and understanding of metaphors and normal indirect figures of speech. An autistic child, asked "can you pass the salt?", is prone simply to answer "yes" rather than treating the question as a request. Such behaviour does not arise due to a lack of intelligence or a misplaced sense of humour.

The three key distinguishing symptoms of autism—impairments in social relations, communication and imaginative activity—seem, at first, and even second glance to be unrelated. As Uta Frith of the Medical Research Council's Cognitive Development Unit in London has observed, it would be possible to find a number of colour-blind, agoraphobic flautists; but no matter how many we found, we would not want to elevate this conjunction into a syndrome. However, Lorna Wing and Judith Gould from the MRC's Social Psychiatric Unit in London established, in 1979, that in autism, social impairment always goes hand in hand with both severe problems in communication and the absence of imaginative pursuits. Thus autism is a true syndrome, rather than being a chance collection of independent symptoms. Any complete theory of the cause of autism, then, has to account for all three symptoms.

Some psychologists still regard autism as either a personality disorder or an emotional disorder in response to psychological stress. Such theories effectively take one of the symptoms, namely, social impairment, and treat it as cause.

For example, one popular view attributes autism to the early breakdown of the relationship between mother and infant: the "refrigerator mother" theory. The idea is that inadequacies on the part of the mother have led to a failure of "bonding" between mother and infant and this, in turn, is alleged to lead to the whole complex of symptoms. One problem with this dangerous theory is that there is not a shred of evidence in its favour. It is instructive as a contrast, however, because it attributes most of the responsibility for autism to the interaction between mother and child, and puts most of the blame on the mother. This leaves little room for any genetic or neurological components in the child.

Other variants of social cause have usually taken the form of suggesting that the child is abnormally anxious about social interactions and has acquired an aversion to other humans. Unfortunately for these theories, such an aversion is rare in autism. What autistics all do have in common in an *abnormality* in their social relationships.

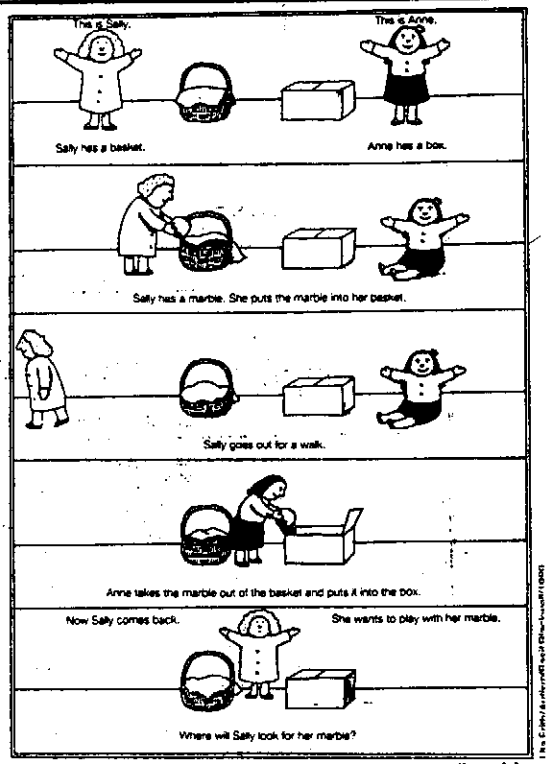
Over the past 10 years it has become clear that autism must have a biological origin. There are several reasons for this. First, people with certain congenital medical conditions, such as maternal rubella, tuberous sclerosis and a chromosome defect known as fragile-X syndrome, are much more likely to suffer from autism. Secondly, autism is strongly associated with mental retardation. In fact, three-quarters of autistic people are mentally retarded. Furthermore, the more retarded the child the greater the likelihood that he or she will also be autistic.

Now, mental retardation stems from some sort of general brain damage or malfunction. The retardation itself cannot cause autism, because many retarded children do not show the symptoms of autism. Down's syndrome children, in particular, are often very sociable. We must assume, then, that the damage that causes retardation will sometimes also cause autism. In addition, in a quarter of cases, we find autism without retardation. This pattern might sound complicated, but we can readily account for it in principle. One model that fits the pattern suggests that some specific brain system, *B*, is necessary for normal, or nonautistic, development. We can view brain damage, in the sense I have used it, as comprised of randomly distributed lesions. The greater the damage, the higher the probability that area *B* will be damaged. This is probably too simple a model, but it serves as an illustrative account.

Our theories, then, must give room for a major biological contribution to autism. Until recently, no one has been able to delineate a causal chain between any biological damage and the complex of symptoms in autism. Now, Uta Frith and her colleagues at the MRC's Cognitive Development Unit have bridged the gap.

In brief, Frith's theory claims that the core problem with autistic people is that they are born with a cognitive deficit—that is, a deficit in some aspect of learning or reasoning. This deficit forces the child onto a deviant path of development from which the complex of symptoms derives. Before tracing this path we need to consider some aspects of normal development. We have a particular interest in how the outside world is experienced by the child, for instance, what kinds of memories the child can create. Learning involves representing events and knowledge concerning the world, and we need to make a distinction between primary representations and metarepresentations.

In "pretend" play, a two-year-old infant pretends that a banana is a telephone and remembers this play the following day. In other words, the infant has a representation of the event in her memory. The memory *<the banana is a telephone>* is a metarepresentation and must at all costs be kept separate from the memory *<the banana is good to eat>* which is a primary representation. One difference between primary and higher order representations lies in the inferences that one can draw. If you know in addition that *<bananas are fruit>* and you also learn that *<oranges are fruit>* when your



Autistic children lack a "theory of mind": they cannot attribute false beliefs to other people, for instance, and so predict, in this puppet show, that Sally will look for the marble in the box

mental machinery will automatically work out that *<oranges are good to eat>* but would not conclude that *<oranges are telephones>*. This is because we treat metarepresentations as exempt from confirmation from other knowledge or from the world; by the same token we do not use them to generate new inferences.

Not all play is pretend play. Take, for example, the case of a child who plays with a toy saucepan on a toy stove, making the kind of movements, for example stirring and shaking, that mother makes on the real stove. This may be merely functional play, a re-enacted memory. However, if the child sniffs and says that there is bacon frying and varies the play according to the dish, then this is pretend.

Alan Leslie, also at the Cognitive Development Unit, has pointed out that the properties of mental representations in pretend play resemble those involved in the use of terms for mental states, such as "believe", "wish", "intend". As a child I may know that *<the moon is made of rock>* and can represent that idea in my memory. At the same time, I can represent the idea *<the moon is made of green cheese>* without contradiction, as long as I ascribe it to someone else, in a form such as "Jamie believes that *<the moon is made of green cheese>*". What is true for beliefs is also true for desires and intentions. Although wide awake, I can represent the idea *<I should go to bed>* without internal contradiction as long as it is embedded in some frame such as "Mother wants . . . ". The use of mental state terms such as these "wants" or "believes" is the manifestation of our ability to represent the contents of other people's minds. The representations involved need to be separated from primary knowledge as they

are not subject to our normal attempts to verify them. They, too, must be secondary.

As normal infants develop, they acquire a set of related skills that can collectively be termed "mentalising"—the ability to predict relationships between external states of affairs and other people's internal states of mind. These skills vary in complexity. The most basic is to represent that other people have knowledge at all. This ability does not require metarepresentations.

But to pretend, or to represent other people's intentions, some basic metarepresentations are needed. These are firmly in place by two years. A more complex ability is to represent someone else's false belief. Suppose you know that your sister has hidden a chocolate in a box. Mother finds it and moves it to a drawer. Your sister comes in. You know *<the chocolate is in the drawer>*. What about your sister? She has a representation *<the chocolate is in the box>*. You must represent her belief as a metarepresentation, otherwise it will conflict with your own knowledge. If you cannot create metarepresentations of this form, you will imagine that your sister *knows* where the chocolate really is. (Note that we take it for granted that normal people cannot simultaneously believe something and its contradiction.) Heinz Wimmer of the University of Salzburg and Josef Perner from the University of Sussex in 1983 investigated the ability to create the kind of metarepresentations which allows you to represent false belief. They showed that we cannot rely on this advanced use of metarepresentations in the normal child until about the age of four.

So much for normal development. What about autism? Well, Frith had noted that autistic children never pretended or lied and, in the light of Leslie's theory, she hypothesised that they were incapable of producing metarepresentations. If this were true, they would not be able to properly understand and use terms such as "believe" nor would they be able to represent other people's false beliefs. With Simon Baron-Cohen, Leslie and Frith went on to test this hypothesis. The basic test was in the form of a puppet show. Sally, one of the puppets, has a marble. She puts the marble in a basket and then leaves the room. Ann, the other puppet, takes the marble out of the basket and puts it in a box. She leaves. Sally comes back. She wants to play with her marble. Where will she look?

We have no problem in knowing that Sally will look for her toy in its original hiding place despite our knowledge that it is not there. This means that we can represent Sally's false belief in our memory as well as representing the true state of things. Normal children can do this task by the time they are about four. Down's syndrome children with a mental age of six can also answer correctly. However, out of a group of 20 autistic children, with a mean mental age of nine, 16 failed the task although they could answer correctly a variety of questions about what happened. They knew where Sally had put the marble, they knew that it was Anne who had moved it and that Sally had not seen the move. Their problem lay neither in memory nor language. Basically, the autistic children could not conceptualise the possibility that Sally believed something that was not true. This is exactly the prediction that Frith and her colleagues made on the basis of the hypothesis that autistic children cannot form metarepresentations.

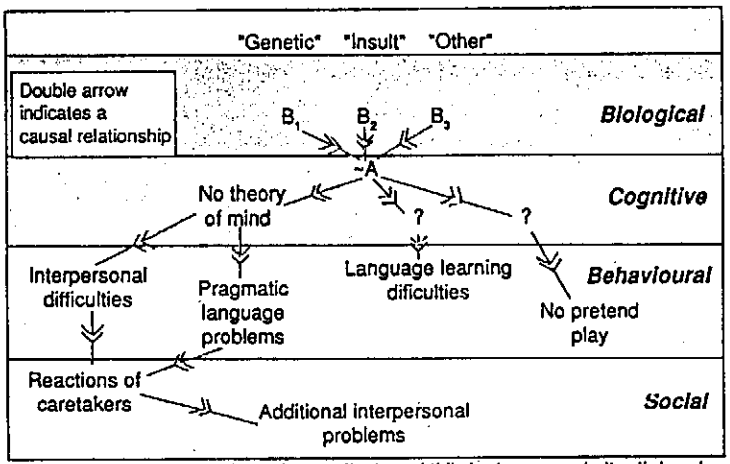
The researchers repeated such experiments several times, and also modified them so that two experimenters took the place of the puppets. The results remain the same. The few autistic children who pass this test tend to be well in their teens and more intelligent. However, they still fail another, slightly more complex problem of the same kind, which any normal eight-year-old can solve and which should have been well within their intellectual capacity.

If we grant that autistic children cannot form and use metarepresentations, how does that account for the whole complex of symptoms? Without such representations autistic

From brain damage to social isolation

THE CAUSAL chain underlying autism is shown in the figure. The first elements are the precursors which leave the infants with their own particular pattern of biological deficits. We have referred to *genetic, insult* (meaning externally caused physiological damage) and *other*. These categories are intended to be portmanteau ones, each with an unspecified number of subdivisions. These precursors lead to biological deficits, labelled $B_{1,3}$. These might correspond to lesions in some part of the brain, some biochemical deficit affecting, say, a neurotransmitter, or the absence of particular connections between two areas of the brain. By definition, all these deficits lead to the loss of the ability to form metarepresentations, *A*.

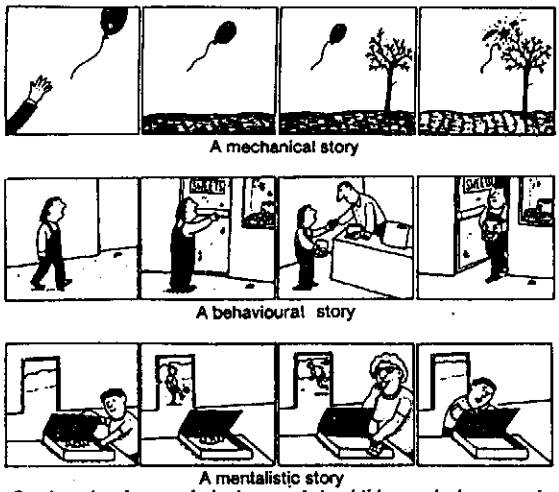
The mark ~ indicates that something is missing. The absence of *A* leads to developmental problems, such as the acquisition of language, and to some permanent deficits, such as a theory of mind. My colleagues and I have also noted in the figure that the abnormality of the child could lead to reactions on the part of those around, which could in turn lead to other problems with relationships. In this way, environmental factors can interact with the biological and cognitive causal chain. The figure indicates four areas in the causal space, dividing the elements into biological, cognitive, social and behavioural. The behavioural space includes the three main features of autism, satisfying the requirement for a theory of autism which I laid down earlier.



A model for autism: an in-built abnormality in a child's brain may underlie all the other characteristics of this complex developmental disorder

If we reject the idea of a biological basis for *A*, we are left with the need to build other causal chains between the known precursors and the lack of *A*. Note, also, what happens if we ignore the cognitive aspects of our causal theory. We would then have to build a series of causal bridges between the biological base and the characteristic aspects of behaviour.

One small problem with the figure as it stands is that it omits certain perceptual and motor characteristics of autism. We need to establish whether they arise from the cognitive deficits or will require a separate causal chain arising from a different biological basis. □



Comic strips for psychologists: autistic children asked to put the pictures in the right order managed the top two, but failed on the story that relies on the attribution of mental states

individuals are unable to create a "theory of mind". This means that they are unable to understand that other people have their own beliefs, desires and intentions which guide behaviour. Without such knowledge social interaction is extremely limited. One autistic man is reported to have observed in amazement that "other people talk to each other with their eyes". Another complained that "other people seemed to have a special sense by which they could read other people's thoughts". Our social interactions are guided by implicit rules from the very beginning. The child who breaks one of these rules or tries to change them by being wilful nonetheless knows about them. The normal child (with approximately normal parents) does not need to be conditioned, through reward and punishment, to learn social rules. These rules are part of the representation the child has of the parents' minds which is independent of the child's representation of the physical world. The earliest form of these representations appears to be present by the child's first birthday.

The proposal, then, is that there is a cognitive deficit underlying autism which amounts to an inability to create certain kinds of representation, particularly of other people's mental states. Triggered by studies of pretend play, the theory successfully predicts an acute limitation in the ability of the autistic child to mentalise, which in turn would lead to an inability to achieve normal social understanding. What of the communication problems? Much of the language we use depends upon appreciating the other person's point of view. Without such ability, we would experience the acute difficulties of communication found in autism.

The final question is how such a deficit would account for delay in the acquisition of language which is a common feature of autism. Very briefly, learning a language depends on categorising utterances correctly. It isn't just a question of what mummy says, but what mummy intends. If a child bangs her plate, mummy might say "plate", "don't bang", "be patient", "I'm coming" or a variety of other things. To know what kind of utterance has just been spoken, the child must take into account mother's state of mind and possible intentions. The autistic child will not be able to do this and so will have a much more difficult task. Frith, in her recent book, *Autism: Explaining the Enigma*, has traced this strand of the causal chain in more detail.

I have here sketched a causal chain from an inability to form metarepresentations to the major symptoms of autism. The origin of the problem is cognitive. Does this rule out the possibility of a biological basis for autism? No. On the contrary, Frith and her colleagues believe that the cognitive skill that is lacking is an innate property of the normal human brain. It is not learned. Indeed, it is doubtful whether the ability to form metarepresentations could be learned. The lack of the skill, then, cannot be due merely to a failure of learning or lack of motivation. It is not a failure of the cognitive apparatus to take advantage of its opportunities but rather a fault in the machinery itself.

Links in the causal chain

Frith has postulated a single cognitive deficit as the cause for autism. A variety of biological causes many produce the cognitive deficit. While we might not be able to specify these, it is clear where they fit in the causal chain (see Box).

Autism is a biological deficit leading to a developmental disorder that unfolds inexorably as the child's biological make-up influences its interactions with others. As such, it contrasts with schizophrenia which appears later in life (usually in the late teens) after an apparently normal early development. While many people have sought a psychological account of the onset of schizophrenia, the current view is that it is the result of what can be labelled a "developmental biological" disorder. With autism, the biological component only defines the starting point.

What are the implications of this analysis for therapy? By and large, therapy aims to affect only factors that are downstream in the causal chain. Because the in-built cognitive deficit in autistic children leads to secondary problems, such as the reaction of parents and subsequent emotional reactions by the child, therapy focusing on these factors could be helpful. But claims that "holding therapy" or similar treatments can "cure" autism indicate either a misdiagnosis (far from uncommon) or misunderstanding of the underlying nature of the disorder.

The emotional by-product of the development of autism may sometimes be the most serious aspect of the condition, and many types of therapy can alleviate such aspects. Beyond that, therapists can help the children to learn language; autistic children may have difficulties in this area simply because they have insufficient individual tuition.

However, the problems with understanding metaphor in language, and the interpersonal difficulties which are at the heart of the autistic condition, will not be cured by anything other than cognitive means. Note that in our tests which delved into a child's "theory of mind", a few of the most intelligent autistic children gave the correct answers. Our most pessimistic view is that all autistic children lack an innate ability that cannot be learned. The successes are due to the child having "hacked out" a solution to a subset of the problematic situations. The best therapy, then, would probably involve systematically presenting the child with a large number of social situations together with the normal resolutions. The idea would be that for each type of situation, children would learn an algorithm which would enable them to predict what could happen next even if they never understand it. This is rather like a child learning that other people like to have "conversations". The child determines a few stock questions to use under suitable circumstances but without really knowing what conversations are about.

When we are feeling more optimistic, we hope that in autistic children the necessary structures are there but have not yet been "hooked up" within the brain. If this is true, therapists should present children with relatively pure demonstrations of "theory of mind" principles that are just beyond their cognitive reach. The experiments illustrated above make a good starting point. □

Professor John Morton is head of the Medical Research Council's Cognitive Psychology Unit in London. **Further reading** *Autism: Explaining the Enigma*, by Uta Frith, Basil Blackwell, Oxford, 1989, £8.95 paperback.