Why study autism?

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I first met autistic children as a trainee clinical psychologist, and I was captivated for life. I thought them hauntingly mysterious. How could they do jigsaw puzzles straight off, and yet never even respond to my simple requests to play with them? What was going on? How could they be tested? Here was a challenge that cried out for basic research.

My mentors, Beate Hermelin and Neil O'Connor, knew how to do elegant experiments with children who hardly had any language and were more than a little wild. I was elated when they offered to supervise me, and I got my dream job in their lab after I finished my PhD. I was hooked on the experimental study of cognitive abilities and disabilities in young children with autism and I wanted to know how they differed from other children. One of the innovations that O'Connor and Hermelin had introduced me to was the mental-age match. They argued that comparing bright and intellectually impaired children would get us nowhere. The brighter would do better, and this told us nothing that we didn't know already. Instead, they compared, say, 8-year-old children who on psychometric tests had a mental age of 4, with 4-year-old, typically developing children with a mental age of 4.

I was proud of one memory experiment I did during my apprenticeship as a PhD student. We observed that autistic children often had a remarkable facility in remembering words by rote. This allowed us to compare autistic and non-autistic children who had the same short-term memory span. What we found gave me a key insight: Typically developing children could remember many more words when

these words were presented in the form of sentences than if the same words were presented in a jumbled up fashion, but autistic children failed to show this advantage. I followed up this finding in experiments with binary sequences, with clear structure, e.g. abababab vs those without, such as aababaaa. The results suggested that structure or 'meaning' allowed stimuli to be packaged into bigger units and thereby extended memory span. Did autistic children not see meaning in the way other children did, I wondered? Did meaning not exert the same dynamic force in their information processing?

This question occupied me for a long time. Some years later, it became a theory that I termed 'weak central coherence.' Briefly, the information we process is usually pulled together by a strong drive to cohere. We like things to make sense, we like a narrative, we like the big picture. In autism, I proposed, this drive is less strong. The downside is that individuals with autism do not see the forest for the trees. But there is also an upside: Not being hampered by a strong drive for central coherence could actually give you far better attention to detail. You are not lured away by an overall Gestalt to forget about its constituents, and you won't fall prey to certain perceptual illusions. For the first time, here was a way to think about autism not just in terms of disabilities but also in terms of special talents.

As I was developing this idea, I was worried that in all our experiments we were missing the social features of autism. My search for a glitch in processing social information would have been a hopeless quest, had it not been for Alan Leslie and Simon Baron-Cohen. Alan had asked the exciting question how young infants were able to understand pretend play while they were still learning about the real world.

How on earth could they distinguish which was what? This reminded me of a finding nobody had paid much attention to: Autistic children show little, if any, pretend play. Alan proposed a cognitive mechanism that could underpin the ability to decouple representations of an event so that they could become second-order representations. They could then be freely embedded into an agent's mental states: the agent can wish, pretend or believe the original event. Could it be that the decoupling mechanism was missing in autistic individuals? In that case, they should not be able to understand that another person can have a false belief.

Why should this matter? Beliefs and other mental states, such as pretense, wishes, and knowledge, are what enable us to predict what others are going to do. We don't predict this on the basis of the physical state of affairs. So, John will open his umbrella because he believes it is raining, regardless of whether it is actually raining. Tracking mental states is grist to the mill of our everyday folk psychology, also known as Theory of Mind. To be able to talk about this ability, we coined a new word, *mentalizing.*

Simon, Alan, and I were excited to find out more about this ability. One of the tasks we developed was the Sally Ann task. It is played out with two dolls, Sally and Ann. Sally has a marble and puts it in her basket. She then leaves the scene. While she is out, Ann takes the marble from the basket and puts it into her box. Sally comes back and wants to play with her marble. The critical question is: 'Where will she look for the marble?' The right answer is, of course, 'in the basket', because that is where she believes it is.

The results amazed us, as they were so clear cut: Typical 4-years olds and older learning disabled children passed this task, while autistic children didn't. They had failed to understand that Sally had a false belief and therefore made the wrong prediction of where she was going to look. This and other experiments threw new light on the social communication problems in autism: If you don't understand mental states, then you wouldn't understand deception nor get the point of most jokes. You wouldn't get the point of keeping secrets, nor would you understand any narratives that depended on 'she doesn't know that he knows' scenarios. It would limit ordinary social interactions in just the way that interactions with autistic people are limited.

With the advent of the new neuroimaging methods, we could now try to visualize this cognitive mechanism in the brain. One of the pioneers in neuroimaging was my husband, Chris Frith, and he and his colleagues were sufficiently interested to set up a then still daring series of studies. We designed stories, cartoons, and animated triangles, which could be presented in carefully matched conditions, which either did or did not require mentalizing. This difference allowed us to see a difference in brain activity in several critical brain regions, forming a mentalizing network. Other labs were able to replicate this.

One disappointment was that we could not immediately see what was different in the brains of autistic people during mentalizing. But to unravel this required many studies by many people in many different labs. This led us to a better understanding of mentalizing, and has already resulted in differentiating two forms: an apparently innate and unconscious form, and an acquired conscious form that is influenced by

culture. This second form can be acquired by autistic people through compensatory learning.

Is there a lesson from my studies beyond the world of autism? I believe that the studies have demonstrated the usefulness of the cognitive level of explanation. The purely behavioral level is not sufficiently transparent for us to deduce the underlying causes; there are just too many. But, we can predict what behaviors might arise if a particular cognitive process were faulty. This was the point of the Sally Ann test: Nobody before had observed that autistic children failed to understand false beliefs. The beauty of this result was that it suddenly made sense of a range of hitherto unconnected behavioral observations, such as the poverty of pretend play, the inability to tell lies, and the incomprehension of irony.

Our concept of autism has changed enormously since the 1960s. There are likely to be many different phenotypes hidden in the autism spectrum. It is now time to split up subgroups and relate specific cognitive processes to specific causes, in the brain and in characteristic patterns of behavior. Mentalizing is not all there is to being social. There are other cognitive processes that underpin our social behavior that might be faulty and give rise to different problems and possibly different forms of autism. We simply need the right theoretical glasses to see differences in the spectrum, which are now blurred. Whether these subgroups conveniently map onto specific biological causes is another question. It is likely that there are hundreds of genetic and other biological causes, too many to make meaningful subgroups. At the behavioral level, each individual is in a class of his or her own. In contrast, at the

cognitive level, there is a nexus, which might hold a manageable handful of phenotypes. My money is on cognition.

Further Reading

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