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Why we need cognitive explanations of autism

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In the 70 years since autism was described and named there have been huge changes in the conceptualization of this enigmatic condition. This review takes a personal perspective on the history of autism research. The origins of the first cognitive theories of autism, theory of mind and weak central coherence, are discussed and updated to inform future developments. Selected experimental findings are interpreted in the historical context of changes that have been brought about by advances in methodology. A three-level framework graphically illustrates a causal chain between brain, mind, and behaviour to facilitate the identification of phenotypes in neurodevelopmental disorders. Cognition is

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placed at the centre of the diagram to reveal that it can link together brain and behaviour, when there are complex multiple mappings between the different levels.

Keywords: History; Cognition; Mentalizing; Detail focus; Compensatory learning.

Seventy years have passed since the first descriptions of autism, and it seems appropriate to begin with a historical perspective on autism research. Having witnessed the beginnings of an experimental psychological approach to autism in the 1960s, I am taking my own experience as a guide for this review. This makes it a narrative account and a subjective one at that. I try to convey some of the toil and excitement of a rocky journey that started off in uncharted waters and led me and my colleagues to some of the first viable cognitive theories of autism.

I was there. I can act as an eye witness of the changes that have taken place in our approaches to autism. This is unusual as most of the highly cited autism researchers are relative newcomers to the field (Feinstein, 2010). Oddly enough, my own lifetime coincides more or less with the presence of autism in the scientific and popular literature. Autism was first identified and labelled by Kanner (1943) and Asperger (1944) during World War II, and understandably at the time the papers were not particularly noticed. I was growing up in a small town in Germany, not so different in age from those first-described cases.

So if you choose to read this review, you will be taken on a personal journey, and you should expect to get a story, rather than an impartial history. Throughout I aim to provide context rather than detailed facts. The latter, after all, can be found in published papers (for a bibliography of my own papers see Bishop, Snowling, & Blakemore, 2007). I hope that my personal account will encourage others to go on future explorations of cognitive processes, because these processes forge the vital links between genetics, neuroscience, and psychology. This exploration has a powerful lure, to find the answers to deep and difficult questions about mind and brain that only autism poses.

Stepping back in time

In June 1968 I experienced one of the happiest moments of my career: Neil O'Connor, newly appointed director of the MRC Developmental Psychology Unit at UCL (University College London), invited me to become a member of his staff. The unit was in a basement flat in an imposing building at the corner of Euston Road and Gordon Street. But there was plenty of space for all: Neil O'Connor, Beate Hermelin, Rick Cromer, myself, a couple of PhD students, a secretary, and a technician. A few more people, research assistants and visitors, joined later. The ethos required that we generally did our experiments ourselves, in schools and hospital units. We wrote in long hand, and only the secretary had a typewriter. New and exciting technology was appearing in the form of a huge Xerox machine for copying. In my own small office, brightened by a big Alphonse Mucha poster and a prolific vine, a sleek Olivetti calculator replaced a clunky mechanical one.

Few people realize that our research on autism at that time was done in a miniature world with very limited resources. After all, autism was considered a rare disorder, with a presumed prevalence of 0.04% (Lotter, 1966), and we thought we were acquainted with most of the autistic children in London. Today autism research occupies a vast and interdisciplinary field, and recent population studies put the prevalence at around 1% (Fombonne, 2009; Parner, Schendel, & Thorsen, 2008). Yet, the research produced in this doll's house broke new ground. The idea that specific cognitive deficits could be key to explaining different developmental disorders was new and is still controversial today. One particularly exciting question was whether specific developmental deficits give clues to the existence of separate cognitive systems in the developing brain.

We never thought then that we would be able to see inside the living brain of autistic individuals, and we despaired of the minimal amount of information that had come from postmortem brain analyses. Nevertheless, although we did not have the tools to study the *brain*, we believed we had tools to study the *mind*. And here was a particularly bold step in the logic of the approach: If brain and mind are the same thing, only seen from different methodological perspectives, it should be possible to identify the *causes* of autism with these tools. Amazingly, such work had to be done without any knowledge of the genetic factors or of the brain pathology of autism, without even agreed diagnostic criteria. Despite this seemingly overwhelming handicap, cognitive studies that were then starting to be done gave substance to the idea that autism was a neurodevelopmental disorder and created the confidence to identify genetic factors and brain pathology as opposed to psychodynamic explanations. The concept of neurodevelopmental disorder itself is still in development, but perhaps historical studies will show that psychological research on autism was the early testing ground for this concept.

In pursuit of information-processing models of the mind

Beate Hermelin and Neil O'Connor, my PhD supervisors, had forged a unique research partnership (for some light-hearted reminiscences on the "avengers of psychology" see U. Frith, 2009). They summarized their seminal work on autism in their book in 1971 and called it simply "Psychological experiments with autistic children." (Hermelin & O'Connor, 1971) It is full of examples of the brilliant application of new paradigms originating from information theory in a field where rigorous experiments were extremely rare. My mentors' idea was to learn about the human mind by studying severe learning disabilities. This may strike many even today as very risky. Still, there were others who had similar goals. Intriguingly, it was the work of Soviet so-called defectologists and of Alexander Luria that had influenced Neil O'Connor. Even though plane travel was still relatively rare, there were also visits from experts on what was then called mental retardation, such as Eric Schopler, Herman Spitz, and Herbert Birch. Beate Hermelin recommended that I read Bernard Rimland's book on the neuropsychology of autism (1964), which was a big inspiration for my work.

One influence on the approach taken by the members of the MRC (Medical Research Council) Developmental Psychology Unit was the then-emerging field of cognitive neuropsychology. Here were interesting paradigms from studies of acquired lesions that could be applied. But of course we did not expect to find the same cognitive deficits in children whose brain pathology had started from before birth as in adults whose brain pathology had started much later in life. We expected instead to learn about plasticity and why it was so hard for these children to compensate for some of the cognitive skills that they were missing through pathology. This question has remained unanswered and is in urgent need of future explorers. But what was the cognitive root of the complex impairments evident in children with developmental disorders? The first theories arose from the idea that these complex impairments were due to faulty sensory (input) processes. Lower level sensory processes might be the cause of "higher level" difficulties through the process of development itself.

This idea seems plausible, but what if it was the other way round? Higher level difficulties might have effects on lower level perception, just as in the now well-accepted effect of context on perception. This effect is now explained in terms of a Bayesian computational model (Yuille & Kersten, 2006), but here a simple example will suffice. In order to hear a word you need to have some idea of what it is you might be hearing. For example, if somebody introduces himself uttering a name you have never heard before, then you may well feel as if you are slightly deaf and ask for the name to be repeated. At this point you might have formed a rough expectation of what kind of name it might be, and now you can hear it properly. Back in the late 1960s I was yearning to study "higher level" processes whose existence could be guessed at by such examples. These sorts of ideas had long been discussed by Gestalt psychologists and had something of a revival at the time, notably in Ulric Neisser's *Cognitive Psychology* (1967).

Beate Hermelin first suggested that my PhD project should address motor problems in autism, something that had not been studied before, and something that could be compared and contrasted with perceptual problems. But I could not help feeling slightly disappointed by such an assignment, which seemed rather dreary to me. Instead I wanted to emulate an experiment by Hermelin and O'Connor (1967) that had made me an ardent admirer of their work. In this study, autistic children were asked to recall strings of words presented in arbitrary order ("what–to–went–ship–sea"), and this was compared with recall of word strings making up sentences ("what–ship–went–to–sea"). When words, instead of being scrambled, are presented in the form of meaningful sentences, then recall is normally much better. Remarkably, this gain was much smaller for autistic children than for other children who were tested as control groups. I was so fascinated by this finding that I did the experiment again with some added twists, and it became my first published paper (Aurnhammer-Frith, 1969).

I did this and other experiments at a school in Ealing, in a large Victorian house, headed by Sybil Elgar, a now legendary teacher of autistic children. This is where I spent many days, often playing fly-on-the-wall in the school and in the playground. The children—as well as the teachers—were absolutely fascinating, and they are still vivid in my memory. Several children never spoke at all, some showed echolalia, a phenomenon that was much more prevalent then. One lovely girl, D., could sing beautifully and spoke in a rather high-pitched monotone. Though generally smiley, she seemed quite oblivious to me when I tried to interact with her. Nevertheless, she often came willingly with me to be tested in a room set aside for experiments. To me the distinct smell of disinfectant in the corridor that led to it is still very evocative. D. seemed happy to repeat the nonsensical word lists of different length and structure that I had devised, and she did not show the memory bonus when the lists were meaningful. Did she not get the meaning? But what was meaning, and how did other, much younger, nonautistic children get it? And how could it be connected with autism?

In my PhD thesis, I tried to address this question by means of patterns that might or might not be detected in binary sequences (U. Frith 1970a, 1970b, 1970c). I thought that the detection of a pattern would lead to the process of chunking, so that fewer, information-richer elements had to be stored. This chunking would lead to the memory bonus. The memory bonus was present in ordinary children and in children with mild intellectual disabilities but not in most autistic children, even if not all performed as perfectly as D. I concluded that children with autism were less good at pattern detection, and—with a great leap of the imagination—that hence they were less good at extracting meaning in general. I read about Bartlett's experiments and loved his phrase "effort after meaning" (Bartlett, 1932).

When further experiments confirmed the early findings (Hermelin & Frith, 1971), I was greatly encouraged. It seemed in principle possible to explain a wide range of impairments in autism by an inability to make sense of the world. I was also very keen to try out the idea that, normally, context could be used to enhance the perception of meaning. In a thought experiment, I made up a test with sentences like these: "He played the lead guitar", "The box was made of lead". You would probably notice nothing at all when reading these sentences aloud. But how did you know how to pronounce "lead" in each case? Presumably you extracted the meaning in a flash before you pronounced the word. The actual experiment had to wait several years before it was carried out with the help of Maggie Snowling (U. Frith & Snowling, 1983). The experiment was later modified and replicated by Francesca Happé (1997). In both cases, autistic children tended to say "leed". In other words, they made less use of the sentence context to disambiguate the key word. The results gave some support for the idea that autistic children had greater difficulty processing information in context. But, as for an inability to extract *meaning*—it was not quite as simple as that.

It is clear that the theory was far too open-ended. Besides, there were other explanations for any difficulties with extracting meaning. For instance there might be problems in the mastery

of language. However, there is a crucial distinction, highlighted by my much-missed friend and colleague, Richard Cromer (1981), between language and its *use*. It was the latter that was problematic in autism. And if you considered the mastery of grammar, apparently well within the grasp of at least some of the children I observed, then this definitely required pattern detection. Actually, the recognition that there could be false trails laid by rashly interpreting experimental findings as due to problems in particular domains, such as language, motivation, or memory, steeled me to having to abandon such trails and liberated me to seek out novel and plain risky hypotheses.

Inspired by the brilliant work of my first PhD student, Maggie Snowling, on dyslexia, I devoted some of my research time to dyslexia, but not as a diversion from my interest in autism. Here was another developmental disorder with a basis in the brain. This idea had to be fought for even more than was the case for autism. But the developmental derailment in dyslexia leads to quite different behavioural symptoms. After a time I realized that to analyse behavioural symptoms with respect to a cognitive—and eventually neural—explanation was the right approach for both disorders. Studying the behaviour by itself would perhaps lead to a continuous refinement of psychometric tests and checklists of signs and symptoms. But there would be no principled way of distinguishing primary and secondary or merely associated problems. However well standardized a psychometric test, the fact remains that it is performance and not competence that is being measured, and performance is open to influences from such factors as motivation, fatigue, or familiarity. To me, a bottom-up approach from elements of behaviour had little attraction. I was disillusioned by inconsistent results of behavioural tests —both in the case of dyslexia and in the case of autism. The inconsistency was serious since the clinical groups I tested seemed to be increasingly heterogeneous. In contrast, what exactly would a top-down approach succeed in doing? A framework was needed that would give guidelines to identify a number of separate questions that might be amenable to empirical research. This was just the task that John Morton would support.

Social cognition and theory of mind

In 1982, Neil O'Connor retired as director of the Developmental Psychology Unit, and John Morton became the director of a new MRC Unit, the Cognitive Development Unit (CDU). It soon moved to a bigger building, with several floors and new colleagues and students. I was extremely fortunate with my gifted PhD students during the 1980s and 1990s: Tony Attwood, Simon Baron-Cohen, Francesca Happé, Amitta Shah, and Digby Tantam. They all made their mark on the now more rapidly developing field of autism research. John Morton encouraged me to sharpen up my still vague ideas. Consequently I focused on the, to me at the time, most nagging question: the coexistence of very poor social skills and very good rote memory skills.

But what exactly was the nature of the poor social skills? There were widely divergent views. In popular opinion at the time, the label "autistic" was synonymous with being globally asocial. For example, a highly emotional TV documentary by Desmond Wilcox (The Visit, BBC, *ca*. 1982) portrayed a bleak picture of autism. It showed a distressed mother struggling to embrace an even more distressed autistic child, who desperately fought the embrace. Eventually, after a very long struggle, the child would relax and meet the mother's gaze. This moment was celebrated as a triumph, as breaking through a barrier to reestablish normal attachment. This "holding therapy" was heralded as a cure. However, it was soon discredited as ineffective and even cruel, and the underlying theory that problems in mother–child bonding and subsequent failure of attachment were the cause of autism turned out to be very wrong (Capps, Sigman, & Mundy, 1994; Shapiro, Sherman, Calamari, & Koch, 1987).

There were many ideas about the roots of the social impairment in autism, and there still are. The main question remains how can such ideas be made amenable to experimental investigation? I always came back to the puzzle of how it was that processing information about the physical world could be so good, while processing information about social world could be so poor. If so, this

meant dividing up hypothetical cognitive functions along some unconventional line, a line that I talked about almost constantly with my new young colleague Alan Leslie. He suggested that the dividing line was created by the ability to form metarepresentations: to "decouple" primary representations of physical reality (it is raining) to become the content of an agent's mental state (John *believes* it is raining). Alan Leslie laid out this theory in persuasive detail in a paper that appeared in 1987. He showed that the ability to form metarepresentation is evident in the second year of life and is responsible for young (typically developing) children being able to understand and enjoy pretend play (Leslie, 1987).

Alan Leslie's ideas hit a nerve for me. They spoke to a surprising finding that robustly distinguished autistic from nonautistic children: extremely poor performance on a standard symbolic play test (Wing & Gould, 1979) where a set of miniature wooden toys allowed children to enact simple scenarios such as using a cup to give a drink to a doll. This finding greatly intrigued me. But what did not being able to engage in pretend play have to do with not being able to interact socially with other people? Leslie's hypothesis made the crucial link: Not being able to understand that another person "pretends" had the same cause as not being able to understand that another person "intends", "knows", or "believes". Clearly, without reference to such psychological states the behaviour of other people would be inexplicable. This was a novel way to look at the social impairments in autism. It entailed a thorough spring clean of older ideas of global and pervasive social failure.

The name for the circumscribed cognitive process that was now seen to underpin certain aspects of social interaction was "theory of mind"—or ToM—and the name has stuck. However, it did not trip off the tongue. A verb had to be coined to refer to "the automatic attribution of mental states, such as desires and beliefs to self and others to predict and explain behaviour". John Morton suggested "mentalizing", and I used this term in my 1989 book on autism (U. Frith, 1989). This book also summarized the work that had led up to formulating and testing the theory that a fault in mentalizing was responsible for the particular failure of social interaction and communication in autism. A recent review of ToM in neurotypical development by Apperly (2012) shows that research in this field is still very active.

Since the field of social cognition was only just emerging (Humphrey, 1983; Premack & Woodruff, 1978; Byrne & Whiten, 1988), there was a dearth of experimental paradigms. By fortunate coincidence, Wimmer and Perner's (1983) now-classic false belief paradigm was published just at the time that Simon Baron-Cohen was ready to start experiments for his PhD thesis, and Alan Leslie was developing his theory. I remember excitedly reading the paper and discussing with my young colleagues how we could make the false belief task suitable for autistic children. Accordingly, we (Baron-Cohen, Leslie, & Frith, 1985) designed the Sally–Ann experiment. The results were remarkably clear, showing that the majority of autistic children did not predict Sally's behaviour on the basis of her false belief, but instead on the basis of reality, while the majority of the children in the comparison groups performed much like the children tested by Heinz Wimmer and Josef Perner. We were amazed. Almost immediately, we (Baron-Cohen, Leslie, & Frith, 1986) devised another quite different paradigm to ask the same question. Here children had to complete a story in the form of a sequence of pictures, and the story involved the inference of either mental states or physical states. Again the results were clear: Children with autism struggled to understand stories that depended on mental events but not those that depended on physical events. The "mentalizing deficit" hypothesis was born—but it would be some time before it grew up.

After the overhaul of our ideas on the social impairments central to autism, a completely new picture came to light. It was exhilarating to see that not all kinds of social interaction are impaired in autism. We contrasted one set of social behaviours, those that required mentalizing, with another set of social behaviours, similar in all respects except that they did not require the ability to mentalize. We called this the "fine cuts" method, and here are some examples:

- Autistic children are capable of sabotage, but not deception (Sodian & Frith, 1992).

- They show and understand instrumental gestures, but not expressive gestures (Attwood, Frith, & Hermelin, 1988).
- They understand seeing but not knowing (Leslie & Frith, 1988).
- They can tell about a fact if asked, but not discriminate whether this fact was novel or already known to the listener (Perner, Frith, Leslie, & Leekam, 1989).
- They can judge what a rotated object will look like, but not what it will look like from another person's point of view (Hamilton, Brindley, & Frith, 2009).

We also found evidence for similar contrasts in everyday life behaviour (e.g., they could be eager to please, but not be able to choose an appropriate present; Fombonne, Siddons, Achard, Frith, & Happé, 1994; U. Frith, Happé, & Siddons, 1994).

In my enthusiasm for the new theory, I frequently overlooked that mentalizing problems can only account for a fraction of social impairments. Social cognition has many more tricks up its sleeve than mentalizing—for example, imitation, emotional contagion, conformity, stereotype formation, social facilitation, reputation management, agency detection, to name only a few. Any or all of these might be impaired in the hugely heterogeneous collection of individuals now diagnosed autistic. What can be the cognitive basis for such variety? These questions are being actively researched now.

Nonetheless, the mentalizing theory was in many ways iconoclastic. It disregarded previous work and previous ideas about the social difficulties of autistic children, and it placed a risky bet on something that had only recently been proposed and only just been made amenable to experimentation with young children. It was not surprising that this novel hypothesis was not accepted readily. Was it set at too high a level, rendered superfluous by more basic impairments (such as a sensory difficulty or a subtle difficulty in the control of movement)? If so, this would be contrary to my belief that it was only by probing high-level cognitive processes that a plausible link could be made to faulty brain circuits.

Further experiments in the 1980s put more substance to the initial claims, as summarized in my 1989 book (U. Frith, 1989; with a revised second edition appearing in 2002). It was tempting to think that mentalizing really was a singular cognitive function underpinned by a singular brain function, and that this brain function was faulty in autism. This would fulfil the long-held hope that modules of the mind might be discovered by studying neurodevelopmental disorders. We sometimes used the name "mind blindness", evoking the analogy to word blindness or object blindness, conditions known to have an origin in acquired brain lesions (Baron-Cohen, 1995; U. Frith, 2001).

In the 1990s, after positron emission tomography (PET) scans had been first used to investigate cognitive functions, the theory became amenable to testing (Fletcher et al., 1995; Happé et al., 1996). But the safety restrictions of PET meant only small numbers of volunteers were scanned. However, as soon as functional magnetic resonance imaging (fMRI) became available, some obstacles to scanning individuals with autism spectrum disorder were removed. There are now replications of the early studies (using varied tasks: cartoons, story vignettes, animated shapes) that revealed the brain basis of mentalizing. They further demonstrated that, for individuals diagnosed with autism, the major components of the brain's mentalizing system are indeed working less well and less in concert (for a review see C. D. Frith & U. Frith, 2006) than those in neurotypicals. The remarkably robust results suggest that the superior temporal sulcus, the temporo-parietal junction, the medial prefrontal cortex, and the temporal poles are all part of a network that is active whenever mentalizing is required.

The idea of a ToM deficit in autism has generated a huge number of empirical studies, and this has led to a number of theoretical modifications (Baron-Cohen, 1995; Leslie, German, & Polizzi, 2005). Many other authors took up the challenge of brain imaging while participants were performing tasks that required reflecting on one's own and others' mental states in neurotypical participants and those with autism (see Van Overwalle,

2011, for a review). Neuropsychological studies of patients with lesions in the brain regions associated with mentalizing also showed failure on typical ToM tasks (for a review see Martín-Rodríguez & León-Carrión, 2010).

Nonsocial cognition and weak central coherence

Of course, this was far from a complete explanation of autism. There are many features of autism that have nothing to do with social interaction, and these were also crying out for an explanation (U. Frith & Happé, 1994). At the time, few ideas from general cognitive psychology were ready to be used to explain these features. Could typical expressions of autistic behaviour, such as restricted interests, mysterious savant abilities, repetitive behaviour, and hypersensitive responses to sensory stimuli be explained by a single cognitive cause? Intriguingly, these features point in fact to unusual cognitive *strengths*, and this was something to celebrate. It even helped overturn the idea that autism is a pervasively devastating disorder.

However, given the problems that many autistic children experience in their everyday lives, I assumed that the cognitive strengths did have a downside as well. But what was the cause? Bartlett's "effort after meaning" still resonated with me and gave rise to the question: What if an individual's drive to discern meaning in the world is weak? Perhaps meaning can be conceptualized as a "whole" in the way the Gestalt psychologists proposed? If so, would a *weak* drive free the mind from the Gestalt laws to which neurotypical perception is shackled? Would it be possible to ignore context so that basic elements stood out that would otherwise simply be suppressed? If so, the world would surely look shockingly different —one would forever see the trees, but not the wood.

I labelled this idea weak central coherence. It proposes that in an autistic brain, while the ability to discern a wide variety of things about the world around is strong, the drive to make these various things cohere is weak. The term was never intended as a value judgement, but it has frequently been objected to on the grounds that something that was able to explain mental strengths should not also be referred to as a mental weakness. An interesting aspect of the theory is that it is not modular like the mentalizing theory. Instead it supposes two general information-processing mechanisms for piecemeal bottom-up and holistic top-down processing in all sensory modalities.

Here is a test that can serve as an illustration of what I mean by weak central coherence and how it can be associated with superior performance. It was based on the Block Design test, which forms part of the well-known Wechsler Intelligence Scales (Wechsler, 1974), and where three-dimensional blocks have to be used to build a pattern that is shown in a two-dimensional drawing. My outstanding PhD student Amitta Shah, who had already shown that autistic children excelled at finding embedded figures (Shah & Frith, 1989) used two types of drawing, as shown in Figure 1 (Shah & Frith, 1993). One type of drawing presents the overall shape only; the other has dividing lines superimposed, which show exactly how individual blocks map onto parts of the design. The two comparison groups found the task much easier when dividing lines were present; they finished their block constructions much more slowly

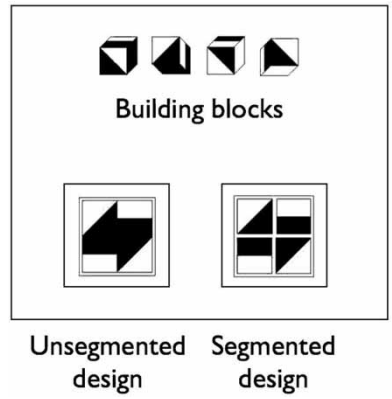

Figure 1. *Examples of unsegmented (left) and segmented (right) design used in the Block Design experiment by Shah and Frith (1993). Autistic children were almost as fast at assembling blocks for unsegmented designs (65 s on average) as for segmented designs (45 s). Children in the control groups took far longer to assemble unsegmented designs (95 s vs. 45 s).*

when only overall shape was shown. However, on this unsegmented version of the task, the autism group performed much better than the controls. Furthermore, they did not show much of a speed gain in the version where dividing lines were present. This result seemed to me closely parallel to the result of the recall tests with nonsensical versus meaningful word strings from my PhD years. But this task shows the other side of the coin. In the case of Block Design, strong coherence—that is, prioritizing overall Gestalt—is a hindrance, while in the case of recall of sentences, prioritizing meaning is a help.

The notion of weak central coherence was one way to account for both strengths and weaknesses of ability as measured by standard IQ tests, and about which we had considerable data. What we saw very typically was an advantage for Block Design and Matrices tests and a disadvantage for Comprehension or Picture Arrangement tests. Weak central coherence lends an advantage in the former two tests because segmentation into parts is helpful, but is a disadvantage to the latter two tests, because here holistic understanding is helpful.

My proposal was rightly challenged, and modifications have become necessary to adjust to findings gained from the much larger and more heterogeneous population of autistic individuals that we know about today. Still, weak central coherence is one of the few existing notions that dares to explain a variety of otherwise unrelated nonsocial features by means of a single cognitive mechanism. Narrow interests, savant abilities, and repetitive behaviours flourish through focus on detail and piecemeal processing, while hypersensitivity to sensory stimuli may stem from a neglect of contextual information.

A framework of three levels of explanations for neurodevelopmental disorders

During my time at the CDU, John Morton insisted that I should explicitly consider the place of cognition in models of cognitive disorders such as autism. This was vital to transform intuitive guesses into hypotheses, especially important as the concept of autism was undergoing great change at the time. In this situation, drawing back from behavioural descriptions was necessary. After all, behaviour, however reliably it is measured, is not revealing about its cause. There is a mapping problem. Many different causes can underlie the same behaviour. On the other hand, behaviour that looks different in different individuals may actually be due to one and the same cause. In this sea of uncertainty, cognitive explanations are like a seaworthy ship to navigate between facts we know about the brain and facts we know about behaviour (Morton & Frith, 1995).

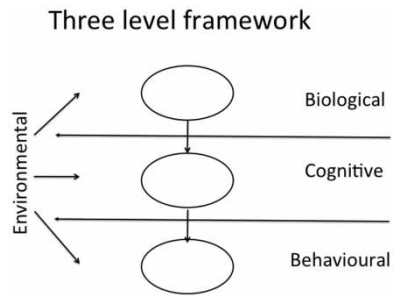

Figure 2. *The most basic form of the three-level framework, defining "cognitive" as the link between brain and behaviour. Given an origin of a developmental disorder in biology the arrows go down towards cognition and from there to behaviour as the visible manifestation of brain abnormalities. The environment interacts with all three levels.*

Figure 2 shows the basic framework. It separates phenomena and their explanation, and it defines the level of cognitive explanation through its location. We can observe behaviour in autism; we can take into account the facts already known about the brain in autism, but these observations are not the same as explanations. To have explanations, hypotheses are necessary, and these tend to be leaps of the imagination. The framework binds these leaps tightly from two directions. First of all they must be a good fit with multiple behavioural observations. Second they must not merely give a new name to a behavioural observation. It would be nonsense to try to explain the behavioural observation that a child does not tell lies by proposing that the child lacks "the ability to deceive", unless this ability would also explain some other previously unrelated behavioural data. Third, the leaps of imagination must fit in with

what we know about brain physiology and evolution. If a cognitive mechanism is claimed to exist and to be vulnerable to abnormality, then there has to be a neural substrate that underpins it. Also, a functional or structural abnormality in this substrate should be discernible, and in an early emerging disorder this may be due ultimately to a programming fault at the genetic level. This is not likely to be a one-to-one mapping. Many different types of genetic and nongenetic risks can be envisaged and are likely to affect a whole range of neurophysiological mechanisms. Yet, they may all converge on a single cognitive mechanism.

Figure 3 illustrates the idea of a many-to-one mapping from brain to cognition and one-to-many mapping from cognition to behaviour. This results in a typical X-shape, and this captures a typical causal chain in the cognitive explanation of the social impairments of autism through mentalizing failure.

The first application of the three-level framework, in a preliminary form, was to understand more precisely what the mentalizing deficit entailed (U. Frith, Morton, & Leslie, 1991). It captures a singularity at the cognitive level, and this level is intermediate between the biological and behavioural levels. At the biological level a variety of causes could be envisaged, and at the behavioural level a whole range of puzzling, and at first seemingly unconnected, behaviours was accounted for by a mentalizing deficit. Crucially, to avoid circularity, new behaviours have to be predicted. This was the case for poor understanding of the Sally–Ann false belief task, an entirely new phenomenon. Figure 4 shows a more explicit model of how mentalizing failure is captured at the behavioural level by a large range of behaviours that at first glance seem unrelated. Evidence for impairments was obtained not merely in clinical anecdotes, but through experimental procedures. The figure lists some of the twinned tasks we used and that produce a "fine cut" between otherwise similar behaviours. The same framework can be applied to dyslexia and indeed to other neurodevelopmental disorders (U. Frith, 1998; Morton, 2004). The three-level framework is a tool that reminds users that *descriptions* are not *explanations*, and that there is a need to link behaviour and mind, just as there is a need to link brain and mind. The gap between behaviour and brain is huge, and inferences from one level to the other are fraught with difficulties. This is why we need the cognitive approach (Morton & Frith, 2001).

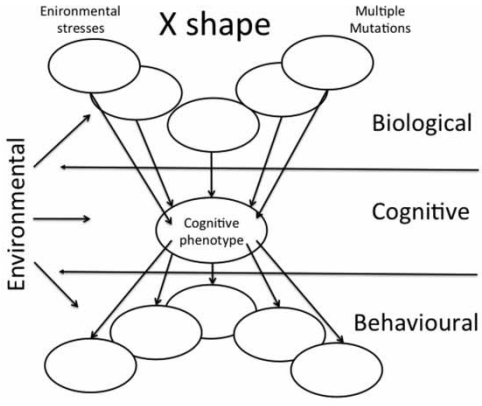

Figure 3. *The X-shape of the three-level framework illustrating a cognitive phenotype where there is multiple mapping between brain and behaviour via a singular node at the cognitive level. Any developmental disorder is likely to have more than one specific deficit, but for simplicity only one is shown in the diagram.*

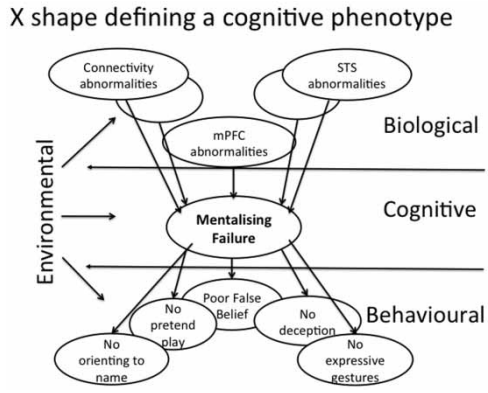

Figure 4. *The X-shape of a "cognitive phenotype" for mentalizing failure, allowing for multiple mapping between brain and behaviour. Some putative brain abnormalities and behavioural signs of mentalizing failure are shown for illustrative purposes.*

From autism to Asperger syndrome

My most striking experience as an eye-witness to the still evolving story of autism comes with the

huge changes in what autism is believed to be like. The shadowy picture that was painted of autism in the early days was very black, settling on mental retardation and psychosis. Now we are aware of the huge individual variation in cognitive ability and severity of symptoms with shining examples of high-achieving autistic individuals. At first we could only vaguely guess that autism would not go away when the children grew up. Now we know much about autistic adults, but we still know very little about the effects of old age on autism. This is also true about infancy and very early childhood.

When I started in 1966 as a PhD student at the Institute of Psychiatry, researchers and clinicians studying autism only saw children, mostly under 10 years of age—and only children exhibiting a "classic" type of autism. I was aware of the intriguing question of whether there was a hidden intelligence in autistic children. I was also aware that this was considered a romantic myth. Instead autistic children were then thought to belong to the large group of disorders that were defined by measurable and often massive intellectual impairments.

But autistic children were different from children with general intellectual impairments in subtle ways. Their key problem seemed to lie in poor language understanding. I remember discussions over tea in the Common Room: If we only could instil language, then we would overcome the social and communication difficulties. We would overcome the sometimes terrifying behavioural problems that must arise from frustration over the impossibility to communicate. Wrong, of course, but actually, I still like the idea of making a huge difference to autistic individuals by providing them with conscious strategies when implicit processes fail to work spontaneously. I believe that, in principle, explicit conscious strategies *can* be taught to enable compensatory learning of many different skills. But this is a more modest proposal.

The initial concepts of autism were dramatically changed in the second half of the 20th century (cf. Rutter, 1968, 2000; Wing, 1981), and the Institute of Psychiatry was the place to be to witness these changes. Besides my admired PhD supervisors, Neil O'Connor and Beate Hermelin, there were other prominent pioneers of autism: Lorna Wing and Michael Rutter. Imagine how surprising it was, in the 1970s, to find more and more autistic children who had fluent language and also high measurable intelligence. I remember when Lorna Wing literally opened my eyes to see that not all autistic children were aloof and withdrawn. As she put it so vividly, some were distinctly 'active but odd' (Wing & Gould, 1979). Imagine how exciting it was to learn about the first genetic study in autism (Folstein & Rutter, 1977). To me this put the nail in the coffin of the notion that autism was a psychogenic disorder (i.e., only in the mind). Now we were strengthened in our belief that autism had a biological basis and originated from before birth (i.e., in the brain and consequently in the mind).

As time passed, it became clear that autism was a lifelong disorder and that there were autistic adults too. Indeed awareness of autism was popularized by the adult portrayed by Dustin Hoffman in the film "Rain Man" (1988). The notion of Asperger syndrome proved an even bigger challenge to concepts of autism. When I translated Asperger's (1944) paper into English (U. Frith, 1991) and annotated the translation to bring out its relevance to autism researchers and clinicians, I had no idea that this would reach a large and receptive audience. It occurred to me for the first time that some potential readers might themselves have Asperger syndrome, while writing the introduction to the book. With some trepidation I addressed a section directly to them. In the same volume, Happé (1991) explored content and form of the first autobiographical writings of people with Asperger's syndrome. Nowadays there are so many books by and for people with autism or Asperger syndrome that a whole new genre has evolved (see Draaisma, 2010, for a review).

Part of the magic of a diagnosis of Asperger syndrome is that it does not paint a severe picture of a potentially devastating disorder. It does not seem like a disorder at all. It acknowledges, and even expects, a different (but not deficient) mind, and often the presence of superior talent. Today we are still debating this issue: Are all conditions

subsumed under the autism label to be seen as disorders in need of remediation, or simply as personality differences? I admit to having it both ways. I believe that the autistic mind is different. I also believe that autistic features, such as social impairments, or obsessive restricted interests, are found in the general population. I have toyed with the idea that many scientists, including myself, show the obsessive detail-focused characteristics of autism. This does not mean that the border between "autism" and "not autism" is hopelessly blurred. The person with autism does not just have the behavioural features of autism, but has different cognitive machinery and hence sees and experiences the world differently at a very central and personal level.

Revisions and updates

As my subjective personal account is coming closer to the present time it is becoming more difficult to identify the most promising advances. I am staying biased by focusing on the work of my own former students and colleagues, but of course cognitive studies of autism are now carried out all over the world, and on a global scale. This brings up an interesting problem. How can we compare studies across time when the diagnostic criteria for autism have widened considerably?

I believe that the children I tested up to the 1980s now only constitute a small subgroup of children typically participating in experimental studies today. It is evident that experimental psychologists nowadays prefer to study older children and adults, and that individuals with average or above-average ability are preferred as participants. This has a number of interesting consequences. One is that certain behaviours are no longer apparent because able adults have found ways of compensating for earlier difficulties. This does not mean that the underlying cognitive difficulties have disappeared. Instead they have to be revealed by more sensitive tests.

In the following section, I want to sketch briefly where our understanding of autism is today in relation to the two cognitive theories that I presented. The shortcomings of these two theories becomes clearer as time moves on, and perhaps one of the most glaring limitations is that they presuppose an "essence" of autism, manifest in unique social and nonsocial phenomena. What if there is no such essence? Having received a very old-fashioned German education, I am compelled to look for a Goethe quotation. In *ca*. 1780 Goethe wrote an "essay on nature", a romantic vision with many contradictions. It seems to me that I share more of this vision than I would like to admit. Here is a quote that seems to fit my dilemma:

> Each of her [nature's] works has an essence of its own; each of her phenomena a special characterization: and yet their diversity is in unity. (p. 313, Vol 30 Cotta'sche Ausgabe in 30 volumes, 1858)

Theory of mind today

As one would expect with any theory that has been going for about 30 years, a number of problems with the theory of mind deficit hypothesis have arisen, and this will no doubt continue until a new theory comes along. One of the most interesting problems was the finding that older and more able children and many adults diagnosed as autistic were perfectly able to pass the Sally–Ann task (U. Frith, 2004). A meta-analysis of a number of studies suggested that the age at passing the test still had a 5-year delay for those with autism compared to neurotypicals (Happé, 1995). Vivid examples of late success can be seen in the many excellent blogs that parents write about their autistic children. For example, one lovely piece reported how an autistic boy, at the age of 12, suddenly discovered lying. This provided a moment of glee for the mother because it proved to her that her son could mind-read, and that she could shout out "so much for the theory".

My own take on this story is very different. It glaringly reveals the huge delay in understanding lying, and it suggests that the mind-reading had been arrived at by means of compensatory learning. I don't doubt that the boy now shows an understanding of lying, but this is not the same as the spontaneous ability to mentalize. To me it seems that there is a gulf between mentalizing learned the hard way and mentalizing learned effortlessly in the neurotypical way. Still, the mother's

contempt of the theory is justified if it had implied that nothing could be done, and her son would forever be unable to understand that there are mental states and how they cause behaviour. It is important to spell out that autistic individuals can learn these things, and that they learn all the time. Most people find that their ability to cope with social interactions improves as they get older. This is equally true of people with autism.

Another criticism of the theory is its silence about the social impairments of autistic children in early childhood. As Hobson (1989) and Mundy, Sigman, Ungerer, and Sherman (1986) pointed out, autism is manifest before the age of three, yet the Sally–Ann test is not passed by neurotypical children until 4 to 5 years. In fact, both these criticisms were addressed once the Sally–Ann paradigm was successfully brought into the infant lab. A new version of the test showed that mentalizing in implicit form is within the capacity even of very young infants. Here, the test is presented in a non-verbal form, and the infant's response is measured in terms of prolonged looking (in surprise) when Sally searches in the place where she could not know the toy now is (Onishi & Baillargeon, 2005). Likewise it could be measured in terms of anticipatory eye movements to the place where Sally must believe the hidden toy to be (Southgate, Senju, & Csibra, 2007). Even infants as young as 7 months (Kovács, Téglás, & Endress, 2010) were influenced by the belief of a protagonist, as revealed in their looking time.

The critical step was then to use these implicit tasks to test autistic adults who were able to pass standard ToM tasks, having learned to mentalize over time. Would they show a tell-tale lack of anticipatory eye gaze? Remarkably this *is* what we found (Senju, Southgate, White, & Frith, 2009). While neurotypical adults correctly anticipate, with their spontaneous gaze, where Sally will search for her hidden toy, adults with autism do not. Thus, it is the early-appearing, *implicit* form of ToM that points to a core problem in autism. This will need to lead to some rethinking of experimental stimuli in the future: Most standard ToM tasks are geared to detecting problems with explicit mentalizing, while implicit mentalizing tasks are scarce. For example, the Frith–Happé animations, which have been widely used in behavioural and neuroimaging studies of mentalizing (Castelli, Frith, Happé, & Frith, 2002; Castelli, Happé, Frith, & Frith, 2000), probably include both implicit and explicit components.

Of course I am aware that apart from mentalizing, myriad other neurocognitive mechanisms have been proposed to underlie the social impairments of autism. And unlike the earlier attempts, some of these are informed by brain science. One example is the idea proposed by Pelphrey and colleagues (see review by Kaiser & Pelphrey, 2012) that deficits in perception of biological agents and their intentions are key. These are thought to be due to malfunctioning of the posterior part of the superior temporal lobe (superior temporal sulcus, STS), a region known to underpin agency perception, and a region that is also implicated in the mentalizing network.

Emotions and empathy

Some critics have claimed that the emphasis on theory of mind led to a neglect of examining emotion and empathy—two key aspects of social interaction. Feelings are themselves mental states, and mentalizing is surely relevant to the expression and recognition of feelings. However, we are dealing here with an unfortunate side effect of the meaning of the word "cognitive", which is often held to exclude anything affective. This is not the way I understand cognition, which to me refers to *all* mental life. In the mind, thoughts and feelings have equal currency, and furthermore they are completely intertwined. It is obvious that autistic individuals have strong emotions, such as high anxiety and angry frustration, but they don't always express these feelings in conventional ways. This leads to the controversial question: Are they aware of their own emotions, and indeed of their own self? Until quite recently, theory of *own* mind, which would appear to be critical for self-awareness, was underrepresented in autism research but there is a new impetus to address just this concept (Happé, 2003; Lombardo et al., 2010; Williams, 2010). One especially intriguing issue is

the relationship between mentalizing and empathy. Ever since parents began to give me examples of empathy they observed in their children, I have been gripped by the idea that an impairment in mentalizing does not entail a lack of resonance with other people's feelings (U. Frith, 1989). It was many years later when it proved possible to test this idea experimentally and to demonstrate a separation of the neurocognitive processes underlying theory of mind and empathy (Blair, 2005, 2008; Jones, Happé, Gilbert, Burnett, & Viding, 2010).

Awareness of own feelings can be assessed by means of questionnaires (containing items such as *I don't know if I am feeling tired or angry*). High scores on such a questionnaire suggest a condition, which has been termed "alexithymia", literally "no words for feelings". We showed that about 50% of the adults with autism who had volunteered to take part in our experiments suffered from alexithymia (Hill, Berthoz, & Frith, 2004). Yet, alexithymia also occurs in people who are not autistic. This allowed for an experimental design of a neuroimaging study where autism and alexithymia were independent factors (Silani et al., 2008). Participants saw pictures of pleasant and unpleasant content and were asked to rate how colourful they were, or, how unpleasant they made them feel. The various contrasts between these conditions produced the following findings: First, everybody showed the same effect in the amygdala when feelings were aroused. Second, only participants without alexithymia showed activity in the anterior insula, a brain structure known for its involvement in bodily awareness. Third, only participants without autism showed increased activity in the regions of the mentalizing system during the introspection condition. From these results we could infer that while *all* our autistic participants *had* the feelings, only about half *were aware of* the feelings, and *none were fully aware that they owned* these feelings. Of course one can talk of "owning" feelings only if one presupposes a self that owns them. These speculative conclusions point to a metacognitive process that is important for self-awareness. It may turn out that precisely this process is impaired in autism.

What about empathy? Tania Singer used her landmark paradigm (Singer et al., 2004), which by its ingenious design reveals brain structures that are active in response to a sharp transient pain that is experienced either by themselves or by a partner. The results were clear: There was no difference between autistic and neurotypical participants, provided they did not suffer from alexithymia (Bird et al., 2010). On the other hand, participants with alexithymia, lacking awareness of their own feelings and showing low activation of the anterior insula, also lacked awareness of others' feelings and lacked an emotional response to these feelings.

Lack of empathy is an unfortunate and inaccurate stereotype of autism. Instead lack of empathy can be said to lie at the heart of a different neurodevelopmental disorder, psychopathy (Blair, 2005). Autism and psychopathy are very different conditions, and one of the main differences is in the ability to mentalize (Blair, 2008). Individuals with psychopathy can mentalize exceedingly well despite their lack of empathy—indeed they use their good mentalizing to manipulate others. Unfortunately, a "double hit" is possible, although probably rare: a person who cannot mentalize spontaneously and also lacks empathy (Rogers, Viding, Blair, Frith, & Happé, 2006). Future comparisons of different neurodevelopmental disorders will no doubt throw more light on the "fine cuts" that may exist in cognitive processes and guide the search for distinctive neural signatures.

Weak central coherence today

The autism community embraced the idea of weak central coherence with great enthusiasm—despite its label. This is not a scientific endorsement of course, but it suggests that it has captured something important that is relevant to understanding autism in daily life. From the mid 1990s onwards, Francesca Happé has been leading the revision of central coherence theory (Happé, 1999). She left behind the idea of a deficit and proposed instead a normal distribution of preference or bias, with a detail-focused strategy at one extreme of the population and the opposite preference at the other.

Clearly, most people would be in the middle of this distribution with no particular preference for either of these strategies. From here it was only a small step to suggest that a detail-focused information-processing style might be typical of the broader autistic phenotype. Accordingly, we conducted a study of parents of children with autism, and parents of children with dyslexia or no disorder as a comparison group (Briskman, Happé, & Frith, 2001; Happé, Briskman, & Frith, 2001). We found that approximately half the fathers, and a third of the mothers, of autistic children—but not those of dyslexic children—showed evidence of a detail-focused processing style. For example, they were faster at finding embedded figures. Most of these parents did not show any signs of social impairment and had never been considered to be on the autism spectrum themselves.

In a review paper several years ago now, Francesca Happé and I were surprised that there were some 60 studies all testing predictions from the weak central coherence theory (Happé & Frith, 2006). We were impressed by the theory's ability to generate debate but we remain agnostic as regards possible underlying neural mechanisms. We are still at the stage of speculation. For example, there is the idea that feedback connections from frontal parts of the brain do not match up to feedforward connections from the posterior parts of the brain (C. D. Frith, 2003). A critical factor may conceivably be a lack of synchronization of neural activity that might be necessary to bind parts into wholes (for a review see Müller et al., 2011).

An interestingly different cognitive account of detail focus is in terms of theories of attention. For example, work by Rinehart, Bradshaw, Moss, Brereton, and Tonge (2001) and Mann and Walker (2003) indicates that it is difficult for autistic individuals to switch their attention away from local detail to global view. Yet, at the same time it *is* easy for them to switch attention away from the global view towards the local detail. Sarah White, another of my talented PhD students, also found this difference in switching attention, but in a completely different task (White, O'Reilly, & Frith, 2009). Furthermore, she showed that a bias towards detail focus may only apply to a subgroup of autistic individuals. Interestingly this subgroup had a brain-based difference—larger head size. Replications of these results are now in progress, and it should be fascinating to see how the theory will develop at the neural level when neuroimaging results become available.

Whatever the underlying neural processes, I am betting on the idea that top-down control of the flow of information in the autistic brain is weak, and I apologize for using the w-word again. I imagine this weakness is tantamount to a reduced influence of prior expectations. This means that bottom-up processes are relatively stronger. One of our brain imaging studies (Bird, Catmur, Silani, Frith, & Frith, 2006) gives some support to the idea that in autism top-down influences on visual perception are weaker than in neurotypicals. When presented with a location cue and very briefly presented images of either faces or houses, neurotypical brains show enhanced activation in precisely the area that is relevant for processing a particular type of stimuli. So the fusiform area of the brain that is concerned with face processing is more active when faces appeared in the indicated location. The same happened in the area that is relevant for processing houses, when houses appeared in the critical location. This modulation of relevant brain areas occurs entirely without awareness and is not associated with improved performance. In line with our hypotheses, we found significantly less of this modulation in autistic brains.

In the introduction to *Autism and Talent* (Happé & Frith, 2010) we argued that detail focus was a possible engine for savant talent. Detail focus could well be the outcome of a dominance of bottom-up processes, which capture raw sensory input, over top-down control processes, which impose prior expectations. As a result of such a dominance, creative and original takes on the world are possible, uncontaminated by social convention.

Conclusions

If my assessment of the current state of autism research is valid, then there is still reason to believe that a small number of cognitive mechanisms can explain a large number of phenomena. A

lack of implicit mentalizing defines a circumscribed cognitive phenotype and details quite specific consequences for everyday social communication. Detail focus seems to capture something about the way autistic people see the world and the nature of their special talents. There does not seem to be a close relationship between these two types of cognitive processes, and indeed recent twin studies suggest that sociocommunicative and nonsocial facets of autism are fairly independent (Happé & Ronald, 2008).

How can cognitive theories guide future research? Can they help us clarify and disentangle the different causes of inconsistent results in experimental studies of autism? I think so, but this is no easy task. Given that there are now large data sets and large samples of participants available, it should be possible to test specific hypotheses about subgroups of autism. However, there is also another cause of inconsistent results, namely the unreliability and insensitivity of our current assessment instruments. Unfortunately, the existing experimental tasks are treated as if they were psychometrically validated tests. In fact, such validation still has to be done with most of the tests initially designed to target theory of mind and central coherence, and this is an urgent task if such measures are going to be used to correlate with potential biomarkers.

Autism can serve as a model for studying other neurodevelopmental disorders. One insight we can hold onto is this: Whatever the brain abnormality in these disorders, it does not prevent learning. In autism, it seems that learning enables the acquisition of *explicit* mentalizing even though *implicit* mentalizing has failed to develop. It would be fascinating to trace the changes in the brain that are brought about by this acquisition, a case of the mind, with external support and teaching, affecting the brain.

Finally, it is obvious from this personal account how much more we know now about autism then when cognitive studies of autism started in the 1960s. When I started out doing autism research, there were only a handful of centres worldwide where such research was carried out. Today, autism research is huge, and there are several whole journals dedicated to the field. Now there are many fruitful theories addressing the signs and symptoms of autism. These deserve separate attention and are not included in this personal account. Indeed the field is so large that a comprehensive review would be a daunting task. Instead I am hoping that the work that I have reviewed here will soon be seen as belonging to the prehistory of autism research.

The progress of scientific knowledge may be slow at times, but it is exhilarating to see the difference new technology makes. In my career two such moments were the use of computers, and the use of scanners. Just imagine how another technological invention could revolutionize our methods. We still don't know the biological causes of autism; we know far too little about the life course from childhood to adulthood and old age, and the role of individual differences. We are still in the dark ages as far as educational interventions are concerned. We even still argue about the diagnostic criteria of autism. There is much work to do, and I hope future researchers will take courage from my story and set out with confidence on the rocky road ahead. New ideas about the mind and brain will redraw our knowledge about autism and will ultimately lead to a better understanding of ourselves.

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