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Book Review

A mature view of autism

Autism: Explaining the Enigma by Uta Frith, Blackwell Publishing, 2nd Edn, 2003. £15.99 (pbk) (249 pages) ISBN 0 631 22901 9

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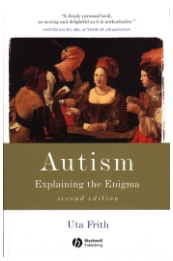

Uta Frith's new, second edition of her beautiful book on autism is very welcome. The first edition, published in 1989, was very well received by both the academic community, practitioners in the autism field, and by families. It was reprinted 10 times in paperback, and has been translated into many languages. How could she improve on it?

And yet she has. She recognized that in the intervening decade, autism research had blossomed, and that this new edition was an opportunity to see quite how well the two main theories that she is well-known for have fared, in the light of a lot of new data.

First, take the mindblindness theory (that people with autism are unable to imagine what other people are thinking). Frith's view is that in the past decade, this theory has been strengthened, and she reviews the evidence in detail and in very accessible style. Further, she goes beyond the extreme picture of mindblindness to hint at those people with autism-spectrum conditions who have 'compensated' for their lack of what she calls an 'intuitive' mentalizing ability by developing what she calls a 'conscious' mentalizing ability. Her conclusion is that now we have a cognitive explanation of the key triad of impairments, shown in Fig. 1a.

Frith then considers the weak-central-coherence theory. In the first edition of her book, she proposed this new idea to explain a set of symptoms in autism that the mindblindness theory could not explain: the narrow attention to detail, the 'islets of ability' or even talents, and the detachment from, or immunity to, context. A decade later, her conclusion is that this theory too has fared well in the light of further experimentation, such that we now have a cognitive account of the 'non-triad' symptoms (Fig. 1b).

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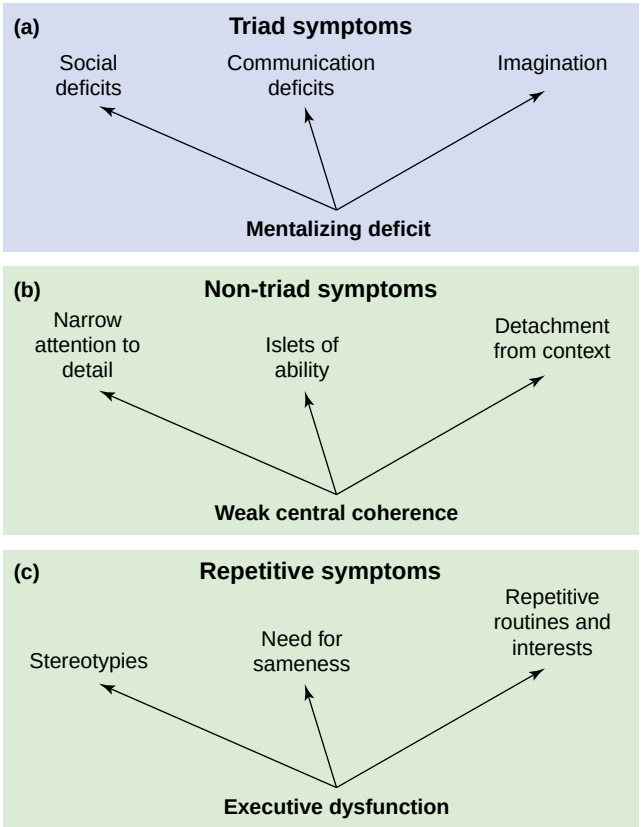

Fig. 1. Symptoms of people with autism, as grouped by Frith.

Since 1989, a third theory has been the focus of autism research, namely, the executive-dysfunction theory, and Frith gives this theory equal space in her new edition. In particular, she argues that an executive dysfunction is posited to be responsible for symptoms such as repetitive routines and interests, stereotypic movements and thought processes, and a need for sameness. Her conclusion is that we now have an explanation for why people with autism are drawn to and show repetition (Fig. 1c).

Overall, she argues persuasively that all three theories are needed in order to account for all the symptoms that need to be explained in autism. And she has done the field an immense service to have written such a readable yet scholarly book, which integrates the three cognitive theories into a single picture of autism. This book is a tour de force.

There are other changes from the first to the second editions. In her masterly review Frith has picked up on all of the other new developments in the autism field since 1989. Autism was once considered rare (~4 in 10 000 children) but now is considered common (~1 in 200); it was once considered categorical but now is considered a spectrum condition; it was once considered to be strongly associated with learning difficulties, but now this is questioned. Autism was once thought of as a modern condition of childhood. Finally, it was once considered to be psychogenic, but the advent of structural and functional neuroimaging demonstrates beyond any doubt that autism is a neurodevelopmental condition.

Each of these new ways of seeing autism is given careful discussion. Regarding its history, Frith argues persuasively that there have almost certainly always been individuals, both children and adults, who had a form of autism. Neuroimaging studies of autism now receive a whole chapter, in which she provides both a clear tutorial on what neuroimaging can teach us, and details the evidence for the brain basis of mentalizing, and the differences in the autistic brain (e.g. in the left medial prefrontal cortex, amygdala, and the fusiform face area).

The impression this book gives is of a field that has reached maturity. And without question, Frith has been driving this field for the last 30 years. Her contributions, which come through in this second edition, are clear: elegant experiments, bold theorizing, and an unstoppable curiousity. In reviewing her book, I am also struck by what a privilege this is for me personally, at two levels. First, Uta Frith has lived out a model of how to supervize and encourage the next generation of researchers, through many individuals, and I am honoured to count myself as one of them. Secondly, this book summarizes the development of her own thought over a whole career, and whilst one might agree or disagree with each individual idea, it is breathtaking to be presented with the whole picture.

This leads us back to her notion of weak central coherence. Frith clearly shows through her writing and her science that she can move effortlessly from the local to the global, checking specific detail whilst never losing sight of the larger goal. Her argument is that people with autism lack this strong drive for coherence, and are instead only able to become curious about fragmented sets of information. Readers of TICS will know that my alternative perspective – the 'empathizing–systemizing' theory – differs from hers in a few minor respects [1]. As I was invited to combine a book review with a debate, I will try to summarize the differences between these two perspectives as well.

Regarding the mindblindness theory, my personal view is that to focus on the term 'mentalizing' is a little too narrow. For me, mentalizing describes the first stage in empathy: it identifies someone who can identify another's mental states when challenged to do so, versus someone who can go on to the second stage of caring about other people's thoughts and feelings. For me, a term that more clearly covers both of these stages is 'empathizing'. This is defined as the drive to identify someone else's mental states (their thoughts or their emotions), *and* to have an appropriate affective reaction to their mental states.

Frith's model also suggests that problems with imagination are a core feature of autism-spectrum conditions. My own clinical experience of adults with Asperger Syndrome, for example, indicates that many develop interests in imaginative areas, such as science fiction, and some of them use their imagination in their work, for example, in design. Rather, if there is any core difficulty related to imagination, I would say that it is in imagining another's mind. I like to think of these as the first triad of symptoms to be explained, as shown in Fig. 2a. If this is compared with Frith's model (Fig. 1a), it will be seen that the differences are subtle and small.

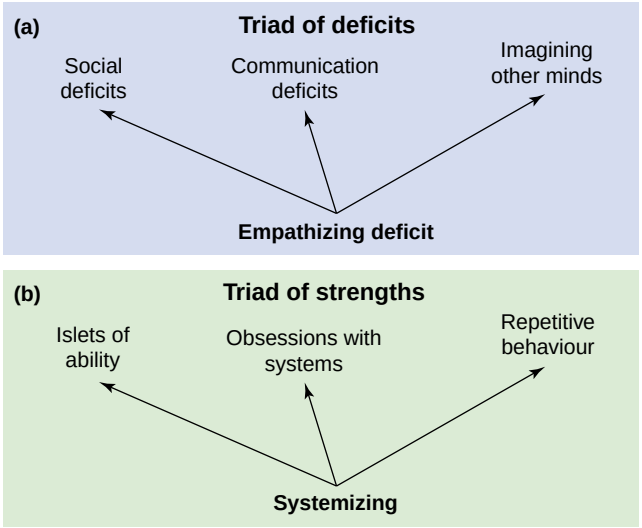

Fig. 2. Triads of symptoms, grouped by Baron-Cohen in the empathizing–systemizing theory [1].

Regarding the notion of central coherence, I read this new edition with a question in my mind, and I find it still needs an answer – at what level should central coherence be taken? At all levels, from the sentence all the way down to the individual letter? From the scene around you all the way down to the objects or the features within objects? Clearly, it cannot be the case that people with autism have weak central coherence at the featural or object level; otherwise they would not see or recognize objects. So although I find the notion of central coherence intriguing, I think it remains to be fully specified.

What of Frith's hypothesis that obsessional interests in autism are simply the result of very local information processing, forever cut off from the bigger picture? My experience of people with autism who become obsessed with a topic is that they are, as Frith suggests, processing the information at a greater level of detail. But I don't think they do this for reasons of weak central coherence. I think they do it in order to understand a topic in depth, to become expert at it, and to understand it as a system. In my terminology, they have a strong drive to 'systemize'. Systemizing is defined as the drive to analyse a system, or build a system (compare Fig. 2b with Figs 1b and c).

I think this drive to systemize can account for what I like to call the second triad characteristics of autism. Systemizing entails a very fine grain of attention, and it allows specialism, creating an expert rather than a generalist. It is why people with autism can develop 'islets of ability' as they become proficient in a system, such as naming prime numbers, or the days of the week any calendar date will fall, or compiling mental lists of birthdates or train timetables.

But as can be seen, I also think one doesn't need to posit the third factor of executive dysfunction to explain core features of autism. My suggestion is that repetitive behaviour – for 40 years described as 'purposeless' – is in fact the very behaviour that one would predict for someone who systemizes to an extreme degree. This is because systemizing is a bit like doing pure science. It is the process by which one tries to understand how a system works, and the best way to do this is to carry out repeatable experiments.

People with autism, I suggest, become captivated by an aspect of reality, and seek to discover the underlying structure or lawfulness of the system, like scientists do. They do this by trying to hold everything else constant, and examining how the output changes as they manipulate one variable at a time. For someone described as 'low-functioning', this might entail running water out of a tap and watching for hours as the water droplets fall in a very specific pattern, as the tap is opened very slightly more. Or it might involve watching the same video over and over again, perhaps hundreds of times, so that one can predict every tiny detail in the next frame: striving for total lawfulness. For someone described as 'high-functioning', this might involve noting how the underlying rock and soil changes as one goes from one part of the country to another. Again, reality becomes entirely predictable, and the laws one discovers become wholly repeatable.

Frith has done an excellent job in reviewing the executive dysfunction theory of autism, but I find that account paints an essentially negative view of the repetitive behaviour of people with autism, suggesting that they repeat their actions because they cannot help it. In a way, I agree they cannot help it, but more in the sense that the non-autistic person cannot help empathizing; people with autism cannot help systemizing, and sometimes it pays off – big time. Despite his Asperger Syndrome, Richard Borcherds (described in [2]), won the Fields Medal for mathematics, the equivalent to the Nobel Prize. To say that his mathematical achievement was the result of an executive dysfunction makes little sense. And to say that it was the result of weak central coherence is to overlook the fact that when people with autism-spectrum conditions such as Asperger Syndrome do in the end achieve expertise in a system (such as their computer, or the syntax of a new language, or the pattern of the weather), it is typically not a fragment of the system, but the *whole* system.

Frith engages with the empathizing–systemizing theory, and suggests that systemizing might be the result of weak central coherence. It is an interesting new twist, but I remain to be convinced. My suggestion is that people with autism try to systemize the whole world, but that some types of information lend themselves to systemizing more easily than others. Information from the social world doesn't – nor do fiction or chatting. On the other hand, train timetables, mathematics, collections of facts, and music all do lend themselves to systemizing, as ultimately they are systems. Good systemizing, just like good science, entails detachment from context. To crack the system one is going to have to start by trying to understand local details, and work outwards. Under certain conditions, this could even give the impression of weak central coherence, if one is zooming in on local features. This raises an interesting new debate: does weak central coherence lead to systemizing, or does systemizing simply give the impression that one suffers from weak central coherence?

In the end, though, these are small points for academic

discussion, and do not detract that this book will be *the* classic text for students, researchers and practitioners in psychology and related disciplines. It will also be indispensable for families of people with autism. In the preface, Uta Frith promises us an insight into her science and her passion, and how these two can be combined, and this reader for one was not disappointed.

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Letter

HERA today, gone tomorrow?

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Habib, Nyberg and Tulving [1] have recently updated their views on the hemispheric encoding/retrieval asymmetry (HERA) model, almost a decade after the idea was first introduced into the memory literature [2]. Broadly speaking, the central tenet of HERA is unchanged; that is, that the left and right prefrontal cortices are disproportionately involved in the encoding and retrieval of episodic memories, respectively. Over the past 10 years, several authors have challenged the model [3–5], and a number of alternatives have been proposed. In their article, Habib and colleagues address these ideas and offer suggestions as to how they might in fact be accommodated within the HERA framework [1].

Although the HERA model has undoubtedly promoted fruitful scientific exchange through the provision of a concrete testable hypothesis, it still leaves many questions about the nature of functional asymmetry in humans unanswered. In particular, the model is based solely on functional neuroimaging data and, to a significant extent, it remains unsupported by data from other methodologies. For example, according to HERA, patients with unilateral frontal-lobe lesions should be differentially impaired at encoding or retrieval depending on the side of their lesion. As Habib and colleagues point out [1], patient studies of encoding and retrieval are often confounded experimentally, although there are certain cases where they might still provide valuable information about these processes. For example, it has been suggested that encoding and retrieval might be assessed relatively independently by testing memory over very short intervals [6], and autobiographical memory (in which information is encoded prior to the time of cortical damage) provides a mechanism for identifying specific encoding impairments in patients (e.g. see [7]). To date, neither of these methods has revealed any robust differences between patients with left and right sided frontal-lobe lesions. Disconnection of the two hemispheres in so-called 'split-brain' patients produces only minor deficits in episodic memory which again suggests that the hemispheric encoding/retrieval asymmetry may be 'more apparent than real' [8].

The data from repetitive transcranial magnetic stimulation (rTMS) studies in healthy volunteers is also equivocal with respect to HERA. For example, in the study by Rossi *et al.* [9], left-sided rTMS during encoding did not disproportionately affect the probability or speed of successful retrieval (relative to right-sided stimulation). The effect of right vs. left-sided rTMS during retrieval reached significance on one of two measures of performance accuracy, and not at all in terms of reaction times.

In short, although each of these alternative approaches is not without its own problems, the lack of significant trends in favour of the HERA model remains at odds with the fundamental nature of the distinction proposed.

However, as Habib and colleagues clearly show, the most significant challenges to HERA in recent years have come from within the functional neuroimaging literature itself (e.g. [3–5], and for review, see [10]). Indeed, in the most comprehensive review of relevant imaging studies to date, Fletcher and Henson [11] concluded: 'The HERA generalization may not be sufficient, however, in that our review included many studies of verbal retrieval that activate both left and right frontal cortex, or even left frontal cortex alone'. In another recent review, Lee and colleagues [10] have reported that between one third and a half of all functional neuroimaging studies of episodic memory encoding do not adhere to the HERA pattern. Of course, at the single study level, there are results that clearly support the predictions of the HERA model, but there are also a similar number of well-controlled, systematic evaluations of the model that do not (see [10,11]).

Notwithstanding these reservations, Habib and colleagues make a number of important recommendations for future research in this area. In particular, they suggest that 'to compute the difference in activity in each hemisphere, the proper reference condition for an encoding task is a retrieval task and the proper reference condition for a retrieval task is an encoding task' ([1], p. 242). The recent functional neuroimaging literature is filled with proposals concerning specialization of function within the prefrontal cortex, although in most cases these claims are based on a single observed association between a particular type of behaviour (or task), and activation in what appears to be a specific brain region. Comparisons between two experimental

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