

## Testing the extreme male brain (EMB) theory of autism: Let the data speak for themselves

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It is a rare pleasure for an author to be invited to reply to their reviewer, so I thank *Cognitive Neuropsychiatry* for encouraging this dialogue. I respect Hadyn Ellis as a cognitive neuropsychologist and I hope that this exchange will clarify some misunderstandings. I also think his review raises some broader questions regarding the intended audience for a popular science book. Perhaps the best way to reply is to go through Hadyn's points one at a time:

1. Hadyn seems to have found it somewhat irritating that I explain at length that sex differences derive from parameters with overlapping distributions. Of course, to a specialist reader, this does not need to be stated at length. But in a popular science book, I needed to avoid the risk of cheap journalism drawing the wrong conclusions, such as “all men are x” or “all women are y”. To guard against this risk, I decided to make the point blatantly clear: That sex differences research only reveals differences between *means*, it says nothing about *individuals*. I would prefer to irritate a specialist by spelling out this very basic point, than risk the book being misinterpreted as stereotyping all males or all females.

2. I was surprised to read his sentence: “Women are empathisers; Men are systemisers”, since these bald overstatements are precisely what I was at pains to *avoid* in the book. My argument has a little more subtlety than that. The (new) claim is that more males than females show a profile of their systemising being stronger than their empathising ( $S > E$ ), and more females than males show the opposite profile ( $E > S$ ). The ratios are about 3:1 (m:f) for the  $S > E$  profile, and about 3:1 (f:m) showing the  $E > S$  profile. So, *difference scores* define these “male” and “female” brain types.

3. Hadyn rightly points out that the “extreme male brain” (EMB) theory of autism echoes Hans Asperger from 60 years ago. Of course, Asperger's idea was just anecdotal. In my book, I show that we now we have a lot more detail and evidence with which to evaluate that idea. Asperger didn't have a definition of the key dimensions of empathising and systemising or any available psychometric assessment of these. So some things are new. We can (and have gone out

to) test whether the profile of people with autism comprises intact or superior systemising, alongside impaired empathising (S>>E).

4. Hadyn writes: ‘‘Baron-Cohen concludes that biology, not environment, makes us what we are’’. I was just a tad shocked to read this statement, as the thesis in the book is far more modest. What I actually say is that *both* biology and experience shape sex differences in the mind. I am amused that Hadyn sets me up as a straw man. Readers will discover (maybe with disappointment) that Hadyn has exaggerated my thesis.

5. Regarding the account of our experiment testing sex differences at birth, Hadyn simplifies the experiment into a contrast between a face and a ball. The ball was in fact a mobile, designed explicitly to have many of the same stimulus properties as a face (size, colour, luminosity, internal features) but it differed from a face in four important ways: (1) it was *inanimate*; (2) the facial features had been scrambled so that the stimulus was *unface-like*; (3) it moved via *mechanical* motion, rather than biological, self-propelled motion; and (4) it did not ‘‘*emote*’’. Finding that female neonates looked for longer at the face and male neonates looked for longer at the mobile could therefore indicate that the two sexes are (on average) attracted by any of these four factors in different ways.

6. But Hadyn is right that the newborn baby study is a key piece of evidence, since the experiment was carried out at 24 hours old; precious little time, if any, had elapsed during which socialisation could shape this observed sex difference. It will be important to try to replicate this finding. Two notes of caution to any scientist thinking of doing this: (a) you need large sample sizes to reveal such sex differences (we tested 101 babies at 24 hours old); (b) you need the right contrast of stimuli to override the face-preference effect that the majority of babies of both sexes will show. Hadyn, for example, asks why the early studies of faces versus checkerboards did not report such a sex difference. Well, this could be due to small sample sizes (it is rare in infancy research to have a sample of 100 babies); it could be because earlier investigators have not tested for a sex difference; or it could just be that checkerboards are not sufficiently interesting to grab the attention of a systemising brain away from faces (towards which we are evolved to orient). The systemizing brain is attracted by something that changes in predictable and lawful ways, to discover input-output relationships. Checkerboards are not really any test of this.

7. Hadyn concedes that the EMB theory might be relevant to the low empathy and strong systemising skills seen in Asperger syndrome (AS), but doubts if it will be relevant to the severe communication deficits seen in classic autism. Nor does he think the EMB theory is going to be able to explain the motor clumsiness seen in some people with AS. However, this is a misunderstanding of what the EMB theory is trying to explain. The theory does not

try to explain every symptom that *can* occur in people on the autistic spectrum (many of which will simply represent *associations*). It only seeks to explain the core or *universal* features that unite all individuals on the autistic spectrum. According to the E-S theory, these core features are impaired empathising (E) and intact or even superior systemising (S). So, to explain the other (non-universal) symptoms that can be observed in some but not all people on the autistic spectrum (such as epilepsy, learning difficulties, language delay, self-injury, gut problems, attention deficit and hyperactivity disorder, tics, clumsiness, etc.) one will need to resort to additional theories. The E-S theory strips away all of these associations, and aims to specify what lies at the core of autism.

8. Hadyn thinks the EMB theory might apply to AS, but not to the majority of people on the autistic spectrum, who he believes have a diagnosis of classic autism. I would question Hadyn's (perhaps outdated) view that the majority of people on the autistic spectrum have classic autism, and only a small minority have AS. The textbooks of the 1970s used to state that 75% of people with autism have the classic kind, which includes learning disability (or below average IQ). More recent population studies are actually suggesting that the truth may be the *opposite* of this: That the majority of people on the autistic spectrum may have AS (75%), with classic autism perhaps only being the minority (25%).

9. From my perspective, the precise proportions of AS to classic autism are not relevant to the theory. The EMB theory characterises all people on the autistic spectrum as having impaired empathising skills (relative to their mental age), alongside intact or even superior systemising skills. Clearly, Hadyn has trouble seeing how this might apply to classic autism, so let me paint a picture:

I have just spent the afternoon with a profoundly learning-disabled boy with autism. He is 9 years old and has about 6 words. He has to be fed, toileted, and dressed by a carer, and spends most of his day—left to his own devices—watching the same video over and over again. Despite his low IQ, he can operate the family video player and knows all of its functions, to the extent that he can override his mother's remote control of the video. This allows him to watch *Pingu* over and over again, even if his mother has tried to stop the machine. In my terminology, he has "systemised" the video recorder. He has also systemised the video itself, so that if his mother (as she does) tries to substitute his well-worn tape with a new copy, he throws a tantrum until he gets his old tape back. Despite all the hisses and crackles on the tape, he knows it—frame by frame—and can predict its behaviour 100%.

When he is not watching *Pingu*, he walks around the room, making little if any eye contact, tapping every surface with the back of his hand, picking up the tactile information of each distinct object surface around the kitchen and the living room. In my theory, this repetitive behaviour is another sign of him systemising—in this case, systemising the lawful properties of object surfaces. He can anticipate with

100% accuracy the feel of each object, and gets evident pleasure from being able to tap the window pane, then tap the window frame, then tap the wall, etc., knowing that they will each make a distinct sound, and each have a distinct texture, hardness, etc. Clearly, this is not systemising at the level of academic physics or computer science, but it is still systemising. And the strength of this drive is stronger than one might see in a child of his mental age without autism. Most importantly, against this profile of intact skills, his awareness of other's thoughts and feelings is significantly reduced.

Back to motor clumsiness. I have already said that the EMB theory is not aiming to explain this, but it is worth adding that Hadyn's notion that motor clumsiness is commonly associated with AS does not tally with my experience. I run a clinic for adults with AS, and over the last three years we have seen about 300 patients. Hadyn is right that a subgroup are dyspraxic or clumsy, but there is another very clear subgroup who are not. Establishing the relative size of these subgroups will be important for future research. Sadly, Hadyn misunderstands the EMB theory if he thinks that a problem for the theory is that David Beckham must have a "female" brain if he is not clumsy. Here is the key point again: The male brain is defined, psychometrically, as  $S > E$ , and the female brain as  $E > S$ . As simple as that. Just because David Beckham has an XY genotype, or just because he has good motor skills, this does not tell us anything about his brain type. He could have a male or a female brain, and that's the point: Without testing a given individual, one would have no way of predicting whether he/she has a male or female brain.

10. Hadyn expresses disappointment that the *extreme* female brain has not yet been identified clinically. Why not, he asks? Most reviewers of this book have commented that the prediction that the extreme female brain should exist is a new and interesting idea. They also found the long discussion in the book about why the extreme female brain may not lead to a clinical disability is likewise quite plausible. The key idea here is that an individual with the *postulated* extreme female brain type (this has not yet been studied) could be a super-empathiser but have *system-blindness*. The reason this might not lead the person into difficulties is that their super-empathy might allow them to have a good social network of support and to enjoy relationships; whilst their system-blindness might lead them to shy away from technical challenges, such as fixing their computer or their car. But faced with such problems, they can simply pick up the phone and get through to a help-desk. Sadly, such a help-desk is not obviously available for the extreme male brain. Who do you phone if you can fix computers but have mind-blindness? But Hadyn is right that the extreme female brain—if they exist—will be interesting to identify and study, and we are attempting to do this at the moment.

I know what Hadyn means when he says that he finds the writing style of *The Essential Difference* patronising at times. Writing popular science is a tough balancing act. Professors of cognitive science may well find the book occasionally patronising, but they are not the main intended audience. The *Guardian* readers who reviewed this book found it readable and, at times, even challenging when led through the neuroscience sections. For similar reasons, some of the questionnaire tests are included because they are accessible, while the statistics concerning reliability and validity are excluded. Ample references to the journal articles are given for the more academic reader who wants to know the size of the standard deviation on the Empathy Quotient result and other technical details.

I confess I am pleased that Hadyn enjoyed the book though he was left not quite convinced by the theory. For me, as a fellow scientist, I get the most fun out of a difference of viewpoints between scientists. As scientists, we are driven by the search for truth—how grandiose is that? So, for Hadyn to say in print that he ultimately thinks the EMB theory will be wrong is music to my ears. It's like sitting down in the pub with him and him betting me a drink that autism will turn out to need a different kind of explanation. I feel not just obliged but excited to take up this metaphorical bet. Having spent over 20 years studying this tiny corner of human nature, I feel this theory has a very good chance of explaining the core aspects of autism. And what gets me out of bed every morning is the fact that this new theory leads to a whole set of predictions, which are either right or wrong. Will females with autism show a male pattern of brain activity when we use functional magnetic resonance imaging (fMRI)? What about their mothers? Will they do so too, if they have the "broader phenotype"? Does fetal testosterone (an important determinant of sexual dimorphism in the brain) play a significant role in the cause of autism? Or some other biological but strongly male-related factor? These questions are very testable, and I'm looking forward to Hadyn buying me that drink when the results come out. And if I'm wrong, the drinks are on me.



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