The Philosophy of Autism

Edited by Jami L. Anderson & Simon Cushing

ROWMAN & LITTLEFIELD PUBLISHERS, INC.
Lanham • Boulder • New York • Toronto • Plymouth, UK

Published by Rowman & Littlefield Publishers, Inc.
A wholly owned subsidiary of The Rowman & Littlefield Publishing Group, Inc.
4501 Forbes Boulevard, Suite 200, Lanham, Maryland 20706
www.rowman.com

10 Thornbury Road, Plymouth PL6 7PP, United Kingdom

British Library Cataloguing in Publication Information Available

Library of Congress Cataloging-in-Publication Data

Anderson, Jami L.
The philosophy of autism / Jami L. Anderson & Simon Cushing.
p. cm.
Includes bibliographical references and index.
ISBN 978-1-4422-1707-2 (cloth : alk. paper) -- ISBN 978-1-4422-1709-6 (electronic)
1. Autism--Philosophy. I. Cushing, Simon, 1968- II. Title.
RC553.A88A53 2012
616.85'882--dc23

2012036099

The paper used in this publication meets the minimum requirements of American National Standard for Information Sciences Permanence of Paper for Printed Library Materials, ANSI/NISO Z39.48-1992.

Printed in the United States of America

Contents

Chapter Three

Autism and the Extreme Male Brain

Ruth Sample

Extraordinary claims require extraordinary evidence. —Carl Sagan

What is autism, and what is "The Extreme Male Brain"? Autism, Simon Baron-Cohen claims, is not only more common in males, but so are "autistic traits," and these traits are, like autism itself, biologically based and generated (at least in part) by fetal hormones. Autism is accordingly the far end of a spectrum of cognitive and affective difference, and this difference is the "essential difference" between men and women.[1] However, this claim is no ordinary causal claim about the relationship between biology and behavior. The hypothesis of the Extreme Male Brain (EMB) moves from a specific understanding of a particular clinical diagnosis to claims of a deeper knowledge of men and women in general. There are interesting and deep philosophical questions here about whether such knowledge is even possible. Here I wish to discuss whether EMB is defensible, as well as the implications of hypothesizing essential differences in mental traits along the lines of gender.

My aim is to investigate the relationship between recent claims made in psychology and neuroscience about autism spectrum disorders (ASD) and the broader claim that there is an "essential difference" between male and female minds.[2] Should we look at autism as an extreme version of the male brain? What, if any, arguments have been produced for this equivalence? More importantly, what would we gain by seeing autism as a gendered disorder?

Over the last decade, many other authors have tried to revive the idea that there are not only basic overall anatomical differences between men and women, but that our brains in particular are different too.[3] These brain differences, they argue, translate into behavioral, cognitive, and affective differences in ways that are consistent with received ideas about boys and girls,

men and women. Furthermore, they claim that because of the differences in our brains, we can explain the differences in the way that men and women are positioned in society: in our schools, our employment patterns, in our hobbies and interests, and in our personal relationships. Baron-Cohen predicates his EMB theory on the idea of a Male Brain. He argues that we should see autism and certain male-typical traits as part of a broader phenotype generated by sex differences in the brain.[4] Some researchers have called this equation of autistic traits and maleness "intriguing."[5] Steven Pinker has enthusiastically endorsed it and, in an unfortunate cover blurb for the paperback edition, wrote that "*The Essential Difference* is essential reading." Others have accepted EMB and expanded his model to include an Extreme Female Brain as a pathological condition.[6] However, the argument equating EMB and autism has not yet been carefully scrutinized.

I shall try to show that although it seems *prima facie* plausible that EMB is true, it is not. The most important basis of this equivalence is an argument that I call the Common Cause Argument. It is an argument that Baron-Cohen never explicitly makes, but I aim to show that is implicit in what he does argue. I think it is the strongest, most plausible argument in favor of the equivalence between autism and EMB. It is structurally valid. It does not, however, succeed. One of the premises *is* true, but that premise is not relevant to establishing the thesis. However, none of the remaining premises that are relevant to the conclusion has been clearly established. Without a sound argument equating autism with the male brain, we should avoid doing so. Even though the prevalence of autism is significantly sexually dimorphic, it would be a mistake to see it as a stronger "dose" of The Male Brain, just as it would be a mistake to see disorders found more often in girls (such as Rett syndrome) as the Extreme Female Brain.

The primary reason for rejecting EMB is that it is not a well-supported hypothesis. Should it turn out the specific claims invoked in favor of EMB are confirmed, the confirmation of those claims would not confirm EMB. "Sexing the brain" adds nothing to our understanding of autism. It adds nothing to our understanding of what causes autism. It adds nothing to our understanding of how to remediate autism. EMB has the potential to divert research funding from other research programs that could enhance our understanding of autism and provide insight into remediation, if not a cure, for autistic symptoms.

In addition, promoting this equivalence has serious social implications that we should not ignore. Baron-Cohen has stated explicitly that science should be distinct from social policy,[7] and has said that some of his critics (such as neuroscientist Cordelia Fine) are merely advancing a feminist political agenda.[8] Instead, even if unintentional, the equation of autism with the male brain advances another agenda: what Erik Turkheimer calls "belligerent defenses of stereotypical masculinity in evolutionary psychology."[9] Baron-Cohen uses EMB to argue that the low representation of women in the natural sciences, mathematics, computer science, and engineering is a product of biological differences in the brains of men and women. This has clear political implications, despite Baron-Cohen's professed neutrality. More recently, he has argued that the male brain is "truth-seeking" in a way that the female brain is not.[10] While his goal may be to valorize certain forms of autism and increase tolerance for poor empathizing, the sexist implications are clear: we should not expect women to succeed at the same rate as do men in the higher-paying, higher-prestige disciplines of math, science, and engineering.

WHAT IS AUTISM?

Important scientific research, including that of Baron-Cohen, in the last two decades has helped us to understand autism—and in particular, to understand the nature of autism as a biologically based neurodevelopmental condition. We now know more about what autism *is not* than ever before. We know that it is not psychogenically produced by "refrigerator mothers," to use twentieth century psychologist Bruno Bettelheim's cringe-making phrase. We know that vaccines do not cause autism. We know that autism has a significant genetic component, with hundreds of genes and epigenetic factors involved. We know that monozygotic twins have a higher rate of concordance in the diagnosis than the concordance of dizygotic twins, but the difference between MZ and DZ twin rates of concordance is much lower than previously thought. A 2011 Stanford University study shows that MZ twins have a concordance rate of .77 and .31 for ASD.[11] ASD is thus moderately heritable, whereas in the past autism was regarded as one of the most highly heritable of psychiatric conditions. Researchers suspect numerous spontaneous, noninherited mutations of having a cumulative causal effect.[12] In rare cases, maternal exposure to certain drugs during crucial periods of fetal development contributes to autism, although toxins account for a tiny minority of cases.[13] We also know more about how to remediate some of the symptoms of autism with behavioral interventions, particularly with various forms of applied behavioral analysis (ABA).[14]

There are several major contenders for conceptualizing autism.

1. "Weak Executive Functioning" theory implicates impairments in the ability to regulate cognitive and affective function.[15] Executive functioning allows us to regulate our emotions, respond to new information and adjust our behavior accordingly, and shift attention when necessary. Broad deficits in executive functioning could explain why

so many people with a diagnosis of ASD do poorly on the Sally-Anne test even when they do not have subnormal IQ.

2. "Weak Central Coherence" theory emphasizes the tendency among those with ASD to focus on particular parts of a situation or visual field rather than grasping the entity as a whole. Thus children with ASD often focus on parts of a face but have impairments in the ability to recognize the face as a whole, and they outperform typical children on certain detail-oriented tasks and the Embedded Figures test.[16]
3. "Mindblindness" emphasizes the deficits in people with autism in the area of recognizing and responding to the emotions of others.[17] Related to this is hypothesis of a hypo-functioning amygdala as the root cause of abnormal social interactions in those with ASD.[18]
4. Most recently, "Intense World Theory" hypothesizes that atypical development of the brainstem leads to multiple deficits in the ability to process sensory information in a modulated way. In essence, this theory attempts to explain *virtually all* the symptoms of autism (including many of those not listed in the last three versions of the *DSM* but widely recognized by researchers and clinicians) as sequellae of the autistic brain's extreme overreaction to stimuli. In contrast to the Mindblindness/Hypo-functioning Amygdala theory, this account postulates a *hyper*-functioning amygdala.[19] It also attempts to account, relying as it does on a theory of atypical brain stem development, for the difficulties with fine and gross motor skill observed in people with ASD, as well as atypical sensitivities to sound, light, taste, touch, and pain.

Still, we cannot yet claim to know what autism is, let alone claim to understand what causes it. The current science of autism is still very much dynamic. Despite the present consensus that autism is not psychogenic but is biologically based and is a disorder of the brain, ASD is always diagnosed symptomatically, usually using the criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) of the American Psychiatric Association. These criteria have evolved over time, and may continue to evolve. Like almost all of the other disorders of the *DSM*, there is no physiological test for autism. No blood test, genetic test, or brain-imaging can diagnose autism, although genetic tests often differentiate between diseases with autistic symptoms (such as Fragile X) and autism. Measurable biological phenomena (such as seizures) and behaviors such as hand-flapping and self-stimulating behavior ("stimming") are associated with autism, but none of these associated phenomena are officially diagnostic. The prevalence of autism appears to be rising, and although some of the rise in prevalence can be explained by factors such as better identification, diagnostic substitution and parental age,

a surprisingly large amount of it (by one estimate, perhaps 40 percent of the increase) remains unaccounted for.[20]

Although researchers and clinicians regard autism as a biologically based disorder, autism is not identified with its underlying biology. In particular, disorders that used to be considered forms of autism (such as the degenerative disorder Rett syndrome) have been reclassified as separate disorders principally because their specific biological basis and etiology has been identified. In the case of Rett syndrome, a specific *de novo* mutation of the MECP2 gene on the X chromosome causes virtually all cases of the disease. Consequently, although it appeared in the *DSM-III* as a type of autism, the fourth incarnation of the *DSM* (*DSM-IV*) removes it and Rett syndrome will not be associated with autism in the *DSM-V*.[21] Other disorders with known chromosomal or genetic causes such as Down syndrome, Fragile X, and tuberous sclerosis may involve some "autistic features" (especially in the case of boys with Fragile X), but are not classified under the general term "autism." In other words, it appears that when autism has a clearly identified genetically based culprit, clinicians and researchers no longer classify it as a form of autism.

Given all of this, it is difficult to make the case that we know what autism is. And yet Baron-Cohen argues that we do know what it is, and that it is a version of the Male Brain. It seems a dangerous error to use the emerging scientific research about autism, when autism is poorly understood, to understand sex differences in cognition and behavior more generally. Conversely, it seems dangerous to view autism through the lens of sex differences. To do so risks distorting both our understanding of autism and our understanding of sex differences.

THE SEXUALLY DIMORPHIC MIND

Philosophers have long attributed sexual dimorphisms in cognitive, emotional, and moral traits to humans, and from the ancient philosophers all the way to the early modern period of philosophy, those dimorphisms were derogatory toward women. Aristotle claimed that all females are a natural deformity, ultimately due to their lack of sufficient heat, and their reasoning faculty is defective in that it "lacks authority." Kant was famous for denying that women had a sense of justice, and worried that they were congenitally incapable of impartiality. Hegel thought that women were incapable of philosophical thinking.[22] These early theories of sexual dimorphism of the mind did not necessarily point to the *brain* as the source of the difference.

A few male philosophers such as John Locke[23] and John Stuart Mill[24] downplayed natural differences between the sexes, as did women such as Mary Wollstonecraft and Harriet Taylor Mill, but philosophers and "natural

philosophers" (the precursors of experimental scientists) continued to defend the existence of sexually dimorphic mental traits well into the twentieth century.

The modern project makes use of research on hormones, brain structure, and various forms of brain imaging—especially functional magnetic resonance imaging (fMRI) in ways not previously possible. Most recently, the human genome itself has been targeted as the source of sex difference, although the idea of "two separate genomes" has been subject to significant criticism.[25] Additionally, some (but not all) contemporary versions of the theory insist that men's and women's brains and minds are fundamentally different, but one is not better than the other. Baron-Cohen argues that neither the male brain nor the female brain is superior to the other, but that these brain differences make us more or less suited for certain occupations and activities, some of which are more socially, culturally, and economically valued than others:

> People with the female brain make the most wonderful counsellors, primary school teachers, nurses, carers, therapists, social workers, mediators, group facilitators or personnel staff. . .People with the male brain make the most wonderful scientists, engineers, mechanics, technicians, musicians, architects, electricians, plumbers, taxonomists, catalogists, bankers, toolmakers, programmers or even lawyers.[26]

There are three parts to the modern version of this project, of which Baron-Cohen's EMB thesis is one example. The first part is to show that there are real and significant sex differences in the cognitive, behavioral, and affective traits of men and women. One scientist or another has argued that the following traits are sexually dimorphic: mathematical and verbal proficiency, performance on tasks of spatial rotation, orienteering, aggression, psychopathy, mind reading and empathizing, interest in competitive sports and competition generally, rough-and-tumble play, interest in children and child care, interest in people, sex drive, sensory-seeking behavior, left-handedness, and interest in color.

The second part is to show that differences in some combination of brain structure, brain functioning, brain development, and brain chemistry can explain the alleged dimorphism, *and* that these brain differences are not themselves due to culture. This is an important qualification. Those who reject the Extreme Male Brain hypothesis can accept that there are sex differences in behavior, affect, and interests that are based in the brain, but may argue that those brain differences are themselves a function of culture. While environmental factors can influence the direction of the structure and chemistry, biology, the argument goes, is the ultimate source of the difference and can explain it.

The third part of the project is to show that there is evolutionary pressure in favor of this dimorphism that explains its existence. Evolutionary psychologists, like the sociobiologists of the 1970s and 1980s, focus on the role the natural selection might play in the development of such dimorphisms. Evolutionary arguments are usually of the form "it makes sense that..." and come after a genetically based difference between the sexes is postulated. A natural selection argument shows how a genetic difference arose. Evolutionary psychologists argue that genetic differences between the sexes have been adaptive for humans by creating a division of labor between the sexes related to their reproductive roles. This third evolutionary part is not essential to the main project of explaining the alleged sexual dimorphism in mental traits, because not every biological difference, whether it is an average difference between, say, adult height among different populations of people, or whether it is an average difference in body mass between men and women, will have served an adaptive function. Some differences are random and have no identifiable function. It could be the case that such a dimorphism, were it to be demonstrated, plays no causal role whatsoever in reproductive success, but is just an accident of nature. Nevertheless, the evolutionary component is important, because scientists often use it as supporting evidence for the first two. Baron-Cohen himself speculates that evolution has played a role in selecting for the dimorphism he ascribes to humans. For example, the weaker empathizing that Baron-Cohen says is characteristic of men might be useful because it "makes it easier for you to hit or hurt someone, or in less extreme ways, simply to push them aside in competition, or abandon them when they are no longer useful to you."[27] On the other hand, stronger empathizing might be beneficial to women because "a high-empathizing female, engaged in childcare, is better equipped to create a community of friends who could watch over her children when she is unable to keep an eye on them all of the time."[28]

The thesis of the EMB fits squarely into a larger group of theories in the history of science. An interesting asymmetry in his theory is the focus on the Male Brain and its "Extreme" version, and not the Female Brain and its "Extreme" version. Why does Baron-Cohen focus on the Extreme Male Brain, and not the Extreme Female Brain? This asymmetry occurs because Baron-Cohen claims that ASD should be understood as an extreme amplification of typically male mental traits, with accompanying deficits in typically female traits. However, there is no developmental disorder identified with the amplification of any "typically female" traits—e.g., being very good at identifying and responding to emotions. Bernard Crespi and Christopher Badcock have defended the idea of schizophrenia as "The Extreme Female Brain" in a recent lengthy paper with commentary in *Brain and Behavioral Sciences*.[29] Badcock has turned the idea into a full-length monograph.[30] In *The Essential Difference*, Baron-Cohen seems to dismiss the idea of an extreme female

brain.[31] However, more recently he seems to have changed his mind and has "postulated" an Extreme Female Brain; as of this writing he has not yet discussed it or defended it at any length.[32]

THE MALE BRAIN AND THE EXTREME MALE BRAIN

Which do we understand first? The Extreme Male Brain or the Male Brain? In places, Baron-Cohen seems to understand typical (i.e., nonautistic) human populations through the lens of autism. Thus it is useful to look at his conceptualization of autism first. He understands ASD as first and foremost, a failure of mind reading: a kind of "mindblindness" in which people have deficits in the ability to accurately ascribe and interpret the beliefs, emotions, and actions of others. These deficits lead to diminished ability to interact with others in normal (i.e., socially expected) ways. Initially, the focus was on the inability of children with autism to pass the famous Sally-Anne test, which he and many others regard as a test of the subjects' ability to accurately identify beliefs in others.[33] Later, the affective response to the mental states of others became the focus: people with autism do not merely have difficulties in understanding other minds, but they also do not have typical responses to the emotions they do identify. They are often less distressed or activated by others who are in distress, even when they *understand that* the other person is in distress.[34] He calls this combination of mindblindness and atypical responsiveness "hypo-empathizing."[35] Empathizing is "the drive to identify another person's emotions and thoughts and to respond to these with an appropriate emotion."[36] This drive, he argues, is underdeveloped in people with ASD.

But autism involves other features as a well as impairments of social interaction. The three main or "core" features presently identified with autistic disorders, according to the *DSM-IV*, are (1) impairments in social interaction, (2) impairments in communication, and (3) restricted interests and stereotyped or repetitive behavior. (The WHO has a slightly different set of criteria, but the *DSM-IV* is more widely used in North America.) However, the diagnostic criteria in the *DSM-IV* are highly disjunctive.[37] There is no simple set of necessary and sufficient conditions for receiving an autism diagnosis. The result is that many people with an autism diagnosis look very different from one another.

"Mindblindness" is Baron-Cohen's way of conceptualizing the impaired ability of people with ASD to understand and respond to what other people are doing and thinking: ASD people have an impaired or absent (or, some might argue, simply *delayed*) "theory of mind." Mindblindness with atypical (dampened) responsiveness to others' mental states conceptualizes the first diagnostic criterion: impaired social interaction. But what about the other

two criteria? The second criterion, having to do with communication, is quite variable and not always pronounced in people with ASD. People with Asperger's do not have a delay in the acquisition of functional language, although sometimes they demonstrate atypical inflection and prosody. Yet those with Asperger's are part of the group of those with ASD, and Baron-Cohen includes them in his theorizing about the Extreme Male Brain. Indeed, they seem to be his *paradigmatic* cases of The Extreme Male Brain. Other people with ASD may have no functional language, and still others can communicate only through a keyboard or via pointing. However, the third criterion includes the restricted interests, and in many cases, the restricted interests of those with ASD involve so-called "static" (as opposed to dynamic) systems: systems of letters or numbers, train tables, actual trains. People—human beings—are Baron-Cohen's paradigmatic example of dynamic systems, because people are constantly changing in response to their inputs, and people cannot be described as functions with unique outputs for a given input. Electrical switches and mathematical formulas are his examples of static, lawful systems.[38] Baron-Cohen says that "systemizing is a new concept," and he is indeed using the term in an unusual way. A system in his sense is "something that takes inputs and deliver outputs [sic]."[39] In particular, systems with "predictive value" are particularly attractive to systemizers: systemizers prefer lawful systems with less variability in the possible outputs given inputs. The minds of people are not, he says, systems in this sense. People do not behave in a law-like, predictable manner; consequently, "[t]his is why systemizing the social world is of little predictive value."[40]

This last diagnostic criterion is the basis of Baron-Cohen's claim that ASD involves hypersystemizing, whereas the first criterion is the basis of his claim that ASD involves deficits in empathizing: the hypoempathizer also tends, he argues, to be a hypersystemizer, focusing on predictable, lawful systems. So Baron-Cohen's hypothesis of the Extreme Male Brain is a modification of his earlier hypothesis that ASD is essentially mindblindness: ASD is mindblindness *plus* inappropriate responsiveness *plus* "system-awareness:" hypoempathizing with hypersystemizing.

Baron-Cohen has argued that we should think of autism spectrum disorders as an extreme form of the kind of brain that men tend to have: a brain that "systemizes" well—or at least a lot—(hence the S-type) but does not "empathize" well—or at least a lot (hence the E-type). People with S-type "male" brains are not attracted to the world of people, but they are interested in machines, mathematics, and scientific, law-like systems. People with E-type or "female" brains are interested in people; they are prone to think about the minds of other people, and they tend to respond more appropriately to them. They find it rewarding to engage with other people. In general, he argues, women tend toward more E and less S, and men tend towards more S and less E, although most people have a more or less "balanced" brain,

somewhere in the middle of the bell curve. Furthermore, Baron-Cohen postulates that those who hypersystemize tend to also be hypoempathizers: in other words, S and E are inversely correlated.[41] The combination of these two features in people with autism is not, he argues, a coincidence, but they are causally linked. Those who have this combination in an extreme enough form may be diagnosed with ASD.

However, Baron-Cohen also argues that his account of the nature of ASD does not just explain atypical or disordered brains, but also explains the distribution of E and S brains among the general population. And this in turn explains why we see gender divisions in social structures: on average, men are better at certain things than are women, and on average, women are better at certain things than are men. Baron-Cohen is emphatic that not all women are pure E and not all men are pure S, and that one's sex cannot serve as a certain predictor of one's brain type: some men have "the female brain" and some women have "the male brain." Rather, he argues that the *average differences* in the brains of men and women mean that women tend to be better empathizers and men tend to be better systemizers, although there is a normal distribution for both men and women of both of these traits. So some men are poor systemizers and strong empathizers, and some women are strong systemizers and poor empathizers. This, he argues, can explain the gendered division of labor in our culture, including the gendered division within family structures and the workplace. It can even explain our choice of toys and hobbies. He points out that he knows some women who are very good with computers and that he knows some men who are very nurturing and caring, and that we should not discriminate against women who want to engage in S-type activities or against men who want to engage in E-type activities. Being male does not automatically guarantee strong systemizing, and being female does not guarantee strong empathizing.[42] Nevertheless, there is a strong tendency of men to be good at what he calls systemizing and poorer than women at empathizing, and a strong tendency of women to empathize well but not systemize so well, and these differences in patterns of thought, emotion, and interest can be explained by biology. Furthermore, he argues that because of this, we should not be surprised when women are not represented proportionately to their numbers in systemizing professions, such as mathematics, computing, engineering, accounting, and the sciences. In fact, he argues that "we should not expect the sex ratio in occupations such as math or physics to ever be 50-50 if we leave the workplace to simply reflect the numbers of applicants who are drawn to such fields."[43]

Baron-Cohen appears to use the case of autism as *evidence* for the Male Brain/Female Brain dichotomy, because autism, in all of its various forms, is much more common among males. There is broad consensus that the ratio of boys to girls with autism is 4:1, and the ratio of boys to girls with Asperger's syndrome is 10:1. People with Asperger's, by definition, have normal IQs

and do not have significant delays in language acquisition. This alone has led some to conclude that male brains are different from female brains because of their proneness to certain pathologies; but Baron-Cohen wants to argue that "non-pathological" (i.e., typical) humans without an ASD diagnosis show the same pattern of gendered traits. Hence the Extreme Male Brain theory appears to be the evidence for the Male Brain Theory.

AN ESSENTIAL DIFFERENCE?

The title of Baron-Cohen's popular book on this topic is *The Essential Difference.* However, what does he mean when he says that this difference between men and women is essential? Baron-Cohen does not explain what he means by "essential" in his book, despite the title. He does not use the word very much beyond the first page. So what could he mean? Baron-Cohen's book is about men and women as kinds of people and the characteristics of those kinds of people. Philosophers often talk about essences with respect to kinds. In this sense, an essence is the set of necessary and sufficient conditions that makes something an example of a kind of thing. For example, one might argue à la Kripke that the essence of gold is its atomic number 79, and the essence of water is its chemical structure H_2O.[44] Being malleable and yellow are not part of the essence of gold; something could have these features and not be gold. Something that is wet, clear, and potable is not necessarily water; it requires the structure H_2O. However, Baron-Cohen never argues that there is an essence, in the sense of necessary and sufficient conditions, of the male brain and he never argues that there is an essence of the female brain. He instead argues that "female brains are predominantly hard-wired for empathy. The male brain is predominantly hard-wired for understanding and building systems."[45] Elsewhere he amplifies this claim: he is talking about average differences between men and women. These are not essential differences.

What about individual essences? "Uniessentialist" views make claims about the unity and identity of individuals, rather than kinds.[46] Maybe having an S-type brain makes a person who he is or who she is, so that person would not be the same person if he or she did not have an S-type brain. Perhaps a person who had an S-type brain would cease to be the same individual if, through some chemical or structural change in his or her brain, that individual no longer had S as a characteristic. Similarly for the E-type brain. We can imagine how a person might argue that if she did not have autism, she would not be the same individual; she would be somebody else, even though she would be the same person if she were an inch taller or if she hated the taste of maple syrup. However, Baron-Cohen never discusses essentialism. He never

makes any claims about whether I would still be me if I stopped being a systemizer or an empathizer, and he never discusses individual essentialism.

Instead, Baron-Cohen seems to be claiming that the differences between male and female brains are basic and fundamental with great significance. But in what way? He is making at least three claims. First, he argues that the differences between male and female brains are biologically based in nature. This might seem obvious—how could brains differ in some way *other* than biologically?—but it is not. For one thing, *environments* can cause biological differences in organisms. Some of these biological differences are then permanent. Environmental exposure to certain chemicals that mimic estrogens, for example, can cause physiological changes in animals—including a change in the animal's sex. Water temperature determines the sex of some amphibians under certain conditions, although the primary mechanism for sex determination is genetic.[47] Recently, it was discovered that female sharks, which normally reproduce sexually, can begin to reproduce parthenogenically when kept in tanks isolated from male sharks.[48] Such a shark is biologically parthenogenic, but its sexual features are not based in the nature or essence of the animal in the sense that Baron-Cohen seems to be using. There is no "essential difference" between the parthenogenic female shark and the non-parthenogenic female one; they are morphologically similar at the start, but dissimilar environment triggers the change in biology.

One might argue that men and women have different brains (in Baron-Cohen's sense) because of the environments they are exposed to. Smiling at girls more, for example, might change their brains to make them more E, and in turn cause behavioral differences. But Baron-Cohen appears to be arguing that in typical developmental environments, *including fetal environments*, male and female brains develop differently. They do so because of the different hormones that are found in the uterine environment. In particular, he is claiming that natural variations in fetal testosterone produce average differences in mental traits between the sexes; and, since on average males are exposed to more fetal testosterone than are females, males have more of the mental traits associated with fetal testosterone. In short, he is arguing that male brains and female brains are *organized* differently because of their naturally different development. This organization is not temporary, but permanent, and has lasting consequences for behavior. Most crucially, occurring *in utero*, it is not subject to the influences of culture. While Baron-Cohen claims that these differences are natural and biological, he does not claim that they are immutable, the way having an X and a Y chromosome throughout one's cells is immutable. It might be possible to eliminate or reduce the differences between female and male brains and the associate behavioral differences, either through behavioral interventions or through biomedical treatments.

Second, he also argues that these differences, while average, have significant and measurable behavioral implications. Higher levels of fetal testosterone, more typically found in the amniotic fluid surrounding male fetuses, predict a more S-type brain, and lower levels surrounding female fetuses predict a more E-type brain. He makes the extremely controversial claim that we can observe differences between female and male babies in the very first hours of life: "from birth, females look longer at faces, particularly at people's eyes, whereas males are more likely to look at inanimate objects."[49] The behavioral differences, he argues, are significant and measurable: both statistically significant (in the sense that the observed differences are not produced by chance) and large. They are not miniscule, insignificant differences between male and female brains.

Third, he argues that not only is the sex difference significant, but also that it is important. You might think that this is a distinction without a difference. But when something is significant, it is more than just a little bit; it is not trivial; it is substantial. So, for example, men tend to be significantly hairier than women, on average. However, one might argue that while there is a significant sex difference in average hirsutism, it is not an important difference; it doesn't matter much in the sense that it does not have important consequences. Baron-Cohen is claiming that the difference here is important in the sense that it has substantial consequences for the functioning of males and females. The preferences, hobbies, habits, and social roles of women are as different as they are because of these significant differences in biology. (It can, he argues, explain the small number of women in certain fields of science.) Moreover, the average brain differences can explain pathologies such as ASD, which are sexually dimorphic in their prevalence. When so many more males have ASD, and ASD is a significant impairment, this is an undeniably important difference.

Thus Baron-Cohen argues that certain differences between male and female minds are biologically based, measurably significant, and highly relevant to our functioning. This is what he appears to mean by "the essential difference." Understood this way, however, one might object that this still does not seem to be "the essential difference" in the ordinary sense of a single trait that makes males the kinds of beings that they are and females the kinds of beings that they are—a Kripkean essence. Calling something "the essential difference" seems much stronger than the interpretation I have offered here. However, because Baron-Cohen never explicitly states that E and S are the single most important traits of men and women, or that E and S are *definitive* or *constitutive* of men of women, the use of the term 'essence' is misleading. The E/S theory is not really a claim about essences. The E/S theory is actually a claim about average differences between the sexes that are said to be biologically based, significant, and have important consequences.

FETAL T AND THE COMMON CAUSE ARGUMENT

How is the "essential difference" or E/S theory relevant to autism? What is the relationship between the claimed average differences in male and female brains, the claimed average differences in the behavior and functioning of typical males and females, and the differential prevalence of ASD among males and females? What causal claim is he making? At times he seems to argue that average behavioral differences between the sexes can "help us to understand" autism.[50] This sounds as if basic sex differences in the brain explain why autistic people are the way they are, and why there are more males with autism than females. At other times, he seems to argue that facts about ASD help to explain why typically functioning men and women are as different as they are. Men are from Mars and women are from Venus, we might say, because men are a little bit autistic. Misunderstandings occur when we don't recognize that there are average differences between what men are good at and find interesting (systems) and what women are good at and find interesting (people). Men do not interpret and respond to emotions the same way that women do, and they enjoy machinery more. And we should therefore not be surprised when the systemizing found in autism is expressed in the general male population as greater mathematical ability: hence the disproportionate number of men in the STEM (Science, Technology, Engineering, and Mathematics) disciplines, particularly mathematics, physics, and engineering.[51] Nor should we be surprised that women, who are less likely to be systemizers and more likely to read and respond to people's emotions appropriately, are found in the caring and helping professions, such as teaching and nursing.

Baron-Cohen's main argument appears to be as follows. Sex differences in mental traits and the symptoms of ASD are both the result of a *common cause*: differences in the brain that begin *in utero*, triggered by fetal testosterone. Brain differences can explain observable behavioral and psychological differences between typically functioning men and women. At the same time, he argues, they also explain the particular symptoms of ASD, the higher prevalence of ASD among boys and men, and the correspondingly lower rate of ASD in girls and women.[52] More men are autistic and more men are mathematicians and engineers for the same reason: fetal testosterone.

Schematically, the argument can be represented as follows:

1. Fetal testosterone is causally relevant to the symptoms of ASD.
2. Fetal testosterone is causally relevant to certain mental traits: higher systemizing and lower empathizing.
3. Both ASD and the mental traits of higher systemizing and lower empathizing are more common in males: they are "male-prevalent."
4. ASD is simply a more intense version of the male-typical mental traits produced by fetal testosterone: they are part of the same broader phenotype.
5. If one set of symptoms is simply a more intense version of another set of symptoms, and they are produced by the same cause, then they are the same phenomenon.
6. ASD = EMB: Therefore, ASD should be understood as the Extreme Male Brain.

This argument asserts that two phenomena *A* and *B* have a common cause; that both *A* and *B* are male prevalent; and that *A* and *B* are different intensities of that same phenomenon. It concludes that because of this, *A* and *B* should be understood as essentially the same thing. The assertion of a common cause, male prevalence, and the interpretation of mental traits as autistic traits are jointly used to make the case that ASD and the male brain are essentially the same thing. As I shall argue below, this argument has a valid structure, although male prevalence should not be used as evidence for EMB. Rather, if EMB is true, it would be an explanation of male prevalence—male prevalence does not itself support EMB.

This raises the question of *causal mechanism*: how does fetal testosterone produce average sex differences and which ones? How could it contribute to the developmental disorders of autism? Most scientists accept that sex hormones, including testosterone, estrogen, and estradiol, have two different kinds of impact on mammalian tissue: organizational and activational. Hormones organize tissue when they create different morphologies (e.g., when they play a role in the development of testes in typically developing boys). They play an activational role when they trigger certain events, such as puberty.[53] Baron-Cohen argues that fetal testosterone is particularly important because it *organizes* the brain early on *in utero*, in addition to activating body and brain changes later in life. Estrogens also play a role in both organization and activation, but as Baron-Cohen notes, this class of sex hormones makes the activational/organizational distinction problematic, because the organizational effects of estrogen continue for a very long time.[54] More importantly, higher levels of testosterone in the developing fetus are, he argues, correlated with more "male typical patterns of behavior."[55] Some of this behavior, he claims, is the *same behavior* seen in autistic people where it is present to a greater degree. Since ASD is a biologically based developmental disorder that by definition develops before the age of three, Baron-Cohen speculates that the same mechanism—fetal testosterone—that produces sex differences in behavior also produces autistic traits.

Why are sexually dimorphic behavioral features and autism not possibly a deficiency or preponderance of estrogen or estradiol? After all, both males and females manufacture androgens such as testosterone as well as the other

sex hormones. Testosterone is not the only hormone that organizes or activates. Briefly, Baron-Cohen finds no evidence that average differences in fetal estrogen correlate with either sexually dimorphic mental traits or ASD, or any other developmental disorders. Boys typically experience a surge in testosterone *in utero* that rises briefly at birth and then dissipates until puberty. Girls do experience a surge in estradiol shortly after birth, and it remains relatively high. Baron-Cohen says "there is little evidence for an effect of estrogen on rough-and-tumble play or reproductive behaviors," while there is more evidence that testosterone affects the expression of these behaviors. Even in a case where the placenta produces a very high level of estrogen, this estrogen is bound to alphafetoprotein in fetal blood, making it impossible for the estrogen to enter the fetal brain;[56] AFP disappears at birth. In addition, in the case of males born with no androgen receptors (Complete Androgen Insensitivity syndrome, or CAIS), their appearance is phenotypically female; their physical appearance, especially in the case of a complete lack of receptors, is female. So it appears that testosterone alone can play a role in masculinizing both phenotypic presentation as well as behavior.[57] If there is a probable common cause among the sex hormones for autism and sexually dimorphic behavior, testosterone appears to be the most likely suspect.[58]

Baron-Cohen does not claim to know the actual mechanism through which fetal testosterone produces dimorphic effects on the brain. So far, he has relied on correlational studies involving amniotic fluid, cord blood at birth, and salivary or serum levels of testosterone in adults.[59] His conceptualization of autism as "extreme male brain" was first published in 1997, when not many correlational studies were available. By 2004, he was able to use eleven correlational studies to discuss the effects of testosterone on behavior, and the results of those studies were quite mixed, as I shall discuss below.

There are some serious problems with this hypothesis. Baron-Cohen cites no studies or data correlating fetal testosterone and *autism*. Further research in this area is ongoing. Neuroscientist Lisa Eliot suggests the opposite conclusion, arguing that "if autism is caused by extreme testosterone exposure, then you would expect to find that boys with the highest prenatal testosterone levels are the ones who end up being diagnosed with the disorder, while boys with lower prenatal testosterone exposure would be diagnosed much less often. This is not the case."[60] Another problem involves the case of those who lack androgen receptors. One would expect to find that those XY persons with CAIS, who, due to a genetic mutation on the X chromosome lack receptors for testosterone entirely, would never have ASD. However, *no one has demonstrated or even reported this*. This is perhaps in part because CAIS is relatively rare, making the prevalence of autism in that population more difficult to study reliably.[61] In other words, there is no research showing that higher fetal testosterone levels *in either sex* increases the probability of the subject receiving a diagnosis of autism. The causal claim that fetal testosterone is causally relevant to autism or ASD is speculative in the absence of this research.[62]

Moreover, even in "idiopathic autism" (i.e., autistic features not caused by a known inherited genetic disorder such as Fragile X or tuberous sclerosis, or some of the genes on chromosomes 15, 22, and 7) many genes have been implicated.[63] In particular, *de novo* mutations seem to contribute to a significant percentage of the cases of autism—upwards of 10 percent.[64] These mutations (like the mutation responsible for Rett syndrome) are not inherited but are spontaneous germ-line anomalies. Since specific genetic anomalies, inherited or not, raise the probability of receiving a diagnosis of autism, it appears that fetal testosterone could at best be a *partial cause* of ASD. Baron-Cohen himself has suggested that the higher levels of fT (fetal testosterone) that produce autism have an inherited genetic basis.[65] Baron-Cohen does not dismiss the heritable genetic component, although he acknowledges it mostly by pointing out that parents and grandparents of those with ASD are more likely to have "autistic traits," rather than pointing to the specific genetic mutations that have been implicated to date.[66] Given the research implicating such a wide range of *de novo* mutations, is it possible that fT is implicated in all of most of ASD produced by these? Whether a genetic factor is inherited or not may ultimately become diagnostically significant, if not dispositive. In fact, it appears that when autism is associated with an inherited genetic disorder (such as Fragile X) and or a *de novo* mutation (such as Rett syndrome), individuals with that disorder are "undiagnosed" with autism (or, more accurately, reclassified according to "diagnostic substitution" and not regarded as appropriate research subjects for studies of autism proper. By such diagnostic substitution, individuals are "sub-typed" out of the class of people with ASD. Indeed, if this practice continues, the class of persons with ASD may be typed more finely until either the disorder no longer exists, or exists exclusively of a subgroup of individuals with *de novo* mutations and some other triggering factor.).

Nevertheless, it is possible that we may find evidence to support the claim that higher levels of fT will be at least one factor in some cases of autism. If so, will that vindicate the Common Cause Argument? As I aim to show, even a positive correlation between fetal testosterone and ASD diagnoses does not justify interpreting autism as the Extreme Male Brain.

IS THE COMMON CAUSE ARGUMENT A GOOD ARGUMENT?

Let's look at the argument in detail.

Premise 1: Fetal Testosterone and Autism

As I mentioned above, the first premise of this argument lacks support. There is no evidence *as of yet* that higher levels of fetal testosterone (fT) play a causal role in the development of autism. Baron-Cohen points out that there is some evidence that 2D:4D digit ratio (the ratio of the length of the second finger to the length of the fourth finger) is on average lower in males and is lower in people with autism than it is in (normal) females. Citing his own study on prenatal hormones and 2D:4D, he (controversially) postulates that the ratio of fT to fetal estrogen is negatively associated with this ratio. Yet this is not direct evidence that fT causes either male-typical 2D:4D or autism. There could, for example, be a confounding factor that produces both higher ratios of fT/fE and lower 2D:4D.

However, future research, such as that being conducted in the Longitudal Foetal Testosterone Project (at the Autism Research Centre, University of Cambridge), could vindicate such a causal role, even if only in a subgroup of people with ASD. So let us provisionally grant that Premise 1 might be true. If it is true, however, we would need to know whether all or some of the symptoms of autism are produced by higher levels of fT. In this case, for the purposes of confirming Baron-Cohen's theory, *we would still need to know that the specific traits of hypersystemizing and hypoempathizing said to be characteristic of ASD are generated by higher levels of fT—not simply the diagnosis itself.* Premise 1 has not been established.

Premise 2: Fetal Testosterone and Sexually Dimorphic Mental Traits

B t what about the second step of the argument: that fT is responsible for male-typical mental traits? Most of Baron-Cohen's research seems to be aimed at providing evidence for *this* hypothesis, not Premise 1, which is about ASD in particular. Do higher levels of fT correlate with more male-typical mental traits—and, more importantly, are these mental traits weaker versions of those identified in ASD?

Baron-Cohen and others have argued that higher levels of testosterone in amniotic fluid are a good proxy for fT, and that amniotic fluid levels do indeed correlate with male-typical behavior. Cordelia Fine has pointed out that, while this is possible, we do not actually know this.[67] Baron-Cohen denounces Fine as an "extreme social determinis[t]" and defends this claim by pointing out that actually trying to extract blood from fetuses to directly test their testosterone levels would be unethical.[68] However, Fine did not suggest that Baron-Cohen should be faulted for failing to conduct such research; she only argues that his claim that amniotic fluid levels are a good proxy for fT is really just a guess. Rebecca Jordan-Young makes an even broader critique of the research associating fT and future gender behavior.[69] The relationship between the two is simply not well understood. In the absence of further evidence, this claim is only a conjecture, not a confirmed hypothesis. However, let us also provisionally grant that amniotic fluid levels

are indeed a good proxy; we may, in the future, devise an ethical way of substantiating this claim.

Yet many questions arise from this premise; I cannot address all of them here. First, how strong is the evidence for the existence of sexually dimorphic traits identified by Baron-Cohen *et al.*? Second, how significant are the average differences identified? Third, how strong is the correlation between amniotic fluid testosterone and the average differences? Fourth, should such differences be understood as differences in systemizing and empathizing? Fifth, are the sexually dimorphic traits most reliably identified plausibly construed as weaker expressions of autistic traits?

The biggest problem with this premise in his argument, itself the conclusion of a very complicated argument, is this: the evidence that Baron-Cohen presents for sexually dimorphic mental traits is not evidence for the S/E dimorphism that is central to Baron-Cohen's argument. Stating the claim in general terms such as "sexual differences in cognition" masks the question of whether the evidence that supports sexually dimorphic traits is evidence for sexually dimorphic performances on tests of S and E. And it does not appear to do so.

Take the very first study that was said to show such dimorphism at a very early age: the test conducted by Baron-Cohen's graduate student. This study is rife with problems. The sample size was rather small at 102. The study was conducted over ten years ago, yet it has never been replicated. There is a substantial chance that the researchers knew the sex of at least some of the test subjects, and on top of it all, newborn infants have very poor eyesight,[70] making it implausible that the newborns of either sex could see the face *as a face*. But even worse, it does not seem to show that boys tend to systemize or that girls are more empathetic, on average. Looking at geometric shapes is not a tendency to systemize. And looking at faces is not empathizing. Even the claim that girls are (somewhat) more interested in faces is suspect, since girls could be more neurologically developed (Baron-Cohen and many others insist that they are) and therefore more able to recognize faces and find them salient. It shows, at most, a slight tendency among girls to look at faces sooner after birth than do boys. This is simply not anything like the empathizing interest and skill said to be a typically female trait. And all of this assumes that the experiment could be repeated without the problems and with the same results—a very big "if" indeed.

Much of Baron-Cohen's research purports to show that fT positively correlates with "autistic traits."[71] The experiment just cited does not try to show this; it only tries to show an innate average difference between the sexes in a behavioral/mental trait: the amount of time spent looking at a representation of a face, rather than a geometric shape, at a specific postnatal age. Should the above results be replicated, it would still not tell us *why* girls looked at the representation of a face slightly longer. Even if future

research shows that fT levels are negatively correlated with looking at such representations of faces, it will not show that fT is correlated with autistic traits, because the above experiment does not show that such behavior is an autistic trait. Yet Baron-Cohen cites this study as an example of innate differences in empathizing behavior—a female-typical mental trait.[72]

In sum, there is not very strong evidence that there are significant dimorphisms in the specific cognitive and affective traits targeted by Baron-Cohen (and others), and in any case, those traits do not seem to amount to differences in systemizing and empathizing.

Premise 3: Male Prevalence of ASD

Interestingly enough, this claim gives the most intuitive plausibility to the EMB hypothesis, and yet turns out to be the least relevant. It is true that diagnosis rates of autism are significantly higher for males than for females at all levels of impairment, but especially for those with "high-functioning autism" or Asperger's. However, this does not provide support for the idea that autism is an extreme form of the typically male brain. There are many disorders that emerge early in development that are sexually dimorphic. The best known of these are X-linked disorders found in males, such as Fragile X and hemophilia. However, there are many other disorders that are sexually dimorphic and not linked to either sex chromosome, some of which are more prevalent in women (e.g., Graves Disease and many autoimmune disorders), and some of which are more prevalent in men. There is no reason to think that we should use sexually dimorphic prevalence as the basis for giving a disease a special status as particularly characteristic of or "essentially" male or female. So why is ASD's male prevalence not only often cited as evidence for EMB, but is usually offered as the *first* piece of evidence?[73] The answer is that male prevalence should not be offered as evidence for EMB, because it is not evidence. Rather, male prevalence is something that *requires* explanation. While he appears at times to think otherwise, the most charitable interpretation of Baron-Cohen's line of reasoning is that male prevalence is explained by the theory of autism as encompassing both clinical and nonclinical populations as expressions of a broader phenotype: the phenotype of the Male Brain, characterized by hypersystemizing and hypoempathizing. "Male prevalence" belongs below the conclusion line, not above it. Indeed, in his most recent writing, Baron-Cohen has offered EMB as the most plausible *explanation* of male prevalence.[74] The other candidates he considers are *de novo* mutations on the X chromosome as well as mutations on the SRY region of the Y chromosome. Male prevalence does call out for explanation. It is misleading to use it rhetorically as evidence that EMB is true. EMB can only explain male prevalence if EMB is true.

Premise 4: ASD and Male-Typical Traits Are Part of the Same Broad Phenotype

If Premise 4 is correct, it would help to explain male prevalence (Premise 3). The problem, however, is that it is hard to see how, on the basis of evidence offered by Baron-Cohen and others, Premise 4 is actually correct. There are several reasons to doubt that it is.

First and probably most fundamentally, there is no single accepted or even dominant theory as to what autism is. There is substantial disagreement as to how to conceptualize ASD, despite the relatively clear diagnostic criteria of the *DSM-V*. Many symptoms, such as extreme sensitivity to noise or other sensory inputs (especially touch), repetitive behaviors (such as hand-flapping and "stimming"), and seizures are not listed as "core" diagnostic symptoms, despite their pronounced co-occurrence with other autistic symptoms. Many people with ASD are mentally retarded (possibly as many as 50 percent), and yet the EMB pays no attention to this, focusing as it does on people with Asperger's who by definition have normal or above-normal IQs. EMB focuses on just two purported symptoms: hypersystemizing (which Baron-Cohen interprets as a version of the "restricted interests" criterion) and hypoempathizing (which Baron-Cohen interprets as the basis of the "impairments of social communication" criterion). But should we see autism as high S, low E?

There are other major accounts attempting to characterize the major or "core" deficits of autism. Executive functioning, central coherence, mind-blindness, and "Intense-World" theory all propose a fundamental way to understand the disorder. Like Baron-Cohen's account, each is brain-based, neurodevelopmental, and attempts to account for the core symptoms of autism. Intense World theory also proposes to account for common but "non-core" symptoms. Furthermore, each is grounded in brain and behavior research: observation and brain imaging of live human subjects, *post mortem* brain tissue samples from those with autism (although there are not large amounts of tissue from ASD subjects available), and research on rats, mice, and monkeys.

The heterogeneity of autism and its syndromic presentation has defied efforts to identify a single mechanism or set of mechanisms for onset and development. As Geschwind puts it, because of this heterogeneity in presentation, "it is not surprising that no unifying structural or neuropathological features have been conclusively identified."[75] In other words, no one can credibly say that we know what autism is, and we do not yet have a candidate for a major causal mechanism for its development. We cannot say that we really know *what the phenotype for autism is*. Therefore we cannot claim that autism is at the extreme end of the phenotype, and that typical males fall closer to that end of the phenotype than do typical females.

Premise 5: The Sameness Condition

Under what conditions can we say that *A* and *B* are the same thing? If *A* and *B* do not share all of the same properties, they are clearly not identical. However, under certain conditions, we might say that *A* and *B* are different versions or expressions of the same thing. The word "expression" is misleading, because it suggests that there is unique chromosomal or genetic cause of autism diagnoses and that different genetic factors (e.g., different numbers of repeats or deletions on a chromosome, or epigenetic factors) would explain the differences between *A* and *B*. No one, certainly not Baron-Cohen, makes this argument. In this case, in order to defend the Sameness Condition Baron-Cohen must be arguing that the symptoms of ASD are simply more intense versions of typically male traits. The concept of color saturation provides a good analogy here. Two colors can be the same, in the sense that we would all agree that they are the same color, respond to them as "red," and perhaps the measurable wavelength of the light would show that they are both predominantly the same (e.g., 650 nanometers). However, one may be more saturated than another, in that less of the light reflected off of the object would be 650 nanometers; that wavelength would be less dominant in a less saturated sample of the color. Yet we say they are the *same hue*.

In the case of a mental trait, we might consider the ability to perceive sound. A person might be completely unable to perceive sound audibly (although able to feel vibrations), and another might be unable to perceive sounds unless they are very loud. Another might be able to hear within a normal range for humans, except when there is a lot of background noise, making it difficult to listen to a conversation in a crowded room. Still another might be able to detect lower-frequency sound waves, but not high-frequency sound. Some people are deaf in one ear, but not both. All of these perceivers might be said to *share the condition of deafness*.

Notice that the condition of deafness does not depend upon a common cause. One might be deaf because of a congenital condition, because of an inherited condition, or because of trauma to part of the brain or inner ear. One might become deaf as a result of repeated exposure to loud noises or simply because of old age. What deaf people share is a functional deficit in the capacity of hearing. The profoundly deaf and the slightly deaf share the same condition, but to different degrees.

On the face of it, the condition of deafness would seem to support the general version of the Sameness Condition. However, classifying all of the above disorders as the same disorder could be misleading. Someone who cannot hear because of a brain tumor may lack functional hearing, but it is unclear that it is correct to say that such a person has the *same condition* as someone who lost her hearing due to a high fever in early childhood, even if the prescribed remediations were exactly the same. Thus it is unclear that symptoms alone would allow us to say that *A* and *B* are the same condition.

Two such people might have much in common. Yet it is not at all clear that they "have the same thing," in the way that two people with Down syndrome have the same thing: three copies of chromosome 21, rather than the typical two copies.[76]

Even two people with Down syndrome might be said to not share the "same thing," in that some people with Down syndrome do not have a trisomy throughout their entire genome (mosaicism), and some people with Down syndrome have a partial duplication of chromosome 21, or a translocation of part of a chromosome.

What about the "common cause" implicit in the Sameness Condition? Must the symptoms be more or less intense versions of each other *and* have the same cause, as in the case of Down syndrome? This seems too strong. If one person becomes deaf because an early trauma to part of the ear, and another becomes deaf due to genetic causes, they are still both considered deaf. Similarly, if one person has ASD produced by, say, exposure to valproic acid, and another has ASD clearly not produced by exposure to any toxin *in utero*, they still share a diagnosis of ASD.

The Sameness Condition seems, as a general rule, too strong. Diseases are usually understood functionally, often without an established etiology. However, even if some features of autism seem like "darker hues" of some stereotypical male behaviors, that doesn't mean that they *do* have a common cause. We cannot assume without better arguments that the S/E paradigm is useful for understanding the etiology of autism. If autism and male-typical mental traits have a common cause, that would not show that they are the same thing. Similarly, if ASD and male-typical mental traits are different shades of the same hue, establishing a common cause is not necessary. However, neither the claim of a common cause, nor the claim that ASD and male-typical mental traits are different intensities of the same phenomenon, has been established. The main problem is not the common cause, but the issue of whether male-typical mental traits are well understood, and whether they are sufficiently similar to autistic traits so that we can see them as different shades of the same phenomenon. I do not think this has been established.

CONCLUSION: WHY EMB NOW?

From all of this I conclude that there is no sound basis for regarding autism as a "male" disease, or as a form of an Extreme Male Brain. It is male-prevalent, but so are many other diseases. The symptoms and causal mechanisms that lead to a diagnosis of ASD are heterogeneous and still poorly understood. In addition, the average differences between male and female brains, and average differences in the cognitive and affective traits in the

typical population simply do not clearly line up with differences between those with ASD and those without. As one group of researchers puts it, "The autistic brain functions differently, sometimes more like men, sometimes more like women, but we should consider that it might actually function in its own unique way."[77]

So how is it the EMB has come to be so widely referenced and enthusiastically endorsed, especially by researchers and lay people *outside* of the field of autism research? First, developing brain science has led us to become more confident that we can understand not only what the brain looks like, but also how it works, and how it is the source of our behavior, both in clinical and nonclinical populations. Autism is known to be a brain-based developmental disorder, and autism research is rapidly expanding as well as one of the more intensively funded areas of research. Second, popular books about sex differences, from John Gray's *Men Are from Mars, Women Are from Venus* to Louanne Brizendine's *The Female Brain*, claim to identify significant biologically based differences in the behavior, interests, and abilities of men and women. Given the male prevalence of ASD, Baron-Cohen was able to forcefully articulate his theory in ways that resonate with popular understandings of sex differences, making EMB a widely accepted understanding of autism. EMB is not really a scientific hypothesis, so much a piece of philosophy that emerged out of scientific research, and it capitalizes on popular theories of sex difference.

However, the influence goes both ways. EMB supports a social agenda that is quietist about the lack of parity in the STEM professions. It tells us not only that male prevalence in autism is natural, but so is male prevalence in these fields. The uncritical acceptance of EMB will lead to more funding to confirm it. Experiments that aim at shoring up EMB will get funding, and the proponents of EMB are more likely to orient their research around confirming it than disconfirming it. And research funding that goes toward shoring up EMB is funding that is not, for example, going toward showing what happens in the brain stems of people with ASD *in utero* (as the Intense World Syndrome account would indicate). It is also funding that is not going toward developing techniques of remediating the worst symptoms of autism or newer, more effective biomedical interventions. Brain science, psychology, and our interest in gender differences in humans led to EMB. And EMB will lead to more research that aims at confirming scientific claims that would support it. EMB's connection with gender will garner attention and allow us to ignore more heterogeneous findings of autism research.

The stakes are high. Funding is limited, and putting our scarce resources into a dubious scientific agenda that is attached to a quietist social agenda is dangerous. While he claims to have no social agenda, Baron-Cohen argues that not only is the male prevalence of autism a product of biology, but so is the male prevalence in math and science. There is no good argument that

autism is just an extreme version of typically male traits, and yet Baron-Cohen and others are happy to conclude that male-dominated science, math, and engineering is largely natural. If we push for parity between men and women in the sciences, it is argued, then we are free to do so, but we are working against nature. This is a powerful disincentive to pursue equity in the STEM disciplines, and it contributes to the already powerful stereotype threat that women in STEM must face as they move through their careers—or drop out of them. Although it is tempting to move from basic scientific research to more general, grand, and popular claims about men and women, this is a temptation that should be resisted. Such extraordinary claims required extraordinary evidence—evidence that simply has not been produced.

REFERENCES

Aldridge, M. A., Stone, K. R., Sweeney, M. H., & Bower, T. G. R. 2000. Preverbal Children with Autism Understand the Intentions of Others. *Developmental Science* 3 (3):294.

Amaral, D. G., Bauman, M. D., and Schumann, C. M. 2003. Review the Amygdala and Autism: Implications from Non-human Primate Studies. *Genes, Brain & Behavior* 2 (5):295–302.

American Psychiatric Association (APA). *Diagnostic and Statistical Manual of Mental Disorders*, ed. IV and V. http://www.dsm5.org/proposedrevision/pages/proposedrevision.aspx?rid=94.

Auyeung, B., Baron-Cohen, S., Ashwin, E., Knickmeyer, R., Taylor, K., and Hackett, G. 2009. Fetal Testosterone and Autistic Traits. *British Journal of Psychology* 100 (1):1–22.

Auyeung, B., Baron-Cohen, S., Ashwin, E., Knickmeyer, R., Taylor, K., Hackett, G., & Hines, M. 2009. Fetal Testosterone Predicts Sexually Differentiated Childhood Behavior in Girls and in Boys. *Psychological Science* 20 (2): 144–48.

Badcock, C. R. 2009. *The Imprinted Brain: How Genes Set the Balance between Autism and Psychosis*. London: Jessica Kingsley Publishers.

Badcock, C., and Crespi, B. 2008. Battle of the Sexes May Set the Brain. *Nature*:1054–55.

Barbeau, E. B., Mendrek, A., and Mottron, L. 2009. Are Autistic Traits Autistic? *British Journal of Psychology* 100 (1):23–28.

Baron-Cohen, S. 1995. *Mindblindness: An Essay on Autism and Theory of Mind*. Cambridge, MA: MIT Press.

Baron-Cohen, S. 2003a. *The Essential Difference: The Truth About the Male and Female Brain*. New York: Basic Books.

Baron-Cohen, S. 2003b. They Just Can't Help It. *The Guardian* (April 17, 2001), August 1, 2011.

Baron-Cohen, S. 2005a. The Essential Difference: The Male and Female Brain. *Phi Kappa Phi Forum* 85 (1):23–26.

Baron-Cohen, S. 2005b. Testing the Extreme Male Brain (EMB) Theory of Autism: Let the Data Speak for Themselves. *Cognitive Neuropsychiatry* 10 (1):77–81.

Baron-Cohen, S. 2007. Sex Differences in Mind: Keeping Science Distinct from Social Policy. In W. M. Williams, and S. J. Ceci (Eds.), *Why Aren't More Women in Science?*, 159–72. Washington, DC: American Psychological Association.

Baron-Cohen, S. 2008. Autism, Hypersystemizing, and Truth. *Quarterly Journal of Experimental Psychology* 61 (1):64–75.

Baron-Cohen, S. 2009. Publish and Be Distorted. *New Scientist* 201 (2701):26–27.

Baron-Cohen, S. 2010. Delusions of Gender—'Neurosexism,' Biology, and Politics. *The Psychologist* 23 (11):904–5.

Baron-Cohen, S. 2011. Inside the Mind of a Man. *New Statesman* 140 (5033):38–39.

Baron-Cohen, S., Auyeung, B., Ashwin, E., and Knickmeyer, R. 2009. Fetal Testosterone and Autistic Traits: A Response to Three Fascinating Commentaries. *British Journal of Psychology* 100 (1):39–47.
Baron-Cohen, S., and Hammer, J. 1997. Parents of Children with Asperger Syndrome: What Is the Cognitive Phenotype? *Journal of Cognitive Neuroscience* 9 (4):548.
Baron-Cohen, S., Knickmeyer, R. C., and Belmonte, M. K. 2005. Sex Differences in the Brain: Implications for Explaining Autism. *Science* 310 (5749):819–23.
Baron-Cohen, S., Leslie, A. M., and Frith, U. 1985. Does the Autistic Child Have a "Theory of Mind?" *Cognition* 21 (1):37–46.
Baron-Cohen, S., Lombardo, M. V., Auyeung, B., Ashwin, E., Chakrabarti, B., and Knickmeyer, R. 2011. Why Are Autism Spectrum Conditions More Prevalent in Males? *PLoSBiol.* 9(6), e1001081.
Baron-Cohen, S., Lutchmaya, S., and Knickmeyer, R. 2004. *Prenatal Testosterone in Mind: Amniotic Fluid Studies*. Cambridge, MA: MIT Press.
Begley, S. 2009. Pink Brain, Blue Brain. *Newsweek* 154 (11):28–28.
Blatt, G. J. 2010. *The Neurochemical Basis of Autism from Molecules to Minicolumns*. Berlin, NY: Springer.
Brizendine, L., 2006. *The Female Brain*. New York: Morgan Road Books.
———. 2010. *The Male Brain*. New York: Broadway Books.
Ceci, S. J., and Williams, W. M. 2007. *Why Aren't More Women in Science?: Top Researchers Debate the Evidence*. Washington, DC: American Psychological Association.
Crespi, B., and Badcock, C. 2008. Psychosis and Autism as Diametrical Disorders of the Social Brain. *The Behavioral and Brain Sciences* 31 (3):241.
Dreger, A., Feder, E. K., and Tamar-Mattis, A. 2010. Preventing Homosexuality (and Uppity Women) in the Womb. Message posted to http://www.thehastingscenter.org/Bioethicsforum/Post.aspx?id=4754.
Eliot, L. 2009. *Pink Brain, Blue Brain: How Small Differences Grow into Troublesome Gaps—And What We Can Do About It*. Boston: Houghton Mifflin Harcourt.
Ellis, H. D. 2005. Book review. *Cognitive Neuropsychiatry* 10 (1):73–75.
Fine, C. 2010. *Delusions of Gender: How Our Minds, Society, and Neurosexism Create Difference*, 1st ed. New York: W. W. Norton.
Frith, C. D. 2004. Schizophrenia and Theory of Mind. *Psychological Medicine* 34 (03):385.
Geschwind, D. H. 2009. Advances in Autism. *Annual Review of Medicine* 60(1):367–80.
Gray, J. 1992. *Men Are from Mars, Women Are from Venus: A Practical Guide for Improving Communication and Getting What You Want in Your Relationships*. New York: HarperCollins.
Grice, D. E., and Buxbaum, J. D. 2006. The Genetics of Autism Spectrum Disorders. *Neuromolecular Medicine* 8 (4):451–60.
Hallmayer, J., Cleveland, S., Torres, A., Phillips, J., Cohen, B., Torigoe, T., Risch, N. 2011. Genetic Heritability and Shared Environmental Factors Among Twin Pairs with Autism. *Archives of General Psychiatry*.
Hayes, T. B. 1998. Sex Determination and Primary sex differentiation in amphibians: Genetic and Developmental Mechanisms. *Journal of Experimental Zoology* 281 (5):373–99.
Hill, A. 2009. Doctors Are Failing To Spot Asperger's in Girls. Guardian News and Media.
Holtcamp, W. 2009. Lone Parents: Parthenogenesis in Sharks. *Bioscience* 59 (7):546–50.
Jordan-Young, R. 2010. *Brain Storm: The Flaws in the Science of Sex Differences*. Cambridge, MA: Harvard University Press.
Keller, F., and Ruta, L. 2010. The Male Prevalence in Autism Spectrum Disorders: Hypotheses on Its Neurobiological Basis. In G. J. Blatt (Ed.), *The Neurochemical Basis of Autism: From Molecules to Minicolumns*, 13–28. Berlin, NY: Springer.
King, M., and Bearman, P. 2009. Diagnostic Change and the Increased Prevalence of Autism. *International Journal of Epidemiology* 38 (5):1224–34.
Kripke, S. A. 1980. *Naming and Necessity*. Cambridge, MA: Harvard University Press.
Kunzig, R. 2004. Autism: What's Sex Got To Do With It? *Psychology Today* 37 (1):66–76.
Locke, J., Yolton, J. W., and Yolton, J. S. 1989.*Some Thoughts Concerning Education*. New York: Clarendon Press; Oxford: Oxford University Press.

Lutchmaya, S., Baron-Cohen, S., and Raggatt, P. 2002. Foetal Testosterone and Eye Contact in 12-month-old Human Infants. *Infant Behavior & Development* 25 (3):327.
Manning, J. T., Reimers, S., Baron-Cohen, S., Wheelwright, S., and Fink, B. 2010. Sexually Dimorphic Traits (digit ratio, body height, systemizing–empathizing scores) and Gender Segregation between Occupations: Evidence from the BBC Internet Study. *Personality & Individual Differences* 49 (5):511–15.
Minio-Paluello, I., Lombardo, M. V., Chakrabarti, B., Wheelwright, S., and Baron-Cohen, S. 2009. Response to Smith's Letter to the Editor 'Emotional Empathy in Autism Spectrum Conditions: Weak, Intact, or Heightened?' *Journal of Autism & Developmental Disorders* 39 (12):1749–54.
Pease, B., and Pease, A. 2000. *Why Men Don't Listen & Women Can't Read Maps: How We're Different and What To Do About It*. New York: Welcome Rain.
Pinker, S. 2008. *The Sexual Paradox: Men, Women and the Real Gender Gap*. New York: Scribner.
Same Difference: How Gender Myths Are Hurting Our Relationships, Our Children, and Our Jobs. 2005. *Future Survey* 27 (6):21–22.
Skuse, D. H. 2000. Imprinting, the X-chromosome, and the Male Brain: Explaining Sex Differences in the Liability to Autism. *Pediatric Research* 47 (1):9–16.
Smith, A. 2009. Emotional Empathy in Autism Spectrum Conditions: Weak, Intact, or Heightened? *Journal of Autism & Developmental Disorders* 39 (12):1747–48.
Turkheimer, E. 2010.The It Strikes Back. *PsycCRITIQUES* 55 (24): No Pagination.
Valla, J. M., Ganzel, B. L., Yoder, K. J., Chen, G. M., Lyman, L. T., Sidari, A. P., Belmonte, M. K. 2010. More than Maths and Mindreading: Sex Differences in Empathizing/Systemizing Covariance. *Autism Research* 3 (4):174–84.
Wheelwright, S., Baron-Cohen, S., Goldenfeld, N., Delaney, J., Fine, D., Smith, R., Wakabayashi, A. 2006. Predicting Autism Spectrum Quotient (AQ) from the Systemizing Quotient-revised (SQ-R) and Empathy Quotient (EQ). *Brain Research* 1079 (1):47–56.
Witt, C."Feminist History of Philosophy." *The Stanford Encyclopedia of Philosophy (fall 2008 edition), Edward N. Zalta (ed.)*.
Witt, C. 2011. *Feminist Metaphysics: Explorations in the Ontology of Sex, Gender and the Self*. New York: Springer.
Zimmerman, A. W. 2008. *Autism Current Theories and Evidence*. New York: Springer.

NOTES

This chapter has benefited from comments from John Collins, Cathy Frierson, Sarah Richardson, Julia Rodriguez, Paula Salvio, and Charlotte Witt, as well as audiences at the University of New Hampshire Law School's Implicit Bias Seminar and the University of New Hampshire Philosophy Department.

1. Baron-Cohen 2003.
2. I will use ASD to refer to the group of three developmental disorders of PDD-NOS, Asperger's, and autistic disorder, as described in the *DSM-IV*. Current experimental research into autism and ASD typically excludes subjects with primary diagnoses that are often associated with autistic symptoms—e.g. diagnoses such as Fragile X, Prader-Willi, and tuberous sclerosis; they also exclude subjects whose autism was subsequent to exposure to environmental agents such as valproic acid, thalidomide, and rubella (Grice and Buxbaum 2006).
3. See, e.g., Pinker 2008; Brizendine 2006 and 2007; Pease and Pease 2000.
4. Baron-Cohen 2008, 78.
5. Keller and Ruta 2010, 15.
6. Crespi and Badcock 2008.
7. Baron-Cohen 2007.
8. Baron-Cohen 2010a, 904.

9. Turkheimer 2010.
10. Baron-Cohen 2008.
11. Hallmayer *et al.* 2011, E1.
12. Zimmerman 2008.
13. Markram, Rinaldi, and Markram 2007.
14. Geschwind 2009.
15. Russell 1997.
16. Frith 1989; Happe and Frith 2006.
17. Baron-Cohen *et al.* 1985; Frifth and Happe 1994.
18. Amaral *et al.* 2003; Baron-Cohen *et al.* 2000.
19. Markram, Rinaldi, and Markram 2007.
20. King and Bearman 2009.
21. See http://www.dsm5.org/proposedrevision/pages/proposedrevision.aspx?rid=94 for *DSM-IV* and *DSM-V* and APA's rationale for the changes.
22. Witt 2008.
23. Locke, John. 1692. *Some Thoughts Concerning Education.*
24. Mill, John Stuart. 1869. *The Subjection of Women.*
25. Richardson 2010.
26. Baron-Cohen 2003, 185.
27. Baron-Cohen 2003, 122.
28. Baron-Cohen 2003, 127.
29. Crespi and Badcock 2008.
30. Crespi 2009.
31. Baron-Cohen 2003, 171–76.
32. Baron-Cohen 2008, 68.
33. Baron-Cohen, 1995. The Sally-Anne test (Wimmer and Perner 1983) evaluates whether a subject will can distinguish between the subject's own belief about the location of an object and another person's. Briefly, the subject watches two actors (in the original experiment dolls were used), Sally and Anne, as Sally places an object (e.g., a marble) in a basket as Anne watches. Sally then leaves the room, and Anne subsequently moves the object to her own box. The subject is asked, "Where will Sally look for the marble?" In some studies, children with autism more often predict that Sally will look in the new location, whereas lower IQ but nonautistic children (e.g., children with Down syndrome), accurately predict that Ann will look in the first location. This is supposed to show that at least some autistic children—those who are functional enough to respond the question—have deficits in the ability to theorize about other minds. Not everyone thinks this test is a good test of "theory of mind." See Bloom and German 2000.
34. Minio-Paluello *et al.* 2009.
35. Baron-Cohen 2008, 68.
36. Baron-Cohen 2008, 65.
37. For example the *DSM-IV* states: "A total of six (*or more*) items from (1), (2), and (3), *with at least two from* (1), *and one each from* (2) and (3)" (emphasis added) (APA).
38. Baron-Cohen 2008, 66; Baron-Cohen does not make the static–dynamic distinction, but I add it in order to clarify.
39. Baron-Cohen 2008, 65.
40. Baron-Cohen 2008, 66.
41. Baron-Cohen 2005, 95–104.
42. Baron-Cohen 2005, 1.
43. Baron-Cohen 2007, 169.
44. Kripke 1980.
45. Baron-Cohen 2005, 1.
46. Witt 2011.
47. Hayes 1998.
48. Holtcamp 2009.
49. Baron-Cohen 2007, 165.
50. Baron-Cohen 2003.

51. Baron-Cohen 2007.
52. Auyeung, Baron-Cohen *et al.* 2009.
53. Goya and McEwen 1980.
54. Baron-Cohen, Lutchmaya, and Knickmeyer 2004, 9–11.
55. Auyeung *et al.* 2009.
56. Eliot 2009, 28.
57. Baron-Cohen *et al.* 2004, 17–19.
58. However, estradiol, which is produced by testosterone through aromatization, has not been ruled out, and some have argued that "oestrogen, rather than testosterone, is the critical hormone to understand the skewed sex ratio in autism" (Keller and Ruta 2010, 20).
59. Baron-Cohen *et al.* 2004, 50.
60. Eliot 2009, 81.
61. Zimmerman 2008, 191.
62. Zimmerman 2008, 201.
63. Durand *et al.* 2007.
64. Geschwind 2009, 370.
65. Auyeung *et al.* 2009.
66. Baron-Cohen 2005, 154.
67. Fine 2010, 108.
68. Baron-Cohen 2010, 904.
69. Jordan-Young 2010.
70. Fine 2010, 112–17.
71. Auyeung *et al.* 2009.
72. Baron-Cohen 2003, 56.
73. Auyeung *et al.* 2009; Crespi and Badcock 2008.
74. Baron-Cohen *et al.* 2011.
75. Geschwind 2009.
76. Even two people with Down syndrome might be said to not share the "same thing," in that some people with Down syndrome do not have a trisomy throughout their entire genome (mosaicism), and some people with Down syndrome have a partial duplication of chromosome 21, or a translocation of part of a chromosome.
77. Barbeau, Mendrek, and Mottron 2009, 27.