Chapter One

Autism: The Very Idea

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There are many pressing questions that one might ask about autism. These include:

How will I know if my child is autistic?
What will be the prognosis if she is?
Is autism treatable?
Is autism curable?
Should autism be eradicated?
Why wasn't autism discovered before the 1940s?
Why have the rates of diagnosis risen so sharply over the past couple of decades?

Before those questions can be addressed, the more fundamental question must be answered: what *is* autism? Answering this question is tricky. For one thing, there is more than one way to take the question. It could be asking what *kind* of thing autism is (the *genus*, you might say): a complex of behaviors? a psychological condition? a kind of neurology? a genetic condition? Alternatively, it could be asking what makes it its own special instance of that kind (the *differentia*): *which* behaviors? *what* psychological condition/neurology/genetics? Call these *categorical* issues. On the other hand, it could be asking whether or not autism is a real knowledge-independent entity in the world for which we have just recently coined a term (on the model of something like Down syndrome) or if the concept is a human artifact that either does not track any real entity (like "neurotic" or other now-abandoned theoretical notions) or tracks behavior that is a product of human culture of a specific time and place (like "hipster"). This is a *metaphysical* question. As Locke might put it: once we establish the *nominal* essence of autism (the content of our concept of autism), we want to know if there is a *real* essence

out there in the world. We want to know if autism is like "water," where it emerged that there really was a distinct kind of molecular substance to the stuff we just used to pick out by its wetness, clarity and ability safely to slake thirst.

THE CRITERIA FOR DIAGNOSIS

Before we can address either categorical or metaphysical questions, we need to have a first pass of a description to work with, and it seems appropriate to begin by returning to the source: the seminal paper "Autistic Disturbances of Affective Contact"[1] in which Leo Kanner first posited the existence of a syndrome that we now know as autism.[2] Commenting on Kanner's article more than forty years after its publication, (now Sir) Michael Rutter wrote:

> There are few scientific papers that have stood the test of time as well as Leo Kanner's first description of the syndrome that came to bear his name. The fact that he was the first person to recognize that this constellation of behaviors constituted a condition that was different from the general run of problems grouped under "mental retardation" or "schizophrenia" was quite enough for the paper to receive an honored place in the history of psychiatry. It was indeed a reflection of Kanner's remarkable clinical acumen that he was able to see so clearly that which had escaped the notice of his many distinguished contemporaries and predecessors… [F]urther research demonstrated that he had been correct in his identification of the key features that held the syndrome together. As Leon Eisenberg commented in his preface to the 1973 collection of Kanner's papers on autism, "The genius of the discovery was to detect the cardinal traits…in the midst of phenomenology as diverse as muteness in one child and verbal precocity in another."[3]

Kanner's achievement, then, was picking out a "constellation of behaviors" that were the "cardinal traits" or "key features" that "held the syndrome together" that has since come to be called autism. So what were these key features? Kanner writes that "even a quick review" of the eleven case studies of children that had been brought to him "makes the emergence of a number of essential common characteristics appear inevitable," characteristics that together "form a unique 'syndrome,' not heretofore reported."[4] Those characteristics (the vast majority of which are today considered either indicators of autism or seen to be commonly related phenomena) included: late speaking; a use of language that was rote and focused mainly on the use of nouns to identify objects, colors, or numbers; excellent rote memory; "delayed echolalia" (delayed because the "parrot-like repetitions" could be stored for later); personal pronouns "repeated just as heard, with no change to suit the altered situation;" common failure to attend to people calling on them; fussiness about food; adverse reaction to loud noises and moving objects; lack of

spontaneity; treatment of people like objects; possession of "good cognitive potentialities" and "strikingly intelligent physiognomies;" clumsiness in gait allied with skill in finer muscle coordination.[5]

What kind of an answer is this to the question we began with? Suppose we say that *this* is autism: the exhibition of the set of characteristics in Kanner's list. Call this analysis the Bundle of Behaviors (BoB) Model. It is the simplest answer to the question "what is autism?" It is the answer: "what Kanner observed." In some ways it is the most optimistic, because it implies that the condition can be discarded in the way that any behaviors can be ended, however difficult that might be, and that successful behavioral therapy would not simply be a way to *mask* autism, but be a cure. Children could be autistic, and then later, not.[6]

Is it a satisfactory answer? Well, not really, for a number of reasons.

First, it seems more likely that the *essence* of autism lies deeper than the surface. Consider an analogous behavioral analysis of what "homosexuality" is.[7] To keep it simple, let's focus on what seems to be the key piece of behavior for homosexuality: same-sex sex acts. This is unsatisfactory as an analysis of what it is to be homosexual, because it seems neither necessary (the concept of a homosexual virgin makes perfect sense) nor sufficient (sex workers who perform same-sex sex acts could perfectly well be heterosexual). The behavior in each case seems to be an *effect* (or "symptom" in the medicalized language that used to apply to homosexuality and still does to autism) of the condition rather than itself *constituting* the condition.

Second, there is the problem of vagueness of boundaries. While some examples of behavior are clear-cut (e.g., misuse of pronouns), others are vague and meeting them would seem to be a matter of opinion and context. Just about every child is fussy about food to a certain extent—what counts as the degree necessary to meet the criterion here? The suspicion is that it is not the *degree* that matters but the *cause*. But referring to a (presumably nonbehavioral) cause means abandoning a simple behavioral model.

Third, we need to know if *every single* item on Kanner's list is necessary for a diagnosis of autism. Should lacking even one of those characteristics be sufficient to establish that one is not autistic? We are accustomed to think that this would be too strict a requirement, that "milder" versions of certain syndromes can lack one or two of the symptoms of the "full-blown" syndrome. But the simple BoB model is just a conjunction of all the "cardinal traits." There is no reason to believe that each trait cannot occur in people who do not have the condition. We cannot know whether or not there is a *separate* condition that lacks just one of the traits (a particular issue if we believe that Asperger syndrome is distinct). A diagnosis of autism would be like a conviction for breaking and entering in this respect: you can only be guilty of breaking and entering if you meet *every* requirement (breaking or entering/a building/without the owner's or tenant's permission/with intent to

commit a felony). Meeting any but the full quota is not a mild version, it is no version at all (which is a relief to those of us who have forgotten our keys, or to firefighters intent on rescuing possible inhabitants of a burning building).

Perhaps the standards might be loosened somewhat. Suppose that I claim to have discovered "William Shatner Syndrome," the compulsive need to behave like that much impersonated actor, and give as constituent elements an exhaustive list of his behavioral characteristics. This would be analogous to the BoB model of autism, in that there is no theorizing about 'a deeper cause:' the syndrome just is the set of behaviors. Suppose further that a patient comes in who is suspected of having the syndrome. Would he have to exhibit every single item on my exhaustive list? Perhaps, instead, we could make a decision on what was the *essence* of "Shatnerosity" and insist that the behaviors that were part of this core cluster were essential, while the others were just "comorbid symptoms," likely indicators of the possibility that one had the syndrome, but not individually necessary. Of course, deciding on where to draw the line between the essential and the accidental characteristics would be a fraught issue. We have seen something like that in the debate over whether or not language delays are essential (ruling out Asperger syndrome as a kind of autism) or not. But who is the "William Shatner" of autism? Would it be the eleven children in Kanner's original study? Surely, though, we want to leave open the possibility that one or more of those was *not* autistic, or at least, even if all were, that they were not so *simply by definition*. However, on the simple BoB model, it would be an analytic truth that they were autistic, because autism would be defined as "behaving like those children."

Clearly this is not the assumption that clinicians have operated on, because it is just not the case that Kanner's list has been seen as sacrosanct. The most authoritative current list of characteristics that merit a diagnosis are to be found in the fourth edition of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders*. This manual is the resource to which a vast number of clinicians turn in deciding whether or not a patient is autistic. It was originally brought into existence to standardize diagnoses that had up to that point varied wildly among clinicians.[8] The entry on "Autistic Disorder" in *DSM-IV* presents an alternative strategy from both the simple method of requiring *every single* characteristic and making a distinction between a set core and an outer ring of nonessentials. The approach might be called the Cluster of Behaviors (CoB) model. Here are the diagnostic criteria:

A. 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):

1. qualitative impairment in social interaction, as manifested by at least two of the following:
   a. marked impairment in the use of multiple nonverbal behaviors such as eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction
   b. failure to develop peer relationships appropriate to developmental level
   c. a lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (e.g., by a lack of showing, bringing, or pointing out objects of interest)
   d. lack of social or emotional reciprocity
2. qualitative impairments in communication as manifested by at least one of the following:
   a. delay in, or total lack of, the development of spoken language (not accompanied by an attempt to compensate through alternative modes of communication such as gesture or mime)
   b. in individuals with adequate speech, marked impairment in the ability to initiate or sustain a conversation with others
   c. stereotyped and repetitive use of language or idiosyncratic language
   d. lack of varied, spontaneous make-believe play or social imitative play appropriate to developmental level
3. restricted repetitive and stereotyped patterns of behavior, interests, and activities, as manifested by at least one of the following:
   a. encompassing preoccupation with one or more stereotyped and restricted patterns of interest that is abnormal either in intensity or focus
   b. apparently inflexible adherence to specific, nonfunctional routines or rituals
   c. stereotyped and repetitive motor mannerisms (e.g., hand or finger flapping or twisting, or complex whole body movements)
   d. persistent preoccupation with parts of objects

B. Delays or abnormal functioning in at least one of the following areas, with onset prior to age three years:

1. social interaction,
2. language as used in social communication, or
3. symbolic or imaginative play.

The CoB approach is to identify key *types* and demand that each be instantiated by one or more *tokens*. No single individual characteristic is essential, but each type must be represented.

However, what this list gains in flexibility, it loses in specificity. Now it seems that a wide variety of differing individuals could all meet the same diagnosis. One person might meet the diagnosis by being A1ab2a3aB1, while another by being A1cd2b3bB2, and so on. Why should we assume that there is some essentially similar syndrome that they share? (The assumption is that each person, while exhibiting different behaviors, is exhibiting the same set of types of behaviors. But if the behaviors are different enough to warrant separate descriptions, then even if they share being *one* type of behavior, each differs from the other in other respects, like being "play" or "communication.")

One could respond that each unique individual who scored above a threshold level on this points system shares a "family resemblance" (as all things called "games" do in Wittgenstein's famous example) to any other, and that this is enough. But that would undermine the status of the diagnosis as a precise scientific one. It begins to seem less likely that this checklist is picking out a condition that "carves nature at the joints," but rather that it is a rough-and-ready way of sorting people into groups for some predetermined social purpose. It seems to be treating an autism diagnosis on the model of entrance criteria into a prestigious university: to make the grade you have to be "well-rounded." Medically, however, it would appear to make more sense to avoid the "catch-all" diagnosis of autism altogether and focus instead on the various individual criteria.

To put it another way: one key problem with BoB was explaining the "string" that bound the bundle of various characteristics into a *syndrome*. Why *those* characteristics? Moving to CoB seemed to make the collection less arbitrary but, conversely, we now need to know why no individual item on Kanner's list is necessary (and some have been dropped altogether). What motivated the shift? (This is especially pressing given that some of the things that Kanner described but that are left out of the new list—sensitivity to various kinds of sensory stimulation for example—should be recognizable to a huge number of parents of autistic children. Why have those criteria been excised?) It wasn't motivated by an attempt to describe the eleven original children more accurately, because only Kanner himself attempted any follow-up.

What is more, the original problem with BoB recurs at a new level: what is it that binds the three *types* together into a syndrome? Why those, and only those *types*?

A FORAY INTO METAPHYSICS

It might seem academic to wonder about the justification for the difference between the diagnostic lists provided by Kanner and the *DSM-IV*—Kanner, after all, was only the first person to talk about the syndrome and could be expected to have made some mistakes that could be corrected by advances made since. Why shouldn't we simply say "we know more about autism now?"

Such a response, however, is not available to a theory that is committed to either a Bundle or a Cluster theory. Recall William Shatner Syndrome: what is the "more" that we could know? Such an account cannot appeal to anything outside of the Bundle or Cluster to justify altering the components. We shall shortly address theories that suggest that the characteristics on the list are *effects* or *symptoms* of autism, rather than constitutive elements of it, but for now the puzzle remains. What is more, the issue of providing a justification for a changed list of characteristics is very pressing because the fifth edition of the *DSM*—the first major revision in seventeen years—is soon to be released, and while there is some dispute about what effect the changes in the definition of what is now called "*Autism Spectrum* Disorder"[9] will have it seems clear that the changes will (and perhaps are intended to) restrict the numbers of people who meet the diagnosis, as the following quotes illustrate:

> "We have to make sure not everybody who is a little odd gets a diagnosis of autism or Asperger disorder," said Dr. David J. Kupfer, a professor of psychiatry at the University of Pittsburgh and chairman of the task force making the revisions, which are still subject to change. "It involves a use of treatment resources. It becomes a cost issue."[10]

> "The changes would narrow the diagnosis so much that it could effectively end the autism surge," said Dr. Fred R. Volkmar, director of the Child Study Center at the Yale School of Medicine and the author of the new analysis of the proposal. "We would nip it in the bud."[11]

Suppose we interpret such statements as suggesting that the changes between *DSM IV* and *V* were partly motivated by financial considerations. How should one respond to that as a justification for changes to the standards for diagnosis? Different responses track different metaphysical views, depending on whether or not it is thought that the essence of autism is to be found solely in the intrinsic properties of those labeled as autistic or to depend also on social facts extrinsic to them.

On one view, this is either irresponsible or only justified by a belief that the earlier standard of diagnosis cast too broad a net. A *realist* would take the

view that the number of people who *really are* autistic is unaffected by words in a book. For the realist, autism should be viewed on the model of conditions like Down syndrome: a biological phenomenon that humans have had throughout the centuries but which has been identified and named only comparatively recently.[12] But a realist would never be happy with the implication that cost *alone* should be a motivation for setting the standards of diagnosis: the goal of a diagnosis would be to capture as closely as possible all and only those people who genuinely have autism.

On the other hand, one could draw a contrasting lesson from this controversy. To the extent that "autism" has reality, it is realized in the individuals who are picked out by diagnosticians as autistic. And exactly who is picked out as autistic depends greatly on the definition given in the *DSM*. So rather than the diagnosis being driven by the attempt to capture an independently existing reality, the explanatory arrow could be reversed: the existence of the phenomenon depends on the diagnosis, where the content of the diagnosis could be driven by ideology or economics more than medical science. This is the view that autism is a "socially constructed" phenomenon.

There are two main ways autism could be socially constructed. It could be like *race*: that is, although there are biological (although not necessarily genetic) bases for things like skin pigmentation, eye and/or nose shape, hair texture and other characteristics that have been taken as race *traits*, there is nothing in nature that marks those traits as distinctively racial. Not only is the collection of traits arbitrary (Why not ear size, belly button type or "tongue rollability?") but there is no "glue" that groups (for example) all the supposedly "black" traits together, so that they either all occur together in particular individuals or not at all. For this reason, biologists have asserted that racial *categories* have no biological reality. "Race" is a socially constructed phenomenon that arbitrarily groups traits together that have no underlying common cause, genetic or otherwise. If autism were like that, then it would lose status as a medically useful category.[13] That would not mean, however, that the conditions that make it up, such as language delays, social difficulties, problems of proprioception, problems with sensory integration *et al.*, would not each be conditions worth studying and treating, just that they are not all linked by being *autism* traits.

(A more extreme social constructivist view of autism would imply that the condition itself is a human creation, that the label has provided a source of identity for people who might otherwise be just awkward.[14] On this view "autism" would turn out to be like "female hysteria"—something whose diagnosis was just self-reinforcing. On this view even the so-called symptoms are not biological realities but culturally produced chimeras. I think this latter view can be dismissed.)

To summarize: the two major metaphysical views about autism are *realism*, the view that what Kanner first identified *is* a genuine cross-cultural

(and possibly even cross-species) psychological/biological phenomenon whose true essence will soon be captured (in the way that the essence of AIDS turned out to be the HIV virus, or the essence of Down syndrome turned out to be an extra chromosome), and *social constructivism*, the view that "autism" is a concept that groups together independently existing, and independently treatable conditions—and no biological basis for *that* will be found, any more than we will find the "fabulousness" gene or the chromosomes responsible for the combination of liking country music at the same time as being Catholic. (Or for acting like William Shatner.) I think it safe to say that the predominant view is realism. One aim of this chapter is to argue that the vagueness surrounding the concept of autism undermines attempts to isolate something intrinsic shared uniquely by those labeled autistic, and that, therefore, the possibility that there *is* no real essence corresponding to the concept of autism, and that it is a social construct, should be more seriously entertained.

However, this conclusion concerning the metaphysical question is premature, as so far we have only considered the behavioral answers to the categorical question.

Recall that both the Bundle and Cluster theories faced the challenge of explaining why it was that we should group the various behaviors (whether tokens or types) under a single heading, and relatedly, how we could ever justify adding or subtracting behaviors from the group. What was it that tied the bundle together?

Obviously if there were a *common cause* of the behaviors then this would answer both challenges. All behaviors that were effects of the same cause should be counted as evidence for the presence of the same condition, and any that were not could be dropped from the criteria for a diagnosis. And the kind of thing that could underlie and explain behaviors in this way would be a *psychological* condition.

PSYCHOLOGICAL ACCOUNTS

Recall the analogy with homosexuality with which I called into question strictly behavioral analyses: a better account of what it is to be homosexual posits a *psychological phenomenon* that would underlie and explain the behaviors taken to be evidence of homosexuality. How about *same-sex sexual attraction*?[15] Locating the essence of homosexuality at the psychological level allows for the phenomenon of being "closeted" and of discovering oneself to be gay without actually having had sex. A psychological phenomenon can cause certain behaviors, but can be counteracted by other psychological states, so that it can be present without obvious behavioral results. Autistic adults can learn to "act normal."

It is important to distinguish between *being in a particular psychological state* and *having a certain psychological tendency*. If we defined homosexuality just as *actually experiencing* same-sex sexual attraction, then, again, homosexuality would be a "gappy" condition: for example, even one prone to excessive arousal would still not be homosexual while in a dreamless sleep. So a more sophisticated, but perhaps somewhat tendentious account (how could you falsify it?) would define being homosexual as "being such that, if one *were* to experience sexual attraction, it would be to someone (whom one perceives as) of the same sex."

What are the implications of a model that locates the essence for a condition at the level of psychological states?

The first is one already mentioned, that one could have the condition and it might not be evident to observers. For example, if one was not yet of a stage in one's development that one experienced *any* sexual attraction, then there would be no evidence that one was homosexual. (Of course, this is what makes the "sophisticated" psychological account suspicious—we cannot tell whether or not the condition is *absent* or "dormant"). And, of course, many children do not display symptoms of autism until at least eighteen months of age.

Second, such an analysis would be *agnostic on the genesis* of the psychological condition. For homosexuality it allows equally that one could be born a homosexual, one could be conditioned into homosexuality, or one might even choose it. Viewing autism in this light leaves open the possibility of "acquired autism," where the relevant psychological states could result from any number of causes (the mother having rubella while pregnant, for example). And, in fact, a distinction is now made between "idiopathic" autism, whose causes are unknown, and what is called "secondary" autism where the causes are said to be understood. That this distinction is made implies that autism itself is not located at the level of causes, even if these were genetic. However, I think our intuitions would rebel at calling somebody autistic who was neurotypical until his thirties but on being the victim of head trauma suddenly displayed Kanner's symptoms.

Third, a psychological model allows for the possibility of a "cure:" whatever one's genetics or history, it is in theory possible for one to cease to have any particular psychological state.[16]

Contrast this with the behavioral account: one is always in the position to choose to perform whatever behavior characterized the condition, but one cannot choose to have an attraction or choose to be "mindblind."[17]

Suppose we agree that autism is essentially a psychological condition (the genus part of categorization). The question then turns to the *differentia*: *what* psychological condition? It has to be one that can explain the behaviors that we are now taking to be *symptoms* of autism (rather than *components* of autism). There are two clear ways a proposed condition could fail to be a

satisfactory explanation. First, it cannot simply be "whatever it is that causes those symptoms" because this begs the question in favor of a single condition and a single cause, while at the same time committing the *virtus dormitiva* sin of pretending to provide an answer without actually saying anything "contentful." Second, it must not simply be that a contentful psychological state is chosen that, on the face of it, cannot explain all of the disparate behaviors that make up the diagnosis of autism, but recalcitrant ones get dropped or artificially redescribed to suggest a unity that was not there before.

Did Kanner see a unity underlying his original traits? In a paper written some twenty-eight years after his seminal paper, he appears to suggest as much: "The outstanding pathognomonic characteristics were viewed as (a) the children's inability from the beginning of life to relate themselves to people and situations in the ordinary way, and (b) an anxiously obsessive desire for the preservation of sameness."[18]

I think there is a little bit of revisionism in his description: as noted in the previous section, in the original paper he describes *behaviors*, and they include a broad range, not easily clustered under only two headings (although he does stray from this in one instance, as I discuss below). But besides this, it is not clear how helpful these groupings are. An "inability…to relate…in the ordinary way" is at best a vague and woolly classification that could fit an enormous range of behaviors. It points to a need to research the cause of the inability without giving any clues as to where that might be found. And "anxiously obsessive desire" is, if anything, a step backwards from the simple description of the behaviors, loaded as the adjectives are with either rather dated or unjustifiably specific psychological notions.

The one glaring instance where the original study departs from a cautiously objective cataloguing of behaviors and indulges in psychological theorizing comes when Kanner attributes to the children "an all-powerful need for being left undisturbed." That the children have this "need" is then used to explain a range of behaviors:

> Everything that is brought to the children from the outside, everything that changes his external or even internal, environment, represents a dreaded intrusion.
>
> *Food* is the earliest intrusion that is brought to the child from the outside…Our patients…anxious to keep the outside world away, indicated this by the refusal of food…
>
> Another intrusion comes from *loud noises and moving objects*, which are therefore reacted to with horror…Yet it is not the noise or motion itself that is dreaded. The disturbance comes from the noise or motion that intrudes itself, or threatens to intrude itself, upon the child's aloneness.[19]

Is Kanner offering up the various kinds of behavior as evidence for the general psychological "need" or is he instead interpreting the behavior in light of a presupposed theory? While the former would be more defensible methodologically, it is of dubious explanatory worth. Furthermore, in the examples above he attempts to give an explanation for sensory issues for which the more likely explanation is biological. It looks like an overreach by a psychologist. In fact, I think we have cause now to question Kanner's descriptions of the traits as prejudiced by a preconceived speculation on an underlying mechanism, a speculation for which he could not have had adequate support.

What are some more recent suggestions for psychological essences or bases of autism? The three major candidates are: impaired theory of mind (the most influential version of which is Simon Baron-Cohen's "mindblindness" theory), weak central coherence, and executive dysfunction. As it turns out, the criticisms of each of these turn on its inability to account for certain symptoms taken to be essential indicators of autism.

In his 1995 book *Mindblindness*, Simon Baron-Cohen contended first, that neurotypical people understand others by having a "theory of mind" and second, that this faculty is impaired in autistic individuals. This view has been criticized from many different perspectives: from the charge that his evidence for this claim (the "Sally/Anne" false belief test) is inconclusive at best (hardly any autistic adult fails the test, while many nonautistic yet intellectually impaired and most deaf children are similarly delayed in their capacity to pass it[20]) to the criticism that the notion that *anybody* has a "theory of mind" is based on a misconception of how the mind works. But perhaps most seriously for an account of autism, the theory fails to account for what are deemed essential elements of it. Many autistic children are obviously so at an age before *any* child is deemed to have a "theory of mind," and the theory has nothing to say about repetitive behaviors, narrow interests and physical issues like being touch averse or hyper-sensitive to sensory inputs.[21]

Baron-Cohen now acknowledges that this theory alone is inadequate, but believes that it can be part of a complete theory, the "empathizing-systemizing" theory. As he ties this account closely with what he calls the "extreme male brain" theory, I will postpone discussion until the section on brain-based accounts of the essence of autism. Let us turn now to the second psychological theory.

Uta Frith suggested the following "hypothesis about the nature of the intellectual dysfunction in Autism":

> In the normal cognitive system there is a built-in propensity to form coherence over as wide a range of stimuli as possible, and to generalize over as wide a range of contexts as possible. It is this drive that results in grand systems of thought, and ultimately in the world's great religions. It is this capacity for coherence that is diminished in autistic children. As a result, their information-processing systems, like their very beings, are characterized by detachment.[22]

A number of experiments led Frith to this conclusion, in particular ones that showed a superior ability to pick out specific details (faces hidden in pictures, for example) which she interpreted as showing that autistic children were not concentrating on the whole, trying to find meaning in it, but instead viewing each part individually.

Frith has modified this "weak central coherence" (WCC) theory substantially over the years, and there is some suggestion that it could link up with theories about the autistic brain that show "short range overconnectivity" but "long range underconnectivity"—that is, that more neurons than in neurotypical brains link local areas in the brain but fewer link distant areas.[23]

WCC appears to show promise in explaining a range of phenomena indicative of autism:[24] Besides enhanced perception of visual detail, there is also hypersensitivity to change (because any detail is noticed) face recognition difficulty (because only the parts of the face are perceived, not the whole face), and a well-known tendency to play with the parts of toys (e.g., the wheels of a toy car) rather than the whole toys. That said, however, the explanation is at best "loose" (are we seriously to believe that the autistic child does not *know* that the whole thing is a car?) and furthermore, several of these conditions are well known, and indeed severe, in people not labeled as autistic.[25] Moreover, however much you modify WCC, it seems that it could, at best, explain a subset of the characteristics first noticed by Kanner, and none of the motor issues so characteristic of autistic children.[26]

"Executive function" is the ability to control activities, both mental and physical. Defects in this function could impair *inhibitory* control (the ability to stop doing what one is doing), *flexibility* (ability to change activities or switch attention to new projects) and the ability to *initiate* new activities. The major features of autism that executive dysfunction is said to explain are the repetitive behaviors, reliance on routine, "obsessive" interests and (purported) lack of creativity,[27] as well as the inability to plan a schedule for oneself. However, it does not seem equipped to explain any sensory issues or the things the other theories purport to explain, viz., social difficulties or enhanced perception of detail, so it is at best incomplete. Also, repetitive behaviors, reliance on routine, etc., are features of other distinct psychological conditions, like Obsessive Compulsive Disorder or Tourette's syndrome, so even if executive dysfunction explained these things it would not be a unique indicator of autism. Finally, as with the other theories, there are many criticisms of the inferences drawn from the various tests that supposedly support the theory.[28]

So we see that no single psychological mechanism yet proposed can capture all of the core features of autism. We must conclude that if there is a

psychological essence to autism it consists of more than any single one of these. We are then faced with a familiar dilemma, only one layer down from before: is autism *simply* a bundle or cluster of psychological mechanisms, or should we look deeper for a common cause that unites all in the cluster? A related but distinct question concerns whether or not the presence of any of these mechanisms alone is sufficient to warrant a diagnosis for autism (even if we allow that autism *consists of* more than the mechanism alone), or if one can have a psychology that exhibits the mechanism and yet not be autistic.

Theories that posit particular psychological modules or mechanisms as explanations of behavior face a further challenge: explain what exactly the module or mechanism *consists in*. Where exactly are these psychological phenomena located? As each of them is a *deficiency*, what *exactly* is it that autistic people lack?

Such challenges inevitably lead us to biology, and, more specifically, neurobiology. Let us turn now to discussion of the "autistic brain."

BRAAAAAAAAINS!

When we talk about the autistic brain, what exactly do we mean? We could mean one of three things: the brain that *results* from autism, the type of brain states that *constitute* autism or the brain that *causes* autism. Which of these it is depends on what level autism is located at, an issue that we have yet to settle. We began with the idea that autism *was* a "constellation of behaviors." But the strange implications of BoB and CoB pointed towards a psychological account. Were there a single unified psychological account, we would still want to know what underpinned that psychological state. Assuming that psychological states *reduce* to states of the brain (that is, psychological states cannot exist except where there are brains in certain states), it is still an open question whether or not we should *identify* autism as a psychological state with a condition of the brain ("identifying" reductivism[29]) or say that, while brain states are all there is to the psychological states in question, one cannot just talk in the language of brain biology and capture the essence of those psychological states ("constitutive" reductivism[30]).

Finally, if it turned out that there was an "autistic gene," then, if we decided that *that* was the essence of autism, then the type of brain that uniquely resulted from that genetic nature would be an *effect* of autism, and not the cause *or* essence.

But, of course, we have not found a satisfactory unified psychological account, which suggests either that autism is not a unified phenomenon, but in fact a bundle of distinct, often comorbid, but actually independent conditions (and that the term "autism" does not carve nature at the joints any more than the term "Coca-Cola" captures a natural kind), or that, if autism is a

single state, then it must be the common cause of these disparate psychological conditions. This suggests that (a) a particular kind of brain is what constitutes autism (just as a particular molecular structure constitutes "water") and (b) for autism to be a real, distinct phenomenon, this should be a single particular kind of brain, and not a set of conditions that can occur independently of each other.

What have studies shown? Sadly, there is as much disunity among brain researchers as we have seen among psychologists. This is not surprising though, because, as Jill Boucher writes:

> The implications of findings on brain function are easily identified because the research is always hypothesis-driven. That is to say, each study is designed to test a specific hypothesis concerning the neural activity that occurs when the person being tested is carrying out a specific task.[31]

In other words, the brain research does not drive the psychological theorizing but rather the reverse. To give a single example, consider the study "Decreased Connectivity and Cerebellar Activity in Autism during Motor Task Performance."[32] This study was referred to in an article entitled "New Insight into the Neurological Basis of Autism," which described the study as follows:

> Researchers used fMRI scans to examine the brain activity of 13 children with high functioning autism and 13 typically developing children while performing sequential finger tapping. The typically developing children had increased activity in the cerebellum, a region of the brain important for automating motor tasks, while children with autism had increased activity in the supplementary motor area (SMA), a region of the brain important for conscious movement. This suggests children with autism have to recruit and rely on more conscious, effortful motor planning because they are not able to rely on the cerebellum to automate tasks.
>
> Researchers also examined the functional connectivity of the brain regions involved in motor planning and execution in order to compare the activity between different brain regions involved in the same task. The children with autism showed substantially decreased connectivity between the different brain regions involved in motor planning and execution. These results add to increasing evidence that autism is related to abnormalities in structural and functional brain connectivity, which makes it difficult for distant regions of the brain to learn skills and coordinate activities.[33]

There are several points worth noting in this study. The first is that this is a study of only thirteen children. (Of course, while this number seems tiny, it is still two more than Kanner's original study.) Second, it is assumed that these thirteen children *all* are autistic, while none of the control group are. This is

worth noting in particular because these children are all "high functioning" (HFA) and thus closer to "normal" children. But that assumes that we *know what autism is*, and can recognize it perfectly when we see it, something it has been a point of this chapter to challenge. (And if we could do that *perfectly*, then what exactly are we discovering with these brain studies? What if we discovered variance among the brains of similarly-behaving children? In fact, we always *do* discover variance, but it is glossed over.)

Then, of course, there is the assumption (inherent in the title of the article, if not the study itself) that conclusions about HFA children can be generalized to all autistic individuals.

Finally, in the study itself (and unremarked on in the article), we find this:

> Some of the children with HFA were taking psychoactive medications, and the potential impact of this cannot be discounted. Future investigations might benefit from exclusion of children taking medications, though this would have a detrimental impact on recruitment of numbers sufficient to examine group differences using BOLD fMRI.[34]

One would have thought that this would be a very serious strike against the study, reminiscent of the study that claimed to show an essential difference between the brains of gay men and those of straight men, but whose "gay brains" were all the brains of men who had died of AIDS.[35] Yet the authors of the study justify proceeding because of the difficulty of recruiting participants. Not ideal circumstances for good science.

Supposing we put these criticisms aside: what lessons should be drawn from a study of reaction times of finger-tapping? The lead author of the study, Dr. Stewart H. Mostofsky, a pediatric neurologist in the Department of Developmental Cognitive Neurology at the Kennedy Krieger Institute, had this to say:

> Tapping your fingers is a simple action, but it involves communication and coordination between several regions of the brain. These results suggest that in children with autism, fairly close regions of the brains involved in motor tasks have difficulty coordinating activity. If decreased connectivity is at the heart of autism, it makes sense social and communication skills are greatly impaired, as they involve even more complex coordination between more distant areas of the brain.[36]

The generalizing assumptions at work here are breathtaking, especially in light of the facts noted about the study above. Why, one might wonder, are such generalizing assumptions necessary? Why study reaction times instead of, you know, the *social and communication skills themselves*? The article author offers an explanation:

> While autism is characterized by impaired communication and social skills, these abilities are hard for scientists to measure and quantify. In contrast, the neurological processes behind motor skills are well understood, and motor tasks can be objectively observed and measured.[37]

One is reminded of F. Lee Bailey's favorite analogy of a person who had dropped money on a dark street, looking for it under a street light some distance away "because the light was better there."

Put aside all the criticisms I have so far raised and assume that this study really does demonstrate a brain difference between thirteen genuinely autistic children and thirteen "perfectly" neurotypical children—perhaps the most damning assumption built into Mostofsky's claim is that all individuals who have the motor skills issues will have communication and social issues as well. That is exactly the "unifying" assumption about autism that we want brain studies to be testing. And note that these are "high functioning" children whose communication difficulties might be less serious. Meanwhile, Kanner himself noted of the children in his study that "[s]everal of the children were somewhat clumsy in gait and gross motor performances, but *all were very skillful in terms of finer muscle coordination,*"[38] suggesting that generalizing across motor abilities in autistic children is inadvisable. A person who worked with many autistic adults once told me that she knew someone who was a regional tennis champion yet was unable to tie his own shoelaces. Perhaps this was evidence of some lack of connectivity, but if so, then it is clearly possible to excel at some tasks while connectivity is impaired, so to suggest that communication difficulties can automatically be explained in this way seems rash.

Earlier I noted that Baron-Cohen linked his recent empathizing-systemizing with what he calls the "extreme male brain" theory. Briefly, as you may imagine, he believes that brains are "sexed," that female brains are different from male brains.[39] One might think that he arrived at this view *from a study of brains*, but one would be wrong. The "brain" theory is actually a theory about ways of thinking. Essentially, Baron-Cohen claims that one can distinguish between empathizing (of which mindreading is the "cognitive" aspect, but it also has an affective component of "having an appropriate emotional reaction to another person's thoughts and feelings"[40]) and "systemizing" ("the drive to analyze or construct systems"). Males, he claims, are better at the latter than the former, and for females it is the reverse. Meanwhile, autistic people are *much* better systemizers than empathizers. Now, Baron-Cohen's theory is open to all sorts of criticisms, including several in this volume, but I will focus here on the following claim:

> Recently the extreme male brain theory has been extended to the level of neurology, with some interesting findings emerging. Thus, in some regions of

> the brain that on average are smaller in males than in females, people with autism have even bigger brain regions than typical males.[41]

That first sentence should give one pause: it is only recently that a theory called the extreme male brain theory has been "extended" to the level of neurology. But that aside, of course, we would want to be very certain that we had correctly identified the full array of types of people who are autistic and compared their brains with the full array of people who are "neurotypical" (recall the study of "gay" brains that were all from people who had died of AIDS), and if, as has been suggested, female autistic individuals tend to go underdiagnosed, then this might be problematic.[42] But this aside, Baron-Cohen follows up the previous bold claim with this rather pathetic qualifier: "not all studies support this pattern but some do."[43]

It would seem that Baron-Cohen's theory is an extreme example of Boucher's assertion that the research is "hypothesis driven." Now, this is not necessarily bad, especially if one espouses a Popperian hypothetico-deductive model of scientific progress. But Popper would, I think, want more effort put towards *falsifying* the claim than in searching for *some* study that appears consistent with it. The fact that only *some* studies "support" this pattern might be enough to undermine the hypothesis already. Furthermore, what we do *not* have here is anything like the evidence we would want to claim that there is a uniquely identifiable autistic brain that explains and unifies the disparate elements of autism, thereby justifying the label of autism as a natural kind of phenomenon.

IN THE GENES

Given all of the problems we have noted, what is to be gained by going one stage further and investigating the genetic basis for autism? We have failed to come to a satisfactory account of what the evidence of autism is, or what people to count as autistic so that we know whose genes to study.

However, there are reasons to believe that autism could be located at the genetic level even with the problems outlined above. Consider the following:

> There is now considerable evidence from family and twin studies that, for a subgroup of autistic individuals, the etiology is mainly genetic. The risk of recurrence of autism in families (i.e., the frequency of autism in subsequently born siblings) is estimated at 6%-8%, or up to 200 times the risk in the general population. Three twin studies of geographically defined populations detected pairwise concordance rates of approximately 65% (the average over the three studies) and 0% in monozygotic and dyzogotic pairs, respectively, producing a heritability estimate of over 90%. Further, there is no convincing evidence that perinatal factors play an important role in the etiology of most cases of autism.[44]

What recent studies seem to show is that autism is *genetic* (like Down syndrome) because of the high rate of concordance in monozygotic twins, *familial* (unlike Down), but that *environmental factors* also must play a part (because in 10 percent of cases one monozygotic twin has autism but the other doesn't at all, and in cases where both have it, the severity can vary widely).[45]

Kanner himself suggested a genetic basis in his original study,[46] but that theory was not investigated for some time because, as Folstein and Rutter noted in the introduction to a landmark 1977 study of twins, "[t]here is no recorded case of an autistic child having an overtly autistic parent and it is decidedly unusual for a family to contain more than one autistic child." That these appeared to be the facts at the time did not undermine a genetic basis because, they argued: "First, it is extremely rare for autistic persons to marry…and there is only a single published report of one having given birth to a child…Second, autism is a very uncommon disorder, occurring in only about 2-4 children out of every 10,000."[47] Both reasons now seem rather quaint. Of course the diagnosis of autism has skyrocketed, but what about the claim that autistic individuals do not marry or even reproduce? This is where the *broader autism phenotype* (BAP) comes in. According to one of the earliest studies to refer to this phenomenon it is "a behavioral phenotype that is qualitatively similar to but more broadly defined than that which defines autism occurs more commonly in relatives of autistic individuals than in the general population."[48] Or, as Baron-Cohen puts it, "mild echoes" of autism:

> This might take the form of being socially withdrawn or confused by social interaction, or mildly obsessive (in the sense of having strong narrow interests or a need for sameness) or having excellent attention to detail and remarkable memory. Although [the close relatives of autistic individuals] don't have autism or Asperger syndrome itself, they have a milder manifestation of the same characteristics.[49]

The existence of BAPs in families of autistic individuals is taken both to lend support to the idea that autism has a familial, genetic basis, and to explain how it is that the reproducing happens, if it is not to be done by severely autistic individuals. It would also explain the apparent high incidence of autism in the children of academics in such disciplines as philosophy and in such places as Silicon Valley: the kind of focus and interest that leads one to succeed in certain intellectual pursuits is itself evidence of a broader autism phenotype.

At the same time, however, this very notion of BAPs might be taken to undermine the idea that autism can have a status as a distinct, self-contained phenomenon. If the conditions that are taken to be evidence for the presence of autism can occur individually, without the others that are needed for a diagnosis of "full-blown" autism, then the bundle is broken, the cluster un-

clustered, and autism just becomes shorthand for a collection of not necessarily related phenomena, rare in the way that left-handed stamp-collecting Elvis-loving archeologists are rare, and equally devoid of a common explanation.

One clear rebuttal to this would be the following suggestion: autism *is* essentially a certain genetic condition that *tends* to produce defects in all the areas noted by the *DSM*, but whose effect can be blunted by other environmental factors or indeed, interaction with other genetic conditions. (This would also make sense of the spectrum model of autism.) Those who do not exhibit *all* the symptoms only do not because other factors have interfered with the influence of their autistic genes on their brain and psychology. On this account, autism is essentially a genetic condition. An "autistic brain" would be a likely (but not essential) effect of possession of the relevant gene(s). Autism would no more be curable than any genetic condition, only eliminable from the population by selective abortion on the basis of an *in utero* or PGD genetic test. One could actually be autistic, however, and "pass" as nonautistic (as, one would assume, huge numbers of individuals "passed" while only the extreme version was counted as autistic), if the influence of the genes were sufficiently blunted.

Conversely, there could be many individuals who exhibited Kanner's symptoms because of nongenetic influences on their brains who would *not* be autistic since their symptoms did not have the specific genetic cause.

Compare with water. We now say that water *is* H_2O, but knowing something is H_2O without knowing its specific environment won't tell us whether or not it is solid, liquid, gas, opaque, scattered or gathered in a sphere. Conversely, things that are not H_2O can have almost identical tendencies (totally identical, if we allow the possibility of a Putnamian twin earth[50]) without being water.

This would achieve the unity of essence for autism in the face of a great range of realizations of the condition, but at the cost of breaking the necessary connection between autism and the syndrome described by Kanner in the first place. It might turn out that "the autistic" include in their number many people who would not meet any behavioral diagnosis, and exclude many who would. At this point one might well question whether or not we really had discovered the genetic essence of autism or instead discovered that autism had to be located at a higher level (behavior/psychology/brain type) and had no real essence. Our position on this would not be a matter that science could settle. Science could (at best) tell us that this particular genetic makeup had this particular effect in these particular circumstances, but there are distinct issues that should affect our decision that are based on human interests.

Consider the issue of biological sex in humans. While throughout history sex determination has usually been made on the basis of genitals and secondary sexual characteristics, it is now generally accepted that to be female is to have XX chromosomes, while to be male is to have XY chromosomes. The trouble is, there are individuals with conditions that mean they are neither XX nor XY, such as Turner Syndrome, or XO, which is when the individual lacks a second X chromosome, and Klinefelter Syndrome, or XXY, where the individual has an extra X chromosome. What are we to say is the sex of such individuals? Furthermore, some individuals have a "mosaic" of sex chromosomes: they have XX in some cells, XY in others and possibly other types in other cells. And finally, there are cases of individuals who outwardly appear one sex but are chromosomally the other.

Consider the case of Maria Patino, twenty-four years old in 1985 and at the time Spain's top female hurdler (or so she thought), who had to take a chromosomal "sex test" in order to compete as a woman in World University Games in Kobe, Japan. Such tests were the norm for athletes at the time and she thought nothing of it until, just before she was to compete in her first race, she was informed that she had failed the sex test: in the eyes of the International Olympic Committee (IOC) she was not, in fact, a woman.[51] It emerged that Patino had Androgen Insensitivity Syndrome (AIS), so named because typically an XY fetus develops male genitalia as a result of exposure to androgen *in utero*, but AIS individuals do not respond to that androgen. As a result, they are usually designated female at birth, and grow up thinking of themselves in that way.

Imagine yourself in Patino's position—how would you react? The reaction that Patino in fact had, was to deny that the chromosomes should be the deciding factor concerning her sex. "I knew I was a woman in the eyes of medicine, God, and most of all, in my own eyes," she insisted.[52] It seems hard to argue with Patino's conclusion, and increasingly it is accepted that "transgender" individuals whose genitals "match up" with their chromosomes, but who identify with the other sex should be allowed to classify themselves how they want. One's sex, then, is not simply settled by one's chromosomes.[53]

Wouldn't the same be true of autism even if we did find an "autism gene?" Clearly one's status as autistic does not play as basic a role in one's identity as one's sex (at least for the majority of people), but it is a status that carries with it certain social and economic riders. So, just as imprisoned transgender individuals are campaigning to be moved to a prison that reflects their self-identity (or at least, removed from one that doesn't), so a self-identified autistic individual might demand rights to certain services that, increasingly, states are requiring be made available to autistic individuals, especially if that individual, while not having the requisite genetic makeup, had the behavioral or sensory challenges we have associated with autism.

All of this assumes that there *is* a simple, definite genetic basis to at least the majority of the conditions we have been associating with autism. Howev-

er, a study of papers published between 1961 and 2003 on the genetics of autism (and that cites over 200 of them) reached the following conclusion:

> Although many genes and proteins have been implicated as causes of autism, too little is known about their functions or their role in brain development to generate a parsimonious hypothesis about the brain dysfunctions that underlie autism. Evidence from multiplex families with the broader autism phenotypes, together with twin studies, indicates that single-gene defects are rare even within families....Despite the profusion of investigations into the genetics of autism, few significant genetic linkages to autism have been identified.[54]

Scientists have been saying for years that the "gay gene" is about to be discovered, and we seem no nearer. And what it is to be gay is, if anything, simpler to state than what it is to be autistic.

CONCLUSION

We do not have a clear conception of what autism is. The closest "official" definition is the diagnosis in *DSM-IV* that is, as befits a diagnosis available to the general practitioner, in terms of observable behaviors. However, one of the several problems with a behavioral definition is that it makes the changes soon to come in *DSM-V* either arbitrary or solely politically/economically motivated. (This should come of no surprise to those familiar with the history of the APA's changing attitude towards homosexuality.) Attempts to locate the essence of autism at a deeper level than observable behavior have been varied but unsatisfactory. Various psychological "modules" either fail to explain all of the behaviors picked out as distinctive of autism, appear present in some people not labeled autistic, or are so vaguely defined that their essence seems simply to be "whatever it is that would underlie the behavior that needs explaining." If autism is to be a *collection* of such modules (or defects in various modules) then we either need to know the justification for grouping them together (the "bundle" problem again) or we need a common explanation at the level of neurology. However, suggested brain conditions are again either specific to only a subset of the symptoms of autism or not distinctive to autism alone. Finally, genetic bases are suggested by the familial grouping of autistic-like symptoms, but if the same genetics explain BAPs as explain "full-blown" autism, then again, the net is cast too wide.

In general, while most people would no doubt think that "science" is driving a deepening understanding of what autism is, I would argue that science is, if anything, demonstrating that there is no such thing as "autism" if we are to understand it on the model of something like Down syndrome. At best there are several conditions that can occur independently of each other,

but seem to co-occur, that can vary significantly in severity, and that seem to cluster in families, but not necessarily always. I propose that we focus instead on specific deficiencies, like sensory processing disorders, communication difficulties or food sensitivities and stop trying to cluster them together as something called "autism." To do so is like dividing hair types into "black hair" and "Caucasian hair." As any hairdresser with a diverse clientele knows, there is huge variety in each, and some types of "black" hair are much closer to some types of "Caucasian" hair than to other types supposedly racially similar.

EPILOGUE: SO WHAT?

Recall the list of pressing questions I gave at the start. Let us look at how each of these is affected by what categorical or metaphysical position one takes on the issue.

How will I know if my child is autistic?

The "true test" will either be behavioral, psychological (which would essentially put the child into a position where her behavior would exhibit a supposed underlying deficit, as in the case of the "Sally-Anne" false belief tests for theory of mind), neurological (say, using "three dimensional brain scans," recently developed by researchers at the University of Pittsburgh Medical Center[55]) or chromosomal, depending on what level one places autism at (the "genus" issue). But either this will mean that "autism" is much more narrowly defined than presently, or being told that one's child is autistic will be remarkably uninformative. But that should be familiar to parents who have been through a diagnosis anyway.

What will be the prognosis if she is?

Again, BAPs significantly cloud the issue. I suspect that a great number of very successful, creative, funny, and/or athletic individuals would turn out to be classifiable within the Broader Autism Phenotype. Conversely, if a brain scan did show a significantly nonneurotypical brain, then I think it would be progress in neuroscience that would enable experts to explain exactly what difficulties the owner of that brain might be expected to have. And I don't think this would be usefully defined as an advance in our knowledge of *autism*, just an advance in our knowledge of *brains*.

Is autism treatable?

That depends. It seems evident that each aspect of autism can be treated, but by different specialists and in different ways. Special diets, both edible and sensory, various speech therapies, and simple loving interaction are all

salutary. If autism is genetic, or consists in "disorganized" brain wiring, then these therapies couldn't really be said to be treating *autism*. But should we care that they are not if they are making life easier and open to more opportunities to the individual so labeled? And the downside to the label is that treatments that might be of benefit to a wide range of children are only provided *on the condition* that one is so labeled.[56] Parents have then to make a decision to strive to get their child a diagnosis in order to get treatment, knowing full well the stigma that comes attached to it. A separate issue is the turf wars that have emerged over treatment of autism that turn on just such disputes about what exactly autism is. Consider the issue of special gluten-/casein-/soy-free diets. On the one hand, there is the claim that switching to a special diet can "cure" a child of ASD. As we have seen, the idea that autism is curable is rejected by scientists who predict that it will be found to have a genetic basis. These same scientists tend to dismiss any claims made by people who purport to offer a cure. But it nonetheless appears clear that for a number of children diagnosed as autistic, a special diet can, like the "sensory" diet, have a calming effect, reduce wild mood swings and instances of acting out, and in general make life better for children and parents. In this respect, some of the apparent symptoms of autism are ameliorated. So this might be viewed as a clash between those who define autism as the behavioral symptoms Kanner listed, such that if some of the more disturbing ones disappear, allowing the child to be calmer and more social, then in a real sense the *autism* is being treated, and those who locate autism at a genetic level, so that a diet could never affect it. One can feel that one is siding with the quacks in attempting a diet regime that could actually help greatly. All of this could be avoided with a more measured and focused description that avoided talk of autism (and the entrenched camps and interest groups that have developed around that term) altogether.

Is autism curable?

The talk of a "cure" for autism seems to be geared towards finding a genetic test for it. This would not, of course, be a way to "cure" it in an individual, but rather as an attempt to prevent autistic people being born in the first place. A better "cure" might be to change society so that it is more accepting of and accessible to individuals with differences of the kind Kanner first noted. Perhaps a first step towards this would be to get rid of the label "autistic." Of course, this suggestion is just like the idea that we should get rid of racial labels, and ignores the fact that some people are relieved to be labeled, either because it "makes sense" of difficulties they have struggled with for a long time,[57] or because it gives them a group to identify with when much of society has regarded them as alien. But more narrow, precise, and syndrome-specific labels could do that too. And just as there is racism, there

are already many negative stereotypes about autistic individuals, in particular that they lack empathy and are dangerously violent.

Should autism be eradicated?

If autism is identified with inability to communicate, self-harming and perseverative activities and all of the most extreme and debilitating indicators, then one can see how this might be appealing. But it matters greatly if this is to be achieved by making therapies available to those whose opportunities in life would widen greatly as a result, or by *eugenics*. If "autism" really is socially constructed, then I'm all in favor of its elimination. The people will still be the same, but they won't be encumbered with a catch-all term weighted down with decades of bad theorizing and "Rain Man" assumptions.

Why wasn't autism discovered before the 1940s?
Why have the rates of diagnosis risen so sharply over the past couple of decades?

It's interesting that social constructivism has an answer to this that would be the same as similar questions about rates of diagnosis of female hysteria or multiple personality disorder. Realists would say that the phenomena were always there (like the famous cases of "feral children," like Victor the Wild Boy of Aveyron in the 1780s) and it's only now that we have developed a concept that maps precisely onto the real essence of a biological condition, so that we can clearly see what was always there. But, as I hope I have shown, our concept is anything but clear, and indeed, progress can only come from abandoning it and starting from scratch. There will never be consensus in all the various brain and chromosome studies if they all compete to be the explanation of *autism*. But if instead they just attempt to explain some narrow feature of the human condition (that has often been lumped in with autism), then the competition dissolves and clarity emerges from discord. But how easy would it be to get grants or to raise funds or to get laws changed without the magic word "autism" to rally behind?

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NOTES

1. Kanner 1943.
2. As is now well known, Hans Asperger was working along similar lines to Kanner in Austria at the time. I do not mean to say that in some sense Kanner "won the race," just that Kanner had a head start on influencing the concept in the English-speaking world, as Asperger's work was not translated from the German for many decades.
3. Rutter 1985, 50–51, emphasis added. Historian Chloe Silverman notes that the continuing relevance of Kanner's descriptions contrasts markedly with contemporary descriptions of other mental disorders that sound hopelessly archaic today (Grinker 2007, 44).
4. Kanner 1943, 41.
5. This list, and each quote, comes from Kanner 1943, 42–48.
6. Kamran Nazeer relates meeting as an adult the teacher who knew him as a child labeled as autistic. When Nazeer exhibited a sense of humor and an apparent ability to interpret her behavior, she said "you're not autistic." Nazeer 2006, 183.
7. Why homosexuality? Because it was a condition first identified by psychologists and labeled as a disorder, and over the nature of which there is continuing debate. Like autism, it was also blamed on "refrigerator mothers." Further parallels are noted in the discussion of brain studies below.
8. See Grinker 2007, 107–121.
9. The notion that autism is a spectrum was first proposed in Wing and Gould 1979 (where it is called a "continuum"). Wing was the person who translated Asperger's work into English and named the syndrome after him. See Boucher 2009, chapter 2, for a discussion of the merits of the "subtypes" (*DSM–IV*) vs. "spectrum" views of autism. It seems that the spectrum view has won out, at least terminologically.
10. Amy Harmon, "A Specialists' Debate on Autism Has Many Worried Observers," *New York Times*, January 20, 2012.
11. Benedict Carey, "New Definition of Autism Will Exclude Many, Study Suggests," *New York Times*, January 19, 2012.
12. Many people who study autism assert a strong parallel with Down syndrome, predicting that autism will be found to have a genetic marker, and an *in utero* test will eventually be available. See discussion of genetics below.
13. It is sometimes claimed that keeping track of race data is medically useful, because of racially specific conditions like sickle-cell anemia. But in fact there is no disease that tracks racial categories, even if some diseases can be traced back to a common origin in a specific region.
14. Consider this diatribe from comedian Dennis Leary on p. 87 of his book *Why We Suck* (New York: Viking, 2008): "There is a huge boom in autism right now because inattentive mothers and competitive dads want an explanation for why their dumb-ass kids can't compete academically, so they throw money into the happy laps of shrinks...to get back diagnoses that help explain away the deficiencies of their junior morons. I don't give a shit what these crackerjack whack jobs tell you—yer kid is NOT autistic. He's just stupid. Or lazy. Or both." (He does go on to talk about "true" autism, so his overall view might be realism.)
15. There might still be behaviorists who want to define "attraction" in terms of empirically observable changes in behavior (blushing, perspiring, increased pulse, et al.), but there remains the possibility that a skilled actor could either hide attraction that was present or exhibit behavior consistent with an attraction he did not have, and that it is the attraction, not the resulting behavior, that is essential.
16. However, because of the first implication noted above, it would be very hard to know if a cure had actually been achieved or if the "therapy" was just masking or counteracting the effects of a still-present psychological phenomenon. Compare the different attitudes to so-called reparative therapy that purports, in flagrant disregard of the APA's ban on such claims, to "cure" homosexuality: one side claims it to be a genuine cure, the other that the "cured" are just in the closet, suppressing their real desires.
17. Contrast also with locating autism at a genetic level: one cannot change one's genes, so talk of a "cure" for autism would on a genetic account be at best science fiction. This is discussed below.
18. Kanner 1971, 229.
19. Kanner 1943, 44.
20. Baron-Cohen himself points out that "a range of clinical conditions show forms of mindblindness (such as patients with schizophrenia, or narcissistic and borderline personality disorders, and children with conduct disorder), so this may not be specific to autism and Asperger syndrome" (Baron-Cohen 2008, 61).
21. "Its shortcoming is that it cannot account for the non-social features" (Baron-Cohen 2008, 61).
22. Frith 1989, 100. Perhaps it is the autistic in me that sees the production of the "great religions of the world" as of dubious merit.
23. Baron-Cohen 2008, 56.
24. Boucher 2009, (102) uses WCC as an example of "one-too-many" explanation, where one theory explains many phenomena.
25. Well-known neurologist Oliver Sacks is so face-blind that he cannot tell if he is looking at his own reflection in a pane of glass or through it at somebody else (until, as he recounted humorously on a radio interview, that person moved). The painter (ironically, of faces) Chuck Close is also almost totally face-blind.
26. See Boucher 2009, 211; 213. My personal complaint is that this theory presents a caricature of the autistic individual as one who cannot see the forest for the trees. However, I have already noted how prevalent I have found autism to be among the children of philosophers, and I would hazard a guess that the "analytic tendency" is just this predilection to focus on the basic building blocks of things. The same would be true among theoretical scientists. Yet

there is no paucity of "grand systems" in philosophy or the natural sciences. I find it odd, indeed, that Frith sees the culmination of systematizing as the "great religions," which seems to me the least systematic modes of thinking. It is worth noting that Baron-Cohen's most recent theory posits instead that autistic individuals are superior "systemizers" [sic] where "systemizing is the drive to analyze or construct systems" (Baron-Cohen 2008, 63, emphasis added). Perhaps both theories could haggle over Wittgenstein (a common choice in the "diagnose the famous autistic person" game): Baron-Cohen could have the Wittgenstein of the *Tractatus*, while Frith could have the *Philosophical Investigations*. Kant has always leaped out to me as someone with autistic tendencies, insofar as there are such things, and nobody could accuse him of lacking systems.

27. I say "purported" because there seems to be ample evidence of creativity even among quite severely autistic individuals. Consider Derek Pavancini, the blind British pianist, who, while he has memorized "an enormous repertoire" of jazz songs, is also able to improvise on the spot (Baron-Cohen 2008, 104). There are more and more examples of autistic artists as well.

28. See discussion of the "windows task" and the Wisconsin Card Sorting Test, Boucher 2009, 171–74.

29. The terminology is Derek Parfit's, see Parfit 1995, 294.

30. Ibid., 295. Parfit gives the example of a statue which, while it is simply made of bronze, is not to be identified with the bronze, because you can melt the statue and still have the bronze but not the statue.

31. Boucher 2009, 139.

32. Mostofsky et al. 2009.

33. http://www.news-medical.net/news/2009/04/29/48951.aspx.

34. Mostofsky et al. 2009, 2421.

35. Byne and Lasco 1999, 116.

36. http://www.news-medical.net/news/2009/04/29/48951.aspx?page=2.

37. Ibid.

38. Kanner 1943, 47.

39. This has interesting parallels in the "science" of homosexuality. According to Byne and Lasco (1999), the vast majority of biological studies on homosexuality have presumed what they call the "intersex assumption," which is the claim that there are male and female brains plus the assumption that what it is to be gay is to have the "wrong sex brain." They point out several devastating problems for this view, most relevant of which for our current interests is the fact that not one of the (over one thousand) brain studies that have been done in the last century that have yielded published results has produced evidence to support the claim that there are distinct male and female archetypes of the human brain.

40. Baron-Cohen 2008, 62.

41. Baron-Cohen 2008, 74.

42. This point, along with several criticisms of the extreme male brain theory are made by Rachel Cohen-Rottenberg, who self-identifies as autistic, in http://www.journeyswithautism.com/2009/07/02/a-critique-of-the-extreme-male-brain-theory-of-autism/.

43. Baron-Cohen 2008, 74. One can only imagine the homosexuality-as-"central nervous pseudohermaphroditism" and autism-as-male-brain theorists arguing over whether or not they were looking at the brain of a lesbian, an autistic female, or a neurotypical male.

44. Piven et al. 1997, 185.

45. Boucher 2009, 118.

46. "We must, then, assume that these children have come into the world with innate inability to form the usual, biologically provided affective contact with other people, just as other children come into the world with innate physical or intellectual handicaps…here we seem to have pure-culture examples of inborn autistic disturbances of affective contact" (Kanner 1943, 50).

47. Folstein and Rutter 1977, 297.

48. Piven *et al.* 1997, 185. As evidence, the authors offer: "Wolff *et al.*… interviewed the parents of autistic children and the parents of nonautistic mentally retarded comparison subjects

and found that the parents of the autistic children were more often judged to lack emotional responsiveness and empathy, show impaired rapport with the examiner, and have histories of oversensitivity to experience, special interest patterns, and oddities of social communication… Gillberg, in a study of the parents of 23 children with Asperger syndrome, reported social deficits in 11 of the 23 fathers that were similar to, but milder than, those seen in Asperger syndrome." Ibid., 186.

49. Baron-Cohen 2008, 93.

50. Putnam 1973.

51. Carlson 1991, 25. See also Fausto-Sterling 2000, chapter 1. Patino agreed to fake an injury at the Kobe games so that the reason for her dismissal did not get out and cause embarrassment, and she was encouraged to keep training. However, four months later, at the first meet of the Spanish indoor season the head of the Spanish federation told her she would have to fake another injury—this time supposedly career-ending—or risk exposure in the media. She refused, won her race, and, as promised, she was exposed in the media, lost her boyfriend and many friends, and all of her records were stripped from the books.

52. Carlson 1991, 27. The International Amateur Athletic Federation (IAAF) finally conceded the point, and in 1992 Patino was allowed to rejoin the Spanish Olympic Squad (and the IOC finally abandoned sex testing in 1999).

53. It might be that "biological sex" is actually a socially constructed notion, just as I have argued that race is, and thus open to change by transgender activists. What constitutes one's sex could be located at the level of outward physical appearance. In that case, sex-reassignment surgery really would achieve a change of one's sex. Alternatively, if sex is psychological, then the point of such surgery would just be to give someone the body that matches what their sex already is. Finally, if sex is at the chromosomal level then sex-reassignment surgery does not achieve a change of sex, but merely enables one to "pass" even while naked. These are important legal matters, of course—see the case of *Littleton v. Prange* (1999).

54. Muhle, Trentacoste, and Rapin 2004, e482.

55. Paul Steinberg, "Asperger's History of Overdiagnosis," *New York Times*, January 31, 2012.

56. "A 1992 United States Department of Education directive contributed to the overdiagnosis of Asperger syndrome. It called for enhanced services for children diagnosed as being on the autism spectrum…The diagnosis of Asperger syndrome went through the roof…The downside to this diagnosis lies in evidence that children with social disabilities, diagnosed now with an autism-spectrum disorder like Asperger, have lower self-esteem and poorer social development when inappropriately placed in school environments with truly autistic children. In addition, many of us clinicians have seen young adults denied job opportunities, for example in the Peace Corps, when inappropriately given a diagnosis of Asperger syndrome instead of a social disability." Paul Steinberg, "Asperger's History of Overdiagnosis," *New York Times*, January 31, 2012.

57. For example, Tim Page, former music critic for the *Washington Post*; see "Parallel Play," *The New Yorker*, August 20, 2007, 36–41.