Smart People Believe Weird Things

Michael Shermer
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In April 1999, when I was on a lecture tour for my book Why People Believe Weird Things, the psychologist Robert Sternberg attended my presentation at Yale University. His response to the lecture was both enlightening and troubling. It is certainly entertaining to hear about other people’s weird beliefs, Sternberg reflected, because we are confident that we would never be so foolish. But why do smart people fall for such things? Sternberg’s challenge led to a second edition of my book, with a new chapter expounding on my answer to his question: Smart people believe weird things because they are skilled at defending beliefs they arrived at for nonsmart reasons.

Rarely do any of us sit down before a table of facts, weigh them pro and con, and choose the most logical and rational explanation, regardless of what we previously believed. Most of us, most of the time, come to our beliefs for a variety of reasons having little to do with empirical evidence and logical reasoning. Rather, such variables as genetic pre-disposition, parental predilection, sibling influence, peer pressure, educational experience and life impressions all shape the personality preferences that, in conjunction with numerous social and cultural influences, lead us to our beliefs. We then sort through the body of data and select those that most confirm what we already believe, and ignore or rationalize away those that do not.

This phenomenon, called the confirmation bias, helps to explain the findings published in the National Science Foundation’s biennial report (April 2002) on the state of science understanding: 30 percent of adult Americans believe that UFOs are space vehicles from other civilizations; 60 percent believe in ESP; 40 percent think astrology is scientific; 32 percent believe in lucky numbers; 70 percent accept magnetic therapy as scientific; and 88 percent accept alternative medicine.

Education by itself is no paranormal prophylactic. Although belief in ESP decreased from 65 percent among high school graduates to 60 percent among college graduates, and belief in magnetic therapy dropped from 71 percent among high school graduates to 55 percent among college graduates, that still leaves more than half fully endorsing such claims! And for embracing alternative medicine, the percentages actually increase, from 89 percent for high school grads to 92 percent for college grads.
EDITORIAL

Knowing Stuart

Stuart is a man in his late twenties who volunteers at Merrymeeting Center for Child Development, where I work. He is proficient in five languages (“if you count Latin, which is a dead language,” Stuart allows), and he’s a World War II history buff.

“In addition to the six million Jews Hitler murdered,” Stuart said, “he exterminated another four million “imperfect” beings of all kinds.”

“I think about that,” I said. “If Jason and Joshua were children in Hitler’s Germany, he would have “exterminated” my sons.” The thought, the injustice, turns my stomach.

“Hitler would have killed me,” Stuart said. Stuart has Asperger’s disorder. We shared a look in silence, contemplating unspeakable horrors.

Stuart is an amazing person. Although he’s struggled with compulsiveness and social cues his entire life, he was not diagnosed as having a disorder until he was 24 years old. I asked Stuart once why he volunteers at Merrymeeting Center. He shrugged, thought about it, and said, “I guess I’m trying to find out more about what I may have been like as a child.” Stuart confesses kinship to our most troubled children, those new to the Center who rave and tantrum and destroy the things around them. “I remember feeling that way sometimes,” he said.

One day I escorted a woman on a tour of the Center. The mother had a child who had been diagnosed as having autism nearly two years previously. The child engaged in severe aggression and tantrum behaviors, was not yet toilet trained, and had no language. The woman sighed, near tears. “I am sure my son will end

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ASAT MISSION STATEMENT

THE ASSOCIATION FOR SCIENCE IN AUTISM TREATMENT WILL:

- Disseminate accurate, scientifically sound information about autism and treatments for autism; and
- Improve access to effective, science-based treatments for all people with autism, regardless of age, severity of condition, income, or place of residence.

WE WILL FULFILL OUR MISSION BY:

- Educating professionals and the public about state-of-the-art, valid treatments for people with autism;
- Supporting certification, to ensure all individuals with autism receive treatment from practitioners who have met minimum standards of competency;
- Forming interactive, supportive partnerships with universities to develop accredited educational programs for autism practitioners, and
- Improving standards of care for people with autism.

VALUES STATEMENT

ASAT is committed to science as the most objective, time-tested and reliable approach to discerning between safe, effective autism treatments, and those that are harmful or ineffective. ASAT supports all scientifically sound research on the prevention, treatment and cure of autism, as well as all treatments for autism that are shown to be effective through solid scientific research, regardless of discipline or domain.
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up in an institution,” she said.

Stuart was entering data into a computer at the back of the room. Hearing this gloomy pronouncement, he lifted his head and smiled. “I’m somewhat autistic myself,” he said. He just left it at that, as if one could draw one’s own conclusions. I introduced Stuart as one of our volunteers. He stood up, shook the mother’s hand and chatted, without apparent self-consciousness, about himself—his skills, his achievements, his challenges, his dreams.

I was flabbergasted. As the mother of two children with autism myself, I marveled at the previous gift of himself Stuart made to this frightened mother intuitively, spontaneously, freely. And it dawned on me: This tall, red-bearded, exuberant man (who speaks five languages and helps special kids) reflects the alpha and omega of our lives; for while Stuart volunteers with our children for a glimpse into his past, he provides the families we serve with a vision of our future.

Thank you Stuart, from the bottom of my heart.

—Lora Perry, MS Editor

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We can glean a deeper cause of this problem in another statistic: 70 percent of Americans still do not understand the scientific process, defined in the study as comprehending probability, the experimental method and hypothesis testing. One solution is more and better science education, as indicated by the fact that 53 percent of Americans with a high level of science education (nine or more high school and college science/math courses) understand the scientific process, compared with 38 percent of those with a middle-level science education (six to eight such courses) and 17 percent with a low level (five or fewer courses).

The key here is teaching how science works, not just what science has discovered. We recently published an article in Skeptic (Vol 9, No 3) revealing the results of a study that found no correlation between science knowledge (facts about the world) and paranormal beliefs. The authors, W. Richard Walker, Steven J. Hackstra and Rodney J. Vogl, concluded: “Students that scored well on these [science knowledge] tests were no more or less skeptical of pseudoscientific claims than students that scored very poorly. Apparently, the stu-

Rarely do any of us sit down before a table of facts, weigh them pro and con, and choose the most logical and rational explanation. We think, but not how to think.

To attenuate these paranormal belief statistics, we need to teach that science is not a database of unconnected factoids, but a set of methods designed to describe and interpret phenomena, past or present, aimed at building a testable body of knowledge open to rejection or confirmation.

For those lacking a fundamental comprehension of how science works, the siren song of pseudoscience becomes too alluring to resist, no matter how smart you are.

**What Caused That?**

*John W. Jacobson, PhD, BCBA  
ASAT Board Member*

One of the questions heard often in the field of developmental disabilities today is, “What causes autism spectrum disorders (ASD)?”

Questions of cause are very difficult to address with full candor and confidence, not only in the case of autism spectrum disorders, but also in other conditions for which diagnosis is made based primarily on behavior. For example, some individuals speculate or assert that biological and/or toxic agents in the MMN vaccination cause autism. This article will focus upon that controversy to explore the complexities of cause and effect.

The scientific method is a process by which researchers seek to answer a variety of questions. Some of these questions do not involve causes, while others do. For example, neurological or neuroscientific studies may attempt to determine differences typically present in the structure or neurochemical features of the brains of children with ASD, compared to the brains of same-aged children without diagnosed disabilities. In other words, the question posed is not what causes autism, but rather what differences exist in autistic versus non-autistic brains.

On the other hand, some studies do attempt to identify causes, such as of a disability. To do so, certain tasks must already have been accomplished. For example, the disability must be well-defined. The decision that the disability is present and that the disability is not present must be accurate. The group that is defined as having the disability must be as homogeneous as possible. That means, for example, researchers should consider the question of what causes autism separately from the question of what causes PDD or Asperger syndrome. This is not to suggest that autism, PDD and Asperger's are not a spectrum as typically thought, but rather that the question “what causes autism?” is a much more specific question than “what causes autism spectrum disorders?” There is also the consideration that, because the diagnosis of autism and other ASDs is based on breadth and severity of effects, logically, differences in brain structure or other features should be more apparent for these children.

Assuming that we can accurately decide whether children have autism, and we are able to identify biological differences between these children accurately as well, we could then approach the question of cause in two ways. Taking into account possible multiple medical or neurological factors, we can conduct a detailed investigation into the backgrounds and developmental history of the children, identifying events that differ between diagnosed and non-diagnosed children. In this approach, we start with a group of children who are identified, and then look back on their histories. This is termed a retrospective study. Retrospective studies are important to conduct, but there are also challenges associated with them, including documenting that reported events occurred, reliance upon incomplete or differing clinical records, and the fact that universal screening for disabilities like autism usually does not exist, so only the children who happened to be identified and referred are included in the study. Children who are referred are likely to differ in several ways from those who are not referred, and some of these ways may be related to risk factors for autism.

The preferred method for conducting a study of causes is to use a prospective approach. Prospectively, one begins by using outreach to screen a population of children for a disability, to detect instances of the disability that might not otherwise be identified, as well as those who would have been identified. Then, background and history data can be collected on a group that is more likely to actually represent children with autism or any other condition. Historical information could include vaccinations and other medical events (e.g., exposure to general anesthesia, recurrent health conditions), as well as information about child development. More ideally, a prospective study would follow children from birth, and all of the information needed to consider causes could be collected as events occur. This would assure that the information is more complete, and in a standardized form. In the case of autism, even though it is now being identified more frequently, this approach is very difficult to carry out, for each child who may develop autism, there may be from 250 to 1,000 other children who need to be screened and followed. For this reason, some researchers may attempt to use prospective approaches to study causes of autism within larger studies that look at child development in large population groups, and consider a variety of disabilities. This often means that information that is specific to risks for ASD may not be fully collected.

Of course, there are other research designs that can shed light on causes of disabilities. Certainly genetic studies can indicate genetic factors that increase risk for a childhood-onset disability. Research with animals that involve brain surgery during early development that result in behavioral changes akin to those typical of a disability may also be suggestive. Basic research at the level of neurons and the effects of toxic substances and side-effects of medications may also be suggestive. But ... there is no substitute for actually studying the occurrence of a condition among children prospectively.
Why do we need scientific studies to indicate what the causes of a condition like autism might be? Why isn’t it enough that some research might identify some differences between children with autism and their peers? First, some differences that are identified initially do not necessarily differentiate children with autism from those who are accurately diagnosed as not having autism. For example, research findings have suggested the unexpected presence of measles virus in the gastrointestinal tracks of children with autism, but subsequently at least one report has found this for children without autism as well. This does not mean, in and of itself, that the initial gastrointestinal findings are not possibly suggestive, but does point out the need for careful assessment of the likelihood that particular factors are plausible risk factors. In this case, scientific research needs to address why, if gastrointestinal measles is a risk factor or cause, or reflects a risk factor, some children are affected, and others are not.

But, if many people develop a consensus that a given event—vaccination, for example—is regularly observed to occur shortly prior to detection of autism, is this not sufficient to warrant research on this issue? The short answer is yes—whether observed by parents, clinicians, educators, or researchers, events that may be plausible causes or risk factors for a disability should reasonably be studied. Parents or others in the lives of children with disabilities may certainly detect events that are not apparent or considered by clinicians or researchers. But the fact that a belief is widely held is not, in itself, evidence that the belief is valid or accurate.

The brains of human beings are structured and function in ways that are the joint product of evolution and experience. One of the well-known biases associated with human perception and thinking is the tendency to conclude that there is a cause and effect relationship between two events, when it can be shown through precise research that this is not the case. Carl Sagan, in his 1997

应用科学于教育

Michele Kule-Korgood, Esq.
ASAT Board Member

在另一大步骤中，确保教育质量，国会通过了《无学习者落伍法》。我们的目标是在医疗治疗中，我们作为一个国家的远景，在教育中。直到最近，教师和学校已不受限制地在方法中教我们的年轻。是它可能时，在一个世纪的宣布，我们将最终将科学方法的承诺以教在教学方法。

有趣的是，我们国家已经发展与更专注于科学，提升它几乎到的三重信条—R’s”，阅读，写作，和算术。在我们的科学课程，从生物学到化学，我们教我们的孩子，可能以检测的真如从猜是通过严格的应用科学方法。但是，当它来决定如何教阅读到的尺寸，或教孩子们与自闭症，基本的建筑块知识，科学方法的教学不参与在选择的多数学校地区。但可能很快。

《无学习者落伍法》注重联邦资金对项目和战略，他们被科学研究。然而，这并不是第一次，国会曾经对科学在选择教学方法。为了，我们必须回头去到的联邦特殊教育法律，个体与残疾人教育法。

在1975年，国会通过了具有历史立法责任到广泛的排斥儿童与残疾从教育。《关于在新年、仓促和证明条件在纽约的Willowbrook机构和集体诉讼的国家对所有手}
Bobby Newman, PhD, BCBA

Dear ASAT: My four year old child is diagnosed with PDD. A recent evaluator recently said that my son was “retarded in addition to being autistic.” What is the relationship between autism spectrum disorders and mental retardation? Is my son also retarded?

This can be a very touchy subject, and the answer you get can vary by the professional you ask. Please understand that my answer reflects my own bias and understanding of the clinical literature on this issue, and I would need a great deal of information regarding your child to give you an answer that was specific to your child.

That disclaimer out of the way, in a nutshell, my opinion is that it is entirely possible that your child may be functioning in the mentally retarded range, without being truly retarded. To expand on what I mean, consider the criteria for the diagnosis of mental retardation. According to the DSM-IV TR, there are three criteria for mental retardation:

1. IQ measured to be two standard deviations or more below the mean.
2. Significant adaptive living skill deficits.
3. Onset before age 18 (in other words, before development is considered to be completed).

Does your child meet these three criteria? A great many students diagnosed with autism spectrum disorders (ASD) do. My central question, however, is whether this measured intellectual deficit is merely a reflection of current behavioral, communicative, and social difficulties. Consider the requirements of standard intelligence tests: They generally require the student to interact with the tester, answer questions, follow directions, imitate, and receptively or expressively identify requested items. Many students diagnosed with autism have simply not learned these skills at the time of testing. Following effective programming, IQ may jump by dozens of points (eg, Lovaas, 1987). Did you magically increase the student’s intelligence, or did you help the individual to develop the skills that allowed the child to participate in the test? I would argue for the latter explanation. Just what is intelligence anyway? Is it some general factor, or a collection of specific factors? A person whose name escapes me once said that intelligence is what intelligence tests measure.

Consider another issue: I do some work in Eire, Ireland, and was in the Dublin airport after one of the consulting trips with my friend and colleague Meredith Needelman (a particularly wonderful speech therapist). She was reading a story to me from a magazine about some movie star. At one point, Meredith realized I was looking at her blankly, smiled and said “you have no idea who I’m talking about, do you?” I’m afraid I didn’t. I couldn’t identify 98% of the celebrities out there if you put a gun to my head. It’s just not my area of interest. Call me a cultural illiterate.

Why do I mention this? Consider that many students diagnosed with ASD are not exposed to many life experiences that provide the knowledge necessary to answer questions on the IQ tests. I’m thinking of a student I tested a few years ago. When I first met the student, he was not toilet trained, could not speak, and only consumed Pediasure for nutrition. After a year of very serious effort by staff and family, all of these deficits were ameliorated, and he was able to participate in an IQ test that included a verbal picture identification component. The child labeled a great many items, but missed others I thought he would know. When I went over the results with his mother, I asked if her son had ever seen one of the farm animals on the test. Mom looked at me like I had eight heads, and asked if, considering his former deficits, I thought they were taking him to petting zoos that frequently. The answer was fair enough, but conditions were different now. So I called a bunch of people into the room who were a lot smarter than me (his teacher, speech therapist, and teacher assistants) and we planned a year’s worth of field trips that the family and school would take each week to expose him to missed experiences. We went over a bridge and through a tunnel, to the zoo and an aquarium, to the baseball game, to the ocean, to the forest, and more. I should also mention that on that same intelligence measure, the child scored three standard deviations above the mean on one of the other subtests that did not directly express language.

To consider the question as regards your son, we need more information. What sort of test was used? Did it have verbal and nonverbal components? Was there a big spread among the subtests? Was it a test appropriate for someone of his age? Was the test ever standardized for people with disabilities? Was the test conducted properly? Was the test conducted by someone with whom the student was familiar, and in a familiar setting, or was the test done in such a way that the student was not comfortable or motivated?

Was there a measure of adaptive behavior collected? These are often assessments that do not directly test the student, but rather interview significant others to compare the child’s behavior to age-standardized norms. If so, was there a spread among subscales? A large spread, particularly with some subscales in or around the normal range, would argue against a mental retardation label. Finally, was the adaptive behavior test done properly? I kid you not, I have heard of such tests being conducted by mailing the questionnaire to parents, a completely inappropriate use of such measures.

I don’t want to be glib, but I think the best course of action at present is to

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act as though the mental retardation diagnosis is simply an artifact of continuing language, interactive, or other skill deficits (as opposed to some inherent and global intellectual delay). Do not speak “around” your child, as though he was not in the room. He may be understanding a great deal more than you realize. Don’t think he is too delayed for you to attempt to teach particular skills. Make sure you teach necessary prerequisite skills, and then go for it. Students surprise us each day.

The late, great Stephen Jay Gould, an annoyingly brilliant individual, once published a book entitled *The Mismeasure of Man*. The book describes some of the historical problems with IQ tests and measures of intelligence in general. It’s useful background when considering this question.

**What Caused That**

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book, *The Demon-Haunted World: Science as a Candle in the Dark*, referred to such tendencies as “irreducible human error.” To err in this manner is human, but to insist that reliance on mere consensus is sufficient to accurately identify causes of events, such as the occurrence of autism, is folly. Errors of this type may be even more likely when the identification of a chain of cause and effect is especially important to the person making a judgment about cause and effect; many parents of children with autism believe that identifying the causes of autism, for their child and other children, is important. This may increase the chances that some or many may conclude that certain, unproven events are causes of the disorder, without solid evidence. However, this is a very human thing to do, and clinicians and researchers are prone to do this as well.

The critical distinction that needs to be made is between correlation and causation. Correlation means that two events tend to occur together. When one does not occur, the other tends not to occur as well (called a positive correlation); or that when one occurs, the other tends not to occur (called a negative correlation). Sometimes correlations, like cause and effect, are perceived accurately, and sometimes they are not. But while necessary for showing cause and effect, correlation does not prove cause and effect. Sometimes correlation might be presumed, because of cultural factors; for example, autism is often diagnosed, by definition, at ages when children are subject to frequent vaccinations. Thus vaccinations and autism could be hypothetically correlated, despite the fact that there is no present scientific evidence that this is the case. Correlation does not in itself show causation, because the fact that two events occur together may be influenced, or caused, by a third factor that has been ignored, or that was not studied.

Causation, on the other hand, requires a higher standard of proof than the fact that two events occur together (that is, have a positive correlation). Proving causation, or that an event is a risk factor for a disability, requires that several conditions be met: (1) the purported cause has to consistently or always occur before the purported effect; (2) when the purported cause occurs, the effect regularly occurs; and (3) when the purported cause does not occur, the effect tends not to occur, is less likely to occur than it does generally, or does not occur at all. Other criteria associated with the strengths of prospective studies also need to be met; for example, that the group of people studied is representative of the larger group of people with the condition (in this case, all children with autism or all children with ASD). This can be done by including all children in a general population with the condition, or by randomly sampling the children with the condition. But, if sampling is used, there also must be a sufficient number of children to generalize to the larger group of children, and the required number to do so increases as the complexity and range of issues under study increases.

Where do we stand today in understanding the causes of autism? It is fair to say that researchers are developing a more complete understanding of the neurological factors associated with autism, but some degree of modesty is also appropriate with respect to the predictions that can be made or confidence with which particular neurological findings can be said to characterize autism. Many neuroscientific studies focus on specific aspects of the brain. Therefore, different aspects of the brain have been studied in different samples; there is seldom concrete evidence that these samples are very much alike, or that they represent a larger group of children with autism. This points out the need for independent researchers to conduct studies with other samples, to verify that the findings with one sample also apply to others.

In addition, many neuroscientific studies include small numbers of subjects. As a result, such studies are not able to detect relatively subtle but consistent differences that may exist between individuals who have autism and those who do not, and the studies may not be representative of children with autism more generally. Advances in research design, including identification of subjects with better measures, are addressing these limitations. Neuroscientific knowledge about autism is steadily advancing, but there are, nonetheless, considerations that affect the strength of the conclusions that can be drawn today.

One must also consider that the group of children diagnosed with autism is heterogeneous: some also have diagnoses of mental retardation, while others don’t; some have seizure disorders, while others don’t; some manifested regression or loss of attained skills, while others did not. Although there is a strong (and warranted) presumption that genetic factors play a strong role in the occurrence of autism, the heterogeneity of children with the condition and current research findings suggest that the relevant genetic factors are complex and multiple in nature. At this point one may reasonably argue that the behavioral condition of autism and ASD are final common pathways, or results, of differing genetic factors—that there is no single genetic factor that accounts for occurrence of the condition. Events prior to birth have also been implicated by neuroscientific studies. It may also be that in some cases, environmental events, such as reactions to toxins, may play a role. It may be that all of these factors, and others, are involved as risks or causes.

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Science in Autism Treatment

*Spring 2003*

ASAT

Providing Accurate, Science-Based Information • Promoting Access To Effective Treatment
Thank you for reading Science in Autism Treatment, and for making it a successful publication. Since the first issue in 1999, our goal has been to share meaningful and useful information on autism research, treatment options and education. With each issue, ASAT reaches tens of thousands of people throughout the United States and around the world. We are excited that the rate of new subscribers continues to grow substantially, which demonstrates that more parents and professionals have access to science-based information about autism.

Because of the continued growth of subscribers, we need to begin collecting $15 per year to offset the publication, printing and mailing costs of Science in Autism Treatment. To continue your subscription, please make your check out to ASAT, note “newsletter” in the memo line, and mail it to ASAT, PO Box 7468, Portland, ME 04112. A portion of the $15 may be tax deductible and you will receive a receipt for tax purposes. If you are unable to pay the $15 fee, please contact Betsy Welch at the ASAT office, who will handle your request for continued service confidentially.

Again, thank you for your role in the growth of Science in Autism Treatment, and for your support of ASAT.

Warm regards,
Lora Perry
Editor, Science in Autism Treatment
Treasurer, ASAT Board of Directors

Order conference tapes
Review past issues of the newsletter
Solid, science-based information
Suggested reading list

ASAT
ASSOCIATION FOR SCIENCE IN AUTISM TREATMENT
YOU CAN FIND US ON THE WEB
www.ASATonline.org

Association for Science in Autism Treatment
P.O. Box 7468
Portland, Maine 04101