

The Sparseness Adaptation Syndrome, Part I
Adaptation to low population density yields a set of traits
correlated with autism and male gender.

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Abstract

Adaptation to low food and population density at inevitable frontiers yields a syndrome that plausibly accounts for major features of autism, including its defining social disconnectedness, correlation with male gender, spectrum of intensities, variety, dependence on multiple genes, rising prominence, and other features. The theory proceeds from thought experiments and simple reasoning and makes testable predictions about measurable quantities.

Introduction

Autism is a neurological condition that has long defied tidy explanation. Here is a partial listing of recently proposed causes of autism:

Vaccines, mercury, environmental toxins generally, Internet use, cold mother, older father, older mother, depressed or stressed mother, prematurity, low birth weight, high birth weight, having a big head, being a twin, smoking during pregnancy, diabetes during pregnancy, birth complications, being born in summer, being a second-born child, being a later-born child, being born close together, in vitro fertilization, childhood immune system malfunction, maternal immune system malfunction, Neanderthal genes, hyperinsulinemia and diabetes, impaired brain connections, impaired sugar metabolism, Clomid, Terbutaline, glycine, mitochondrial dysfunction, malfunctioning epigenetics, living near a freeway, too little oxytocin, too much androgen, parental wealth, Lyme disease, maternal antidepressant use, etc.

A simple Internet search easily verifies and extends this list.

Most of the proposed causes adopt a fairly narrow view of autism's origin. In what follows we see that taking a step backward to encompass a larger view resolves major questions about autism with a single hypothesis: Autism, at least in its less severe forms, is one result of adaptation to low population density at topologically inevitable and unrelenting sparse frontiers. The sparseness adaptation hypothesis does not account for all of autism, but it may account for much of it. The hypothesis forms a natural null hypothesis against which others can be tested.

The sparseness adaptation theory of autism makes testable predictions about measurable quantities: If those predictions are not born out then whatever confounds them is important in the history of brains.

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Autism

What is autism? So many varied traits are associated with this neurological condition that it is easy to become confused about what exactly defines it. Here is a list of some traits commonly associated with autism:

Poor eye contact, monotone speech, odd gait, preoccupation with objects, preoccupation with parts of objects, echolalia, odd posture, clumsiness, preternatural physical skill, lack of empathy, extreme sensitivity to small sounds, extreme sensitivity to bright or flickering lights, not liking to be touched, lack of a theory of others' minds, lack of speech, precocious speech, talking like a little professor, social ineptitude, unusual ability to perform arithmetic or calendric computations, inappropriate interpersonal distance, liking of strict routine, blindness to social convention, inappropriate voice volume, rocking, head-banging, wrist-flapping, foot-tapping, intestinal disturbances, having frequent tantrums, lining up toys, etc.

Simple Internet search verifies and extends this list as well.

To see what does define autism we return to the original uses of the term and to the question of why the name *autism* was adopted in the first place.

Once autism had no name. It is useful to remember this when crafting a theory of autism because it underscores the obviousness of autism's central defining feature: *social disconnectedness*. The name *autism* derives from the Greek word *auto* for self, and proclaims the apparent mental involution or self-absorption of autistic people. Historically the word autism was invoked independently three times (by E. Bleuler,³ L. Kanner,⁸ and H. Asperger¹ respectively) to name clinical syndromes. In all three cases it was the startling social disconnectedness of the people described that prompted use of the term autism: The three founders clearly saw other features of autism as secondary to social disconnectedness.

When we speak of well-developed social ability usually we mean an ability to connect socially with people regardless of who exactly they are: It is a *generic* ability. **Autism is first defined by a relative absence of generic social ability**, and it is this absence that appears as social disconnectedness and apparent self-containment.

Because social disconnectedness is the central, eponymous feature of autism it is the primary feature for which a theory of autism must account. A good theory of autism may also account for some secondary features. Indeed, some subsidiary facts of autism beg for explanation. As I write it is common to hear autism referred to as a mystery, and one surrounded by other mysteries, for example:

Why does autism —

- exist in the first place?
- affect more males than females? ^{1, 16}
- occur in so many varieties? ¹⁷
- have a spectrum of intensities? ¹⁷
- depend on so many different genes? ⁷

Why are there autistic savants? ¹⁴

Why is autism becoming more prominent? ¹⁸

We shall see that the present reasoning addresses all of these questions and demystifies the origin of social disconnectedness in autism.

The Argument

The basic argument of the sparseness adaptation theory of autism is simple: Frontiers that are sparse by topological necessity constantly destroy socially adaptive genes, thus feeding social-gene-depleted genomes into the larger population and fostering autistic traits in the general population. As it stands this makes a pretty story, but there are many pretty stories. Rudyard Kipling's fanciful accounts of evolution in his renowned *Just So Stories* aptly demonstrate this.⁹ Prettiness is no guarantee of validity: We want something solidier than a *Just So* story.

A comment by Albert Einstein is famously paraphrased as, "An explanation should be as simple as possible, but no simpler."⁴ I intend the following propositions to be almost incontrovertible, yet to be simple without being simplistic:

1. Brain parts need food to build, maintain, and use.
2. Some social abilities depend on particular brain parts that depend on particular genes.
3. Where food is sparse brain parts promote either food gathering or death.
4. Both food and population are sparse at topologically inevitable frontiers.
5. The probability that social ability promotes food gathering declines with increasing sparseness of food and population.

It follows that topologically inevitable frontiers constantly destroy genes conferring social ability, thus feeding social-gene-depleted genomes into more populous areas and fostering autistic traits in the general population.

The Argument Examined

Let's look at the propositions more closely.

1. Brain parts need food to build, maintain, and use. Without food no babies are born and no brains arise in the first place. Also, things fall apart and brain parts are no exception: Without food to maintain them brain parts decay like the rest of the body. Finally, per unit weight brains use ten times as much food energy as does the rest of the body. At every level brains and their parts — and indeed all parts of an organism — depend intimately on food for building blocks and energy.

2. Some social abilities depend on particular brain parts that depend on particular genes. People who have strokes often become abruptly acquainted with the previously unnoticed complexity of tasks like walking, speaking, and social relationship. All of these abilities depend on particular brain structures that in turn are shaped by particular genes. Stroke or other harm to particular brain parts can damage some social abilities while leaving other abilities unaffected, showing that the social abilities depend on particular brain parts.⁵ Studies of identical twins separated at birth confirm that major features of personality are

strongly shaped by genes.¹³ Genes do not fully determine who we are. However, the present reasoning requires only that *some* genes are necessary to brain structures that are in turn necessary to *some* social behaviors.

3. *In sparse areas brain parts promote either food gathering or death.* Where food is scarce brains must find food quickly or die. Given that all organism parts need food to build, maintain and use, any organism part that does not directly or indirectly promote the arrival of food acts as a drain on stored food reserves, and so hastens death. This applies to brain parts as it does to any other organism part.

4. *Both food and population are sparse at topologically inevitable frontiers.* This is the linchpin of the sparseness adaptation theory. From the simple fact that food and populations do not exist everywhere it follows that there exist frontiers that are sparse in both food and population. (If there is a crowd in one place and empty space in another then somewhere in between is a frontier where population is relatively sparse.) This is like having an x-ray into all past history. Without having been present we know that human ancestor populations were constantly exposed to the effects of sparse surrounding frontiers.

5. *The probability that social ability promotes food gathering declines with increasing sparseness of food and population.* We consider sparseness of food first. The harsh realities and social effects of scarce food at an extreme frontier are made clearer by a simple thought experiment. (This is a *thought* experiment. Do not perform it on living creatures.)

The Sparse Box

Suppose that a hypothetical creature can survive on one food pellet a day, but not on less food. The creature lives in an impervious box and is fed its requisite one pellet per day so that all is well. Now suppose a second creature with identical needs is also put into the box. What happens? There is not food enough in the box for both of its inhabitants to survive. Only two outcomes are possible: Either one creature dies or both die. The only way life can persist in the sparse environment of the box is if one of the creatures prevents the other from eating. The one can do this in two ways: by grabbing food pellets more quickly than does the other or by slaying the other creature outright. Either way one creature kills the other. In the *Sparse Box* at least one creature *must* die, and generosity and camaraderie are exactly suicide.



Mobility is an essential trait in sparse regions because of the need to gather widely scattered food. Nevertheless human ancestors can move only so far in a given time and hence a sufficiently large sparse area is like a box to them: Once any carried food runs

out they must find food within a short distance or perish. The limited range acts like the walls of a box and the inhabitants are forced into deadly competition for the few resources there. Where there is food enough for one but not two no strategy can reliably transmit cooperative genes, but the genes of the deadliest competitor endure.

The deadly competitor who survives at an extreme frontier lacks not only *generic* social ability, but almost any aptitude for social connection whatsoever. By any definition **the extreme outlier is socially disconnected — and functionally autistic** in the sense of the early eponymous definition. Just how thoroughgoing and persistent this autism is becomes clear in the full light of the five propositions taken together. We continue with the fifth, examining the further effects of food scarcity.

Tribal Autism

It is easy to see that connective social ability is a liability in an extremely sparse area. One wonders though about life immediately *within* the desolate frontier. After all, at some population density cooperative behavior becomes enough of a norm that it can sustain crowds. Revisiting the *Sparse Box* addresses the question.

Suppose that *two* creatures live in an impervious box and are fed the requisite two pellets per day so that again all is well. When a third creature is introduced with no additional food, however, once again at least one must die.



Obviously the original two treat a newcomer as a potential teammate at the risk of their own lives. *Any* group living at subsistence level near a frontier, and that hopes to survive intact with its genes, must treat newcomers as deadly rivals for food. Where only n can survive the $n+1$ st is a mortal foe. Thus even near an extreme frontier genes fostering generic sociability are a liability to a group living at subsistence level.

The ability to relate cooperatively to people outside of one's own small group is a social ability, and this ability is a useless *liability* in the near-frontier. Because near-frontier tribes are mutually exclusive, at the inter-tribal level they are functionally autistic. I call this form of social disconnection **tribal autism**. Tribal autism is expected in areas near frontiers just as individual autism is expected at the extreme frontier itself.

Population sparseness compounds the effects of food sparseness. In the absence of modern technology most social abilities depend on nearby neighbors to be of any use. An ingratiating manner and easy way with words won't feed anyone marooned like Alexander Selkirk (the model for *Robinson Crusoe*).¹¹ At the extreme of population

sparseness social ability and any brain structures dedicated to it bring no food. This is not a matter of there being too little food: It's a matter of there being no one around to cooperate *with*, and so having to survive alone where social abilities (with underlying brain parts and genes) are simply a drain on resources.

The food-gathering value of most social abilities declines with interpersonal distance because two creatures must be within hailing distance of each other to form, and in many cases to execute, a joint food-gathering plan. The odds of a social plan succeeding decline with distance to neighbor. Wherever creatures near a frontier don't simply huddle in a group social abilities that depend on a nearby neighbor — which is to say most social abilities — become useless.

Population sparseness also compounds the tribal autism expected from the *Sparse Box* thought experiment. Near a frontier there are by definition few groups of any kind so the probability that *inter-group* social ability promotes food procurement also declines with increasing sparseness.

The net effect of the two *Sparse Box* thought experiments is that social disconnection between individuals is a prerequisite of survival at an extreme frontier, and social disconnection between *groups* is a prerequisite *near* frontiers. Allowing that small bands near frontiers likely contain genes from extreme outliers (mating once being the sole social requirement for propagation of outlier genes) it is easy to see that **sparse frontiers and their environs are a veritable forge of autistic traits**. It does not take many generations to concentrate these traits: Outliers either have them or they are dead.

Surviving frontier organisms occasionally transmit their sparseness-adapted genes to organisms well within the frontier, and in this way asocial genes make their way into crowded areas. There the axe does not fall on the genes immediately because food is more plentiful and competition less fierce. The social disconnection expected from the inward-moving sparseness-adapted genes is the defining characteristic of autism and this inward diffusion can account for autistic traits even where cooperative behavior is common.

Statistics and Probabilities

I have caricatured frontier life somewhat for the sake of clarity. The reality is that individuals and groups move and mix as they respond to changing conditions. However, this does not greatly alter the expected genetic effects, as we now see:

A genome reflects a statistical composite of its environmental history. The more time a type of organism spends in a particular environment the more genes ill-suited to that environment are culled from its genome. Odds are that an ancestor in a sparse area will spend more time alone in sparse conditions than another in a lush area, *regardless of whether the ancestor also spends time in a band or tribe*. The minute a social gene moves into an area where it does not contribute to its own self-sustenance the axe of probability begins to fall on that gene: It is draining the host's resources and increasing the likelihood of the host's and its own demise. So it is with genes at every scale and degree of food or population sparseness, and particularly where a step away from a group is a step into desolate isolation.

At some distance from an extreme frontier social bonds become inevitable. However, working bonds in a band very near a frontier are likely to be socially rudimentary, ambivalent and not at all generic — socially rudimentary because in very sparse areas there is little time for anything but interaction directed to objective survival; ambivalent because the slightest dip in food levels turns a teammate into a deadly competitor; and not at all generic because per the *Sparse Box* outsiders remain deadly.

Well-fed city dwellers, like many readers of this essay, often have little feel for the actual demands of frontier survival. A weekend in the woods with a few friends and a backpack full of food is not the same as frontier survival. The social ability needed near a sparse frontier is probably more like that of a soldier under fire than that of a gracious host.

Conclusions

The resounding conclusion of the forgoing reasoning is that both at and near frontiers genes favoring generic connective social ability are a drain on resources and an invitation to death at the hands of competitors. The fourth proposition gives force to this conclusion: Ancient populations could not escape sparse frontiers. Without having been present we know that human ancestors were exposed to the unrelenting effects of topologically inevitable frontiers. That is why the sparseness adaptations are expected to be genetic and not only learned. **It is also the practical certainty of the fact that sets the sparseness adaptation theory of autism apart from pretty *Just So* stories.**

The five propositions together form an implacable engine: Social abilities depend in part on brain parts; brain parts depend in part on genes; and the lot depends on food. At inevitable sparse frontiers generic social abilities do not bring in food, thus lethally draining resources, and they interfere with fierce competition, so effectively courting suicide. Therefore both at and near frontiers brains with diminished social ability thrive. Without this logic the proposed origin of autism is another *Just So* story. With the logic, though, one is forced to deny firm propositions in order to deny the conclusion that sparseness adaptation bred autistic traits into the human ancestral population.

The Sparseness Adaptation Syndrome

A syndrome is a set of correlated traits. The listed propositions and *Sparse Box* thought experiments show that adaptation to frontier sparseness produces a set of correlated traits. Compared to brains with long histories in lush and crowded areas, sparseness-adapted brains are:

1. Socially disconnected
2. Competitive, and
3. Mobile.

These are basic traits of the **Sparseness Adaptation Syndrome**. Further correlated traits can extend the list.

One of the great attractions of the sparseness adaptation theory is that it neatly addresses the major questions about autism given earlier.

Addressing the Mysteries

Why does autism exist in the first place? We have noted that the primary defining characteristic of autism is social disconnectedness. The sparseness-adapted outlier is by heritable necessity more socially disconnected, i.e., less a generic social bonder, than a cousin from a lush and crowded place. **Generic sociability declines with increasing sparseness, and the extreme absence of generic sociability is autism.**

If social ability depends on brain structures then any damage to those structures can lessen social ability. As a result social disconnectedness, and autism, can have many causes. However, few if any other causes are as unrelenting as the sparseness of population surfaces and voids, or cause atrophy *specifically of social structures* while sparing others. When social disconnectedness is part of a general cognitive impairment it is probably more descriptive to call the condition a general cognitive impairment than to call it autism.

Autism that is adaptive is rarely noticed, precisely because it *is* adaptive: It takes a mismatch between brain and environment to bring autism to notice. However, autistic genes diffusing from the population frontier can combine in ways that are poorly adapted in any circumstance. This may account for some of the more noticeable forms of autism.

Why does autism have a spectrum of intensities? This is because population and food resources exist in a spectrum of densities ranging from sparse to lush and crowded. There are genes and gene combinations well adapted to every zone in the density spectrum.

Why does autism occur in so many varieties? The constraint that produces autism is global: It does not specify exactly *how* connective social ability will atrophy — only *that* it will atrophy. Social ability is complex and it can atrophy in many ways. It follows that autism will occur in many varieties. This is important because it shows that a search for an "autism gene" — or even only a few such genes — almost certainly is futile. Furthermore, **because of its spectrum and variety autism will always defy clear diagnosis.**

Why does autism depend on so many different genes? This is because social ability depends on so many different genes: It is the varied loss of these genes that manifests as autism.

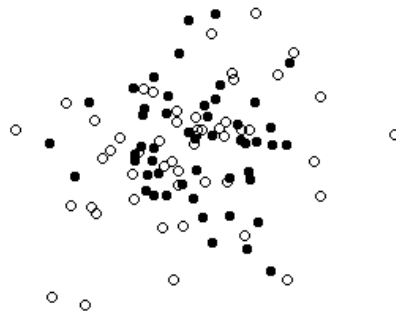
Why are there autistic savants? In sparseness an organism has little use for generic social ability, but it has plenty of use for ability with objects and edible or dangerous organisms. Most autistic savant abilities have little to do with social bonding but some have potential value in a world of objects and alien organisms. It is likely that with increasing sparseness some brain social structures become "re-purposed" to more object-related uses, similar to what evidently happened when the legs of ancient hippopotami found new use as the fins of whales.^{3,5} From the point of view of a hippopotamus a whale is a disabled hippopotamus but a swimming savant.

Savant abilities suggest a second and likely important origin of autism: Brains may, like computers, belong to a class of systems whose capacities are limited by finite bandwidth and storage capacity. If so then at a frontier social ability is like a computer program that steals memory and processor speed, thereby degrading brain performance on other tasks. In other words great social ability in a sparse area not only drains stored food reserves, it may also drain brain *computational* resources needed for non-social purposes like tool-building and game tracking. This would compound the destruction of social genes in sparse areas and also favor genes that re-purpose social brain structures to non-social uses.

Why is autism more prevalent in males than in females? To address this question consider the following thought experiment:

Suppose 100 identical things are dumped in a tight pile on a landscape. Suppose also that all of the things are identically inclined to move about randomly like tiny staggering drunkards. What happens? They all stagger about bumping into obstacles and each other, and the heap slowly spreads outward on the land. By sheer randomness some stagger to the margins and some stagger about near the starting point, while others travel outward and back again, and so on.

Now suppose, however, that starting at the outset half of the initial 100 things are periodically weighted as in a handicap horse race so that they cannot travel as fast. What happens then? What happens is that most of the things in the sparsest regions are the *unburdened* things, because they travel faster and so tend to move about and outward faster. The overall average distance-to-neighbor for the unburdened group will be larger than that for the burdened. As a result **the unburdened group must be on average more sparseness-adapted, hence more autistic, than the burdened group.**



In this simulation some of the burdened (solid-colored) entities are present at the frontier, but they are a minority there.

Half of the human ancestral line has been periodically burdened. We call the delightful burdens *babies* and they have encumbered countless generations of females during pregnancy and nursing. The physical origins of this arrangement are beyond the present scope. It suffices that in the human line **males have collectively spent far more time at greater distance from neighbors than have females** and so carry more sparseness-adaptive genes than do females. **This makes autism more prevalent in males than in females.** This brings us to the final listed mystery.

Why is autism becoming more prominent? Two explosions have occurred on Earth in the past ten thousand years: The explosion of human technology, and the resulting explosion of human population. Where each human stood several millennia ago there now stand hundreds.¹⁰ There may be many reasons that autism is becoming more prominent, but one of them is that sparseness adaptations that accumulated over many thousands and even millions of years now have far less sparseness in which to be adapted, and adaptations with a long history are slow to disappear. As mentioned earlier, it takes a mismatch between brain and environment to bring autism to notice. **Autism is becoming more prominent because there is less sparseness to which the brain structures of autism are adapted, and burgeoning crowds expose the social limits of autistic people.** This explanation is doubtless incomplete but perhaps germane.

The specific implications of the sparseness adaptation theory of autism suggest a further question:

Given that a tendency to kill is sparseness-adaptive why is violence not a more prominent feature of classical autism? This is an excellent question, and several answers suggest themselves. One possibility is that autistic violence is heavily driven by actual food sparseness, so that it is not seen in well-fed populations. A second possibility is that the very sparseness that selects for killing also selects for killing by means that use little food energy — in other words indirect forms of violence. Beating rivals to food in a sparse box is a simple example of this and behaviors like it do not necessarily appear violent. A third possibility is that violent forms of autism do exist but are artificially separated from the less violent forms, like separating people who are tall and plump from those who are merely tall even though both have similar foundational structures (skeletons).

A similar question could be asked about the absence of great competitiveness from the classical picture of autism, with similar possible answers. A fourth possible answer, however, is that **sparseness adaptation defines a fourth class of autism overlapping but distinct from the classes defined by Bleuler, Kanner and Asperger:** The expected traits of the Sparseness Adaptation Syndrome — including social disconnectedness, fierce or deadly competitiveness, and the ability and tendency to move about — define **Sparseness-Adaptive Autism.**

Whether or not it defines a new class of autism it is highly unusual for a single simple hypothesis to squarely address so many basic questions about the condition, and this suggests that existing classes of autism are at least strongly shaped by sparseness adaptation. The sparseness adaptation hypothesis forms a natural null hypothesis against which others can be tested.

A major attraction of the sparseness adaptation hypothesis is that it yields the testable predictions essential to rigorous science. The following section is necessarily more technical in order to confirm that the present theory is more than a *Just So* story.

The Hard Science of Prediction

A rigorous scientific theory makes testable predictions about measurable quantities. The present sparseness adaptation theory of autism has its roots in measurable quantities like distance to neighbor, organism and population persistence times, population densities, genetic composition and details of environment and brain structure. These quantities can be measured in different ways, but overall the theory makes the following predictions:

- **Autism-associated genes are more prevalent in lineages with long histories in sparse areas.**
- Autism is more prevalent in males: The observed gender difference is not a bias artifact.
- Autistic people and men spend more time at greater distance from neighbors than is average.
- Autism is pervasively polygenic.
- Autism correlates with distinct brain structures, but these exist in great variety.
- Brain scans will show that brain parts activated by social tasks in most neurotypical people are activated by non-social tasks in some autistic people.
- Maladaptive autism often results from or is compounded by a *concentration* of relatively common sparseness-adaptive genes.
- Language in sparse areas differs radically from that in lush areas and disproportionately concerns objects, techniques and strategies of rapid food acquisition, and subduing or escaping competitors. Sparseness also favors language that conserves time and energy.
- Autism correlates with an ability to hunt animals and survive alone in wilderness (this likely seen more clearly in relatives of very autistic people).
- Autism is a feature of brain history in all extraterrestrial biospheres.

There are many other predictions and the ones given can expand into more technically detailed predictions. I give further predictions in later parts of this essay. The lineage history predictions will become testable as gene sequencing becomes more common. Significantly, gene sequencing should make it possible to construct accurate histories of gene flows and mixing in populations, thereby establishing how strongly stirring effects influenced sparseness adaptation. Random proximity and communication measurements — e.g. checking how far autistic and non-autistic people (or men and women) are from neighbors at random moments — can begin to test the behavioral predictions. Some predicted correlations will be more clearly observable in *relatives* of very autistic people because extreme autism can be disabling in ways that lead to confounding compensations imposed by neighbors.

As complex as life is, it is much harder to fashion strongly predictive theories in the realm of biology than it is in, say, atomic physics. By the standards of biological theory, then, the sparseness adaptation theory of autism is remarkably predictive, particularly when compared to pretty and beguiling *Just So* stories. If the theory's predictions are not born out then whatever confounds them is probably important in the history of brains.

In Sum

That then is the gist of the theory that a Sparseness Adaptation Syndrome gives rise to autism. To deny the conclusion is to deny at least one of the five propositions leading to

it. In constructing counterarguments it is not enough to show that there are moderating influences: That is obvious from the existence of crowds, and *Parts III* and *IV* of this essay explore these influences. A proper counterargument, though, must show either that unrelenting sparse frontiers leave no genetic trace or that such traces cannot underlie even in part a syndrome of social disconnectedness.

Doubtless there are flaws in this essay's reasoning, and it does not pretend to account for all of autism. However, if something even remotely *like* the story happened in the history of human brains, the result would be that autism- or autism-like genes diffused from sparse areas into the larger human gene pool, bringing with them something very like what we know today as autism.

NOTE

The sparseness adaptation hypothesis does not support the view that any group of brains is globally superior to others, and any attempt to distort the reasoning to that end is an abuse of the hypothesis and associated theory.

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