The Sparseness Adaptation Syndrome, Part I

Adaptation to low population density yields traits
correlated with autism and male gender.

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Abstract

Charles Darwin's theory of evolution predicts evolutionary histories only weakly. The theory now presented addresses some of the predictive weakness in the following way: Adaptation to low food and population density at topologically necessary frontiers inevitably yields a syndrome that accounts for major features of classical autism, including its defining social disconnectedness, correlation with male gender, spectrum of intensities, variety, dependence on multiple genes, rising prominence, and other features. It also produces a *generic autism* that affects all humans. The theory proceeds from thought experiments and simple reasoning, and it makes testable predictions about measurable quantities.

Introduction

Darwin's theory of evolution is strong in retrodiction and weak in prediction: It does a credible job of showing where organisms come from and a poor job of telling where they will go. According to Darwin evolution is like a backward-flowing river: All waters can be traced to the river's mouth, but proceeding uphill from the mouth it's impossible to say where any drop of water will end. Will water in a backward-flowing Mississippi end up in New York or in Alberta? Will a monkey become a man or a marmot? In common use Darwin's theory is overwhelmingly presented as if it were predictive, as in "Evolution accounts for, explains, or causes these particular traits," when in fact the theory is almost entirely *un*predictive.

The mild predictions Darwin's theory does allow are equivalent to predicting that a drop flowing uphill from New Orleans will still be in the Mississippi some miles north of the city *as long as no tributaries branch off* along the way – but it is exactly the branching tributaries that are of greatest interest and importance in evolutionary history, and those Darwin's theory cannot predict. A gradually shrinking finch beak or darkening moth wing are trips up a river without tributaries.

It was Darwin's *retro*diction that angered critics who could not or would not accept that humans arose from apelike ancestors. Lost in the hubbub was the remarkable weakness of Darwin's theory as a predictor of evolutionary courses: Accurate prediction is classically a hallmark of strong scientific theory. I explore the strengths and weaknesses of Darwin's theory in more detail in a later installment of this essay.

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[2] 1st version 2000; See autismtheory.org/sparse1.pdf for the latest version of this paper.

The theory of Sparseness Adaptation presented here addresses some of the predictive failure of Darwinian evolutionary theory. In doing so it plausibly accounts for major features of autism, including its defining social disconnectedness and its correlation with male gender. The theory does this with a single hypothesis: **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. 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 simple argument described 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 solider than a *Just So* story. In any case solid details are forthcoming. The Sparseness Adaptation theory of autism also makes testable predictions about measurable quantities.

Autism's Defining Feature

Once autism had no name. It is useful to remember this when crafting a theory addressing 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. Asperger [1] 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.

For the forgoing reasons I define **generic autism** as a profound tendency to social disconnectedness, including for example a relative absence of generic social ability and disinclinations to communicate, cooperate and behave empathetically – compared to the population as a whole. The general trend of this paper is that classical autism is an *instance* of generic autism, and that identifying autism too closely with specific features like echolalia or pronoun reversal is like identifying automobiles with features like power windows or crank starters. The origins of echolalia and pronoun reversal may be important but are beyond the scope of the present theory. For simplicity in this paper (Part I) the word *autism* refers to both classical and generic autism unless stated otherwise.

Sparseness Examined

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 five 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 increasingly extreme 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.

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.[14] It is impossible to *learn* social behaviors without genetically-shaped brain structures that permit social learning: You can't teach a rock to socialize. 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.

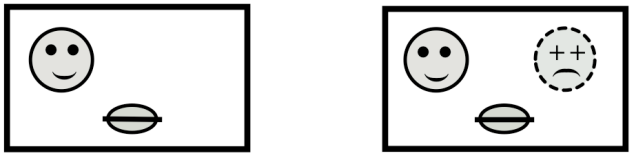
3. In sparse areas brain parts promote either food gathering or death. Where food is scarce brains must find food quickly or die. Given that all organism parts need food to build, maintain and use, any organism part that does not directly or indirectly promote the arrival of food acts as a drain on stored 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. If you can put it in a big enough bag it has a frontier. 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 increasingly extreme 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 cooperative strategy can reliably transmit genes, but the genes of the deadliest competitor endure.

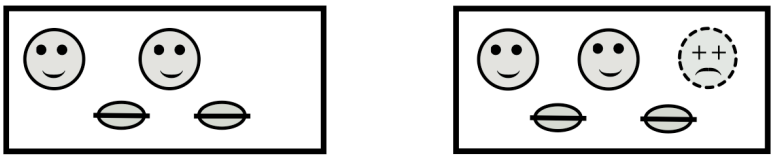
The deadly competitor who survives at an extreme frontier lacks not only *generic* social ability, but almost any aptitude for social connection whatsoever. By any definition **the extreme outlier is socially disconnected – and functionally autistic** in the sense of the early eponymous definition (as detailed later). 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 proposition, 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+1st 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 *li*ability 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*).[12] 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. Whatever social ability durable frontier bands possess cannot be generic. 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.

It bears noting that even if in a sparse area an unused or uncompetitive gene for sociability results in only a one percent decline in reproductive success in a each generation the prevalence of that gene will drop to one percent of its initial value in fewer than ten thousand years, which is not a long time in the ancient history of brains. For convenient reference let's call this **gene erosion**, analogous to the erosion of wood during wood chopping: Though the amount of wood removed with each stroke is small a few hundred strokes reduces a log to kindling.

In another gradual but unrelenting process surviving frontier organisms transmit their sparseness-adapted genes to organisms well within the frontier, and in this way asocial genes make their way into crowded areas. This is particularly likely when globally declining nutrient levels send frontier organisms back into the more nutrient-rich population center. There the axe does not fall on frontier 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. In this way autism comes to affect all human societies.

The Sparseness Adaptation Theory implies first the *destruction* of useless, draining and outright dangerous social abilities in sparse areas. It happens that this is sufficient to account for many features of autism. However, there is also increased demand to *construct* non-social abilities in sparse regions – for example to enable better hunting, survival-gear manufacture, and the like. At the frontier there is thus a "selective pressure" to shift genetically-disposed brain resources from social- to object-related abilities. This is a secondary, constructive, aspect of the Sparseness Adaptation hypothesis. The destruction of social abilities is straightforward: The construction of fresh abilities is more complex. While the theory does account for the potential freeing-up of brain resources, it does not supply details of their redirection toward object-related abilities.

Exceptions and Objections

Sparseness effects are inevitable in any real population, but their magnitudes can vary. When organisms have similar average exposures to sparseness this blunts sparseness effects – as happens when a population approximates either of two ideal cases now detailed.

Two ideal populations are immune to effects of frontier sparseness – the perfectly spherical and the perfectly stirred. Objects uniformly distributed in a perfectly spherical shell (or ring) are all effectively at the frontier and so all have identical exposures to sparseness. A perfectly-stirred population also avoids sparseness effects because over time its members have the same average exposure to sparseness.

Perfectly spherical and perfectly stirred populations do not exist in nature, nor even approximately among humans and their ancestors. Most observed populations, including all primate populations, are far from spherical most of the time so this is unlikely to have greatly mitigated sparseness effects in human history. There is also no perfect population stirring, also not even approximately among humans and their ancestors. If human populations mixed perfectly the geographically-distributed racial differences we see today would not exist: At the least everyone would be of a uniform color.

Although there are no perfectly spherical or stirred populations among Earth's creatures, some organisms do live in constrained *niches* that expose members to similar levels of sparseness overall. However humans are not among these highly-constrained species. This is key. Humans became in many ways the dominant species on Earth precisely because their ancestors were *not* confined to highly-constrained niches. Even organisms in tight niches feel the vise of the Sparse Box, but humans and human ancestors do radically more so. **If any creatures were expected to show sparseness adaptation effects it would be humans.**

Sharp Boundaries

Some human ancestral territories had sharp boundaries, for example ocean shores. To a human ancestor unable to get food from the sea a shore was the edge of a lethal zone. Even when the land was lush right up to the shore, though, sparseness adaptation effects were to be expected because shore-dwellers lacked neighbors in the direction of the sea. Also, a non-fishing shore-dweller could gather food only in the direction away from the ocean, whereas one dwelling somewhat inland potentially could gather food in all directions. Where human ancestors did gather much food from the sea a resource gradient potentially declined inland from the coast, shifting sparseness adaptation effects inland.

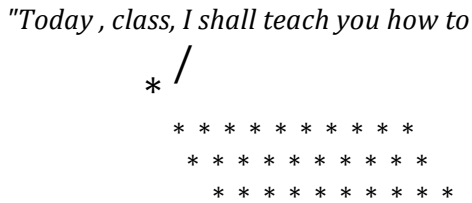
The steepness and topography of resource gradients affect sparseness adaptations and these adaptations can vary in topography and scale but probably cannot disappear. In any case, as we shall see in later discussions of gender and global resource distribution, resource gradients abounded in human ancestral environments and were on a scale to have marked adaptation effects.

Nature vs. Nurture

To this point we have discussed the effect of sparseness on genes, but inevitably in this context the question arises, "Why can't these behavioral differences simply be *learned*, so that all humans or human ancestors are fundamentally alike?" This is the familiar "nature/nurture" question.

The idea that what is learned is in no way inherited betrays a certain naiveté about the genetic and neural underpinnings of learning. For readers unfamiliar with the history of thought on this subject I have attached an *Appendix on Inborn Brain Structure.*

There are many reasons to expect that major sparseness effects are genetic rather than learned, but the most obvious one is that organisms at the extreme frontier don't have the luxury to learn what they must know always and immediately in order to survive. The situation is analogous to proposing that the human instinct to breathe might be learned rather than genetic: The person who didn't know how to breathe at birth wouldn't make it to the first breathing class! In the same way the brain that, at an extreme frontier, did not know instinctively to grab food first and waste none of it would quickly succumb to one that did know this.



An Arctic Exception?

Consider a collection of living things so interdependent that none can survive long far from the group. If you are human your own body fits this description: Without special handling cells removed from your body quickly perish. When a living thing is like a cell in a body and flatly cannot survive alone then there is no opportunity for it to be fully sparseness-adapted. Life appears to be like that in some *societies* and environments, for example in the Arctic.

Among ancient Arctic dwellers banishment from society probably meant death for all but the hardiest exile. In such societies tribal autism remained possible, but individual autism had little opportunity to flourish. At first this seems an exception potentially fatal to the Sparseness Adaptation theory of autism. However, *to prevent individual sparseness adaptation absolutely lone survival must be impossible absolutely* and this is obviously not the case in general (allowing for the bare minimum of social contact needed for gene transmission). It is silly to suppose that no Arctic exile ever survived for a time as a lone seal-hunter.

It is not possible to escape all effects of the fourth proposition above – that all populations have surfaces where population density is relatively sparse. Like the earlier-mentioned shore-dwellers, even the outermost soldiers in a phalanx live in relative sparseness because they lack outer neighbors, and their genes feel the effect of their exposure. As also mentioned earlier a shoreline, the typical home of Arctic-dwellers, is not the nadir of resource availability because oceans supply bounty to fishers and hunters there. One cannot escape sparseness effects on the Arctic coast but it is also not the place to look for the most pronounced sparseness effects.

Sparseness Adaptation continues to affect people who remain in a group whether in the Arctic or elsewhere. *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 limited, ambivalent and not at all generic – socially limited because in very sparse areas there is a premium on communication 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. Urban-dwellers often have such a hard time imagining life at the frontier that they concoct schemes whereby a team of comrades will carry the day at the frontier. This is more for psychological comfort than for any reality as a survival strategy: Where there is food for one there is food for one. Keep in mind that if a hunting strategy requires three times as many people to kill an animal for food, then the strategy must be at least three times as effective as a lone hunter's in order to feed all the mouths involved. In any case the social ability needed near a sparse frontier is probably more like that of a soldier under fire than that of a genial 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 and deadly 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** – and from the common lay misunderstanding of Darwin's theory of evolution. In a moment we shall see too that far more than Darwin's theory the Sparseness Adaptation theory makes testable predictions about measurable quantities.

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 existing questions about autism while raising challenging new ones.

Because social disconnectedness is the central, eponymous feature of autism it is the primary feature for which a theory of autism must account. A strong 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, 18]
- occur in so many varieties? [19]
- have a spectrum of intensities? [19]
- depend on so many different genes? [7]

Why are there autistic savants? [15]
Why is autism becoming more prominent? [20]

The Sparseness Adaptation hypothesis addresses each of these questions.

Addressing the Mysteries

Why does autism exist in the first place? To recap, 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 is 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? 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 and this yields an **autism 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.**

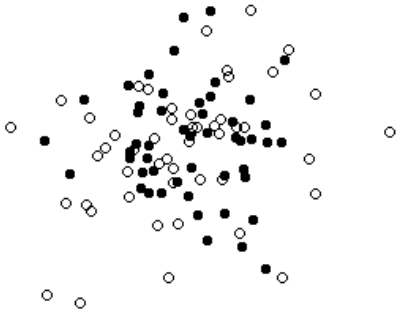
Why does autism depend on so many different genes? This is because social ability depends on so many different genes: It is the varied loss of these genes that manifests as autism.

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 *un*burdened 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.[11] 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 Sparseness Adaptation hypothesis raises a **new question:** *Given that a tendency to kill is sparseness-adaptive* ***why is violence not a more prominent feature*** *of classical autism?* Several answers to this troubling question 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 bones.

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.

Testing the Theory: 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.
• Brain scans will show than brain parts activated by object-related tasks in most people are also activated by social tasks in some autistic people.
• Inbreeding in sparse areas exposes recessive genes to great sparseness-adaptive pressure

and so sparseness-adaptive traits may be disproportionately recessive. (?)
• 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.

Perhaps the main prediction of the Sparseness Adaptation theory is that people who tend to social disconnection today are more likely than others to have ancestors who dwelled at length in sparse regions. There are, however, two main ways to measure present-day social ability: 1) Use existing diagnostic measures of clinical (classical) autism, and 2) Use new measures of social ability de-emphasizing traits with little bearing on social connectedness. Correlations with sparseness of ancestral habitat are predicted in both cases, but will likely be stronger in the second case.

Compensations for autistic social disability are a fascinating area of potential study. In general the Sparseness Adaptation theory expects that when faced with social problems people with social-gene-depleted genomes will compensate by using brain regions adaptively honed for non-social tasks. In other words the autistic person freed from frontier survival demands may relate to other people by using brain parts primarily adapted for survival among objects, a few neighbors, and non-human species. When technology frees people generally from frontier survival demands entire "autistic societies" with cultures based on learned object-oriented approaches to social relationship become possible. It may be that this is a norm at some places on Earth.

Learned compensations for autistic social disability are a secondary form of social ability. The Sparseness Adaptation theory thus predicts two broad, overlapping categories of social ability: the innate and the learned, and expects their brain activity signatures to differ and to correlate with differing degrees of ancestral-homeland sparseness. These categories distinguish between people inclined to relate warmly to others more or less at birth and people who *learn* to treat others kindly. Distinguishing the two forms of social ability complicates but does not prevent testing basic predictions of the Sparseness Adaptation theory.

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 later parts 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.

Appendix on Inborn Brain Structure

Much of human intellectual history, extending at least to Plato's doctrine of forms, has involved the discovery that one must know something to learn something. Around the year 1700 C.E. Leibniz described the realization this way: "I have made use also of the comparison of a block of marble which has veins, rather than of a block of marble wholly even, or of blank tablets, i.e. of what is called among philosophers a *tabula rasa.* For if the soul resembled these blank tablets, truths would be in us as the figure of Hercules is in the marble, when the marble is wholly indifferent to the reception of this figure or some other. But if there were veins in the block which should indicate the figure of Hercules rather than other figures, this block would be more determined thereto, and Hercules would be in it as in some sense innate, although it would be needful to labor to discover these veins, … Thus it is that ideas and truths are for us innate, as inclinations, dispositions, habits or natural potentialities…"[10] Where Leibniz wrote *soul* one could as easily read *brain*. It bears noting in passing that even a blank slate has structure inasmuch as a slate has finite size and edges – and writing on it can be neither too small or piled upon itself if it is to be legible. The quote is of course a description of Leibniz' conclusion and not of the reasoning that led to it.

The following technical reasoning confirms Leibniz' conclusion: Each character in a simple text is commonly coded as a single digital *byte* consisting of 8 information *bits* each with a value of 0 or 1. This allows each byte to represent an upper- or lower-case letter in English, some punctuation and also an indication of whether the character is bold or italic. Suppose that a brain is shown a sequence of characters obeying a rule that merely forbids certain characters and the brain is challenged to learn that rule. Sample rules might be, "The input character must be italic," or "The character must be either an A or a Z but never bold." In a single byte of information there are 2^8 = 256 possible bit arrangements, any one of which can be allowed or disallowed by a particular rule. There are thus 2^256 possible rules that the brain might be required to learn. Note that this is the number for simple rules concerning only the allowability of each byte, not the vastly greater number of possible rules about a byte's probability or its relation to other bytes in the input stream.

The forgoing simple learning task corresponds to a brain maintaining a bank of 256 switches and flipping one whenever a previously unseen character is observed. Of course a brain can never know that a previously unseen character might not yet appear, but it must nevertheless be able to hold 256 switch states if it is in principle to learn any of the possible rules governing its byte-sized input. This is a rudimentary form of learning and yet in general for n input bits there are 2^(2^n) possible such simple rules and 2^n switches required.[17] 2^n exceeds the number of proton-sized volumes in the observable universe for n less than 500 bits, which is significantly smaller than the number of bits coding this sentence. There is room for only about 2^142 proton-sized switches in a human brain. That is not enough to check for all possible allowed-byte-character patterns in a sentence this long: "Out, out, brief candle." Bear in mind that just one other possible input of that length is: "**G***[*s*k***t**E*b*^**q*L*~t**ej***bQ***P**a**}***l***b**K**.**" In other words a brain that can *learn* the rules governing the characters in even a simple phrase must *exclude from consideration* a vast number of possible rules *a priori:* It has no room to do otherwise. This is brain structure in its most elementary form, structure present at birth and required for learning.

It may be that "laws of the universe" prevent certain brain input patterns from ever occurring – that certain sensible patterns in fact will never occur. If so the brain must have inborn structure excluding these sensible patterns from needless representation in its parts. This in itself is brain structure present at birth and required for learning. It is structure controlled by, and in the weakest case influenced by, genes.

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simple and as few as possible without having to surrender the adequate representation of a single datum of experience."

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