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Experts evidencelessly parrotting about a "disorder"

[[Afterthought to this chapter: Some readers may feel that my talking of "experts parrotting" is cheeky language symptomatic of too much conceit and too little respect. And indeed, who am I to speak thus? I too read things and then repeat them to others. Nevertheless, I suggest that the majority of research professionals are still somewhat rightly criticised here, as they often treat their own parrotting much more seriously than they ought to. There's a difference between merely saying "researchers have found that xyz" and insisting that that xyz is an established fact which doubters only doubt because they are stupid or ignorant non-professionals.]]

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> *"New scientific ideas never spring from a communal body, however organized, but rather from the head of an individually inspired researcher who struggles with his problems in solitary thought."* – Max Planck

In a previous chapter I have explained how research professionals are highly selected and trained to become very skilled at mindlessly parrotting the received pseudo-wisdoms without the inefficiency of stopping to think whether they might actually be a load of rubbish anyway. The results of these defective social arrangements are all too substantial in the outputs of almost all the professional researchers in the autism causation field (even though many have with some ingenuity and honesty discovered many things despite the handicaps of their defective educations).

Competent science is absolutely dependent on competent, careful use of language. Terminology must not be used in ways which presumptively imply that there is greater knowledge than is in honest reality actually known. And while it would be generally preferable for the language used to be pleasant and positive, it most certainly should not be euphemistically clouding over some realities or other, nor underhandedly steering the reader to one or other hoped-for interpretations of the facts.
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The matters to be explained in this chapter are not some mere trivialities of "mere semantics" or personal taste preference of labelling. Rather the terminology used by these autism experts indicates that they have not even grasped the basic essence of what autism "is", but have instead encased their thinking in a fallacious misunderstanding. There is little prospect of ever understanding the causation of autism if you haven't even reached the most basic starting point of understanding what sort of thing autism *is* anyway. And autism is hard enough for the public to understand without experts talking rubbish language to complicate the matter even more.

Words mean different things to different people, and vitally fundamental proper meanings tend to get overlaid by more simplistic ones as the intellectual decadence of academia continues. This muddling has been happening to the vitally useful word that is "syndrome" but I shall continue here anyway.

The word *syndrome* derives from the classical Greek σύνδρομον, meaning "concurrence". A syndrome is a descriptive-observational sort of thing – the observation that certain symptoms or features or characteristics tend to be associated together. One might thus talk of the "biggic syndrome", of greater tallness, longer arms and legs, larger hands, larger feet, larger chest, larger head, and so on. That "biggic syndrome" does not exclude the fact that some people have large heads but average feet, and so on, but such persons would be recognised as being relatively atypical, only marginally biggic, rather than typical or central examples of "biggism".

Within health science, various syndromes have been recognised. For instance, carpal tunnel syndrome tends to involve numbness, tingling, or burning sensations in the thumb and fingers and loss of grip strength. Such a syndrome does not necessarily correspond to a single causality. But meanwhile a number of syndromes have indeed been identified as caused by particular genetic abnormalities, such as Down syndrome, Turner syndrome, and Williams syndrome. In those cases the syndrome word is used in the usage manner of modern genetics to indicate that specific causality.

Note that a syndrome (in its traditional descriptive meaning) is a statistical characteristic of a population rather than something which a particular individual can "have". By contrast, a person may indeed have the abnormality of a genetic syndrome (in the word's modern genetics meaning), for instance may have the relevant deletion from chromosome 7 and consequently be properly said to *have* Williams syndrome. Or they may have an extra chromosome

21 and consequently be properly said to *have* Down syndrome. But note that in respect of autism there is not any such genetic or molecular characteristic which autistic individuals *"have"*.

Which brings us to an even more problematic word, namely "diagnosis". In respect of the autistic syndrome this word causes much more confusion than enlightenment. In just about all other fields of medicine a diagnosis means the identification or at least inference of *the cause* (or at least some aspect thereof). For instance you don't diagnose that "you have a headache" but rather you diagnose that that headache is caused by a blow to the head, or nervous tension, B-vitamin deficiency, or whatever. The diagnosis thus goes beyond mere observation to a deeper understanding, hopefully not too inaccurate. The diagnosis word could also be properly applied in respect of Down syndrome, because the causation by trisomy 21 can be established.

Unfortunately it has become customary to use the exact same word – "diagnosis" – with an entirely different meaning in psychiatry. Psychiatry is notorious for its controversial syndromes, such as schizophrenia, bipolar, depression, and of course the autistic syndrome (and or "Asperger syndrome" of which more further on). From my own studying I am satisfied that all the main syndromes are more-or-less valid observations of real-world phenomena. And in that respect I am very much a non-heretic here, unlike a significant number who insist that those labels correspond only to social constructs. But the notion that individuals can be "diagnosed" as "having" these conditions I find to be nonsense. The diagnosis word thereby insinuates what is not true. Such psychiatric "diagnoses" do not identify any originating cause or even any aspect of causality. All they do is sort the individual's behavior into one or other of the syndrome clusters. They are purely descriptive and unenlightening of anything deeper. The statement that "your child has been diagnosed as autistic" really means little more than that "your child behaves the non-standard way he does, and some other children also behave somewhat similarly". It doesn't mean they've seen some worms crawling around in his brain, some gene has "mutated", or necessarily anything more. (It probably would indicate that the child would better benefit from certain specialised educational provisions, but you would almost certainly have concluded that already anyway regardless of the "diagnosis".)

And this brings us to the most seriously unhelpful misuse of language about autism, namely the constant parrotting that "autism is a disorder", which some people supposedly "on the spectrum" supposedly "have" or are "with".

Just about every proudly PubMed-indexed paper about autism starts off with the required declaration of faith that autism is a "disorder" or even a "severe disorder", which certain persons "have" or are "with". No evidence is ever cited for this supposed fact. Which doesn't surprise myself as there isn't any such evidence but instead considerable evidence pointing in the alternative direction of autism very definitely *not* being a "disorder", as I will now start to explain.

Autism can be disabling and can be distressing. But so can an IQ of 100 by comparison with a more useful IQ of 130. It doesn't follow that the 100 IQ must be a disorder or disease or pathology or caused by something "gone wrong" in the brain.

Autism can be *caused by* a disorder such as viral infection, or may sometimes be *associated with* disorder, but it doesn't follow therefrom that the autism *itself* is a disorder *per se*.

I inquired on this point of one of the autism researchers who has contributed the most to our knowledge of the brain atypicalities associated with autism, namely Prof Manuel Casanova. He's not been particularly fanatical about the "disorder" concept himself, but anyway he suggested in response various observations such as:

> *"When neurons do not migrate from the periventricular germinal zone they form nodular heterotopias. These are unorganized islands of neurons present under the ependyma of the ventricles."*

But even if that were established as being a manifestation of disorder, it would still not follow that that abnormality was itself the autism, or that autism itself is a disorder per se. All that the various researchers have shown is that autism can *involve* or be *caused by* disorder. (I should emphasise that I don't think Dr Casanova has been personally responsible for originating or promoting this "disorder" language.)

Two clarifications on the above. Firstly, many people have been interested in finding whether there was something different about the brain of Albert Einstein. And yet if they did find such a difference they would not then conclude that it showed Einstein had a brain "disorder". The brains of Obama and Trump are almost certainly visibly different from one another, but it doesn't follow that one or other of them must have a "disorder". Secondly, all humans have a shrunken, non-functional appendix. But it doesn't follow that they can be properly described as having "shrunken appendix disorder". And the gaps between our fingers are created by death of the cells between those fingers. These sorts of "gone wrong" facts in no way evidence let alone prove that a "disorder" or even maladaption is involved.

I am not here making an impossible demand for evidence. If all autistics were shown to have some clearly pathological biological marker in common, such as high levels of a toxin, a specific gross genetic abnormality, a part of the brain rendered dysfunctional by a circulatory stoppage, or whatever, then the standard declarations that "autism is a disorder" would be justified. But even after 70 years of autism research no such marker has been identified, and that is why the "diagnosis" still consists merely of looking at the person's behavioral features and expressing an opinion about them.

And I here confidently declare that no such marker will ever be found anyway, because all the evidence tells me that autism is not a disorder anyway. Rather it is merely, or more accurately, it IS an important part of the non-pathological variability of being human (or alive more generally). Indeed I will go further and explain why autism can never be defined solely in terms of the brain, or even the body, but only in relation to the environment outside of the person or other organism. I emphasise this: autism is not a characteristic of the brain or of the body, but only of the organism (person or animal) *in relationship to a specific environment*.

I'll first just point out some facts that don't sit comfortably with the notion of autism being a disorder (*per se)*.

Some of these facts were already pointed out more than 20 years ago in my 1993 publication (Chapter 7 here), even though at that time I did not have any thoughts of this "disorder" dogma which only got canonised into Holy Writ later on. I quote (in which *SES* means Socio-Economic Status, and *bimodal* means like a graph with two peaks on the same curve.):

> "The only epidemiological survey of the IQ of parents (Lotter, 1967) found substantially above-average scores on the Mill Hill Vocabulary Scale ($p < 0.005$) and the Standard Progressive Matrices ($\chi_2(2, N = 15) = 98.7, p < 10^{-20}$). The other studies of parental IQ have given similar, though less marked results (Cantwell, Baker, & Rutter, 1978). Members of Mensa (IQ > 148) have been found to have three to six times the normal frequency of autistic siblings and children (Sofaer & Emery, 1981). though the significance of this is somewhat limited by the small number of cases. Because there is a substantial correlation between IQ and SES, and because this theory proposes similar bimodal distributions for both, these findings must be set in the context of the preceding discussion of evidence concerning SES."

And that evidence concerning SES involved substantial and highly significant associations of autism with high SES including peculiar

bimodal distributions (i.e. double-peaked graphs), none of which can be merely dismissed in terms of sampling (reporting) bias. (Fuller details are in Chapter 7.) The lower-SES peak of the bimodal graphs can be understood as being caused by pre-natal or peri-natal adversities suffered by lower class mothers (and the next chapter here will be discussing a "health" technology which is very much forced on the lower classes to this day).

Those associations with high IQ and high SES are perfectly in line with the central concept of the antiinnatia theory of autism (Chapter 7). Namely that antiinnatia factors in the normal range of intensity cause high IQ (and tend to raise SES) and are exactly the same factors which cause autism in a higher range of intensity. Certain other observations which further support that concept were also cited in the 1993 paper, such as:

> "Immaturity of general appearance and unusual symmetry of face. (Attractive appearance, and intelligent appearance...."

(this in the context that as predicted by the antiinnatia theory, facial symmetry has since been found to be correlated with high IQ, as referenced in Chapter 16.).

And:

> "Skills that do not involve language, including music, arithmetic, dismantling and assembling mechanical or electrical objects, fitting together jigsaw or constructional toys. (Some very retarded can read words out loud.)"

And a study of 137 parents of autistic children found that 28% believed their children met the criteria for a savant skill, defined as a skill or power "at a level that would be unusual even for 'normal' people" (Howlin et al., 2009).

And:

> "An unusual form of memory: the ability to store items for prolonged periods in the exact form they were first experienced."

Meanwhile some professors such as Temple Grandin have been "diagnosed" as or otherwise considered to be autistic. And there have frequently been suggestions that creative geniuses have some elements of autism. Indeed the antiinnatia theory was from its very start a theory of IQ, genius, and autism, with all being caused by one or other level of antiinnatia factors.

And add to this the finding of autistics being more rational than non-autistics (Allman et al., 2005; DeMartino et al., 2008).

Plus the findings that in the first two years they have larger brains and more neuron connections in those brains. (Their brain growth later slows down but that would be expected to happen in

consequence of the grimly unstimulating but stressful lives the more severe children tend to experience.)

Plus the fact that many autistics strongly object to being described as having a "disorder" or even "having" or being "with" anything for that matter. Instead they are proud to be what they are, namely autistic, and thereby as they see it often superior to what they see as the inferior normals. Regarding which maybe this would be a good place to tell you about my discovery of Neurotypicalism Spectrum Disorder ("neurotypical" being a word invented by autistics to refer to "normal" people). I quote from my account on the 2009 Awares autism online conference:

> "Neurotypicality is a disorder with desperately tragic symptoms, some of which are indicated below.
> Many neurotypicals, especially male ones, spend endless hours obsessed in intense fascination at people they will never meet or even communicate with kicking leather spheres around an area of grass for hours at a time.
> Meanwhile the female neurotypicals spend endless hours in intense fascination reading about people who don't even exist, or avidly watching tv series about such non-existent people.
> Another neurotypical symptom is a great preoccupation with which group, "class", movement, etc, which they or others supposedly belong to. Some even become obsessed with the obsessions of others about which groups etc the others are obsessed about...."

And note that the autism pride (neurodiversity) movement is a peculiar anomaly. There's never been any "psychosis pride", "neurosis pride", "depression pride", "attention deficit pride", etc.

And here is another quotation about the autism "disorder", this time from a newsletter email I got from Karen Simmons of AutismToday.com on 30th Aug 2014:

> "In fact, I thought Jonathan was extraordinarily bright since he began reading at the age of 2 1/2, when he read the word "recycle" off of a truck. At 3, he would memorize songs like it was nothing too. One song in particular included all the letters of the alphabet."

And recent-ish research has found superior pitch discrimination hearing (Bonnel, Mottron, et al., 2003; O'Riordan & Passetti, 2006; Heaton et al., 2008). And superior touch sensitivity (Blakemore, Sarfait, et al., 2006). And greater ability to detect odours (Ashwin, Chapman, et al., 2014). There have also been reports of greatly enhanced visual acuity though there are contrary views as to whether they have been well-founded or not.

Meanwhile, autistics have also been found to be far cleverer than they seemed (which goes strangely harmoniously with that theory I published years ago claiming that autism was caused by exactly the same factors as high IQ, and involving exceptionally low levels of "IQ impairers"....). The Raven's Progressive Matrices (RPM) is considered the ultimate measure of the most essential, general aspect of intellectual ability involved in problem-solving and other processing tasks. Hayashi et al. (2008) found that Asperger autistics had RPM scores higher than controls, leading them to suggest "that individuals with Asperger's disorder have higher fluid reasoning ability than normal individuals, highlighting superior fluid intelligence." And various other studies have reached similar conclusions (Dawson, Soulieres, et al., 2007; Soulieres et al., 2011) and that autistics solve the RPM items much faster, and also had 31% faster performance on "inspection time" tasks compared to controls matched on the WISC IQ test (Barbeau et al., 2013). The studies I have cited here are web-accessible and will point you to others which find more or less the same. And notably the Dawson, Mottron, Soulieres, et al. team share my own rejection of the "disorder" terminology along with the hypocritical "persons with autism" nonsense, as does Jim Sinclair (1999).

And now putting all those preceding facts together, namely special skills, abnormally accurate memory, better-looking, more symmetrical, more rational, less emotionaically jerkic, larger brains with more connections, superior hearing, touch, and smell, high fundamental intelligence, faster brain speed, no pathological criterion found after 70 years of research, association with higher IQ and higher SES, and being something which many of the "victims" consider themselves proud to <u>*be*</u> (rather than be "with" or "having" or "on") anyway.... on what basis can this be ASSUMED to be obviously a "disorder" such as to justify just about every "scientific" paper ever listed in PubMed beginning with that evidence-free recitation that "Autism is a disorder....." ? ("Well we all got our PhDs at Harvard so it must be true....")("Baah!")

In a later chapter here you can read the only theory of autism (and IQ and genius) which actually successfully grapples with all the key facts and questions. And it has no need to resort to any far-fetched presumption that autism is a disorder, indeed rather its neglect for so many years could raise a question of whether *Academism* Spectrum Disorder is very much more the real disorder.

By way of moving on to what I suggest to be a more competent understanding of the matter, here's another quote from my 1993-published paper:

> "the existence of a continuum ranging from severe autism through the much milder and more common Asperger's syndrome (Gillberg & Gillberg, 1989; Frith, 1991) to normality."

Re which please consider the dimension of personality from extraversion to introversion, specifically in people who are a bit inclined also to above-average neuroticism. An extremely extravert person would tend to be "pathologically" impulsive and consequently doing stupid things such as reckless criminal offences or dangerous acts. And an extremely introverted person would tend to be "pathologically" shy and averse to commonplace noise and excitement. Both these extreme persons have serious problems but they are in no way due to a "disorder" they "have". They just are as they are, by reason of natural variation (due to genes and or environment or something in the water).

Likewise some people have lower IQs than others. Yet there is no level of IQ which can be said with scientific justification to be a boundary between "having" or not "having" of "low IQ disorder" ("mental retardation" or whatever the latest squirm-word is nowadays). Rather if we look at progressively lower levels of antiinnatia factors the brain becomes progressively slower and more error-prone, hence the lower IQ, as explained in Chapter 7. And conversely, with progressively higher levels of antiinnatia factors, the brain first becomes progressively faster and error-free, and then other things start happening which give us firstly a narrow window of creative genius-potential merging into marginal autism (including "Aspergers"), and then onwards to severe autism and ultimately non-viability manifesting as stillbirth.

In the first chapter here I explained how just about all academics have a severe unlearning disability, and consequently many of them are going to be unable to unlearn their parrotting of the "disorder" word and the faulty notions underlying it. They will soothe their cognitive dissonance by claiming that the conception I have outlined above is wrong in some way or other.

One of the points they will raise to rationalise away their denial of their inability to unlearn will relate to yet another problematic terminology commonly used about autism, namely *"de novo mutation"*.

You probably already know that a mutation is a change in an organism's DNA sequence of genes. If you don't already know about this it would be best if you study about it via a biology textbook or encyclopedia or equivalent online information before continuing here.

A *de novo mutation* is a mutation which is not present in either parent, hence has arisen "de novo", that is newly, in the individual

in question. Actually in this case the terminology is not being incorrectly used. What is incorrect is what is being implied about and inferred from those de novo mutations.

Certain sectors of autism research have as their greatest preoccupation the finding out of "what has gone wrong" to cause the "disorder" which is autism/ASD/ Aspergers. From the perspective of a career-cautious researcher, it makes a lot of sense to try to blame a gene or a virus for "what has gone wrong", because genes and viruses cannot get angry at you for blaming them and cannot start legal action for libel compensation. By contrast if you blame some product put in peoples' mouths, then the makers and marketers of that product might indeed get angry at you and start legal action and other bother against you. So there's a very important principle in medical research that it's far better if you can blame a gene or virus.

Indeed it gets much better. If you can blame one or more genes, not only can those genes not sue you but you can then patent everything about them and the tests to detect them and ways to change them and patented drugs to block them, and thereby make a recurring income-stream fortune of trillions of dollars. Not to mention all the research jobs created in the process.

In respect of autism, there has for many years been evidence that a virus such as rubella can increase the risk of becoming autistic. But only in a minority of cases. So for the researchers it's very important that we go on to find those evil (but highly profitable) genes which are hoped to be behind "what's gone wrong" (even though in reality nothing has "gone wrong" in the autistic brain anyway).

In the 1993-published paper I indicated my conclusion that most or much autism before that time (before the increase) had been mainly due to genes, and that a great many different genes would be involved. *And* that they would be exactly the same genes which cause raised IQ and raised SES and in some rare circumstances also cause creative genius. I'll now suggest there was much wisdom in the comment many years later from Simon Baron-Cohen that seeking to abort autistics could be greatly misconceived on account of it also tending to eliminate rare valuable talents from our populations.

But meanwhile most research money has been staked on finding the evil genes causing this "disorder". A few years back, a huge study was published in *Nature*, the most prestigious of all journals. The list of authors alone filled several pages. And yet the genes and genetic anomalies they (reckoned to have) found could only account for a very small minority of autism cases.

An important part of the evidence which researchers assume to be supporting their "bad genes" theory of autism relates to ages of parents. It has been found that older mothers and older fathers tend to have a higher probability of autistic children. But curiously the studies in question give notably differing results in different countries (Sandin et al., 2015), which should hint to us that there may be something partly or entirely cultural going on rather than entirely or partly genetic. (A very competent review of the evidence is given by Zhou (2015), who with much understatement concludes: "All this suggests that social factors may be more at play in these figures than it simply being a question of paternal or maternal age.")

But why let an inconvenient fact get in the way of a convenient one? The convenient fact in question is that the number of de novo mutations increases with age of the father. Which seems certainly true. But it does not follow that those de novo mutations are mainly causing or a main cause of autism, and even less that they represent a bad thing happening to the genes anyway.

Cutting-edge science is difficult to get perfect and it makes fools of even the cleverest of other people from time to time. There is a well-known concept of evolution by natural selection as follows. There is first the accidental generation of random changes in the DNA, that is random de novo mutations, and then the resulting slightly-changed organisms are subjected to the filtering effect of natural selection such that those with disadvantageous mutations get rarer or even eliminated.

The Mona Lisa after modification
by a few "de novo mutations"?

It would be useful to think here of a famous painting such as Leonardo's Mona Lisa portrait. The mutation process could be thought of as analogous to a blind child randomly dabbing a paint-brush at that painting. The point is that the Mona Lisa painting has been the result of much patient work and developed skill, and so just about any random change to it would be a deterioration rather than an improvement. Likewise randomly loosening or tightening bolts on a car engine would be much more likely to make it less functional than more functional. And the standard (assumed by most scientists) reasoning about mutations proceeds likewise reasonably to the conclusion that mutations will almost always be deleterious (bad) rather than advantageous (good), considering that our existing genomes are the result of millions of years of constant natural selection towards "perfection". The familiar talk of radiation tending to cause harmful mutations is seen in this same light of mutations being bad.

But..... (with sincerest apologies to those Cambridge medical graduates) But...., well, to explain this I will use another of my analogies. This involves two elderly Bechstein pianos of my acquaintance, the one a youthful 122 and the other 123. Various springs, strings, and weakly brass bridgepins died many years ago and have had to be carefully replaced. But their soundboards live on, sounding extremely much like high-quality new ones would.

And yet piano soundboards are noted for often degrading over the years (even some from S...you-know-who). The sound can be dependent on some very precarious engineering, where a difference of less than a millimetre can make the difference between excellent and abysmal. Anyone who's played around many pianos knows that

even new ones can have poor tone, and that many have turned into key-controlled drum-kits or worse a long time before they reach 80 years old let alone more. Yet these two pianos have certainly not had a cocooned pampered life, but on the contrary been grievously abused by previous ungrateful owners.

The secret of these pianos as I see it is that by 1892/3 Carl Bechstein had been progressively refining his design (the cheapo Model 5 upright in this instance) through 40 years and through the experience of many thousands of instruments produced, and as a result he had "evolved" a production formula which was robust and would still sound good even with the occasional random change of something or other.

And the relevance of this analogy about those pianos is that the DNA's production formula of the human body has likewise been refined by evolution, but not merely over 40 years and 30,000 serial numbers, but for vastly longer and more. And it is to be expected that the human body would, like those pianos, have evolved to be something that is robust and not easily defuncted by just a bit of change. I'll now go into this in more detail.

The first thing is that genes are not all equal units. Some genes have vitally important effects such as relating to sickle-cell anemia. Many other genes appear to have much more marginal importance or even none at all. It follows that mutations cannot be all equally important either.

The second thing is that not all changes of genes are equally likely to occur.

And a third, key, thing is that by reason of the stabilising refinement (in humans as in Bechstein's pianos), the genome *itself* will have evolved, such that easy but bad changes are few and far between, whereas easy but non-deleterious changes are very common.

And a fourth, even more key thing is that there is no such thing as the ideal "perfect" person with "perfect" genome. As I explained back in the 1993 paper if more than a handful had bothered to read it, there are reasons why genetic diversity (and hence genetic change, hence those nasty *de novo mutations*) can be actually *advantageous*.

Here another illustration may be useful, this time an actuality rather than mere analogy. Anyone with experience of Olympics-level fly-swatting will be well aware that flies do not all behave the same. Some keep crawling around on the window-pane, others jiggle around a bit then have a rest below on a book instead, and so on in various variations. Clothes moths have even more diverse personalities despite their flour-grain-sized brains. Without such

variable unpredictable behavior the flies and any other such species or group would quickly get eliminated by predators or other enemies who can easily anticipate what they are going to do next. There is also advantage in not having all individuals competing to fill exactly the same niche (locationally or occupationally or food preference etc).

So evolution can be expected to actually *favour* some appropriate de novo mutations, especially in respect of behaviour, and the notion that they are most likely to be deleterious (under normal conditions such as without intense radiation) is unsound.

This positive importance of genetic change is reflected in the work of Nobel laureate Werner Arber (2014), who refers to "natural strategies of genetic variation", and "a multitude of specific molecular mechanisms to contribute to the overall spontaneous genetic variation.", and "reports on cross-species gene transfer, as well as recent DNA sequence comparisons, speak clearly in favor of a general validity of the relevant natural laws of genetic variation for all living organisms."

Some further elaboration about this corrected understanding of genetics and mutation is provided in an appendix to this chapter.

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(You may at this point wish to turn to that appendix to this chapter, pages 70-77, then return back here thereafter.)

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The appendix shows that the latest research confirms that my understanding is correct, and that the outdated but still-predominant would-be wisdom is mistaken.

Thus the evil genes autism theory is unsoundly founded on a false assumption of how mutations fit into the processes of evolution.

I indicated above that one should expect the main reasons for the associations with parental age to be cultural rather than genetic. But let's first take a silly idea to its logical conclusion. The idea swimming around is that older parenthood causes inferior offspring and so parents should be encouraged to have their children earlier. There's a slight problem with this advice, because the age association with autism and with those de novo mutations goes near-linearly right back to age 15 or earlier. So on this logic we should be advising 15 year-old boys and girls to have families straight away before legal adulthood so as to avoid those supposedly horrible autistics being born. Meanwhile in the real world.....

In the real world I explained several decades ago that autism is associated with higher IQ, higher SES, more beautiful faces, and higher biological superiority generally, because the antiinnatia genes tend to produce all of those. Oh.... and "immaturity of general appearance" (Wing, 1976 cited in Clarke, 1993). The average 45-

year-old man in many countries is an ageing slob who (a) is no longer attractive to youngish women, (b) probably has enough on his hands already children-wise (in terms of energy and money earning means), (c) is probably not physically up to any more fatherhood anyway, or at least not much bothered, and (d) quite likely dead and buried already anyway. Meanwhile the high-SES (and "immature-looking") 45-year-old is just getting into his stride as a self-made zillionaire, or semi-celebrity, or starting a second family after a first has flown. Thus the reason why the older father has more autistic children is not because his genes are inferior but precisely because his genes are (biologically) *superior*. And likewise the under-privileged teenage schoolkidparents family have fewer autistics precisely because they have less of the antiinnatia genes for biological superiority. By the way, I haven't said anything about moral/ethical/cultural superiority here. I am merely referring to biological propensities to successfully continue the lineage to grandchildren and onwards.

For those whose brains did not stretch sufficiently to take on board the previous paragraph, I shall add here that I have just now entirely by chance encountered an article about Jeffrey Skoll, a 47-year old billionaire, who says "*I don't have kids yet, but when I do,* there's only so much I think they should have. They can make their path their own." (Grant, 2012).

And here's another. Al Pacino, described as "one of the greatest actors in all of film history". His three children were born when he was 49 and 61 years old. "The joy of work is what keeps me going."

And "Millionaire nightclub owner Peter Stringfellow is to become a father again at the age of 72."

And add these too: Frank Skinner (55), Gordon Brown (55), Sir Paul McCartney (61), Rod Stewart (66), Clint Eastwood (66). And Rupert Murdoch having two children in his seventies.

Reality dawned yet?

And so by marrying that cute guy in the same student year as yourself you may minimise the risk of your children turning out to be geniuses or superstars, or even just really nice guys and gals.

In summary about this supposed "disorder", there is no evidence that autism is a disorder, there is huge obvious evidence that it is not, and the only theory that actually accounts coherently for the syndrome explicitly endorses the concept of autism as being just a part of the normal variation of human (and non-human) life.

Interestingly Simon Baron-Cohen shares my disinclination towards the disorder word, preferring to refer to Autism Spectrum Condition, ASC. This probably reflects that he like myself has come to autism from studying psychology rather than psychiatry. The

psychiatrists have trained at med schools so they see every difference as a disease to be treated, whereas the psychologists see it as part of the multi-dimensionality of human diversity to be treasured and wondered at.

Not everything the autism researchers do is wrong. The latest edition of the Diagnostic and Statistical Manual (DSM-V) has made some progress, eliminating Asperger syndrome and adding five or so levels of severity to the "diagnosis" of autism. This is properly justified because there is no scientific basis for a distinction between autism and Asperger's. It was merely a historical accident that Kanner and Asperger made simultaneous rediscoveries of approximately the syndrome described by JL Down in 1887. But the DSM-V is still light-years off course in still containing the pseudo-scientific notion of "Autism Spectrum Disorder" aka "ASD".

Here's two last paragraphs about the "disorder" word, namely of why such inexcusable baseless false language is being used. Parents who have autistic children are in many cases very upset about them (and very tired by the extra demands involved). They hoped their children would grow up to be clever and social and get on in life but instead they find them problematic in various ways. They think that their child ought to have been "normal" and that something has "gone wrong" such that they "have" this something "wrong" with them which is not what they should have been. We have to sympathise with the parents using this language but it is simply not factually correct. And so it must be rejected, even though the situation is not entirely that simple as I will now explain.

I will present in a later chapter the evidence that most cases of autism nowadays are being caused by mercury vapor poisoning from dental amalgams. In such cases, something has indeed "gone wrong", namely they have got mercury poisoning, and they do indeed "have" something, namely excessive mercury. But it remains the case that the excessive mercury is not the autism. And some cases of autism could be entirely due to very high levels of the antiinnatia genes, especially where two ultra-high-IQ ultra-classy parents are involved. In such cases the child simply IS autistic and does not "have" anything other than too much of a good thing genetically. I don't know for sure whether there is or can be any cure or treatment for such a condition; maybe yes maybe no. I hope to have more to say on the matter at a future date.

(By the way, and this is a most important point here, as most autism is now caused by mercury poisoning, researchers who think they are studying autism are as often as not actually unwittingly studying mercury poisoning instead. And in consequence they become even more convinced of their false notion that autism must

be some disorder of the body. There's even a book titled "Autism: Oxidative Stress, Inflammation, and Immune Abnormalities." (Chauhan et al., 2009), which expertly overlooks that that's three major consequences of mercury listed in its title!)

In addition to the parents' preference of terminology, there is the researchers' preference. Saying that your research is about a terrible "disorder" sounds a lot more important and prestigious than saying you are just studying some atypicalities of behavior. And the false "disorder" language gives your research career a free pass to the medical charities funding world. And why bother with mere scientific truthfulness when you can get promotion so much easierly by means of false propaganda drivel-words?

Oh, but!.....surely the researchers are merely using the terminology officially established in the DSM (Diagnostic and Statistical Manual), as they should? But this notion is incorrect. The researchers are supposed to be the leading edge of understanding. It is the DSM that is supposed to be following the researchers rather than the other way round. And furthermore, the DSM is very far from being the uncontroversial, evidence-based tome of accumulated competence which it tends to be assumed to be. The book *Cracked* by James Davies (2013) does a good job of discussing the not-so-impressive reality underlying the DSM. And even the chairman of DSM-3, Allen Frances, came out of retirement to publish a similarly scathing condemnation of DSM-5.

The DSM is profoundly misconceived in another respect. It is trying to serve three distinct purposes and ends up serving none properly. We need:

(a) A ("scientific") answer to the everyday question of "what is (the proper definition of) autistic"? (or "how does autism manifest itself?");
(b) A working criterion for researchers to use to sort people into "autistic" and "control" in their studies, for instance studying whether autistic people have longer fingers than controls;
(c) A working criterion for clinicians and administrators to decide who should qualify for disability services and support.

And there is no reason why those three things should be anything like identical. Indeed the latter criterion would properly take major account of the practical impact of any disabilities, which is certainly not any proper part of the other two. What a ridiculous muddle. (Though at least it was written by graduates.)

Update: Unlike most academics Professor Casanova has a quite good blog on the public internet, titled "Cortical Chauvinism".

Subsequent to my sending him an early draft of this chapter and now having finished writing the rest of this book, I notice that he has meanwhile put up a blogpost defending the standard notion that autism is a disorder and not an aspect of normal variation (as the antiinnatia theory entails). I guess he's waiting for me to reply to it!

Anyway, it appears to me that his most cogent (/least uncogent) new point is that a high proportion of autistics have seizures (epilepsy). To which I have two points of rejoinder. Firstly, most autism nowadays is caused by mercury, and seizures are known to be one of the symptoms which mercury can cause. The brain processes causing seizures appear to be poorly understood anyway, but a websearch of { mercury seizures } shows up numerous people identifying mercury as a cause of seizures, including a report titled "Effects of continuous low-dose exposure to organic and inorganic mercury during development on epileptogenicity in rats" (Szasz et al., 2002; Klinghardt, 1998). This is of course *yet more* evidence in support of my claim that perinatal mercury has caused the autism increase – while also causing that epilepsy as well.

Secondly, an outright "disorder"-like symptom can still be the result of pure "normal" variation. This is exemplified by variation in height of humans and indeed of other creatures. Above a certain height, the gravity forces on the bones become excessive such that they tend to break or have joint failures. Of course in practice that causes natural selection to disfavour people being too tall, such that such problems in practice are rare. (I can think of other examples of the same principle but they would take too much space to explain here, especially as one such suffices anyway.)

Finally here, I have not yet explained my point about autism not being a characteristic of just the brain or even just of the body. It would be better if I leave that explanation until after you have read the presentation of the antiinnatia theory first so I can take it from there. Meanwhile here are some examples of de-irrationalised language for you to practice with.

Autistics with childness
Persons with degrees (usually incurable, sadly)
Women with Blackness
Men with Muslimness
Persons with Professorships
Persons with Doctorates
Researchers with Academism Spectrum Disorder

"And now please let me introduce our speaker with distinguish-edness Irva Hertz-Etc, who is a Person with Doctorate and Person with Professorship, and may soon also be awarded a diagnosis as a Researcher with Academism Spectrum Disorder...."

Yet more abusive language from academics

A further word which many researchers have become routinely accustomed to misusing is *hypothesis* (or in plural, *hypotheses*).

In later chapters here I will be presenting one or more theories. In the first chapter here I have already explained the evidence of how the medical science bureaucracy's research has become grossly perverted by a hostility to new theories to such an extent that it barely merits recognition as genuine science any more. A symptom of that perverted hostile attitude is misuse of the word "hypotheses" to refer to what are actually theories.

It is impossible for a theory to be a hypothesis and impossible for a hypothesis to be a theory, because they are categorically different things.

The "fuzzy" sciences such as psychology, sociology, and clinical and epidemiological medicine use what is known as "inferential statistics" to ascertain whether a "statistically significant" effect or relationship has been observed in a study. Every student of those fields learns about testing the *"null hypothesis"*, which is.... well please let me explain from the beginning.....

You can generally tell a hypothesis from a theory by the fact that a hypothesis can't have the word *"because"* or *"causes"* incorporated into it. For instance:

"Autistics look cuter" = hypothesis.

"Autistics look cuter <u>because</u> the antiinnatia suppresses the gene-expression of idiosycracies of their appearance" = theory.

"There is a higher prevalence of autism in Las Vegas" = hypothesis.

"There is a higher prevalence of autism in Las Vegas <u>because</u> it has been invaded by Martians recently." = (somewhat daft) theory.

"Banging a hammer on your finger is followed by more pain" = hypothesis.

"Banging a hammer on your finger is followed by more pain because the pressure causes injury to the finger, which activates pain receptors which then send impulses down nerves to your brain where the impulses are interpreted as pain." = theory.

"Higher mercury intake <u>is associated with</u> higher autism scores" = hypothesis.

"Mercury <u>causes</u> autism" = barest-bones basic theory.

"Mercury causes autism by (/because of its) selectively suppressing gene-expression" = slightly more developed theory.

"Mercury causes autism because [process a; process b; process c...] as is evidenced by [observation a; observation b; observation c...] which are logically related to those processes by [reason a; reason b; reason c...]" = highly developed theory but which *still might be a load of rubbish*. <u>But can never be a "hypothesis" just as an apple can never</u>

be a lunchbreak.

Another useful way of understanding it is that a hypothesis is a putative answer to a "What is the fact of the matter?" question, whereas a theory is a putative answer to a "Why is it so?" question.

Now back to the students studying inferential statistics, which is a very important part of your own education here. Most research studies are investigations of whether more of x is associated with more (or less) of y, such as "Is there more autism in areas where more of the cars are red?" Or something similar. The *null hypothesis* is the (alternative) hypothesis that there is no such association or difference. So from the examples above we can see null hypotheses to test such as:
"Autistics DON'T look cuter."
"There is NOT a higher prevalence of autism in Las Vegas."
"Banging a hammer on your finger is NOT followed by more pain."
"Higher mercury intake is NOT associated with higher autism scores."

At this point I shall hypothesise that I have sufficiently explained to you the categorical difference between a hypothesis and a theory. Pending evidential confirmation of that hypothesis, I shall now move on to the way the word is regularly abused by academics.

Things vary in quality and worth. A brand new Ferrari is a car but a rusted bashed 20-year old Ford with broken windows and clattering engine and worn tyres is likewise a car, and not some sort of "carpothesis".

Likewise theories vary greatly in their quality and worth. But it gets more complicated. You will often encounter the phrase "The theory that ....." (usually followed a bit further on by "has been disproven by numerous studies"). This is where you can get confused. Consider for instance "the theory that mercury causes autism". This is not the same as "the theory that mercury causes *all* autism" or "the theory that mercury causes *some but not all* autism", or even the same as "the theory that mercury is one of the factors in the causation of autism". Such distinctions are important, because these are simply not the same theories even in basic outline.

But I have made a mistake in the preceding paragraph. Did you notice it? It's this. There is not one single "theory that mercury causes autism". Rather there can be many. Including a sub-collection of "theories that mercury kills braincells and thereby causes autism". Those ones I don't personally rate very highly. Then there is the theory that "mercury randomly binds to DNA and thereby acts as an antiinnatia factor and thereby causes autism". Which is one which I argue for in later chapters here.

A theory such as "Autism is being caused by visiting Martians

because Martians are yellow-striped and the yellow stripes are very relaxing and that relaxation causes people to learn foreign languages which is the main symptom of autism." is a load of rubbish. And yet it is a theory none the less, just a very rubbishy theory. Likewise "Autism is now being caused by the absence of mercury from most modern vaccines" is a theory of very low credibility. But it still cannot be a hypothesis.

And here's a crucial point. Even a very decent theory well-supported by evidence and argument, if it is nevertheless new and thus has not yet undergone some process of bureaucratic herd endorsement (baah!), tends to be treated with the greatest of hostility as evidenced in the preceding chapter. And as part of the process of contempt, it is belittled as supposedly not even being a theory anyway, but as something supposedly categorically different, namely a mere "hypothesis", just as a Person with Professorship or Person with Doctorate is somehow categorically different in their very essence from a mere unqualified "person". In the status-obsessed mindset of academics, a theory can only be an outstanding great discovery by a Darwin or Einstein, whereas Simon Baron-Cohen's disputed theory that high fetal testosterone is a major cause of autism can only be a piddling "hypothesis" (not that I rate it much myself either).

Wikipedia declares that "A theory is a well-substantiated explanation...", and "Theories are the single highest level of scientific achievement". Meanwhile some famous journals such as the Lancet and Nature have a category of articles they call *"Hypothesis"*, and yet they clearly must be having in mind would-be theoretical explanations given that they are required to have not yet been "tested" and yet take up up to 1500 words whereas just about any genuine hypothesis can be stated in a single sentence such as the examples above. It is impossible to take a whole article to propose the hypothesis that "Vitamin C intake below 2 mg is associated with autism a year later (but *no-one has tested it yet!*)". I've just done it fully in that sentence there. But it could take an article to propose the theory that "Vitamin C intake below 2mg causes autism because [process a; process b] as supported by [reasoning c; reasoning d]".

And that position of Wikipedia and the academic parrotting is arguably a very harmful terminology, because just about every great theory starts its life as a mere rough idea to which only later is there attached more and more evidence and reasoning. And the last thing we need for the advancement of science is such a false pseudo-categorising barrier blocking even more the way to recognition of new and better understandings.

And the other last thing we need is such sloppy abusive use of the terminology which makes it all the harder for readers to accurately understand what writers are talking about.

And no, the fact that x million people have sloppily abused some language for decades does not make it acceptable or valid via some rationale that "words only mean what people mean them to mean anyway". Which people, when, where, why? Certainly not myself. I have higher standards and will not be lowering them for any number of superior experts.

The autism increase controversy

The following Chapter 3 contains a discussion of the notion that there has not really been an increase of autism in recent decades. That notion is also revisited in Chapter 12. But it would be useful add some further prefatory content here for unpicking the false reasonings of those denying the increase.

Firstly it might be useful to be clear about some motivations. Such motivations do not necessarily cause any bias, but being aware of them can enable due scepticism in one's reading of the debate.

A first motivated group are those of the medical establishment, who have a very heavily-developed reflex which could be well-characterised as a perversion of the Hippocratic Oath: "First do not admit to doing any harm". This reflex is particularly liable to be excited whenever the word "mercury" is in the air (for reasons that might have something to do with Chapter 3 here). These establishment people are strongly motivated to deny there has been an increase because that helps towards denying that themselves the medics have caused the catastrophe revealed by the increase.

A second motivated group consists of the more fanatical of the "Neurodiversity" or "autism pride" people, according to whose viewpoint autism is never a problem or disability and certainly not a disorder or illness, and "therefore" it cannot have been caused by some adverse factor, and "therefore" an increase cannot have been caused. (This is an example of a very common phenomenon of human mentation, namely adjusting the "facts" to fit the prior theory (chosen for emotional reasons) rather than correcting the theory to fit the actual facts.)

A third motivated group is some parents of autistic children, who are somewhat motivated to find someone to blame and to seek compensation from (though many parents are more motivated to just find out the truth of what caused and what could un-cause the autism). It is only some of this third group who might be motivated to falsely perceive an increase rather than deny one.

The idea that there had indeed been an increase originated

with facts noted by researchers in the field. There were the direct observations of people who had been in the field for decades, such as Bernard Rimland, Sally J Rogers, and Lisa Blakemore-Brown, who insisted that there had been a real increase of behaviours. And there were the statistics from surveys of autism prevalence, which showed sharp increases of numbers (and continued to do so).

In reaction against these reports, speculations were put forward by some observers, basically three in number: the increase could be due to (1) widening of the diagnostic criteria; (2) increased awareness; (3) diagnostic substitution (from "mental retardation" to "autism" or "ASD"). Or a combination.

The notion of substitution from mental retardation was argued for in a study by Croen et al., but subsequently Dr Croen ended up agreeing that substitution could not explain the increase of numbers. A decade later the substitution concept was revived in a paper by Polyak et al., whose graph features in Chapter 6 here along with my explanation of its gross incompetence and unsoundness. And autism is very unlike ordinary mental retardation, which is usually characterised by high empathy and sociability and low "cuteness".

The dismissal of the increase in terms of widening of diagnostic criteria is also unsound. A wider category of "autism spectrum disorder" does indeed now exist, but the increases have also been observed in respect of the two diagnostic concepts separately (Blaxill 2004) and with care taken to keep the criteria constant.

The remaining ground for doubting the increase, namely in terms of increased awareness, seems very credible at first glance for anyone who has not actually been involved in the field for more than a few years.

And yet on only slightly more reflection, that attempted explain-away in terms of mere increased awareness is laid bare as the most utterly absurd one, as shown in Chapter 12 here. That some prominent "experts" have resorted to such claptrap and are still doing so, is symptomatic of the forcefulness of the malign motivation of "do not admit to doing any harm", which was the first on my list above here.

In support of the official increase-denialism quackery there has been created a whole mythology of a fictional "lost generation" of older autistics, as discussed in Chapter 12. Under this crackpot pseudo-science, we are asked to believe that huge numbers of severely disabled children were somehow never noticed before and that all preceding generations of parents and pediatricians were grossly incompetent in failing to notice them. And that thousands of these non-verbal head-banging incontinents somehow managed to sneak successfully through the normal school systems and on into

normal employment. Autism is very different from ordinary low IQ.

Meanwhile a number of studies have confirmed the reality of the increase. These include Nevison (2014) and three of the studies she cites (namely Hertz-Picciotto et al. 2009, Mind 2002, and California 2002). And to all this evidence we can add the further observations I make in Chapter 3. There isn't really a scientific debate here, but something more like a corporate agenda in alliance with the fanatical ideology held by some very vocal "neurodiversity" advocates. A severely-flawed "award-winning" "best-seller" book, *Neurotribes* (featuring a promotional plug for Janssen's Risperdal®), has been heavily promoted recently. Its over-arching theme is a grossly one-sided mis-portrayal of the supposed non-increase.

Appendix to Chapter 2: More advanced understanding of how the processes of mutation are subject to natural selection

> "When you put English text into [the code], it generates very frequent stop codons in the genetic code and won't produce big proteins .... *It's designed to be biologically neutral*."
>
> – Nobel Laureate microbiologist Hamilton Smith

After I was mercury-poisoned by distinguished experts I became severely mentally disabled, and in subsequent decades other people then prevented me from continuing via the usual social means my education and research efforts. So I generally had to resort to working things out for myself, alone. This tended to involve the application of logic to facts. And oftentimes my personal conclusions turned out to be already confirmed findings of professional specialists. This can be seen in at least two instances in the theory paper presented in Chapter 7 here. Firstly, I perceived in autism the re-emergence of characteristics lost millions of years earlier, and this turned out to be a well-established phenomenon which biologists called atavisms. Secondly, I reasoned to the conclusion that there would be greater reliability of expression of more advantageous characteristics. And now, here in respect of my thoughts of those "evolving" Bechstein pianos we have a further example of such concurrence of my own naive inferences with specialist experts.

In my reasoning-obsessed worldview, it simply stands to reason that the cellular processes affecting mutation would themselves be subject to evolutionary pressure of "survival of the fittest"; and that this evolution of mutational processes would be such as to make deleterious mutations less easy to occur and advantageous mutations relatively more likely to occur; and that such evolution of the

mutational processes would be expected to occur in practice rather than just theory; and therefore most mutations would not be harmful, even though the grand evolutionary process overall would be like a randomly-driven "blind watchmaker" with no teleological guidance towards purposive ends.

With these ideas I enter into a hotly "controversial" area, because a great many people have learnt "the facts" in their university courses (as per an earlier chapter here), and don't see any merit in learning something which conflicts with them and so "therefore can't be true". (And those minds have been made even more rigidified due to their confrontation against creationist would-be-science.) But in recent years the field of study of genomes and evolution has become rather turbulent, because of the arrival of so many new facts enabled by the development of technologies for genome-sequencing and genome engineering. And I remind you of that quote from Max Planck:

> "…. A new scientific truth does not triumph by convincing its opponents and making them see the light, but rather because its opponents eventually die, and a new generation grows up that is familiar with it."

And in this case a lot of those funerals will be required. Because as a cheeky guy called Ron Maimon says (Maimon, 2013): "All these things are things that biologists got wrong, because they were going by stupid dogma." And: "The RNA "brain" is also making "knowing" modifications in the DNA through the action of reverse transcriptase."

Keynes famously said that politicians were the slaves of defunct economists. Likewise, most authors of medical research papers are slaves of defunct, superceded, genomics theorists. Not least with the simplistic theoretical notion that just about all mutations are nasty evil things creeping up to make you ill.

The understanding of DNA and mutations is currently undergoing a substantial revolutionary paradigm shift, from a simplistic flawed 20th-century model to a more complex more accurate 21st-century model. As is usual with these changes, those holding the outdated views continue to hold on to them and tend to be dismissive of those presenting the new understanding. So rest assured that some "leading" people will tell you that the following is just rubbish!

DNA and mutations are matters which just about every medical scientist has to learn about, even if they are not reckoning to be a cutting-edge genetic theorist themselves. It's hardly surprising that most scientists even including those working in genetics fields tend to be stuck with the outdated 20th-century model

they learnt at school, rather than having the most modern understanding.

Meanwhile some more words from Nobel laureate Werner Arber (2011) on the matter:

"....evolution genes. Some of their gene products act as variation generators, others as modulators of the rates of spontaneous genetic variation. ...." " .....increasing evidence .... indicates that principally the same natural strategies of genetic variation, local sequence changes, intragenomic DNA rearrangements, and DNA acquisition, are also in action in higher multicellular organisms. ..... one can postulate that all kinds of organisms living today on our planet earth dispose of a set of evolution genes that had become fine-tuned in the course of long periods of past evolution...." So you see that a Nobel prizewinner thinks alike with this Nobody no-prizes-winner.

This field is currently very much in flux due to new data coming from the improving technologies. But the newer thinkers are tending to reckoning like myself that the mutation profile of a species evolves to a steady state of *"balanced mutation"* with "a balance between slightly deleterious and slightly advantageous" (Razeto-Barry et al., 2012; Ohta & Gillespie, 1996). Like those Bechstein pianos.

Another analogy might be useful here. It is well-known (at least to experts in these things) that the southern end of the island called Great Britain is gradually sinking into the sea, at a rate of about five millimetres per decade. Plus the sea is gradually rising due to the melting ice caps. Meanwhile the tide goes in and out daily. And furthermore there are waves coming up the beaches, typically about 20 cm high and moving at walking speed. Now, aware of claims that England is progressively sinking below the waves, an ignorant amateur geologist might check by doing some measurements on a beach. He measures the rise of the water as a wave comes in. And wow!, the water rises 20 cm in less than a second. At that rate the whole of Norfolk and Kent will be drowned by tea-time!

People looking at data about mutations of DNA could be likewise assuming they are observing a whole picture of relentless long-term negativity of mutation when in reality all they are noticing is some more transient change, analogous to those waves coming up on a beach, without grim longer-term trend implications.

A fundamental problem with this mutation and parental ages research is that it is analogous to that silly geologist measuring the rising side of the wave but ignoring its falling side. Likewise these mutation researchers look at only one side of the matter, namely the negative effects (profitable for the drugs industry of course!). But

suppose (for mere sake of argument....) that I am a person who is exceptionally reasonable and patient, exceptionally slow to anger or take offense, exceptionally resistant to losing my head in traumatic circumstances, exceptionally capable of enduring and coping with decades of stressful adversity and sneering demeanment, and then reacting only with creative positivity and ingenious sense of humour.... (indeed a person such as described in Chapter 8 here?).... Perhaps thus we could say that Robin P Clarke has "mental superiority syndrome"** ("MSS"). Which might have been caused by some of those balancing advantageous mutations mentioned by Razeto-Barry a few paragraphs back. But who's doing any studies of the mutational or parental age correlates of my MSS? Precisely no-one. And such abnormally positive people are potentially of huge value to society (at least if it is capable of letting them be).

(** Or at least "mental conceit syndrome".)

And there is another way the researchers are only looking at one side of the matter, as follows. They look for de novo mutations occurring when a non-autistic parent has an autistic child. But they don't look to see whether there could be exactly as many de novo mutations occurring in the opposite event, when an autistic parent has a non-autistic child. Which is precisely what would be happening under the balanced mutation situation reckoned to be the reality by the new thinkers.

At this point it could be useful to recall that a key concept of the unfaulted and unrivalled antiinnatia theory of autism is that the genetic (and other) antiinnatia factors which contribute to "risk" of autism are basically the same ones that also contribute to "risk" of high IQ.

From that point of view, any de novo mutations which "cause" autism would also be causative of higher IQ (and genius) in other individuals. (An exception to this might be if there were some mutations with extra-large antiinnatia effect, just as while most potatoes are a sensible size for eating, a potato as big as a cow would be quite harmful to eat in one meal.)

Now here is where this subject gets quite amusing, at least for my own perverted sense of humour. At the same time as some researchers are reckoning to confirm that those nasty paternal mutations cause increased risk of autism (which I say is caused by *high* level of antiinnatia factors), concurrently some other researchers are reckoning to confirm that the nasty paternal mutations are also causing increased risk of .... lowered IQ (which I say is causally the exact opposite of the autism, caused by *low* antiinnatia instead). But "strangely" these researchers' research isn't going to plan! As follows.

Arslan et al. (2014) have recently done a study in this field which is particularly superior because it controls for the IQs of the fathers of the children (to rule out that as a possible confounder). Their report includes a good discussion of the theoretical issues, of which I will give a reasonably unmangled summary here.

They observe that intelligence is regarded as an attractive trait in mates across cultures, and also that it has had survival value in recent times. This leads to a question of why low intelligence has not become extinct, and why high intelligence has not become "fixated", by which they mean everyone now having the genes for maximal IQ.

On this question they point to the (mistaken) ideas of an earlier paper. Which (mistakenly) suggests that the reason why high IQ has not become fixated is that there is a substantial stream of unhelpful new mutations keeping the IQ low. And that there is consequently a *"mutation-selection balance"*, with new nasty mutations tending to make the population's IQ go down while *"directional selection"* tends to make it go up. (But all this is mistaken!)

I think what people are overlooking here is that the output of a human brain is subject to the quirks of a stupendous number of neuronal connections and those quirks are in turn dependent on a huge number of individual genes in each neuron and their varying expression under a huge number of varying conditions inside those neurons. Scientists who ought to know better assume the brain is some sort of amazing super-computer, whereas it should more reasonably be thought of as a would-be computer rather randomly emerging from lumps of not-entirely-organised meat. Computer chips are carefully designed by expert engineers, and formed of highly stable solids such as copper and silicon. By contrast brains are not designed but rather emergent from random mutations and blind selection, and are made from very unstable living cells. Information input to a computer does not change the hardware. But information input to a brain *does* change the brain's "hardware", in terms of new or altered synapses and so on (more than actually known at present). Computers are designed to be boringly logical. Brains are evolved to be seriously prejudiced.

Now here's the crucial problem for evolution. You can't evolve your way out of all that quirkyness of the brain functioning, because there are vastly too many genes and non-genetic factors involved. Even if you could de-select them from the cells of the inherited germline, there would still be many "somatic" de novo mutations among the billions of individual cells forming the brain.

There is a lot of potential for quirky error in the outputs of those neurons, or in other words in the gene-expressions of those neurons, and there needs to be some means for editing down their contributions to reduce the unhelpful rubbishy noise output. In Clarke (1993) it was argued that antiinnatia factors (including antiinnatia genes) would have their main function in suppressing such unhelpful error-causing expressions (which I called a class of "innatons" namely "IQ impairers"), and thereby tending to raise IQ. But these same antiinnatia factors would have the downside of also tending to suppress the advantageous innatons and making people a bit autistic-y. And that "but" is where these mutation researchers go wrong.

Sure, high IQ is an important quality in human affairs. But so have been "Sense and Sensibility", not to mention "Pride and Prejudice" and "Persuasion". Consequently these antiinnatia genes which these researchers are unwittingly studying are under evolutionary pressure from two directions. For any given environmental situation, there is an optimum level of antiinnatia: between being too retarded on the one hand and being too geeky on the other. And so the genes for IQ are not under that "directional selection" leading to "mutation-selection balance", but instead under something more like "stabilising selection".

Arslan et al. (2014) say of this stabilising selection that it: "leads to a buffering against both deleterious and beneficial changes (robustness)" and that "higher robustness would imply smaller effects of new mutations".

Which brings us back to the concept of "balanced mutation", which I mentioned earlier is becoming the new main paradigm of mutational change. And back to my thoughts about the Bechstein pianos.

Meanwhile, poor old Prof Arslan and colleagues were still thinking along the old lines, so they were expecting their results to confirm the "mutation-selection balance" assumptions instead. Quite often, when scientists get "wrong" results they are very embarrassed about it, and fearful that they will be laughed at and even demoted for their "incompetence". So they tend to squirm about with their study, briefly mention the "wrong" thing only on page 7, or hide it away altogether. But to their credit the Arslan team didn't, but instead just stated their surprise at their result:

> "We did not find support for our hypothesis that higher paternal age at offspring conception, as an indicator of more new, harmful mutations, would predict lower offspring intelligence."

Which is exactly what you would expect if those mutations are analogous to those waves going both up *and down*, rather than to a one-way trend threatening to drown the entire populations of Canterbury and Cambridge before the end of the day.

In summary about the Arslan et al. study, they ended up confirming my own theory of the genetics of IQ even though they didn't even know it existed and they were expecting to prove the opposite anyway. Thus so much more becomes clear once you dump the evidence-defying parrotting of "autism is a disorder".

Meanwhile, those older parents are also survivors of many more years of natural selection, to the extent that the positive selection from that surviving could entirely cancel out any deleteriousness from some of the added mutations. If older paternity really was very disadvantageous one would expect that it would have been naturally selected away millennia ago, with human paternity starting and ending at the same age as for horses and dogs.

We should also bear in mind the fallacy of averages. It is a standard dogma of corporatised medical propaganda that large studies of thousands of people are the best form of evidence, while the wonderful experience of Fred after taking some herbal tablets bought by his mom is supposedly no evidence at all. These studies of increasing parental age fall into this same fallacy of averages. Sure, as Mr Average gets older he is more likely to need a walking stick and to get cirrhosis of the liver. But those of us who don't drink alcohol and who take (expert, haha) care of our bone nutrition are not Mr Averages and those results have no relevance to us. Many older people have not so much spent x years alive as having spent x years living self-abusively in various unhealthy ways almost guaranteed to damage their children, but nothing to do with age per se.

And the list of other conditions most asserted to be associated with parental age is notable. Schizophrenia commonly begins around the time of becoming adult, and hence can prevent a young man from becoming registered as a young husband. But thereafter a proportion recover or become more stable, so the former schizophrenic may then become one of those "older fathers" which the researchers are finding. And again, in respect of manic-depressive illness, that condition has been notably associated with valuable creativity and higher social class, which again is associated with older parenthood, with no need for adverse mutations to be involved.

If later paternity were really genetically harmful, as per my silly mutilations of the Mona Lisa, then we would expect the

resulting children to be weak, sickly, stupid and cancer-ridden. But they aren't. A review by Tournaye (2009) concluded that the absolute risk of genetic anomalies from older paternity is low, and that "there is no clear association between adverse health outcome and paternal age".

And finally, any genetics which ignores the context of changing environmental factors is half-baked. A hugely important factor is dental mercury. In Chapter 3 here I show how just about all of this autism, schizophenia, manic depressive, and more, can be accounted for as the consequence of the introduction of dental amalgam in the 19th century followed by its "improvement" from the 1970s with the even worse non-gamma-2 amalgams. Get rid of that dental mercury and just about all this disability caused thereby disappears, nothing whatsoever to do with genes being harmful per se, but merely genes conceivably making a person vulnerable to an abnormal environment which would not be there anyway if fewer "distinguished experts" were liars.

The bottom line here is that the proper understanding of the genome and mutations does not at all correspond with the still-predominant assumptions of the outdated simplistic model. And too many people in autism genetics research are assuming that it does.

[P.S.: Ruben Arslan has commented: "You refer to me as "poor old Prof Arslan". I'm neither a Prof nor a Dr, I'm still a PhD student. The "poor" is quite right though." However, the very next month I noticed some other research of his reported in the *New Scientist*. Some "student".

He also informed me that that Arslan et al. 2014 "wrong" result has since been confirmed by "a much bigger study (D'Onofrio et al., 2014)...."]

(The main text of this chapter continues back at page 60.)

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