

***A sample including the
Prefaces, Chapter 1, Chap-
ter 2, and References taken
from,***

A Hole in Science:

**An Opening for an
Alternative Understanding of
Life**

By Ted Christopher

Preface to the 2016 (2nd) Edition

A number of sentences in the original edition of this book were cleaned up some for this edition. Also some significant changes to portions of Chapter 6 and Chapter 7 were introduced. The book is still quite conceptual in a number of places - including Chapter 1 - and in combination with a somewhat terse writing style, that encourages slow reading.

Preface

The modern scientific understanding of life is built upon the belief that all features of life - including of course consciousness - are completely describable in terms of molecules and their activities. From this perspective, living beings can be viewed as simply constituting a particular subset of the material universe, and as such are presumably constrained by the same laws of physics and chemistry. This material-only hypothesis is usually referred to as scientific materialism or materialism, and it is essentially a modern intellectual fixture. The corresponding blueprints for organisms are of course believed to be given by a particular large molecule - deoxyribonucleic acid (DNA).

A couple of preliminary orientation notes. Hereafter the term “materialism” (or one of its synonyms) will only refer to the belief in the material-only nature of life (not the more general hypothesis about the universe). A second orientation note is that the focus herein will be primarily on humans, with secondary consideration given to other animals.

Now with regards to scientific materialism there are two basic points presented in this book. The first is that materialism has a big hole in it. This gap is centered on the unfolding “absolutely beyond belief” failure of DNA to determine some basic innate characteristics of individual humans and this possibility was apparent before the frustrating follow-up to the Human Genome Project. The second point is that this hole appears to be consistent with the common premodern transcendental understanding of life (“transcendental” will be used herein in place of a number of existing terms including “transmigration” and “reincarnation”). Starting with this preface I will lead with the first set of arguments and then follow up with some possible transcendental-based explanations. At a minimum I hope to get readers to see some serious challenges facing science’s material-only vision, and also to encourage them to consider alternatives.

I get the ball rolling by taking a quote from Sam Harris' *Free Will* in which he pointed out that:

And now your brain is making choices on the basis of preferences and beliefs that have been hammered into it over a lifetime - by your genes, your physical development since the moment you were conceived, and the interactions you had with other people, events, and ideas [Harris S. 2012, p.41].

Harris' message naturally flows from the materialist model. From that model’s perspective, you are lucky to be alive given your dependence upon a very unlikely conception that produced your particular DNA blueprint, a blueprint that as Richard Dawkins put it, "created [you], body and

mind" [Dawkins, p.20]. Continuing, that DNA blueprint (including the gene portions), defined your "nature" component and your subsequent environmental exposures formed your "nurture" component. From materialism's viewpoint that nurture dynamic was essentially updating or refining your innate programming. Thus, the material-only entity that is you; your particular behavior; and of course as a bio-robot, lack of free will. The particular molecules that constitute you just carry out their molecular activities and thus Harris can simply characterize you as a "biochemical puppet". Some of the potential significance of this materialist's perspective was made apparent in a June 2014 *Scientific American* article that pointed out some of the ways in which "skepticism about free will erodes ethical behavior" and how this could conceivably culminate in societal "anarchy" [Shariff and Vohs].

Now compare the previous quote with the following description of a musical prodigy found in Darold A. Treffert's *Islands of Genius*:

By age five Jay had composed five symphonies. His fifth symphony, which was 190 pages and 1328 bars in length, was professionally recorded by the London Symphony Orchestra for Sony Records. On a *60 Minutes* program in 2006 Jay's parents stated that Jay spontaneously began to draw little cellos on paper at age two. Neither parent was particularly musically inclined, and there were never any musical instruments, including a cello, in the home. At age three Jay asked if he could have a cello of his own. The parents took him to a music store and to their astonishment Jay picked up a miniature cello and began to play it. He had never seen a real cello before that day. After that he began to draw miniature cellos and placed them on music lines. That was the beginning of his composing.

Jay says that the music just streams into his head at lightning speed, sometimes several symphonies running simultaneously. "My unconscious directs my conscious mind at a mile a minute," he told the correspondent on that program [Treffert 2010, pp.55-56].

Treffert's book contains a number of other examples supporting his conclusion that prodigal (including prodigious savant) behavior typically involves "know[ing] things [that were] never learned". Such behaviors provide clear rebuts to the materialist vision and thus Harris' statement about free will. Treffert also considered the phenomenon of acquired savant syndrome in which savant behaviors appear in the wake of central nervous system setbacks. Needless to say, it is unlikely that puppets (or robots) would acquire skills as a result of physical damage.

An under-appreciated problem for the carved-in-scientific-stone, materialist vision is that there have always been counterexamples available in the form of unusual (and noncontroversial)

behavioral phenomena. Additionally monozygotic twins - with shared DNA blueprints - are far too different. For example, take Harris' logic and then consider that with very similar environments and the same DNA, it turns out that if one monozygotic twin is gay then the likelihood that his twin brother will also be gay is only 20-30% (ironically found in Francis Collins' *The Language of Life: DNA and the Revolution in Personalized Medicine* [pp.204-205]). This is an example of the large differences found between monozygotic twins, differences which suggest a behavioral mystery potentially affecting all of us and which led Steven Pinker to acknowledge, "something is happening here but we don't know what it is" [Pinker 2002, p.380].

The big question, though, is how well can science in general confirm their Nature-plus-Nurture, material-only understanding of life? In September 2008, the geneticist David Goldstein (then at Duke University) was quoted regarding the outcome of thorough comparisons between the million or so common genetic (or DNA) variations and the apparent inheritance patterns associated with the occurrences of common complex diseases [Wade 2008]. It had naturally been assumed that some of these common variations amongst our DNA blueprints would be correlated with the patterns of susceptibility to common diseases (and of course to other innate differences between individuals). This assumption was also bolstered by estimates that very little DNA - perhaps 0.1% or about 3 million nucleotide elements - appears to differ between any two humans [Schafer; Green; Kingsley]. Somewhere then amongst this small subset of variable DNA there should be the origins of our innate differences, and this assumption is the basis of the fields and big expectations of personal genomics and behavioral genetics. But Goldstein pointed out that:

[a]fter doing comprehensive studies for common diseases, we can explain only a few percent of the genetic component of most of these traits. For schizophrenia and bipolar disorder, we get almost nothing; for Type 2 diabetes, 20 variants, but they explain only 2 to 3 percent of familial clustering, and so on.

Goldstein then added:

It's an astounding thing that we have cracked open the human genome and can look at the entire complement of common genetic variants, and what do we find? Almost nothing. That is absolutely beyond belief.

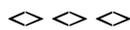
Subsequently, in 2011 the director of the Bioscience Resource Project, Jonathan Latham, offered his own assessment:

The most likely explanation for why genes for common diseases have not been found is that, with few exceptions, they do not exist ... The likelihood that further searching

might rescue the day appears slim. A much better use of the money would be to ask: if inherited genes are not to blame for our commonest illnesses, can we find out what it is [Latham]?

This surprising DNA deficit has been reflected not only in the lack of DNA breakthrough-headlines but also in the extraordinary failure of the associated biotechnology industry [Sheldrake, pp. 168-171].

Nonetheless confident news may continue - such as with the junk DNA headlines of September 2012 - but significant findings with regards to our individual inheritances are still missing (the junk DNA news bonanza was later characterized by neurobiologist Athena Andreadis as a big “orchestrated PR campaign” [Andreadis]). Thus, in a 2012 blog contribution, geneticist Kevin A. Mitchell acknowledged that a “debate is raging in human genetics” over this missing heritability problem [Mitchell 2012/02]. This under-communicated scientific impasse offers a fundamental challenge to materialism, an understanding which might simply have been questioned based on some behavioral enigmas.



The second argument in this book can be approached thru findings from studies of the natural religious or spiritual understanding of young children. In the book, *Born Believers - The Science of Children's Religious Belief*, Justin L. Barrett laid out some of the growing evidence that infants tend to possess an innate understanding of the existence of souls/God/gods, to be believers in what Barrett termed a “natural religion” [Barrett]. The book contained some striking examples including ones in which the positions of atheists’ were rebutted by their young children. As Barrett wrote “[c]hildren are prone to believe in supernatural beings such as spirits, ghosts, angels, devils, and gods during the first four years of life” [p.3]. He later added:

Exactly why believing in souls or spirits that survive death is so natural for children (and adults) is an area of active research and debate. A consensus has emerged that children are born believers in some kind of afterlife, but not why this is so [p.120].

These striking findings were simply placed within science’s vision, though. Barrett, even as a practicing Christian, concluded that these are simply delusional tendencies derived from evolution and nurture - “biology plus ordinary environment”. How our evolution-formed DNA blueprints could have resulted in such beliefs appears to be quite a mystery, though. At the beginning of his book Barrett did offer an alternative explanation that had been provided confidently by an Indian man he encountered on a train. In Barrett’s words the man had explained:

[T]hat on death, we go to be with God and are later reincarnated. As children had been with God more recently, they could understand God better than adults can. They had not yet forgotten or grown confused and distracted by the world. In a real sense, he explained, children came into this world knowing God more purely and accurately than adults do [p.2].

Some of the possible implications of that transcendental view will be explored herein, in particular in the context of the growing mystery of the origins of our innate individual specifics. This view appears to have been a common premodern understanding as described in *M'Clintock and Strong's Cyclopaedia of Biblical, Theological and Ecclesiastical Literature*, “[t]ransmigration, dating back to a remote antiquity, and being spread all over the world, seems to be anthropologically innate, and to be the first form in which the idea of immortality occurred to man” [Head and Cranston, p.170]. This belief has two aspects, the intuitive continuity of behavior/personality part and the much more puzzling cause-and-effect (or popularly “karma”) part. Of these two logically distinct hypotheses it has been claimed that they were historically “in fact ... virtually always conjoined” [Head and Cranston, p.10]. Perhaps the apparent continuity of personalities across lives in small and undistracted populations initiated and then amplified the credibility of the continuity belief. Additionally, perhaps the karma hypothesis then followed on occasion from observing an individual encountering their apparent just deserts across lives. In a personal sense, a transcendental understanding would assert that at a core or foundational level, each of us is a non-material self or soul - a center of consciousness - which in the long run travels back and forth between embodied and disembodied existences (and thus transcends any particular one), and further that there is some continuity between these sequential existences.

In this book I will argue that in addition to offering a straightforward explanation for our natural religion, a transcendental perspective also provides traction on some scientific conundrums including prodigies, transgender individuals, and the surprising variations in personality found amongst a number of species; a simple explanation for the mysteries associated with monozygotic twins; a backdrop for some controversial phenomena including near-death experiences; and finally a consistent framework for the missing heritability problem. In brief, the missing origins for a number of our innate individual specifics could be understood as carryover from previous lives and with some standout behaviors - as with prodigious savants and prodigies - there could be some additional carryover consistent with some of the remarkable descriptions of the intervening disembodied state.



This book serves the Religion-versus-Science debate by offering counterarguments to science's material-only, bio-robotic vision of life. In so doing it helps open a door to alternative understandings. In exploring some potential explanations provided by the premodern transcendental understanding I consider evidence for a transcendental soul and in so doing offer some bottom-up support for what I think is a religious perspective. On the other hand, the usual top-down approach of pursuing objective arguments for the existence of God is apparently very difficult. Furthermore, even if someone succeeded - perhaps with a physics-based effort like *The God Theory* [Haisch] or an evolution-required-some-intelligent-intervention effort like *Darwin's Doubt* [Meyer] - how would that ultimately change science's puppet-like vision of you and your life? A chapter taking a uniquely critical look at both religion and science is included.

The discussions in this book can also be seen as complementary to some of the established approaches to breaching scientific materialism. A sampling of these established efforts include investigations of extrasensory perception [Tart; Radin]; possible cases of reincarnation [Stevenson; Tucker 2005 and 2015]; near-death experiences [Holden et al; Alexander]; the totality of psychological challenges as chronicled in a thorough text like *Irreducible Mind* [Kelly et al]; and also more broadly-based challenges to materialism [Sheldrake]. This book on the other hand, focuses on materialism's inheritance problem along with some consistent explanations available with the common premodern transcendental understanding. Some possible reincarnation cases will be used herein, though, as they offer some support for general transcendental hypotheses.

The perplexing range of personalities found among people and other species; the surprising differences found between identical twins; and also our innate religious inclinations; all challenge scientific materialism. Together with some behavioral conundrums and the unfolding "beyond belief" missing heritability problem, it is time to question the completeness of biology's view of life. In *The Sacred Depths of Nature*, this view was confidently characterized by the author/biologist Ursula Goodenough, as "relentlessly mechanical", "bluntly deterministic", and by extension without free will [Goodenough, pp.46-47]. As will be shown herein, though, contradictions to this view are easy to find.

Chapter 1 - The Quietly Unfolding Missing Heritability Problem

Arguably the ground floor of the modern intellectual point of view is science's material-only understanding of life. With the possible exception of those making inferences drawn from near-death experiences or parapsychological studies, the modern mechanistic paradigm enjoys thorough intellectual confidence. It would be a challenge to try to identify academic efforts in the last 50 years that have questioned this ground floor of science's vision, perhaps in particular that our deoxyribonucleic acid (DNA) "created us, body and mind" and therefore that our individual existences came against gargantuan odds. This view of DNA is made explicit in the title of Francis S. Collins' 2010 book about DNA, *The Language of Life*. This vision of life is of course nested within the modern scientific vision of the universe - vast and meaningless.

Some DNA Basics

Before considering some recent efforts to identify the DNA origins of particular characteristics of individuals, it is worth considering some relevant aspects of the DNA landscape. I start here with a somewhat detailed example to warm up to some evolutionary dynamics of DNA and their constituent genes. A relatively concrete and significant example of those dynamics was the development of color vision in our primate ancestors. As a result of that development humans and a number of other primate species differ from most mammalian species in our increased capacity to distinguish the visual spectrum. In particular, our eyes' retinas come equipped with three distinct visual pigments, one of which responds strongest to short-wavelengths of light and the other two respond strongest to longer wavelengths. Those responses of the three pigments correspond to the colors blue, green, and red, respectively. With their differing spectral sensitivities, these pigments together provide the necessary input so that our brains can provide relatively good color vision. Details of how the brain's basic visual processing occurs are still being worked out, but the evolutionary development and significance of our trichromatic vision (or trichromacy) appears to be understood. The relevant source here - a fine *Scientific American* article, "The Evolution of Primate Color Vision", by Gerald H. Jacobs and Jeremy Nathans - included a revealing picture of a frog as our trichromatic vision would see it and another from the corresponding typical mammalian dichromatic viewpoint [Jacobs and Nathan]. The article also pointed out that a consequence of our trichromatism "is that computer and television displays can mix red, green, and blue pixels to generate what we perceive as a full spectrum of color".

A pigment consists of a light-absorbing molecule derived from vitamin A together with a protein bearing a particular sensitivity to optical or light stimulation. Such pigments when housed in one of our retinas' cone cells can be stimulated by some light and that response is then relayed to the brain for producing an image. The specification for a visual pigment's protein is found in the DNA. It appears that the DNA specification for our shorter-wavelength pigment protein is essentially shared with many other vertebrates. Thus it appears that this DNA protein-specification (or gene) apparently had its origins a long time ago in evolutionary history.

It is with the DNA specifications for our two long-wavelength pigment proteins that some interesting history and the specific origins of our color vision can be found. These two genes - and the resulting pigment proteins - are very similar, and in fact, the two proteins differ in only 3 out of their constituent 364 amino acid elements. Many other vertebrates also have a single visual pigment similar to these but somehow in evolutionary history one or more primates got an update, an almost-duplication, resulting in a second long-wavelength pigment specification and with it an opportunity for improved color vision.

The initial step towards our trichromacy appears to have occurred over 40 million years ago in some primate ancestors and involved successive changes or mutations in the original long-wavelength pigment gene. These mutations left these primate ancestors with three variations (or alleles) amongst their long-wavelength gene which was located on their X chromosomes. Since there was only one such specification per X chromosome, though, the males in this group (having only one X chromosome) were still limited to two color-sensitive retinal pigments and thus some form of dichromatic vision. Some of the females, on the other hand, were fortunate in that their two X chromosomes bore different long-wavelength pigment genes and thus they experienced trichromacy. This original gender-dependent step towards trichromacy is still present in the New World primates of South and Central America.

After the separation of the New and Old World primates (via the moving apart of South America and Africa) about 40 million years ago, there appears to have been a rare error in the production of a subsequently fertilized egg cell within the Old World primate lineage of Africa and Asia. That error apparently occurred in the chromosome-swapping (or recombination) process used to form an egg cell and occurred in a female with DNA that bore two different long-wavelength pigment genes. The particular error apparently left two distinct long-wavelength genes on one X chromosome. Thus, that resulting egg cell's DNA became a ticket for its offspring to possess trichromatic vision regardless of their gender.

The resulting vision update was likely helpful to those primates including with their efforts to distinguish ripe fruit. Over subsequent generations then this dual long wavelength gene package spread widely to provide the trichromatic vision that is now standard equipment amongst Old World primates including humans. The original arrangement with only one longer-wavelength pigment gene on the X chromosome would have fared poorly under natural selection, and thus was eventually lost from the Old World primates' gene pool.

The evolutionary dynamic exhibited by this development of trichromatic vision in Old World primates was perhaps somewhat complicated with its sequence of mutations followed by a recombination error. But the underlying changes over time in DNA blueprints and then the subsequent response of natural selection - in this case positive selection - was not unusual. Thus as is currently understood, by happenstance a gender-independent trichromatic DNA specification was come upon long ago in the Old World primate lineage and it was a natural selection-winner so that over subsequent generations that trichromatic specification became the norm. (A subtle point being glossed over here is that the mammalian brain - as demonstrated in experiments with mice - apparently can readily incorporate the additional input of a second long-wavelength visual pigment and thus utilize a trichromatic opportunity).

What is of general note here is the very elemental, undirected, and long term nature of the color vision evolutionary process. Another example of such a process can be found with the receptors responsible for our sense of smell which were cumulatively built over time and involved about a thousand genes. Each of those genes was acquired during evolutionary history and each produces a distinct protein which is used as chemical receptor in our nose and then also as a guide to ensure that the corresponding neural connection is correctly made in our brain [Pinker 2002, p. 93]. Like the addition of the third visual pigment gene, the additions of each of these smell receptor genes was likely helpful in a reproduction-and-survival sense (perhaps in helping to distinguish a threat), and thus spread widely.

One additional complication associated with the genetics of our color vision, though, is that it is imperfect. Most significantly, there is some evolution-given variation in the two long wavelength genes found within the human genome. As a result of that variation about 1 in 12 males of European ancestry suffers from some form of deficiency in their ability to distinguish within the red-to-green light spectrum. Because these genes are located on the X chromosome, females are less likely to experience this condition and for the corresponding female population the deficiency figure is only about 1 in 200 [Columbia, p.626]. For other groups the occurrence rates appear to be lower. This second genetic dimension, involving DNA defining the differences between indi-

viduals within a species is of course important in and of itself, and is also fundamental to the ideas considered within this book.

Some other examples of DNA's confirmed individual import were given in David M. Kingsley's *Scientific American* article, "From Atoms to Traits" [Kingsley]. Kingsley pointed out several physical traits and their confirmed DNA origins. Sometimes these origins simply involve single element (or letter) changes in the DNA as in the case of short-versus-tall pea plants. In another example it entailed a big singular change involving the substitution of an 800-base-pair sequence into a gene of a pea plant resulting in wrinkled pea skins in place of smooth ones. The effects of these DNA variations had been noted by Gregor Mendel in the mid-nineteenth century. Along these lines Kingsley also pointed out that:

bigger muscles, faster running ability or improved ability to digest new foods have all arisen from simple new arrangements of atoms in the DNA sequence of ... dogs and humans.

Amongst physical traits the DNA connection is increasingly being confirmed.

But it is also worth noting, though, that how an elemental DNA dynamic - like that found in our color vision history - could have formed something like complex instinctive behavioral tendencies is not easy to see. As Rupert Sheldrake pointed out:

[g]enes are not selfish and ruthless, as if they contained gangster homunculi. Nor are they plans or instructions for organisms. They merely code for the sequences of amino acids in protein molecules [Sheldrake, p.163].

Nonetheless, a number of species exhibit an unlearned or instinctive fear of snakes and it is a challenge to imagine discrete changes in the DNA code resulting in an automatic ability to become alarmed over the sight of a snake. Strongly detecting a particular frequency of light is a bit of a one-dimensional challenge for evolution, but detecting the visual form of particular objects is much more difficult. As non-infants most of us effortlessly see a visual field full of particular objects (including trees, dogs, roads, garbage cans, and clouds), and that process is built upon our past efforts to learn the appearances of those objects.

Continuing with a behavioral commentary, the particular context for humanity's mostly-mental evolutionary trek was nicely framed by Steven Pinker when he wrote:

[o]ur minds are adapted to the small foraging bands in which our family spent ninety-nine percent of its existence, not the topsy-turvy contingencies we created since the agricultural and industrial revolutions [Pinker 1997, p.207].

Further, he characterized the corresponding environmental selection pressure as “a camping trip that never end[ed]” [Pinker 1997, p.207] Very little about this evolutionary scenario, though, appears to suggest that there could be genes - or more generally DNA - making substantial contributions to complex modern behaviors like playing the piano, being a Democrat, being adept to the demands of the Industrial Revolution, or being a natural whiz at calendar calculations. Thus, the associated DNA-based logic of behavioral genetics would appear to be a challenge for the underlying elemental nature of evolution. Nonetheless, science has put its faith in the individual-defining import of DNA. As a frank testament to this faith, Steven Pinker confidently pointed out that:

[i]dential twins separated at birth [with maximal DNA similarity and gross environmental differences] share traits like entering the water backwards and only up to their knees, sitting out elections because they feel insufficiently informed, obsessively counting everything in sight, becoming captain of the volunteer fire department, and leaving little love notes around the house for their wives [Pinker 1997, p.20].

Such optimism about DNA’s behavioral impact appears to be commonly assumed and thus the cliché “it’s in the DNA”.

Another optimistic assessment was made by an editor of *Nature* who proclaimed that in the coming century:

[g]enomics will allow us to alter entire organisms out of recognition, to suit our tastes ... [and] will allow us to fashion the human form into any conceivable shape. We will have extra limbs, if we want them, and maybe even wings to fly [Nature 1997].

Putting aside such opinions, a number of basic assumptions about DNA’s role are being tested thoroughly as (existing) DNA specifications are being compared against the corresponding individual outcomes.



A return to one of the Preface’s quotes by Duke University’s geneticist David Goldstein is good place to embark on an overview of the missing DNA situation. In that September 2008 *New York Times* article Goldstein was quoted on the outcome of thorough efforts to try to identify the commonly occurring genetic variations (or common variants) which were presumed to contribute to the inheritance patterns found amongst the occurrences of common complex diseases like diabetes, cancer, heart disease, and mental illnesses [Wade 2008]. Part of the logic of this effort is

reflected in the routine medical practice of asking patients about possible family histories of complex diseases since such diseases do tend to run in families. On the DNA side, the particular variations considered were single letter or nucleotide sites that commonly differ amongst individuals and also tend to coexist with larger variations at nearby gene portions of the DNA. These single nucleotide variations are called SNPs (“P” is for polymorphisms) and they tend to flag potentially significant changes in a nearby genes. But after thorough studies Goldstein concluded that:

It’s an astounding thing that we have cracked open the human genome and can look at the entire complement of common genetic variants, and what do we find? Almost nothing. That is absolutely beyond belief.

This was a rare frank assessment of the failure of the initial big scientific push to establish the “miracle” of personalized medicine via identifying the underlying DNA variations believed to be responsible for the common complex disease susceptibilities.

In October 2010 came another sober report from the personalized medicine front in an article in *Scientific American* entitled “Revolution Postponed” by Stephen S. Hall [Hall]. By this time Goldstein had some company in his negative assessment of the common variants hypothesis (“common” usually implies that a particular DNA code variation is present in at least 5 percent of humans), although the lack of positive connections notwithstanding, there were other geneticists still confident in the original hypothesis. The article touched on the significant rift existing between the two camps of geneticists. The alternative approach of others including Goldstein was then to pursue the complementary rare variants hypothesis and with it to assume that in a relatively short evolutionary period that natural selection had successfully weeded out the unhealthy common DNA variations present in the human gene pool. Commonly occurring disease-prone designs were thus assumed by this rare variants camp to have been consistent natural selection failures and thus lost in our reproductive history.

Amongst those resigned to the failure of the original common variants hypothesis, was Walter Bodner “who was among the first to propose the genome project in the 1980’s and is a pioneer of the association studies that have dominated recent genomics”. Bodner claimed that “[t]he vast majority of [these common] variants have shed no light on the biology of diseases” and also that the associated search was “scientifically wrong”. David Botstein of Princeton University called the underlying search for SNPs (specifically the HapMap project) a “magnificent failure.” And in the summer of 2010 Goldstein had said of the common variants hypothesis, “[w]e have entered and left that field, which explained less than a lot of people thought it would.”

In the “Revolution Postponed” article the remaining supporters of the common variant hypothesis mentioned included Eric S. Lander of the Broad Institute at M.I.T. Lander claimed that over the previous three years findings into the genetic origins of diseases had been “mind blowing” and further that “we haven’t even scratched the surface of common variants yet.” His take was that the common variants hypothesis would eventually pan out. Another prominent supporter cited in Hall’s article was Francis Collins, head of the National Institute of Health, who had claimed on an appearance on PBS’s *The Charlie Rose Show* that via some new common variants findings “we have ... already ... change[d] our entire view of how to develop new therapeutics for diabetes, for cancer, for heart disease”.

The gist conveyed in Hall’s article, though, was of very limited success up to that time from the common variant-based genome studies. One enumerated example given was for type 2 diabetes. According to David Goldstein for this condition the:

association studies analyzing 2.2 million SNPs in more than 10,000 people have identified 18 SNPs associated with the disease, yet these sites in total explain only 6 percent of the heritability of the disease - and almost none of the causal biology.

The closing reference to causal biology is a practical point since understanding this is viewed as essential to developing medical treatments to combat the disease susceptibilities. With the above numbers one might wonder about the statistical creditability of finding a 6 percent contribution distributed amongst 18 contributors (from amidst 2.2 million possible contributors). Those are pretty thin slices of the heritability pie and thus perhaps the worries of some researchers that these thin slices were really statistical illusions [Wade 2010].

Perhaps due to the solid logic behind it and also substantial investigative momentum, the common variants hypothesis subsequently extended itself into a search for “a large number of small-effect common variants” [Gibson]. The idea here was to try to find genetic causation through the sum of many small common variant contributions. Given the lack of significant findings reported in the subsequent years, though, it appears that neither of the common or rare variant camps (or variations thereof) has found much. And thus as mentioned earlier, a 2012 blog contribution by a neurogeneticist from Trinity College Dublin, Dr. Kevin A. Mitchell, acknowledged that:

[a] debate is raging in human genetics these days as to why the massive genome-wide association studies [GWAs] that have been carried out for every trait and disorder imaginable over the last several years have not explained more of the underlying heritability [Mitchell 2012/02].



Now stepping back from the DNA heritability search front, it is worth considering some more of the DNA landscape. In a May 2009 *Scientific American* article by Katherine S. Pollard entitled “What Makes Us Human?” the initial findings of efforts to determine the DNA differences underlying the species divide between humans and chimpanzees was presented [Pollard]. Pollard pointed out:

[a] humbling truth emerged: our DNA blueprints are nearly 99 percent identical to theirs. That is, of the three billion letters that make up the human genome, only 15 million of them - less than 1 percent - have changed in the six million years or so since the human and chimp lineages diverged.

Pollard then went on to point that:

[e]volutionary theory holds that the vast majority of these changes had little or no effect on our biology. But somewhere among those roughly 15 million bases lay the differences that made us human.

An additional point could well have been made here and it is that somewhere amongst this inter-species defining collection of DNA letters (or nucleotides) there should also be a subset that is very relevant to the differentiating of our individual inheritances. This would seem to be particularly true for mental inheritances since our large brains are our big evolutionary distinction and as such were central to Pollard’s article.

The above point about evolutionary theory is also significant to the basics of the dynamics of DNA. That theory asserts that the process of genomic change is in large part a haphazard one, as one evolutionary biologist, T. Ryan Gregory of Ontario’s University of Guelph, stated, “[a]t its most fundamental level, it’s a mess”. This quote was given in an aptly entitled *New York Times Magazine* article, “Is Most of Our DNA Garbage?” [Zimmer 2015]. Thus in addition to functional dynamics similar to the previous ones dealing with vision and smell, there are also plenty of minimally functional dynamics too. Thus in a gross sense you can find relatively simple species like an onion or a broad-footed salamander with genomes far larger than those of humans - five and fifty times larger, respectively. From this perspective it appears that much of any species’ genome - including ours - is simply “junk” or noncoding letter sequences that just happened to have hitched a ride on the DNA. It also appears that in some cases junk code segments had previously lost their functional or coding status via mutations during evolutionary history but nonetheless stayed on board the DNA. In a bottom line quote, the evolutionary biologist Gregory

could assert that only “8 percent, plus or minus 1 percent” of the human genome is likely to be functional, and that the remainder, “doesn’t seem to matter that much”.

Finally, on the most relevant point from the DNA landscape, it turns out that *Homo sapiens* have been referred to by geneticists as a “small species” since there is relatively little genetic variation amongst us, and such limited variation is typical of a species with a small population [Pinker 2002, p.142-143]. That lack of genetic variation apparently followed from our having literally been a small species not too long ago as we survived through a period in which our population became tiny. An insufficient amount of time - evolution-wise - has since elapsed for that limited pool of DNA variation to expand much (unlike our population). Thus again, some estimates have it that any two human beings are about 99.9% identical in terms of their DNA blueprints, which translates to being different in about 3 million bases or letters [Schafer, Green, Kingsley]. It is also worth noting that even amongst this 0.1% variable portion of our genomes, there could be plenty of irrelevant junk. Thus, amongst the often cited three billion nucleotides figure there would seem to be a pretty small subset of our DNA, in particular amongst the variable portion, that should be home to our missing heritability. This was likely relevant to Goldstein’s “absolutely beyond belief” characterization in 2008 of the initial failure of the common variants hypothesis.

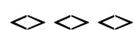


Now moving on for a focused behavioral look at the state of the heritability situation, it is worth considering the search for the origins of the variation in human intelligence. An October 2008 *Scientific American* article “The Search for Intelligence” offered this, as well as providing some context for intelligence which is arguably the biggest item on the behavioral genetics queue [Zimmer 2008]. The common variants hypothesis had again not panned out as it only appeared to offer a possible 0.4 percent explanation for the heritable - and thus presumed genetic - differences found amongst the intelligence scores considered. The article included University of Virginia psychologist Erik Turkheimer bolstering our intuitions in pointing out that intelligence “[i]s something that everybody observes in others” (unlike disease susceptibilities) and further that “intelligence and intelligence test scores are in many ways the best predictor in all of psychology”. Intelligence, or perhaps more specifically academic intelligence, is certainly a big deal in modern societies. In the article it was pointed out that there appear to be neural (or brain) correlates associated with the variations in intelligence. Thus, perhaps in the future there could be a brain-imaging alternative to IQ tests. These could offer visual evidence letting you know how smart you are or should be and, in so doing, sidestep complications with regards to test-taking vagaries.

Moving on four years and that search received an update in an article by David Dobbs in which he reported on findings that had apparently more than doubled the DNA origins identified to 1 percent [Dobbs]. Some might argue against the ultimate significance (or survivability) of that 1 percent but even an insider like psychologist Robert Plomin of King's College London sounded resigned to the situation, "[i]f it's this hard to find an effect of just 1 percent what you're really showing is that the cup is 99 percent empty". Needless to say this is very surprising conclusion from the scientific perspective.

The same Dobbs article described a theory accounting for the apparent lack of a DNA basis for the variation in the innate portion of IQ. The geneticist Dr. Kevin Mitchell, had proposed in a July 2012 blog effort that we should consider flipping the intelligence heritability logic around [Mitchell 2012/07]. Instead of looking at the genetics of intelligence we might consider high intelligence as the standard genetic equipment and consider the reverse genetic dynamic of dumbing instead - in particular dumbing via random genetic (or DNA) mutations. In this theory high functioning intelligence was a big evolutionary winner (perhaps akin to color vision amongst primates) and thus as offsprings of that process the DNA code responsible for high intelligence is found in most if not all of us. Then to cover the substantial variation in our innate intellectual capacities, Mitchell suggested we consider that random mutations - the numbers of which do vary amongst individuals (with an average of perhaps 100 mutations) - are the dynamic factor in the intelligence realm. This dumbing hypothesis is built upon the basic assumption that the impact of a DNA mutation is much more likely to be harmful than helpful. Random mutations do seem unlikely to beget high intelligence.

As suggested in Dobbs' article, Mitchell's theory of the dumbing down of intelligence appeared to have some traction amongst geneticists. Given the circumstances - little if any regular DNA support for such a big variable and largely innate behavioral attribute - it is not clear what alternatives were scientifically plausible. But stepping back, how much sense does this theory really make? When reading evolutionary psychology pieces, they tend to make both evolutionary sense and also immediate personal sense. For example a number of the various sexual behavioral theories are backed by clear evolutionary logic. The purported behavioral tendencies likely would have been advantageous to our ancestor's reproduction efforts and thus any underlying responsible genes would have spread widely. And in an intimate sense one can readily observe in oneself and others some of these behavioral tendencies. But does Einstein's intellect seem plausible as an evolutionary outcome? This topic and some relevant intelligence-related conundrums will be returned to in the following chapter.



In another behavioral domain, the earlier David Goldstein quote pointed out that the search for common genetic variations had uncovered “almost nothing” in the way of connections to the occurrences of bipolar disorder and schizophrenia. An update on the schizophrenia genetic search can be gleaned from an article by Stanford University’s Tanya Marie Luhrmann in the Summer 2012 issue of the *Wilson Quarterly* [Luhrmann]. In that “Beyond the Brain” article Luhrmann chronicled the general failure of the treatment of mental illness via what is referred to as the “bio-bio-bio model”. That biology-oriented approach had focused on such illnesses as expressions of a “brain lesion, [with a] genetic cause, and [an associated] pharmacological cure”. This has not worked well thus far and in particular the underlying “search for a genetic explanation fell apart”. Luhrmann described that:

[t]he number of implicated genes is so great that Schizophrenia Forum, an excellent Web site devoted to organizing the scientific research on the disorder - the subject of 50,000 published articles in the last two decades - features what [one leading researcher Ridha] Joobar has called a “gene of the week” section. Another [prominent researcher], Robin Murray ... has pointed out that you can now track the scientific status of a gene the way you follow the performance of a sports team.

These investigators apparently were finding no significant DNA basis for the inheritance of the susceptibility to schizophrenia, but the enormous momentum of the process had them chasing down statistical noise. This could well be happening elsewhere as expressions of the missing heritability problem.

A good deal of Luhrmann’s article resonated with a message in Ethan Watter’s critical look at mental illness in the modern world, *Crazy Like Us: The Globalization of the American Psyche*. Essentially the scientific assumptions - in particular the underlying materialist model with its presumed genetic origins - may be accurate but the implementation has thus far not worked. Traditional approaches to episodes of schizophrenia seem to have worked better than those of modern medical science. As Luhrmann pointed out, “[s]chizophrenia has a more benign course and outcome in the developing world”. She also touched on the underlying downside of science’s bio-robotic perspective (a perspective captured in the title, “Faulty Circuits”, of an April 2010 *Scientific American* article by the director of the National Institute of Mental Health, Thomas Insel). In our country people with schizophrenia commonly spend a lot of time homeless in part because “[t]hey dislike the diagnosis even more than the idea of being out on the street, because for them the idea of being ‘crazy’” is worse. Luhrmann also wrote that “Indian families don’t treat people with schizophrenia as if they have a soul-destroying illness.”

Identical Twins

One intuitive vantage point from which to see a number of the difficulties confronting the DNA model - and also the encompassing materialist vision - is with monozygotic twins. These so called identical twins in fact present a number of basic mysteries. The cause of their origin - the initial split or division of a single cell zygote - is a mystery. So is its occurrence only in some species. Further, similar appearances and biological-presumptions aside, the empirical realities of the resulting twins constitute a significant challenge to the sacred DNA "created us body and mind" logic that motivated the Human Genome efforts and associated excitement.

Although DNA replicas, such twins whether they were raised together or separately, have been observed on average to be more different than alike personality-wise. Thus these clones can closely share the same environment or inhabit separate ones (with whatever differences in epigenetic implications) and still they appear to have comparably different personalities. Notice this environment (or nurture) minimizing finding is also consistent with the conclusions of adoption studies. With some personal exposure to identical twins and/or after reading relevant study data, it should not come as a surprise that one conjoined (attached) monozygotic twin commented that "[w]e are two completely separate individuals who are stuck to each other. We have different world views, we have different lifestyles, we think very differently about issues" [Harris J., p.1]. This mystery motivated Judith R. Harris' *No Two Alike* and was discussed at some length in Steven Pinker's *The Blank Slate* [Pinker 2002].

Yet in the case in which identical twins were separated at birth, they can still share remarkably specific behavioral tendencies as mentioned in an earlier quote from Pinker. From the small and questionable stuff (look enough and any pair of humans might find they share some little preferences) to the big and life-defining stuff - like becoming very dedicated volunteer firemen [Segal, p.14]. Such phenomena have been used as strong evidence for the life-defining import of DNA, but set against the seemingly innate personality differences they could be just another mystery.

Large health differences between monozygotic twins were described in a 2006 *New York Times* article by Gina Kolata [Kolata 2006]. That article opened by describing a healthy and active 92 year old and her identical twin. The latter "is incontinent, she has had a hip replacement, and she has a degenerative disorder that destroyed most of her vision ... [and] has dementia". Yet the sisters have the same DNA, grew up together, and lived in the same place. They also had markedly different personalities and ambitions.

The centerpiece of Kolata's article was a description of a large study comparing the variations in longevities found amongst identical and same-sex fraternal (or dizygotic) twins. Since the former share all of their variable (and potentially individual-differentiating) DNA and the latter only half, comparisons like this are standard practice for determining the relative import of DNA, as in behavioral genetics. Continuing, together the study's twin populations totaled 10,251 pairs. The study found that the identical twins died only a little closer together than the fraternal twins and, specifically, the deaths of the identical twins averaged "more than 10 years apart". Consistent with this, one of the twin study's authors commented "[h]ow tall your parents are compared to the average height explains 80 to 90 percent of how tall you are compared to the average person [but] only 3 percent of how long you live compared to the average person can be explained by how long your parents lived". The relevant *Nature Review Genetics* paper gave the inferred genetic component as covering - "about a quarter of the variance in adult lifespan" - and pointed out that somewhat surprisingly, "the genetic influences on lifespan are minimal before the age of 60 and only increase after that age" [Christensen et al]. As representative of the intellectual momentum associated with DNA (and science), though, Kolata's article had been titled "Live Long? Die Young? Answer Isn't Just Found in Genes".

Kolata's article also mentioned that "randomness" had been found amongst the longevity of genetically identical lab animals. Additionally, Dr. Robert Hoover of the National Cancer Institute was quoted from an editorial on the cancer connection, "there is a low absolute probability that a cancer will develop in a person whose identical twin - a person with an identical genome and many similar exposures - has the same type of cancer."

It is noteworthy, though, that these very significant and unexpected findings (and article) seem to have been minimally integrated into relevant scientific communication. This is possibly common for results that challenge materialism. For example, four years later in a *Scientific American* review article on the lack of findings in personal genomics the word "twin" never appeared [Hall]. If you are trying to find DNA which is significant to the different health outcomes of individuals who are broadly separated by environment and their variable portions of DNA, wouldn't it be good idea to gauge these efforts - and more generally inform others - on the large health differences found between individuals who are minimally separated by environment and share their DNA specifications?

Another monozygotic conundrum is the remarkable bond that tends to exist between them. As pointed out by Steven Pinker, "when separated at birth and reunited as adults, ... say they feel like they have known each other all their lives" [Pinker 2002, p.47]. In one of my childhood neigh-

borhoods I can't even remember the local twins being apart. Given that siblicide is common in nature does this really make sense for siblings [Tenneson]?

Finally, another monozygotic-mystery can be found with regards to male exclusive homosexuality. This behavioral tendency appears to be established by birth and of course is a challenge to evolutionary reasoning. The DNA contributions can not be big, though, since when one monozygotic twin is gay then the other twin is gay in only about 20 to 30 percent of cases (against a backdrop overall gay frequency of 2 to 4 percent). Additionally, it appears that the likelihood of a male having a homosexual orientation increases by about 30 percent for each older brother preceding him. As mentioned earlier this information was found in Francis S. Collins' confident DNA book, *The Language of Life* [Collins, pp.204-205]. Thus science's gay explanation has to identify a loose DNA contribution, find the means to ramp up the gay likelihood by 30 percent per older brother (presumed to be caused by the mother's previous male-pregnancy experiences), and also perhaps identify some other subtle prenatal environmental influences to wedge apart the twins' orientations. Does this seem "bluntly deterministic" or consistent with the earlier cited programmatic assessment of Sam Harris? In fact these findings, like others not related to appearance, are contrary to DNA-based expectations and suggestive of a broadly-based mystery.

Transcendental Introduction

I switch gears here now to introduce a possible transcendental take on the DNA impasse as well as some neighboring mysteries. This alternative take is based on the common premodern transcendental understanding of life in which there is also an underlying soul reincarnating from life-to-life, and this non-material aspect can have far-reaching effects. On the prevalence as given previously from *M'Clintock and Strong's Cyclopaedia of Biblical, Theological and Ecclesiastical Literature*, "[t]ransmigration ... being spread all over the world, seems to be anthropologically innate" [Head and Cranston, p.170]. This belief has two components, the relatively intuitive behavioral continuity aspect and the very puzzling cause-and-effect (or popularly "karma") aspect. As mentioned earlier, these two distinct hypotheses were claimed to have been historically packaged together [Head and Cranston, p.10]. Perhaps within small isolated groups the appearance of the continuity of personality and interests across lives helped to establish the continuity hypothesis. Also the karma hypothesis might have followed from instances in which individuals exhibiting such continuity also appeared to experience consequences consistent with the previous individual's (or incarnation's) efforts.

A transcendental continuity understanding would be consistent with individual cases suggestive of young children experiencing an explicit recall of a previous life [Stevenson; Tucker; Leininger] and also with our innate dualism. The continuity aspect also offers explanations for a number of surprising behaviors including those found amongst prodigies, transgender individuals and adoptees. Further it could provide insight into the very surprising personalities found amongst a number of species [Siebert, Angier 2010]. Continuity also offers a simple explanation for the behavioral differences found between monozygotic twins and more generally a consistent framework for the behavioral side of the missing heritability problem. With the second - the cause-and-effect - transcendental component, there appears to be consistency with the unexpectedly large health differences found between monozygotic twins and moreover with the disease susceptibility portion of the missing heritability problem. Thus, the missing DNA origins for a number of individual characteristics could be viewed as expressions of carryover from previous lives and with some exceptional behaviors - as with prodigies and prodigious savants - there could be some additional carryover consistent with some premodern descriptions of the disembodied state.

A further suggested generalization is that transcendental import would likely be overlapping with, as well as complementary to, the import of DNA. If, as some traditions assert, the incarnating soul is drawn to their parents-to-be, then that soul might tend to find some continuity in the DNA specifics produced by conception - beyond the species and sex default codes. This could include DNA-determined unusual conditions. But beyond this overlapping aspect, though, the odds that the crapshoot of conception would deliver a variable DNA match for a soul's overall trajectory is zero. The DNA definition would have to be breached in many ways. Thus to the degree that science can show that DNA plus realistic environmental impacts define individuals, then this would minimize the import associated with possible transcendental phenomena. In this regard, efforts to confirm the DNA expectations of behavioral genetics and also to account for the surprising differences found between monozygotic twins are of particular interest.

A complication being glossed over here is that in a number of cases the DNA blueprint's import is hypothesized by science to be probabilistic and thus the relevant confirmation here would have to be carried out as an averaging-exercise over many individuals. For example, from this perspective there should be one or more DNA codes specifying for the tendency to be a transgender individual and thus individuals bearing such a code should be more likely to experience the transgender sense (but are not guaranteed to). The rationale for such probabilistic hypotheses must in part be to try to explain the differences between so-called identical twins.

Continuing, a transcendental explanation for monozygotic twins could be as follows. Identical twins had been close before their current life, perhaps having recently been siblings, close friends, coworkers, or spouses. Such scenarios are consistent with some reports from modern investigations into possible cases of reincarnation [Stevenson, pp.171-172]. This previous closeness brought them together to be born as monozygotic twins and was the underlying cause of the initial split of the single cell zygote. Continuity of behavioral tendencies tends to result in their roughly similar personalities - as is often found between those who are close - and also could provide for some shared behavioral preferences. Their previous connection, perhaps including their shared disembodied experiences, provides a basis for their unbelievable closeness. Superficially, such twins are material-only replicas produced by the same DNA blueprint, but underneath there are two separate beings with mostly separate backgrounds accounting for much of their otherwise surprising differences. The issue of male exclusive homosexuality amongst monozygotic twins will be returned to in a later chapter.

For a Western historical perspective on such views consider the following observations from the 1600s from Joseph Glanvill, Chaplain to King Charles II:

Every soul brings a kind of sense with it into the world, whereby it tastes and relisheth what is suitable to its particular temper What can we conclude but that that the soul itself is the immediate subject of all this variety and that it came prejudiced and prepossessed into this body with some implicit notions that it had learned in another? To say that all this [individual] variety proceeds primarily from the mere temper of our bodies is methinks a very poor and unsatisfying account. For those that are the most alike in the temper, air, and complexion of their bodies, are yet of a vastly differing genius [tendencies] What then can we conjecture is the cause of all this diversity, but that we had taken a great delight and pleasure in some things like and analogous unto these in a former condition [Head and Cranston, p.122].

Has any modern researcher read or considered such a perspective?

Behavioral Genetics Reconsidered

In one of Steven Pinker's intellectual epics, *The Blank Slate*, he offered some concluding thoughts on the modern intellectual movement characterized by extreme environmental assumptions [Pinker 2002]. This nurture-only perspective in Pinker's opinion "became part of the secular faith and appeared to constitute the common decency of our age". Pinker then summarized the

downside, including that this perspective “torments mothers who work outside the home and parents whose children did not turn out as they would have liked” and that it “blinds us to our cognitive and moral shortcomings”. He then went on to write that:

[r]egardless of its good and bad effects, the Blank Slate is an empirical hypothesis about the functioning of the brain and must be evaluated in terms of whether or not it is true. The modern sciences of mind, brain, genes, and evolution are increasingly showing that it is not true [p.421].

Into the intellectual vacuum of that fading movement appears to be a tsunami of its own - the modern DNA-based, bio-robotic vision of life. This biology-based belief views all of life as material-only expressions. In geneticist J. Craig Venter’s recent book, *Life at the Speed of Light: From the Double Helix to the Dawn of Digital Life*, Venter’s answer to “a question at the heart of biology: ‘What is life?’” was provided as “DNA-driven biological machines” [Venter, p.6]. The associated big picture view is of a collection of evolved species-defining DNA pools and the corresponding view of an individual is as the outcome of a lottery-like event within one of those pools. One could argue about some of the unhealthy import of this mechanized view of life - as with regards to the treatment of mental illness - but perhaps it is the overall certainty with which it is pushed that should be most objectionable.

One key line of observations that bolstered the modern faith in the behavioral import of DNA, was studies of monozygotic twins that had been separated in infancy. Observations from such studies were then compared to those from studies of raised-together monozygotic twins and nature-versus-nurture inferences drawn. Specifically, if the differences in a behavioral attribute seen between raised-together and raised-apart twins was small, then the corresponding nurture contribution was also presumed to be small. Also of note here was that the degree of similarity found between the raised-apart twins on a particular behavior (that correlation) was assumed to be an approximation of the corresponding genetic (or nature) contribution. Thus, in a very significant 1990 *Science* article entitled “Sources of Human Psychological Differences: The Minnesota Study of Twins Reared Apart”, Thomas J. Bouchard, Jr. *et al* had written:

[o]ur findings support and extend those from many family, twin, and adoption studies, a broad consilience of findings leading to the following generalization: For almost every behavioral trait so far investigated, from reaction time to religiosity, an important fraction of the variation among people turns out to be associated with genetic variation [Bouchard et al].

And then more particularly they wrote:

[o]n multiple measures of personality and temperament, occupational and leisure-time interests, and social attitudes, monozygotic twins reared apart are about as similar as are monozygotic twins reared together [Bouchard et al].

The similarity noted here supported the first quote on the behavioral import of DNA. One particular focus in that paper was on intelligence and for that behavior attribute the calculated genetic contribution was “70% of the variance in IQ”. Based on such results one might well surmise that we are far from environmentally-defined “blank slates”.

Returning to the earlier Pinker quote, his focus on veracity as the bottom line was certainly appropriate. A pillar of the modern materialist vision is that DNA blueprints specify for various individual outcomes. In a general species- and gender-defining sense that import appears quite likely to be the case, although little has been confirmed. For certain specific conditions there are also confirmed DNA origins. (Additionally, in the case of tumors, specific DNA mutations have been found to be driving particular growth dynamics.) But for the broadside of who we are as individuals - including much of the territories staked out by behavioral genetics and personal genomics - the large scientific efforts have identified “almost nothing” and thus the ongoing silence. Also predating DNA searches there have been a number of obvious questions for this materialist view, in particular in the behavioral realm. With the transgender phenomenon, some people do spend their entire lives wishing that they were the opposite sex and a recent study found that many of them who have undergone sex-change efforts (transitioned) “knew they had been born into the wrong gender from childhood?” [Landau]. Did biologists and psychologists really think they were going to find a DNA - or environmental - explanation for this phenomenon?

So what kind of response has this DNA impasse generated? Apparently very little, except perhaps some scrambling around to try to hold down the bio-fort. In a 2011 lonely assessment of the analogous personal genomics situation, Jonathan Latham and Allison Wilson pointed out that with few exceptions (including previously identified genes for cystic fibrosis, sickle cell anemia, and Huntington’s disease; and also including genetic contributions for some instances of Alzheimer’s and breast cancer):

according to the best available data, genetic predispositions (i.e. causes) have a negligible role in heart disease, cancer, stroke, autoimmune diseases, obesity, autism, Parkinson’s disease, depression, schizophrenia and many other common mental and physical illnesses that are the major killers in Western countries [Latham and Wilson].

They went on to ask (in italics) “[h]ow likely is it that a quantity of genetic variation that could only be called enormous (i.e. more than 90-95% of that for 80 human diseases) is all hiding in what until now [circa 2010] had been considered genetically unlikely places?” Latham and Wilson then conjectured that the missing contributors must be environmental but this appears to be an enormous stretch. Although they might have been right about the potential worth of increasing attention on environmental factors, their neglect of innate contributions doesn’t make sense, in particular beginning with the health differences found between monozygotic twins. Nonetheless, Latham and Wilson appropriately pointed out that “[b]y all rights then, reports of the GWA [genome wide assessments] results should have filled the front pages of every world newspaper for a week”. Needless to say that big missing heritability coverage hasn’t happened.

Rather, the media coverage went another way. For example, in the fall of 2012 there was an avalanche of media stories about big developments from unexpected regions of the DNA. The *New York Times* reported that the “junk” DNA findings were “considered a major medical and scientific breakthrough, [and have] enormous implications for human health” [Kolata 2012]. Also reported was that one prominent researcher had commented that the junk DNA developments were “a stunning resource” and that his “head explode[d] at the amount of data”. The seemingly hyped nature of the junk reports was then confirmed in a very unusual guest blog write-up at *Scientific American*. Biologist Athena Andreadis in a September 17, appropriately titled entry, “Junk DNA, Junk PR”, described the origin of the junk DNA reports as “a huge, painstakingly orchestrated PR campaign” by the relevant scientists and that the developments didn’t “alter the current view of the genome” [Andreadis]. This criticism appears consistent with the earlier assessment by evolutionary biologists that most of our DNA is in fact nonfunctioning junk. Thus Andreadis’ critical comments were apparently representative of the big rift that this resuscitation of junk DNA by geneticists set off between themselves and evolutionary biologists [Zimmer 2015].

Now returning back to Steven Pinker’s *The Blank Slate*, there is a nice section laying out some of the basic findings of behavioral genetics [Pinker 2002, pp.372-381]. The first two laws of behavioral genetics describe the inferred inputs from nature and nurture. In modest fashion he introduced the second of these laws - “[t]he effect of being raised in the same family is smaller than the effect of the genes [i.e., the first law]” [p.373]. This could have been better stated as, “short of traumatic effects and some nominal allegiances (like political affiliation), being raised in the same family has negligible impact”. A negligible familial contribution was in fact Pinker’s conclusion. Thus the big argument against the Blank Slate (or nurture dominates) hypothesis is that empirical data have suggested that the influence of nurture is in many ways very small.

Continuing, Pinker had written that the Third Law of behavioral genetics claims that “[a] substantial portion of the variation in complex human behavioral traits are not accounted for by the effects of genes or families” [p.373]. This might have been better stated as “a little over half of an individual’s particular behavioral tendencies do not make nature plus nurture sense”. Thus in a concrete example, an individual’s particular behavioral trait - say tendency towards introversion - is likely to only loosely follow from the state of introversion of their parents and also their family environment. Following the apparently standard routine, Pinker formalized the hypothesized origin of this Third Law by calling it the “Unique Environment”. This presumed environmental impact - which most tangibly is posited as the individual environmental experience of one identical twin which made it different than the other twin (and the process somehow produces similar outcomes whether the twins were raised together or were separated at birth) - completes the behavioral genetics model. Pinker offered a nice alternative take of the three laws in writing that “identical twins are 50 percent similar whether they are grow up together or apart” [p.381]. He also acknowledged, though, the mystery of this unique environmental contribution with a Bob Dylan-influenced, “something is happening here but we don’t know what it is”. Note that this same mysteriousness also limits the traction available for the behavioral import of DNA’s recently uncovered and hyped sidekick - the epigenome.

Yet despite the unfolding DNA deficit situation, scientific confidence continues as apparently does all of the genetic reasoning and expenditure based on it. As a relevant snippet, one *New York Times* article offered an individual’s conflicting experience with genetic testing [Peikoff]. To solidify the analysis the author had gotten opinions from various relevant scientists. One point repeated was that the current genetic testing wasn’t thorough enough. One academic pointed out that the tests are “missing 99.9 percent of the letters that make the genome”. The genome pioneer J. Craig Venter was quoted in pointing out that Piekoff’s conflicting results “are not the least bit surprising” and that “[a]nything short of [whole genome] sequencing is going to be short on accuracy - and even then, there’s almost no comprehensive data sets to compare to”.

What wasn’t touched on in the Piekoff article was the additional significance of 99.9% - an estimate of the DNA equivalence between any individuals. Additionally, how much of the complementary (0.1%) individual-differentiating DNA have science’s considerable search efforts already checked? And how likely is it that those DNA searches would have found “almost nothing” to this point?

Returning to Pinker’s behavioral genetics coverage we can see that science’s explanation of one’s particular behavioral tendencies is essentially based on a presumed DNA contribution plus a

hypothesized unique environmental impact. So what happens if this missing DNA situation continues on its “almost nothing” trajectory? Will scientists acknowledge and perhaps emphasize what would constitute a profound mystery? Would a scientist write a follow-up article to Pinker’s “My Genome, My Self” [Pinker 2009], perhaps one entitled “My Genome, Basically So What?”.

Chapter 2 - A Collection of Intelligence Conundrums

The previous chapter briefly touched on the surprising situation facing scientists attempting to account for the variations in human intelligence. For this significant and basic behavioral attribute, DNA searches have only identified about 1 percent of the expected underlying DNA basis (or genetic component) as of the fall of 2012. During the following August a behavioral genetics researcher in reviewing a manuscript of mine acknowledged a resignation similar that to that conveyed in the same 2012 article (i.e., “99 percent empty”). That such a prominent and variable individual feature could well be de-coupled from a basis in DNA is a stunning development. What then made someone like Albert Einstein so intellectually superior?

In that same *New York Times* article discussed in chapter 1, the author David Dobbs had described a new theory in which the DNA dynamic for intelligence was reversed. As mentioned earlier, in that theory proposed by the neurogeneticist of Trinity College Dublin, Kevin Mitchell, intelligence was a big evolutionary winner and thus high intelligence was the evolved standard equipment [Mitchell 2012/07]. The variations found in human intelligence were then proposed to come from the inevitable random mutations found in each of our genomes, with such mutations seen as much more likely to hurt rather than help intellectual abilities. Thus humans with the highest innate intelligences simply had minimal contamination of their intelligence blueprint, while the rest of us suffered significant losses via DNA mutations. More subtly, Mitchell had proposed that the apparent inheritance of intelligence reflected an underlying heritable mutation rate. An individual born into a family with a relatively low rate of random mutations present in their genomes, would tend to have a high IQ. Conversely, an individual born into a family which had a higher rate of random mutations would tend to have a lower IQ.

Mitchell’s intelligence proposal raised additional questions, though. How could evolution have resulted in something akin to a genius’ intelligence as standard equipment? Does the variability apparent in group (average) intelligences then reflect differences in the average DNA mutation rates found amongst groups? Coincidentally, eight days after the Dobbs article the *New York Times Magazine* contained a lengthy article by Andrew Solomon entitled “How Do You Raise a Prodigy?” [Solomon 2012/10]. In it were descriptions of high intelligence kids who then from Mitchell’s proposed perspective would then have carried minimal dumbing mutations. Here then might be descriptions of kids operating close to the proposed default high intelligence state. Here is an excerpt:

Drew Petersen didn't speak until he was 3 1/2, but his mother, Sue, never believed he was slow. When he was 18 months old, in 1994, she was reading to him and skipped a word, whereupon Drew reached over and pointed to the missing word on the page.

Drew went on apparently to learn to read quite a bit of sheet music on his own, skip the first of six months of formal piano lessons at age 5, and then within the year was "performing Beethoven sonatas at the recital hall at Carnegie Hall". On the way to kindergarten at one point Drew asked his mom, "[c]an I just stay home so I can learn something"? His mom had commented, "[h]e was reading textbooks this big, and they're in class holding up a blowup M". Additionally, Drew and some of the other prodigies considered had exhibited enormous self-determinations which left one mom commenting "it's not for me to be proud; [she] who does this herself".

As conveyed in the prodigy article, such examples of high intelligence appeared to be overwhelmingly innate. Drew's parents did not appear to be standout intellectuals, also had a non-prodigy child, and sensibly seemed to avoid hyping the genius business. But again does such relatively high intelligence make sense as an evolutionary outcome? From an evolutionary standpoint is it even likely to be found in our evolutionary cognitive card collection, let alone as a standard outcome? As previously touched on, a framework for contemplating the evolutionary forces that shaped our intelligence was presented in Steven Pinker's *How the Mind Works* [Pinker 1997, pp.186-190]. That description consisted of humans having found a cognitive niche specializing in how to outsmart and then often eat other species. As described above, Pinker had pointed out life for our "ancestors [was like] a camping trip that never ends" without modern equipment. Pinker had appropriately introduced this concept with a chapter title "Revenge of the Nerds". A simpler evolutionary dynamic was the development of trichromatic vision within our primate heritage which could have been helpful for identifying ripe fruit. But is the proposed cognitive evolutionary dynamic - with natural selection weeding out DNA that was lousy for campings' cognitive tasks and also preserving and spreading some fortuitous DNA camping gems - likely to make possible, let alone standardize the kind of high intelligence exhibited by prodigies? How natural selection could have produced a little kid capable of playing Beethoven sonatas, and being sufficiently self-motivated to do so, is quite the conundrum.



The remainder of this chapter considers a collection of three intelligence-related mysteries - a childhood behavioral syndrome, the Einstein Syndrome; the somewhat overlapping phenomena of prodigies and savants; and then the Flynn Effect or the observed historical rise in IQ's. In isolation each of these mysteries provides plenty of puzzlement, but in the context of the lack of DNA

support for our variations in intelligence it will be argued that together they represent a very significant challenge to the modern understanding of humans. The material that follows is an updated version of some material presented in a paper published in the medical online journal, Cureus.com in August 2013 [Christopher].

The Einstein Syndrome

Thomas Sowell's book *The Einstein Syndrome - Bright Children Who Talk Late* considered a very interesting behavioral phenomena named for the late physicist Albert Einstein (Sowell is a well known author and economist) [Sowell 2001]. Sowell pointed out that children with this condition have "speech development [which] lags far behind that of other children their age, while their intellectual development surges ahead of their peers" [p.1]. These children often are very strong willed, late in toilet training, relatively weak socially, and their intellectual strengths are focused in analytical areas and/or music. They also tend to possess exceptional memories. Simply put such kids appear to be born quite strongly nerd-inclined ("nerd" is not a slight). Another prominent characteristic is that they are almost always born into families with a strong technical and/or musical presence.

The Einstein Syndrome considered children fitting this description whose parents had come together in two groups. One group represented the experiences of 43 biological families and was connected with Sowell, while the other group represented 232 biological families under the auspices of Professor Stephen Camarata, a speech pathologist at the Vanderbilt University Medical Center [pp.4-5]. With the inclusion of a few families with multiple late-talkers, the respective counts of biological children were, 45 and 236. The median age of beginning to speak in the smaller group connected to Sowell was four years old, while the figure for the children in Professor Camarata's group was three and a half [p.107]. In the smaller group most kids "did not make a statement using more than one word until they were at least three and a half years old and their first complete sentence was spoken when they were four" [pp.17-18].

For comparison, the normal development of speech progresses from single word utterances and then at "around 18 months the child starts to combine single words into two word sentences" [Smith et al]. Subsequently, their "[v]ocabulary typically grows from around 20 words at 18 months to around 200 words at 21 months" [p.304]. Furthermore, the large Stanford-based

Terman study (1925-59) of gifted children (with IQ's of about 140 and higher) found they tended to talk earlier than their lower IQ peers [p.472].

Sowell had previously written a book, *Late-Talking Children* [Sowell 1998], on this subject and had a son who had exhibited this syndrome. Professor Camarata also had had a son with this syndrome and he himself had demonstrated it as well. Almost 90% of the children in these groups were boys [Sowell 2001, pp.9-10]. Also noteworthy was that 26 percent of the children in Sowell's group had a close relative who had exhibited this syndrome, while the corresponding figure for Camarata's group was 48 percent [p.9].

A "striking" characteristic found with the Einstein Syndrome was that the associated families "are highly atypical - and highly analytical" in their occupations [Sowell 2001, p.5]. Of the late-talking children considered, almost three quarters "had at least one close relative who was either an engineer, a scientist, or a mathematician" [p.5]. Close relatives in this context were limited to parents, grandparents, aunts, uncles, and additionally for Camarata's group, siblings. The children in the two study groups were about 10 times as likely to have fathers who were engineers as were late-talking children in general that had been considered in a British study [p.7]. It is noteworthy that this association with a family type perhaps mirrors a weaker correlation observed between the occurrence of autism and technical families considered by Simon Baron-Cohen in a November 2012 *Scientific American* article.

Also observed was also a big musical connection. In both groups about three-fourths of the kids had a close relative who played a musical instrument. In Camarata's group 28% of them had a close relative who was a professional musician and in Sowell's group that figure was 26% [Sowell 2001, pp.7-8]. This appeared to be another relatively focused activity correlated with the occurrence of this syndrome within a family.

Some of the stories involving these children exhibiting the Einstein Syndrome were amazing. In one instance the three year old "silent" son of a professor was involved in the following:

The older boy, now five, had learned to read and would entertain his doting parents by doing so aloud. One evening he came upon a word he did not recognize, and struggled with it. At which point his brother toddled over, peered at the text and read out the sentence perfectly. Following that, he again lapsed into silence for several months and only then began to speak easily [p.19].

In another case a toddler "became deeply absorbed in listening to Bach, to the point of being moved to tears" [p.85]. Sowell also wrote that "one of the five-year-old pre-schoolers in my

group helped both his mother at home and his teacher at school when they had problems using the computer (circa the 1990's). He could also play the piano with his eyes closed" [p.12]. Extraordinary child lock breaking abilities were exhibited by Sowell's own son prior to the age of one [p.41].

In a detour from Sowell's *The Einstein Syndrome* coverage, the earlier considered prodigy Drew Petersen appeared to fit the syndrome profile in so far as demonstrating the late-talking attribute and also in having an engineer for a father [Solomon 2012/10]. As described in Solomon's book, one of Drew's piano teachers later recalled her reaction to hearing him for the first time:

[he] could barely reach the pedals, but played with every adult nuance you'd ever want. I thought, 'Oh my God, this really is a genius. He's not mimicking and not being spoon-fed. His musicality comes from within.' [Solomon 2012, p.418]

Thomas Sowell also considered some earlier experiences of adults who apparently had had the syndrome (including Albert Einstein). One was the pianist Arthur Rubenstein who demonstrated a remarkable draw to the piano as a young child:

[he] became fixated on the piano. Whenever he was asked to leave the drawing room where [it] was kept he screamed and wept. He began playing the piano at age three. When his father later brought him a violin to play, little Arthur reacted by smashing it, earning himself a spanking [Sowell 2001, p.39].

Sowell wrote that such strong-willed behavior "will be all too familiar" to Einstein Syndrome parents. Further Rubenstein:

[a]fter hearing a performance of the first suite of Edvard Grieg's *Peer Gynt*, [he] returned home 'to play almost all of it - to the amazement of the family'. At this point Rubenstein was not yet five years old and had not yet begun formal instruction under a professional musician. At age seven, he gave his first public performance [p.40].

Another remarkable developmental syndrome, Williams Syndrome (roughly the opposite of the Einstein Syndrome), was also touched on.

These amazing behaviors led Sowell to title his explanatory chapter, "Groping for Answers". There he carefully laid out some hypotheses about possible brain developmental dynamics which could have produced the specific patterns of the observed aptitudes. Beneath this he favored a DNA/heredity basis with some support coming from the earlier occurrences of the syndrome amongst close relatives and also indirectly by the analytical-orientations of the families.

Within families with instances of the Einstein Syndrome most other siblings were normally developing, though [Sowell 2001, p.97 and epilogue]. Additionally, of course, most high aptitude technical and/or musical people have not followed this pattern. Given the rarity of the syndrome a DNA explanation would seem likely to involve some form of a mutation, but is it realistic for a mutation to produce this set of behaviors? Is it plausible for a bio-molecular code to specify for a particular obsession such as with the piano? Additionally, how could the relevant mutations be localized to this type of family?

A possible explanation from the transcendental perspective is as follows. A being who became highly focused in a previous technical and/or musically-occupied life was reborn and brought along some of their behavioral tendencies and capabilities. This behavioral skew was also carried over and reflected in their brain as well and this combination could have contributed to the delayed speech. Additionally, a symptom associated with a person being very intellectually focused is that they also tend to be out of touch with their body and this could have been reflected in the phenomenon of delayed toilet training. The fact that such children were predominantly found in technical and/or musically-connected families reflected the tendency of an incarnating-being to be drawn to similar and/or previously-related parents. From this perspective, one might expect that this syndrome would not be found within cultures that do not support analogous careers. Have there been any Albert Einstein's born in groups that don't support scientific-like careers (or more generally distant from such groups)?

The above transcendental take on the Einstein Syndrome portrays some basic elements of a possible transcendental dynamic in which the incarnating soul tends to be drawn to local, similar, and/or previously-related parents. This dynamic would be consistent with the crude heredity patterns that underlie DNA expectations, for example in the field of behavioral genetics. Further, although the *The Einstein Syndrome* did not report on measured brain characteristics, it is likely that the children exhibiting these unusual characteristics also had correlated brain features. Likewise, there have been observations from brain imaging studies in which some features apparently connected to high intelligence stood out, yet the observed correlation between intelligence scores and variations in DNA is again only about 1 percent [Zimmer 2008, Dobbs]. This could be viewed as an example of a transcendental Lamarckian-like effect. With such a transcendental dynamic a very focused individual could then pass on some of their acquired characteristics to their next incarnation, as opposed to their offspring as was proposed with the Lamarckian evolutionary dynamic. Such a transcendental Lamarckian dynamic might also involve the production of supporting mutations as was suggested for the cause of the initial zygotic split leading to monozygot

ic twins. (Also of note here is that the Lamarckian concept has been scientifically resuscitated in connection with possible epigenetic inheritance effects).

Finally on a potentially related note, bright children are much more likely to experience myopia [Sowell 2001, p.90] and among autistic or retarded musical prodigies “a majority ... have been either congenitally blind or severely visually impaired” [p.102]. Sowell had used these points in part as supportive of a heredity-based explanation for the Einstein Syndrome. From a transcendental perspective such visual impairments could be viewed as symptomatic of rebirths that were extremely focused on music and thus involved corresponding losses of neglected capacities.

Savants

The second challenge considered here involves the behavior of savants as depicted in Darold A. Treffert’s very interesting book, *Islands of Genius* [Treffert 2010]. In addition to traditional autistic savants this book also considered the recently recognized acquired savant syndrome in which savant behavior appears in the wake of a central nervous system setback. Although not considered here, Treffert’s book also considered sudden savant syndrome in which savant skills seem to appear spontaneously. Treffert’s preface provided the following introductions to some of the savant terrain:

Kim Peek, the inspiration for the movie *Rain Man*, memorized 12,000 books. He is the Mt. Everest of memory with bottomless factual recall in multiple areas of expertise including history, geography, literature, music, sports, science and religion, to name only some. He became a living Google. But as a child, his parents were advised to put him in an institution. One doctor suggested a lobotomy.

Matt Savage, who couldn’t stand noise or being touched as a child, very quickly mastered the piano at age 6 1/2 and had his first CD of jazz composition at age eight. Matt is recognized worldwide now as “the Mozart of Jazz,” a title conferred on him by the famous jazz artist Dave Brubeck. At age 17 he is the leader of the Matt Savage Trio, giving concerts around the globe. He recently recorded his eighth CD.

Leslie Lemke is blind, severely cognitively impaired and has cerebral palsy. Yet he played Tchaikovsky’s Piano Concerto No. 1 flawlessly after hearing it for the first time at age 14. Leslie, who has never had a music lesson in his life, is a musical genius.

After a 15-minute helicopter ride over London, Stephen Wiltshire, in a five-day drawing marathon, produced a spectacularly accurate four meter long sketch which captures with mind-boggling fidelity seven square miles of London - building by building, street by street, window by window. Diagnosed with autism at age three, he was described as a “rocket of young talent” on the scene at age eight. Stephen was invested by Queen Elizabeth II as a Member of the Order of the British Empire and now has his own gallery in the Royal Opera Arcade in London.

These extraordinary people, and others like them ... have savant syndrome, a rare but remarkable condition in which incredible abilities - “islands of genius” - coexist side by side, in jarring juxtaposition, to certain disabilities within the same person [pp.XIII-XIV].

Also in the preface Treffert suggested that:

no model of brain function, including memory, will be complete until it can fully incorporate and explain this jarring contradiction of extraordinary ability and sometimes permeating disability in the same person. Until we can fully explain the savant, we cannot fully explain ourselves nor comprehend our full capacities [p.XIV].

For Treffert there appeared to be no doubt that these remarkable behaviors arose solely from physical processes in the brain. The central mystery for him was the origins of those savant-functioning capabilities. His explanation involved what he called “genetic memory” and in particular that savants have somehow tapped into our shared DNA-based storage of knowledge and skills and then implemented them in the hardware of their brain. He thus offered a technical analogy of the factory-installed software on a computer. His specific storage vehicle was the epigenome, the conditioned side-kick of the genome (via molecules effecting the enfoldment of the genome’s packaging or chromatin).

In about half of the cases savant syndrome occurred concurrently with an autistic disorder and in the rest the underlying disorder was a brain injury or disease. Of particular interest were the prodigious savants whom Treffert believed would have been characterized as geniuses or prodigies if they didn’t have the coexisting disability. In this regard there is some overlap here with Einstein Syndrome.

Some distinguishing characteristics of prodigious savants include extraordinary memories and also exceptional but narrowly focused skills. Such savants are believed to be very rare with “probably fewer than 100 known prodigious savants living worldwide” [p.25]. Treffert described

five areas that the skills of savants appear to focus on - calendar calculating (usually finding the day of the week associated with a specified date), music, art, mathematical and number skills (including super-fast calculations), and mechanical or spatial skills [pp.19-22].

Treffert detailed some of the brain changes that have been found to be associated with savant syndrome [Treffert 2010, pp.48-54]. These typically involve some damage to the left hemisphere and then subsequent compensatory changes and efforts on the part of the brain's right hemisphere. Here is his somewhat concise description:

disruption of typical left hemisphere function from prenatal influences - such as detrimental hormonal effects on the cortex from circulating testosterone - or other injurious prenatal, perinatal or postnatal development in children and adolescents, or from later brain injury or disease in adults. These injuries produce compensatory right brain skills and abilities to offset left brain dominance. In addition there is, simultaneously, probably from those same detrimental factors, injury to the cortico-limbic (cognitive or semantic memory) circuits with substitution and reliance on (habit or procedural) memory circuits. This combination of left brain and cortico-limbic circuitry damage, with compensatory right brain skills and reliance on habit and procedural memory, produces the clinical picture that is savant syndrome [p. 54].

That there are corresponding changes in the brain is perhaps only part of the functional story. Was the memory exhibited by Stephen Wiltshire after his 15 minute helicopter ride or Kim Peek's book recall really feasible with a brain-only realization? In the *Blank Slate* Steven Pinker pointed out the intuitive when he wrote that "learning *is* a change in some part of the brain" [Pinker 2002, p.45]. You can sense this when you try to memorize a phone number or more subtly as you acquire a new habit. But can the biology of the human brain provide the extremely rapid changes implied by the recalls exhibited by Kim Peek and Stephen Wiltshire?

Another striking memory feat presented by Treffert came from Oliver Sacks' book, *An Anthropologist on Mars*. It involved a man named Franco Magnani who experienced a serious but unknown illness which had effects including "delirium" and "perhaps seizures" [Treffert 2010, pp.198-199]. After recovering "Magnani began painting immaculately accurate scenes from the village of Pontito [Italy] where he had grown up, but had left at age 18." In addition to the "digital-fidelity recall" the painting skills and interest appeared to come out of the blue. Magnani was quoted, "Fantastic. How could I do it? And how could I have had the gift and not known about it before?". Other examples of "massive autobiographical memory" or hyperthymesic syndrome

were also given. Again does such memory seem brain-only feasible? What about the associated energy demands?

In an apparent parallel, observations of non-savants with hyperthymesic phenomena was the focus of a February 2014 *Scientific American* article, “Remembrance of All Things Past” [McGaugh and LePort]. That article opened with an excerpt from an e-mail that the lead author James McGaugh had received from a woman named Jill Price:

As I sit here trying to figure out where to begin explaining why I am writing you ... I just hope somehow you can help me. I am 34 years old, and since I was 11 I have had this unbelievable ability to recall my past ... I can take a date, between 1974 and today, and tell you what day it falls on, what I was doing that day, and if anything of great importance ... occurred on that day I can describe that to you as well. I do not look at calendars beforehand, and I do not read 24 years of my journals either.

The authors then followed up and extensively tested Price’s recall of events and her memory was eventually proved faulty in one case - the day of the week of one of the previous 23 Easters (and Price is Jewish). Along the way she “corrected the book of milestones for the date of the start of the Iran hostage crisis at the U.S. embassy in 1979”. During tests of less significant dates Price:

correctly recalled that Bing Crosby died at a golf course in Spain on October 14, 1977. When asked how she knew, she replied that when she was 11 years old, she heard the announcement of Crosby’s death over the car radio when her mother was driving her to a soccer game [note there must be an error or typo in the article since Price couldn’t have been 11 years old in both 1974 and 1977].

She demonstrated an “immediate recall of the day of the week for any date in her life after she was about 11 years old”. Yet she “has trouble remembering which of her keys go into which lock” and “does not excel in memorizing facts by rote”. The remainder of McGaugh and LePort’s article chronicled their subsequent confirmation of similar extraordinary memories in about 50 people. Such memories were found to be “highly organized in that they are associated with a particular day and date” and that it occurred “naturally and without exertion”. The authors also did not find evidence that the phenomena tended to have a family history and thus some implied support for a genetic explanation. In any case, such phenomenal memories could be prompting some basic questioning amongst neuroscientists and others.

Back to Darold Treffert’s *Islands of Genius* where substantial efforts involved trying to account for the mysterious savant learning. The examples given in the book appeared to strongly

support his contention that “they indeed know things [and exhibit skills] that they never learned” [Treffert 2010, p.59]. In a 2014 *Scientific American* article Treffert stated that “[b]y 2010 I had assembled a worldwide registry of 319 known savants, of whom only 32 had the acquired form” [Treffert 2014]. In that article he concluded that “[a]cquired savantism provides strong evidence that a deep well of brain potential resides within us all”. In his *Islands of Genius* book he wrote that he believes that the epigenome (in particular as optimistically portrayed in the *NOVA* TV episode, “Ghost in Your Genes”) is the vehicle for such transmission and claimed simply “[b]ottom line: genetic memory exists” [Treffert 2010, pp.60-61]. There do not appear to be reports suggesting anything like this capability is plausible, though, and in fact researcher Eric Nestler acknowledged in an interview with *Scientific American* that any epigenetic inheritance effect is “controversial” [Nestler podcast].

I detour here into some descriptive content on the epigenome as it is relevant to Treffert’s genetic memory claim (and to some other extraordinary claims). A fine description of what is known of the epigenome, its mechanics, and possible mental health implications can be found in Eric Nestler’s *Scientific American* article, “Hidden Switches in the Mind” [Nestler]. The epigenome consists of the additional chemical markers which can indirectly effect the expression of genes by changing the packaging of the encompassing DNA (together the folded-up DNA and its supporting proteins are called chromatin). In particular, if the epigenetic markings influence the shape of the chromatin in such a way that some of the genes are tightly packaged (or tucked away) and thus not readable by RNA, then the expression (or copying) of those genes will be minimized (or switched off). In general as Nestler pointed out, “[t]he environment can influence gene activity by regulating the behavior of epigenetic writers and erasers - and thus the tagging, and restructuring, of chromatin”. This packaging dynamic appears to be part of normal genetic functioning - as for example in differentiating the gene expressions found in different cell types (i.e., liver cells versus muscle cells). This epigenetic dynamic also appears capable of long-term, unhealthy, conditioned impacts as potentially with addictions and mental illnesses. The potential for any heritable effects - and thus conceivably with prodigal inexplicable learning - is difficult to imagine, though.

Treffert’s confident claim - “bottom line: genetic memory exists” - would presumably entail the recording and downloading of complex neural patterns believed to embody high level learning to the germ line’s (or germ cell’s) epigenome; the subsequent reproductive passage of that encoding; and ultimately the uploading of that epigenetic encoding to the appropriate neural circuits to realize the otherwise inexplicable learning. More subtly, such epigenetic encoding would

presumably have to not interfere with normal epigenetic functioning and as part of this survive the two-fold stripping of epigenetic marks that is believed to accompany the reproductive process. If this weren't challenging enough, there would still have to be a historical reproductive lineage to support the specific learning. To explain Jay, the previously considered cello-playing prodigy, via such a "genetic memory" theory, you would still have to identify one or more ancestors who were involved with cello-playing and composing. As Treffert pointed out, though, in the realm of prodigious savants there are some in which there appear to be no "family history of special skills" [Treffert 2010, pp.37-39]. More generally, Treffert's optimism about the possibility of most or all of us having access to such epigenetic-encoded genius is highly unlikely from this perspective as there have been so few intellectually exceptional individuals in history (and unlike bacteria we can't simply transfer our DNA).

Before considering a possible transcendental take on some of the savant phenomena some comments on the principal source here, *The Tibetan Book of the Dead (TBD)*, are in order [Fremantle and Trungpa]. This book was apparently written in the 8th century by a Buddhist religious teacher named Padmasambhava and it contains instructions to aid a dying or recently deceased person in dealing with the presumed subsequent (post-death) intermediate or bardo state. This text was thus often read at the bedsides of the dying or recently deceased. The intermediate state was believed to be quite tumultuous but it also offered great potential to at minimum secure a good rebirth. The coauthor and late Tibetan teacher Chogyam Trungpa offered a modern synopsis in his commentary:

there is something which continues, there is the continuity of your positive relationship with your friends and the [religious or spiritual] teaching, so work on that basic continuity, which has nothing to do with the ego. When you die you will have all sorts of traumatic experiences, of leaving the body, as well as your old memories coming back to you as hallucinations. Whatever the visions and hallucinations may be, just relate to what is happening rather than trying to run away. Keep there, just relate with that [p.40].

Trungpa's commentary also emphasized an interpretation of underlying energies in the bardos. Another Tibetan teacher, Tulku Thondup, characterized the bardo experience as "like a dream journey, fabricated by our own habitual mental impressions" [Thondup, p.10]. Much of the TBD involves very explicit suggestions in particular to help the deceased realize their own ultimate nature and alternatively to simply avoid a bad rebirth (and written apparently for those with Tibetan Buddhist-flavored "habitual mental impressions"). The intentionality of the soul within the bardo

is viewed as a key and thus the repeated instructions to maintain an altruistic attitude dedicated to the betterment of “all sentient beings”.

It is the associated description of the post-death or bardo soul that is of particular interest here. It is stated several times that “in the bardo state the mind becomes nine times more clear” and also that the associated memory is such that even if the TBD was “heard ... only once and the meaning not understood” then after death “it will be remembered with not even a single word forgotten” [Fremantle and Trungpa, pp.167-168]. This claimed clarity and memory capability, though, would presumably compete against the claimed “visions and hallucinations”.

A crude transcendental explanation of savant syndrome could then begin with simple transcendental continuity and thus the inexplicable learning and interests were carried over from past lives. More particularly, if a person had been very interested and strongly habituated to an activity such as music then that tendency might continue in the intermediate state and ultimately result in a rebirth with very focused behavioral tendencies. In a physics-sense they caught a resonance and this carried over strongly into their subsequent life. Perhaps such a process could allow for some of the underlying “nine times more clear” soul-mind to shine through in a focused way and thus produce some of the spectacular prodigious savant feats. Analogously, the acquired savant syndrome could reflect neural setbacks that inadvertently opened a window for the functioning of the underlying soul-mind. On this note the similarity between some pre-epileptic seizure experiences (involving an “intense heightening of awareness” and not “abnormal or fantastic visions”) and some transcendental mystical awakening experiences [Sekida, pp.14-15] could then be literal and not neural-only phenomena (including of course hallucinations [Sachs]).

Finally, an additional very specific bardo description and possible savant connection comes from Tulku Thondup’s book, *Peaceful Death, Joyful Rebirth* [Thondup]. In it he wrote that:

[s]ome people relive their dying experiences, exactly as they went through them, on every seventh day after their death, again and again, especially if it was a tragic death. That is why every seventh day is observed by survivors with prayers and dedications [p.88].

Facing such a scenario would likely whittle down your perspective on things and strongly frame time in a cycle-of-7 (or modulo 7) days perspective. With savant syndrome the most common - “almost universally, present” - unusual ability and focus is with calendar calculating. This phenomena apparently is also present amongst hyperthymesic individuals (and such individuals also “scored higher on a test of obsessive personality traits”). Why and how this calendar calculating

happens is an enormous mystery. Central to it appears to be a fixation on time through a day of the week (or modulo 7) perspective.

The Flynn Effect

The third and final mystery considered here is the Flynn Effect. Philosopher James Flynn (and some less noted earlier researchers) noticed that IQ scores in many countries appeared to be rising during the twentieth century [Pinker 2011, Folger]. Although there is no shortage of controversy here, the apparent rising IQ's are not in question. The "bombshell" as Steven Pinker put it "is that the Flynn Effect is almost certainly environmental" [Pinker 2011, p.653]. How such an environmental dynamic could have evaded previous studies - and everyday perception - of intelligence is truly amazing.

The apparent intelligence gains are not subtle:

[a]n average teenager today, if he or she could time-travel back to 1950, would have an IQ of 118. If the teenager went back to 1910, he or she would have had an IQ of 130, besting 98 percent of his or her contemporaries. Yes, you [read] that right: if we take the Flynn Effect at face value, a typical person today is smarter than 98 percent of the people in the good old days of 1910. To state it in an even more jarring way, a typical person of 1910, if time-transported forward to the present, would have a mean IQ of 70, which is at the border of mental retardation. With the Raven's Progressive Matrices, a test that is sometimes considered the purest measure of general intelligence, the rise is even steeper. An ordinary person of 1910 would have an IQ of 50 today, which is smack in the middle of mentally retarded territory, between "moderate" and "mild" retardation [Pinker 2011, p.651].

The underlying gains have been largely in the abstract reasoning portions of intelligence tests such as those containing similarities, analogies, and visual patterns (including Raven's Matrices). Little if any gains occurred in the traditional main topics of education - knowledge, math, and vocabulary [Folger; Pinker 2011, p.651]. Thus, arguments connecting these gains to improvements in schooling appear to be inadequate.

Flynn feels that these increases in IQ scores reflect a pervasive shift in modern societies towards more focus on abstract reasoning [Flynn; Pinker 2011, pp.653-654]; Folger]. In particular, Flynn hypothesized that this shift involved "scientific reasoning" infiltrating "everyday thinking" on an increasingly wide scale [Flynn; Pinker 2011, p.655]. Steven Pinker offered an explanation - albeit perhaps an optimistic one - that many modern people have apparently "assimilated hun-

dreds of these [scientific] abstractions from casual reading, conversation, and exposure to the media, including *proportional, percentage, correlation, causation, control group, placebo, representative sample, false positive, empirical, post hoc, statistical, median, variability, circular argument, tradeoff, and cost-benefit analysis*” [Pinker 2011, p.655]. With such a hypothesized modern shift towards abstraction, Flynn suggested that “we developed new cognitive skills and the kind of brain that can deal with them” [Flynn].

The mystery associated with the Flynn Effect is how this could have happened given the relatively fixed nature of an individual’s intelligence quotient. As intelligence researcher Linda Gottfredson put it, “decades of genetics research have shown, ... [that] genetic [or innate] endowments are responsible for much of the variation in mental ability among individuals” [Gottfredson 1999]. Additionally, Gottfredson pointed out that:

[A]lthough shared environments do have a modest influence on IQ in childhood, their effects dissipate by adolescence. The IQs of adopted children, for example, lose all resemblance to those of their adoptive family members and become more like those of the biological parents they have never known.

Certainly some adoptions - perhaps particularly international ones - would seem to have realized in an environmental-sense something akin to the time-travel hypothesized by Pinker. So why hasn’t the Flynn Effect also been apparent via some of the environmental dynamics experienced by some individuals?

Additionally, the limited real-world import of these apparent gains in aptitude was vividly suggested by Linda Gottfredson’s citing of the complexity barriers encountered in a 1993 literacy survey of American adults [Gottfredson 2012]. Included was the observation that only 17 percent were able to use “a bus schedule to determine the appropriate bus for a given set of conditions” and only 3 percent were able to “answer the most complex questions, like determining the total cost of carpet to cover a room (using a calculator)”.

Nonetheless, the puzzling gains in our aptitude for abstract reasoning beg an explanation. From a transcendental perspective this could be explained as a Lamarckian-like effect due to the increased emphasis on abstract reasoning in the modern world. From this perspective there has been a transcendental boost in innate abstract reasoning abilities as souls have cycled thru (human) lives with more and more emphasis on abstraction. This would be similar to the earlier proposed explanations for the gains found with the Einstein Syndrome and savants, but without the large focal boosts. This would also be consistent with the innate intelligence differences found

between individuals (and group averages), differences which so far have been minimally correlated with DNA. Thus the differences in the intellectual demands, possibilities, and pursuits across transcendental trajectories could have produced different cumulative Lamarckian-like contributions to the innate intellectual capacities of individuals. If Gottfredson's complexity example is representative, though, the hypothesized transcendental boost in aptitudes has not been matched by a critical boost in intellectual motivations.

In any case such a transcendental process would place the environmental influence for contemporary intelligence score gains in the previous lives of individuals, with the most recent life perhaps being the dominant contributor. The alternative of trying to account for contemporary gains amidst modern society's increasingly distracted norms and reduced physical activity is difficult. Flynn in fact commented, "[t]o my amazement, in the 21st century the increases are continuing" and went on to add, "as if guided by an invisible hand" [Folger].

Discussion on Intelligence and its Conundrums

A prominent feature of any human being is their intelligence. This might be the biggest item on the behavioral genetics queue. In some cases an individual's intelligence really stands out, somewhat analogous to extremes in height. The basis of the variations in this basic human feature was supposed to be largely found in DNA, even apparently in the cases of exceptionally high intelligence. The inability to confirm that DNA basis is certainly a big deal and a good example of the missing heritability problem. Given the significance of the development of intelligence in our evolutionary history as evidenced by our much increased brain size, scientists certainly must have expected to find a number of pieces of DNA supporting that species dynamic (and initial support was described in [Pollard]) and further for its variation amongst individuals.

Does the explanation involving a default high level of intelligence with the variations being imposed in a downward fashion via random mutations seem plausible? A look at the remarkable high intelligence terrain poses immediate challenges to that explanation. Even without the particular conundrum of inexplicable learning, it is difficult to imagine how our evolutionary history could have resulted in codes for such high levels. At the very least the mysteries associated with our intellectual abilities should receive more recognition in scientific circles and be more commonly communicated to the public. The usual sense of certainty associated with scientific materialism should give way to an acknowledgement of some of these mysteries.

In another relevant academic area, one would think that philosophers would be jumping on such mysteries, particularly ones so close to knowledge. With the exception of one philosopher (who had demonstrated the temerity to argue for the existence of free will), though, none of my efforts to pass on this intellectual tip produced any response from the realm of philosophy. Moreover, I have seen no evidence that any philosopher is pursuing anything connected to the unfolding DNA mystery. Not too long ago as evidenced by the work in [Head and Cranston] some philosophers wondered about a possible transcendental process and its heritable implications. Also from studying possible cases of reincarnation, Stevenson and Tucker naturally wondered some about those implications [Stevenson; Tucker 2005. See for example the closing chapters].

My own perspective on the transcendental hypothesis has been somewhat conjoined to my interest in Buddhism. There must certainly have been numerous other earlier beliefs on the nature of a possible transcendental process, although perhaps only a few of them are still available in the modern world. On this point, I introduce some descriptions of the soul as possibly relevant to a transcendental explanation. In the *Tibetan Book of the Dead* there is a duality in such descriptions between a passive (or “emptiness”) aspect and an active (or “luminosity”) aspect. Here is an excerpt describing the disembodied post-death state:

[t]hese two, your mind whose nature is emptiness without any substance whatever, and your mind which is vibrant and luminous, are inseparable: this is the dharmakaya of the buddha. This mind of yours is inseparable luminosity and emptiness in the form of a great mass of light, it has no birth or death ... [Fremantle and Trungpa, p.87]

This appears to be an elemental description of the underlying transcendental soul. Of note in this depiction is that I am avoiding the longstanding muddled self-versus-no-self philosophical debate which entertains some Buddhists (although this certainly doesn't appear to describe a non-self).

Somewhat of a complement to the TBD can be found in the far-ranging and earnest discussions of the book *I AM THAT* based on talks with the late Indian teacher Nisargadatta. These discussions, like Nisargadatta, had a somewhat loose affiliation with the Hindu tradition. That tradition includes teachings from both a top-down or God perspective and also a bottom-up or soul perspective. In any case, the emphasis in those discussions appeared to be in moving towards a direct experience of - and hopefully ongoing foothold in - the soul's perspective (and thus the title of the book). Briefly, the depicted very big challenge was to maintain close observations of living experiences so as to appreciate phenomena as-is, without getting caught up in memory-based detours. Here is a simple subjectively-oriented excerpt in which Nisargadatta answered a question on personal differences:

[t]here is no difference between us; nor can I say that I know myself, I know that I am not describable nor definable. There is a vastness beyond the farthest reaches of the mind [i.e., workings of the brain]. That vastness is my home; that vastness is myself. And that vastness is also love [Nisargadatta, p.530].

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