

Bringing schizophrenia into the Darwinian fold

Eric Wright

A search through the psychology literature reveals a number of current popular evolutionary theories on the origin of schizophrenia. These Darwinian theories assume the fact of evolution, and then from this starting point go on to make unsubstantiated and implausible speculations, often untestable or practically so. Where testable, the scenarios are easily refuted, as they do not correlate with research findings about schizophrenia.

Schizophrenia is a debilitating mental disorder in which a person's contact with reality and normal functioning is severely impaired due to some of the following symptoms: delusions (unshakable false beliefs), hallucinations (false sensory experiences), disorganized and illogical speech, bizarre behavior, and negative symptoms (lack of emotion, speech or initiative).¹ The word means 'splitting in the mind',² but not split or multiple personality.

The underlying cause and even site in the brain of the disorder is not known and there is no test to confirm or deny diagnosis. Thus, defining what is or is not 'schizophrenia' for research and treatment purposes may be difficult because 'the diagnosis of schizophrenia is based on the combination of several clinical features, none of which is unique to schizophrenia'.³ It has even been argued that all psychotic disorders (where there is 'loss of contact with reality'), including schizophrenia and bipolar disorder (manic depression), should be conceptualized as part of the same continuum of psychotic illnesses.^{4,5} The fact there are a number of subtypes of schizophrenia, as well as great variety in the age of onset and course of the illness, suggests multiple causes.⁶ Theories include genetic, developmental, viral, immunological, biochemical, nutritional, and stress causes, but they are not all mutually exclusive.⁷ For example, biochemical theories of schizophrenia usually have a genetic basis. Also, the neurodevelopmental hypothesis, which is currently the favoured model, where abnormality in early brain development is cited as the main cause of schizophrenia, several factors have been suggested or implicated in developmental defects, including genetic and epigenetic, obstetric factors, and intrauterine environmental stresses such as *in utero* exposure to influenza.⁸⁻¹⁰

A strong argument for the genetic influence of schizo-

phrenia is that in twins the concordance rate for schizophrenia is about 50% in monozygotic (identical) twins, who share 100% of the genes, compared to 15% for dizygotic (fraternal) twins and a risk of about 1% for the general population.¹¹ In addition, when monozygotic twins have been reared apart their concordance rate is similar to that of monozygotic twins reared together, a finding that rules out a more similar environment for monozygotic twins as a causative factor in the different concordance rates between fraternal and identical twins.¹² However, as the concordance rate for schizophrenia is not 100% there must also be non-genetic factors involved. As single major locus models, consisting of one dominant or recessive gene, appear inadequate to explain many observations about schizophrenia, models allowing for several genes, or a combination of genetic and environmental factors, have been proposed.¹³ Although genetic linkage analysis has implicated many loci, on nearly a dozen chromosomes, there are as yet no definitively identified schizophrenia susceptibility genes.^{14,15}

Allen and Sarich acknowledged in 1988 that

'While it would be misleading to state that the evolutionary perspective has never been applied in the study of schizophrenia, it is fair to say that it has had little or no influence on the conventional thinking about schizophrenia.'¹⁶

However, the failure of the medical model (the current prevailing view) to adequately explain the cause of schizophrenia, and its mixed success in treating the illness, has recently seen a push from different directions to put evolutionary theory on center stage. For example, books advocating evolutionary psychiatry have been written arguing that evolutionary approaches are best equipped to treat and explain mental disorders.^{17,18} '*Evolutionary psychiatry* is the application of the disciplines of ethology, sociobiology, and neo-Darwinistic anthropology to human psychiatric populations'.¹⁹ This article critically examines evolutionary theories on the origin of schizophrenia. These theories are based on naturalism, a philosophy that excludes the supernatural and spiritual, and views man as just another animal without soul, spirit and purpose.

Bicameral minds

The late psychologist Julian Jaynes, a teacher who lectured in the Psychology Department at Princeton University, published some very speculative and unorthodox ideas about human consciousness. Jaynes suggested that language evolved during the late Pleistocene, approximately from 70,000 BC to 8,000 BC, a period when he believed the 'hominid population exploded out of the African heartland'.²⁰ During this time when language was said to have evolved, Jaynes suggested that verbal auditory hallucinations 'were a side effect of language comprehension which evolved by natural selection as a method of behavioral control'.²¹ The beginning of the bicameral mind, considered as an

'offshoot of the acquisition of language',²² was suggested by Jaynes to have occurred with the invention of names at about 10,000 BC to 8,000 BC, which is around the time when he proposed that there was archaeological evidence for the beginning of civilization. Jaynes hypothesized that civilization only became possible with 'the social organization provided by the bicameral mind'.²³ So, what is a bicameral mind? By bicameral mind Jaynes is referring to his idea 'that at one time human nature was split in two, an executive part called a god, and a follower part called man. Neither part was conscious'.²⁴ The man part of the bicameral mind was situated in the left or dominant hemisphere, with the god part of the bicameral mind supposedly structured such that 'the speech of the gods was directly organized' in the area of the right hemisphere that corresponds to the left-sided Wernicke's area 'and 'spoken' or 'heard' over the anterior commissures to or by the auditory areas of the left temporal lobe'.²⁵

The bicameral mind supposedly lasted until about 1,000 BC, with its subsequent breakdown resulting in man acquiring consciousness for the first time. According to Jaynes, the bicameral mind started breaking down as a result of several causes, including 'the weakening of the auditory by the advent of writing; the inherent fragility of hallucinatory control; the unworkableness of gods in the chaos of historical upheaval ... and a modicum of natural selection'.²⁶ The moderate amount of natural selection was that the advent of consciousness offered survival value to those most able to learn it.

The bicameral mind hypothesis of Jaynes was in large part based on a subjective and controversial interpretation of ancient literature, sculptures, and other artifacts of earlier civilizations. He viewed the prophecies of the Bible as the product of hallucinations from bicameral minds, and the decline of prophecies in the post-exilic period of Judaism as due to the loss of the bicameral mind. The Fall of Man, as described in Genesis, was suggested as a possible myth 'of the breakdown of the bicameral mind and the coming of consciousness'.²⁷ Instead of God speaking to Abraham or Moses, Jaynes attributed what they heard to bicameral voices, for example, 'the bicameral voice of Moses possibly has a sudden impulse to kill him (Exodus 4:24) for no reason at all'.²⁸ It should be noted that Jaynes failed to mention the phenomenon of fulfilled Bible prophecy,²⁹ which completely demolishes the argument that the inspiration of the prophecies were bicameral voices, as these voices, attributed to being purely a product of the brain (excluding any external source), could not have accurately predicted events in the distant future. The idea that humans before 1,000 BC lacked consciousness yet managed to build pyramids, develop complicated social structures and write the books of Job and Psalms is ludicrous. Also, the post-exilic lack of prophecy did not commence until about 400 BC. Between 1,000 BC and 400 BC prophetic words were received by Isaiah, Jeremiah and the Minor Prophets. If their prophecies were bicameral hallucinations, then other men who lived during this time



If the bicameral mind theory of Julian Jaynes is true, then these pyramids were built by robot-like 'pre-conscious' humans.

in history such as Buddha, Aesop, Confucius and Socrates might be accused of also being 'pre-conscious'!

In Jaynes' theory 'schizophrenia, at least in part, is a vestige of bicamerality, a partial relapse to the bicameral mind'.³⁰ Other partial vestiges of the hypothesized bicameral mind include hypnosis, possession, and artistic practices such as music and poetry. Auditory hallucinations in right handed people with schizophrenia are viewed as originating in the right hemisphere and are seen as reflecting the role of the god part of the bicameral mind, 'that of initiating and guiding the patient's behavior'.³¹ Jaynes' theory is considered to be outside mainstream psychiatric thought, with Asaad and Shapiro stating 'The difficulty which we find with Jaynes' hypothesis is that the conclusions he draws have a questionable basis in neuropsychiatric fact'.³² They went on to write, 'Most clinicians who have worked with psychiatric patients would realize how simplistic an explanation the bicameral mind hypothesis is when faced with the myriad forms of hallucinations'.³³ Right hemisphere involvement in auditory hallucinations has some support,³⁴ but so does left hemisphere involvement. A recent review of the literature concluded that 'Functional imaging data are inconsistent, yet point to the left superior temporal gyrus as one of the neural substrates for AVH [auditory verbal hallucinations]'.³⁵ Most likely the phenomena of AVH are not confined to the involvement of a single cerebral hemisphere. Currently all ideas on the origins of auditory hallucinations in schizophrenia are very speculative, but the bicameral mind theory postulating a transitional form of humans is refuted by historical facts. Applying a debunked theory to schizophrenia is therefore illogical. Even if some research findings of schizophrenia match those predicted by the theory these can never validate the hypothesis.

Paying the price for language

Timothy Crow, a psychiatrist at the Warneford Hospital in Oxford, UK, has arguably developed the most prominent evolutionary theory on schizophrenia to date. A reason why Crow proposes a Darwinian theory on the origin of schizophrenia is that a procreative disadvantage is associ-

ated with the disease and hence, if the illness has a genetic basis then natural selection should have selected the genes for schizophrenia out of the population within a few generations.^{36–38} To explain the persistence of schizophrenia genes in the population, in a manner consistent with evolutionary theory, he suggests that there must be a genetic advantage in human populations retaining genes that predisposes to schizophrenia, although not to the sufferers of the illness themselves.³⁹ In Crow's hypothesis the schizophrenic illnesses

'are a manifestation of genetic diversity in the evolution of the specifically human characteristic of language, an innovation that has occurred by a process of progressive hemispheric specialization—the establishment of dominance for some critical component of language in one or the other hemisphere'.⁴⁰

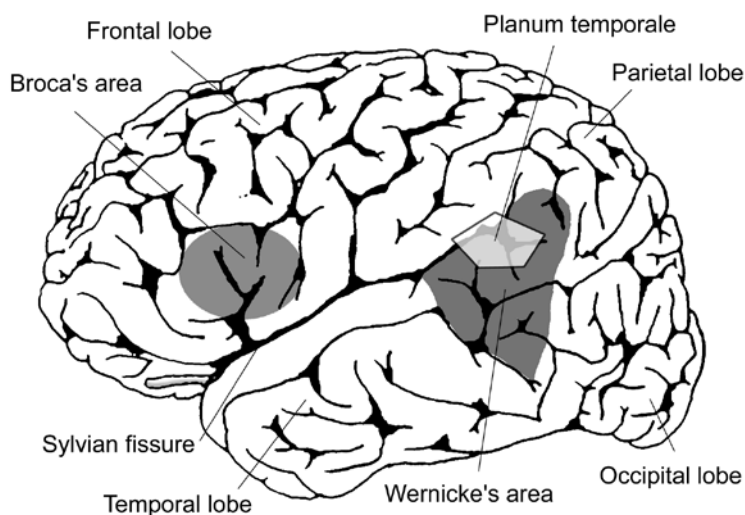
Crow argues that schizophrenia is the price *Homo sapiens* pay for the acquisition of language, that is, 'language and psychosis have a common evolutionary origin',⁴¹ with language tracing its origin back to the speciation event in East Africa, between 100,000 and 250,000 years ago, which is believed by many evolutionists to have given rise to modern *Homo sapiens*. It is suggested that this speciation event, and hence the origin of language and psychosis, was triggered by a critical mutation of a gene on the sex chromosomes that permitted the two cerebral hemispheres to develop asymmetrically.⁴² According to the theory, the disadvantage of a predisposition to schizophrenia is balanced out by the advantage (language) that this genetic variation gives to the population as a whole.

A cornerstone of Crow's theory is that there is a gene for cerebral asymmetry carrying the variation predisposing to psychotic illness, that this asymmetry gene is present in homologous form on both the X and Y chromosomes, and that language, psychosis and cerebral asymmetry have a common evolutionary origin in a critical change occurring on this sex chromosome gene.^{43–45} Although skeptical of the positive claims of linkage to autosomes made by others, Crow admits that the case for a determinant of cerebral asymmetry being 'relevant to genetic predisposition to psychosis is weak in that linkage findings on the X chromosome ... are no stronger than a number of the above claims relating to autosomes'.⁴⁶ This skepticism regarding linkage findings to autosomes has some justification, as many of the positive claims for linkage have later failed to be replicated.^{47,48} A prominent researcher has commented in regards to the genetics of schizophrenia: 'The crisis apparent in psychiatric genetics today is how one detects the true from the false "positives" and how to design studies that will lead to progress from this point on'.⁴⁹ The finding by DeLisi *et al.* 'that linkage for schizophrenia or

schizoaffective disorder on the X chromosome is absent'⁵⁰ is strong evidence against Crow's theory that a gene related to cerebral asymmetry on the sex chromosomes is implicated in the genetic predisposition to schizophrenia.

Important to Crow's argument is that individuals with schizophrenia show lesser anatomical asymmetries compared with the general population, with this reduced lateralization possibly reflecting the 'dominance failure' for language.⁵¹ According to Crow the planum temporale (PT), which has a pronounced asymmetry of the human brain, is the obvious candidate to carry 'a change in schizophrenia that is lateralized'.⁵² The PT forms the upper surface of the posterior region of the temporal lobe and is part of Wernicke's receptive language area.⁵³ Apart from being a key language area, the PT is larger on the left side in about two thirds of normal human brains.⁵⁴ With regards to anatomical asymmetry of the PT in schizophrenia some magnetic resonance imaging studies have indicated a reversal of the typical left greater than right asymmetry in the PT, or a volume reduction in the left PT.⁵⁵ However, it has been suggested that the diversity in results for PT data in schizophrenia can be explained by several variables not being adequately controlled for.⁵⁶ A recent study⁵⁷ on a large sample found no support for a reduction in leftward asymmetry of the PT in schizophrenia. Hence, the case for a reduction in anatomical asymmetries of the brain in schizophrenia, as needed by Crow's theory, is far from established.

Even if evolution were valid, which it is not, a major problem with Crow's evolutionary scenario is the strong evidence that the cerebral hemispheres were already asymmetric in one of the most obvious language regions before the



Lateral view of the left hemisphere of a human brain. The cerebral cortex (the outer grey matter) of the brain is divided up into four main regions (the frontal, parietal, occipital and temporal lobes). Broca's area and Wernicke's area are two key language related areas of the human brain. The planum temporale lies below the surface within Wernicke's area, and is visible only when the Sylvian fissure is opened (after Geschwind).⁹²

supposed acquisition of language, as indicated by findings of PT asymmetry in chimpanzees and other great apes.^{58,59} Gannon *et al.* found left hemisphere predominance of the PT homologue in the temporal lobe of chimpanzees paralleling that of humans (asymmetry of the PT was believed to be unique to humans).⁶⁰ Hence, how can the origin of language be attributed to the development of cerebral asymmetry in the PT if this region is already asymmetric in apes, which do not have the capacity for language? Whether the hypothetical common ancestor of apes and humans, which supposedly lived around 6 million years ago, had cerebral asymmetry, or apes evolved it independently, neither scenario gives credence to the idea that brain asymmetry is critical to language. Crow's response to the findings by Gannon *et al.* was to express skepticism concerning the PT measurements, citing disagreements as to how it should be measured in man, as well as pointing out inconsistencies in the literature regarding primate asymmetries.⁶¹ However, the even more recent finding⁶² of asymmetry in three great ape species of a homologue of Broca's area, which is a key region for speech production in humans, with a left hemisphere dominance similar to that seen in the equivalent area of humans, is further evidence against Crow's argument that cerebral asymmetry and language have the same origins.

Essential components of Crow's evolutionary hypothesis are refuted when examined in the light of available scientific evidence. Even if some predictions of Crow's theory fit the facts about schizophrenia, that the cornerstone of Crow's arguments have been invalidated renders further attempts at squeezing data about schizophrenia into this debunked theory illogical.

Evolutionary psychiatry

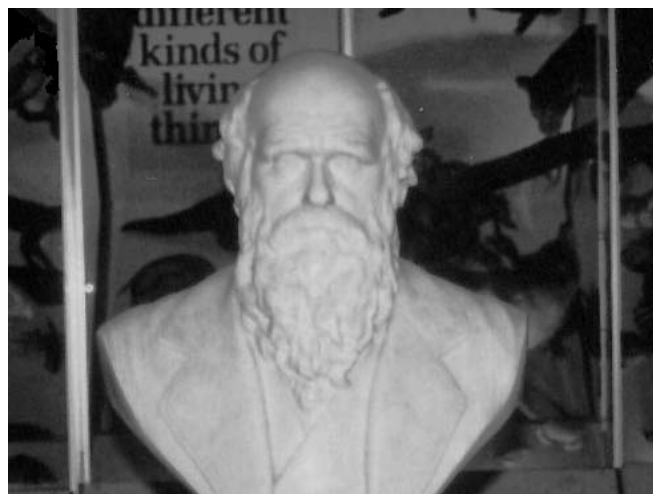
In their book *Evolutionary Psychiatry*, Anthony Stevens and John Price speculate on the causes of human psychopathology based on the argument 'that psychiatric symptoms are manifestations of ancient adaptive strategies which are no longer necessarily appropriate but which can best be understood and treated in an evolutionary and development context'.⁶³ In their 'group splitting' hypothesis of schizophrenia the authors suggest that schizotypal leaders, and hence their genes, evolved as an ancient evolutionary strategy to split up human groups that became too large. Some members of the old large group would follow these schizotypal charismatic leaders to form a new group, while others remained with the old group. By being accepted as a leader the schizotypal individual, who may well be predisposed to developing schizophrenia,

'is protected from psychosis by the adulation, affirmation and authority conferred on him by his group. It follows that if for any reason his charismatic career should be cut short, the resultant loss of status could induce a breakdown, whether schizophrenic, schizo-affective, or purely depressive in nature.'⁶⁴

In general, their hypothesis is based on speculations that cannot be tested. For example, how can it be known what caused the split up of the hypothetical ancient hunter-gatherer groups. Any number of reasons could be suggested, including family disputes, scarcity of resources, wars, etc. It would not necessitate one or both of the leaders of the newly formed groups being schizotypal charismatic leaders. However, such opposing argument story telling is unnecessary, as all such speculations are 'just so stories' based on the assumption that evolution has occurred. Take away the assumption of evolution, and the evidence for the 'group splitting' hypothesis is gone also.

Darwinian psychiatry

Michael McGuire and Alfonso Troisi, in the book *Darwinian Psychiatry*, 'argue that evolutionary theory can and should serve as the general theoretical framework for explaining and treating mental disorders'.⁶⁵ In general, the book is abundant in broad or vague generalizations based entirely on the assumption that evolution is a fact. If evolutionary theory were to form the basis for treating mental disorders then evolutionary explanations for disorders must be backed up with hard evidence, a point that seems to have gone unnoticed by the authors. Describing conditions or disorders, and then offering unsubstantiated evolutionary speculations, based on the assumption that evolution is a fact, as to why people with these conditions or disorders behave the way they do, as much of the book does, is not evidence that evolutionary explanations are correct. The same conditions and disorders can be made to fit just as well or better into other non-evolutionary models. Saying that evolutionary theory addresses strategies where, for example, 'Internal strategies might include hallucinations



A bust of Charles Darwin at The Natural History Museum, London. The veneration and influence of Charles Darwin continues non-stop. Since the publication of *Origin of Species* over 140 years ago, few areas have been left without some sort of Darwinian interpretation, and schizophrenia is no exception. There is even an emerging 'Darwinian Psychiatry'.

to create a private and safer world',⁶⁶ tells us nothing about the brain mechanisms involved in hallucinations, or how these mechanisms supposedly evolved.

In their evolutionary analysis section of their short chapter on schizophrenia the authors suggest that it is likely there are multiple causes of the 'suboptimal traits' which are responsible for the majority of disorders such as schizophrenia, but that 'information-processing deficits' is the net effect. 'In this model, genetic loading is not specific for schizophrenia, but for suboptimal traits, and depending on the type of loading and subsequent events, different conditions occur'.⁶⁷ What they do not explain is why evolution has kept on preserving in the human population a disorder such as residual schizophrenia, which is viewed as 'minimally adaptive'. In reading their *Preface* one learns that this is not necessary, as

'to reason this way is to misread the workings of evolution, which do not promise species perfection, only that some genes and traits are more likely to be preserved than others. Moreover, those features that are preserved are often far from ideal, at least from a psychiatric perspective.'⁶⁸ If you still do not get it then 'Said another way, it is an error to assume that *Homo sapiens* has been selected to be mentally healthy.'⁶⁸

In summary, the evolutionary approach offered by McGuire and Troisi is so vague, plastic and unsubstantiated as to be useless as a framework for explaining schizophrenia, or other mental disorders for that matter, let alone treating people with such disorders.

Abnormal connections

In one of the less prominent theories it has been suggested that schizophrenic symptoms are due to abnormal functional connections between specialized brain regions, caused by nerve fibers linking functional areas in the brains of schizophrenic people that would not be connected in normal people.⁶⁹ In this hypothesis abnormal brain connections are viewed 'to be a natural, inevitable, and essential consequence of the evolutionary expansion and advancement of the brain'.⁷⁰ Basically Randall suggests that in order for evolution to have brought about the emergence of new brain functions, such as language capability, the brain would have needed to increase its complexity, which would have involved forming more connections within and between existing functions by biological trial and error. Hence, these

'New and unique ('extra') connections could thus arise which could confer a special talent or ability on the individual who possessed them; equally possible is that new connections could produce an aberration of integration either within the function concerned, or between it and one or more others.'⁷¹

This 'aberration of integration' was suggested as

the mechanism underlying the schizophrenia syndrome. It has not been established that there are abnormal brain connections in people with schizophrenia, but even if this were the case, attributing it to a consequence of brain evolution without additional evidence, based entirely on the assumption of evolution, amounts to little more than another evolutionary 'just so story'.

Nocturnalism

According to the nocturnal theory presented by Jay Feierman

'schizophrenia is caused by genetically nocturnal variant individuals being *normatively coerced* to be active during the light of day or under conditions of artificial lighting. Under this coercion, such an individual would be processing information while awake with a brain that is processing information as though he were asleep.'⁷²

After suggesting that the brain is functionally in sleep mode while the individual is awake, Feierman then uses this as a basis to speculatively explain certain observations and symptoms characteristic of the schizophrenia syndrome. For example, while awake with his brain functionally in sleep mode, the author suggests that the schizophrenic individual could be 'aware of the information transformation and processing between short-term and long-term memory'.⁷³ This could lead to the individual experiencing hallucinations, which are one of the characteristic symptoms of schizophrenia. As to the selective advantage that allowed this genetic trait for schizophrenia to enter the human gene pool, and subsequently be maintained in the pool, a previous selective advantage in hunter-gatherer bands in which humans evolved is suggested. In this hypothesis 'nocturnal individuals, who resulted from the random mating of diurnal carriers, may subsequently have provided vigilance for their night-sleeping kin'.⁷⁴ Hence, the protection, through night vigilance, given to the hunter-gatherer band by having a nocturnal individual around whilst the diurnal individuals were sleeping, would be the advantage. A heterozygote selection advantage, such as heterozygote carriers of the nocturnal gene benefiting by having a greater chance of being closer spatially during sleep to a night vigilant relative, than either nocturnal or diurnal homozygotes would be, was suggested as a mechanism that could maintain the nocturnal (schizophrenia) gene in the human gene pool.

A testable prediction of the theory was that under conditions of constant darkness 'many of the major signs and symptoms of clinical schizophrenia' would disappear.⁷⁵ However, Feierman was later forced to abandon this prediction, and hence modify his original theory, as the case study of a schizophrenic patient who had enucleated both his eyes (removed the eyeballs) indicated no obvious signs of improvement in the patient several years after he became blind.⁷⁶ In that later publication Feierman acknowledged, 'that at this time there is minimal evidence supporting

specific elements in the author's particularly evolutionary theory of schizophrenia and its generative hypothesis', and also that the theory lacked 'any simple way to test its utility'.⁷⁷ Hence, the onus should not be on others to debunk this implausible evolutionary tale, but on Feierman to provide evidence for it.

Creativity and sexual selection

In his book *Strong Imagination* Daniel Nettle argues there is a link between increased risk of psychosis and being gifted in the creative arts. Nettle suggests that the 'bad genes' for psychosis are not, and were not, in the past eliminated by natural selection because of the compensatory advantages they bring to the human gene pool, that is, the traits of enhanced creativity. It is argued that being gifted with creativity is highly attractive, and therefore the traits for creativity would be maintained in the population by sexual selection at higher levels 'than would be adaptive for more utilitarian reasons'.⁷⁸ Nettle believes it is unlikely that there is a single psychosis gene, but instead that 'there are many genes, the variants of which contribute to variation in the human personality',⁷⁹ including a dimension which he refers to as psychoticism. In the theory, it is the relatives of a psychosis patient, those that carry the genes for psychoticism but who avoid developing its negative side (e.g. schizophrenia), that reap the enhanced creativity benefit (e.g. 'the ability for unorthodox, divergent, lateral thinking'⁸⁰) of the genes for psychoticism. While viewing the underlying cause of psychosis as due to genetics, Nettle also emphasizes an important role in the environment in terms of triggering the development of psychosis in individuals that are high up on his psychoticism scale, and hence at greater risk of developing psychotic disorders.

Nettle admits that his 'creativity-benefit' argument only works if creative achievement was as valuable to our supposed hunter-gatherer ancestors as it is said to be for us. Sexual selection theory, as put forward by Geoffrey Miller,⁸¹ is employed to provide a theoretical framework for keeping the traits for creativity, and hence the genes for psychosis, in the human gene pool in the past when they would have provided little practical benefit. David Horrobin, in his book *The Madness of Adam and Eve*,⁸² argues there is a link between creativity, schizophrenia and the speciation event that led to modern humans, but rejects sexual selection theory as a primary explanation. On Miller's sexual selection theme Horrobin states 'he has provided no mechanism for how selection might specifically have generated change, nor for how sexual selection pressures could explain the many genetic features which make us human'.⁸³ Horrobin uses the terms 'evolutionary fairy tales' and 'Kiplingesque just-so stories' to describe some of the concepts that have been used to explain how human brains evolved, arguing that because they lack precise details about genetic changes the 'hypotheses are effectively untestable and unprovable'.⁸³ Although not addressed directly by Horrobin, evolutionary

fairy tale aptly describes Nettle's sexual selection theory of psychosis.

Creativity and fat mutations

Horrobin suggests that schizophrenia entered the human race prior to the 'out of Africa' diaspora and that it was the development of several mutations, the last one perhaps between 150,000 and 130,000 years ago, which made us human, but also brought us schizophrenia. 'Genetic changes in the chemistry of the fat inside our skulls',⁸⁴ particularly phospholipid metabolism, are proposed as the key mutations. At the time the last mutation occurred it is suggested that the pathological behavior brought about by schizophrenia was relatively mild because the diet at the time, rich in fatty acids, attenuated the effects of the biochemical changes caused by the mutations. At the same time a surge of creativity is suggested as being unleashed as a result, this being the positive aspect of the mutation. However, in more recent times, the attenuation effect are said to be no longer as evident because of a change in diet, and so psychosis is much more prevalent in our society.

Horrobin's idea of giving essential fatty acids to schizophrenia patients may have a limited therapeutic effect in some patients.⁸⁵ However, even in the study in question the majority of patients still required standard drug therapy to control their condition in addition to the fatty acid treatment. Whilst Horrobin would claim any therapeutic effect of fatty acids as support for his evolutionary theory, the benefits of the fatty acids support nutritional theories,⁸⁶ and need not be tied in with an evolutionary approach.

The first of the 'fat' mutations were suggested by Horrobin to have occurred in our last common ancestor with the chimpanzee, about 5–6 million years ago. From this he believes that a testable element of his 'just-so story' can be known within 20 years, that is, 'whether the key mutations that differentiate us from the great apes are in the genes coding for proteins which regulate fat metabolism'.⁸⁷ This might be so if evolution is the only explanation allowed, which unfortunately is the case in most of academia. However, if creation theory was given a voice in this setting then it would make the point that an Intelligent Designer could have created differences in the genes between apes and humans, including those that code for proteins which regulate fat metabolism. Hence, Horrobin's prediction would be useless in terms of ruling out intelligent design.

Other evolutionists have questioned aspects of Horrobin's theory. McCrone comments that there is a huge hole in the central premise of Horrobin's book because it 'never spells out how a faulty metabolism could change the connectivity of a schizophrenic brain'.⁸⁸ Nettle states, 'if schizotypal genes have an overwhelming selective advantage, as Horrobin supposes, then they should become universal in the population'.⁸⁹ If this had happened, then the recent change in diet for the worse, no longer rich in

fatty acids, resulting in the negative aspects of the ‘fat mutation’ being unleashed, should have resulted in a far greater incidence of schizophrenia in the population than about 1%. Hence, if Horrobin’s hypothesis is still correct then what about the majority of the population who display no signs of psychosis? Are most of us ‘still relatively dull and boring’⁹⁰ pre-humans, missing the final fat mutation, or are we still psychosis prone, waiting for the environmental trigger to drive us over the edge? More likely it is Horrobin’s hypothesis that is wrong.

Conclusion

The evolutionary scenarios examined all start with the assumption that evolution is a fact. The authors have obviously not examined (or are ignoring) the evidence refuting evolution.⁹¹ Whatever the shortcomings of current explanations and treatments for schizophrenia, applying evolutionary theories to the problem is analogous to turning off a dim light in a windowless room in order to see better. This is because evolutionary theories on the origin of schizophrenia start off by assuming the answer that they should instead be trying to prove, that is, whether evolution is true and hence whether it is even possible for schizophrenia to be a product of evolution. The evolutionary scenarios examined are also wildly implausible, often presented with little or no supporting evidence, and some are untestable or practically so. Where testable the scenarios are easily refuted as they do not correlate with research findings about schizophrenia. Since evolutionary explanations of schizophrenia are groundless, treating it as a Darwinian disorder is hardly the best model to use in trying to discover the cause or cure for the illness.

Rather, getting sidetracked down an evolutionary dead end could harm such efforts. There is also the danger that some remedies from an emerging Darwinian Psychiatry, based on the flawed assumption of evolution, could harm rather than benefit the patient. Also, would a Darwinian psychiatry result in the patient being indoctrinated ‘cult-like’ into believing the false ideology of Darwinism?

Another concern is that the false notion of schizophrenia as an evolutionary disorder will open a Pandora’s Box. There is the danger that the mentally ill, and subsequently other groups in society, will become falsely perceived by elements of some societies, and/or by totalitarian regimes in some countries, as being less evolved, and hence genetically or biologically inferior. The dark history of eugenics, particularly the atrocities of the Nazis, is a warning.

The lack of evidence for evolutionary theories of schizophrenia brings up a point that cuts to the heart of Darwinism. Most researchers believe there is a genetic component to schizophrenia, as well as an environmental factor. How is it then that a genetically transmitted disorder, which occurs in similar proportions across cultures and is associated with a procreative disadvantage, can persist in the population for thousands of generations if it is being

selected against? Granted, there are many unknowns with respect to schizophrenia, but we will look in vain to find the answers in Darwinism.

References

1. *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, DSM-IV, American Psychiatric Association, Washington D.C., p. 285, 1994.
2. Reber, A.S., *The Penguin Dictionary of Psychology*, Penguin Books, London, p. 666, 1985.
3. Carpenter, Jr, W.T., Buchanan, R.W., Kirkpatrick, B., Tamminga, C. and Wood, F., Strong inference, theory testing, and the neuroanatomy of schizophrenia, *Archives of General Psychiatry* **50**:826, 1993.
4. Crow, T.J., A continuum of psychosis, one human gene, and not much else—the case for homogeneity, *Schizophrenia Research* **17**:135–145, 1995.
5. Crow, T.J., Is schizophrenia the price that *Homo sapiens* pays for language? *Schizophrenia Research* **28**:127–141, 1997.
6. Carpenter, Jr, W.T., Buchanan, R.W., Domains of psychopathology relevant to the study of etiology and treatment in schizophrenia; in: Schulz, S.C. and Tamminga, C.A. (Eds), *Schizophrenia: Scientific Progress*, Oxford University Press, New York, pp. 13–22, 1989.
7. Torrey, E.F., *Surviving Schizophrenia*, Revised Edition, Harper & Row, New York, pp. 129–169, 1988.
8. Bassett, A.S., Chow, E.W.C., O’Neill, S. and Brzustowicz, L.M., Genetic insights into the neurodevelopmental hypothesis of schizophrenia, *Schizophrenia Bulletin* **27**:417–430, 2001.
9. Waddington, J.L., Schizophrenia: developmental neuroscience and pathology, *The Lancet* **341**:531–536, 1993.
10. Weinberger, D.R., From neuropathology to neurodevelopment, *The Lancet* **346**:552–557, 1995.
11. Keefe, R.S.E. and Harvey, P.D., *Understanding Schizophrenia: A Guide to the New Research on Causes and Treatment*, The Free Press, New York, p. 82, 1994.
12. Yoneda, H., Chihara, S., Kidogami, Y., Tsuji, T., Ishida, T., Nonomura, Y., Inayama, Y., Kono, Y., Asaba, H. and Sakai, T., Molecular genetics in schizophrenia; in: Nakazawa, T. (Ed.), *Biological Basis of Schizophrenic Disorders*, Japan Scientific Societies Press, Tokyo, pp. 231–240, 1991.
13. Straube, E.R. and Oades, R.D., *Schizophrenia: Empirical Research and Findings*, Academic Press, Inc., San Diego, pp. 372–376, 1992.
14. Bray, N.J. and Owen, M.J., Searching for schizophrenia genes, *TRENDS in Molecular Medicine* **7**:169–174, 2001.
15. Tsuang, M., Schizophrenia: genes and environment, *Biological Psychiatry* **47**:210–220, 2000.
16. Allen, J.S. and Sarich, V.M., Schizophrenia in an evolutionary perspective, *Perspectives in Biology and Medicine* **32**:133, 1988.
17. McGuire, M. and Troisi, A., *Darwinian Psychiatry*, Oxford University Press, New York, 1998.
18. Stevens, A. and Price, J., *Evolutionary Psychiatry: A New Beginning*, Second Edition, Routledge, London, 2000.
19. Feerman, J.R., A testable hypothesis about schizophrenia generated by evolutionary theory, *Ethology and Sociobiology* **15**:263, 1994.
20. Jaynes, J., *The Origin of Consciousness in the Breakdown of the Bicameral Mind*, Houghton Mifflin Company, Boston, p. 130, 1990.
21. Jaynes, Ref. 20, p. 134.
22. Jaynes, Ref. 20, p. 216.
23. Jaynes, Ref. 20, p. 149.
24. Jaynes, Ref. 20, p. 84.
25. Jaynes, Ref. 20, p. 105.
26. Jaynes, Ref. 20, p. 221.
27. Jaynes, Ref. 20, p. 299.

28. Jaynes, Ref. 20, p. 304.
29. Morris, H.M. and Morris, H.M. III, *Many Infallible Proofs*, 2nd Edition, Master Books, Green Forest, pp. 189–207, 1996.
30. Jaynes, Ref. 20, p. 405.
31. Jaynes, Ref. 20, p. 411.
32. Asaad, G. and Shapiro, B., What about the bicameral mind? *American J. Psychiatry* **144**:696, 1987.
33. Asaad and Shapiro, Ref. 32, p. 696.
34. Shergill, S.S., Brammer, M.J., Williams, S.C.R., Murray, R.M. and McGuire, P.K., Mapping auditory hallucinations in schizophrenia using functional magnetic resonance imaging, *Archives of General Psychiatry* **57**:1033–1038, 2000.
35. Stephane, M., Barton, S. and Boutros, N.N., Auditory verbal hallucinations and dysfunction of the neural substrates of speech, *Schizophrenia Research* **50**:61, 2001.
36. Crow, Ref. 5, pp. 130–131.
37. Crow, T.J., Schizophrenia as the price that *Homo sapiens* pays for language: a resolution of the central paradox in the origin of the species, *Brain Research Reviews* **31**:119, 2000.
38. Crow, T.J., A Darwinian approach to the origins of psychosis, *British J. Psychiatry*, **167**:12, 1995.
39. Crow, Ref. 5, p. 131.
40. Crow, T.J., Schizophrenia as failure of hemispheric dominance for language, *Trends in Neuroscience*, **20**:339, 1997.
41. Crow, Ref. 5, p. 127.
42. Crow, Ref. 5, pp. 127, 138–139.
43. Crow, Ref. 5, p. 138.
44. Crow, T.J., Current status of linkage for schizophrenia: polygenes of vanishingly small effect or multiple false positives? *American J. Medical Genetics (Neuropsychiatric Genetics)* **74**:99–103, 1997.
45. Crow, Ref. 37, p. 125.
46. Crow, Ref. 44, p. 101.
47. Bray and Owen, Ref. 14, p. 172.
48. DeLisi, L.E., Critical overview of current approaches to genetic mechanisms in schizophrenia research, *Brain Research Reviews* **31**:187–192, 2000.
49. DeLisi, Ref. 48, p. 188.
50. DeLisi, L.E., Shaw, S., Sherrington, R., Nanthakumar, B., Shields, G., Smith, A.B., Wellman, N., Larach, V.W., Loftus, J., Razi, K., Stewart, J., Comazzi, M., Vita, A., De Hert, M. and Crow, T.J., Failure to establish linkage on the X chromosome in 301 families with schizophrenia or schizoaffective disorder, *American J. Medical Genetics (Neuropsychiatric Genetics)* **96**:335–341, 2000.
51. Crow, T.J., Schizophrenia as failure of hemispheric dominance for language, *Trends in Neuroscience* **20**:339–343, 1997.
52. Crow, T.J., Sexual selection as the mechanism of evolution of Machiavellian intelligence: a Darwinian theory of the origin of psychosis, *J. Psychopharmacology* **10**:81, 1996.
53. Geschwind, N., Language and the brain, *Scientific American* (April): 76–83, 1972.
54. Geschwind, N., Human brain: left-right asymmetries in temporal speech region, *Science* **161**:186–187, 1968.
55. Shenton, M.E., Dickey, C.C., Frumin, M. and McCarley, R.W., A review of MRI findings in schizophrenia, *Schizophrenia Research* **49**:24, 2001.
56. Shapleske, J., Rossell, S.L., Simmons, A., David, A.S. and Woodruff, P.W.R., Are auditory hallucinations the consequence of abnormal cerebral lateralization? A morphometric MRI study of the sylvian fissure and planum temporale, *Biological Psychiatry* **49**:686, 2001.
57. Shapleske *et al.*, Ref. 56, pp. 685–693.
58. Gannon, P.J., Holloway, R.L., Broadfield, D.C. and Braun, A.R., Asymmetry of chimpanzee planum temporale: humanlike pattern of Wernicke's brain language area homolog, *Science* **279**:220–222, 1998.
59. Hopkins, W.D., Marino, L., Rilling, J.K. and MacGregor, L.A., Planum temporale asymmetries in great apes as revealed by magnetic resonance imaging (MRI), *Neuroreport* **9**:2913–2918, 1998.
60. Gannon *et al.*, Ref. 58, p. 221.
61. Crow, T.J., Why cerebral asymmetry is the key to the origin of *Homo sapiens*: how to find the gene or eliminate the theory, *Current Psychology of Cognition* **17**:1244–1245, 1998.
62. Cantalupo, C. and Hopkins, W.D., Asymmetric Broca's area in great apes, *Nature* **414**:505, 2001.
63. Stevens and Price, Ref. 18, p. i.
64. Stevens and Price, Ref. 18, p. 156.
65. McGuire and Troisi, Ref. 17, p. vii.
66. McGuire and Troisi, Ref. 17, p. 277.
67. McGuire and Troisi, Ref. 17, p. 210.
68. McGuire and Troisi, Ref. 17, p. ix.
69. Randall, P.L., Schizophrenia, abnormal connection, and brain evolution, *Medical Hypotheses* **10**:247–280, 1983.
70. Randall, Ref. 69, p. 273.
71. Randall, Ref. 69, p. 272.
72. Feierman, J.R., Nocturnalism: an ethological theory of schizophrenia, *Medical Hypotheses* **9**:462, 1982.
73. Feierman, Ref. 72, p. 466.
74. Feierman, Ref. 72, p. 465.
75. Feierman, Ref. 72, p. 470.
76. Feierman, Ref. 19, p. 266.
77. Feierman, Ref. 19, p. 264, 272.
78. Nettle, D., *Strong Imagination: Madness, Creativity, and Human Nature*, Oxford University Press, Oxford, p. 182, 2001.
79. Nettle, Ref. 78, p. 85.
80. Nettle, Ref. 78, p. 211.
81. Miller, G., *The Mating Mind: How Sexual Choice Shaped the Evolution of Human Nature*, Heinemann, London, 2000.
82. Horrobin, D., *The Madness of Adam and Eve: How Schizophrenia Shaped Humanity*, Bantam Press, London, 2001.
83. Horrobin, Ref. 82, p. 59.
84. Horrobin, Ref. 82, p. 10.
85. Horrobin, Ref. 82, pp. 220–222.
86. Torrey, Ref. 7, pp. 148–151.
87. Horrobin, Ref. 82, pp. 237–238.
88. McCrone, J., The human stain, *Guardian Unlimited*, 28 April 2001. (Available at <www.guardian.co.uk/Archive/Article/0,4273,4176749,00.html>, 13 May 2003)
89. Nettle, D., Eccentric origins of creativity, *Nature* **412**:119, 2001.
90. Horrobin, Ref. 82, p. 236.
91. Sarfati, J., *Refuting Evolution 2*, Master Books, Green Forest, 2002.
92. Geschwind, N., Specializations of the human brain. *Scientific American* **241**(3):180–199, September 1979.

Eric Wright has a Ph.D. in neuroscience. He has worked in the area of neuroscience for several years, including research into schizophrenia. He has had an interest in Creation/evolution issue for a long time, particularly the subject of human origins.