Brain Disorder and Rock Art

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Prompted by numerous endeavours to link a variety of brain illnesses/conditions with the introduction of palaeoart, especially rock art, the author reviews these proposals in the light of the causes of these psychiatric conditions. Several of these proposals are linked to the assumption that palaeoart was introduced through shamanism. It is demonstrated that there is no simplistic link between shamanism and brain disorders, although it is possible that some of the relevant susceptibility alleles might be involved in some shamanic experiences. Similarly, no connection between rock art and shamanism has been credibly demonstrated. Moreover, the time frame applied in all these hypotheses is fallacious for several reasons. These notions are all based on the belief that palaeoart was introduced by 'anatomically modern humans' and on the replacement hypothesis. Finally, the assumption that neuropathologies and shamanism preceded the advent of palaeoart is also suspect. These numerous speculations derive from neglect of the relevant empirical factors, be they archaeological or neurological.

This article owes a great deal to a recent paper by Bullen (2011), critiquing the attribution of rock art to bipolar disorder, and the subsequent elaboration by Helvenston (2012a) and Bullen's (2012) response (see below for details). This author is in agreement with most of the points made in that discussion, so this is not to present counterpoints or to canvas any substantive disagreements, but to follow Helvenston's example and expand the scope of the discussion. For instance there have been other debates, as in this journal, of the nexus between rock art and brain illnesses, and the generic issue of subjecting palaeoart, and most especially rock art, to more or less capricious interpretations, which is certainly a topic in need of more comprehensive treatment.

Bullen (2011) responded to the key propositions by Whitley (2009) — which were that (a) shamans introduced palaeoart, (b) shamanism derives from bipolar disorder, (c) this disorder confers enhanced creativity on the patient, and (d) this illness explains the origins of artistic production — by plausibly and convincingly refuting them. Here the author wishes to delve more deeply into the underlying issues by considering genetic, etiological, evolutionary and neurological aspects of such contentions, but above all review the epistemological issues that lead to the formulation of such opinions by rock-art commentators.

Having elsewhere dealt in some detail with the first of Whitley's crucial propositions, that shamans introduced palaeoart, the author will examine the topic of the origins of what is simplistically termed 'artistic production' — 'palaeoart' would be a better word for the phenomenon in question because it is unknown whether this corpus was an 'art' in the sense of that expression.

Before the origins of palaeoart can be properly considered, the 'cult of Palaeolithic art' developed over more than a century **ago?? OR 'developed over the last century'??** needs to be reflected upon. Much research on Palaeolithic art concentrates on that of Western Europe ignoring the far greater body of Pleistocene rock art elsewhere (e.g. in Australia, southern Africa or India). In addition, most of the world's surviving Pleistocene rock art was made by people of Mode 1, 2 or 3 technological traditions (Foley & Lahr 1997) rather than Mode 4. This research has also disregarded the lack of evidence that the early part of the much-cited Franco-Cantabrian corpus must be the work of 'anatomically modern' humans, whereas in fact there is good evidence that

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it was made by Robusts (Bednarik 2007; cf. Sadier *et al*. 2012). Forensic evidence which permits the artists' ages to be estimated has also been ignored. They were mainly juveniles or teenagers, and the great majority of imprints of human body parts in the caves are of children (Bednarik 2008a). Instead, a vast mythology of meanings of the images was created. Indeed, the much-debated shamanism which both Bullen and Helvenston discuss derives from such searches for meaning. It also remains profoundly unknown at what point in human history the practices defined as shamanism were introduced, despite isolated claims for Holocene evidence (e.g. Porr & Alt 2006). But there are alternative, logical methods of investigating the role of shamanism in rock art. The author is not aware of a single report of a shaman having produced rock art in the literature of ethnography, nor have such reports been cited by those advocating shamanism in rock art. There are, however, numerous cases of rock-art production having been observed and recorded or where the authors of the 'art' may be known to us (e.g. Bednarik 1998, 26; Haskovec & Sullivan 1986; Mulvaney 1996; Novellino 1999). In all such cases no shamans were involved, and the utilitarian or ceremonial purpose of the rock art, where it is known, lacks any connection with shamanism. Indeed, one of the most obvious prerequisites for considering what the characteristics of shamanic art might be is a definition of its ethnographically demonstrated idiosyncrasies. In the absence of such an explicit index *vis-à-vis* rock art we lack any definitive way of identifying authentically shamanic art traditions in rock art; we simply do not know the properties of shamanic rock art. Moreover, most of the world's rock art occurs in regions from which no shamanic practices are known ethnographically. Although none of this demonstrates that no rock art was ever produced by shamans, the proposition that significant quantities of rock art are the work of shamans (Lewis-Williams & Dowson 1988) is unwarranted by the empirical data, and it is of course untestable. Thus the null hypothesis, that most rock art is not shamanic, has empirical support; the shamanic hypothesis has none.

Helvenston (2012b) most pertinently observes that 'when faced with uncertainty and the unknown the San resort to supernatural powers, when faced with familiar tasks like gathering food, or building huts, their approach is scientific'. In a very similar way the author has observed that when the members of a strictly Islamic society are faced with taking a life-threatening risk, they may turn to the local shaman for protection. The mere continuation of shamanism in such religiously rigid societies speaks for itself, and illustrates the continuing innate power of shamanism. The communal dancing of the San Bushmen and even their healing trance are entirely different from the phenomenon of authentic shamanism.

Autism and rock art

Margaret Bullen (2005) once pointed out that there are features of deep trance that mimic autism, quoting Bogdashina (2003) to the effect that deprivation of sensory stimulation can lead to autistic-like behaviours. The human brain condition autism (Allman *et al*. 2005; Balter 2007; Baron-Cohen 2002; 2006; Bednarik & Helvenston 2012; Brasic 2009a,b; 2010; Burack *et al*. 2009; Frith 1989; Grinker 2007; Helvenston & Bednarik 2011; Hermelin & O'Connor 1970; Hughes *et al*. 1997) has often been proposed to have been instrumental in introducing **stimulating?? producing??** Pleistocene palaeoart (Bogdashina 2010, 159–60; Haworth 2006; Humphrey 1998; Kellman 1998; 1999; cf. Marr 1982; Spikins 2009; Treffert 2010).

Although Humphrey's 1998 paper presents no convincing case for a nexus between Pleistocene cave art and autism he does raise some very pertinent and interesting points. One concerns the ingrained belief that the Upper Palaeolithic artists shared our modern 'mind'. Pleistocene archaeologists often use such terms as 'modern behaviour' or 'modern mind' but it is becoming increasingly apparent that there is no agreement as to what they mean. Some authors refer to human modernity as a set of variables one can reasonably expect to find a million years ago, even earlier (Bednarik 2011a,b). Others favour a much narrower definition, attributing a 'pre-modern mind' even to the cave artists of the early Upper Palaeolithic (see Humphrey 1998 and the debate therein) and suggesting that the 'modern mind' postdates 20 kya. Bearing in 'mind' that it is not clear what the mind is (what is its appearance, weight or composition?) and that this is probably intended as a shorthand generic term for mental processes occurring in the human brain, the concept of 'modernity of mind' needs to be clearly defined. If 'mind' refers to the state and operation of the neural structures that are involved in moderating behavioural patterns, these must have been essentially modern at least since the end of the Lower Pleistocene (Bednarik 2011a). But human behaviour is not only determined by the intrinsic neural and endocrine systems giving rise to it. These are influenced by ontogenic experiences of the individual and their effects on these neural configurations (Maguire *et al*. 2000; Draganski *et al*. 2004; Smail 2007; Malafouris 2008). The brain of literate conspecifics is differently organized from that of any pre-literate individual (Helvenston in press), and there are even significant

differences between present individuals of 'magical thinking' and those with well-integrated cause-and-effect reasoning. The archaeological claims placing the advent of the 'modern mind' 30 or 40 millennia ago are therefore false, however it is defined; 'We have never been modern' (Latour 1993).

It is doubtful that a scientific (testable) case can be made for a connection between the exceptional skills sometimes (but very rarely) found in autistics (Happé & Vital 2009; Mottron & Belleville 1993; 1995; Mottron *et al*. 1999; Waterhouse 1988) and the abilities of the graffitists of the Franco-Cantabrian caves. Perhaps a better case could be presented by examining the very limited evidence that the cave art involved adults (Bednarik 2008a), but this has not yet been attempted. Humphrey's challenge to the archaeologists' 'received view' (Dennett 1998) — to show why they assume that Upper Palaeolithic palaeoartists must have shared present-day perception and reality — is of particular interest. So is Dennett's observation that '[i]t will be interesting to see if the defenders of the received view have such facts in reserve to salvage their case, or whether they will have to fall back on simply citing various eminent opinions in favour of the received view'. Certainly the responses of archaeologists following the presentation of Humphrey's hypothesis in this journal have failed to offer such 'facts'.

Another fascinating aspect of Humphrey's contentions arises when he quotes Mithen as stating 'that modern humans … were capable of the type of symbolic thought and sophisticated visual representation that was beyond Neanderthals'. Two issues arise from this statement. First, the art of the 'Aurignacians' provides no testable proof of symbolic thought. It only provides evidence of depiction, no more. That is not to say that the 'Aurignacians' were not capable of symbolic thought, but the proof for that is to be (and can be) found elsewhere. Second, we have no evidence of any kind that 'Aurignacian' palaeoart was produced by 'anatomically modern humans', because all Late Pleistocene human remains of Europe predating, say, 26 kya are either of Neanderthals or of intermediate forms (Bednarik 1995; 2007; 2008a; 2011a,b). Therefore Mithen's claim is probably wrong on both counts and merely expresses the inherent defects of the replacement hypothesis.

Humphrey presents only a single example of an autistic child with advanced artistic abilities (Selfe 1977), although some others have long been known (e.g. Happé & Frith 2010; Kellman 1998; 1999; Pring & Hermelin 1993), and he seems unaware of other authors pursuing the same issue. Moreover, his hypothesis suffers from his lack of awareness that such abilities in children are certainly not limited to autistic savants, but are also well known as 'precocious realism' in the art of non-autistic children (Drake & Winner 2009; O'Connor & Hermelin 1987; 1990; Selfe 1983; *contra* Snyder & Thomas 1997). Seen in that overall context, Humphrey's hypothesis loses its appeal, and he might consider **should reconsider??** it in the light of the sound empirical evidence that a major part of the palaeoart he considers was made by juveniles. His suggestion that the palaeoart of the early 'Upper Palaeolithic' implies an absence of language use because of its naturalism, looks absurd when applied to the realistic rock art of the San Bushmen. Similarly, he seems to be unaware that throughout the world, the images we tend to regard as naturalistic are preceded by traditions that lack iconographic elements. Finally, the extremely rare occurrence of autistics of exceptional depictive abilities does not explain why 99.99 per cent of autistic spectrum disorder (ASD) patients lack them. After all, ASD has recently become a very common illness affecting one in 110 children (Weintraub 2011; a more reliable study reports it being as high as one in 38: Kim *et al*. 2011). The epidemic increase in this diagnosis, from one in 5000 in 1975, cannot be entirely attributed to changing diagnostic criteria (cf. Buchen 2011). The explanation offered by Bednarik (2011a; exponential increase in human neuropathology due to suspension of natural selection) is perhaps the most eligible.

Asperger's syndrome and rock art

Spikins's (2009) 'different minds theory' endeavours to explain 'modern behaviour' as the rise in cognitive variation within populations through social mechanisms for integrating 'different minds'. She focuses particularly on one form of autism, Asperger's syndrome, because it does not inhibit the effective use of language or cognitive development, and the associated attention to detail enables patients to compensate for a deficiency in empathy. Subjects with autistic conditions (as well as in schizophrenia: Brüne 2006) have cognitively based deficiencies in 'theory of mind' (ToM), which defines the ability to attribute mental states — beliefs, intents, desires, pretending, knowledge etc. — to oneself and others and to understand that others have beliefs, desires and intentions that are different from one's own (Baron-Cohen 1991; Baron-Cohen *et al*. 1997; Frith & Happé 1994; Happé 1997; Happé *et al*. 1996; Jacques & Zelazo 2005; Jarrold *et al*. 2000; Ozonoff & Miller 1995).

Spikins's main contention is that autism is a spectrum of differences displayed across the modern population, and that modern behaviour arose when autistic modes of thinking were integrated into the

practices of human societies. Focusing on Asperger's, a form of 'mild autism' (Bednarik & Helvenston 2012; Rodman 2003), she emphasizes the analytical and mathematical thinking it involves and attributes to it the changes she detects in technology: 'Rigid analytical thinking (both by autistic individuals and through their influence) might improve technology and foraging efficiency' (Spikins 2009, 190). She cites projectile weapons, bladelets, bone artefacts, hafting, 'elaborate fire use', exploitation of marine resources and large game, apparently unaware that all of these have been demonstrated from the Lower Palaeolithic, together with palaeoart and 'personal ornamentation'. Nevertheless, she feels that these are all attributable to the 'attention to detail, exceptional memory, a thirst for knowledge and narrow obsessive focus' of autistics, particularly when coupled with their desire for social isolation.

However, these proficiencies are obviously not limited to people with ASD, a condition that also happens to include diagnostic characteristics such as inflexibility in thinking, difficulty with planning and organization, and rigorous adherence to routine (Pickard *et al*. 2011), which impede originality and innovative thought. The creativity Spikins invokes is impoverished in ASD patients (Craig & Baron-Cohen 1999; Frith 1972; Turner 1999) unless fostered, and the savant skills ascribed to a very few of them need to be nurtured and are specific to the ordered cultural context of modern life (Baron-Cohen 2000; Folstein & Rosen-Sheidley 2001; Thioux *et al*. 2006). Moreover, the neuropsychiatric disorders of humans, absent in other extant primates (Bednarik & Helvenston 2012; Enard *et al*. 2002a; Marvanová *et al*. 2003; Olson & Varki 2003; Rubinsztein *et al*. 1994; Sherwood *et al*. 2011; Walker & Cork 1999), are a deleterious by-product of more recent 'evolution' (Bednarik 2011a,b; 2012**NOT IN REFS**; Bednarik & Helvenston 2012; Helvenston & Bednarik 2011). Finally, the phylogenetic timing of the introduction of ASD is the crucial issue here: to influence society the illness had to exist, but to do so society and selective processes had to first tolerate it. The lack of social skills typical of ASD in societies heavily reliant upon social dynamics would tend to select against it, socially as well as genetically. Thus Spikins's hypothesis runs up against the classical Keller and Miller paradox (see below), the resolution of which will be considered below because it applies to all neuropathologies.

Schizophrenia and rock art

Another such effort, Whitley's (2009) attribution of shamanism to bipolar disorder, was preceded by implicating the similarly severe neuropsychiatric condition schizophrenia (e.g. Demerath 1942; Devereux 1956; Kroeber 1940; Le Barre 1970; 1972; Scheff 1970; Silverman 1967). The altered states in (North American) shamanism were perhaps first recognized by Oesterreich (1935, 295). Peters and Price-Williams (1980, 397) examined them across 42 cultures. Loeb (1924), Radin (1937) and Devereux (1961) defined shamans variously as epileptic, hysteric or neurotic, whereas Silverman (1967) introduced the notion that shamanism is an acute form of schizophrenia. His hypothesis attracted criticism immediately (Boyer 1969; Handelman 1968; Weakland 1968) and was followed by later work rejecting it. Lex (1984) suggested that the popularity of the notion that schizophrenia provides an explanation for shamanic experiences and behaviour appears to emanate from distorted and 'romantic' interpretations of the significance of hallucinatory symptoms. Noll (1983), in examining altered states of consciousness, demonstrated that the anthropological 'schizophrenia metaphor' of shamanism and its altered states is untenable. Significant phenomenological differences exist between the shamanic and schizophrenic states of consciousness. Despite these authoritative rebuttals the notion that there is a connection between shamanism and schizophrenia continued to be pursued in recent years (e.g. Polimeni & Reiss 2002; El-Mallakh 2006).

Twin and adoption studies have conclusively shown that schizophrenia (Os & Kapur 2009) is a genetic disorder (Cardno & Gottesman 2000; Kennedy *et al*. 2003; Riley & Kendler 2006). However, because its underlying physiological abnormalities remain inadequately understood, a properly integrated etiological and pathophysiologic model does not yet exist. Although a number of schizophrenia susceptibility genes have been identified[1] they have small or non-detrimental individual effects; the illness is of a polygenic nature. These genes may prompt changes in attention, memory, language or other cognitive functions through small effects on neurotransmitter function, cerebral structural organization, brain metabolism or connectivity as they interact with non-genetic factors. Susceptibility alleles only constitute increasing risk for schizophrenia through aggregating, be it by chance, assortative mating or by other mechanisms (Cannon 2005). They may be individually associated with normal or increased fertility or be operating under positive selection, unlike fully-fledged schizophrenia. Carriers of small numbers of schizophrenia susceptibility genes are far more numerous (about 15 per cent of any population) than cases of the actual disorder (0.3–1 per cent), and the advantages selected for in first-

degree relatives of schizophrenia patients have been suggested to include creativity (Horrobin 2001). Thus schizophrenia, 'the very embodiment of maladaptive traits' (Keller & Miller 2006), is most likely the result of complex polygenic inheritance and environmental susceptibility factors.

Crow (1997) perceives a connection between schizophrenia and language, and that the 'speciation event' defining modern humans also introduced language. According to this hypothesis, schizophrenia and language are linked to cerebral asymmetry, and the hemispherical dominance for language led to the collateral hemispheric lateralization and psychosis (Crow 1995a,b). However, this notion, theorizing that genetic drift can occur more frequently on the Y chromosome, is countered by several indices, not only the error of linking language origins with the speciation of Graciles (see above, and Bickerton 1990; 1993; 1996; 2010**NOT IN REFS 2009?**; Dunbar 1996; Falk 2009). For instance, the planum temporale, presenting a left–right asymmetry favouring the left (Geschwind & Levitsky 1968), which has been related to language reception, is also present in apes (Gannon *et al*. 1998; 2001). Moreover, the detection of the *FOXP2* gene on chromosome 7 of Robusts (Krause *et al*. 2007; cf. Enard *et al*. 2002b; Sanjuán *et al*. 2006; Zhang *et al*. 2002) but the absence of schizophrenia susceptibility alleles, such as *NRG3,* refutes the idea (in fact schizophrenia may have appeared very much later than Graciles: Bednarik & Helvenston 2012).

The records of the UK National Childhood Development Study (Crow *et al*. 1995; Karlsson 1984) suggest that children later diagnosed with schizophrenia had persistent reading impairment and low IQ scores. Schizophrenia occurs in all cultures, all of which perceive it as a serious maladaptive dysfunction (Pearlson & Folley 2008). Introvertive anhedonia, a typical symptom of schizophrenia (Schuldberg 2000), decreases creative activity significantly, thus providing a clear separation between creative and clinical cohorts. Therefore the notion that schizophrenia fosters creativity or artistic production has little or no credibility, and if shamanism derived from that illness, the explanation of rock art as the work of shamans loses all support.

However, the relationship between these three factors is much more complex and this may explain the apparently competing models. As in autism, there is a spectrum within which schizophrenia is merely the extreme form. For instance, first-degree relatives of psychotic patients have been consistently shown to be notably creative (Heston 1966; Karlsson 1970). Elevated levels of some of the schizotypal traits are commonly observed in individuals that are active in the creative arts (Brod 1997; Nettle 2001; Schuldberg 2000; Schuldberg *et al.* 1988). Schizotypal diathesis, which may lead to actual illness under specific environmental factors (Tsuang *et al*. 2001), but in most cases does not, is therefore more convincingly implicated in creativity, much in the same way as mild forms of autism can yield high-performing individuals. It is through polygenic mutation-selection balance that mental disorders reflect the inevitable mutational load on the thousands of genes underlying human behaviour. The data on the factors of increased risks of mental disorders with brain trauma, inbreeding and paternal age on mental-disorder prevalence rates, the fitness costs of the illness and the rarity of susceptibility alleles all indicate this.

Of significance — although of no direct bearing on the issue of the involvement of shamans — is that schizophrenia is associated with 'drastically reduced probability of reproduction' (Bassett *et al*. 1996; also Avila *et al*. 2001; Nettle & Clegg 2006), through significantly diminished fertility, mediated by reduced survival and social competence (Brüne 2006), reduced attractiveness for mating and lower marriage rates, as well as possibly via reduced fertility once married. The notion that artistic production has its origins in 'costly displays' (Miller 2000; 2001; Varella *et al*. 2011) would therefore also seem to exclude the involvement of schizophrenic artists. Of potential interest would be how schizotypy relates to mating behaviour, e.g. through an increase in extra-pair copulations, a higher turnover of relationships, or less time between relationships. However, the involvement of schizophrenia or schizotypy in shamanism deserves further examination. The discovery of the rubber hand illusion (RHI) in schizophrenic patients (Peled *et al*. 2003) has considerable implications for the notion of out-of-body experiences (Thakkar *et al*. 2011). It has been suggested that a weakened sense of the self may contribute to psychotic experiences. The RHI illustrates proprioceptive drift, which is observed to be significantly greater in schizophrenia patients than in a control sample, and can even lead to out-of-body experience, linking 'body disownership' and psychotic experiences (e.g. floating outside one's own body). This derives from the internal body image in the superior parietal lobule in the right hemisphere and the operation of mirror neurons (cf. somatoparaphrenia, anosognosia). Although there is no credible empirical evidence linking schizophrenia with palaeoart production, just as there is none linking shamanism with it or with schizophrenia, susceptibility to proprioceptive drift can be shown to be linked to schizotypy, and may well account for certain experiences of shamans.

Bipolar disorder and rock art

Only Whitley (2009) has proposed this connection so far, and apparently with much less justification than any of the above proposals. Whitley's collation of 'mad geniuses', 'first religion', shamanism and mood disorders may well derive from his long-standing dedication to proving the shamanistic origins of rock art. Bipolar disorder has been less popular as an explanation of shamanism because its etiology renders it less likely. It also differs from schizophrenia in a number of crucial ways. For instance, although chronic it is not neurodegenerative with advanced age, in contrast to schizophrenia. In schizophrenia there is increased neuronal density in the prefrontal cortex, whereas in bipolar disorder there is decreased neuronal and glial density in association with glial hypertrophy (Rajkowska *et al.* 2009). Both illnesses are highly heritable (Edvardsen *et al*. 2008), as shown by monozygotic twin studies (Kieseppä *et al*. 2004), and they are clearly polygenic as indicated by the wide spectrum of their manifestations. The bipolar range stretches from bipolar I through bipolar II and to mild forms of cyclothymia. It is reflected in the lack of resolution in decisively determining the genetic basis, although regions of interest identified in linkage studies include chromosome 18, 4p16, 12q23-q24, 16p13, 21q22 and Xq24-q26 (Craddock & Jones 1999; Craddock *et al*. 2005; Saito *et al*. 2001), and genes *DRD4*, *MAOA* and *SYNJ1* have been implicated (Andrés *et al*. 2004; Cho *et al*. 2005; Jansson *et al*. 2005; Muglia *et al*. 2002; Preisig *et al*. 2005; Stopkova *et al*. 2004). Just as autism and schizophrenia comprise spectra rather than discrete illnesses, much the same applies to bipolar disorder, and probably for the same reasons: numerous genetic predispositions (Schulze 2010) and a range of environmental factors determine any patient's specific condition.

Bullen (2011; 2012) and Helvenston (2012a) have already responded adequately to Whitley's proposition, and negated both his key notions, that shamans are bipolar and that the Pleistocene cave art of France and Spain is the work of such bipolar shamans. Helvenston has rightly emphasized that

> bipolar illness is a very serious disorder and even today it is not well controlled in a large group of people suffering from it. During the manic or depressive phases, the individual is almost completely disabled, and unlikely to be creating anything, let alone distinguished art. Sufferers are frequently unable to care for themselves during these phases and would only be productive during remission. During remission they often have to cope with the consequences of what they have done during the manic or depressive phases (Helvenston 2012a, 109).

She also insists that 'no ape has ever shown any illness even remotely resembling bipolar illness', citing Mason and Rushen (2006). Bullen, however, maintains that extant non-human primates are burdened by brain illnesses, and that we

> cannot ask a baboon if it has low self esteem or if it feels hopeless but it can portray those perceptions in its observed helplessness. Genetic studies in primates may help to elucidate which genetic variants associated with affective disorders could have been present in the early hominid genome (Bullen 2012, 111).

However, the presence in apes of genes thought to be involved in human neuropathologies is irrelevant to detecting such illnesses in them. Mere presence of individual genes does not result in brain illness. A paper sometimes cited as presenting evidence of neuropathology in non-human primates (Marvanová *et al*. 2003) in fact merely reports the incidence of similar genes in the brains of *healthy* humans, apes and monkeys. For instance some of the genes thought to be involved in Alzheimer's are concordant in humans and other primates, while others are not. Moreover, most of the data used in this report derives from another study (Enard *et al*. 2002a) that was entirely of specimens that were all free of mental disorders and brain abnormalities detectable by autopsy. While epilepsy and stroke do occur in non-human primates, other brain illnesses have not been reported from natural settings or free-roaming populations (Bednarik & Helvenston 2012; Olson & Varki 2003; Rubinsztein *et al*. 1994; Walker & Cork 1999). It is true that when captive apes and monkeys are deprived of environmental stimulation and the company of conspecifics they often present symptoms resembling obsessive-compulsive behaviour. However, such behaviour is not attributable to inherent defects of the brain, but to protracted enforced conditions, i.e. of a totally different etiology. Moreover, variations of such aberrant behaviour are also evident in many captive non-primate species. Chimpanzees do not experience the significant atrophy in the size of brain and other internal structures that inevitably accompany aging in humans (Sherwood *et al*. 2011). Consequently, the human susceptibility to neuropathologies such as Alzheimer's disease is unique in the animal world, and is attributable to the wear and tear of an excessively complex brain, most of whose neurons cannot be renewed. Indeed, the cognitive ability of humans may have prompted the reduced apoptosis (the process of 'programmed' cell death) of neurons, relative to chimpanzees. This has been proposed to cause the higher risk in humans of cancer and other diseases associated with reduced apoptotic function (Arora *et al*. 2009).

One of Bullen's (2011) chapters is titled 'Are we any nearer to knowing how it all started?' — referring to the origins of palaeoart. As she notes, Whitley contends that both shamanism and bipolar disorder tend to run in the family, i.e. if two discrete characteristics are heritable they must be connected. On this reasoning any heritable characteristic would be related to bipolar conditions. Indeed, a careful analysis of this pattern of interpreting rock art would be immensely valuable in learning to understand modern reactions to rock art: why are its interpretations as the work of extraterrestrials or of shamans so popular? More fundamentally, why are modern people so strongly inclined to interpret rock art? Modern beholders of some forms of ancient palaeoart tend to delude themselves into believing that it communicates with them, through a form of autosuggestion, and this process deserves careful analysis. The shamanic hypotheses play on this susceptibility, and on the perception that such keys to rock-art interpretation (see Le Quellec 2006) provide codes of meaning.

Brain disorders in human evolution

Perhaps the most fundamental problem with the shamanic, bipolar, schizophrenic, Asperger's and autistic explanations of rock art is that their advocates make no attempt to determine whether these conditions actually applied in the Pleistocene. As Helvenston continues in her authoritative commentary:

> If there had been manic-depressives during the Palaeolithic, they would have been completely disabled prior to modern medication, so it is highly unlikely that they produced any great art and there is no more reason to suppose that a manic-depressive created the Palaeolithic cave paintings than that a normal person did — in fact there is less reason (Helvenston 2012a, 109).

This is not just a question of clarifying when neuropathologies began to have a significant impact on the human genome, but more importantly why they were not selected against. The mental and cognitive developments in the human brain rendered humans vulnerable to neurodegenerative diseases as well as frontal lobe connectivity problems, to demyelination or dysmyelination, Mendelian disorders — in fact to thousands of syndromes and disorders endemic to humans. Why their rise was not vigorously selected against by natural evolution is the Keller and Miller (2006) paradox, which was resolved only recently (Bednarik 2007; 2008b,c). In a species fully subject to the canons of natural selection such numerous disadvantageous mutations would surely tend to be suppressed vigorously. They include thousands of Mendelian (single gene) disorders, but also countless somatic changes, such as cleidocranial dysplasia or delayed closure of cranial sutures; malformed clavicles and dental abnormalities (genes *CBRA1* and *RUNX2* refer); type 2 diabetes (*THADA*); the microcephalin D allele, introduced in the Final Pleistocene through a single progenitor copy (Evans *et al*. 2005); or the *ASPM* allele, another contributor to microcephaly, which appeared around 5800 years ago (Mekel-Bobrov *et al*. 2005). Indeed, most changes from Robusts (such as *Homo sapiens neanderthalensis*) to Graciles (*Homo sapiens sapiens*) have been maladaptive: significantly reduced brain volume (by *c.* 13 per cent) and cranial as well as other skeletal robusticity, and greatly reduced physical strength. Why did natural selection fail to cull the alleles underlying hominin neotenization and 'devolution' that marks the last forty or fifty millennia of human 'evolution'? (In reality, evolution is dysteleological, therefore biological devolution is impossible, whereas cultural devolution certainly does occur.)

The suspension of human evolution determined by natural selection has remained completely unrecognized until recently because Pleistocene archaeology and palaeoanthropology have pursued the replacement hypothesis with such enthusiasm, when it has no genetic, skeletal or cultural justification whatsoever (Bednarik 2008b) and is attributable to a hoax. That hypothesis demands that natural selection and genetic drift (Bednarik 2011c) governed recent evolution and speciation, when in fact the emergence of the Graciles involved no speciation; they derive from Robusts via intermediate forms (as implied by the findings of Green *et al*. 2010), gracilization being a gradual process. The distinctive changes during the final third of the Late Pleistocene are almost entirely the result of self-domestication caused by the determination of breeding patterns by rising cultural imperatives that have been defined (Bednarik 2008c). Domestication promotes unfavourable alleles (e.g. Andolfatto 2001; Horrobin 1998; 2001; Lu *et al*. 2006), and it can even account for other unexplained features, such as the abolition of oestrus in females. Assuming that it was under the auspices of this process that predispositions for brain illnesses were protected from natural selection, which is the rational explanation, such pathologies must postdate these developments. It would then be expected that most appeared less than 40 kya and are endemic to *Homo sapiens sapiens* (Bednarik 2008b; Helvenston & Bednarik 2011). Where relevant genetic indications are already available, they confirm this prediction. For instance the genes *AUTS2* and *CADPS2*, involved in autism, appear with Graciles, and *NRG3* (schizophrenia) is also absent in Neanderthals (Voigt *et al*. 2006). Using the human

haplotype map to test for selective sweeps in regions associated in genome scans with psychosis, such as 1q21, is promising (**Voigt *et al*. 2006??**). Such selective sweeps tend to yield relatively recent etiologies, of less than 20 kya. Some conditions, such as schizophrenia, have been suggested to be much more recent (Bednarik & Helvenston 2012), and so far no known relevant susceptibility alleles have been reported from Neanderthaloid remains.

Therefore at this stage none of the more severe brain illnesses should be expected to be found in hominin populations prior to the suspension of natural selection, which on present indications may have begun, initially on a small scale, between 40 kya and 30 kya. It is therefore unlikely that by the time the Chauvet or Aldène rock art was created (Bednarik 2007; Sadier *et al*. 2012), any of the brain diseases to which it has been attributed could have even taken root. The shamanism that is claimed to have given rise to it must have been established much later if it derived from neuropathologies, and its occurrence in any Pleistocene society has not been demonstrated or even proposed, except via circular reasoning. As noted, all types of the European Pleistocene palaeoart that are capable of providing empirical evidence about the age of the artists were demonstrated to have been produced by children or teenagers, which renders it highly likely that the same applies to much of the rest of this palaeoart. This hypothesis offers testability, whereas the hypotheses involving brain illnesses are lacking in falsifiability or sound epistemology. Whether it is Asperger's syndrome, bipolar syndrome, autism or schizophrenia, neurosis, hysteria or epilepsy, none of them seem to account for either palaeoart or shamanism, nor does shamanism account for palaeoart.

Note

1. *ADCYAP1*, *ALDH1A1*, *ANK3*, *BDNF*, *CD9*, *CHRNA-7*, *CNR1*, *COMT*, *CPLX2*, *DISC1*, *DRD2*, *DTNBP1*, *FABP7*, *GABRB3*, *GAD1*, *GNB1L*, *GRIA1*, *GRIA4*, *GRIN2B*, *GRMS*, *GSN*, *HINT1*, *HOPA12bp*, *HSPA1B*, *HTR2A*, *IMPA2*, *KALRN*, *KIF2A*, *MBP*, *MOBP*, *NCAM1*, *NDUFV2*, *NRCAM*, *NR4A2*, *NRG1*, *NRG3*, *PDE4B*, *PRKCA*, *RAB18*, *RELN*, *RGS4*, *SLC1A2*, *SLC6A4*, *SNAP-25*, *SYN2*, *TCF4*, *TNIK*: see Ayalew *et al*. 2012; Cho *et al*. 2005; Li *et al*. 2006; Spinks *et al*. 2004; Xu *et al*. 2006; Yoshikawa *et al*. 2001.

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