Author's Response

Elaborating the social brain hypothesis of schizophrenia

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Abstract: I defend the case for an evolutionary theory of schizophrenia and the social brain, arguing that such an exercise necessitates a broader methodology than that familiar to neuroscience. I propose a reworked evolutionary genetic model of schizophrenia, drawing on insights from commentators, buttressing my claim that psychosis is a costly consequence of sophisticated social cognition in humans. Expanded models of social brain anatomy and the spectrum of psychopathologies are presented in terms of upper and lower social brain and top-down and bottom-up processes. Finally, I argue that cerebral asymmetry evolved as an emergent property of primary intrahemispheric reorganisation in hominoids.

During the two years that have elapsed since I first submitted my target article "An evolutionary theory of schizophrenia: Cortical connectivity, metarepresentation, and the social brain" to *BBS*, I have examined and re-examined many aspects of the hypothesis. My ongoing research as well as the critiques of academic colleagues, not least the 14 commentaries submitted in this journal, has caused me to broaden my thinking and grapple with a number of troublesome questions. For example, the enigma that lies at the heart of schizophrenia research and which initially prompted me to write this paper: Why does a disorder such as schizophrenia, associated with reduced evolutionary fitness, survive in the human genome? This is the curiosity that has perplexed those engaged in study of the disorder for nearly a century. I am grateful to **Crow**, himself a pioneer in schizophrenia research, for reminding us of Huxley and Mayr's early attempts to solve this problem (cf. Huxley et al. 1964). The current debate has a long history, and many of the ideas raised in both the target article and the commentaries are by no means novel. However, I maintain my view that biomedicine is knee-deep in quicksand when it comes to unravelling the complexities of mental disorder, and that a comprehensive understanding of psychosis necessitates a broad sociobiological and evolutionary approach. Delbrück (1949) has said, "The animal or plant or micro-organism . . . [a mature physicist] . . . is working with is but a link in an evolutionary chain of changing forms, none of which has any permanent validity." Mayr (1988) adds, "There is hardly any structure or function in an organism that can be fully understood unless it is studied against this historical background." This is why I reject **Aleman & Kahn**'s position that "the evolutionary framework in which Burns' hypothesis is embedded might be superfluous."

R1. A methodology borrowed from archaeology

Aleman & Kahn quote Lewontin, underlining their scepticism regarding the possibility of a scientific theory of human cognitive evolution. I would agree with these authors that it is probably impossible to achieve such a theory if one relies solely on a narrow empirical method derived from reductionist physics. The construction of a sound evidence base for evolutionary hypotheses is not always easy. How does one generate data about the behaviour and mental state of our ancestors? Relationship dynamics, emotional states, and cognitive processes do not readily fossilise like bones, to be examined and analysed and presented as data. This is a problem that several authors have addressed. Lewis-Williams, a South African cognitive archaeologist and expert on the rock art of the San, recently published an intriguing book entitled *The Mind in the Cave*, in which he interprets the Palaeolithic art of Western Europe in terms of emerging consciousness in early humans (Lewis-Williams 2002). His task is similar to mine in that he faces the same constraints when assembling evidence for his hypothesis. He explains that there are too many gaps in the archaeological record to establish a clear line of argument and this prevents the scientifically reified formal, sequential testing of hypotheses. His solution to this problem is to draw on the work of Alison Wylie, a philosopher of science. Wylie describes a methodology that incorporates important scientific principles of hypothesis testing and that is well suited to the challenge of theorising about archaeological matters. This method she terms *cabling*. Unlike some arguments that form a logical "chain" of sequential links, the cabling method entails the intertwining of numerous strands of evidence. Wylie explains that very often, archaeologists construct an argument by drawing in a number of different strands of evidence from varied scientific sources. For example, the utility of an excavated structure might be elucidated by drawing upon ecological, ethnographic, and anthropological facts that have a bearing on the site. Lewis-Williams makes use of this method in his enquiry, drawing on evidence from extant hunter-gatherer traditions, from psychology and from neuroscience, in his construction of a hypothesis. He argues that the cabling method is sound in that it is both *sustaining* (a strand may compensate for a gap in another strand) and *constraining* (it "restricts wild hypotheses that may take a researcher far from the archaeological record").

In response to **Aleman & Kahn**'s scepticism regarding the possibilities of reconstructing human cognitive evolution, I would argue that such a cabling methodology is valid and indeed appropriate within evolutionary biology. These authors request evidence for "a heritable variation for social cognition in our remote ancestors, . . . and that those who possessed this ability in the remote past left more offspring by virtue of that ability." They are asking the impossible, because social behaviour does not fossilise. We need to approach this problem with a broader perspective than that derived from physical science. Byrne has listed the establishing of a reliable pattern of descent as one part of a methodology for inferring the history of primate cognition (Byrne 2000). Many authors have confirmed the close evolutionary relationship between simian and ape species and modern Homo sapiens, with strong data from comparative psychology, molecular biology, and physical anthropology. Thus, cladistic analysis provides us with living relative species with which we can test the hypothesis that there is a heritable variation for social cognition that increases fitness. Very recently Silk et al. published such a study in *Science* (Silk et al. 2003). They

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analysed a huge database documenting social behaviours in 108 female baboons over 16 years. The results showed that a composite index of sociality was highly correlated with infant survival. This study provides the first direct evidence of the selective advantage of sociality in primates. I hope more studies of this nature will follow in other primate species, because this clearly is a research strategy that is feasible and that has the potential to verify the social brain hypothesis.

R2. The evolutionary genetics of schizophrenia

Several commentators have drawn attention to the vagueness of the model I proposed to explain the "survival" of this maladaptive disorder. **Hardcastle** and **Weisfeld** take the extreme view that schizophrenia is a benign trait not subject to natural selection, because reproduction occurred at an earlier age than onset of the disorder in ancestral times. I cannot agree with Weisfeld's somewhat romantic image of the Palaeolithic environment, with abundant food, nurturing families, and limited stress on predisposed individuals. This harkens to a past era where anthropologists idealised the "noble savage" and is in contradiction to most evidence that supports a harsher and more stressful ancestral lifestyle (Bogin 1999): A more severe world where drought, disease, and threat of predation was the norm would have pushed the reproductive age into or beyond the usual age of onset of schizophrenia, thus rendering the disorder subject to natural selection.

Panksepp & Moskal suggest that schizophrenia "is not actively maintained in the genome," and that certain genes make one vulnerable to "epigenetic and environmental factors that promote schizophrenic phenotypes." I certainly agree that the genetic basis of schizophrenia should best be conceptualised as conferring a vulnerability to disorder rather than a disorder itself. Twin studies have shown that genes contribute no more than 50% to aetiology, leaving a major role for developmental and environmental factors (although **Crow** would apparently disagree here). However, this is not sufficient reason to exclude an evolutionary scenario, since one would still expect genes that confer a 50% risk of vulnerability to an "unfit" phenotype to be subject to negative selection and therefore removed from the human genome. The enigma remains, and a putative mechanism for the survival of these genes is still required. In my view, to attribute both past and present survival of schizophrenic phenotypes to "cultural spandrels" is to avoid this central challenge.

Sullivan & Allen favour a balanced polymorphism model with some advantageous behavioural trait exhibited in relatives. Likewise, **Brüne** is prepared to consider a heterozygous advantage and cites new evidence that might support the advantage being located outside the CNS, thus resuscitating Huxley et al.'s original theory (Huxley et al. 1964). If, as Sullivan & Allen state, the 5% advantage required to maintain the polymorphism is difficult to demonstrate, then advocates of this model confront the same problem pointed out by **Crow** in respect of my model, namely, that it is difficult to test and validate.

Keller, **Nesse**, and **Hoffman**, **Hampson**, **Varanko**, **& McGlashan** (**Hoffman et al**.) have offered some fascinating ideas regarding the evolutionary genetics of schizophrenia that help to clarify and strengthen my hypothesis. While I agree with the criticism raised by some commentators – that my genetic argument is vague and ambiguous – I am not prepared to abandon my thesis that the genes for schizophrenia have survived natural selection owing to their association with genes responsible for the evolution of the social brain in our species. On the contrary, the insights of these authors now provide me with material with which I can formulate a more specific and robust model. Although their positions may differ, there appears to be some overlap and the model that follows attempts to integrate this common ground.

Keller rightly asks for clarity regarding my use of the word "genes" in the target article and in reply I confirm that my intended meaning was "allele" (rather than "locus"). I am grateful to this commentator for updating me on recent advances in evolutionary genetics, in particular his clear exposition on the concepts of *mutation-selection balance* and *balancing selection.* I agree that a simple pleiotropic model is inappropriate with regard to schizophrenia and that these two mechanisms may better explain the persistence of susceptibility alleles. While a mutation-selection model may well be suitable, especially in the light of Houle et al.'s (1996) work on "downstream traits," Keller's suggested *antagonistic pleiotropy* model seems to find common ground with **Nesse**'s concept of *cliff-edged fitness* and **Hoffman et al.**'s pruning model. Nesse also considers *antagonistic pleiotropy* a viable model. Consider the following attempt to integrate these ideas into a single model:

1. All humans have at least one susceptibility allele (SA) for schizophrenia because these alleles have been selected for their pleiotropic contribution to the evolution and development of the social brain.

2. There is variation between individuals in the number of SAs, and the presence of increasing numbers of SAs enhances reproductive fitness up to a threshold.

3. An increasing number of SAs corresponds with an increase in the magnitude of the phenotypic trait. In this model the trait is increasing cortical connectivity with associated neural pruning at the histological level and increasingly sophisticated social cognition at the behavioural/psychological level.

4. At a certain threshold (or cliff-edge), the presence of increasing numbers of SAs results in a sharp decrease in the fitness effects of the phenotype. This phenotype constitutes the schizotypal-schizophrenic spectrum. As suggested by **Hoffman et al**., both the schizotypal and schizophrenic phenotype exhibit reduced fitness. Since an increasing number of SAs corresponds with an increase in synaptic connections (both normal and abnormal) and increased peri-adolescent pruning, the schizotypal-schizophrenic brain is characterised by reduced final cortical connectivity. Thus, the diffusion tensor imaging (DTI) findings of reduced FT and FP connectivity are predicted by this model (and not a problem finding, as alleged by **Verleger & Lencer**).

5. As suggested by **Hoffman et al**., the at-risk carrier (the schizotype) exhibits normal or reduced fitness, thus negating the need for a balanced polymorphism model. Additional SAs, environmental factors, and epigenetic effects convert some of these at-risk individuals to full-blown disorder.

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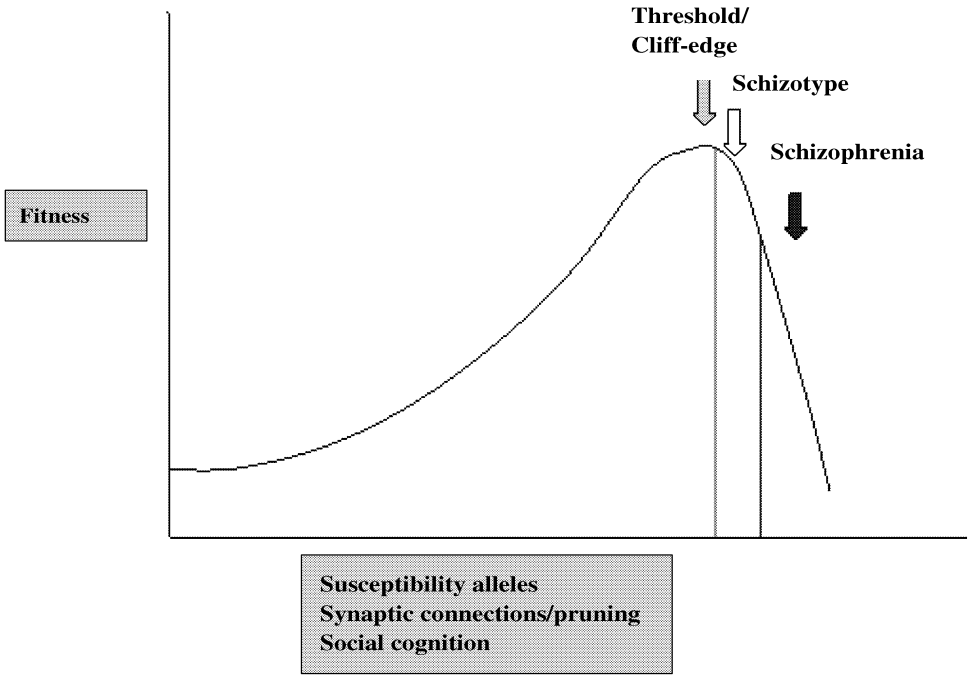

Figure R1 (Burns). Evolutionary genetic model for the "survival" of schizophrenia susceptibility alleles.

This model is depicted in Figure 1 and incorporates **Nesse**'s concept of "cliff-edged fitness" effects as well as **Hoffman et al**.'s proposal that both "at-risk" schizotypes and those with schizophrenia fall beyond the threshold and therefore exhibit reduced fitness effects. It also acknowledges the role of environmental and epigenetic effects in the conversion of the at-risk phenotype to the disorder phenotype, as stressed by **Panksepp & Moskal**. Finally, the model is consistent with **Keller**'s account of antagonistic pleiotropy.

R3. Linking genes to phenotypes

Crow and **Panksepp & Moskal** ask for predictions regarding which genes could be responsible for hominid-specific dysconnectivity in schizophrenia and how in fact these gene effects disturb the structure or function of the cortex. **Gilbert** highlights the difficulty encountered within schizophrenia research in linking specific genes to specific phenotypes. The clinical heterogeneity of the disorder, the variability in neuropathological findings, and the lack of progress in identifying specific gene mutations means that we are dealing with a complex multidimensional syndrome (rather than a specific disease entity) that probably breaks down into a number of disease processes with variable aetiologies. Crow has become an advocate for a single gene mutation model of schizophrenia – a bold and lonely stand in the face of overwhelming evidence to the contrary. This seems to contradict his earlier significant and well-supported concept of a spectrum of psychosis. It is true that protocadherin X and Y have been subject to positive selection in the hominid line, making this an attractive candidate for human-specific traits. However, there must be many other hominid-specific mutations as yet unidentified that could equally likely have played a role in the evolution of human cognition. Until the chimpanzee genome has been mapped entirely and compared against the human genome, we will not be in a position to predict the genes responsible for human cortical dysconnectivity. Furthermore, as I stated in my target article, a simple comparison of human and chimpanzee genomes will not necessarily yield these answers either, because the cognitive differences between us may well be a result of altered gene expression rather than gene mutation.

Furthermore, the chimp is derived from a common ancestor and therefore has undergone its own mutations, so differing loci identified in a side-by-side comparison of genomes may be hominid-specific or chimp-specific. In terms of predicting genes responsible for cortical dysconnectivity, then, I think it is premature to speculate beyond proposing that susceptibility alleles (SAs) for schizophrenia are likely to be among those that have a role in cortical generation. There are likely to be multiple SAs that regulate neurogenesis, differentiation, arborisation, synaptogenesis, myelination, and possibly apoptosis (see my discussion in the target article, sect. 6.2). Of interest is a recent study of caspases (apoptotic proteins) in schizophrenia, which showed that temporal cortical cells are vulnerable to apoptosis in the disorder (Jarskog et al. 2004). This might suggest that there is disordered circuitry prior to adolescent pruning, as suggested in the target article.

R4. The anatomy of the social brain

A number of commentators critique the cognitive bias of my model of the social brain and its dysfunction in schizophrenia. **Panksepp & Moskal** make a case for greater focus on the role of the "foundational social circuits of the mammalian brain," whereas **Gilbert** feels I have stressed "top-down" processes to the exclusion of "bottom-up" effects on social cognition. **Weisfeld** argues for a greater in-

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tegration of the ethological perspective in constructing a model of the social brain, and he joins Panksepp & Moskal in advocating an analysis of basic limbic-driven social emotions and motives in order to understand hominid-specific social cognition. While I fully acknowledge the bias in my target article toward cognitive aspects of social behaviour and cognition, I disagree with the assertion that, like other "cognitive/evolutionary psychological views," I have "ignored too many of the foundational social circuits of the cross-mammalian limbic brain" (Panksepp & Moskal). In section 3.3 of the target article, I included extensive discussion of the amygdala and OFC, structures generally accepted as limbic and of major evolutionary significance. Furthermore, I have acknowledged the role of basic social functions such as affiliative bonding, emotion regulation, and the representation of choice bias. It seems Panksepp & Moskal have misunderstood my conceptualisation of the social brain as an integrated, connected system and this may account for their comments. They group my approach together with "most modular views of evolutionary psychology," and this is unfair in my opinion, for I have explicitly attempted to move away from the modular/evolutionary psychology paradigm as espoused by Fodor (1983), Cosmides & Tooby (1992) and others. In section 4 of the target article I have formulated (vis à vis Mithen 1996) a model characterised by a "breakdown in this modularisation," "cognitive fluidity," and the "integration of specialised information" (target article). Perhaps a little clarification is required, because it was not my intention to ignore the derived limbic contributions to the social brain.

It may be useful to consider the social brain a system of integrated circuits, including both limbic and cortical structures and functionally operating in terms of both "top-down" and "bottom-up" processes. Within such a system one could, anatomically, identify both an *upper social brain* and a *lower social brain,* the former being the cortical aspects and the latter the subcortical aspects. In terms of my model this distinction is artificial; I do not support a modular view but rather an integrated, "fluid" view. However, this may be a useful model, because it acknowledges both primitive and newer aspects in the system. Furthermore, it may coincide with a dimensional approach to psychopathology (see discussion below). Top-down processes include the function of the heteromodal cortex "elaborat[ing] theories of mind and complex sociocognitive strategies" as suggested by **Panksepp & Moskal** and involve attentional, working memory, and executive functions. Bottom-up processes originate in the primitive subcortical regions and regulate basic emotions, motives, and drives (as discussed by **Gilbert** and **Weisfeld**). So, in schizophrenia, for example, Gilbert's "threat and safety systems," which are limbic-based, might interact in a bottom-up fashion with higher cognitive processes to give rise to malevolent voices and paranoid ideation.

This more generalised model of the social brain would also go some way to addressing the concerns of **Bosman**, **Brunetti**, **& Aboitiz** (**Bosman et al**.) who argue that schizophrenia is a disorder of generalised (rather than localised) connectivity. If the social brain is conceptualised in broader terms as an integrated system of both primitive (subcortical) and recently evolved (neocortical) components, then the deficits in attentional, perceptual, and higher functions that characterise the disorder are compatible with the hypothesis that schizophrenia is a disorder of the social brain. In the target article, I acknowledged the role of these generalised cortical functions in social cognition, theory of mind, and the psychopathology of schizophrenia. Furthermore, I believe that Bosman et al.'s discussion of neural synchrony and their work on "anterior-posterior networks regulating top-down and bottom-up processes" does not conflict with a broader view of the social brain. It may be that "high-frequency neuronal synchrony" operates as "a binding mechanism" in a bidirectional manner between the upper and lower social brain. Neural synchrony, according to these commentators, may be responsible for the integration process termed "cognitive coordination" by Phillips & Silverstein (2003). These concepts seem identical to concepts discussed in section 4 of the target article.

Likewise, the well-documented, generalised neurochemical abnormalities in schizophrenia are no longer a problem for the social brain hypothesis if one conceptualises the social brain in broader terms. **Bosman et al**. cite the role of inhibitory GABA interneurons in "the maintenance of reverberatory circuits in large scale networks," and both GABA hypofunction and dopamine hyperfunction in schizophrenia have been correlated with functional dysconnectivity in the disorder (Carlsson et al. 2001; Dolan et al. 1999; Heinz et al. 2003). Abnormal neuronal connectivity in both cortical and subcortical components of the social brain in schizophrenia is likely to correlate with neurotransmitter receptor abnormalities in these circuits, thereby accounting for the "established neurochemical vectors" of the disorder (**Panksepp & Moskal**).

I agree with **Panksepp & Moskal** that the upper social brain (USB) is in part epigenetically derived and is in part "guided by limbic socioemotional functions." But the converse is likely to be true also: The lower social brain (LSB) is unlikely to be wholly exempt from epigenetic modulation (given its long evolutionary history) and since we know that frontotemporal and frontoparietal cortical systems have ancient origins within the primate line, it seems likely that the LSB has been subject to "guidance" by these cortical systems during hominid descent. To limit the role of genes to the LSB and attribute USB components of the social brain solely to "epigenetic programming" is, in my view, reductionist and erroneous. Furthermore, Panksepp & Moskal are incorrect in stating that I have considered the "sociocortical connections unique to humans." In fact, in section 8 of the target article I extensively detailed a continuum of evolved connectivity in both ancestral and extant hominoids.

Likewise, in response to these commentators' comments on the evidence for psychosis in animals, I would draw their attention to section 2.3 of the target article, where I discuss **Crow**'s theory and his assumption that "other species do not have a capacity for psychosis"; this sentence continues: "to date, this is neither proven nor disproven" (cf. target article). In a sense **Panksepp & Moskal** are correct – I do believe "*schizophrenic* genotypes and phenotypes are restricted to our own species"; schizophrenia, as we know it and currently understand it, is a complex polygenic disorder with multiple aetiologies, including environmental and epigenetic processes uniquely evolved in Homo sapiens. Schizophrenia is the result of having highly evolved social brain circuitry. I certainly do not believe, however, that other species do not have the *capacity for psychosis.* The fact that I have elaborated the continuum of connectivity

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that exists in primates implies that I would support Panksepp & Moskal's thesis that cortical derangement would "impair mice less than men" and would give rise to a vulnerability to psychosis-like behaviour. But I don't think one can call the syndrome of stereotypic disorganised behaviour induced by amphetamines and other psychotogenic substances an animal form of schizophrenia. In our psychiatric nomenclature, schizophrenia is a functional disorder, and we are hesitant to diagnose the disorder in the presence of acute drug intoxication. To my knowledge, there is very slim and mostly anecdotal evidence for "spontaneous" psychosis in other species. And even if there were, the absence of language and complex social cognition outside our species means such a syndrome would only approximate the disorder we recognise in humans.

R5. Psychopathology and the social brain

Brüne quite rightly points out that an evolutionary theory of schizophrenia must account for all possible symptoms, and his discussion of mirror neurons and catatonia is a useful addition. Brüne goes on to address the problems posed by the clinical heterogeneity of mental disorders for an evolutionary theory of schizophrenia. He stresses the need for cross-culturally similar prevalence rates of the disorder if an evolutionary perspective is to have relevance and then highlights the problems we have with identifying a core "disease entity." In their fascinating report of their work in Micronesia, **Sullivan & Allen** tell us of great variability in both prevalence rates and clinical presentation in their study population. Does this mean that an evolutionary perspective is rendered meaningless (as argued by **Aleman & Kahn**) and that **Hardcastle** is correct in suggesting that schizophrenia is "a benign trait"? I do not think so, but I do think Brüne gives us cause to reconsider what we mean by the term *schizophrenia.* In section 2.1 of the target article, I refer to the notion of "continua of variation" between schizophrenia and the affective psychoses and between "disorder" and "normality." Clearly, as Bentall (2003), Brüne (2004; and see Brüne's commentary in this issue) and others have maintained, all of the symptoms we attribute to schizophrenia manifest in other psychiatric disorders as well. And, of all of these symptoms, impaired social cognition is probably the most protean and widely found, as Brüne (2004; Brüne et al. 2003; and present commentary) has observed. What are the consequences of these troublesome facts for the social brain hypothesis of schizophrenia?

I believe that an expanded model of the social brain (as detailed above), with both upper and lower components and top-down and bottom-up processes, provides us with a framework within which to explain most mental disorders in terms of a spectrum of social brain dysfunction. **Brüne** tentatively suggests that "virtually all psychiatric disorders fall into the category of 'social brain disorders,'" and he advocates a nosological shift from syndrome- to symptom-based diagnosis: two potentially bold statements, both of which I support. There is good evidence for social brain dysfunction in autism (Baron-Cohen et al. 1985), bipolar disorder (Kinderman 2003), psychopathy (Mealey & Kinner 2003), and dementia (Snowden et al. 2003), and in time further research may well demonstrate similar problems in other psychiatric disorders. Clearly, the aetiological factors responsible for dysfunctional social cognition may vary according to specific expressions of psychopathology (see discussion in Brüne et al. 2003), but I would suggest that the anatomical and functional location of specific disorders within the structure of the social brain may also vary. Since the social brain is a broad system of interconnected cortical and subcortical structures, it is feasible that social brain disorders manifest differently from one another according to where in the system their focal point of pathology lies. For example, anxiety and depression are likely to be an expression of predominant lower social brain (LSB) dysfunction, based in a primary limbic and brain stem pathology with bottom-up processes leading to secondary cognitive disturbance. On the other hand, psychotic illness might be understood in terms of both lower and upper social brain (USB) pathology with bottom-up and top-down processes giving rise to a range of primitive (e.g., threat vs. safety judgements) and recently evolved (e.g., paranoid delusion) symptoms. This model would accommodate and possibly help explain the subgroup of schizophrenia sufferers referred to by **Gilbert** where post traumatic stress disorder is aetiological. Within the spectrum of schizophrenias, one might surmise that those individuals with prominent positive and affective symptoms (whose symptomatology may overlap with bipolar and unipolar mood disorders) have predominant LSB dysfunction, while those with negative schizophrenia have predominant USB dysfunction. See Figure 2.

R6. Auditory hallucinations and theory of mind

Hoffman et al. argue that the major psychotic symptom of auditory hallucinations is not accommodated by my social brain hypothesis of schizophrenia. These authors have modeled auditory hallucinations using intriguing computer pruning experiments that produce "attractor states that intrude into information processing." While I agree with their notion that pruning of developing circuits contributes to structural and functional dysconnectivity in social brain circuits in schizophrenia (see sect. 6.2 of the target article) and that this is the basis of psychotic symptoms, I must convey my scepticism regarding the use of computer technology as an accurate model of brain function. Conrad (1989) was a pioneer in investigating biological information processing and strongly supported what he termed the *brain-machine disanalogy;* that is, that computer modeling cannot be absolute in replicating complex brain function. Despite rapid sophistication in this field, most of his views have been confirmed in the decade since he published his major thesis (Ziegler 2002). Hoffman et al. maintain that since patients with schizophrenia attribute auditory hallucinations to an "other," it follows that they must have intact theory of mind (ToM), because they can distinguish "self" from "other." But in my model of schizophrenia, I explicitly stated that this disorder is characterised by the development of a ToM that later becomes disrupted as the psychosis ensues. So, yes, people with schizophrenia can differentiate self from other (because this ability developed during childhood), but with the emergence of neural dysconnectivity and *cognitive malintegration* (or *disjunction* [Cleghorn & Albert 1990]), the relationship between self and other is misinterpreted, giving rise to positive and negative symptoms.

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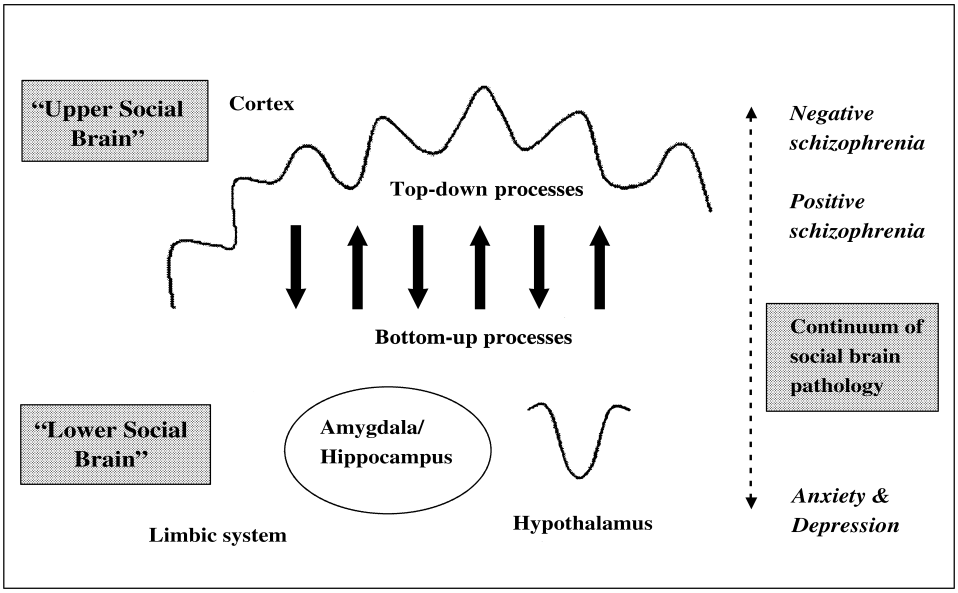

Figure R2 (Burns). Model of the social brain showing different components, processes, and the predominant location of pathology for a continuum of "social brain pathology."

R7. The question of cerebral laterality

Several issues arise from the commentaries regarding the question of cerebral laterality, interhemispheric connectivity, and the ontogeny of orbitofrontal asymmetry (**Crow**, **Aleman & Kahn**, and **Rotenberg**). Crow dismisses the social brain hypothesis of schizophrenia in favour of the language/asymmetry theory he developed; yet he offers no reasons other than that the latter is "a more precise and heuristic evolutionary theory." The three areas of deficiency he identifies in my theory are in my opinion redundant; all are addressed in the target article. He states: "It provides no explanation of the cortical changes," and "dysconnectivity of what, and why?" I protest! In section 9.2, I suggest that *sequential hypermorphosis* may "alter the pattern of expression of individual developmental genes across the cortical plate." This hypothesised mechanism might account for the abnormalities of cortical connectivity in frontotemporal and frontoparietal white matter systems associated with the schizophrenic brain. And surely the detailed discussion of *cognitive malintegration* in section 4 constitutes a thorough explanation of nuclear symptoms? Crow's third point is addressed, both in the target article and in section R3 above; perhaps he and I must agree to differ since the gap that separates us is based upon a fundamental theoretical divergence. He believes in a speciation event during the evolution of Homo sapiens, and this necessitates his adherence to a single gene mutation model. I believe in the gradual emergence of modern human cognition, and this necessitates my adherence to a multiple gene effect model. Therefore, I cannot predict how any one gene might give rise to structural dysconnectivity. Crow is asking me to defend something I don't believe in!

Aleman & Kahn complain that I have ignored the abnormalities of transcallosal white matter connectivity demonstrated in some studies of schizophrenia. Their complaint is justified and I agree that any theory of schizophrenia must acknowledge and account for the findings of both *interhemispheric* and *intrahemispheric* dysconnectivity. In my discussion of the evolution of cerebral asymmetry in section 8.3, I argue that asymmetry has ancient roots within the hominoid lineage and that it emerged as a result of decreasing interhemispheric connectivity and increasingly lateralised specialisation of functions. Therefore, there seems to have been a reciprocal relationship between inter- and intrahemispheric connectivity. If, as I have suggested, the elaboration of intrahemispheric tracts was associated with an increase in developmental vulnerability of these emerging networks, then it is no surprise that interhemispheric tracts would be similarly vulnerable to developmental insults. In schizophrenia where we find abnormal FT and FP connectivity, it follows logically, therefore, that there should also be some differences in transcallosal white matter. I would suggest that FT and FP abnormalities are primary and are genetically determined and that transcallosal abnormalities are a secondary developmental consequence of faulty wiring within the hemispheres. This relationship between inter- and intrahemispheric connectivity accounts too for the findings of reduced asymmetry in the disorder, since aberrant wiring within the hemispheres means that discrete functions are inadequately lateralised during development. In summary, therefore, I am suggesting that intrahemispheric dysconnectivity is primary in schizophrenia and that the findings of both interhemispheric dysconnectivity and reduced asymmetry are a secondary developmental consequence.

Crow does not respond in his commentary to the recent demonstration of directional asymmetries in extant ape species, but he has responded previously to this potential problem for the language/asymmetry hypothesis of schizophrenia (Crow 1998c; 2003; 2004a). Crow questions the validity of the methodology employed in studies showing directional asymmetry in apes. He argues that accurate measurement of, for example, the planum temporale, is dif-

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ficult and that "the apparent asymmetries of function in the above studies (may be) secondary to differences in lesion topography that relate to asymmetries of the cerebral vasculature extrinsic to the brain rather than to asymmetries of the brain itself" (Crow 1998c). He may be right, and as he suggests, "systematic studies are clearly required." As is the case with all groundbreaking discoveries that force us to rethink accepted "truths," only replication of these findings will conclude the matter. Personally, I believe that directional asymmetry has early origins in hominoid descent and the discoveries of Gannon et al. (1998) and others will be vindicated.

Finally, we must address the issue, highlighted by **Sullivan & Allen**, of variability between the sexes in terms of prevalence and age of onset of schizophrenia. Why do males in general have earlier onset of the disorder and why, in Micronesia, does schizophrenia predominate in males and have greater social dysfunction than in females? **Crow** argues that the psychosis gene is subject to sexual selection and that this accounts for these gender differences. However, I think that the contribution from **Rotenberg** in this volume is instructive on this issue and may help to resolve this question without resorting to sexual selection. He refers to the specific ontology of the OFC, differentiating right and left hemispheres in terms of their respective functions and development. He maintains that right OFC maturation commences earlier, progresses faster, and continues longer than left OFC maturation, and he identifies the right frontal hemisphere as responsible for full integration in the "polysemantic context." Furthermore, he states that males have prolonged brain maturation relative to females, providing the potential for marginal increases in creativity but a corresponding increase in vulnerability to pathology.

In terms of the social brain hypothesis, I would suggest that the evolution of the hemispheres progressed as follows: We know from the work of Rilling and Insel (1999a) that intrahemispheric connectivity increases disproportionate to increasing brain size and that interhemispheric connectivity decreases, leading to these authors' conclusion that directional asymmetry was an emergent property of primary intrahemispheric reorganisation and localisation of functions (Hopkins & Rilling 2000; Rilling & Insel 1999a) – see discussion in target article, section 8.3. It follows that the ontological and functional features specific to the right hemisphere (as described by **Rotenberg**) are a consequence of the evolutionary processes described by Rilling and colleagues – that is, they are emergent properties of primary intrahemispheric reorganisation. The notion that the right hemisphere is responsible for the polysemantic context is thus compatible with my hypothesis that evolving FT and FP connectivity in hominid ancestors gave rise to a complex neural net responsible for social cognition in modern Homo sapiens. Likewise, Rotenberg's argument that the right hemisphere matures longer than the left, especially in males, is compatible with my thesis that increasing connectivity and capacity for sophisticated social cognition was associated with increasing vulnerability to developmental insult. It also explains why in certain contexts, males should be more vulnerable than females to neurodevelopmental disorders such as schizophrenia, and why these disorders generally manifest earlier in males than in females. If prolonged cortical development renders the phenotype more vulnerable to pathology, then it is no surprise that males show a disadvantage, since they have prolonged cortical maturation relative to females.

R8. Conclusion

I am grateful to the commentators for forcing me to address certain issues that were either vaguely or inexpertly handled in the target article. Likewise, I appreciate the insights and suggestions offered by those closer to the rock-face of brain research than I. These insights (for example, on current thinking in evolutionary genetics) have, I believe, enriched and strengthened my original thesis. Clearly, there are a host of unresolved and controversial viewpoints, and I make no claim to be nearer the truth than anyone else. However, it remains a fact that the concept of an evolved social brain in our species is gaining support from innovative research methods and the growing acceptance of social processes as a driving force in human descent. The social deficits that characterise most psychopathologies illustrate the unambiguous importance of mature social cognition for healthy individual and interpersonal functioning. Even if I am wrong in some of my speculations regarding the origins of schizophrenia, I hope that this dialogical process printed in the pages of this journal has helped to focus attention on the devastating social dysfunction suffered by individuals living with mental disorders such as schizophrenia.

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Letters "a" and "r" appearing before authors' initials refer to target article and response respectively.

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