The Descent of Madness - Evolutionary Origins of Psychosis and the Social Brain

By Jonathan Burns

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A review by David Kibel

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Jonathan Burn's The Descent of Madness- Evolutionary Origins of Psychosis and the Social Brain- is a very brave book. It is also proudly South African. It is courageous because not only does it attempt to navigate the complex conceptual and linguistic terrain of evolutionary theory but also apply these concepts to the equally complex definitional, psychopathological, developmental and genetic issues that plague our understanding of schizophrenia (broadly and rather unfortunately in my view, referred to as 'madness').

The book builds upon a primary thesis that schizophrenia exists in our species as a 'costly legacy in the evolution of cortical connectivity and social cognition'. Complex cerebral interconnectivity and specialized neural circuits evolved in the hominid line in order to regulate social cognition and the intellectual demands of group living. 'The advantages of becoming socially adept were gained at the cost of this enormous vulnerability of the developing circuits to both genetic and environmental events.'

The book squares up with several vexing questions, which it attempts to marry.

1. What is the engine driving primate and especially human brain evolution given that natural selection does not usually give rise to unnecessary complexity in an organism?

To answer this, Burns follows Humphrey's idea that social cohesion is fundamental to a context in which the transmission of learning skills and knowledge necessary for survival can occur. Social cohesion within the group depends on the possession of complex social cognitive skills by member of that group. The brain's capacity to acquire these skills is accordingly, the evolutionary pressure behind human brain evolution. Group dynamics are not static- they are often ambiguous and fluctuate constantly. Thus, in order to survive, group members need to be skilled in the arts of detection, interpretation and calculation of the relative benefits, and costs of chosen behaviors. Each individual or 'social gamesman' needs to be capable of special sort of forward planning and mind reading- anticipate various alternative responses, plan ones' rebuttal and maintain group cohesion to deal with inter-group conflicts.

Burns inspirationally, makes use of the concept of Ubuntu to encapsulate the profoundly social aspect of our brains. Ubuntu ('a person is a person through other persons') compasses human compassion, forgiveness, love, interconnectedness, and recognizes the fundamental human need to belong and the implication of rejection or isolation.

The new focus on social adaptation driving brain evolution represents a departure from existing theories e.g. as an co-evolutionary response to increasingly sophisticated tool or fire use, spatial memory for foraging, meat-eating, climatic changes or language. Evidence for increasingly sophisticated social intelligence within the repertoire of monkeys and apes is well demonstrated by their extensive use of : alliances and cooperation when directly competing for resources; acquiring dominance on the basis of support given by others; showing long lasting 'friendships' which predict the distribution of mutual help; devoting considerable time and effort to others via social grooming; showing repair of social relationships perturbed by conflict via targeted reconciliation; show knowledge of personal characteristics and affiliation of other members; use social manipulation to gain personal ends including deception.

2. For how long has the capacity for true psychotic states been present?

Amphetamine induced hyperdopaminergic psychotic states are well known amongst mammals. Evidence is presented, though somewhat tenuous, for the existence of naturally occuring 'prepsychotic behaviour' in our nearest extant primate relatives (including speculation that this was 'likely' in hominids such as Austalopithecus, Homo habilis and Homo erectus). However the assumption is that only with the evolution of a mature social mind in modern humans did a capacity for true psychotic illness arise.

Insights from the archaeology and anthropology of Paleolithic cave art, psychotropic substance use and shamanism practices of altered states of consciousness are interpreted to reveal the extent to which early humans were acquainted with quasi-psychotic phenomena and were likely familiar with full-blown psychosis too. The evidence suggested is that shamans themselves were not frankly psychotic, as their function required full social integration and competence.

3. What is the evidence for a neural basis of social cognition in hominids?

Burns presents interesting supportive evidence to illustrate how the social brain evolved during the course of hominid evolution, through a process of brain reorganization and

increasing intra-hemispheric white matter connections – the frontotemporal and frontoparietal connections (uncinate, arcuate fasciculus and anterior cingulum). The inter-hemispheric connectivity apparently has diminished during hominoid phylogeny. Several others structures (anterior cingulate and amygdala) have undergone strong adaptive pressure adapted to group living.

The ability of primates to represent the mental states of others or mind read ('theory of mind' TOM, a term first coined by Premack and Woodruff in 1978 to describe chimpanzees' capacity for deception) refers to the assumption that others have mental states and minds like our own. Imaging studies (including PET, SPECT and fMRI) using TOM paradigms have concluded that neural structures and regions active during TOM tasks correspond broadly, with those implicated in social cognition (ie interconnected regions of the prefrontal, temporal and parietal association cortices).

At a neuronal level however, the search for a neural substrate for social cognition, empathy and bi-directional transfer of information between two individuals has been hugely advanced with Rizzolatti's discovery in 1992, of the remarkable 'mirror neuron' (originally discovered in the ventral motor F5 prefrontal cortex of macaque monkeys, homologous to Broca's speech area in the human brain). These cells revolutionized understanding of leaning and interpersonal behaviour because mirror neurons discharge both when the macaque performs a particular goal-directed action and when it observes another individual performing a similar action. Thus different sets of mirror neurons serve to mirror different actions and intentions (as well as sounds) within the observer. A motor image of the observed action is thus formed in the brain. When the observer later unconsciously plans and prepares to imitate this action, a motor image has already been formed. Learning by observation, feeling for others, theorizing about others intentions are thus embodied within the observer.

Subsequent studies in humans have confirmed this mirror-matching system located in a cortical network consisting of Broca's areas, the premotor cortex, the superior temporal sulcus and posterior parietal cortex. This implicit, automatic and unconscious process of 'embodied simulation' enables the observer to penetrate the world of the other, a multimodal representation or 'shared manifold of intersubjectivity' that underpins our capacity to share feelings and emotions with others. . Thus through the mirror neuron system we have an embedded mechanism for actively engaging in and responding to interpersonal stimuli emanating from the social world.

Further, the posterior and medial aspects of the OFC are considered to be part of a circuit connecting the limbic temporal lobe that is relevant to emotion, particularly related to social stimuli. Ablation of this area in wild monkeys results in significant reduction and losses of behaviours that are considered important for the maintenance of social bonds. Furthermore the socially isolated orang-utan apparently has decreased representation of the limbic OFC and a relative immaturity of the frontal limbic cortex. This may relate to its more solitary life and less complex social organization of this primate.

In humans and primates, the anterior cingulate plays a critical role in evaluation of social performance and conflict monitoring (evaluating choices) as well attachment and bonding, the precursors of human sociability. In humans it is also be shown to be involved in forgiveness and empathy. The hominoid anterior cingulate gyrus has evolved a unique set of projection neurons, the spindle cell. These cells possibly relate to the integration of inputs with emotional overtones and are involved in self-awareness, attention, emotional control and communication.

The primate amygdala contains neurons that respond selectively to facial expression and eye gaze and when surgical lesions are placed in this structure, the animal fails to evaluate new stimuli and puts itself at risk. The corticobasolateral (CBL) nucleus of the amygdala has undergone disproportionate enlargement and connections in higher primates. The amygdala forms an interface between the information processing (eg facial recognition, eye gaze direction etc) of the neocortex and the subcortical emotional systems (primitive threat/defense systems). Thus it is well placed to perform its function as part of the brain's emotional-regulation system. In particular the amygdala seems to play a role in recognition and evaluation of threat and danger i.e. mediating an evolutionary ancient fear response.

4.What is the evidence for functional deficits of social cognition in schizophrenia, and their neural basis?

To answer this question, Burns returns to the original Bleularian meaning of the disturbance of affect, ambivalence and autism in schizophrenia. He points out that this notion has been lost. Rather than a loss of or absence of affect and emotion, the 'negative' or deficit state, fails to convey the distress subjectively experienced by schizophrenics with negative symptoms. A better description of this state is a disturbance described by Louis Sass and others, that of loss of ipseity - the experiential sense of being a 'vital and self-coinciding subject of awareness and experience'. This sense of alienation encapsulates the fist –person perspective on the world (ipse is Latin for 'self' or 'itself').

Loss of ipseity is associated with excessive self consciousness, a loss of 'natural self evidence' as well as the loss of the usual common-sense orientation to reality, that normally enables a person to take for granted so many elements and dimensions of the social and practical world. Patients focus on aspects or processes of action, which would normally go unnoticed. This is both exhausting and distracting and can account for the appearance of apparent detachment. It is also has the effect of objectifying thoughts and feelings'.

This phenomena is born out of research that shows that although patients with schizophrenia do well on may intellectual tasks requiring logical and abstract thought, they have particular difficulties with more common –sense or practical problems, especially problems relating to the social world (with hypertrophy of intellectual tendencies). Thus the primary problem the schizophrenic experiences is a sense of detachment from or loss of natural embeddedness in the world.

Evidence for deficits in social cognition in schizophrenia cited (apart from an interesting personal observation from Darwin observing patients in an asylum), include a rare and fascinating ethological study of schizophrenic behaviour. Schizophrenic patients showed restricted repertoire of defense of personal 'territory', maintaining rigid social

hierarchy and avoidance of any body contact. Imaging evidence is also cited for a relative failure to activate prefrontal cortex on fMRI and failure or distortion in the development of the mirror neuron system. Theory of mind abnormalities have been demonstrated in schizophrenia although interestingly, less severe than in autism.

Of clear interest to Burns are the structural and functional disconnectivity syndromes in schizophrenia visualized on DT-MRI. This technue is able to demonstrate disconnectivity of white matter, myelin sheaths, axonal membrane and microfilaments, reduced functional integrity of the white matter tracts connecting prefrontal cortex to temporal, parietal and anterior cingulate. This has been shown in both schizotypal personality disorder as well as schizophrenia. This evidence supports the central argument that psychosis exists as a spectrum of anatomical disconnectivity.

Thus, because cognitive fluidity depends on healthy function of these social brain circuits, these disturbances render the individual vulnerable to partial or complete mental disembodiment from the 'embodied self'.

The loss of cognitive fluidity has implications for Mithen's proposal that the modern conceptual mind represents a breakdown of modularization allowing for fluid integration of information. The loss of fluidity also has implication for the very interesting idea of the 'mind as theatre'. Psychotic voices like actors of a drama, may represent the misinterpretation of modular 'selves' evolved to enact a plurality of social roles within our own self.

5. If schizophrenia is associated with lower fertility why has it survived evolution and continues with a relatively universally stable incidence?

A proposed genetic mechanism to account for the continued survival of genes conferring risk for schizophrenia, despite its disadvantages to an individual, includes 'increasing numbers of susceptibility alleles' (SA's) up to a threshold corresponding to increased cortical connectivity and increasing risk for psychosis. Beyond the 'cliff-edge' threshold, excess developmental pruning of these 'social brain circuits' results in increasing disconnectivity, reduced reproductive fitness and 'schizotypal' vulnerability to full-blown psychosis. This approach is consistent with a dimensional concept of a continuum of psychotic disorders (schizoptyal spectrum from normal to frank psychosis).

This view is subtly different from a 'balanced polymorphism model' in which unaffected individuals or kin in the schizotypal spectrum, are at some kind of advantage, thus compensating for the apparent disadvantage of the psychotic phenotype (a balanced polymorphism model). These approaches have drawn links between schizotypy and genius, and between divergent thinking and creativity.

Burns differs also from a group selectionist approach (evolution acting at a group level rather than individual gene). For example Stevens and Price argued for a 'group-splitting hypothesis' of schizophrenia. In the ancestral environment they suggest, a group would reach a critical size at which it began to outgrow its resources. At this point a schizotypal individual having undergone a 'mazeway resynthesis' and spurred on by his or her idiosyncratic and iconoclastic ideas and possible 'voices of the gods' would offer a vision of a new and better 'promised land' to those who would follow. The followers would enter his or her delusional world and the group would split, dispersing human ancestors across the planet and creating new cultures. This approach is appealing because individuals like David Koresh and Jim Jones indeed fathered many children and infact many cults have survived and continue to flourish.

In both previous cases, Burns argues that epidemiological evidence fails to demonstrate such a reproductive advantage.

Finally Burns takes issue with Crow's theory of psychosis which relies on a sudden speciation (saltational) leap or event occurring 100-150 000 years ago in which a 'porocadherin' gene initiated the beginnings of lateralisation in the human brain and specialization of the language area on the left side. For Burns this theory is implausible firstly because it is not consistent with evidence of the existence of a primitive form of psychotic behavioural syndrome in non-human primates. Secondly it relies upon discontinuity in the evolution of language, rather than a gradualistic model for language development. A gradualistic model supports the idea that articulate speech emerges from a gradual process of evolving communication in higher mammals.

6. How can the developmental abnormalities in schizophrenia be used as a tool and incorporated into this model?

A particularly complex and rather difficult chapter is devoted to the developmental issues (ontogeny) in schizophrenia. Proposed here is that a simple gene switch in the homonid line has resulted in an ever-increasing extension of each phase of development or prolonged neotony. (A process neologistically named sequential hypermorphosis). This had enabled the expansion or lengthening of learning periods and skill development, leading to the evolution of the prolonged phase of human childhood.

The pruning and apoptosis later seen in adolescence appears to be excessive in schizophrenia or reveal a prior abnormal developmental. Interestingly, Hoffman's using computer models have demonstrated that excessive pruning may lead to the emergence of 'attractor states that intrude into information processing', and possible auditory hallucinations. Circuits most severely affected by this process appear to be those that evolved most recently and comprise the very same areas posited as the social brain.

Discussion

The social brain model is suggested as continuum between lower and upper brain areas such that:

''…anxiety and depression are likely to be an expression of predominant lower social brain dysfunction, basic in a primary limbic and brainstem pathology with bottom-up processes leading to secondary cognitive disturbance. Psychotic illness might be understood in terms of both lower and upper social brain pathology giving rise to a range of primitive threat vs safety judgments and recently evolved (paranoid) symptoms.''

For me, this kind of conceptualization of the social brain though interesting, is too lose and all encompassing. It tends to water down the power of the model for schizophrenia specifically. He is correct however to draw attention to the exquisite sensitivity of people with depression and anxiety

disorders to social issues, social threats, cues and losses. These must involve theory of mind (eg worry about what other's are thinking)

I also felt a need for a deeper discussion (apart from mirror neurons and theory of mind) of the specific role of the prefrontal cortex in achieving the kind of social cognitive fluidity that is argued for. For example the capacity for sustained attention; guiding behaviour by internal representation (ie not 'out of sight out of mind'), resolving ambiguity, deciding which set of questions to ask, search engine function etc. are all aspects of executive function relevant for the social brain model.

Further, mention could have been made of an important ratio, that of- prefrontal/rest of cortex -(accurately measured by a variety of methods including areas receiving projection from the mesocortical dopamine system). By this, mammals and primates show exponential increases - 3.5% in cats, 7% in dogs, 11.5% in gibbons, 17% in chimps, and 29% in humans.[1] Small increments along this scale have ever increasingly larger capacity for sustained attention, flexibility and internal representation with obvious implication for the social brain model.

Although the social brain model has a 'top-down' emphasis and deals especially well with the issue of negative symptoms, to be an even more credible model requires it to incorporate a fuller understanding of 'bottom up' issues e.g. the dopaminergic projections to distant/recent cortical areas, salience and the role of dopamine in making sense of environmental contingences, extracting meaning from circumstances. As Panksepp put it 'When dopamine systems are overdriven as in schizophrenia our imagination outstrips the constraints of reality'; we confuse correlations with causality and thereby form delusions'.[2]

The cortico-cortico connectivity discussed is similar to Edelman and Tononi's 'reentrant circuits', the very specialized heteromodal, and reciprocal cortical connections, considered to form the basis of consciousness.[3] Also, Whitehead's brilliant article 'Social Mirrors and Shared Experiential Worlds' elaborates the idea of the mind as theatre or 'theatre of mind' and well worth integrating into this model.[4]

Finally Burns' somewhat repetitive restatement of his differences with Tim Crow appear somewhat churlish and irritating. The notes and references are excellent and the diagrams and table very helpful. The cover of this book is beautifully chosen.

This book is a valuable addition to the fledgling subject of evolutionary psychiatry. I highly recommend this book and hope it is read.

References

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