Understanding the symptoms of "schizophrenia" in evolutionary terms

Martin Brüne

Centre for Psychiatry and Psychotherapy, University of Bochum, 44791 Bochum, Germany. **martin.bruene@ruhr-uni-bochum.de**

Abstract: An evolutionary theory of schizophrenia needs to address all symptoms associated with the condition. Burns' framework could be extended in a way embracing behavioural signs such as catatonia. Burns' theory is, however, not specific to schizophrenia. Since no one single symptom exists that is pathognomonic for "schizophrenia," an evolutionary proposal of psychiatric disorders raises the question whether our anachronistic psychiatric nosology warrants revision.

Burns' "social brain" hypothesis of schizophrenia sets up new standards of evolutionary thinking in psychiatry by integrating evolutionary ontogeny, cerebral connectivity, psychopathology, and molecular genetics. A couple of additional issues relating to a possible evolutionary background of schizophrenia are worth mentioning.

Firstly, referring to Burns' pleiotropy model of schizophrenia, it is by no means clear that the alleged adaptive advantage of heterozygous carriers of genes predisposing to schizophrenia would lie within the realm of the nervous system (i.e., cognition, emotion, behaviour). Huxley et al.'s (1964) proposal, for example, originally suggested that such an advantage could also be unrelated to the CNS. This possibility has been discussed in relation to other genetically transmitted diseases that primarily affect the nervous system, such as Tay-Sachs disease and Huntington's disease (Polimeni & Reiss 2003). With respect to schizophrenia, Yovel et al. (2000) have shown a greater activity of natural killer cells in patients with schizophrenia, a finding that could buttress earlier hypotheses of a greater resistance in schizophrenic patients and their relatives against infectious diseases, and therefore deserves further consideration.

Secondly, an evolutionary theory of schizophrenia ought to account for all possible symptoms associated with the condition. Whereas most evolutionary hypotheses, including Burns' theory, focus on Schneiderian first-rank symptoms such as thought insertion, thought withdrawal, or delusions of mind-reading, they relatively neglect psychomotor symptoms – for example, catatonia. Burns rightly highlights the putative role of so-called spindle cells located in the anterior cingulate cortex (ACC) that only recently emerged in the great apes and humans but have been found absent in lesser monkeys (Nimchinsky et al. 1999). Although the exact function of spindle cells is not known, they are probably involved in complex tasks of motor control and control of cognitive impulsivity. Bilateral lesions of the ACC, on the other hand, lead to akinetic mutism and insufficient suppression of automatic subroutines (Paus 2001). These pathologies strikingly resemble catatonic symptoms such as stupor, mutism, and stereotypies (Brüne 2004a). In extension to Burns' theory, a second cell type found in the prefrontal and temporal cortex in primates' brain areas that constitute parts of the social brain may shed further light on the origin of catatonic symptoms. Rizzolatti and coworkers (Rizzolatti & Arbib 1998; Rizzolatti et al. 2002) have described what they have called "mirror neurons" located in the ventral premotor cortex of monkeys. These cells have also been found in Broca's area and the superior temporal sulcus in humans. Their functional significance clearly lies within the social domain: they are selectively active when observing hand and mouth movements in conspecifics and when imitating the observed behaviour (Rizzolatti & Arbib 1998). The existence of mirror neurons in Broca's area of the human brain has fuelled the hypothesis that human language evolved in the first place from gestural communication, and that the human Brodmann area 44 is also involved in action understanding and imitation (Rizzolatti et al. 2002). I have speculated elsewhere that a disinhibition of mirror neuron activity could be involved in a subset of catatonic echo phenomena, namely echopraxia, echolalia, and automatic obedience, as well as *mitgehen* and *mitmachen* (Brüne 2004a). To my knowledge this hypothesis has not yet been empirically tested.

Third, there is a more serious problem with evolutionary hypotheses of schizophrenia in general. It follows from what I have outlined above – in line with Burns' proposal – that a symptom or syndrome-based approach to psychosis in evolutionary perspective is crucial to the understanding of schizophrenia. According to Bentall et al. (1988), however, not a single one sign or symptom exists that is specific or pathognomonic to schizophrenia. Although it has been suggested that the prevalence rate of "core schizophrenia" is cross-culturally similar (Jablensky 2000) and thus relatively independent of environmental influences (which would support a common origin of the disorder), we simply do not know whether schizophrenia or its subtypes represent true "disease entities," or, as Burton-Bradley (1990) once put it, whether our diagnostic system is too narrow for a valid cross-cultural comparison. The assertion of cross-culturally similar prevalence rates of schizophrenia is, however, vital for every evolutionary hypothesis that argues for compensatory advantages in heterozygous gene carriers, regardless whether favouring a pleiotropy model, as Burns does, or a single mutation model like Crow's (1995b). Burns' social brain theory of schizophrenia implicitly assumes that schizophrenia is a useful concept in cross-cultural perspective. On the other hand, I am wondering whether it is specific enough to schizophrenia. Functional impairments of social brain connectivity have similarly been described in bipolar affective disorder and in obsessive compulsive disorder (OCD). Brain imaging studies in OCD, for example, have revealed an increased metabolism or regional blood flow in the orbitofrontal cortex, the anterior cingulate, the basal ganglia, and the thalamus (Saxena & Rauch 2000). Furthermore, in a functional brain imaging study using a symptom provocation paradigm in patients with OCD, Breiter et al. (1996) found a significant bilateral activation of the anterior and posterior orbital gyri, the superior, middle, and inferior frontal gyri, the anterior cingulate cortices, the temporal cortices, the right caudate and left lenticulate, as well as in the left insula and bilateral amygdala – precisely those brain structures which Burns identifies as being involved in the pathogenesis of schizophrenia.

I am tempted to say that virtually all psychiatric disorders fall into the category of "social brain disorders." If we as clinicians would dare abandon the anachronistic psychiatric nosology (this is what we already do in clinical practice, e.g., regarding psychopharmacological treatment), Burns' framework of social brain evolution could be a significant contribution to an empirically testable evolutionary conceptualisation of the dimensions of psychiatric disorders.

Language and asymmetry versus the social brain – where are the testable predictions?

T. J. Crow

SANE POWIC, Warneford Hospital, Oxford OX3 7JX, England.
tim.crow@psychiatry.oxford.ac.uk
www.psychiatry.ox.ac.uk/powic/index.htm

Abstract: I agree with Burns that an evolutionary theory is required, but I question his multifactorial premise. The arguments for an evolutionary theory are stronger, and one that is more precise than that presented by Burns has already been formulated. This theory, that schizophrenia is "the price that Homo sapiens pays for language," (Crow 1997a; 2000b, 2004c), generates testable predictions absent from Burns' presentation.

I concluded that an evolutionary explanation was necessary when I convinced myself (Crow 1984) that the theory that schizophrenia is due to the spread of a slow virus between genetically predisposed individuals (Crow 1983) was wrong (for data and argument see Crow & Done [1986]. Not only is there no evidence for

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