Precursors of psychosis as pointers to the *Homo sapiens*-specific mate recognition system of language

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Malmberg *et al* (1998)† have made a significant contribution to the now quite substantial literature on the 'premorbid' personality characteristics of those who in adult life develop disturbances of psychological function that are described as psychotic or schizophrenic. Their own study of Swedish conscripts as they point out, has strengths, but also some weaknesses (notably confined to males, it provides no information on gender differences). To suggest an interpretation that differs significantly from that of the authors is not to diminish the value of the study. It can be viewed from different perspectives.

For instance, one can suggest that the medical perspective that there are risk factors that predispose to a disease called 'schizophrenia' is only one way to look at the findings. That there is a categorical entity as originally envisaged by E. Kraepelin and E. Bleuler is highly doubtful. Different sets of criteria for defining an illness as 'schizophrenia' identify different populations of subjects (Endicott *et al*, 1982). Rather, it seems there is a spectrum of psychotic states which merge one into another without it being possible to establish non-arbitrary boundaries between states. Recent interest in dimensions of psychotic symptoms (positive, negative, affective) illustrates one aspect of the problem – why if there are true entities are there also dimensions? What are the dimensions variations of?

KRETSCHMER'S ALTERNATIVE

Diametrically opposed to the original Kraepelinian disease entity concept is that of Kretschmer (1925). He wrote:

> "we can never do justice to the endogenous psychoses so long as we regard them as isolated unities of disease, having taken them out of their natural heredity environment, and forced them into the limits of a clinical system. Viewed in a large biological framework the endogenous psychoses are nothing other than marked accentuations of normal types of temperament."

From this perspective the personality characteristics of those who later develop psychotic disturbance ought to be indices of the relevant dimensions of temperament. Unfortunately, such concepts as we have of temperamental dimensions (introversion–extroversion, neuroticism, conscientiousness, openness, etc.) lack the type of biological and genetic framework that Kretschmer sought. What are the dimensions of variation of human psychological structure? Why are they preserved? What function is being selected for?

My own view is that the phenomena of psychosis, because they are so incomprehensible and because they apparently defy simple genetic principles, present us with a paradox that, if directly addressed, provides the key to the origin of *Homo sapiens* and its intrinsic diversity. Malmberg *et al* (1998) conclude that their results are "consistent with a multi-factorial aetiology for schizophrenia and offer tentative support for a psychological disturbance mediating genetic and environmental effects on the causal pathway to the illness". But the evidence for an environmental factor is insubstantial. None of the variables they identify in their own study as most strongly associated with the diagnosis (prefer small groups, more sensitive than others, no steady girlfriend) can plausibly be regarded as an independent risk factor. Nor have the cohort studies (Done *et al*, 1991, 1994; Jones *et al*, 1994) succeeded in identifying such factors. Rather, they and the case–control studies point to a constellation of characteristics that are more commonly or more strongly seen in those who later develop psychosis. But how are these characteristics to be described? What is their fundamental significance?

THE PRICE THAT *HOMO SAPIENS* PAY FOR LANGUAGE

This is where the phenomena of psychosis itself, viewed as suggested in the Kretschmerian quotation above but also in an evolutionary context, become crucial. Nuclear schizophrenic symptoms, we can now be reasonably sure on the basis of the World Health Organization Ten Country Study (Jablensky *et al*, 1992) and other evidence (Crow, 1995) occur in all societies at about the same frequency. They are a characteristic of human populations, but they are also biologically disadvantageous in the sense that individuals who develop such symptoms are less likely than the rest of the population to pass on their genes. Why are these genes not selected out? I suggest that the relevant variation (a) crosses the population as a whole (i.e. is not confined to a sub-set of the population); (b) has been present since the origin of modern *Homo sapiens* (i.e. represents a stable polymorphism) and (c) is associated with the speciation characteristic of language. In other words schizophrenia is the price that *Homo sapiens* pays for language.

This theory casts light on the nature of the symptoms. What these represent is language at the end of its tether. Thus interpreted, the nuclear symptoms of schizophrenia tell us about the relationship between thought and speech, and that the dimension of 'indexicality' (the distinction between the 'I' of the speaker and the 'you' of the hearer, and related phenomena) is fundamental to the organisation of language (Crow, 1997*a*).

LANGUAGE AS THE SPECIFIC MATE RECOGNITION SYSTEM

If we assume that a dimension of linguistic competence is central to the problem, one can now ask at what point does this become relevant to gene selection? The obvious answer is at the point of mate selection – genes are passed on by those who are successful in finding mates. What is clear from the study of Malmberg *et al* is that the predictors of psychosis are all correlates of social competence and that the best predictor, lack of a girlfriend, points directly to the central paradox: this genetic variation is relevant to the mate selection process, and predicts failure, yet the variation is retained in the population.

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Therefore, one can argue that the variation is an integral component of the diversity associated with what Paterson (1985) describes as "the specific mate recognition process", that is the defining characteristic of the species.

For this reason the restriction of the sample of Malmberg *et al* to males is a limitation. Gender differences in schizophrenia, for example in age of onset, need to be explained (and have been cited as evidence for a role for sexual selection in *Homo sapiens* by Gould & Gould (1989)). As noted by Malmberg *et al* there are indications that the precursors of psychosis in childhood are gender-specific. In his School Report Study, Watt (1978) described the girls who subsequently developed schizophrenia as being emotionally unstable, introverted and passive and the boys as emotionally unstable but also disagreeable and defiant of authority in their early school years. In the UK National Child Development cohort we found the boys who subsequently developed schizophrenia to be more anxious and hostile towards children and adults at ages 7 and 11 but this was not true of the girls, who became depressed and withdrawn at the age of 11 years (Crow *et al*, 1995).

Is there a single variable that could account for these deviations, including the gender differences? A finding in the National Child Development Study that was stable across ages of testing was reading impairment. This may reflect a defect in inter-hemispheric integration, and there is other evidence both anatomical (Crow, 1997*a*,*b*) and functional (Crow *et al*, 1996) that individuals with schizophrenia are delayed or arrested in cerebral lateralisation. Gender differences are present in handedness and anatomical asymmetry and can be explained by a gene that is present in homologous form on both the X and the Y chromosome (Crow, 1993; Corballis *et al*, 1996). There is a case that such a gene played a critical role in human evolution.

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PSYCHOSIS AND LANGUAGE

Kretschmer's view that the endogenous psychoses are nothing other than marked accentuations of normal types of temperament is proposed as the framework within which the relationship between premorbid adjustment and the phenomena of psychosis should be considered. The variations, it is suggested, are genetic and intrinsic to human populations – they represent diversity in the developmental course of the *Homo sapiens*-specific characteristic of language. A single variable, degree of cerebral lateralisation, can account for some anatomical and functional deviations (e.g. reading delay) and is also relevant to gender differences in precursors and onset. Language in *Homo sapiens* is the specific mate recognition system: the genetic diversity associated with this function represents variation that reflects on the origin of the species. The phenomena of psychosis itself are the key to the genetic and neural diversity associated with language.

REFERENCES

Corballis, M. C., Lee, K., McManus, I. C., *et al* (1996) Location of the handedness gene on the X and Y chromosomes. *American Journal of Medical Genetics (Neuropsychiatric Genetics)*, **67**, 50–52.

Crow, T. J. (1993) Sexual selection, Machiavellian intelligence and the origins of psychosis. *Lancet*, **342**, 594–598.

— (1995) A continuum of psychosis, one human gene and not much else – the case for homogeneity. *Schizophrenia Research*, **17**, 135–145.

— (1997*a*) Is schizophrenia the price that *Homo sapiens* pays for language? *Schizophrenia Research*, **28**, 127–141.

— (1997*b*) Schizophrenia as failure of hemispheric dominance for language. *Trends in Neurosciences*, **20**, 339–343.

—, Done, D. J. & Sacker, A. (1995) Childhood precursors of psychosis as clues to its evolutionary origins. *European Archives of Psychiatry and Clinical Neuroscience*, **245**, 61–69.

—, — & — (1996) Cerebral lateralisation is delayed in children who later develop schizophrenia. *Schizophrenia Research*, **22**, 181–185.

Done, D. J., Johnstone, E. C., Frith, C. D., *et al* (1991) Complications of pregnancy and delivery in relation to psychosis in adult life: data from the British perinatal mortality survey sample. *British Medical Journal*, **302**, 1576–1580.

—, Crow, T. J., Johnstone, E. C., *et al* (1994) Childhood antecedents of schizophrenia and affective illness: social adjustment at ages 7 and 11. *British Medical Journal*, **309**, 699–703.

Endicott, J., Nee, J., Fleiss, J., *et al* (1982) Diagnostic criteria for schizophrenia: reliabilities and agreement between systems. *Archives of General Psychiatry*, **39**, 884–889.

Gould, J. L. & Gould, C. G. (1989) *Sexual Selection*. New York: W. H. Freeman.

Jablensky, A., Sartorius, N., Ernberg, G., *et al* (1992) Schizophrenia: manifestations, incidence and course in different cultures. A World Health Organization Ten Country Study. *Psychological Medicine – Monograph Supplement*, **20**, 1–97.

Jones, P., Rodgers, B., Murray, R., *et al* (1994) Child development risk factors for adult schizophrenia in the British 1946 birth cohort. *Lancet*, **344**, 1398–1402.

Kretschmer, E. (1925) *Physique and Character*. London: Kegan Paul, Trench, Trubner.

Malmberg, A., Lewis, G., David, A., *et al* (1998) Premorbid adjustment and personality in people with schizophrenia. *British Journal of Psychiatry*, **172**, 308–313.

Paterson, H. E. H. (1985) The recognition concept of species. In *Species and Speciation* (ed. E. S. Vrba), pp. 21–29. Pretoria: Transvaal Museum Monograph.

Watt, N. F. (1978) Patterns of childhood social development in adult schizophrenics. *Archives of General Psychiatry*, **35**, 160–165.

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