and the relationship between psychosis, personality disorder and creativity.

A major stumbling block for the theory was the overlap it predicted between the diatheses for schizophrenia and high-functioning autism as at that time it was generally accepted that the two syndromes were unconnected. It is therefore most gratifying and opportune to read Dr Wolff's conclusion, also in the November issue, that schizoid disorders and autism are likely to be on a continuum (*Journal*, November 1991, **159**, 621–625).

Finally, I would like to draw attention to a little known work by Henry Maudsley who appears to have been the first to see the significance of genetic diversity for psychiatry. I used the following passage as the introduction to an earlier unpublished manuscript (Farley, 1974).

> "In the long run it is perhaps better for the species that there is here and there a family stock of such constitutional instability and tendency to variation, even though variation fated to go astray, than it would be for every stock to rest in the stable equilibrium of a set adaptation to its surroundings, bee-like in busy, or sheep-like in placid routine of automatic existence". (Henry Maudsley, 1908).

FARLEY, J. D. (1976) Phylogenetic adaptations and the genetics of psychosis. *Acta Psychiatrica Scandinavica*, **53**, 173–192.

MAUDSLEY, H. (1908) *Heredity, Variation and Genius*. London: Bale & Danielson.

REVELEY, A. M., REVELEY, M. A., CLIFFORD, C. A., *et al* (1982) Cerebral ventricular size in twins discordant for schizophrenia. *Lancet*, **i**, 540–541.

SUDDATH, R. L., CHRISTISON, G. W., TORREY, E. F., *et al* (1990) Anatomical abnormalities in the brains of monozygotic twins discordant for schizophrenia. *New England Journal of Medicine*, **322**, 789–794.

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AUTHOR'S REPLY: Dr Farley is correct in supposing that I had not read his 1976 paper. Now that I have done so I would like to draw attention to the logic of the first paragraph of that paper that I find persuasive:

> "It is paradoxical that as the evidence for inherited factors in the aetiology of the functional psychoses becomes more complete, so theories as to the nature of what is genetically transmitted grow more indefinite. The high prevalence of functional psychosis, the distribution of cases, and evidence for some continuity between psychotic and normal behaviour, have all led to the increasing acceptance of polygenic theories at the expense of the heuristically more valuable monogenic theories. In the case of schizophrenia, the major proponents of the polygenic position suggest only that the genes predispose the subject specifically to develop this disease (Gottesman & Shields, 1972). No indication is given as to their function in the normal subject, who, by definition, also carries them. Bearing in mind, firstly, that the biological value of polygenes is to introduce graded variation into adaptations of survival value and, secondly, that the reproductive rate is reduced in overt schizophrenia, one would anticipate that these genes would also be responsible for transmitting something of considerable biological importance to man."

This seems to me to describe the nature of the problem succinctly, and to indicate the directions in which we should look for an answer. I would like to think that the polygenes to which Dr Farley refers constitute allelic variations at a single locus (as argued for example by Allen & Sarich, 1988), and therefore will be more accessible to investigation than additive variation at a number of unrelated genetic loci, but there is as yet no strong evidence on this point.

Of additional interest is the emphasis that Farley (1976) places on the studies of Odegaard (1963) and Mitsuda (1967) on the genetics of psychosis. Neither of these authors adopts a Kraepelinian interpretation (i.e. the view that schizophrenia and manic–depressive psychosis are distinct entities) but rather construe their findings in terms of continuous variation. In this respect the findings, and Dr Farley's theory, are in line with the recent studies from Mainz (Maier & Lichtermann, 1991) and with continuum concepts of psychosis (Crow, 1986, 1990*b*).

Dr Farley's contribution provokes the question of who first formulated an evolutionary theory of psychosis, and in what way do his and my own (Crow 1990*a*, 1991) hypotheses relate to earlier concepts. I am aware of a number of hypotheses of this type, and am working on a review of this neglected field. I am grateful to Dr Farley for drawing my attention to aspects of which I was unaware.

ALLEN, J. S. & SARICH, V. M. (1988) Schizophrenia in an evolutionary perspective. *Perspectives in Biology and Medicine*, **32**, 132–153.

CROW, T. J. (1986) The continuum of psychosis and its implication for the structure of the gene. *British Journal of Psychiatry*, **149**, 419–429.

— (1990*a*) Temporal lobe asymmetries as the key to the aetiology of psychosis. *Schizophrenia Bulletin*, **16**, 433–443.

— (1990*b*) The continuum of psychosis and its genetic origins. *British Journal of Psychiatry*, **156**, 788–797.

— (1991) The origins of psychosis and 'The Descent of Man'. *British Journal of Psychiatry*, **159** (suppl. 14), 76–82.

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