

variously as volume change, shape change or a result of other processes altering voxel intensity, we dispute the simplistic assertion that region of interest methodologies are 'more accurate' – such methodologies have their own difficulties, in particular with interrater reliability and the optimal parcellation boundaries chosen for structures, and the two methodologies are perhaps better viewed as complementary. Region of interest analyses of a similar sample demonstrated that volume deficits of the hippocampus and amygdala characterise schizophrenia but not bipolar disorder (Marshall *et al*, 2004; McDonald *et al*, 2006). This is consistent with our computational morphometry study – and with Kraepelin's seminal dichotomy.

Craddock, N. & Owen, M. J. (2005) The beginning of the end for the Kraepelinian dichotomy. *British Journal of Psychiatry*, **186**, 364–366.

Marshall, N., McDonald, C., Schulze, K., et al (2004) Amygdala volume in patients with schizophrenia or bipolar disorder from multiply affected families and their unaffected relatives. *Schizophrenia Research*, **67** (suppl. 1), 235.

McDonald, C., Bullmore, E. T., Sham, P. C., et al (2004) Association of genetic risks for schizophrenia and bipolar disorder with specific and generic brain structural endophenotypes. *Archives of General Psychiatry*, **61**, 974–984.

McDonald, C., Marshall, N., Sham, P., et al (2006) Regional brain morphometry in patients with schizophrenia or bipolar disorder and their unaffected relatives. *American Journal of Psychiatry*, in press.

Strakowski, S. M., Adler, C. M. & DelBello, M. P. (2002) Volumetric MRI studies of mood disorders: do they distinguish unipolar and bipolar disorder? *Bipolar Disorders*, **4**, 80–88.

C. McDonald Department of Psychiatry, National University of Ireland Galway, Galway, Ireland. E-mail: colmmcdonald@nuigalway.ie

E. Bullmore Brain Mapping Unit, University of Cambridge, Department of Psychiatry, Addenbrooke's Hospital, Cambridge, UK

R. Murray Division of Psychological Medicine, Institute of Psychiatry, King's College London, UK

Social defeat and schizophrenia

Selton & Cantor-Graae (2005) relate schizophrenia to social defeat. Given Darwin's theory of intrasexual selection, social defeat is inevitable for a proportion of any population, and it is not unlikely that we are seeing this unselected or deselected proportion in the psychiatric clinic. The response to social defeat is variable. In chimpanzees there is conditional reconciliation, in which the defeated animal engages in affiliative behaviour with the one who has defeated him (Aureli *et al*, 2002). The hugging and

kissing ritual relieves post-conflict anxiety (indicated by scratching and other self-directed acts), so that in the chimpanzee world the sun goes down on no one's wrath. In partially migratory species of birds, such as the robin, the defeated birds who have no territories migrate, and if they return in the spring they may find that the winners have succumbed to the cold. In partially hibernating species the defeated animals hibernate. In general, in territorial species defeated animals disperse, whereas in group-living species they stay in the group in a subordinate role.

I think that defeated humans have the alternative defeat strategies of either dispersing or staying in the group. The 'schizotypal' appears to be a dispersal phenotype, modified from the usual mammalian dispersal phenotype because of the uniquely cohesive structure of human groups, which are held together by common belief systems. When a person with this phenotype is defeated, they develop a new belief system, recruit followers and take them off to a new location (Stevens & Price, 2000). This appetitive behaviour may well require stimulation of the dopamine reward system, as was found in defeated mice, which being territorial disperse when defeated. However, when defeated the depression-prone human stays in the group in a subordinate role. He may be happily reconciled to this subordination or he may use the depressive strategy of 'deceiving downwards' in which he develops the cognition that he is not such a useful member of the group as he thought he was (Hartung, 1987). This depressive strategy may involve some downregulation in the hippocampus, as occurs in defeated rats, which are group-living animals (McEwen, 2005).

In general, we think people with the schizotypal phenotype become depressed when dispersal is blocked whereas those who are prone to depression become depressed when reconciliation is blocked. People with the schizotypal phenotype and depression also have their new belief system, which in the absence of followers is likely to be labelled delusion, and the unworldly prophet is then looked after not by adoring acolytes but by psychiatric nurses.

Aureli, F., Cords, M. & Van Schaik, C. P. (2002) Conflict resolution following aggression in gregarious animals: a predictive framework. *Animal Behaviour*, **64**, 325–343.

Hartung, J. (1987) Deceiving down: conjectures on the management of subordinate status. In *Self-Deceit:*

An Adaptive Strategy (eds J. Lockard & D. Pulhus), pp. 170–185. Englewood Cliffs, NJ: Prentice-Hall.

McEwen, B. S. (2005) Glucocorticoids, depression, and mood disorders: structural remodelling in the brain. *Metabolism*, **54**, (suppl. 1), 20–23.

Selton, J. P. & Cantor-Graae, E. (2005) Social defeat: risk factor for schizophrenia? *British Journal of Psychiatry*, **187**, 101–102.

Stevens, A. & Price, J. (2002) *Prophets, Cults and Madness*. London: Duckworth.

J. S. Price South Downs Health NHS Trust, Brighton General Hospital, Elm Grove, Brighton BN2 3EW, UK. E-mail: johnscottprice@hotmail.com

Selton & Cantor-Graae (2005) proposed that long-term experiences of social defeat may sensitise the mesolimbic dopamine system, increasing the risk for schizophrenia. Regrettably they continued the tradition of ignoring the distal evolutionary perspective. An underemphasised observation is that although neurological illnesses have lifetime prevalence rates in the order of thousands, prevalence rates for psychiatric illness often lie between 1% (as for schizophrenia) and about 20%. When considering highly disabling conditions such as schizophrenia, depression or anxiety, one must consider the survival implications. Over evolutionary time if there were not some adaptive advantage these genes would have been eliminated. The suggestion that these conditions are products of modern culture is untenable, as they are found in all cultures and have been observed back in time as far as history permits. Furthermore, animals certainly have depression and anxiety.

Selton & Cantor-Graae could have referred to the book by Stevens & Price (2000) on the evolutionary adaptiveness of social subordination and schizophrenia. They proposed that schizotypal individuals at times of social crises may come to the fore and lead individuals with similar genes in new directions. Similarly, work by Gilbert (1992) and Sloman (2000) on depression and defeat warrant consideration.

Evolutionary perspectives often suggest obvious but new directions for gene-environment research. For example, I have proposed a model of post-traumatic stress disorder (PTSD) based on mammalian defences (Cantor, 2005). An understanding of these suggests that looking for genes for the entity PTSD is misguided. The six mammalian defences operate under different selection regimes, therefore greater evolution of one will be associated with a