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Correspondence

Contents: Strength of the genetic effect in schizophrenia/Cognitive-behavioural therapy for schizophrenia/Obsessive-compulsive symptoms and clozapine/Making the most of interpreters/ Prodromal symptoms of schizophrenia/High-dose antipsychotic medication/Epilepsy in Down's syndrome/Defeating depression in Zimbabwe/ Glucocorticoids, serotonin and mood/Seizures and antidepressants/Darier's disease cosegregating with affective disorder/Social competence and adolescent psychosis/ICD-10: a neuropsychiatrist's nightmare?/A catecholamine model of fatigue/ Dystonia induced by amphetamine and haloperidol/Adrenoceptor activity and adenylate cyclase inhibition in depression.

Strength of the genetic effect in schizophrenia

SIR: While one assumes that McGuffin *et al* (*BJP*, May 1994, **164**, 593–599) set out to be provocative in maximising the genetic component while minimising the environmental component in the aetiology of schizophrenia, their paper cannot pass without comment.

Many of the conclusions they reach are based on concordance rates for schizophrenia of 46% between monozygotic twins. Clever formulae then boost their 'heritability' of schizophrenia to 89%. It is now more than 20 years since Campion & Tucker (1973) pointed out just how different monozygotic twins are, in that, compared with their dizygotic counterparts, they have lower birth weights and their perinatal death rate is three times higher. Reveley & Reveley (1987) noted that twins are no longer regarded as a suitable population on whom to study the genetics of epilepsy or congenital abnormalities and that a concordant pair of schizophrenic twins "could be concordant equally on the basis of birth injury as on the basis of genetic predisposition".

Kendler *et al* (1993) used a preferable genetic method, in that they interviewed first-degree relatives of schizophrenic probands in Ireland. This study confirmed the indisputable genetic predisposition towards schizophrenia. Among the interviewed parents of schizophrenic people, 1.3% suffered from the disorder, as did 9.2% of siblings. Thus, while rates of schizophrenia were several times higher than among controls, schizophrenia among close relatives was relatively rare: in only 16% of families studied was there more than one schizophrenic individual.

Following their hypotheses on monozygotic twin concordance, McGuffin *et al* go on to describe 'stochastic' factors which appear to be genetic phenomena which provide sources of dissimilarity in genetically related individuals. Thus, while genetics explain concordance between monozygotic twins, they can also explain discordance. One should note that these stochastic events occur 'randomly', that is, independently from any environmental variable. At this point, I began to wonder if the paper had been intended for publication on April Fool's Day.

In the paper immediately before that of McGuffin *et al*, Crow (*BJP*, May 1994, **164**, 588–592) urges proponents of environmental aetiologies of schizophrenia to use "straightforward hypotheses and simple analyses" in support of their contentions. Perhaps the geneticists should also follow this advice.

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JOHN M. EAGLES

Ross Clinic
Aberdeen AB9 2ZF

Cognitive–behavioural therapy for schizophrenia

SIR: Kingdon *et al* (*BJP*, May 1994, **164**, 581–587) overlook two problems in recommending cognitive–behavioural therapy (CBT) for schizophrenia. The first relates to the nature and origin