back-pedalled away from acknowledging that the discovery occurred in therapy. One mother of a complainant said she had always known, but had "put it on the back burner." Another, whose counsellors were recorded as pursuing the theory that she had experienced childhood abuse (initially with some difficulty because she was fond of her kindly father), developed her first ideas on a day in between therapy sessions. About 1000 hours of therapy later, she had an extensive belief system, including at least as many occasions of abuse as hours of treatment. A colleague in Quebec has observed the same outcome with rightful acquittals in six other trials. What juries and judges in Canada have just learned, Dr Brewin wishes us to unlearn.

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Genomics

SIR: Neuropsychiatry continues to leave me dumbfounded at its lack of connectedness with human experience. This 'cut-offness' was clearly demonstrated in Farmer & Owen's article (1996). First they sell the latest false dream. They conclude that "there is little doubt" that the genetic aetiology of "common familial disorders including the major psychiatric disorders" will be discovered, and that this knowledge "will radically alter clinical practice." A brief review of the history of psychiatry reveals how many previous false dawns there have been: hormone, pharmacotherapy, cognitive–behavioural therapy, neuroimaging, molecular biochemistry. Farmer & Owen point out the huge ethical dilemmas, not just about the potential misuse but the history of actual misuse of genetic notions of human history, culture and difference. This history and potential should not surprise us. The sense of omnipotence that accompanies such beliefs can never be far from the surface. Genetic science is saying, "I understand what the ideal gene pool should look like, I know therefore what the perfect human being should consist of." What parents, if told their baby had genes that put them at risk for a psychiatric disorder, would not want them changed? Farmer & Owen also suggest that knowledge of the genetics of psychiatric disorders could lead to pre-symptomatic testing. Some of their suggestions are simply laughable. "Advice can be given to individuals with high genetic loading for these disorders regarding exposure to environmental precipitants such as use of street drugs." Other suggestions terrified me. Pre-symptomatic identification of high-risk individuals is suggested. Imagine this: you are told that you have a high risk for schizophrenia. The warning signs have much to do with your thoughts. Suppose now that you get angry at someone, so angry that your thoughts are erratic, destructive and irrational. Will you question whether this is the first signs you were warned about? Worse still, will others now interpret this as a sign that you are developing the illness? (Oh dear, we were warned about this, we'd better get him down to the doctors, he has no insight.) Such a risk-factor culture is also an invitation for a spurious identification, a kind of self-fulfilling prophecy.

FARMER, A. & OWEN, M. J. (1996) Genomics: the next psychiatric revolution? *British Journal of Psychiatry*, **169**, 135–138.

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SIR: Farmer & Owen (1996) gave a subtle description of the various reactions of psychiatrists with respect to the expansion of genetics in recent years. As do most articles addressing the developments in the combined field of psychiatry and genetics, those authors emphasise the detection, comprehension and prevention of the major entities in adult and geriatric psychiatry, and the ethical and psychological problems related to genetic counselling in the context of

predictive genetics. However, no mention is made of the science of behavioural and cognitive phenotypes of genetically determined syndromes. Oddly ignored or unrecognised, this none the less important patient population is of direct concern to the child psychiatrist as well as to the adolescent and adult psychiatrist.

Since the early 1990s a number of syndromes known in clinical genetics have found a molecular explanation. To cite but a few (OMIM, 1996), the genetic alterations discovered and understood include those responsible for syndromes such as fragile X (incidence 1 per 2000 children), VeloCardioFacial/DiGeorge/CATCH 22 (0.5–2.5 per 10 000), Prader–Willi (1 per 25 000), Angelmann (1 per 25 000) and William's (1 per 20 000). All these syndromes, the physical phenotype (dysmorphism) of which may be inconspicuous, also express a characteristic psychiatric phenotype. Presently, it is possible to establish genotype–psychiatric phenotype correlations. At the psychiatric level such investigations lead to knowledge which, in turn, allows for a more targeted guidance or psychoeducational patient approach. Furthermore, in a medium-term perspective, such research enables the development of study models for future investigations of cohorts of children carrying susceptibility genes, notably for schizophrenia, bipolar disorder or obsessive–compulsive disorder.

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Depression and the safety of antidepressants

SIR: The Defeat Depression Campaign might be expected to improve the detection of depression. However, if as a consequence potentially suicidal patients receive older, potentially more toxic tricyclic drugs rather than newer, safer antidepressants, then the risk of life-threatening or fatal overdose may reduce the likelihood of a successful treatment outcome.

Basic details of all deliberate self-harm (DSH) episodes reporting to Derby Royal Infirmary Casualty Department have been recorded on a database from 1990. A retrospective, case-note survey was conducted of deliberate antidepressant overdoses registered at the same department from 1 January 1995 to 31 December 1995. There were 179 self-poisonings with either an older tricyclic, a selective serotonin reuptake inhibitor (SSRI), or a 'novel', non-SSRI (either trazodone or lofepramine). Lofepramine or trazodone were equally likely to be used by those with or without a history of DSH (13 and 12 episodes, respectively). There was no significant differential association between overdose by older tricyclic or SSRI and a record of previous DSH (49 tricyclic and 41 SSRI overdoses in those without a history of DSH; 30 tricyclic and 34 SSRI overdoses in those with such a history, $\chi^2=0.858$, $P>0.1$). Of course, some overdoses may have involved medication not currently prescribed to the patient.

There appears to be considerable scope to promote the prescribing of the newer, less toxic antidepressants among those at risk of repeated self-poisoning, if depression is to be treated effectively but without undue risk.

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Incidence of mental disorder

SIR: When reporting on the incidence of psychiatric disorders in Finland, Lehtinen *et al* (1966) indicate that information from other studies is limited and refer for comparison only to the Lundby Study in Sweden (Hagnell *et al*, 1990) and the Epidemiologic Catchment Area (ECA) Program in the USA (Eaton *et al*, 1989). Findings from at least two other studies would have filled out the picture of available evidence and strengthened their conclusions. One of these is a study of women in Scotland (Surtees *et al*, 1986). The other is an investigation my colleagues and I have carried out in Atlantic Canada, the Stirling County Study (Murphy *et al*, 1988).

Lehtinen *et al* (1996) conclude that "there are huge differences" in results about incidence. Average annual incidence of all types of mental disorders grouped together was 15 per 1000 in the 16-year Finnish study, 18 per 1000 in the 25-year Swedish study, but nearly 100 per 1000 in the one-year USA study.

Depression is used in the following to illustrate similarities and differences when the Canadian and Scottish studies are also brought to attention. The Stirling County Study is similar to the Finnish study in the size of the population investigated and the use of a 16-year follow-up period. Average