

have, as appropriate, also included other categories of external cause deaths such as accidental poisonings and burns (the latter being of particular relevance for suicides among Asian women). In both these papers, for completeness and consistency cross-checks, we examined suicide verdict data as well. Although extending the definition significantly increases the numbers of suicides, it is important to note that patterns of relative suicide risk across ethnic groups are remarkably consistent in all four of our papers covering national data for the 1970s, 80s and 90s – in particular, the high risk in young Asian women, and the low risk in Asian men and African–Caribbeans.

The caveats associated with mortality analyses using country of birth as a proxy for ethnicity are acknowledged but unavoidable until ethnic data become available. Until then, these large national data sets are an essential complement to local studies such as that of Neeleman *et al.* As suicide is a relatively rare event, the former provide a much larger database for robust statistical analysis, while the latter facilitate in-depth investigation not otherwise possible. It is noteworthy that the pattern of high/low suicide risk (i.e. direction of differential risk) across ethnic groups is in fact similar in both Neeleman *et al.*'s and our approaches.

**Department of Health (1997)** *Public Health Common Data Set 1996* (including Health of the Nation indicators). Guildford: National Institute of Epidemiology, University of Surrey.

**Neeleman, J., Mak, V. & Wessely, S. (1997)** Suicide by age, ethnic group, coroners' verdicts and country of birth. A three-year survey in inner London. *British Journal of Psychiatry*, **171**, 463–467.

**Raleigh, V. S. (1996)** Suicide patterns and trends in people of Indian subcontinent and Caribbean origin in England and Wales. *Ethnicity and Health*, **1**, 55–63.

— & **Balarajan, R. (1992)** Suicide and self-burning among Indians and West Indians in England and Wales. *British Journal of Psychiatry*, **161**, 365–368.

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### Opportunities for psychiatry from genetic findings – some concerns

**Sir:** Genetic aspects of psychiatric disorders have been discussed, sometimes quite emotionally, ever since the re-discovery of the Mendelian laws around 1900. The eugenic programmes pursued in Germany during the Nazi regime, but also in other European

countries and in the USA, led to deeply rooted concerns about possible misuse of genetic knowledge. The advent of molecular genetics during the past decade has opened up a new avenue of perspectives that has reinforced the ongoing debate. We agree with the opinion expressed in the recent review by Rutter & Plomin (1997) that the public discussion is sometimes blurred by misconceptions and false hopes associated with genetic findings. However, we are less optimistic than they are that precise genetic knowledge might prevent its abuse. Back in the 1920s population data gathered by Ernst Rüdin, the major representative of eugenic psychiatry in Germany, suggested that affective and schizophrenic disorders involve multiple recessive genes. Rüdin himself based on these findings favoured sterilisation programmes, whereas his co-workers Hans Luxenburger and Bruno Schulz correctly argued that sterilisation of phenotypes would not be effective in removing these disorders from the population; finally, the German eugenic programmes included about 100 000 people with schizophrenia (Weber, 1993).

Hence, it is not calming to read in the paper by Rutter & Plomin (1997) that “susceptibility genes for mental disorders should not provide the basis for a *major* [emphasis added] expansion of the grounds for termination of pregnancy”. The term *major* might well be subject to interpretation in the future. In addition, Rutter & Plomin (1997) chose questionable examples to support their claim that an abuse of genetic knowledge is unlikely. People with Down's syndrome, for example, would be very good targets for eugenic programmes, not because they have inherited their disease but because prenatal diagnosis enables their selective abortion. Moreover, we are not as confident as the authors that “it is most unlikely to be ethically acceptable to terminate a pregnancy on the basis of a disorder that may leave the person functioning well for much of their life” in the light of the estimated 1 000 000 abortions and infanticides performed in India between 1981 and 1991 due to a genetic variant called female gender (Dasgupta & Bhat, 1997).

Although Rutter & Plomin (1997) did an excellent job of describing misconceptions about genetic findings, they may themselves have a misconception about the relationship between science and society. Once science has made its achievements available to society the use or abuse of these

achievements depends on interpretation that varies over time and is entirely independent of scientific reasoning. Therefore, abuse of genetic knowledge will not be prevented by eradication of scientific misconceptions, but by clear-cut and generally accepted ethical guidelines that have yet to be established.

**Dasgupta, M. & Bhat, P. N. M. (1997)** Fertility decline and increased manifestation of sex bias in India. *Population Studies*, **51**, 307–315.

**Rutter, M. & Plomin, R. (1997)** Opportunities for psychiatry from genetic findings. *British Journal of Psychiatry*, **171**, 209–219.

**Weber, M. M. (1993)** *Ernst Rüdin: Eine Kritische Biographie*. Berlin: Springer.

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**Authors' reply:** Drs Pollmächer and Weber note that the prevention of abuse of genetic knowledge requires the combination of good science and careful detailed consideration of the ethical issues. We agree very strongly and, indeed, have argued for the need to take discussion of the ethical issues further forward (Plomin & Rutter, 1998).

**Plomin, R. & Rutter, M. (1998)** Child development, molecular genetics, and what to do with genes once they are found. *Child Development*, in press.

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### Fluoxetine–terfenadine and sexual dysfunction

**Sir:** We report a case of recovery of orgasm after terfenadine ingestion in a patient taking the selective serotonin reuptake inhibitor antidepressant, fluoxetine.

A 55-year-old man with a 13-year history of depressive symptoms during the winter months meeting DSM–IV criteria for seasonal affective disorder (American Psychiatric Association, 1994) had been taking fluoxetine every winter for six years. Each year since commencement of the treatment with the selective serotonin reuptake inhibitor antidepressant he had experienced anorgasmia which had recovered spontaneously on discontinuation of the drug. During the last episode, unlike previous years, he continued to take the antidepressant well into the summer months. During this period