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.

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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

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he took terfenadine 50 mg once a day for hay fever concurrently with fluoxetine 20 mg once a day and experienced orgasms on several occasions. He also reported anergia and mild drowsiness while taking this combination of drugs. Despite remission of his hay fever symptoms the patient chose to remain on terfenadine in order to counterbalance the unwanted sexual side-effects of fluoxetine.

Fluoxetine has been reported to cause sexual dysfunction. The cause of this is unclear but may be related to stimulation of the serotonergic system (Aizenberg *et al*, 1995). A number of adjunctive pharmacological strategies including yohimbine, cyproheptadine, bethanechol, amantidine, and bupropion have been employed previously for the treatment of this dysfunction (Labette & Pollack, 1994). There are no published reports of restoration of normal sexual function with the use of terfenadine. Terfenadine is an antihistamine and has no reported effect on serotonergic function. Thus, our patient's recovery of sexual function appears to be due to the peripheral action of terfenadine. Terfenadine and its active metabolite fexofenadine do not cross the blood–brain barrier. Thus, an antihistaminic effect may be important in resolution of serotonergic-induced sexual dysfunction.

Fluoxetine inhibits cytochrome P450 (Nemeroff *et al*, 1996) which metabolises terfenadine. This causes an elevation in plasma levels of terfenadine when used in conjunction with fluoxetine. Cardiac toxicity has been observed in patients taking fluoxetine and terfenadine concurrently (Marchiando *et al*, 1995). Therefore, we would recommend caution in the treatment of fluoxetine-induced sexual dysfunction with terfenadine.

Aizenberg, D., Zemishlany, Z. & Weizman, A. (1995) Cyproheptadine treatment of sexual dysfunction induced by SSRI. *Clinical Neuropharmacology*, **18**, 320–324.

American Psychiatric Association (1994) *Diagnostic and Statistical Manual of Mental Disorders* (4th edn) (DSM–IV). Washington, DC: APA.

Labatte, L. A. & Pollack, M. H. (1994) Treatment of fluoxetine induced sexual dysfunction with bupropion: a case report. *Annals of Clinical Psychiatry*, **6**, 13–15.

Marchiando, R. J., Cook, M. D. & Jue, S. G. (1995) Probable terfenadine–fluoxetine associated cardiac toxicity (letter). *Annals of Pharmacotherapy*, **29**, 937–938.

Nemeroff, C. B., DeVane, C. L. & Pollock, B. G. (1996) Newer antidepressants and the cytochrome P450 system. *American Journal of Psychiatry*, **153**, 311–320.

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Data on the Mental Health Act

Sir: We have been commissioned by the Department of Health to perform a systematic review of all data pertaining to the Mental Health Act 1983. In order to avoid publication bias, we invite any readers who have unpublished data (including audits into the use of the Act) to contact us so their data can be included in the final report. Readers with such data should contact Ms Wall at the address below, or electronically on: s.wall@iop.bpmf.ac.uk.

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Complementary medicine discussion group

Sir: I wish to contact Members and Fellows with a special knowledge of complementary (or alternative) medicine who would be interested in forming a discussion group on aspects of this topic related to the education and guidance of psychiatrists and general practitioners.

Recent surveys in several countries that possess extensive modern health services show that complementary treatments are used by as many as a third of the adult population more than once, or even regularly. In addition, estimates suggest that the total amount of money spent on complementary treatments is of the same order of magnitude as that spent on orthodox medicines.

The large size of this industry means that many patients seen by doctors of all types must also be using some form of complementary treatment, and this applies particularly to people with long-standing illnesses who are often being cared for by psychiatrists.

The initial purpose of a discussion group would be to try to identify some reading matter which would help psychiatrists to offer some useful guidance to patients who ask them about complementary treatments, or who turn out to be using both orthodox and complementary treatments at the same time. At the moment very few medical schools or postgraduate courses in psychiatry even mention this subject in the programmes offered to trainees. Many psychiatrists might be glad of some guidance, so long as it is seen to be on the cautious side.

It needs to be made clear from the outset that the purposes of such a discussion group would include neither the recommendation nor the rejection of complementary treatments as a whole, since the term as used at present covers a very wide variety of different treatments and procedures. Depending on the circumstances, some are potentially useful, but others are likely to be useless or even harmful.

I should be interested to hear from Members and Fellows who already have some knowledge of the topic and who are interested in a cautious but constructive approach with the first objectives as outlined above.

In view of the potentially controversial nature of this topic and the rather definite viewpoints that are likely to be encountered, I am taking this first step as an individual rather than as a member of a Department of Psychiatry or of a Faculty of the Royal College. If the early stages go well, it is just possible that any discussion group formed might be regarded as within the Faculty of General and Community Psychiatry of the College, but at this stage this is only a conjecture.

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