

# Remedies for brain disorders

SIR — The dismay of several distinguished scientists<sup>1</sup> at reading your leading article sceptical of the applications of our new knowledge to human disease<sup>2</sup> is understandable. *Nature* is, however, essentially correct in saying that we are still quite far from appropriate and effective solutions to the problems created by various severe neurological and mental illnesses. At best, we are now at the end of the beginning, thanks to the development of sophisticated tools that may be useful for the understanding of disease processes and eventually for the development of better remedies. We should not repeat the costly mistakes made in the 'war on cancer', when some important but limited successes led to a premature announcement of the beginning of 'victory'.

In the case of the major psychoses, there is no scientific agreement whatever on aetiopathogenesis, let alone on some critical nosographic problems. Specifically, the statistical distribution of symptoms and of the varied course of disease in large groups of psychiatric patients does not show the peaks and valleys essential to support the hypothesis of distinct pathological conditions (categorical models). Rather, the data speak of a continuum from 'pure' bipolar disease and psychotic depression to 'pure' schizophrenia (dimensional model)<sup>3</sup>. Yet most hard research of a biomedical kind takes one or other categorical model as a basic assumption; such practices may be unavoidable, but the limitations and biases should be more explicitly declared.

The available therapies serve almost exclusively to control symptoms, undoubtedly an important goal in certain circumstances. But the cost is often high. With the neuroleptic drugs in particular, as well as the high risk of irreversible neurological damage causing tardive dyskiasias, there is growing concern about the contribution that treatment may make to the development of organic dementia<sup>4</sup> and the marked unpleasantness of the drugged stage in a substantial number of the treated patients (neuroleptic dysphoria)<sup>5</sup>. The last phenomenon is clearly supported by reports that some neuroleptics evoke strong aversion in animal studies<sup>6</sup>, while some authors support the view that there may be a link between neuroleptic use and the development of a chronic dysphoria-depression syndrome<sup>7</sup>. In fact, abating the unpleasantness of psychiatric therapies is one of the goals proposed by the World Health Organisation<sup>8</sup>.

I gradually gained a painful and sobering awareness of these and other related problems thanks to several years of close

contact with practising neurologists and psychiatrists, clinical psychologists, nurses, social workers and some of their more difficult patients — an exercise that may be strongly recommended to anyone doing basic research in the neural and/or the behavioural sciences. I agree that some of the statements in *Nature*<sup>1</sup> may have been a bit too strong. In our culture, however, this is the only tool we have to hammer an important point; therefore we must appreciate, rather than resent, your decision to break the glass on the alarm.

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1. *Nature* **358**, 184 (1992).
2. *Nature* **357**, 348 (1992).
3. Jablensky, A. in *Epidemiological Impact of Psychotropic Drugs* (eds Tognoni, G., Bellantuono, C. & Lader, M.) 71–97 (Elsevier/North-Holland, Amsterdam, 1981).
4. Breggin, P. R. *J. Mind Behav.* **11**, 425–464 (1990).
5. Emerich, D. F. & Sanberg, P. R. *Biol. Psychiat.* **29**, 201–203 (1991).
6. Bignami, G. *Biol. Psychiat.* **30**, 844–845 (1991).
7. Harrow, M., Fichtner, C. G., Grossman, L. S., Yonan, C. A. & Sands, J. *Biol. Psychiat.* **30**, 845–847 (1991).
8. Sartorius, N. *Croat. Med. J.* **33**, 3–8 (1992).

## Genetic influence

SIR — Christopher Anderson (*Nature* **358**, 357; 1992) accurately describes the controversy surrounding the National Institutes of Health (NIH) funding freeze for the forthcoming genetics and crime conference, but says little about the conference itself. As its organizer, I would like to fill that gap. The conference will offer a public forum for examining the direction and impact of research concerning genetic influences on criminal behaviour, and for debating the validity, interpretation and applications of that research. Far from endorsing the assumptions or goals of research programmes in human behavioural genetics, the conference will subject them to close critical scrutiny.

The conference will address many of the questions its opponents are now raising. Do genetic explanations of behaviour undermine or refine environmental explanations? Does genetic research focus on some kinds of crime to the exclusion of others? Will that research divert attention from social causes of crime? How can genetic factors explain socially defined behaviour? What uses will be made by the criminal and juvenile justice systems of the claims of genetic influence and genetic predisposition likely to emerge from current research? And how will those claims affect public perceptions and broader social policy? For those concerned about the

public oversight and policy implications of scientific research, this conference presents an opportunity, not a threat.

According to Anderson, Dr Bernadine Healy, director of NIH, claimed she could not defend the conference against public criticism for want of a "ringing endorsement" by its reviewers. In fact, the conference did receive a ringing endorsement from its reviewers: the study section described the proposal's analysis of the issues as "superb" and the array of speakers as "impressive". It concluded that the conference was "likely to be considered a landmark in the field". Healy's selective contact with members of the study section represents a further subversion of the NIH review process.

I am concerned about the way in which this conference has been perceived by some members of the black and activist communities, and I was consulting the National Center for Human Genome Research to modify the brochure and agenda well before Healy imposed her illegal freeze. Her politically motivated response has hampered our efforts to address important issues about the social impact of behavioural genetic research.

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## Adders multiply

SIR — Madsen *et al.* (*Nature* **355**, 440–441; 1992) ask "Why do female adders copulate so frequently?" Reproductive success in the adder depends strongly upon the substrate, and the abnormally high frequency of copulation that the authors observed may in fact merely reflect the lack of suitable substrate in the Swedish meadowlands where the observations were made. The importance of this variable is related in the following biblical legend.

When Noah's ark landed on Ararat, he ordered the departing animals, in the name of the Lord, to be fruitful and multiply. All his charges were more than happy to comply, save for the pair of adders, who reminded Noah that since they were mere adders, he could not very well expect them to multiply. Not one to take no for an answer, Noah took the adders to a vacant room, placed them on a table, and told them he was locking them in until they obeyed the Lord's command to multiply. When Noah next looked in on them, they had indeed managed to multiply, for he had wisely placed them on a log table.

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