The mother, aged 32 years, had been commenced on sertraline 150 mg daily, increasing to 200 mg within 2 weeks, for a depressive illness. Within 3 months she became pregnant and remained on sertraline 200 mg throughout her pregnancy. She also took lithium and thioridazine for the first 6 weeks of her pregnancy only, unaware at this time of the pregnancy. The pregnancy proceeded without complication. After a normal full term delivery of a healthy boy she continued with sertraline until 3 weeks postpartum, when this was stopped abruptly. She had breastfed since delivery.

The baby, previously feeding and developing well, after one day developed symptoms of agitation, restlessness, poor feeding, constant crying, insomnia and an enhanced startle reaction. These symptoms were intense for approximately a further 48 hours and then began to subside over the next few days. The mother remained well with no adverse symptoms after stopping sertraline.

Although withdrawal symptoms may have been expected from shortly after birth, it is possible that breast milk concentrations were sufficient to prevent the symptoms noted after complete cessation. The half-life of sertraline of around 26 hours may account for the onset of symptoms in the infant about one day later. Unfortunately, we were unable to measure breast milk sertraline concentration. In addition, the manufacturing company, Invicta Pharmaceuticals, have no data regarding breast milk concentrations or the transplacental transfer of sertraline, and no studies have examined these factors. In view of the popularity of the SSRIs we would call for the manufacturing companies to investigate these important parameters.

LOUIE, A. K., LANNON, R. A. & AJARI, L. J. (1994) Withdrawal reaction after sertraline discontinuation (letter). *American Journal of Psychiatry*, **151**, 450–451.

PYKE, R. E. (1995) Paroxetine withdrawal syndrome (letter). *American Journal of Psychiatry*, **152**, 149–150.

SZABADI, E. (1992) Fluvoxamine withdrawal syndrome (letter). *British Journal of Psychiatry*, **160**, 283–284.

L. S. W. KENT
J. D. D. LAIDLAW

Mother and Baby Unit
Queen Elizabeth Psychiatric Hospital
Birmingham B45 2QZ

Behavioural toxicities of antidepressants

SIR: We were surprised to read the editorial by O'Hanlon & Freeman in April's *BJP* (1995). It is essentially a précis of a paper published elsewhere by the same writers, with substantially the same content, references and, indeed, verbatim quotations Authors of contributions to the *BJP* are required to avow that "their (article's) substance has not been published or submitted for publication elsewhere". Perhaps this rule should apply also to editorials.

Readers of the full article, published as a 'review' in the *Journal of Drug Development and Clinical Research* (1995) will also be aware that the work was sponsored by the manufacturer of an antidepressant that has been found to be behaviourally toxic. This information was not supplied in the *BJP* editorial. This is puzzling, as one of the authors has castigated contributors for submitting articles without declaring the contributions of potentially interested parties (Freeman, 1993).

In the future, if editorials are written as the result of commercial sponsorship, then this information should be available to readers. They can then judge for themselves whether there is a potential conflict of interest.

FREEMAN, H. L. (1993) Effective and acceptable treatment for depression (Letter). *British Medical Journal*, **306**, 1126.

— & O'HANLON, J. F. (1995) Acute and subacute effects of antidepressants on performance. *Journal of Drug Development and Clinical Research*, **7**, 7–20.

O'HANLON, J. F. & FREEMAN, H. L. (1995) Categorising the behavioural toxicities of antidepressants. Proposals and requirements. *British Journal of Psychiatry*, **166**, 421–423.

J. S. KERR
D. B. FAIRWEATHER
I. HINDMARCH

HPRU, University of Surrey
Surrey GU7 1UF

AUTHORS' REPLY: The purpose of any editorial is to express the authors' opinions, within a limited space, concerning an issue of important scientific interest. It is entirely different from that of a lengthy review of research, even if the conclusions of that paper also reflect the same opinions. Our major review on antidepressants was read by the Editor, but he invited the editorial instead, on grounds of space. This clearly did not preclude the later submission of the full study to another journal.

Of course, neither our editorial, nor any other in the *British Journal of Psychiatry*, was written with any sort of "sponsorship". To suspect the same implies either naiveté or a conspiratorial outlook on life.

We find it regrettable that Kerr *et al* refer to "an antidepressant that has been found to be behaviourally toxic". Whether the drug they refer to (dothiepin) is behaviourally toxic was not crucial to our conclusions. Our major point in both the review

https://doi.org/10.1192/S0007125000065181 Published online by Cambridge University Press

and the editorial was that such labels should not be so casually applied. In fact, we did not find evidence to support their contention about dothiepin, especially from the studies they themselves have conducted.

It is profoundly disappointing that Kerr *et al* chose to ignore completely the serious scientific and clinical issues raised in our editorial. Instead, they have focused on our motivation; this *ad hominem* tactic reflects no credit on them. Neither does it encourage progress towards identifying true behavioural toxicity and establishing it on a scientific basis.

J. F. O'HANLON
Institute for Human Psychopharmacology
Abstraat 2A, 6211 LS Maastrict
The Netherlands

H. FREEMAN
21 Montagu Square
London W1H 1RE

CORRIGENDA

CROW, T. J. (1995) The Darwinian Approach to the Origins of Psychosis. *BJP*, **167,** 12–25. On page 25, the reference to MacLean & Triune (1973) should read:
MACLEAN, P. D. (1973) *A Triune Concept of Brain and Behaviour*. Toronto: University of Toronto.

SHEPHERD, M. (1995) Two faces of Emil Kraepelin. *BJP*, **167,** 174–183. The picture of Kraepelin on page 174 is from the *American Journal of Psychiatry*, **151,** 423, 1994. Copyright 1994, the American Psychiatric Association. Reprinted by permission.

A HUNDRED YEARS AGO

Society for the Study of Inebriety

At a meeting on July 4th, Dr Norman Kerr in the chair, Dr A. Mansfield Holmes of Denver, U.S.A., gave an interesting account of the records of the Dalldorf Asylum at Berlin. The proportion of cases of hereditary insanity to acquired insanity was from 2 to 1 to 3 to 1 in different periods. Of the former group, 9 per cent of the cases were directly traced to drunken parents; of the latter, 10 per cent. The Director, Dr Rust, placed heredity as the chief cause of insanity, and alcoholism the second. His conclusions were that alcohol excited to acquired insanity and produced hereditary insanity by bringing about organic changes in the ancestor which are transmitted to the children in the form of predisposition. Periodical drinking to excess was more detrimental to the individual parent and habitual to the offspring. Dr Kerr, with reference to the objections urged in the House of Lords as to undue interference with the liberty of the subject and other abuses of a law for the restraint of habitual drunkards, said there would be no difficulty, by definition, ample inspection, power of appeal, etc., in so safeguarding the interests of habitual drunkards as to render any such abuse practically impossible.

British Medical Journal, July 1895, 117.

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Epsom, Surrey

https://doi.org/10.1192/S0007125000065181 Published online by Cambridge University Press