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Genetic basis for transsexualism

SIR: In their brief report on a female monozygotic twin pair discordant for transsexualism (*Journal*, December 1992, **161**, 852–854), Garden & Rothery have drawn the surprisingly sweeping conclusion that "this case ... refutes the notion that there is a simple genetic basis for the disorder". Their evidence shows a much more modest conclusion, namely that in this single case of transsexualism, genetic factors are irrelevant.

Garden & Rothery go on to offer a "psychodynamic hypothesis" to explain this case, namely that "in the father's absence, the mother uses the child as a confidante". As the child in question was a mere five years old when he first noticed that he was really a boy, he was hardly old enough to be his mother's "confidante".

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'Hidden' spending on community services

SIR: As a clinician manager in our mental health directorate, I was interested to read James Raftery's article (*Journal*, November 1992, **161**, 589–593). I work in a district general hospital (DGH) and all the psychiatric in-patient facilities are concentrated there, all the community services reach out from there, and we have had no dependent link upon Hellesdon Hospital in Norwich for eight years. My registrar and I hold six clinics a week in a community hospital 15 miles away, and I and another consultant in general psychiatry hold clinics in nearby general practices. Our community psychiatric nurses (CPNs) have personal attachments to local practices, are hospital-based for coordination, but work from three (hopefully soon five) community resource centres which host weekly multidisciplinary meetings of the community mental health teams.

We are aware that our community facilities are comparatively well developed, so I was surprised when performance indicator figures for the East Anglian region put us in the lowest ranking insofar as money is spent on working in the community.

Being a budget holder now, and since we are attached to a hospital trust, I winced at the information and looked into it. We are not a community unit but provide virtually all the community services with some help from voluntary bodies and local social services. The Health Authority puts money into two of the day centres and into a Drugs and Alcohol Advisory Service. It seems that that is the only part of our budget deemed to be devoted to community work. My salary and those of our CPNs and my consultant colleagues are all designated as hospital costs – not community – although our DGH is not outside the community but very much part of the community it serves. If 80% of my salary was listed as devoted to community work, which is a correct reflection of how my time is spent, this then would be a more accurate performance indicator.

Our beds are not 100% full, and as I transfer patients to CPN care with periodic out-patient reviews, my clinic numbers may fall and the problems of misinformation may expand. There are not many finished clinical episodes (FCEs) because we have an extensive rural network of support for those in need of long-term care.

Hence, I feel that James Raftery, when he writes "Direct spending on mental health services remains largely in-patient based and has not fallen with bed numbers", has failed to unravel the problems accountants and information systems have of keeping track of where the work is really done.

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Professional scepticism towards multiple personality disorder

SIR: The diagnosis of multiple personality disorder (MPD) remains controversial and the correspondence (Fahy, *Journal*, August 1992, **161**, 268–270), in

response to Merskey's authoritative paper (*Journal*, March 1992, **160,** 327–340), testifies to the anger which sceptical reactions to MPD can provoke. This is not good science. The issue of the validity of MPD is not going to be settled by such affect-laden exchanges, nor by unverified assertions about the number of psychopathologists who believe this phenomenon is a discrete, naturally occurring, clinical entity. Fahy suggests that Merskey sought publication in a British journal because most North American psychopathologists do not share his scepticism. However, Paul Chodoff, a North American psychiatrist, equally pre-eminent and respected by British psychopathologists such as Merskey, has written in the *American Journal of Psychiatry* (1987):

> "I have been in the practice of clinical psychiatry for the past 40 years. . . . During this extended period, my experience with multiple personality disorder has consisted of one very doubtful case. Interested by the spate of current reports, I have conducted a very informal poll of my colleagues. Uniformly, they report seeing no instances, or no more than one or two, in their careers up to now. . . . I cannot accept that my colleagues and I have been so singularly lacking in diagnostic acumen as to miss recognising an entity now being reported in the hundreds, even thousands. . . . Iatrogenic influences operating on suggestible patients in the interests of secondary gain (for psychiatrists as well as patients) cannot be excluded as a possible reason for this disorder".

My own more extensive poll, conducted through the correspondence columns of the Bulletins of the Royal College of Psychiatrists and the British Psychological Society in 1988, produced not a single unequivocal case of MPD.

Lal Fernando, a psychiatrist at the Metropolitan General Hospital, Ontario, has written:

> ". . . according to [Ross] we can deduce that 5% of all psychiatric admissions in Britain or South Africa will carry DSM–III–R diagnoses. . . . Considering the fact that the majority of psychiatrists have never seen or diagnosed a case of MPD on both sides of the Atlantic, I find these figures and predictions incredible".

Thigpen himself (1984) reports seeing no further cases of MPD in the 30 years since his seminal treatment of *Eve*.

Although MPD does seem a curiously North American phenomenon, the very favourable review of my book *Multiple Personality: An Exercise in Deception* in the prestigious American journal *Contemporary Psychology* (1991, **36,** 624–625) further testifies to more sympathy for the sceptical position than Fahy cares to admit. The same can be said about the October 1992 workshop organised for the Hospital and Community Psychiatry Institute in Toronto by Dr Richard Warner, Medical Director of the Mental Health Center of Boulder County, Colorado. The workshop was on the "Overdiagnosis and overtreatment of personality disorders" and Dr Warner is in the process of editing a forthcoming book on the iatrogenesis of a variety of disorders in suggestible individuals.

It simply will not do for believers to accuse sceptics of inexperience or clinical shortcomings not only because this is unfortunately *ad hominem* rather than *ad rem*, but also because so many witnesses frankly admit their own lack of experience prior to their first diagnosis of MPD.

In order to persuade the sceptic, believers in MPD frequently resort to reporting the burgeoning numbers of diagnoses. Despite its superficial validity, this position is comparable with arguments for the existence of unidentified flying objects (UFOs) which rest on an increase in their alleged sightings.

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HIV infection, serotonin, and sexual dysfunction

SIR: I read with interest the article by Catalan *et al* (*Journal*, December 1992, **161,** 774–778). I would like to comment on the development of sexual dysfunction during the human immunodeficiency virus (HIV) infection.

Serotonin has been implicated in the control of sexual behaviour, and the administration of the serotonin precursor 5-hydroxytryptophan, in combination with a peripheral decarboxylase inhibitor, has a facilitatory effect on the sexual behaviour of male mice (Svensson *et al*, 1987).

HIV infection significantly reduces serum tryptophan (Werner *et al*, 1988; Larsson *et al*, 1989) and blood serotonin (Larsson *et al*, 1989). This dramatic degradation of the essential amino acid tryptophan results in decreased serotonin biosynthesis and neurotransmission in the brain (for review see Green & Costain, 1981). These changes are thought to be due to the induction of the enzymes indoleamine 2,3-dioxygenase and tryptophan 2,3-dioxygenase by