host of clinical studies which he mixes up (like apples and pears) with the handful of community based studies that exists. One of the few methodologically acceptable community based studies of anorexia nervosa (see Treasure, 1990; Patton & King, 1991) – the one performed in Göteborg, Sweden in the 1980s (Råstam *et al*, 1989) – was excluded from Fombonne's analysis for "obvious reasons". It seems these "obvious reasons" were (1) that the material of the Göteborg study was presented in sufficient detail to allow specific analysis of whether DSM–III or DSM–III–R criteria applied, (2) that partial syndromes – later meeting full DSM–III–R criteria (Gillberg *et al*, 1994) – were included as a separate group in the original study, and (3) that the birth-cohort was followed up for a few years leading to the appearance of new cases. The findings were presented in a way which has made it possible for Fombonne to calculate all sorts of rates needed for a thorough review. He himself complains that several studies have not provided enough information about the diagnostic criteria used, and that few authors have looked at cohorts in a longitudinal fashion, so we had some difficulty understanding what was so "obvious" about the reasons for excluding this study.

The prevalence rate of anorexia nervosa in the community based studies was considerably higher than the median rate calculated by Fombonne. Again, this should not be taken as evidence that there has been an increase in prevalence rate over the years. However, it is essential that conclusions be based on the most reasonable data sets rather than those that, according to the standards set out by the author of a review/meta-analysis, are less than adequate.

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M. RÅSTAM
C. GILLBERG

University of Göteborg
S-41345 Göteborg, Sweden

Periodic psychosis of puberty

SIR: The article by Abe & Ohta (1995) regarding adolescent onset brief periodic psychoses raises a number of important issues which must be clarified before this condition can be so confidently defined. It is not clear from the report whether the cases they describe met criteria (ICD–10 or DSM–IV) for other psychiatric diagnoses. Certainly, if the subjects cross-sectionally met criteria for depression or mania, it should come as no surprise that these disorders would recur.

Without a description of family history, natural course, comorbid symptoms/diagnoses, or other external validators, it is difficult to assign any diagnostic validity to the concept of a "periodic psychosis of puberty". The reported relationship of worsening psychotic symptoms associated with menses is well recognised in adolescent in-patient units in which severely ill teenage girls with mania or depression are treated. This phenomenon does not of itself qualify for a unique diagnostic label.

In ten consecutive years of adolescent in-patient practice, we have only ever seen one teenager (white, male, age 16) with a non-substance induced, non-affective or non-schizophreniform brief psychosis. This is one of about 1000 admissions. Perhaps there is a cultural diversion to this type of presentation in psychiatrically disturbed teenagers that differentiates Canadian from Japanese youth.

ABE, K. & OHTA, M. (1995) Recurrent brief episodes with psychotic features in adolescence: periodic psychosis of puberty revisited. *British Journal of Psychiatry*, **167**, 507–513.

S. P. KUTCHER

Dalhousie University
Halifax, Nova Scotia
Canada

Treatment of PTSD

SIR: Busuttil *et al* (1995) suggest that their case series strongly endorses the use of psychological debriefing (PD) in the treatment of PTSD. However, the 63 hours of "formal work sessions" in a residential setting described in their paper seems excessive and difficult to justify for PTSD sufferers who have not first tried out-patient treatment. Briefer therapies can work. For example, Foa *et al* (1991) described a randomised controlled trial in which PTSD sufferers experienced a marked reduction in symptoms after nine 90 minute exposure therapy sessions at three and a half month follow-up.