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MICHAEL SOYKA

Psychiatric Hospital  
University of Munich  
Nussbaumstrasse 7  
80336 Munich, Germany

### Psychopathology and ‘ecstasy’

SIR: Maguire *et al* (*BJP*, September 1994, **165**, 391–395) have presented an interesting, if small, study comparing psychiatric symptoms in patients who have taken MDMA (‘ecstasy’) with a control group who showed no evidence of drug use. However, having presented useful data they draw conclusions that cannot be sustained by their evidence. The authors appear to believe that the symptoms displayed by the 13 patients had a causal link with the use of MDMA. They report that the symptoms displayed by the eight psychotic patients did not differ from the matched controls. Four of the eight psychotic patients had discontinued MDMA use four or more weeks prior to psychiatric presentation, and one patient had discontinued 26 weeks earlier. There was no clear relationship between MDMA use and the onset of symptoms. Little information is offered as to the course that the patients’ illnesses took, other than in case 8 where it would appear that symptoms continued irrespective of MDMA use.

We suggest that, from the available evidence, it is equally likely that these patients were suffering from one of the major psychoses. While it is conceivable that MDMA use precipitated or coloured the symptoms, a firm causal link is not sustainable. Use of drugs of abuse, including MDMA, has become widespread in the general population and the coincidence of a psychotic breakdown with drug-taking is bound to happen frequently. It would be dangerous to draw the conclusion that these disorders should necessarily be classified or managed any differently to other psychotic disorders.

The Clunis Report (Ritchie *et al*, 1994) describes in detail a case where a history of supposed drug use

was uncritically taken as an explanation for the persistently abnormal mental state of a man suffering from schizophrenia. This contributed, albeit in a minor way, to the inadequate management of a man who went on to kill. The case illustrates the potential dangers of mistaken attribution of psychotic states to drug use. It is important that the published literature should not exaggerate the strength of the evidence for putative causal relationships to the exclusion of other plausible explanations.

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R. G. POOLE  
C. J. BRABBINS

Windsor Mental Health Resource Centre  
Liverpool L8 7LF

### Cognitive therapy

SIR: We would like to bring to your attention a significant omission from Scott’s otherwise excellent article on cognitive-behavioural therapy or CBT (*BJP*, July 1994, **165**, 126–130). While she does justice to its application to mood and anxiety disorders, she makes no mention of its application to eating disorders and bulimia nervosa in particular. This omission is surprising since the evidence supporting this specific application of CBT is at least as strong as in other areas, if not more so.

The rationale for using a cognitively-oriented treatment for bulimia nervosa arises from the prominence of these patients’ extreme concerns about shape and weight and their likely role in the maintenance of the disorder (Wilson & Fairburn, 1993). This application of CBT has been the subject of over 15 randomised controlled trials and their findings support its use (Fairburn *et al*, 1992). Once maintenance of change is taken into account, it is clear that the effects of CBT are superior to those of all other treatments studied, with the possible exception of interpersonal psychotherapy (Fairburn *et al*, 1993a). Between a half and two-thirds of patients make a full and lasting recovery and many of the remainder are much improved.

A detailed manual describing CBT for bulimia nervosa has been available for many years – the latest version was published in 1993 (Fairburn *et al*, 1993b). The treatment has been adapted to suit patients with anorexia nervosa (Garner & Bemis, 1985) but this adaptation has yet to be adequately evaluated. The role of CBT in the

treatment of other eating disorders remains to be studied.

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CHRISTOPHER G. FAIRBURN

Department of Psychiatry  
University of Oxford

G. TERENCE WILSON

Graduate School of Applied and Professional  
Psychology  
Rutgers University, USA

W. STEWART AGRAS

Department of Psychiatry and Behaviour Sciences,  
Stanford University, USA

fore, internal calibration by appropriate comparison groups is an overriding necessity. The simple fact is that cognitive therapy has not as yet demonstrated that it is clearly superior to placebo in a sample that has also been shown to be medication-responsive.

That this design necessity cuts across diagnosis is indicated by Black *et al.* (1993), who assessed the cognitive therapy of panic versus fluvoxamine versus placebo. There have been many reports that cognitive therapy of panic is remarkably successful. However, in this trial (the only one that compares cognitive therapy to both medication and placebo) cognitive therapy was barely distinguishable from placebo, whereas fluvoxamine was markedly superior. This design is capable of cutting through much ambiguity and wishful thinking. Elkin *et al.* is a methodological standard that the field should adhere to.

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DONALD F. KLEIN

Columbia University  
College of Physicians and Surgeons  
Unit 22, 722 West 168th Street  
New York 10032

SIR: Scott, in her article on cognitive therapy (*BJP*, July 1994, **165**, 126–130), expresses surprise that the NIMH treatment study (Elkin *et al.*, 1992) indicates that CBT was barely distinguishable from placebo. She states: “It is hard to reconcile these findings with all the previous data (27 outcome studies) supporting the efficacy of CBT in major depression.”

However, 27 experimentally inadequate studies do not outweigh one well-designed one. Elkin *et al.* (1992) is the only study that made certain that the sample studied was medication-relevant, as shown by the superiority of medication to placebo. Having thus calibrated the sample, the insignificant difference of CBT from placebo achieves trenchant significance.

Studies that do not include both a placebo and a medication arm are irretrievably ambiguous. Unfortunately, our diagnostic rubrics allow for enormous heterogeneity and are only loosely linked to the prediction of therapeutic effect. There-

#### Childhood abuse and psychosis

SIR: In their study on childhood abuse in first-episode psychosis, Greenfield *et al.* (*BJP*, June 1994, **164**, 831–834) found that history of abuse was associated with significantly more dissociative symptoms. Unfortunately, the article does not indicate the extent to which the subjects were aware of the abuse aspect of the study at the time they completed the dissociation questionnaire. Council (1993) found that childhood trauma and dissociation were correlated only when the trauma survey preceded the measurement of dissociation, suggesting that the relationship between these variables may be an artefact of the context within which they are assessed. It should also be noted that the significant correlation between dissociation and trauma has been shown to disappear after statistically controlling for family pathology (Nash *et al.*, 1993).