interviewed in this study did so. Could this unusual feature be in some way related to their being the relatives of depressed probands rather than random community sample cases?

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References

BEBBINGTON, P. E., TENNANT, C. & HURRY, J. (1982) Adversity and the nature of psychiatric disorder in the community. *Journal of Affective Disorders*, **3**, 345–366.

BROWN, G. W. & HARRIS, T. O. (1986) Stressor, vulnerability and depression: a question of replication. *Psychological Medicine*, **16**, 739–744.

SIR: We are grateful to Dr Craig and Professor Murphy for their close reading of our paper and the opportunity to resolve an apparent ambiguity. Their second paragraph is quite correct. In Tables III and IV of our paper, the life events referred to were in the three months before onset for those relatives with an ID level of 5 or greater, and three months before interview for the rest. We hope that this partially relieves their puzzlement referred to in the paragraph, and that we can fully relieve their perplexity by explaining that the same definition of life event was used for both proband and relative in Table III. They should note, however, that we are not making a case that life events have greater impact on probands than their relatives (and we agree that it would be difficult to sustain such an argument on the basis of the data described in our set of papers). The main point is that life events are strongly associated with the onset of depression in a community sample, but not in a sample of first-degree relatives.

The third question relates to the frequency of life events and the comparison we make between the community sample and the first-degree relatives. In our Tables IV and V we use the same definition of life events in both groups. Although we do not provide the results in the paper we did in fact find that threatening life events of any definition were more frequent in relatives than in the community. The apparent discrepancy between the event rate in the community group and that given by Bebbington *et al* (1981) is due to our use of figures which are weighted back to Bebbington's orginal sample in order to achieve an accurate estimate of the population frequency of recent events. This is necessary because of the two-stage sampling strategy employed by Bebbington *et al* (1981).

The final point raised by Dr Craig and Professor Murphy is an interesting one, and the question of why there should be a comparatively small proportion of chronic cases among first-degree relatives has also occurred to us. We are inclined toward a more mundane explanation than the one they offer. It seems likely to us that the dating of onset of very broadly defined depression is an inexact procedure and one where we may have adopted a different definition of time of onset from previous workers who have focused on community samples. We used a Past History Schedule (PHS) in conjunction with the Present State Examination (PSE) (McGuffin *et al*, 1986). The PHS/PSE interviews identify past episodes and define the most severe occurrence if multiple episodes are evident. It may be that this approach more clearly delineates the episodic nature of depression than does a less structured enquiry about past PSE-type symptoms over an extended period.

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References

BEBBINGTON, P., HENRY, J., TENNANT, C., *et al* (1981) Epidemiology of mental disorders in Camberwell. *Psychological Medicine*, **11**, 561–579.

MCGUFFIN, P., KATZ, R. & ALDRICH, J. (1986) Past and present state examination: the assessment of 'lifetime ever' psychopathology. *Psychological Medicine*, **16**, 461–466.

Obsessive-compulsive rituals

SIR: Regarding Katz *et al's* letter from CIBA-GEIGY (*Journal*, December 1988, **153**, 845) about our clomipramine study (*Journal*, April 1988, **152**, 522–534), we can understand that our findings may be unwelcome to them. Their letter contains some confusion and mistakes that need clarification and correction.

In our literature review we wrote "There is no evidence that clomipramine is significantly better than other tricyclic drugs in OCD despite a widely held belief to the contrary." CIBA-GEIGY claim that

clomipramine was 'therapeutically' superior to desipramine in treating OCD; in the trial they cite of Zohar & Insel, of the nine clomipramine patients three had not been randomised, so that group was not properly controlled. Nor do CIBA-GEIGY cite the only other controlled comparison in OCD of clomipramine and desimipramine (Rapoport *et al*, 1980), where the two drugs differed neither from one another, nor from placebo. Our point holds that in no properly randomised controlled study of OCD has clomipramine been *significantly* superior to another tricyclic drug.

It is strange that CIBA-GEIGY refer readers to Kasvikis & Marks (1988) to see that E (therapist-accompanied exposure) was added from week 8. In our original paper we state this clearly in the Abstract, in Fig. 1, and in the Design.

CIBA-GEIGY are wrong in writing "effects beyond week 8 are confounded by virtually asymptotic performance of the clomipramine and placebo groups, and a concomitant change in methods"; the mistake about the asymptote is repeated later in the letter. Firstly, Fig. 2 of our paper shows a clomipramine and the placebo group (Ce_2E and PeE) continuing to improve from weeks 8 to 17 on five of the six measures shown. It is this improvement in the placebo group that makes the drug effect disappear after week 8. Secondly, adding E from week 8 onwards to a clomipramine and the placebo group, far from confounding their comparison, actually strengthened it by testing drug effect while controlling strictly for psychological treatment. It is thus incorrect for CIBA-GEIGY to state "only the initial 8 weeks of this trial offer a relatively unbiased estimate of the therapeutic effect of clomipramine v. placebo". Moreover, there was no significant effect of adding E to self-exposure from week 8 onwards once the levels at week 8 had been controlled by ANCOVA, so this addition is irrelevant in comparing drug with placebo. Given the highly significant superiority of self-exposure over anti-exposure and the limited and transient superiority of clomipramine over placebo, we have to conclude that self-exposure was the most potent therapeutic factor in our trial.

CIBA-GEIGY are correct to note, as we did, that from weeks 0 to 8, clomipramine was superior to placebo, but fail to say this was on only 5 of 14 measures of rituals and with mainly low levels of significance, whereas self-exposure had an effect on fully 11 of those same 14 measures and mainly at a far higher level of significance. They ask, "What would be adequate in the authors' minds to consider a drug v. placebo difference as clinically significant or durable?" The answer is that significance requires an effect on most relevant measures, and durability requires persistence of that effect: we found neither.

The letter is inaccurate in saying that our patients were "preselected to ensure that they would be responsive to behavioural intervention". We included many extremely severe and chronic cases with widely ramifying handicaps whom we knew would be very difficult to treat. Only 5 of 124 patients suitable for the trial were excluded because previous behavioural treatment had failed, and eight more refused such treatment. In contrast, fully 22 patients refused trial entry because they did not wish to take drugs, so our study preselected patients who were relatively compliant in taking medication.

CIBA-GEIGY write that exposure patients "were required to undergo up to 3 hours of self-exposure therapy each day. It is unlikely that any other treatment would have much effect, or for that matter could have much effect, given this magniture of exposure." The authors thus seem to accept our conclusion that self-exposure turned out to be a major therapeutic factor that might swamp any others. Clomipramine did have a mild effect nevertheless, but only briefly and only when self-exposure was added, whereas clomipramine plus three hours of self-antiexposure a day had virtually no effect.

Inclusion of the antiexposure group vitiates the letter's claim that our design was "set up as a comparative study of the adjuvants to exposure". Our design was adopted precisely in order to compare clomipramine without and with exposure, and to examine the effect of adding E to self-exposure. Limitation of resources prevented us from also testing clomipramine with no instructions about exposure ('Do whatever you feel is right for you'). This turns out to be probably unimportant, as OCD cases coming for treatment are usually very chronic (a mean of nine years' duration at presentation), no study has shown benefits from clomipramine or any other drug lasting more than a few months, many authors have noted the frequency of relapse on stopping medication, in the only two controlled studies to have long-term follow-up there was no clomipramine effect at one-and two-year follow-up (Mawson *et al*, 1982; and the present one – Kasvikis & Marks, 1988), and exposure confers benefits usually lasting at least four years.

Our findings agree with others in the literature that clomipramine can have a 'significant' effect. However, merely saying effects were significant is not enough. How significant were they, and for how long in a chronic condition? On both counts clomipramine was unimpressive in our study. Progress will be faster when drug companies and other bodies support more studies with long-term follow-up well

beyond the four- to eight-week designs that are usual. This will probably only happen when the CSM and FDA insist on chronic studies to justify chronic prescription for chronic disorders.

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References

KASVIKIS, I. M. & MARKS, I. M. (1988) Clomipramine and exposure for OCD: 2-year follow-up. *Journal of Anxiety Disorders*, **4**, 291–298.

MAWSON, D., MARKS, I. M. & RAMM, E. (1982) Clomipramine and exposure for chronic obsessive-compulsive rituals: III. Two-year follow-up. *British Journal of Psychiatry*, **140**, 11–18.

RAPOPORT, J., ELKINS, R. & MIKKELSEN, E. (1980) Chlorimipramine in adolescents with OCD. *Psychopharmacology Bulletin*, **16**, 61–63.

Near-death experience

SIR: The article by Roberts & Owen (*Journal*, November 1988, **153**, 607–617) called to mind a recent article by the philosopher Sir Alfred Ayer (1988), entitled "What I saw when I was dead". Sir Alfred's heart evidently stopped beating for four minutes after he choked on a slice of smoked salmon. On recovering he described the experience to a French friend: "Did you know that I was dead? The first time I tried to cross the river I was frustrated, but my second attempt succeeded. It was most extraordinary. My thoughts became persons." He says further, "I was confronted by a red light, exceedingly bright and also very painful even when I turned away from it. I was aware that this light was responsible for the government of the universe. Amongst its ministers were two creatures who had been put in charge of space."

In analysing the experience, Sir Alfred says it "could well have been delusive. A slight indication that it might have been veridical has been supplied by my French friend, or rather by her mother, who also underwent a heart arrest many years ago. When her daughter asked her what it had been like she replied that all she remembered was that she must stay close to the red light."

Sir Alfred's experience corresponds significantly to the description of NDE provided by Greyson (1985), incorporating parts of the 'transcendental component', i.e. encountering guides, coming to a border of no return (in this case the river), and parts of the affective component, i.e. being surrounded by a brilliant, warm (in this case, red) light. His recollection, "my thoughts became people" is reminiscent of the experience that we, as psychiatrists, have with psychotic patients; i.e. there is a correspondence between their thoughts and the verbal productions/forms of their hallucinated objects.

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References

AYER, A. J. (1988) What I saw when I was dead. *National Review*, **40**, 38–40.

GREYSON, B. (1985) A typology of near-death experiences. *American Journal of Psychiatry*, **142**, 967–969.

Teenage depressive stupor

SIR: We read with interest Powell *et al's* report of depressive stupor in a 13-year-old boy (*Journal*, November 1988, **153**, 689–692). The authors' claim that there are no published descriptions of stupor in this age group is, however, incorrect, as case 4 of our series of ten cases of adolescent bipolar psychosis also presented with stupor at the age of 13 years (Hassanyeh & Davison, 1980).

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HASSANYEH, F. & DAVISON, K. (1980) Bipolar affective psychosis with onset before age 16 years. Report of 10 cases. *British Journal of Psychiatry*, **137**, 530–539.

Defining personality disorder

SIR: The validity of personality disorder (PD) as a mental illness has recently been the subject of several articles and letters (Blackburn, 1988; Chaloner, 1988; Cook, 1988; Gunn, 1988; Lewis & Appleby, 1988), with the majority favouring its rejection. While statistical cluster techniques and reliable personality-trait inventories support the existence of fixed deviant personalities, they cannot address the question of whether or not they are illness *per se*, as any such conclusion relies on the prior definition of mental illness. In the introduction to their study on the pejorative implications of the label 'personality