

Statistical design, analysis and correspondence

SIR: A statistician should know better than to become involved in disagreements between psychiatrists, particularly when these involve aspects of statistical design and analysis. But having read some of the statements in recent letters by Welch & Lewis and Marks *et al* (*BJP*, January 1995, 166, 120–122), I thought I should have the courage to take the role of the fool rather than that of the angel and point out some of the more obvious nonsense both in their remarks, and in the analyses reported by Marks *et al* (1994) that prompted their correspondence.

It is often hard to persuade psychiatrists (and others) that statistics is a thriving, constantly evolving body of knowledge. Techniques taught to today's consultant psychiatrist in the dim and distant past are very likely to have been superseded by recent developments. One example of an area that has altered dramatically in the past five years is the analysis of longitudinal studies, particularly when missing data occur. This is not the place to go into detail (two recent references are Everitt, 1995 and Diggle *et al*, 1994), but briefly, powerful and sophisticated modelling techniques are now available (with suitable accompanying software in most cases) that can undertake informative and appropriate analyses of longitudinal data, including dealing with missing observations in the correct fashion. Employing such methods would avoid both the suggestion of Welch & Lewis that using the 'last observation carried forward' approach to replacing missing values is sensible (it is not!), and the claim by Marks *et al* that 'repeated tests of significance . . . had to be done' (they did not and the results from them are extremely likely to mislead!).

Why is it, I often wonder, that psychiatrists are so ready to pontificate on topics statistical, whereas few statisticians write to psychiatric journals claiming that they know the best treatment for depression? I suggest readers regard this question as rhetorical.

DIGGLE, P. J., LIANG, K. & ZEGER, S. L. (1994) *Analysis of Longitudinal Data*. Oxford: Oxford Science Publications.

EVERITT, D. S. (1995) The analysis of repeated measurements: a practical review with examples. *The Statistician*, in press.

MARKS, I. M., CONNOLLY, J., MIYEN, M., *et al* (1994) Home-based versus hospital-based care for people with serious mental illness. *British Journal of Psychiatry*, 165, 179–194.

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Predictors of outcome in the behavioural treatment of OCD

SIR: The attempt by Keijsers *et al* (*BJP*, December 1994, 165, 781–786) to determine which factors predict outcome in the behavioural treatment of obsessive-compulsive disorder (OCD) is welcome, given the paucity of studies in this area. The authors provide a critique of extant work in this field, correctly pointing out the major flaws in many published studies. However, they themselves fail to address two fundamental methodological difficulties.

First, the number of patients in the study ($n=40$) is simply too low to allow robust statistical analysis of such issues as the possible effect of medication on the results. For example, the authors boldly state that, given no significant antidepressant drug \times treatment interaction effect, "there is no reason to suspect that unmedicated patients improved less than medicated ones". Reference to Table 1 of their study, however, shows that only 11 patients were taking antidepressants, resulting in very low statistical power to assess any such interaction effect. Second, we are told that 51 patients were approached to participate in the study, but that only 40 completed the study. No attempt is made to control for bias which might have been introduced by the attrition of 22% of the patient sample.

Despite these limitations, Keijsers *et al* do delineate a number of variables which appear to have some predictive value. Most of the variables considered were "complaint related" items such as duration of symptoms and severity of complaints, and not amenable to alteration. Of the "non-specific treatment variables", only "quality of the therapeutic relationship" (a rather vague construct) would potentially be amenable to change by the therapist. It is thus important to assess which elements of the therapeutic relationship are important in terms of predictive value, and this will be very difficult to do.

It seems reasonable to suggest that research such as this should consider variables which can potentially be altered. In this regard, in a study conducted by my colleagues and me at the Institute of Psychiatry, London (Castle *et al*, 1994), having a co-therapist was (surprisingly to us) the most powerful predictor of outcome in 178 OCD patients treated with behavioural psychotherapy; interestingly, the effect was robust only for women. Again, it will be important to ascertain what it is about a co-therapist that is of benefit. Much further work needs to be done in this area, so that more

patients with this often debilitating condition can be treated.

CASTLE, D. J., DEALE, A., MARKS, I. M., *et al* (1994) Obsessive-compulsive disorder: Prediction of outcome from behavioural psychotherapy. *Acta Psychiatrica Scandinavica*, **89**, 393–398.

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Cognitive therapy for panic

SIR: We are grateful to Drs Clark *et al* (*BJP*, October 1994, **165**, 557–559) for clarifying some points in their paper (*BJP*, June 1994, **164**, 759–769). Their conclusions about cognitive therapy, however, remain problematic; their results can be explained non-cognitively just as well.

The Oxford cognitive therapy group's better outcome may reflect its cognitive therapy less than its getting firmer and more detailed exposure instructions (albeit in a cognitive cloak): attention to (a) stopping safety behaviours, which is a form of exposure; (b) behavioural experiments, which are a form of brief exposure; (c) interoceptive exposure; (d) diaries of behavioural experiments amounting to brief exposure (applied relaxation and imipramine patients were not asked to keep exposure diaries). Such elements constitute a good behavioural analysis and implementation.

The above procedures resemble Bandura's so-called mastery treatment which gave detailed teaching of exposure and reduced phobias. Clark *et al*'s cognitive therapy group improved without being asked to do prolonged exposure. They may have used an ingenious way to do brief effective exposure; this needs testing without cognitive components. A good test would be a comparison of their full cognitive-behavioural package with one containing only their behavioural elements and omitting cognitive elements such as their rationale, identification of misinterpretations of bodily sensations, challenging of evidence, and substitution of more realistic interpretations and restructuring images.

The authors say they did not aim to rule out the possibility that non-cognitive variables might also predict outcome, but rigorous ruling out is vital if cognitive theory is to be credible. Avoidance was not predictive. What about other non-cognitive variables such as Hamilton Anxiety, Beck Anxiety, general tension and anxiety, Beck Depression?

Change of beliefs may reflect general improvement rather than cause it. Moreover, a cognitive theory has to show that change in beliefs precedes improvement in other measures.

The Oxford group's non-severe cases "whose attacks were thought unlikely to be completely eliminated by situational exposure alone" were an easier-to-treat sample. Less severity (avoidance, anxiety, depression, disability, but not panic) predicts more response to various treatments (Basoglu *et al*, 1994a). Exposure to external cues may be unsuitable for the very few panic disorder patients who have neither avoidance nor situational panics, but such cases can respond to interoceptive exposure, which is not only "consistent with a cognitive theory of panic" but also with non-cognitive theories. The exclusion of severely agoraphobic cases remains puzzling. Despite most London-Toronto (LT) cases being severe, they were treated successfully in the clinic; very few were so house-bound that they could not attend.

In most panic disorder patients, panic reduction is a weak yardstick unless disability is overcome too. Patients can become panic-free just by staying home. Unlike avoidance reduction, panic reduction relates little to lessening in work/social disability or global improvement (Basoglu *et al*, 1994b). Panic improved markedly with placebo, not only in the LT study (74% after 8 weeks treatment, when the double-placebo dropout rate was only about 5%) but also in the Upjohn Phase I and II multinational studies. The absence of non-pill groups in those studies does not vitiate this finding. The Oxford argument for spontaneous remission in panic in those studies seems unconvincing for patients who had been ill for a mean of 5–8 years and who with placebo improved more on panic than other ratings.

Clark *et al* also wonder if panic improved with placebo in LT patients because panics increased transiently after stopping drug before trial entry. This idea is disconfirmed by a fresh analysis. At week 0, compared to patients who had had no prior medication, LT patients who stopped drug before week 0 had a similar number of situational and spontaneous panics, anticipatory anxiety, and illness severity. There was thus no overinflation in week 0 scores from recent drug withdrawal. At entry *all* LT patients had chronic panic disorder with agoraphobia by DSM-III criteria, having a mean of 5.2 major panics a week.

It remains unclear why the Oxford study used contrast groups with such a weak form of exposure (no exposure in weeks 1–4, no asking patients to stop safety signals and so stop avoiding, no note of