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Psychological treatments for hypochondriasis

Sir: Clark *et al*'s (1998) loose use of terms is misleading. Their "cognitive" therapy was in fact cognitive–behavioural therapy (CBT) with (p. 219) "A mixture of cognitive and behavioural techniques" including "behavioural experiments" by imaginal exposure ("inducing symptoms by deliberate body focusing or dwelling on fearful thoughts"), live exposure ("increasing engagement in activities that were avoided because of illness beliefs (for example, exercise)"), and "response prevention for repeated bodily checking and prevention of reassurance seeking . . . others who were normally involved in the provision of repeated reassurance were included in the response prevention programme and were given instructions in how to deal correctly with any further requests for reassurance". Homework included exposure and response prevention (ERP).

In contrast, "behavioural stress management" included only weak exposure *without* mention of 'cognitive' therapy's strong behavioural components of: ERP in the first few sessions; behavioural experiments and response prevention by patients and others to deal with checking and reassurance seeking; and exposure homework. It did include anti-exposure reassurance ("remind patients that previous physical investigations had proved negative and their doctor was convinced they did not have a serious illness"). The procedure is best termed stress management with a small behavioural component late in therapy.

The design's having more behavioural (ERP) experiments in the cognitive (80%) than in the behavioural therapy (0%) sessions shows in Table 1. The Table does not mention exposure homework, but the description (see above) suggests this too was advised more in the cognitive than the behavioural sessions. Because the authors' cognitive therapy was also more behavioural (had more ERP) than their behavioural treatment, their design cannot support the claim that cognitive therapy was a specific treatment, unlike behavioural stress management. They compared CBT (cognitive restructuring plus ERP) on the one hand with stress management including limited exposure and additional methods on the other. The early superiority of their CBT (which was not sustained) could be explained by its greater use of ERP than the stress management protocol which introduced exposure later in treatment.

It is possible that cognitive therapy alone, without behavioural experiments and ERP, may have produced similar improvement, but the study has no such contrast group. What was specific about a form of cognitive therapy that included strong behavioural methods in a design which had no treatment group that omitted both cognitive and behavioural components?

Clark *et al*'s design is out of date, as controlled studies have found in several anxiety disorders, including hypochondriasis, that exposure alone and cognitive therapy alone were each therapeutic in their own right. In depressive disorders too, purely behavioural (without cognitive) methods were just as helpful. None of these controlled studies is cited.

Clark *et al*'s preoccupation with cognitive effects leads them to ignore recent findings that neither cognitive nor behavioural therapy is crucial for improvement. Sufficient yes, necessary no. One or the other can do the trick, and each may be an unwitting way of using other effective ingredient(s) that are as yet unidentified. Future studies are more likely to advance knowledge if they separate cognitive from behavioural components and test whether they work by similar or different mechanisms or in ways that are neither cognitive nor behavioural.

As an aside, on Fig. 1's measure none of the follow-up differences between the two treatments was significant.

Clark, D. M., Salkovskis, P. M., Hackman, A., *et al* (1998) Two psychological treatments for hypochondriasis. A randomised controlled trial. *British Journal of Psychiatry*, **173**, 218–225.

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Authors' reply: Our controlled trial demonstrated that two new treatments, developed by our group, produce substantial improvements in hypochondriasis. Professor Marks quibbles with the labels chosen for the treatments and our use of the term "specific treatment effect". Personally, we are more concerned with effectiveness than with labels. However, our terminology was not inappropriate.

The term 'cognitive therapy' was introduced over 30 years ago and from the start denoted a cognitive theory-based treatment involving verbal disputation *and* behavioural procedures, both of which had the *explicit* aim of changing patients' dysfunctional beliefs (see Beck, 1970). Our cognitive therapy for hypochondriasis has these characteristics. Some people prefer the term cognitive–behavioural therapy (CBT). We chose cognitive therapy not because we think our behavioural procedures are unimportant: quite the contrary. Instead, it was because the term CBT is used in a variety of different, and potentially confusing, senses. For some people it equals cognitive therapy as defined above. For others, such as Marks, it includes a mixture of procedures that are each given with different rationales, viz. an anxiety habituation rationale for exposure and a belief change rationale for verbal disputation.

The term 'specific treatment effect' also has a long-standing meaning, which we adhered to. At least since Gelder *et al*'s seminal paper (1973) on specific and non-specific effects in psychotherapy, the term has been used to denote a demonstration that the effects of a therapy cannot be accounted for simply by a series of specified procedures that would be present in any well-conducted psychological treatment, irrespective of orientation. Our cognitive therapy programme clearly passed this test as it was superior to behavioural stress management on 7 out of 10 hypochondriasis measures at post-treatment, despite behavioural stress management involving the same repeated assessments, being administered by the same therapists for the same amount of time, involving systematic out-of-session homework, and being rated as equally credible by patients. This demonstration of specificity seems rather more convincing than Marks' own claims for specificity in his recent trial of treatments for post-traumatic stress disorder (PTSD) (Marks *et al*, 1998). In that trial, exposure was only superior to the control treatment (relaxation) on three out of nine primary PTSD measures and there was no evidence that patients

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thought relaxation was as credible as exposure.

With respect to Marks' comments about likely active ingredients in our cognitive therapy programme, it would appear that he has not read the article carefully. We make no claims about the relative potency of cognitive and behavioural procedures and explicitly state (p. 224) that the study design did not allow us to determine which of the many cognitive and behavioural procedures that distinguished cognitive therapy from behavioural stress management were responsible for the former's superiority. Marks also appears not to have noticed that assessment of the session tapes detected "no instances of either in-session or homework exposure to avoided illness-related situations (hospitals, television programmes, etc.)" and that "reassurance . . . was not often detected and the two treatments did not differ" in this respect (p. 220). Finally, we did not cite any controlled studies demonstrating that exposure alone or verbal disputation alone are specific treatments for hypochondriasis because none exists. After acceptance of our paper, data that these procedures are better than no treatment were produced by a Dutch group but there is no evidence that they have a specific effect (i.e. are better than an attention placebo condition).

Like Marks we are very interested in the question of which cognitive/cognitive–behavioural procedures are most effective. However, we differ in our views on the best ways to answer this question. Marks *et al* (1998) favour large-scale component analysis treatment trials. Because of their failure to deal with dose response issues, and other logical and variance control problems inherent in their design, we consider such trials insensitive instruments for detecting additive effects of cognitive and behavioural procedures. For this reason, we favour much tighter, single-session experiments (see Salkovskis *et al*, 1999, for a successful example of this methodology).

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Antipsychotic polypharmacy and early death

Sir: The article by Waddington *et al* (1998) is an example of careful audit over a prolonged period. For this, the authors are to be thanked, especially since such 'captive' populations and hence the possibility of such studies, is fast disappearing. However, critical comments are necessary. First, the major conclusions are presented in terms of statistical significance and one of these, the finding that 'polypharmacy' is a contributory cause of early death, is so alarming that it may well have been taken up by a vituperative press seeking to vilify psychiatry and all its works. However, the statistical information presented is so weak as not to be regarded as having significance. It is a curious and contradictory observation that both polypharmacy and withdrawal from medication both contributed, in the same direction, to demise. The article would have been more helpful if actual numbers, or at least median values, had been presented. Means and standard deviations, even with the addition of ranges, provide no clear information. For instance, apparently medication had been stopped in some patients but in how many and for what reason is not stated. I should like to believe that the series of investigations to which I contributed (Andrews *et al*, 1976), which demonstrated that continued medication in such a chronic population was of no value, had had some influence. Then the absence of information regarding clozapine (with its recognised lethal potential and for which careful monitoring is *de rigueur*), is a defect of the study; it is possible that earlier demise occurred in just such a context. Finally, it is a pity that all causes of death were lumped together; the opportunity has been missed to contribute to knowledge as to whether or not high-dose antipsychotic medication is verified as a cause of cardiac disease and death.

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Authors' reply: We appreciate the controversial nature and possible unpalatability of some of the associations that we report, but are disinclined to accept a number of Dr Snaith's strictures. Regarding statistical issues, our major findings are *not* presented in terms of significances but, rather, in terms of relative risks with 95% confidence intervals, in accordance with the 'statistics' section of the *Journal*'s 'Instructions to Authors'. We do not find that antipsychotic polypharmacy is a *contributory cause* of early death; that is one of several interpretations of our finding of an *association* between antipsychotic polypharmacy and early death. Our statistical approach and data presentation are conventional (Altman & Bland, 1998), with Cox proportional hazards modelling accepted as a method of choice for examining a set of variables for independent predictors of survival. There is no contradiction in both antipsychotic polypharmacy and time since final withdrawal of antipsychotics predicting reduced survival. As stated in our article, the index of polypharmacy is the maximum number of antipsychotics given concurrently, to cover instances where this occurred prior to the index evaluation such as when antipsychotics had been withdrawn; both are identified by Cox modelling as independent predictors of reduced survival (i.e. each variable is associated with reduced survival after controlling for the influence of the other). It was not always straightforward to specify on an individual basis the reason(s) for antipsychotic withdrawal (of which there were 20 instances); we accept Dr Snaith's point that a lack of perceived value in continuing antipsychotic treatment may have contributed to its withdrawal in some patients, in addition to our own speculation in terms of terminal physical illness replacing psychiatric disorder as the primary focus of medical care.

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