

Finally, the study did not assess stress, which may constitute a further, important uncontrolled bias. A recent cohort study of stress and the common cold concluded that all four dimensions of stress investigated — stressful life events, negative affects, positive affects and perceived stress — were significantly related to occurrence of the common cold.<sup>7</sup> Stress may also have significantly affected symptom severity and participants' perception of their symptoms.

Certainly, the trend observed in the placebo group of shorter duration of some symptoms and lower mean severity could have been due to less severe symptom history, compounded by a lower degree of overall stress.

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**IN REPLY:** Precisely because of the temporal variation in symptom severity described by Vitetta and colleagues, we judged that medical professionals are not as well able, in a variably timed interview, to quantify patients' cold symptoms as the patients themselves can do on a continuing basis. Therefore, we consider that our study<sup>1</sup> would have been no more valid if the detailed symptom severity cards had been supplemented by one or more physical examinations. In that respect, we are in good company with others who have studied the common cold over many years.<sup>2</sup>

We agree that double-blind randomisation does not necessarily distribute all relevant variables equally. That is why, in Box 2 of our study report, we presented four variables — age, sex, mean number of colds in the previous year, and mean number of days unwell with colds in the previous year.<sup>1</sup> The likelihood that stress,

smoking and alcohol status would have been sufficiently maldistributed in this large group to mask a significantly beneficial effect in even one of the three groups which received high-dose vitamin C seems vanishingly small. Nevertheless, we acknowledge that the study would have been stronger if we could have reported the distribution of these three potential confounders.

We contest the view of Vitetta and colleagues that the evidence from randomised controlled trials of vitamin C in treating the common cold conflicts significantly (see Box 1 of our article<sup>1</sup>). The overview finding — that mega-doses of vitamin C for prophylaxis produce a relatively trivial reduction in cold severity but no reduction in incidence<sup>3</sup> — was the stimulus for our own study. No community studies of this issue have been flawless, but the mounting collective evidence suggests that we should look elsewhere for a cold panacea.

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## Evolving evidence and continuing uncertainties for eating disorders

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**TO THE EDITOR:** We are writing in response to the editorial of Ben-Tovim et al.<sup>1</sup> Although we agree that more research into treatment efficacy in eating disorders is needed, we believe that the study to which reference is made<sup>2</sup> is seriously flawed. The study should not be presumed to provide evidence about the effect of treatment on outcome, particularly as the majority of patients studied received no treatment. The high death rate (3/95 [3.2%] among patients with anorexia nervosa and 2/37 [5.4%] among patients with "eating disorders not otherwise specified") in such mildly ill patients (few of whom would have warranted hospitalisation on the basis of their weight) approximates that of seriously

emaciated patients in longer-term studies of treatment outcome<sup>3,4</sup> and could more properly be said to illustrate the results of having no treatment or inadequate treatment.

Exactly what constituted specialised treatment is never actually described in the original article,<sup>2</sup> in which "extended inpatient treatment" is defined as treatment lasting more than two weeks and "extended outpatient treatment" as three or more visits. Thus, the so-called "resource intensive treatment" the authors refer to would not necessarily represent even adequate management of these conditions.

In our own 6–10-year outcome study<sup>5,6</sup> cited by the authors, 61 emaciated patients with anorexia nervosa received, on average, 11 weeks of inpatient treatment consisting of nutritional rehabilitation and psychotherapy. Only one patient died (of suicide) and, of the patients fully assessed, 41/50 (82%) had a good or intermediate outcome. The degree of weight restoration achieved by the end of treatment correlated with the degree of osteoporosis 10 years later.<sup>7</sup>

In other studies, duration of illness and early intervention have been shown to significantly influence outcome.<sup>4</sup> This contrasts with the findings of Ben-Tovim et al,<sup>2</sup> which may have been skewed by an unusual level of chronicity in the study group. A recent study of 69 patients with eating disorders treated in our own multidisciplinary program showed that, on 12–18-month follow-up, 48/69 (70%) had improved and 34/69 (49%) no longer had an eating disorder diagnosis. Mean levels of all but one of the major behavioural and psychological features rated by the EEE-C (Eating and Exercise Examination by Computer) instrument<sup>8</sup> were significantly reduced.

The advice given by Ben-Tovim and colleagues to the parents of the hypothetical 15-year-old girl with anorexia nervosa is regrettably nihilistic and, if based on their *Lancet* study,<sup>2</sup> not founded on sound or generalisable evidence. Parents should be referred to a program for which good outcomes have been demonstrated, treatment accords with published guidelines, the clinicians are suitably experienced, and in which early intervention is the aim.<sup>4</sup>

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**IN REPLY:** The commitment of Russell and Abraham to their own program has distracted them from accurate reporting and sound epidemiological principles. They say that the majority of patients that we studied received no treatment. Not so. We clearly stated that only 34 of the 220 patients studied received no treatment.<sup>1</sup> They then draw a range of inferences from the fact that "3/95 (3.2%)" patients with anorexia nervosa died. In fact, only 2 of 95 patients with anorexia nervosa died as a consequence of that disorder during the five years of our study. At 2.1%, this is similar to the crude death rate of 1/61 (1.6%) that they describe in their own study.

However, it is only acceptable to use a rare outcome as a measure of the efficacy of a treatment program if the clinical characteristics that put patients at risk for such an outcome are known and accounted for. We do not know the specific factors that put people at particular risk of dying from anorexia nervosa. Without such knowledge, small differences in crude death rates can not of themselves inform us whether treatment programs diminish or accentuate such risks. Unfortunately, there are no other studies against which to compare the outcomes of the patients with "eating disorders not otherwise specified" in our study. We have dealt with issues such as the representative nature of our study elsewhere.<sup>2</sup> I stand by our work and the conclusions we draw from it.

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