Authors' reply: As Mushtaq & Minn-Din correctly point out, Bijl *et al*[1] did not find associations between prevalence rates of any psychiatric disorder (including anxiety disorders) in children and anxiety-related symptoms in parents. However, one must be aware of several methodological differences to our investigation: the results cited by Mushtaq & Minn-Din are based on 12-month prevalence rates and multivariate logistic regression analysis additionally controlling for childhood adversities and socio-demographic characteristics. We would like to clarify that the results of the Bijl *et al* paper are much more comparable with our study and that the results we are actually referring to are those based on life-time prevalence rates of psychiatric disorders in children without controlling for childhood adversities and reported separately for the various offspring disorders. Here, Bijl *et al* clearly report associations between anxiety in parents and children.

In addition, it is true that the adult children in the Bijl *et al* study were considerably older (18–65 years) than the offspring in our study (17–21 years at follow-up). We would like to add that there are other substantial ways in which the studies differ; for example, our use of assessment via direct interviews *v*. family-history information.[1] Nevertheless, we do not see why our claim that we confirm and extend the Bijl *et al* study should be problematic, especially when taking into account the low median for age at onset of anxiety disorders.[2]

We would also like to point out that both studies were community-based so that the use of the term 'patients' by Mushtaq & Minn-Din is slightly misleading.

1 Bijl RV, Cuijpers P, Smit F. Psychiatric disorders in adult children of parents with a history of psychopathology. *Soc Psychiatry Psychiatr Epidemiol* 2002; **37**: 7–12.

2 Cross-national comparisons of the prevalences and correlates of mental disorders. WHO International Consortium in Psychiatric Epidemiology. *Bull World Health Organ* 2000; **78**: 413–26.

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Cognitive–behavioural therapy for self-harm

We read Slee *et al*'s[1] article with interest and concern. We believe there are major biases towards the treatment arm of this study which may invalidate their conclusions. Furthermore, our experience of working in a liaison psychiatry team receiving more than 1500 self-harm referrals a year leads us to question the applicability of the intervention given the characteristics of the study group.

At the outset, there are more participants in the treatment-as-usual (TAU) group shown to be depressed and this difference reaches statistical significance from the first follow-up at 3 months and gradually grows with each follow-up. Hence, it can be argued that the difference in outcome is a mere difference in depression and anxiety, which we know respond well to cognitive–behavioural therapy (CBT). Moreover, as the authors themselves admit, there was a trend from the beginning of higher suicidal cognitions in the TAU group, which assumed statistical significance from the first follow-up at 3 months. Furthermore, the authors have not attempted to match the extra time spent with participants in the CBT group with a similar amount of therapist/contact time in the TAU group. Masking (as acknowledged) of follow-up assessments was not undertaken. Therapists in the treatment group very actively pursued participants; this may have been the active ingredient rather than CBT. Sending postcards alone as an intervention significantly reduces the frequency of hospital-treated self-poisoning events.[2] All these factors bias the results in favour of the treatment group. Despite these biases, the reported benefit in reducing self-harm was marginal and only statistically significant at 9 months, with questionable clinical significance.

The participants in this study differ very significantly from the individuals seen after self-harm by routine liaison psychiatry services. The self-harm definition used was very wide, including punching and head banging, which are not usually defined as self-harm by clinicians and not proven to be associated with higher suicide risk, unlike self-poisoning and self-cutting. No data are reported on the proportion of self-harm in the study which was of this milder nature. Right from the recruitment phase, participants with alcohol and drug misuse were eliminated. This clearly skews the population enormously since a very high proportion of our patients have comorbid issues. The treatment group in particular lost eight individuals before CBT was started, and all assessments and therapy sessions were then completed. We contend that this was a highly motivated and selected group likely to benefit from the intervention, and unrepresentative of the clinical population.

Short-term interventions for self-harm have not generally proved significant when explored in large-scale studies.[3] It is therefore crucial that small randomised trials of CBT or other interventions are carefully designed to minimise bias, and we feel this study fell short of the design and reporting standards we would expect. We are also concerned that high-profile publication of such studies may lead to unwarranted implementation of interventions whose effect is unproven, and whose opportunity costs are great.

1 Slee N, Garnefski N, van der Leeden R, Arensman E, Spinhoven P. Cognitive–behavioural intervention for self-harm: randomised controlled trial. *Br J Psychiatry* 2008; **192**: 202–11.

2 Carter GL, Clover K, Whyte IM, Dawson AH, D'Este C. Postcards from the EDge: 24-month outcomes of a randomised controlled trial for hospital-treated self-poisoning. *Br J Psychiatry* 2007; **191**: 548–53.

3 Crawford MJ, Kumar P. Intervention following deliberate self-harm: enough evidence to act? *Evid Based Ment Health* 2007; **10**: 37–9.

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Authors' reply: Kripalani *et al* express their concerns about biases towards the treatment arm of our study and the characteristics of our study group of patients who self-harm. With respect to biases towards the treatment arm, it should be noted that at the start of treatment no significant differences in anxiety, depression and suicidal cognitions were evident. Thus, the gradually growing difference in depression and suicidal cognitions from the first follow-up at 3 months and in anxiety at the 9-month follow-up in our opinion reflects a treatment effect. Just because the effects on secondary measures were stronger than on the target variable, we concluded that, as hypothesised, CBT primarily targeted maintaining factors of self-harm and that the specific self-harm effect was a secondary effect. Moreover, our study results remain silent on whether the treatment effects observed are attributable to specific ingredients of CBT or to the total package of CBT in addition to TAU. We agree with Kripalani *et al*, however, that the fact that assessments were not carried out masked to treatment