Perhaps this debate needs to move on to a creative engagement with this process.

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Sertraline and exposure therapy in social phobia

I read with interest the article by Haug *et al* (2003), but was puzzled by the conclusion they drew from their data.

After a 24-week study comparing sertraline, sertraline plus exposure, exposure plus placebo, and placebo in patients with social anxiety disorder (Blomhoff *et al*, 2001), patients were followed up at week 52. In the summary the authors conclude that 'Exposure therapy alone yielded a further improvement during follow-up, whereas exposure therapy combined with sertraline and sertraline alone showed a tendency towards deterioration after the completion of treatment'. This seems to be a misleading interpretation of their data.

Haug and colleagues did not mention the primary efficacy measures of their study in their paper. Reading the original paper by Blomhoff *et al*, I find that the primary efficacy measures were numbers of responders or partial responders on the Clinical Global Impression – Social Phobia (CGI–SP) and the Social Phobia Scale (SPS). In the first study, treatment with sertraline was superior to placebo, but exposure was not. For example, 45.5% of the patients treated with sertraline plus exposure were responders compared with 33.0% of the patients treated with exposure plus placebo. I wonder why it was not mentioned in the second paper whether the three active groups differed from placebo and from each other on the primary efficacy measures.

Instead, Haug *et al* report only relative changes of mean scores without adjusting for the large absolute differences at termination of the acute study (week 24). After 52 weeks, exposure patients only caught up to the already better scores of the sertraline groups. From both papers, I calculated the following total mean changes for weeks 0–52 by adding the mean changes for weeks 0 to 24 and the ones for weeks 24 to 52 and found: 1.68 for placebo, 2.02 for sertraline plus exposure, 1.92 for sertraline, and 1.88 for exposure plus placebo on the CGI–SP overall severity. For the SPS, I found the following mean changes: 12.09 for placebo, 15.56 for sertraline plus exposure, 14.12 for sertraline, and 15.91 for exposure plus placebo. These scores may change a little bit after correction for participants who withdrew from the trial. I doubt that any of these scores differs significantly from each other or from placebo. By no means is it true that 'Exposure therapy given alone is more effective in the long term than when given in combination with sertraline'. The opposite is the case: it takes 1 year for the exposure patients to reach the level of improvement that the sertraline and the combination patients have already reached after half a year. Perhaps the patients treated with exposure only showed further improvement during the 'treatment-free' follow-up period because one-fifth of them now received treatment with selective serotonin reuptake inhibitors. Remarkably, there was no deterioration in the sertraline groups on the primary efficacy measures, despite the fact that only one-fifth of this group remained on medication.

I have calculated a Bonferroni-corrected critical P-value of 0.0073 when seven scales are used. Thus, all P-values <0.05 and <0.01 given in the paper may be not significant.

I would suggest that the authors analyse their primary efficacy measures and reinterpret their data.

Declaration of interest

B.B. is or has been a speakers' bureau participant with Aventis, AstraZeneca Pharmaceuticals, Bayer AG, Boehringer-Ingleheim GmbH, Bristol-Myers-Squibb, Eli Lilly and Company, GlaxoSmithKline, Janssen-Cilag, Lundbeck, Meiji-Seiko Pharmaceuticals, Novartis Pharmaceuticals Corp., Organon, Pfizer Inc., Roche, Sanofi-Synthélabo, Solvay, and Wyeth Pharmaceuticals.

Blomhoff, S., Haug, T. T., Hellstrøm, K., *et al* (2001) Randomised controlled general practice trial of sertraline, exposure therapy and combined treatment in generalised social phobia. *British Journal of Psychiatry*, **179**, 23–30.

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Author's reply: The primary efficacy measures from our paper about treatment effect at week 24 (Blomhoff *et al*, 2001) are reported in the method section of the paper about the follow-up study (Haug *et al*, 2003). In the pairwise comparisons, combined sertraline and exposure and sertraline alone were significantly superior to placebo, while a non-significant trend towards increased efficacy of exposure alone compared with placebo was reported.

The four study groups had a significant reduction in scores on all social phobia scales from baseline to follow-up. Furthermore, there was no significant difference in scores on primary efficacy measures between the active treatment groups in any of the time-point analyses between week 0 and week 24. In the follow-up analyses we were therefore mainly interested in the changes after cessation of treatment. For the exposure group and the placebo group there was a further improvement in scores on social phobia from week 24 to week 52 and the changes on several of the subscales were highly significant. On SF–36, which demonstrates changes in a more global functioning, there was a significant improvement for the exposure alone and the placebo groups, while there was a significant deterioration in both the sertraline-treated groups. Changes in scores on other social phobia scales for the sertraline-treated groups were non-significant, but there was a tendency towards deterioration (Tables 1 and 2, pp. 314–315). We agree that the changes in sertraline-treated groups during the follow-up period were marginal. However,

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contrasting these minimal changes with the significant improvement in the exposure-treated group, we find it appropriate to conclude that exposure therapy given alone seems to be more beneficial in the long term. Longer follow-up could have added valuable information to this issue. In all groups about 20% of the patients were treated with sertraline during the follow-up period so this could not explain the differences in scores between the groups at week 52.

Declaration of interest

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Premature conclusions about depression prevention programmes

In my opinion, the meta-analysis by Jané-Llopis *et al* (2003) suffers from some methodological flaws that misguided the authors to draw premature conclusions on predictors of prevention in depression prevention programmes.

First, many of the selected studies did not target the prevention of depression but examined therapeutic or preventive strategies for other primary disorders and used depression scores as secondary outcome measures. For example, Bisson *et al* (1997) studied the efficacy of psychological debriefing on the development of post-traumatic stress disorder (PTSD) in victims of acute burn traumas. They showed that psychological debriefing may even worsen the long-term course of burn victims. But while psychological debriefing may have been mistakenly considered helpful for preventing PTSD in the past, no reasonable therapist or researcher has ever claimed that massive emotional confrontation would represent a promising strategy for depression or depression prevention.

Second, the coding of respective methods looks rather inconsistent, and I wonder how the authors were able to reach such a high interrater reliability across codes. For example, the psychological debriefing method used by Bisson *et al* (1997) was coded as 'behavioural, cognitive and educational' (p. 389), while the code 'cognitive' was missing for Seligman *et al*'s (1999) intervention based on cognitive therapy. Similarly, four research groups using similar variants of the *Coping with Depression Course* by Lewinsohn *et al* (1984) were coded differently (e.g. 'cognitive and competence', 'behavioural, cognitive, educational and social support', 'cognitive', and 'behavioural, cognitive, competence and educational' (pp. 386–391)). Finally, the coding category 'behavioural methods' incorporates very heterogeneous strategies. For example, behavioural strategies found to be helpful in cognitive–behavioural therapy for depression focus on increasing pleasant activities and social skills training (Lewinsohn *et al*, 1984), whereas the delivery of peer support telephone dyads by lay persons, as used in the studies by Heller *et al* (1991), may be regarded as a very specific behavioural strategy which has so far not been recommended as a helpful intervention by the research community. In Jané-Llopis *et al*'s meta-analysis, respective interventions from the studies by Heller *et al* (1991) had negative effect sizes and therefore may have substantially accounted for the missing or even negative effect of the 'behavioural' component of preventive measures.

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Homicide data

I am writing to query the homicide statistics quoted by Dr Salib (2003). The figures he quotes for total annual homicides suggest a fall in homicide between 1979 and 2001. The source for his figures is quoted as the Office for National Statistics (ONS).

Homicide statistics are easily available through the website of the ONS and from various other sources, including Home Office statistical bulletins and the House of Commons Library. For example, Richards (1999) describes homicide trends between 1945 and 1997, demonstrating the dramatic rise in rates of offences initially recorded as homicide seen over that time from around 300 or 400 a year in the 1950s to more than 700 a year in the late 1990s. The recent Home Office Statistical Bulletin (Simmons & Dodd, 2003) shows a continuing rise in this trend with 1048 deaths initially attributed to homicide in 2002/2003, although these figures are based on date of notification and thus can include deaths that actually took place in earlier years.

Dr Salib's paper appears to use data on death registrations from the ONS where there has been a conviction for murder or for manslaughter. However, the ONS assigns a temporary ICD–9 code for cause of death for deaths where death was violent, unnatural or suspicious or pending the outcome of inquests and legal proceedings, which are of course often prolonged. The ONS site itself states that it is difficult to present accurate statistics on number of homicides using death registrations, which is what Dr Salib has seemingly attempted to do.

As psychiatry is faced with a Government currently determined to medicalise as far as possible the growing problem of violence in our society, it is essential that psychiatric journals present statistics on this subject in a meaningful fashion. Dr Salib's paper, although not specifically about trends in homicide over time, presents misleading data on this subject, which are neither helpful nor informative to the wider debate on violence in society.

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Author's reply: Dr Rowlands raises an important question, triggered by homicide data in my recent paper on the effect of

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