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Author's reply: There were no statistically significant differences between the groups at pre-treatment (as can be read from Table 2, means and standard deviations were very similar across groups). However, for several reasons we found it appropriate not to report *P*-values of baseline data. Analyses were conducted using ANCOVAs, holding pre-treatment values as covariates. Moreover, when *n* is small, considerable variation between groups can be the case without reaching statistical significance, because of limited power. Consequently, several scientific journals (e.g. *Annals of Internal Medicine*[1]), advise against the use of *P*-values when comparing baseline data in randomised controlled trials.

As for the name of the treatment, we view the term internet-based cognitive–behavioural therapy (CBT) as most suitable. The treatment's theoretical foundation and its components are based on learning theory and cognitive theory. As stated in the Method and the Discussion sections, the rationale for including a mindfulness exercise was to reduce avoidance behaviours related to bodily sensations and to enhance exposure. Also, as the term CBT has been used for describing a plethora of treatments with substantial inter-treatment variability, the addition of 'modified' would probably be misleading rather than clarifying. In fact, a recent paper presents mindfulness-based cognitive therapy as 'a newer variation of cognitive behavioral therapy'.[2]

Regarding the control group, I agree that participating in a discussion forum hardly can be viewed as the optimal control condition. However, as the present study is the first ever to investigate internet-based CBT for health anxiety, a comparison with conventional CBT would have been premature. Such a comparison would have meant conducting a non-inferiority trial presenting difficulties regarding criteria for non-inferiority as well as the inherent assay sensitivity problem. In addition, far more participants would have needed to be randomised to internet-based CBT (because of power issues), which would have been ethically questionable. That is, far more patients would have been exposed to a potentially non-effective or even unsafe treatment. As I see it, the ideal control condition would rather have been an internet-based psychological placebo arm providing the same amount of therapist attention and treatment credibility without targeting the central proposed mechanisms of change.

When it comes to recruitment, I consider advertisements and an article in a newspaper as two quite different forms of attention. The former is under complete control of the researcher while the latter is not. As a consequence, I find it reasonable to assume that the two forms of attention have differential effects in terms of recruitment and that they therefore should be reported separately.

As for generalisability of the findings, Udo *et al* state that our paper tells us little as to whether internet-based CBT works in acute psychiatry settings or in an in-patient psychiatric context. I can only say that I absolutely agree. The clinic at which the present study was conducted is an out-patient clinic and internet-based CBT is not different from conventional CBT in the sense that one should be vary cautions in generalising findings from one healthcare context to another.

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Childhood psychotic symptoms: link between non-consensual sex and later psychosis

Numerous studies have established a link between trauma early in life and psychosis in adulthood.[1] In particular, non-consensual sex in childhood appears to robustly predict the occurrence of psychotic symptoms later in life.[2] Bebbington *et al*[3] add to this literature by demonstrating a large potential role of non-consensual sex in the development of psychosis in a large representative sample of English adults. However, although the authors take several steps to adjust for residual confounding, they make no attempt to correct for the presence of psychotic symptoms in childhood. This is a potentially critical error as reverse causation remains a distinct possibility. Children who exhibit psychotic symptoms may be at high risk of sexual victimisation owing to their poor social skills, paucity of social relationships, and for numerous other reasons. Thus, initial mental health may explain the link between sexual abuse and adult psychosis.

In an analysis of over 3500 British adults reported elsewhere,[4] I showed that non-consensual sex at age 16 or earlier placed females at a substantial risk of auditory and visual hallucinations at age 29 (OR = 8.51, 95% CI 0.99–73.28). However, females who experienced hallucinations in childhood were also likely to have been forced to have sex by age 16. When the presence of initial psychotic symptoms was taken into account the link between non-consensual sex in childhood and hallucinations in adulthood was diminished to non-significance (OR = 2.43, 95% CI 0.09–62.88). These findings suggest that childhood sexual abuse may not be related to psychosis in adulthood over and above psychotic symptoms in childhood, at least in the domain of visual and auditory hallucinations.

Thus, when patent non-causal explanations have not been tested, vigilance is required prior to inferring that the link between sexual abuse and psychosis may be causal. Although the design utilised by Bebbington *et al* was cross-sectional, it would have been possible to ask participants to retrospectively gauge the age at onset of their psychotic symptoms. This would have allowed the researchers to produce a more methodologically robust assessment of the potential causal effect of sexual abuse.

Bebbington *et al* also identified anxiety and depression as partial mediators of the relation between sexual abuse and psychosis. However, poor initial mental health may have determined both childhood abuse and later experiences of depression, anxiety and psychosis. It is therefore of the utmost importance that those assessing the role of environmental risk factors in predicting psychosis endeavour to assess the presence of psychosis and subclinical psychotic symptoms and mental health more generally at baseline. This will allow the contribution of early environmental risk factors to psychosis to be evaluated and will provide a robust evidence base for clear policy-relevant recommendations.