Previous studies[2] consistently indicate increased prevalence of dementia in older African–Caribbean people when compared with the indigenous White population in the UK. The magnitude of this difference between these populations is not clear. Hence, there is a definite need for well-planned epidemiological studies to determine the actual burden of disease. Surprisingly, Adelman *et al*'s study[1] presumed that vascular factors such as hypertension and type 2 diabetes are likely to increase the burden of dementia in the African–Caribbean population. However, the possibility of other risk factors such as depression, illiteracy and prevalence of apolipoprotein 4, which, presumably, increase the chances of subsequent dementia, needs more emphasis.[3,4] Current data from sub-Saharan Africa and India[4] suggest that age-adjusted dementia prevalence estimates in 65-year-olds are low (1–3%) compared with other low- and middle-income countries. It appears that there is a need to identify potentially modifiable environmental/genetic factors to explain the increased prevalence of dementia when this population migrated to the UK. Therefore, future studies are needed to identify these risk factors in this migrant population.

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Authors' reply: We agree that it is helpful to emphasise that we do not know whether vascular factors are the primary aetiology behind the increased prevalence of dementia in this population. We considered literacy to be a risk, and this (like our earlier study) controlled for education[1] and found no difference between ethnic groups. Similarly, depression rates in older Black and minority ethnic populations have not been found to be raised;[1] nor has the prevalence of apolipoprotein 4 when compared with their White counterparts.

However, there are contradictory findings about whether the expression may be the same.[2–5] Thus, although all these factors may relate to the rates of Alzheimer's dementia, there was no clear evidence to suggest they are responsible for the increased rate in the African–Caribbean group. Finally, there is no evidence that the prevalence of dementia in the participant's country of birth (Caribbean Islands) is lower than that for the UK. A Delphi consensus study estimated that the rates for Latin America and the Caribbean are at least as high as for Western Europe.[6] We agree, however, that more research is needed to consider the possible aetiology and modifiable risk factors.

Declaration of interest

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Internet-based CBT for severe health anxiety

Having appraised the evidence regarding the article by Hedman *et al*,[1] we write to comment as follows.

First, it is not possible, from the article,[1] to tell whether the comparison group was similar to the experimental group, as no statistical tests were done.

Second, the treatment described by the authors as internet-based cognitive–behavioural therapy (CBT) involved components of mindfulness and may have been more appropriately described as internet-based modified CBT.

Third, given that defined psychological approaches, including CBT are accepted as treatment for health anxiety,[2–5] CBT delivered as usual may have been a more appropriate control treatment than the online discussion forum. An online discussion forum is not recognisable or recommended treatment for health anxiety.

Fourth, the description of participant recruitment is contradictory: 'There were no advertisements in newspapers or in other media. However, an article about the study was published in a major nationwide newspaper'.

Fifth, we note that the power in per cent is not stated explicitly in the study such as to inform respective clinician's appraisal of this study as regards applicability of results to various clinical settings.

In light of the above, there is a need for cautious interpretation of the evidence presented, which we feel has limited therapeutic value in the acute psychiatry settings, such as crisis resolution and home treatment teams and in-patient wards, in which we work. However, we value this paper as adding to the limited body of knowledge available about treatments for health anxiety and expanding the notion that this disorder is treatable.

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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.