Need to identify modifiable risk factors of dementia in the older UK African–Caribbean population
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How to interpret different results for CRHTT data

Jacobs & Barrenho\(^1\) used the same data as Glover et al\(^2\) when they were comparing admissions in primary care trusts with and without crisis resolution and home treatment teams (CRHTTs). However, they employed different methods for their analysis and reached conflicting conclusions. According to Jacobs & Barrenho, the introduction of CRHTTs did not have a statistically significant influence on the number of admissions, while Glover et al found a significant reduction especially for CRHTTs offering a 24-hour service.

In their article, Jacobs & Barrenho\(^1\) do report a reduction in admissions (e.g. Fig. 4) but state that it was not statistically significant. They do not mention power calculations. There were usable data available from 229 primary care trusts (PCTs) and the authors conducted various complex analyses by using a number of control factors and by studying trends over time. It could be that their lack of statistically significant findings is because of a lack of power. If this is the case, there is no fundamental difference between their findings and the previous analysis.\(^2\)

At the end of their article, the authors make the suggestion that perhaps data should be analysed at the level of CRHTTs and not at the level of PCTs, given that there is huge variation between CRHTTs. We concur with that suggestion and we would like to go even further and suggest that future studies look at the service actually provided to individual patients in terms of how many visits are undertaken over a specified number of days. This information is readily available from most electronic notes systems. Further study is needed to investigate the types of interventions provided, such as whether medication was prescribed and administered, whether specific psychological treatments were offered, and so on. The availability of such data will allow an informed decision to be made about what is required to avoid admission to hospital and whether a CRHTT is the best organisational format to deliver that care.


Previous studies 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 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. Current data from sub-Saharan Africa and India 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 in this migrant population. When the population migrated to the UK, future studies are needed to identify these risk factors in this migrant population.


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 and found no difference between ethnic groups. Similarly, depression rates in older Black and minority ethnic populations have not been found to be raised, 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. 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. We agree, however, that more research is needed to consider the possible aetiology and modifiable risk factors.

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Internet-based CBT for severe health anxiety
Having appraised the evidence regarding the article by Hedman et al, we write to comment as follows.

First, it is not possible, from the article, 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, 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.


from one healthcare context to another.

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internet-based CBT is not different from conventional CBT in

present study was conducted is an out-patient clinic and

acute psychiatry settings or in an in-patient psychiatric context.

paper tells us little as to whether internet-based CBT works in

groups). However, for several reasons we found it appropriate

ences between the groups at pre-treatment (as can be read from

using ANCOVAs, holding pre-treatment values as covariates.

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limited power. Consequently, several scientific journals (e.g.

when comparing baseline data in randomised controlled trials.

focal papers: ‘cognitive behavioral therapy’ .2

ably be misleading rather than clarifying. In fact, a recent paper

relationships, and for numerous other reasons. Thus, initial

mental health may explain the link between sexual abuse and adult

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 mindful-
ness 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 prob-
ably 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 cautious in generalising findings
from one healthcare context to another.

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 al3 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.
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author’s reply: Dr Daly argues that the link between child sexual abuse and adult psychosis may be the result of confounding by psychotic symptoms in childhood or adolescence. He adduces evidence for this from his secondary analysis of data from the 1970 British Birth Cohort sample. Of the female sample, 1.6% indicated that they had been forced to have sex by the age of 16, and this was associated with an elevated risk of visual and auditory hallucinations at age 29 (OR = 8.5). However, after controlling for the experience of such quasi-psychotic symptoms before the age of 16, the odds ratio fell to a non-significant 2.4. Daly interprets this as indicating that this relationship exists because children with quasi-psychotic symptoms are more at risk of abuse and also at greater risk of developing psychosis as adults.

Nevertheless, Dr Daly’s conclusion must equally be tentative. First, the British Birth Cohort sample apparently does not provide temporal discrimination between the occurrence of sexual abuse and the development of quasi-psychotic symptoms. Second, given that this is so, the diminution of the odds ratio after controlling for quasi-psychotic symptoms in adolescence could indicate mediation. In other words, the sexual abuse leads to adolescent symptoms which are then associated with adult symptoms. I find this explanation more plausible than the suggestion that psychotic symptoms themselves have a major effect in increasing vulnerability regarding treatment.

Revascularisation in patients with mental illness

Mitchell et al must be congratulated on their systematic review of myocardial revascularisation in patients with mental illness. As physicians performing revascularisation procedures, we were disappointed by the inferior treatment received by patients with mental health problems. Fortunately, these patients account for only a minority of those presenting to acute cardiology services with symptoms and signs suggestive of acute coronary syndrome. However, when they do attend, they present cardiologists with a number of challenges, which ultimately can influence the decision regarding treatment.

Revascularisation remains an important treatment for those patients with myocardial necrosis, providing both symptomatic and prognostic benefit. Importantly, however, it can only be performed following invasive coronary angiography – a procedure which carries a risk of vascular complication, myocardial infarction, stroke or even death of 0.2–1.0%. Clearly, patients must give appropriate consent before coronary angiography is undertaken, and this can represent an important hurdle when treating patients with mental health problems.

A second important challenge which should be considered prior to undertaking angiography, and must be considered prior to performing definitive revascularisation, is the issue of adherence to medication. Frequently, revascularisation can be performed percutaneously at the time of angiography. This procedure usually necessitates the implanting of coronary stents, which are small permanent metal scaffolds that help maintain coronary vessel patency. There are many advantages to using these devices; however, in recent times stent thrombosis has emerged as the most serious and worrying complication of their use. This condition is fortunately rare, but it remains a devastating, unpredictable event that has a significant morbidity and mortality; up to a third of patients will die. Research has identified that early or premature discontinuation of dual antiplatelet therapy is one of the most important risk factors in stent thrombosis. Consequently, cardiologists are reluctant to implant stents in patients who they feel are unlikely to comply with dual antiplatelet therapy. Unfortunately, patients with mental illness have been shown to be less adherent to medication, a factor which certainly has an influence on revascularisation decisions.

These issues represent important challenges (and not excuses), which must be overcome to allow our patients to receive the most appropriate treatment. The differences in treatment certainly deserve to be highlighted and as recommended by Mitchell et al the reasons behind them require more in depth investigation, especially within the confines of the National Health Service.


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than for antipsychotics.3 One important question here is whether probably lower for hypoglycaemic and antihypertensive drugs non-adherence rates among patients with severe mental illness is not significantly different from those with diabetes and hypertension. It is worth noting that suggesting persistent refusal may be overestimated, accounting for the very small proportion of patients who cannot initially consent because of acute mental illness are always given a second chance to consent once well? Better links between physicians and psychiatrists would no doubt help here. Even in those with mental ill health, the vast majority of problems with day-to-day adherence are caused by accidental omissions and rational non-adherence to medication adherence among patients with schizophrenia and comorbid diabetes and hypertension. Garg & Garg rightly highlight what factors underlie these apparent deficits in received cardiac care. Garg & Garg raise two issues that we agree deserve further comment. A first is the importance of non-adherence among patients with severe mental illness. Generalised spike-and-slow-wave complexes without seizures in schizophrenia. There has been long discussion about the increased prevalence of electroencephalogram (EEG) abnormalities and their significance in patients with schizophrenia.4,5 Although interictal epileptiform discharges presumably indicate a higher risk for seizures,5 such abnormalities alone in a clinical case of schizophrenia are generally not regarded as having strong implications for antipsychotic therapy. Here, we report the case of a 17-year-old student who over a period of several months developed a paranoid-hallucinatory syndrome, feeling persecuted, sidelined and out-casted by his peers. He also experienced changes in auditory perception, reported supersensitive hearing and auditory hallucinations of backbiting whispering voices of his peers. There was a prodromal phase with increasing social withdrawal, affective flattening and a drop in school grades over a period of 2 years prior to the diagnosis of schizophrenia by an out-patient psychiatrist. Treatment with 250 mg quetiapine led to some improvement in his symptoms or magnetic resonance imaging symptoms or signs. A routine clinical EEG showed infrequent 3 Hz spike-and-slow-wave complexes (SWCs). Video telemetry for 3 days clearly showed 3 Hz SWCs with a duration of between 200 and 3500 msec and an average frequency of about 8 per hour and a peak frequency of 18 per hour without clinical seizure correlates. Assuming
that the EEG findings might play a role in the genesis of schizophreniform syndrome, medication was changed to valproate monotherapy. This resulted in full clinical and cognitive remission and considerable improvement of the EEG within a few weeks. Subsequently, the patient's school grades returned to top levels.

The clinical relevance of such an EEG finding in a patient with schizophrenia is still an unresolved question. In spite of an intensive historical discussion of this issue, to our knowledge this is the first description of a clinical case of schizophrenia with generalized 3 Hz SWCs and excellent clinical response to valproate monotherapy. In our view, this case illustrates three clinically important points: (1) it is worthwhile doing EEG studies in patients with schizophrenia; (2) non-ictal SWCs might play a pathogenetic role in a small subgroup of patients with schizophrenia; and (3) in clear-cut cases of SWCs in patients with schizophrenia but without clinical seizures, a therapeutic trial with anticonvulsant medication might be warranted.