British Journal of Psychiatry (1995), **167**, 818–826

Correspondence

Contents: Cognitive functioning with chronic fatigue/Comorbidity and suicide risk/Five group comparisons of treatments for anxiety disorders/Homicidal behaviour and mental disorders/Seizure threshold in bilateral and unilateral ECT/Late-life depressive order/Keeping count for the community/Severe apathetic hyperthyroidism with normal thyroid hormone levels/Antidepressants in pregnancy and breastfeeding/Neuropsychological function in manic-depressive psychosis/Visual fields in Gilles de la Tourette syndrome

Cognitive functioning with chronic fatigue

SIR: Cope *et al* (1995) claim that cognitive impairments associated with chronic fatigue "probably reflect psychopathology rather than a post-viral process". They based this conclusion on findings from a small, heterogeneous group of patients, some of whom may have been cases of true post-viral fatigue syndrome, but a significant number of whom may have been suffering from previral fatigue. Given that the documented infections in the latter cases were probably coincidental, why was it assumed that their symptoms might have been caused by encephalitis? Surely, if the researchers had really wanted to assess the role of a "post-viral process" on cognitive functioning, they should have restricted their sample to patients with unambiguous post-infectious fatigue and included at least one relevant measure of immune status at the time of testing.

Although British doctors have generally been reluctant to distinguish subgroups of patients with chronic fatigue syndrome, it is noteworthy that where research has focused on more strictly defined samples, objective evidence of cognitive impairments has been found (Riccio *et al*, 1992; Sandman *et al*, 1993). Indeed, neuropsychiatric dysfunction is included as a major criterion in several case definitions of this syndrome (Lloyd *et al*, 1990; Lapp & Cheney, 1995). Even the Oxford guidelines used by Cope *et al* require that the fatigue must affect "mental functioning".

Since the results of this study indicate that the subjective reports might have been exaggerated and even inaccurate, shouldn't the researchers have reconsidered their diagnosis of the 11 subjects with CFS? Conversely, if they took the patients' own assessments as being valid and reliable, why do they not extend this to their views of aetiology and opinions concerning exercise?

Finally, we note that Cope *et al* described the sample studied by Riccio *et al* (1992) as "depressed CFS patients". In fact, these researchers focused on myalgic encephalomyelitis (ME) and it is clear from the paper that not everyone would have fulfilled the existing diagnostic criteria for CFS. As for their psychiatric status, our copy of the article states that only 22% suffered from depression. Cope *et al*'s rediagnosis of other researchers' patients would not have bothered us had it not preceded a jibe at experienced colleagues whose reasoned interpretation of the data they dismissed as a "misuse of neuropsychological test results".

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AUTHORS' REPLY: Goudsmit & Howes take issue with the conclusions from our study, namely that subjective cognitive complaints are a prominent feature of chronic fatigue, (while objective cognitive and MRI abnormalities are not), and that such complaints probably reflect psychopathology rather than a post-viral process.

https://doi.org/10.1192/S0007125000066186 Published online by Cambridge University Press

We do not understand what the correspondents mean when they describe our patient group as "heterogeneous" since they are carefully described and come from a clearly defined epidemiological population. The role of viral illness in causing chronic fatigue remains undetermined and it is at present impossible to state with any certainty whether a patient's fatigue is a consequence of a viral infection. It was never "assumed that their symptoms might have been caused by encephalitis".

The correspondents comment that "the results of this study indicate that the subjective reports might have been exaggerated or even inaccurate". It is not clear on what evidence they are basing this opinion – if it is because few deficits were revealed on objective cognitive testing, it is precisely this discrepancy which has been noted consistently by other researchers. Goudsmit & Howes, however, suggest that we should reconsider our diagnosis of CFS subjects (of whom there were 21 not 11 as they state) post-hoc because of it. This would be scientifically invalid. Such comments also demonstrate precisely our point in exercising caution in the use and interpretation of neuropsychological tests to confirm or refute an organic basis for CFS.

The study by Riccio *et al* (1992) is commendable as being one of the first in the area. However, the authors described their results as preliminary; their nine patients with ME had significantly higher scores than controls on the depression subscale of the HAD, and 2 were defined as retarded depression using the PSE. It is now clear that studies of this kind must take into account coincident depression and anxiety in subjects with severe fatigue when assessing cognitive performance. Another advance in research in this area has been the near universal adoption of operational criteria for chronic fatigue syndrome, in preference to the potentially misleading term myalgic encephalomyelitis.

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Comorbidity and suicide risk

Sir: Hornig & McNally (1995) argue that we did not control optimally for comorbid disorders known to increase suicide risk. In our initial paper (Weissman *et al*, 1989), we controlled statistically for major depression, alcohol and drug abuse, and agoraphobia separately, and for the latter three disorders, in combination. The odds ratio did not change significantly with all of these combinations.

In a follow-up report based on the ECA data (Johnson *et al*, 1990), we directly estimated the risk of suicide attempts in individuals with uncomplicated panic disorder compared to comorbid conditions. We also conducted similar analyses for major depression. We showed that the lifetime rates of suicide attempt in individuals with uncomplicated panic disorder (7.0%) were higher that for persons with no psychiatric disorder (1.0%), giving a significant odds ratio of 5.4 with 95% CI of 1.9–15.4. (Although the numerator of this rate of suicide attempts in individuals with uncomplicated panic disorder consists of four individuals with a lifetime diagnosis of uncomplicated panic disorder who attempted suicide, the total sample size (denominator) on which this rate is based is the 57 individuals with uncomplicated panic disorder, which in our opinion is a reasonable sample size.) We also showed that the lifetime rate of suicide attempts in individuals with uncomplicated major depression was 7.9%, a figure which is clearly not significantly greater (either statistically or clinically) from that of the rate for individuals with uncomplicated panic disorder. More importantly, we showed that individuals with comorbid panic disorder have rates of suicide attempts equal to that of individuals with comorbid major depression.

Hornig & McNally have now compared the rates of suicide attempts in individuals with specified "uncomplicated" psychiatric disorders to the rates of suicide attempts in individuals with only that specified disorder and panic disorder (their Table 2). Their interpretation of their results is that "uncomplicated" panic disorder does not confer any additional risk for suicide attempts over and above that associated with the specified uncomplicated psychiatric disorder. However, one could use the data presented in their Table 2 using their same logic to argue that the presence of a psychiatric disorder, such as major depression, in addition to panic disorder does not increase the risk of suicide attempts in those with uncomplicated panic disorder. This may be done by comparing the value of 7.0% (the rate of suicide attempt in those with uncomplicated panic) to that of 0% (the rate of suicide attempt in individuals with only major depression and panic) to conclude that the presence of comorbid major depression does not increase the risk of suicide attempts in individuals over that associated with uncomplicated panic. Clearly, this implausible result, which is inconsistent with results from a number of clinical studies, is due to

https://doi.org/10.1192/S0007125000066186 Published online by Cambridge University Press