decreased need for others. The tortoise, although not a mammal, provides the perfect example: its extraordinary defensive shell reduces the need to flee or fight to virtually zero.

Furthermore, the possibility arises that future gene therapies may have serious untoward consequences (Watson & Andrews, 2002). The genes associated with social defeat may encourage a battered wife to yield to her brutal husband, albeit at the price of chronic depression. Should her relevant genes be deleted by future gene therapy, potential consequences include being killed as a result of not yielding, killing her abuser in retaliation or at least a heightened state of domestic abuse. The potential for such untoward consequences must be considered.

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Authors' reply: We thank Drs Price and Cantor for their interesting reactions. We welcome the prophet as a much more colourful 'endophenotype' for schizophrenia than a dysfunction in eye-tracking or prepulse inhibition. However, whether the increased reproductive fitness of a small number of prophets compensates for the reproductive impairments in patients with schizophrenia and their lonely schizotypal relatives remains to be demonstrated.

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Schizophrenia, cancer and imprinting: early nutritional influences

We read with interest the important findings of Goldacre *et al* (2005) on the association between schizophrenia and cancer morbidity. Compared with the general population, they found a reduced rate of cancer of the colon in the schizophrenia cohort (adjusted rate ratio 0.72, 95% CI 0.50–1.01) with a trend towards significance (P=0.06). Rates of rectal cancer were significantly reduced in people with schizophrenia (rate ratio 0.57, 95% CI 0.33–0.93, P=0.03). In their discussion, they emphasised the reduced rate of skin cancer in the schizophrenia cohort (rate ratio 0.56, 95% CI 0.36–0.83, P=0.004).

Recent studies suggest that abnormal insulin-like growth factor-2 (IGF-2) imprinting is aetiological in the development of colorectal cancer (Jirtle, 2004). Genomic imprinting occurs following epigenetic modification of the germ line, which results in parent-of-origin dependent, monoallelic gene expression in somatic cells. Epigenetic changes in the genome are stable but reversible alterations in a CpG dinucleotide or histones, for example through changes in methylation. The genome of colonic epithelium from patients with colorectal cancer is hypomethylated compared with normal colonic epithelia (Feinberg & Vogelstein, 1983). Hypomethylation results in the loss of IGF-2 imprinting. We proposed abnormal imprinting (deletion of paternally expressed IGF-2) as a possible mechanism associated with schizophrenia risk (Abel, 2004). Early nutritional influences (prenatal/maternal) may stimulate changes in cytosine methylation to which imprinted genes such as IGF-2 seem susceptible. Early nutrition may influence susceptibility not only to adult obesity, diabetes and cardiovascular disease (Waterland & Jirtle, 2004) but also to schizophrenia. This suggests that early nutritional interventions aimed at preventing chronic disease are an exciting possibility in schizophrenia. This view is supported by Dutch and more recent Chinese data which indicated that rates of schizophrenia doubled following prenatal exposure to famine (St Clair *et al*, 2005).

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Post-traumatic stress disorder after life events

In their interesting article Mol *et al* (2005), who conclude that life events might cause as many post-traumatic stress disorder (PTSD) symptoms as do traumatic events. The design and conclusions of the study raise some doubts.

Our first concern relates to the assumption that a particular symptom or disorder may be diagnosed in the absence of the fundamental nature or essence of the disorder. The authors claim to identify PTSD in the absence of (a) a traumatic event and (b) a response involving intense fear, helplessness or horror. Both these criteria are diagnostic A criteria of the DSM–IV and essential features of PTSD. Diagnosing PTSD with disregard for a traumatic event is like diagnosing a major depressive disorder in the absence of depressed mood.

Our assumption regarding the broad use of the PTSD diagnosis is strengthened by the instrument used for assessing PTSD symptoms, which has high sensitivity but lacks specificity (Carlson, 1997). In addition, it is questionable whether a self-report scale can assess the clinical relevance of symptoms. Our concern is corroborated by the results in Table 4. The only item on which the life events group scored higher than the traumatic events group was the non-specific symptom of 'having trouble concentrating', whereas the traumatic events group scored higher on 'trauma-specific items' such as amnesia and hyperarousal.

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Another concern is the selection of traumatic events. Accidents, sudden death of a loved one and witnessing violence are categorised as traumatic events but gave relatively low PTSD scores. In our opinion, such events may evoke a range of reactions such as guilt, anger, sadness, anxiety and apathy. Again, if criterion A2 – a response involving intense fear, helplessness or horror – has not been assessed, it is questionable whether these experiences were really traumatic.

The conclusion that life events can generate as many PTSD symptoms as traumatic events is unjustified. At most it could be concluded that some of the PTSD items might not be specific to trauma but are more general stress-related symptoms.

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Authors' reply: Drs de Bruijn & Denys are concerned about identification of PTSD in the absence of the A1 and A2 criteria of the DSM–IV. However, we did not diagnose PTSD but we looked at PTSD symptomatology related to the worst event experienced by participants (including traumatic and non-traumatic events). We did not include the A1 criterion because we wanted to investigate whether the 17 symptoms that are thought to typically occur in those who have experienced a traumatic event, as defined by DSM–IV, are indeed specific for that type of event or occur as frequently following non-traumatic events. In order to study this we inevitably chose events that did not fulfil the A1 criterion (otherwise we would not have had a control group of events). Regarding the A2 criterion, it would be interesting to study respondents' subjective appraisal of the event in terms of fear, helplessness and horror. This would clarify whether the A2 criterion is also as specific for traumas as is often argued and how it is related to the 17 B criteria of the DSM–IV. We would not be surprised if non-traumatic major events could also evoke the emotions of fear, helplessness and horror.

Drs de Bruijn & Denys were also concerned about the somewhat low specificity of the self-report scale we used to measure PTSD symptoms (the Post-traumatic Stress Symptom Scale – Self-Report version; Foa *et al*, 1993). However, it is conceivable that our results are owing to the lack of specificity of PTSD symptoms in general for diagnosing PTSD, as was demonstrated in a recent study by Gold *et al* (2005).

Concerning the results in Table 4: the traumatic events groups did score higher on several items (3 out of 17) but these differences were not significant, indicating that no specific items were more strongly related to traumatic events than to life events.

In summary, our main conclusion that life events can generate as many PTSD symptoms as traumatic events is upheld.

Declaration of interest

The Achmea Foundation for Victim Support in Society paid the salary of S.S.L.M. but had no influence on the methodology or analyses of the study.

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Psychiatric comorbidity and chronic fatigue syndrome

Prins *et al* (2005) assessed psychiatric comorbidity in chronic fatigue syndrome (CFS) using the Structured Clinical Interview for DSM–III–R. Comorbidity was remarkably low compared with similar investigations, and in particular the apparent absence of current post-traumatic stress disorder (PTSD) was striking. The authors speculated that the low comorbidity rates might result mainly from a lack of 'psychiatric bias' of the examiners. They also found that psychiatric comorbidity did not predict the outcome of cognitive–behavioural therapy.

Without doubt, diagnosing comorbid depression and anxiety disorders in CFS is useful because both are highly treatable emotional reactions to the illness. The relevance of somatoform disorders (such as somatisation disorder) for CFS is more doubtful, given their inherently dualistic character (Mayou *et al*, 2005). Most importantly, the very low lifetime incidence of PTSD reported by Prins *et al* (2005) emphasises the value of descriptive psychiatric diagnoses in CFS. In my experience many patients with CFS report victimisation during childhood and/or adult life, and this has been confirmed by a controlled questionnaire-based study (Van Houdenhove *et al*, 2001). However, most victimised patients have 'sub-threshold' symptoms that do not meet diagnostic criteria of clinical PTSD. It is important to listen carefully to the patient's life history (Van Houdenhove, 2002) in order to shed light on any aetiological role of traumatic experiences in CFS and the resulting personality disturbances that may negatively influence treatment.

In summary, psychiatric evaluation of patients with CFS should not be limited to establishing a diagnosis of psychiatric comorbidity but should first involve narrative strategies (Greenhalgh & Hurwitz, 1998).

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