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### Bad medicine: the menopause

I completely concur with Dr Des Spence: Big Pharma is manipulating doctors and exploiting patients, and Big Medicine is neglecting its role to protect patients.1

I can remember, shortly after becoming a GP Principal in 1987, a drug representative trying to persuade us to prescribe evening primrose oil capsules for itching in eczema. The glossy presentation and neurolinguistic programming attempts to persuade failed because of my training. A study of n = 20 can prove anything. So how did it get a licence? Of course, after many years its licence was withdrawn as there was no evidence of positive effect.

Later I asked drug representatives two questions: What is the number needed to treat (NNT)? And what is the number needed to harm (NNH)? The response was mostly gobbledygook, or I'll get back to you, which they never did.

Looking at this more seriously, if referees and journal editors insisted on NNT and NNH figures in research/review papers, instead of relative risk everybody would understand the results more easily. It is depressing that many health media correspondents do not know the difference between actual risk and relative risk, and so misinform the public on the effectiveness of treatments. Unfortunately this is unlikely to happen as even NICE refuses to adopt NNT and NNH as a type of outcome description.

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# Chronic fatigue syndrome: is the biopsychosocial model responsible for patient dissatisfaction and harm?

Geraghty and Esmail have done well to draw attention to the substantial biomedical literature now extant around chronic fatigue syndrome (CFS).1 They are right to point out that the dominant model has been a biopsychosocial (BPS) one, and that this has led to persistent disagreement between doctors and patients. What is particularly salutary is the absence of any advance in therapy, so that they recommend, for example, cognitive behavioural therapy (CBT), but with the caveat that it might not be helpful in individual cases. CFS continues to challenge GPs by its resilience to treatment, but colleagues will be well advised to take on board the changing evidence for pathophysiology set out in this article. Many of the cited papers are accessible and worth reading, such as the authors' reference 8,2 and in that paper, reference 38.3

The bottom line for me is respect for the patient, and humility in the face of lack of knowledge about the precise causes of CFS, which is clearly anyway a heterogeneous group of conditions.

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## Chronic fatique syndrome and the biopsychosocial model

I was pleased to see this article highlight the potential harms of graded exercise therapy (GET) and CBT for patients with myalgic encephalopathy (ME).1 I myself was diagnosed with ME as a 20-year-old student in early 1984 by a consultant neurologist in Glasgow. My illness was triggered by Coxsackie B4 virus — there was an outbreak of Coxsackie in the West of Scotland at this time. Since my own diagnosis with this poorly understood illness, I have been baffled — and shocked to see the criteria of ME diluted in the early 1990s and the consequent conflation with unexplained 'chronic fatigue'. Moreover, the adoption of the biopsychosocial model of 'ME/ CFS' has certainly not been beneficial to my own experience of illness. I am hopeful that, with the dedicated international biomedical researchers we now have, there will be effective therapies in my lifetime. There are many, many people who truly suffer with this dreadful illness.

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### Hypertension in surgical patients: the role of beta-blockers

We read the editorial on 'Preoperative blood pressure measurement: what should GPs be doing?' with great interest.1 In support of the lack of evidence that reducing blood pressure helps, the authors quote the POISE study,2 stating that beta-blockers were used to reduce blood pressure preoperatively and the data suggested that it did more harm than good. POISE was not designed to test the effects of reducing blood pressure before