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*BMJ* 2004;329:928-929
doi:10.1136/bmj.329.7472.928

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What causes chronic fatigue syndrome?

Infections, physical inactivity, and enhanced interoception may all play a part

Chronic fatigue syndrome, also known as myalgic encephalomyelitis, is an illness of unknown nature and cause, but most medical authorities now accept its existence. Research about its cause has been hampered by the absence of a biological marker, the heterogeneous nature of the illness, and difficulties in differentiating cause from effect. Yet, some progress has been made, particularly when causes are divided into predisposing, triggering, and maintaining factors.

Women get chronic fatigue syndrome more commonly than men for unknown reasons, although increasing evidence suggests a genetic influence on the illness.1 Premorbid mood disorders are replicated risk markers for chronic fatigue syndrome;1–3 the risks may be inflated by shared symptoms or they may be markers for those patients with comorbid mood disorders.1–3 Another replicated premorbid risk marker is increased consulting of a doctor for minor illnesses up to 15 years before diagnosis,1–3 suggesting a general vulnerability for either ill health or seeking health care, the latter possibly being mediated by comorbid anxiety.

C certain infectious illnesses, such as Epstein-Barr virus, Q fever, and viral meningitis, can trigger chronic fatigue syndrome.4–6 Common upper respiratory infections do not.4–6 Little evidence exists of persistent infection in patients with chronic fatigue syndrome.4–6 An immune cause has not been established, although preliminary research suggests that immune responses to exercise in these patients may be abnormal. Adding the doctor’s caring to medical care affects the patient’s experience of treatment, reduces pain, and may affect outcome. This survey makes it clear that doctors continue to use placebos, and most think they help. We cannot afford to dispense with any treatment that works, even if we are not certain how it does.

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Competing interests: None declared.

who later developed chronic fatigue syndrome were not sedentary, although relying on self reports of chronic fatigue syndrome might have weakened predictions by including misdiagnoses.

If the finding was not a chance one, could being sedentary itself predispose people to chronic fatigue syndrome, or is being sedentary a marker for something else? We know that ambulant patients with chronic fatigue syndrome are at least as physically deconditioned as sedentary but healthy people, even though studies have excluded the most physically disabled patients because of the difficulty in measuring their fitness. Lack of fitness and inactivity consistently predicted the later development of chronic fatigue syndrome after infectious mononucleosis, suggesting that either premorbid or early deconditioning is a risk factor in some patients. Getting fitter is not necessary to feel better after a graded exercise programme, so is there another explanation?

Patients with chronic fatigue syndrome perceive activity as more of an effort than healthy controls and underestimate their cognitive and physical abilities, while being more aware of their internal physiological state, a phenomenon called interoception. How might this be related to being sedentary or having a disabling illness in childhood? Inactivity increases perception of effort with exercise, through both physiological deconditioning and the related cognitive, emotional, and sleep disturbance from being sedentary. This may enhance or sensitise interoception, perhaps in a similar way to that hypothesised in the related disorder of fibromyalgia. The corollary is that this enhanced bodily awareness or interoception may itself cause sedentary behaviour. When an appropriate trigger supervenes in later life, enhanced interoception may predispose some people to chronic fatigue syndrome. Trials of prevention are required to test this idea. Treatments that “reprogramme” interoception and increased activity, such as graded exercise therapy and cognitive behaviour therapy, seem to help most patients.

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Competing interests: PDW does consultancy work for the Department for Work and Pensions and Swiss Re, a re-insurance company.


Reforming the consultant contract again?

Issues related to financial incentives have changed little and need to be addressed

A fter protracted negotiations, the BMA and the consultant body voted in favour of a new contract, implemented with effect from April 2004. This contract applies to new practitioners and existing consultants who choose it. During negotiations, the Department of Health sought, but failed to obtain, professional support for a system of fee for service payments, for surgeons in particular. Despite this rejection, professional support among surgeons may now be emerging for the introduction of such a system.

The government’s NHS Plan requires considerable increases in medical and surgical activity. This can be achieved by changing the size of the medical workforce or by using incentives to stimulate existing doctors to produce more activity. Contracts of NHS hospital consultants include systems of reward and regulation, and both influence activity. Regulation has developed with the evolution of job plans and appraisal, and it will develop further with revalidation and more performance management by trusts. Reward systems and financial incentives, however, have changed little in the new consultant contract.

The current lack of transparent accountability of consultants is unlikely to be altered by the new contract. This concern, together with fundamental changes in the private healthcare market, may lead to a revision of the consultant contract. Consultants’ income from private practice is currently being undermined in terms of volume of private activity and the price paid for it. The government’s ambitious access targets, in reducing waiting times, could affect the volume of activity by reducing private purchase of health insurance, the attractiveness of company health insurance, and the number of people who pay out of pocket. Fees derived from private practice, currently higher in Britain than in Germany, the United States, and Australia, are also under threat. The government