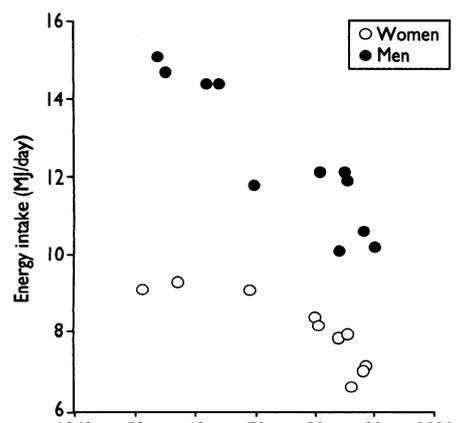


critical determinant of excess weight gain for some people. This is why we have emphasised negative proxies for activity (ownership of cars and labour saving devices, television viewing, etc). We dispute Morris's claim that television viewing is declining to any great extent. According to figures published by the Central Statistical Office, average viewing was 26 hours 32 minutes a week in 1986 and 26 hours 44 minutes in 1992.⁴ Over the same period the number of households owning a video recorder rose from 9 million to 17 million, which implies that secondary viewing will have risen sharply.

With respect to food intake, space constraints prevented us from presenting cross sectional data that corroborate the apparent fall in energy intake recorded by the national food survey. The figure shows a compilation of weighed dietary surveys of adults in Britain. Overall intakes are higher than that found in the national food survey, reflecting the fact that weighed surveys record all foods and beverages, but the downward trend is similar.



Compilation of cross sectional dietary surveys of adult energy intake in Britain (original citations are available on request)

Whatever the exact trajectory of energy intakes may be, it seems clear that per capita food intake has not increased and therefore that low levels of physical activity must be implicated in the current rising trend in obesity.

In our original paper we also presented an analysis of the social class trends in obesity, which again implicated physical inactivity. It is the combination of these different lines of evidence that we find particularly persuasive.

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Rising trend may be due to "pathoenvironment"

EDITOR.—The prevalence of obesity has reached epidemic proportions in the United States and many European countries. Andrew M Prentice and Susan A Jebb provide convincing evidence that "simple gluttony" cannot account for this increased prevalence since over the past 20 years food intake has decreased whereas body mass index has continued to increase in Britain.¹ The authors conclude that physical activity has decreased even faster over the same period and so

obesity can be blamed on "sloth." Many readers may incorrectly conclude from this study that obesity is simply the result of a lack of the willpower to control eating and exercise.

Epidemiological and genetic studies suggest, however, that obesity in a given population is largely determined by the environment, whereas the variability among individuals within a given environment is largely determined by the individual genetic responses to that environment.^{2,3} I therefore propose that the high prevalence of obesity in some environments is the consequence of normal, genetically determined physiology in a "pathoenvironment." Throughout most of its history, humankind has evolved in a restricted environment characterised by scarcity of food and a need for high levels of physical activity. Survivors of those times were probably people with a "thrifty genotype," which made them fatter during times of plenty so that during lean times they could survive on their own energy stores.⁴ More than 30 years after the proposal of the thrifty genotype hypothesis,⁵ however, we are still waiting for the identification of specific "survival" genes, which are likely to be common among many native populations with a high prevalence of obesity, such as Pima Indians, Australian Aborigines, and Pacific Islanders. Some people in Western societies may also have inherited stronger metabolic drives to eat more or exercise less than others and will, therefore, become obese in the present pathoenvironment.

To combat obesity on societal levels, public health strategies should be designed to make the environment less pathogenic by reducing the energy density of readily available food and increasing physical activity. These programmes should be targeted at children as early as the primary grades in school. Intervention programmes will not, however, be accepted until it is recognised that obesity is not simply due to the gluttony and sloth of undisciplined people but often results from genetically determined metabolic drives to eat more and exercise less in a pathoenvironment.

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Monitoring the safety of herbal remedies

Herbal remedies have a heterogeneous nature

EDITOR.—Herbal remedies are becoming increasingly popular with the public as they are perceived as being beneficial, free of side effects, and complementary to Western medicines.¹ There have been a number of reports of hepatic toxicity involving different herbal products.^{2,3} Guy Vautier and R C Spiller report fulminant hepatic failure due to a Chinese herbal remedy labelled "eternal life."⁴ We report a case related to the same remedy.

A 27 year old insulin dependent diabetic man presented with a two week history of progressive jaundice after consuming "eternal life." His peak bilirubin concentration was 458 $\mu\text{mol/l}$, alanine transferase 2230 IU/l (normal range <30), and

peak prothrombin time 19.3 s (normal range <15). A causal relation between the herbal tea and this patient's hepatitis was supported by the exclusion of viral, autoimmune, hereditary, and biliary diseases. Liver biopsy showed inflammation around the portal tract and piecemeal necrosis. The patient's clinical condition improved over the next four weeks. We sought advice about the constituents of our patient's tea from a local Chinese herbalist. Alarming, although the product is called eternal life, it does not always contain the same plant extracts. Apparently each prescription is tailor made to suit the patient's needs.

The heterogeneous nature of herbal products renders monitoring of adverse reactions difficult. We share Vautier and Spiller's concerns about herbal remedies. If we wish to have an effective national surveillance and licensing policy, however, we must first seek the cooperation of the practitioners of alternative medicine. It is only with their help that we can know accurately what the formulations of these herbal remedies are. To enforce legislation on them is likely to drive them underground.

Our case also shows the importance of asking specifically about the use of alternative medicines by patients who present with jaundice.

We thank Dr R C Read, senior lecturer in infectious diseases, for permission to report this case.

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WHO project is under way

EDITOR.—Guy Vautier and R C Spiller report a suspected severe hepatotoxic reaction to *Dictamnus dasycarpus*.¹ They make the important general suggestion that suspected reactions to herbal or natural medicines should be reported in the same way as are those to other drugs.

The World Health Organisation Collaborating Centre for International Drug Monitoring has received, from different national centres, over 5000 reports of suspected adverse reactions that have mentioned herbal medicines. This probably represents considerable underreporting, given the total size of the database of over 1.4 million reports related to conventional drugs.

In addition to the underreporting there is a great problem in categorising such products, which often have multiple ingredients of uncertain or variable amount or activity. Without the registration of herbal products the only recourse is to record each recognisable ingredient. We have a project under way to classify known, common toxic ingredients. The project is in an early phase but includes experts from around the world. The international programme on chemical safety has provided a forum for discussion so far, and additional funding is being sought by a consortium of poison control centres and experts in plant taxonomy from the Royal Botanical Gardens, London, and similar groups in other countries. Dr P A G M de Smet has provided much support for developing an extended anatomical (site of action) and therapeutic classification in conjunction with