The author has declared no conflicts of interest.

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Reply

First we thank Frederick Wolfe for seriously reading and criticizing our article from the outside of Plato’s cave [1]. Second, after reading his comment, it seems we have to make our statement more clear. Wolfe states that we said fibromyalgia does not exist and that we should give some proof for this statement (or rather: we should give proof, like he does, that fibromyalgia really does not exist). However, neither in the quote of Wolfe in our article (‘Wolfe’s assertion that fibromyalgia will always be with us like fibromyalgia. We may also be able to study how to prevent the diagnosis from being made.

Wolfe stated he has tried to give us some proof of the existence of fibromyalgia from the outside of Plato’s cave. First, by applying the criteria he stood in the middle of the cave. Second, do we still remember that there was supposed to be a supernatural world outside Plato’s cave? In reply to Wolfe’s last sentence: philosophers who are also clinicians know this. Is it that Wolfe does not?

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Significant proportions of our patients with a high cholesterol/HDL ratio were not taking statins, and these included five patients with IHD. More than 50% of patients had a high BMI (>25). It is debatable whether we should counsel all RA patients regarding cardiovascular risk factors and check their fasting lipids and glucose. It may be impossible to assess the risk factors in busy rheumatological out-patient clinics, but we could advise general practitioners to do this. There are no data available suggesting the threshold value for treating dyslipidaemia in RA patients. While hypertension and diabetes are commonly identified and treated, dyslipidaemia is frequently forgotten as an important risk factor. So the question unanswered is whether we should have a lower threshold for treating dyslipidaemia in RA patients.

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Reply

It is encouraging to see the growing interest of the rheumatological community in the prevention of cardiovascular disease (CVD) in rheumatoid arthritis (RA). The findings of Saravana et al. are not surprising. In a similar study of CVD risk factors in rheumatology out-patients, we found even higher levels of comorbid CVD than that detected by Saravana et al. (total cardiovascular comorbidity 34%; angina 16%, previous myocardial infarct 8%, cerebrovascular accident 4%, peripheral vascular disease 2%, treatment for hypertension 28%, and treatment for hypercholesterolaemia 4%). Of more concern is that in the remaining RA patients with no known CVD we also found a high prevalence of hypertension (systolic blood pressure >140 mmHg in 56.4%), hypercholesterolaemia (total cholesterol >5 mmol/l in 71.3%) and obesity (mean body mass index 27.6 kg/m²). Also, 22.3% were current smokers and 5.3% had diabetes mellitus [1]. These results are similar to those of other groups in the UK [2] and the USA [3].

We entirely agree with Saravana et al. that lipids and obesity are important factors in the development of CVD, but we believe that they should not be viewed or managed in isolation. We would advocate using a composite scoring system for the calculation of CVD risk to prevent undue emphasis being placed on any one risk factor to the detriment of others. There are several risk calculators based on the original Framingham data and modified over the ensuing years, as more accurate risk prediction models have been developed. In our clinics we use the Joint Societies Risk calculator [4], which is widely available at the back of the British National Formulary books and as an on-line version, and is rapid and easy to use in the clinical setting.

We recognize that implementing CVD risk assessment in busy rheumatology out-patient departments is difficult. However, we feel that at present this is by far the most appropriate setting. Even though evidence for increased cardiovascular mortality in RA has been around for a long time, awareness of it has started to become widespread amongst the rheumatological community only recently. It is likely (indeed this has been our experience locally) that awareness of this problem in primary care will lag even further behind. Once CVD risk assessment in RA is established in secondary care, and some peculiarities of it in the RA population have been sorted out through further research (e.g. the significance and interpretation of lipid levels during active inflammation), education and support for primary care physicians in undertaking this activity will be easier.

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Symptom concealment—a new phenomenon in patients treated with biological therapies?

Sir, The articles and discussion by Kroesen et al. [1] and Moots et al. [2] on the association of ‘infection in general’ with biological therapies raise many important issues for clinical rheumatologists. At St George’s NHS Trust we have treated 60 patients with infliximab, etanercept or adalimumab since 1999. Two patients have been admitted with severe infections, one with Haemophilus influenzae empyema and another with Pseudomonas pneumonia. Both patients were smokers but had had no previous history of symptomatic pulmonary disease or of significant chest sepsis. In both cases a delay of several weeks occurred between the onset of symptoms and admission to hospital. Part of this delay included a period of blind treatment by the general practitioner with antibiotics, without reference to the rheumatology department. However, in both cases a significant period of delay also occurred before the patient consulted the general practitioner, despite repeated warnings from our department to seek medical advice promptly should infective symptoms occur. We have ascertained that in these cases the patients consciously delayed seeking medical help, against advice, because they feared that this would lead to...