However, our patient differed in one intriguing aspect: she had an associated SS.

This association raises several issues. First, marked chronic immune activation (Table 1) and antibodies to histones with no anti-CD4 antibodies suggest CD4+ apoptosis as a possible pathogenetic mechanism. This is reminiscent of AIDS [5, 6], but no retrovirus could be found. Second, at least three viruses (HIV, HTLV-I and hepatitis C) are known to be associated with a SS-like syndrome [7]. These have been ruled out here, and HBV without HTLV-1 and hepatitis C are known to be associated with a SS-like pathogenetic mechanism. This is reminiscent of AIDS [5, 6], but no retrovirus could be found. Third, patients with unexplained CD4+ infection has not been previously reported. The triad of SS, severe CD4 lymphocytopenia and protracted immune activation with no evidence of retroviral infection has not been previously reported. Third, patients with unexplained CD4+ lymphocytopenia should be carefully evaluated for an occult SS.

Haematological or immunological presentations of SS already include a wide spectrum [2], including lymphoma. CD4 depletion can be an important underlying condition. Two studies identified a 5% prevalence of CD4 lymphocytopenia in SS, below the level used in the case definition of the CDC [8] and ~13% had anti-CD4 antibodies [9]. However, the two are not correlated, pointing to another aetiology, in agreement with our findings. In HIV, CD4+ T-cell depletion is more strongly correlated with markers of immune activation than with the viral load [6]. Perhaps our patient’s immune activation and the enhanced production of cytokines known to occur in SS [10] mediate apoptosis and T-cell depletion in some patients.

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From the outside of Plato’s cave

SIR, Hazemeijer and Rasker’s entertaining and erudite essay on fibromyalgia [1] contains many threads, but one thread, repeated several times and summarized as ‘...Wolfe’s assertion that fibromyalgia will always exist regardless of the name given to the syndrome … is unlikely to be true,’ stimulates me [Wolfe] to respond. I have been in the forefront of those who recognize that the fibromyalgia appellation has not served patients or society well [2–4]. But to say that it doesn’t exist should perhaps require proof by the authors rather than assertion.

So here is some proof that it does exist. In response to Hazemeijer and Rasker’s article, I studied 1000 randomly selected RA patients from the US National Data Bank for Rheumatic Diseases. To my knowledge, none had been diagnosed as having ‘fibromyalgia’. I then applied proposed survey fibromyalgia diagnostic criteria (regional pain score ≥8 and visual analogue fatigue ≥6) [5]. Seventeen per cent of RA patients satisfied these criteria. As shown in Table 1, the RA patients identified by survey fibromyalgia criteria had all of the characteristics found in patients who meet formal American College of Rheumatology (ACR) criteria: widespread pain and high levels of pain, fatigue and sleep disturbance. All rheumatologists who care for RA patients are aware of this group of patients, even though they do not apply the fibromyalgia diagnostic label—QED.

Those of us who played a part in the identification of fibromyalgia almost 3 decades ago did so because we, in caring for our patients, saw pain and distress that did not fit into the then, and now, prevalent model of organic disease. For a physician to identify fibromyalgia rationally is to mean that he or she is aware of the importance of biopsychosocial aspects of illness, for tender points are only a diagnostic shorthand. When we approach RA patients with ACR and disease activity score (DAS) criteria [6, 7] in randomized trials, we perforce ignore psychosocial issues. But in the clinic, we do this at our peril. Note in Table 1 that the difference in health assessment questionnaire (HAQ) scores between those with and without survey fibromyalgia is greater than that seen in clinical trials involving the best of the biological agents. Clinicians know this. Is it that philosophers do not?

Table 1. Fibromyalgia among patients with RA

<table>
<thead>
<tr>
<th>Variable</th>
<th>Fibromyalgia (+ve)</th>
<th>Fibromyalgia (−ve)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain (0–10)</td>
<td>6.9</td>
<td>3.3</td>
</tr>
<tr>
<td>Sleep disturbance (0–10)</td>
<td>6.5</td>
<td>3.3</td>
</tr>
<tr>
<td>Depression (ever) (%)</td>
<td>41.2</td>
<td>18.7</td>
</tr>
<tr>
<td>Depression (now) (%)</td>
<td>30.0</td>
<td>9.3</td>
</tr>
<tr>
<td>HAQ (0–3)</td>
<td>1.8</td>
<td>1.0</td>
</tr>
<tr>
<td>Lifetime co-morbidity score (0–11)</td>
<td>2.4</td>
<td>1.5</td>
</tr>
<tr>
<td>Symptom intensity (0–32)</td>
<td>14.3</td>
<td>6.1</td>
</tr>
<tr>
<td>Helplessness (5–25)</td>
<td>17.4</td>
<td>11.0</td>
</tr>
<tr>
<td>Low back pain (%)</td>
<td>70.0</td>
<td>41.2</td>
</tr>
<tr>
<td>Fatigue (0–10)</td>
<td>7.9</td>
<td>3.8</td>
</tr>
<tr>
<td>Regional pain score (0–19)</td>
<td>12.8</td>
<td>3.9</td>
</tr>
</tbody>
</table>
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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]. Secondly, 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 [...] regardless of what name the syndrome has, is unlikely to be true’), nor elsewhere in our article, do we say that fibromyalgia does not exist. There are specific people complaining of pain, sleeping disorders and fatigue who have been ‘diagnosed’ as fibromyalgia patients. We are neither denying the existence of these patients nor their complaints.

So, we also know that fibromyalgia exists, but not in the same way an object in Plato’s world of ideas is supposed to exist outside the cave. The form of representation called fibromyalgia must not be confused with a shadow of a Platonic idea. There is no such objective thing as fibromyalgia, in contrast to measurable inflammation factors or any other organic disease. What clinicians, by using classification criteria (although often called diagnostic criteria), recognize as fibromyalgia is a way of behaviour, a phenomenon, a representation of complaints changing in time. By applying ‘diagnostic’ criteria to a data bank Wolfe tries to prove the existence of fibromyalgia. But isn’t that similar to how we know fibromyalgia exists (and even in a way is produced)?

The proof of the existence of fibromyalgia is the proof of relationships in a therapeutic domain and the processes of assigning meaning. By only looking backwards and applying criteria on already ill diagnosed and classified people in a huge data bank, it is not possible to find the proof of the existence of certain processes and relationships. That is Whig history: explaining the past from the knowledge of the present.

In a prospective field study in a society in which the diagnosis of fibromyalgia does not exist, we may study which people will be going to complain of fatigue, pain and different somatic symptoms in the future. In such a setting we may be able to study the processes and relationships that lead to the diagnosis of a syndrome 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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Ischaemic heart disease in rheumatoid arthritis patients

Sir, We read with an interest the recent editorial by Kitas and Erb [1]. Patients with rheumatoid arthritis (RA) have a reduced life expectancy when compared with the general population. Recent studies showed that cardiovascular death is considered to be the leading cause of mortality in patients with RA [2]. It is thought to be due to accelerated atherosclerosis via persistent inflammation. Wallberg-Jonsson et al. [3] examined cardiovascular morbidity and mortality in a cohort of seropositive RA patients. They found that 34% of their cohort had a cardiovascular event during 15 yr of follow-up. Del Rincon et al. [4] showed that the incidence of cardiovascular events in RA was 3.43/100 patient-years vs 0.59/100 patient-years in controls. Risk factors for cardiovascular events are not properly addressed in our busy rheumatology outpatient clinics. So we did a small study to check traditional cardiovascular risk factors in RA patients in out-patient clinics. We recruited 98 successive patients from our rheumatology outpatient clinics. We gave particular importance to the fasting lipid profile and body mass index.

Twelve out of 98 patients (12.24%) had a personal history of ischaemic heart disease (IHD). Most of the patients were taking aspirin and anti-anginal medications. The lipid profile was analysed in the fasting blood sample. The mean cholesterol level was 5.3 mmol in patients with a history of IHD and 5.6 mmol in other patients. Forty-nine patients had a mean cholesterol/high-density lipoprotein (HDL) ratio of more than 4.44 mmol (upper limit of normal is 4.44 mmol), including five patients with IHD. Thirty-five patients with a cholesterol/HDL ratio of more than 4.44 were not taking statins, including two patients with a history of IHD. It is interesting to note that the mean body mass index (BMI) was 27.96, and 56 patients had a BMI of more than 25. Lipids and hypertension may relate to obesity and a sedentary lifestyle. These factors are now considered as major ischaemic risk factors.