The authors have declared no conflicts of interest.

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The use of a cannabis-based medicine (Sativex) in the treatment of pain caused by rheumatoid arthritis

SIR, We were interested to read the report by Blake et al. [1] in your journal.

Between 1998 and 2002 we surveyed a sample of 2969 people with various chronic diseases in the UK, with the aim of investigating the reasons for the medicinal use of cannabis [2]. In that survey, arthritis was reported to be among the five most common reasons for the medicinal use of cannabis, and we reported that of those who had reported using cannabis for the relief of their symptoms, 21% did so specifically for the relief of the symptoms of arthritis. The publication of your recent article prompted us into a review of the data collected, in order to determine what proportion of those respondents suffered from rheumatoid arthritis, and how their symptoms responded to the use of cannabis.

Of the 2969 respondents to the survey, 784 (26%) stated that they had ‘arthritis’. Of these, 247 patients indicated that they suffered from rheumatoid or osteoarthritis (the remainder did not specify). A total of 155 respondents stated that they continued to use illicit cannabis for the purpose of symptom relief. Around 111 of them (46%) had rheumatoid arthritis. Respondents were asked to indicate how their condition was affected by the use of cannabis. Of those patients with arthritis who responded to this question, 172 stated that it made them ‘much better’, 53 stated that it made them ‘a little better’ and five stated that it ‘made no difference’. None of the patients indicated that their arthritis was worsened by the use of cannabis.

Of those respondents who used illicit cannabis for rheumatoid arthritis, 100% indicated that they found it made them either ‘much better’ (72%) or ‘a little better’ (28%). We can conclude from this review of the original data, that the illicit use of cannabis by patients with rheumatoid arthritis is widespread, and that there is anecdotal evidence of effectiveness.

At that time, the authors pointed to the need for clinical studies of quality-controlled cannabis preparations to explore these conclusions in a more scientific and systematic way. We now note that the results of a formal randomized and controlled clinical study of the use of a medicinal grade of a cannabis extract validates this anecdotal evidence of the benefit of cannabinoids in the relief of the symptoms of rheumatoid arthritis. It will be of considerable interest to observe whether other conditions for which only anecdotal evidence currently exists may be validated in a similar manner in clinical trials of standardized cannabis preparations.

The authors have declared the conflicts of interest as G.G. is the Executive Chairman and founder of GW Pharmaceuticals. M.W. has participated in and received honoraria for CME articles from Bayer, Valeant and Solvay, and has received grants from GW and Valeant. S.W. is a full-time employee of GW.

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patients’ opinions, are of little or no value to anyone but tabloid journalists.

That no one said their arthritis worsened needs to be contrasted with side effect profiles from other studies of cannabinoids that are of concern and suggest caution.

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Obesity and cardiovascular risk factors in rheumatoid arthritis

SIR, We read with interest the review of Hall and Dalbeth [1] on the influence of disease-modifying drugs (DMARDs) on cardiovascular risk. The article covers the effects of pharmaceutical interventions, such as corticosteroids, TNF-α blockade and statins, and the influence of lifestyle variables, such as the Mediterranean diet. We believe that direct interventions on body mass index (BMI) might also have an important role to play. We recently assessed a range of cardiovascular risk factors including BMI, and measures of disease activity and impact in 100 rheumatoid arthritis (RA) patients (20 male, 80 female, median age 60 yrs (95% CI 57.0–62.0), median disease duration 12 yrs (95% CI 9.0–17.0) attending an out-patient clinic, and demonstrated strong associations between BMI and both hypercholesterolaemia and diastolic blood pressure.

Median BMI was 27.0 (95% CI 26.0–28.0). Of the patients, 68% were overweight (BMI > 25) and 31% obese (BMI > 30). There was a positive correlation between BMI and diastolic blood pressure ($R = 0.37$, $P < 0.01$), a positive correlation between BMI and cholesterol:HDL ratio ($R = 0.36$, $P < 0.01$), and a negative correlation between BMI and HDL ($R = 0.31$, $P < 0.01$). There was no association between BMI and daily exercise or current steroid use. Overall, 40% of the patients had a diastolic blood pressure $> 80$ mmHg, and 18% had $> 90$ mmHg. We, established that 54% of the patients had a fasting cholesterol $> 5.2$ mmol/l, and the median fasting cholesterol was 5.27 (95% CI 5.05–5.50).

It has been shown that even modest reductions in BMI can have positive implications for cardiovascular health [2]. Moreover, in addition to dietary advice, a range of pharmaceutical interventions is now available for the treatment of obesity. We agree with the authors that RA ought to be treated as an additional risk factor when calculating 10-yr risk of a CHD-event; if this is the case, then should it, for example, qualify as a comorbidity as defined by the NICE guidelines [3] for the use of the weight-reducing drug orlistat?

Rheumatologists should be instrumental in leading intervention on cardiovascular risk factors in RA, as discussed by Hall and Dalbeth [1]. We believe that direct intervention on obesity, which is common in our RA population, offers further opportunities to modify associated risk factors, such as hypertension and hypercholesterolaemia.

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Re: Obesity and cardiovascular risk factors in rheumatoid arthritis

SIR, Armstrong and colleagues raise the important issue of optimizing body mass index (BMI) in patients with rheumatoid arthritis (RA), as part of a cardiovascular risk reduction strategy. They include some interesting data from a local RA cohort, which revealed that 68% patients had a BMI $> 25$ and 31% had a BMI $> 30$. They also report positive correlations in this cohort between BMI and both diastolic blood pressure and cholesterol: high-density lipoprotein cholesterol ratio, both of which are established risk factors for cardiovascular disease. Since population data suggest that a modest reduction in BMI is associated with a substantial reduction of cardiovascular morbidity, Armstrong and colleagues advocate that rheumatologists adopt a more aggressive approach to overweight and obesity in RA patients. They suggest that this include prescription of orlistat, which reduces the absorption of dietary fat.

In the general population, obesity is clearly associated with increased mortality [1, 2], and emerging evidence links obesity and inflammation [3]. It is intriguing to speculate that the optimization of BMI may have beneficial effects on disease activity in chronic inflammatory diseases, such as RA. We, therefore, have sympathy with the proposal of Armstrong and colleagues and have included in our algorithms the objective of achieving a BMI $< 25$ in patients with RA in all risk groups. A potential problem with this recommendation is the lack of studies on the relationship between BMI and cardiovascular risk, specifically in the rheumatoid population. Indeed, Escalante and co-workers [4] have shown an inverse relationship between mortality in RA and BMI. This relationship disappears when adjustment for RA disease activity is made, and it therefore seems likely that the adverse association with low BMI in RA patients reflects the cachexia associated with severe systemic disease. Furthermore, the adverse effect of a low BMI mainly reflects the effect of having a BMI $< 20$. Based on current evidence, we believe that it is sensible to recommend aiming for a BMI $< 25$, but only in RA patients with well-controlled disease. Similarly, we think that orlistat would be a useful therapeutic adjunct in patients with a BMI $> 30$, provided their disease is well-controlled, and we agree with Armstrong and colleagues that it would be worthwhile including controlled RA to the list of comorbidities in the NICE guidelines [5] for the use of orlistat. An additional consideration is that the use of orlistat may reduce the absorption of fat-soluble vitamins, including vitamin D. Since patients with RA are at an increased risk of osteoporosis, it is important to ensure that they do not become osteomalacic. Monitoring of vitamin D at 3–6 monthly intervals would therefore be a sensible precaution in RA patients treated with orlistat. If calcium/vitamin D supplements are

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