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The fibromyalgia problem. A Latin American point of view: reply

We thank Dr Caballero-Uribe for seriously reading and criticizing our article from inside the therapeutic domain [1]. His comment will certainly give the therapeutic domain more depth, and will be of added value for good medical practice; the same applies to the comments of others, including Wolfe [2], Procee [3] and Harth and Nielsen [4], and our replies [5, 6].

Dr Caballero-Uribe, in stating that ‘fibromyalgia still is a poorly recognized entity’, presupposes that fibromyalgia exists, now and forever, but has sometimes been hidden or overlooked by most doctors. We warned against this suggestion in our article: ‘Syndromes like fibromyalgia are thus not waiting below the surface until they are discovered by a researcher or brought to light by the medical gaze’ [1]. The fact that 7% of the Amish community fit the ACR criteria for fibromyalgia proves only that we can make many persons ill by labelling them with this diagnosis.

In philosophy it is called a ‘whig history’ if one presupposes that something (in this case fibromyalgia) exists and is caused by factors that can be described and explained. This is a positivistic point of view, a circular argument beginning with what is supposed to be evident in the present and explaining it from past events [1, 7]. We postulated the use of another form of discourse to discuss fibromyalgia syndrome: the discourse of aesthetic representation [1]. The rise of fibromyalgia is a process in which meanings arise and change, and doctors and patients repetitively assign symptoms and complaints to other events. Fibromyalgia as a representation is as real as the real difference between a painted portrait and the person who is portrayed.

We agree with Ehrlich that everybody has pain sometimes, and perhaps even chronic pain, during a lifetime. In Western cities, fibromyalgia tends to be diagnosed when no other reason is found for the pain. No one has fibromyalgia until it is diagnosed [8]. Patients diagnosed as having fibromyalgia are probably vulnerable cases whose behaviour is facilitated by the therapeutic domain.

We want to make clear that we never stated that people diagnosed as having fibromyalgia do not exist or would not suffer. We never described fibromyalgia as a ‘mind disease with rheumatic complaints’, as quoted wrongly by Dr Caballero-Uribe. These people—we cannot repeat it often enough—are real patients and do have serious complaints, but the form of appearance of their complaints changes during their medical history (as well as during the history as studied by historians) parallel with a changing therapeutic domain or social setting. In our review we mentioned as examples of rising and declining disorders not only fibromyalgia syndrome, but also railway spine, the rise and fall of the Australian epidemic of repetitive strain injury around 1986 and the arc de cercle of classical hysteria, which was widespread around 1900 but nowadays is never seen.

Dr Caballero-Uribe suggests that widespread pain, chronic pain and fibromyalgia are identical. There is no proof for this statement. Fibromyalgia is what the (ACR) criteria say it has to be, and it came into being styled by just these criteria. We will explain this.

Neurohormonal alterations and substance P levels in cerebrospinal fluid cannot be candidates for markers of the existence of fibromyalgia or candidate disease markers, as Dr Caballero-Uribe suggests. Some investigations found neurohormonal alterations in patients who were diagnosed (we would say ‘classified’) as fibromyalgia patients [9]. Dr Caballero-Uribe, like many participants in this debate, uses a circular argument. No one knows for sure whether the alterations appeared before or after the person was classified as having fibromyalgia syndrome. Recently we argued, using a theory of Kandel [10], how alterations in neurohormonal levels and the origin of fibromyalgia can be explained [6]. When a doctor is talking to a patient about fibromyalgia, they are both acting in a therapeutic domain. It is known that psychotherapy, a more or less structured way of talking with a patient, induces the same biochemical effects as psychopharmacological treatment [10]. The theory to explain this is that every thought and behaviour is basically an epiphenomenon of gene expression. Altered thoughts, beliefs and behaviour are connected with alterations in gene expression. The latter means altered production of proteins, neurotransmitters, hormones, and so on. The origin of fibromyalgia can be understood as the same kind of process as that which we have just explained. Acting with chronic pain in a therapeutic domain may induce changing behaviour and altered gene expression, and so give rise to alterations in, for example, substance P levels in the cerebrospinal fluid [6].

We still do not know what pain is [11]. We do not know how pain behaviour is explained. Why is pain perpetuating in some but increasing in others? It remains uncertain whether the (dys)functioning of the nociceptive system in these patients is the cause or the result of what is called fibromyalgia syndrome [12].

We have difficulty in the use of an algorithm for the treatment of a disorder that by definition has no objective abnormalities and that is mostly not curable.

We believe that by making use, in such an algorithm, of tender point scores (which have been proved to be unreliable), the Fibromyalgia Impact Questionnaire (rather a functional index than a psychological index) the proposed treatment will intensify the belief of fibromyalgia syndrome victims that they are really very ill, making further referrals even more necessary in their opinion. We know that the long-term effect of medication in FMS patients is very disappointing, if it has any effect at all. What further hope can an interested rheumatologist give after making his victims ill?

Dr Caballero-Uribe’s letter illustrates one of the fibromyalgia syndrome-producing principles of a therapeutic domain. He presupposes that fibromyalgia is a real disease, hidden below the clinical surface, unseen by ignorant professionals other than rheumatologists. From the point of view of the sociology of knowledge, it is clear that fibromyalgia would never have existed if it had not been invented, or diagnosed. Doctors and society are each responsible for causing or prolonging the misery of the patients with fibromyalgia syndrome [1]. It would be wise to stop using the ACR criteria in the clinic [13]. At the very least, doctors in all clinical specialities must be wary of causing harm by unwarranted investigations and treatments [14]. For the prevention and treatment of fibromyalgia, we have to start by fundamentally changing the therapeutic domain. In such a renewed setting, fibromyalgia would not become manifest in an individual, and thus fibromyalgia would no longer exist [1].
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Comment on review on T cells in bone biology

Sir, A recent review on T-cell involvement was published in Rheumatology [1] and the authors are to be congratulated on their paper. We have published two papers relevant to this topic recently [2, 3]. One paper examined the expression of RANKL protein in synovial tissue from patients with rheumatoid arthritis (RA), who had both active and inactive disease, as well as patients with a seronegative spondyloarthropathy, osteoarthritis (OA) and normal subjects. We clearly demonstrated in this paper that RANKL was expressed to a similar extent in synovial membranes from patients with active RA and seronegative spondyloarthropathy, with much less RANKL expression in inactive RA and OA synovial membranes, which was similar to that seen in normal synovial tissue [2]. In addition, we demonstrated by dual immunohistochemistry that the CD45Ro-positive T lymphocyte was the major cell expressing RANKL at the protein level, with around 40% of macrophages also expressing RANKL but few if any type B lining cells (fibroblast lineage) expressing RANKL. In a companion paper [3], we demonstrated that OPG was not seen in active RA synovial tissue, unlike the extensive expression of OPG in patients with seronegative spondyloarthropathy, OA and normal synovial tissue. We also demonstrated that synovial tissue from patients with inactive RA had extensive OPG expression and that this expression was predominantly on type A lining cells (macrophage lineage) and endothelial cells. We raised the possibility that OPG expression in RA may be a major determinant of osteoclast formation and bone erosion and therefore an attractive therapeutic target in RA.

These two papers were published in December 2002 [2] and January 2003 [3], well before the review [1] was submitted for publication, so it is surprising and rather disappointing that the authors did not feel that these papers were relevant enough to be included in a review of the subject which contained 121 references.

For those readers of this journal who are interested in this subject, we have also published on RANKL and OPG expression in tissue from patients with other bone resorbing conditions, periodontal disease [4] and peri-implant bone loss and failure [5].

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Comment on review on T cells in bone biology: reply

Sir, We have read with interest the comments from Professor Smith following the publication of our review on T-cell interactions with the osteoclast [1]. In reviewing this topic, we concentrated on describing the current state of knowledge in this field and on presenting research from areas not normally read by the rheumatologist. Unfortunately, it was not feasible to catalogue all the work that has been carried out in this area. Papers were typically cited when reporting the earliest description of a new finding, where the work resulted in the development of a new