

## Letter

# Is fibromyalgia a cardiovascular disease? A comment on Martinez-Lavin's review 'Stress, the stress response system, and fibromyalgia'

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See related review article by Martinez-Lavin, <http://arthritis-research.com/content/9/4/216>

Fibromyalgia syndrome (FMS) is a mysterious condition with uncertain physiopathogenesis. Despite its hermeneutic problems, FMS and related afflictions are responsible for the majority of family medicine and rheumatological medical office appointments. In spite of the skepticism of some, it seems that the medical science community has accepted FMS as a valid, unique clinical diagnosis and the main theory explaining it (the central sensitization model (CSM)) may relate to many chronic pain states.

The CSM actually labels not a single unified hypothesis but, rather, a group of evidence-based central nervous system (CNS)-centered physiopathogenesis hypotheses for FMS. It constitutes the only model capable of explaining the majority of symptoms of FMS and predicts that CNS-acting agents are the most effective drugs in its treatment. Despite its success, many questions still need clarifying. What is the exact nature of the CNS alteration that is expressed as central sensitization? Why does it occur? What are its causes? And are there any genetic alterations that predispose to it?

Martinez-Lavin's review on FMS and dysautonomia [1] proposes a model that could explain FMS in terms of a sympathetically maintained neuropathic pain syndrome. It has the virtue of explaining the possible mechanisms behind CSM. Besides its conceptual and experimental gaps, we must not discard it without meticulous scrutiny through experimentation. The effort to get closer to a mechanistic, complete model of the CSM and FMS will certainly be rewarding since the need for a theoretical framework is critical in the study of FMS and other related chronic pain states.

We recently reported our own FMS causation model based on the experimental findings of Martinez-Lavin and others [2].

Briefly, we noted that there is probably a close relationship between FMS and related diseases and cardiovascular alterations, especially dysautonomia. The pattern, as noted also by Martinez-Lavin, involves not only sympathetic basal overactivity, but also sympathetic blunted responses to stimuli such as orthostatic challenge. Stewart and colleagues [3] have published many articles on orthostatic intolerance and its relationship to chronic fatigue syndrome, a disease closely related to FMS. His team and others think that dysautonomic alterations in orthostatic intolerance and chronic fatigue syndrome (described as similar to the findings associated with FMS) stem from microvascular reactivity alterations that lead to venous pooling in dependent extremities. Their main concept is of a 'relative thoracic hypovolemia' that determines secondary dysautonomia in orthostatic intolerance and probably chronic fatigue syndrome. Based on these and other findings, we proposed a cardiovascular FMS model, relating cardiovascular and nociception neural modulation as a possible cause of the CSM.

Do the experimental data as reviewed by Martinez-Lavin provide support for a cardiovascular model of FMS and related diseases? It may be so, but we need more experimentation to try these new ideas. Recent publications suggest that FMS patients more frequently have metabolic syndrome, and a hyperadrenergic state with blunted epinephrine secretion [4]. In addition, elevated body mass index correlated with secondary FMS [5]. Finally, neuroimaging techniques are disclosing a possible relationship between FMS and neurodegeneration [6]. Could it be that FMS patients have a neurovascular disease associated with CSM? Only time and more knowledge will tell.

## Competing interests

The authors cite their own work on the subject that is currently in press. There are no potential sources of conflicts of interest.

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