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Editorial

Muscle Failure and Immune Dysfunction in Patients with Chronic Fatigue Syndrome: The Chicken or the Egg Dilemma

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Editorial

Despite a renewed interest from the scientific community, Chronic Fatigue Syndrome (CFS), also known as Systemic Exertion Intolerance Disease (SEID), remains an unsolved medical condition [1]. Many efforts have been done to investigate possible causes of CFS among various hypotheses including infectious, endocrine, psychiatric, metabolic and immune studies [1,2]. In the last decade, although available evidence has been often conflicting, the general idea that an immune dysfunction could be the primary cause of CFS has gained weight in the scientific and patients community [1,3]. Unfortunately, to date, all the prospective controlled trials using immune-modulatory approaches have been negative, including two recent studies targeting interleukin IL-1 and B lymphocytes [1,4]. These disappointing results should give us the opportunity to rethink the currently accepted model where an immune dysfunction would precede the occurrence of CFS manifestations. Indeed, CFS various clinical symptoms are exacerbated by physical activity, defining the post-exertional malaise included in diagnostic criteria [5]. Also, even though the observed benefits do not concern all CFS patients, exercise therapy has shown positive results in controlled randomized trials [6,7]. Then, from a research perspective, we should not forget that a muscle failure has been demonstrated in CFS patients [8,9]. CFS patients are characterized by an impaired muscle conduction velocity assessed by the reduced amplitude and lengthened duration of the evoked muscle potential (M wave) during and after exercise [8]. Also, in most CFS patients, M wave alterations are associated with an accentuated oxidative stress in response to exercise [8,10] that is responsible for the formation of lipid hydro peroxides impairing the muscle membrane excitability [11]. More, heat shock protein (Hsp) 27 and Hsp70 formation is markedly reduced in CFS patients [12]. In contracting muscles of healthy individuals, the formation of Hsp protects the muscular cells against the deleterious effects of reactive oxygen species [13]. Hsp also promotes the IL-6 production which in turn elicits the release of anti-inflammatory cytokines (IL-1Ra, IL-10) [14]. Animal studies have shown that neural information from a fatiguing muscle triggers the Hsp production in non-contracting muscles and also in other organs including the brain [15]. Thus, the dysregulation of Hsp production could explain the absence of an adapted anti-inflammatory response in the exercising muscle, as well as the systemic characteristics of CFS. To conclude, we wish to raise the point that the interplay between muscle and the immune system is probably more complex than previously thought. Much evidence suggests that the observed muscle failure in CFS patients may not be the expression of a primary systemic immune dysregulation. Instead, especially in CFS patients for which the disease was preceded by intense sports practice or severe acute infection, and in which we observed the lowest level of Hsp [12,16], the muscle failure could be at the origin of CFS manifestations, including a possible impact on the immune status. So, after the recent failure of promising therapeutic strategies targeting the immune system, it seems that the chicken or the egg debate is more open than ever and that sufficient clues lead to reconsider the place of muscle in the CFS research field.

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