THE INVOLVEMENT OF AUTONOMIC NERVOUS SYSTEM AND INFLAMMATORY MECHANISMS IN CHRONIC FATIGUE: PERSPECTIVES FOR FUTURE STUDIES

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We read with interest the well-designed study performed by Kristiansen *et al.* (2019) and take this opportunity to commend the authors. This is since autonomic dysfunction has now been shown to be related to several disorders.

As an important conclusion, the authors reported only minor sympathetic predominance in chronic fatigue, which may partially explain increased serum C-Reactive Protein levels. This now allows us to consider some issues for future studies:

1. The parasympathetic anti-inflammatory pathway has been previously described (Borovikova *et al.*, 2000). More recently, Abe *et al.* (2017) reported that C1 neurons, which are involved in sympathetic regulation; mediate anti-inflammatory reflexes induced by stress in animals. Thus, we propose the following question: Would not the clinical interactions between sympathetic or parasympathetic actions and the inflammatory processes affect the chronic fatigue symptoms?

2. Similarly, non-invasive parasympathetic activation was revealed to improve inflammatory markers (Kong *et al*., 2018). So, is it logical to investigate this treatment for these chronic fatigue symptoms?

The purpose of this cross-examination is to achieve a better understanding of the physiological roles of interactions between autonomic and inflammatory mechanisms.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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