Dangerous exercise. The detrimental effects of exertion and orthostatic stress in Myalgic Encephalomyelitis and chronic fatigue syndrome

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Exercise, especially regular exercise, is known to have various beneficial effects on the health status [1,2]. However when immunological, stress and other responses to physical activity are dysregulated, exercise can become detrimental to health [3].

Looking at the evidence so far, this latter observations seems also to be applicable to Myalgic Encephalomyelitis (ME) and chronic fatigue syndrome (CFS). Although ME and CFS are often declared to be synonyms [4], the diagnostic criteria define two distinct clinical entities. ME is primarily defined by muscle weakness (and myalgia) after a minor exertion lasting for days and characteristic neurological features, while CFS is primarily defined by (unexplained, incapacitating) chronic fatigue [4]. Although ME and CFS are to be considered to be two distinct, partially overlapping, diagnostic labels [4], the Institute of Medicine [5] confirmed that post-exertional “malaise”, defined as a prolonged aggravation of typical symptoms (e.g. “brain fog”/cognitive deficits and muscle and joint pain) is the hallmark feature of “ME/CFS”.

Despite this methodologic hurdle, i.e. research into mixed ME and/or CFS patient populations, various characteristic aberrations have been found repetitively. Exercise-related abnormalities [6] are associated with a) energetic abnormalities, which seem to be amplified by exertion, including mitochondrial dysfunction [7-10], (very) low oxygen uptake [11-13], reduced oxygen supply to muscles [14] and brain [15], and excessive acidosis and recovery from exercise-induced acid [13,16,17]; b) muscular abnormalities related to exercise [18-20]; c) increased pain sensitivity and lower pain threshold induced by exercise [21-23]; d) long-lasting oxidative stress in response to exercise [19,21,24]; e) cardiovascular abnormalities [25-27]; f) deviant autonomic responses to exertion and orthostatic stress [28-30]; g) neurological abnormalities in relation to exercise [31-33]; h) immunologic abnormalities after exertion [32,34,35]; i) exercise-induced gastro-intestinal abnormalities, including bacterial translocation [36-38]; j) attenuated stress responses to exercise [39,40]; and k) ion channel dysfunction during rest and in response to exercise [41-43]. These abnormalities can plausibly explain post-exertional “malaise” in ME/CFS [44].

Physical deconditioning is not a perpetuating factor in ME/CFS [45]. Deconditioning also cannot account for the aberrations observed, especially since various studies have used sedentary controls, e.g. [11,13,46]. Deconditioning also cannot explain the profound fall in exercise capacity (maximum oxygen uptake/workload and/or oxygen uptake/workload at the anaerobic threshold) at a second exercise test and the inability of ME/CFS patients to reproduce VO2peak 24 hours later [47-49].

Interestingly, recent studies observed a hypometabolic state [50,51], which was characterized by one study as ‘dauer’ [52]. Although male and female patients seem to compensate differently, these studies show ‘inadequate ATP generation by oxidative phosphorylation and excessive lactate generation upon exertion’ [53].

So, while the deconditioning hypothesis seems tempting, graded exercise protocols seem to have detrimental effects on many patients [54]. In the case of ME/CFS it is wise to acknowledge the potential dangers of exercise to avoid iatrogenic harm.

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