

**Skeletal muscle metabolism in the chronic fatigue syndrome. In vivo assessment by 31P nuclear magnetic resonance spectroscopy.**

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**BACKGROUND:** Previous study of patients with chronic fatigue syndrome (CFS) has demonstrated a markedly reduced dynamic exercise capacity, not limited by cardiac performance and in the absence of clinical neuromuscular dysfunction, suggesting the possibility of a subclinical defect of skeletal muscle. **METHODS:** The in vivo metabolism of the gastrocnemius muscles of 22 CFS patients and 21 normal control subjects was compared during rest, graded dynamic exercise to exhaustion and recovery, using 31P nuclear magnetic resonance (NMR) spectroscopy to reflect minute-to-minute intracellular high-energy phosphate metabolism. **RESULTS:** Duration of exercise was markedly shorter in the CFS patients (8.1 +/- 2.8 min) compared with the normal subjects (11.3 +/- 4.3 min) ( $p = 0.005$ ). There were large changes in phosphocreatine (PCr), inorganic phosphate (Pi), and pH from rest to clinical fatigue in all subjects, reflecting the high intensity of the exercise. The temporal metabolic patterns were qualitatively similar in the CFS patients and normal subjects. There were early and continuous changes in PCr and Pi that peaked at the point of fatigue and rapidly reversed after exercise. In contrast, pH was relatively static in early exercise, not declining noticeably until 50 percent of total exercise duration was achieved, and reaching a nadir at 2 min postexercise, before rapidly reversing. There were no differences in pH at rest (7.08 +/- 0.04 vs 7.10 +/- 0.04), exhaustion (6.85 +/- 0.17 vs 6.76 +/- 0.17) or early (6.64 +/- 0.25 vs 6.56 +/- 0.24) or late recovery (7.09 +/- 0.04 vs 7.10 +/- 0.05), CFS patients vs normal subjects, respectively (NS). Neither were there intergroup differences (NS) in PCr or Pi. Although, quantitatively, the changes in PCr, Pi, and pH were marked and similar in both groups from rest to exhaustion, the changes all occurred much more rapidly in the CFS patients. Moreover, adenosine triphosphate (ATP) was significantly ( $p = 0.007$ ) less at exhaustion in the CFS group. **CONCLUSIONS:** Patients with CFS and normal control subjects have similar skeletal muscle metabolic patterns during dynamic exercise and reach similar clinical and metabolic end points. However, CFS patients reach exhaustion much more rapidly than normal subjects, at which point they also have relatively reduced intracellular concentrations of ATP. These data suggest a defect of oxidative metabolism with a resultant acceleration of glycolysis in the working skeletal muscles of CFS patients. This metabolic defect may contribute to the reduced physical endurance of CFS patients. Its etiology is unknown. Whether CFS patients' overwhelming tiredness at rest has a similar metabolic pathophysiology or etiology also remains unknown.

PMID: 1446478 [PubMed - indexed for MEDLINE]