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Letter to the Editor

Is Physical Training Beneficial In Patients Harboring Mtdna Deletions?

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We read with interest the excellent article by Taivassalo et al., (2006) on the effect of endurance training and detraining in eight patients with chronic external ophthalmoplegia (CPEO) due to large-scale mtDNA deletions (Taivassalo, T. et al., 2006). After 14 respectively 28 weeks of a standardized training a beneficial effect on physiological outcome and quality of life was observed without a change in mutation loads or mtDNA copy number. We want to add the following comments:

Obviously, patient #1 became severely handicapped after an accident at onset of the detraining period. The authors admit that her results significantly influenced some of the outcome variables (Taivassalo, T. et al., 2006). Why was this patient not excluded from the study or at least from statistical analysis?

The study group was homogenous for mtDNA deletions, for CPEO, for exercise intolerance, but not for muscle weakness, reported in two patients. Was involvement of the limb muscles also confirmed by elevated creatine-kinase, or electromyography? The authors mention that there was considerable difference in the severity of the clinical histochemical and biochemical abnormalities between the patients. How much contributed this variability to the results?

In the early stages of the disease CPEO is confined to the periocular muscles, but may, as most mitochondrial disorders, turn into multi-system disorder with disease progression (Zeviani, M., & Di Donato, S. 2004). Was exercise intolerance due to limb muscle involvement or due to cerebral involvement? Were imaging studies of the cerebrum carried out to assess a possible cerebral origin of exercise intolerance?

Training obviously reduced serum lactate levels at rest and during exercise, being explained by the oxidative phosphorylation improved from training (Taivassalo, T. et al., 2006). How to exclude that the lactate lowering effect was due to improved lactate clearance in the liver?

To expect a change in the rate of heteroplasmy already after 28 weeks of training does not take into account that mutation loads increase only slowly with age (Szibor, M., & Holtz, J. 2003) and that an increase in the mutation load after long-term exercise has not been reported.

Particularly, after immobilization due to hospitalization or previous surgery, patients with mitochondrial disorders frequently report that detraining results in loss of physiological adaptation. Such patients often complain about significant deterioration of their physical abilities, in case their regular demand of exercise had not been addressed. Most patients report improvement in psychological well-being and physical fitness if they regularly perform exercise without reaching or exceeding their individual limit of capacity.

Most patients with mitochondrial disorders at least temporarily, take vitamins, antioxidants, L-carnitin, creatine, L-arginine, or hormones (Finsterer, J. 2004). For cardiac involvement they may require cardiac medication and for seizures antiepileptics. Under which regular medication were the included patients?

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How to explain that the amount of increase from baseline was equal to the decrease from the trained state concerning peak work capacity, peak oxygen utilization, and peak oxygen extraction? Is this an incidental finding?

The showiest clinical feature of CPEO is ptosis and ophthalmoparesis. Were these clinical manifestations influenced in any way by the 28 weeks of training?

Though we still cannot convincingly recommend exercise training to patients with mitochondrial disorders, because of lacking long-term studies and the individuality of mtDNA mutations, there is increasing evidence that exercise training could become a promising therapeutic option for these patients. As long as physicians can neither recommend nor disapprove such therapy, patients themselves must find out, which is the level of physical activity most beneficial with regard to their individual maximal physical abilities.

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