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Delayed central fatigue after fatiguing submaximal exercise in adaptation to endurance training

Firas Zghal[1], F Cottin[1,2], I Kenoun[3], H Rebai[3], W Moalla[3], M Dogui[4], Z Tabka[5] and V Martin[1,6]
[1]University of Evry Val d'Essonne, France
[2]University of Paris-Sud, France
[3]EM2S Research Unit, Sfax, Tunisia
[4]Sahloul University Hospital, Tunisia
[5]Sousse University, Tunisia
[6]Blaise Pascal University, France

Purpose: The purposes of this study were to evaluate the effect of endurance training on central fatigue development and recovery.

Methods: A control group was compared to a training group which followed an 8-week endurance-training program consisting in low-force concentric and isometric contractions. Before (Pre) and after (Post) the training period, neuromuscular function of the knee extensor (KE) muscles was evaluated before, immediately after and during 33 minutes after an exhausting submaximal isometric task at 15% of the maximal voluntary contraction (MVC) force. After training, the trained group performed another test at iso-time, i.e., with the task maintained until the duration completed before training was matched (Post 2). The evaluation of neuromuscular function consisted in the determination of the voluntary activation level during MVCs from peripheral nerve electrical (VAPNS) and transcranial magnetic stimulations (VATMS). The amplitude of the potentiated twitch (Pt), the evoked (motor evoked potentials, cortical silent period (CSP)) and voluntary EMG activities were also recorded on the KE muscles.

Results: Before training, the isometric task induced significant reductions of VAPNS, VATMS and Pt and an increased CSP. The training period induced a 3-fold increase of exercise duration, delayed central fatigue appearance as illustrated by the absence of modification of VAPNS, VATMS and CSP after Post 2. At Post, central fatigue magnitude and recovery were not modified but Pt reduction was greater.

Conclusion: These results suggest that central fatigue partially adapts to endurance training. This adaptation principally translates into improved tolerance of peripheral fatigue by the central nervous system.\

zghal.firas2012@yahoo.fr

Genetic and developmental perspective of language abnormality in Autism and Schizophrenia: One disease occurring at different ages in humans?

Haoran George Wang
University of Toronto, Canada

Language and communication through it are two of the defining features of normally developed human beings. However, both these functions are often impaired in Autism and Schizophrenia. In the former disorder, the problem usually emerges in early childhood (~2 years old) and typically includes a lack of communication. In the latter condition, the language problems usually occur in adolescence and adulthood and presents as disorganized speech. What are the fundamental mechanisms underlying these two disorders? Is there a shared genetic basis? Are the traditional beliefs about them true? Are there any common strategies for their prevention and management? To answer these questions, we searched PubMed by using Autism, Schizophrenia, gene, and language abnormality as keywords, and we reconsidered the basic concepts about these two diseases or syndromes. We found many functional genes, for example, FOXP2, COMT, GABRB3, and DISC1, are actually implicated in both of them. After observing the symptoms, genetic correlates, and temporal progression of these two disorders as well as their relationships more carefully, we now infer that the occurrence of these two diseases is likely developmentally regulated via interaction between the genome and the environment. Furthermore, we propose a unified view of autism and schizophrenia: a single age-dependently occurred disease that is newly named as Systemic Integral Disorder, if occurring in children before age 2, it is called Autism; if in adolescence or a later age, it is called Schizophrenia.

ghrwang@gmail.com