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**INTEREST OF A COMPARATIVE PROTEOMIC APPROACH TO UNRAVEL THE AETIOLOGY OF DOMS IN HUMANS**
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Intense or unusual physical muscular exercise frequently leads to muscle soreness that appear after some delay which is associated with histological lesions and which is known as Delayed Onset Muscle Soreness or DOMS. We examined the muscle proteome to understand the molecular and cellular consequences of a strenuous eccentric physical exercise and of a specific training aimed to prevent DOMS. Six healthy male subjects were submitted to three needle biopsies in the vastus intermedius muscle in three conditions: (1) at rest (control), (2) 24 hours after a provocation session of 3 sets of 30 maximal eccentric contractions of the quadriceps on an isokinetic dynamometer, (3) 24 hours after a similar eccentric session following 5 sessions of isokinetic submaximal eccentric training. The DOMS were assessed before and 1 day after both provocations by three indirect markers of muscle damages: the plasma level of creatine kinase (CK), the muscle tension and the subjective presence and intensity of pain evaluated using a visual analogue scale. The first provocation induced a great elevation of the CK (11484 17195.1 UI/l), a decrease of the quadriceps muscle extensibility and subjective pain (5.78 1.2 a.u.). After the second provocation, the symptoms of DOMS were reduced confirming the protective effect of a specific submaximal training. The 18 muscles biopsies were subjected to a 2D-DIGE analysis coupled with the MALDI-MS-MS protein identification. We observed a statistical variation of abundance of 174 proteins and identified 129 spots corresponding to 40 different proteins. The majority of them were contractile and metabolic proteins. Surprisingly, we observed that three contractile proteins, which already decreased after the first provocation, are decreased at an even lower level together with other contractile proteins after the second provocation. This observation suggests that the DOMS are not the consequence of a decrease of the contractile proteins expression. Furthermore, our results showed a decrease of the expression of several glycolytic enzymes only after the second provocation. One hypothesis to explain such an observation is an increase of the oxidative metabolism at the expense of the glycolytic metabolism in response to training. On the basis of these findings, we demonstrate the utility of the application of a proteomic approach on human muscle samples and we show that the loss of contractile proteins is not responsible for the onset of DOMS.

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