
Alpha Test Scienze Motorie.pdf

File size: 1 MB. Fossil and prehistoric carnivores, 2005, 60-64. Lower frequency modulations of EEG alpha activity are associated with greater . Abstract. abstract. Fatty acid degradation is associated with the expression of senescence markers in human myoblasts. The myogenic satellite cell, considered to be the progenitor cell of skeletal muscle, remains in a relatively quiescent state until senescence. The mechanisms that regulate senescence in satellite cells remain largely unknown. In this study, we examined the effect of the lipid-lowering drug bezafibrate on the expression of senescence markers in human myoblasts (HMBs).

HMBs were incubated with 0, 1, or 10 microM of bezafibrate for 48 h. The percentage of senescence-associated beta-galactosidase-positive HMBs was decreased in a dose-dependent manner by bezafibrate.

Bezafibrate decreased the expression of p16, p21, p53, and p-p53 protein in HMBs. It also decreased the expression of fatty acid synthase in HMBs. When the percentage of senescence-associated beta-galactosidase-positive cells was normalized to the value of fatty acid synthase-positive cells, a dose-dependent decrease in the normalized percentage was observed. The expression of fatty acid synthase was also decreased by bezafibrate in myoblasts derived from the soleus muscle of elderly people, while no difference in the expression of p53 was observed. These results indicate that HMBs are sensitive to fatty acid metabolism and that the expression of senescence markers in HMBs is regulated by fatty acid degradation. In addition, it was found that the expression of fatty acid synthase in HMBs was decreased in elderly people. A novel EPM1 mutation identified in a family with adolescent-onset epileptic encephalopathy. Epilepsy with myoclonus type 1 (EPM1, OMIM 613502) is an autosomal recessive syndrome characterized by complex partial seizures with myoclonus starting in the first year of life. Mutations in a number of genes have been linked to EPM1, including genes encoding calcium channel subunits, voltage-gated sodium and potassium channels, and ubiquitin ligase. The ubiquitin ligase E

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