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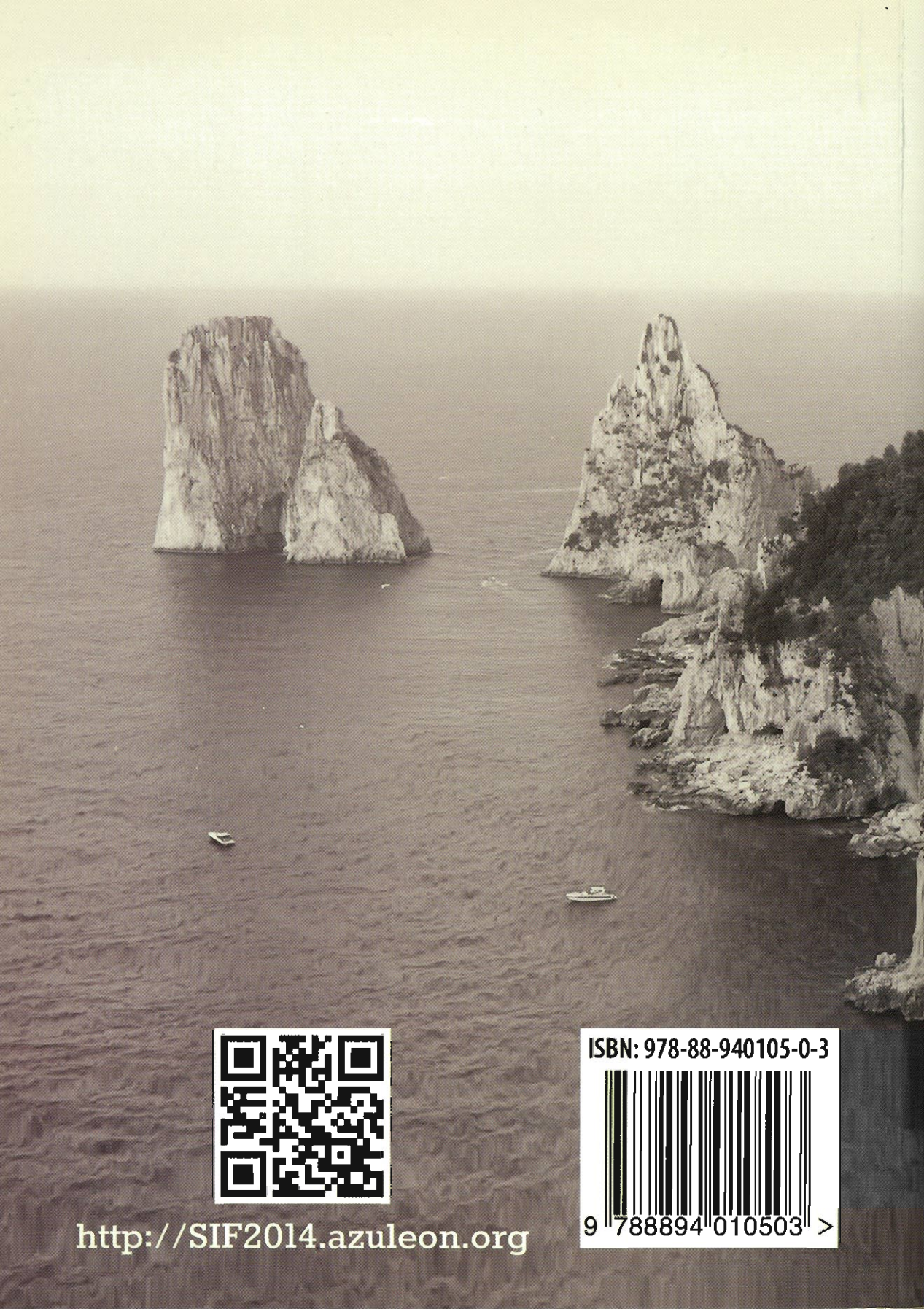

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Quadriceps muscle proteomic profiling of exercise versus sedentary mdx mice

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In our recent study has been shown following low-intensity endurance exercise a significant recovery of damaged skeletal muscle in mdx mice, probably by reducing the degeneration of dystrophic muscle (Frinchi M. et al 2014). Consequently to this finding, in the present work we aimed to identify proteins involved in the observed reduction of degenerating fibers. To do this end, we used proteomic analysis to evaluate changes in proteins profiling of quadriceps dystrophic muscles of exercised versus sedentary *mdx* mice. Four protein spots were found significantly changed and were identified as three isoforms of Carbonic anhydrase 3 (CA3) and as superoxide dismutase [Cu-Zn] (SODC). Protein levels of CA3 isoforms were found significantly up-regulated in quadriceps of sedentary *mdx* mice and were completely restored to wild type values in quadriceps of exercised *mdx* mice. Protein levels of SODC were found down-regulated in quadriceps of sedentary *mdx* mice and were significantly restored to wild type values in quadriceps of exercised *mdx* mice. Western blot data were in agreement with those obtained using proteomic analysis and revealed the presence of one more CA3 isoform significantly changed. Based on data found in the present study, it seems that low-intensity endurance exercise, by counteracting oxidative stress and pH regulation, may in part contribute to reduce cell degeneration process in *mdx* muscles.

P3.3

Study of molecular mechanism involved in neuronal plasticity induced by magnetic stimulation in cultured hippocampal neurons

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Although a large number of investigations have shown that transcranial magnetic stimulation, a non-invasive method of brain stimulation with minimal side effects, is able to induce neuronal synaptic plastic change, very few studies have examined the molecular mechanisms of magnetic stimulation involved in synaptic plasticity. Since it is well known that neurotrophins and their receptors regulate synaptic strength and thereby mediate plasticity, in this study we have investigated the effects of low-frequency (1 Hz) magnetic stimulation, at different intensities, on the activation of neurotrophic factors receptors and relative intracellular pathways in primary cultures of hippocampal neurons. The results showed that one single exposure to magnetic stimulation, low-frequency and 1.55 tesla intensity, activates Glial cell-derived neurotrophic factor receptor (RET), Brain-derived neurotrophic factor receptor (TrkB), Fibroblasts growth factor 2 receptor 1 (FGFR1) and PI3K/Akt pathway in primary cultures of hippocampal neurons after only a short time (5 minutes). These data may explain, at least in part, the mechanism through which magnetic stimulation enhances synaptic plasticity. Our current studies are characterizing the mechanism of neurotrophic factor receptor activation following magnetic stimulation, including the role of neurotransmitters release.