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Effect of spinal cord stimulation on the development of atrophic processes in rat leg muscles during hind limb unloading

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Restriction of muscle functional activity as a result of musculoskeletal damage, CNS injuries, in conditions of weightlessness, is invariably accompanied by the development of atrophy, the prevention of which is an urgent task of neurophysiology and medicine. In the present study, we evaluated the effect of activation of spinal cord neuronal networks in rats on the development of hind limb muscle atrophy while limiting their functional activity. Laboratory animals were divided into the following experimental groups: UN - functional muscle unloading; UN+EES – muscle unloading and daily epidural spinal cord stimulation; UN+MS - muscle unloading and daily non-invasive magnetic spinal cord stimulation. Functional muscle unloading was modeled by hanging the hind limbs. Spinal cord stimulation (EES and MS) was carried out at the L2-L3 level for 90 minutes (10 min stimulation, 10 min break), with a frequency of 3 Hz, threshold intensity for hind limb muscle contraction (determined individually). After 7, 14 and 35 days of exposure to experimental conditions, the wet and dry weights of the soleus, gastrocnemius, and tibialis anterior muscles were determined. It was found that the most pronounced atrophy, manifesting after 7 days of unloading and increasing by 35 days, is observed in the tonic soleus muscle. Weight reduction of mixed gastrocnemius and fast tibialis anterior muscles is observed after 14 and 35 days of unloading. Electrical and magnetic stimulation of the spinal cord limits, but does not prevent, the development of atrophic processes in the hind limb muscles.

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Hydrogen sulfide as the potential therapy for Duchenne muscular dystrophy

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Duchenne muscular dystrophy (DMD) is an inherited X-linked neuromuscular disorder caused by a lack of functional dystrophin, as the result of more than 7000 patient-specific mutations in the largest human genes, *DMD*. We hypothesized that hydrogen sulfide (H_2S), through its antioxidant, proangiogenic, and anti-inflammatory properties, may potentially provide a novel therapeutic strategy to attenuate DMD pathology.
Proteomic analysis revealed that the level of cystathionine β-synthase (CBS) and 3-mercaptopyruvate sulfurtransferase (MPST), the enzymes generating H_2S, is lower in the diaphragm of 6-week-old *mdx* mice, a model of DMD. RNA-seq analysis indicated that endogenous H_2S biosynthesis is diminished also in muscle satellite cells isolated from dystrophic animals. Daily treatment with 100 μmol/kg body weight of NaHS (rapid H_2S donor) for 4 weeks neither affected body weight nor influenced complete blood cell count in *mdx* animals. Although we did not observe any improvement in the grip strength, the activity of creatine kinase, a serum marker of muscle injury, tended to be decreased after NaHS delivery. The protein level of osteopontin, a recently described biomarker of DMD associated with regeneration, inflammation, and fibrosis, elevated in *mdx* mice, was decreaesd by NaHS treatment. Morever, H_2S donor restored decreased expression of proangiogenic factors in *mdx* animals. Ongoing experiments are focused on investigating whether this gaseous mediator can mitigate muscle-related symptoms of DMD.

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