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Dietary NaCl supplementation prevents muscle necrosis in a mouse model of Duchenne muscular dystrophy.

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Abstract

The mdx mouse is an animal model for Duchenne muscular dystrophy. Mdx mice fed a 12% NaCl diet from birth up to 20 days of age (mdx-Na mice) had an approximately 50% reduction in serum creatine kinase (CK) activity compared with mdx mice fed a standard diet. Most notably, necrotic fibers in tibialis anterior (TA) muscle of mdx-Na mice were reduced by 99% and were similar in control mice. These mdx mice displayed significantly elevated blood Ca2+ and Na+ levels, while the total calcium content of their TA muscle was reduced to the level of control mice. In addition, mdx-Na mice had elevated zinc and magnesium contents in their TA muscle. These results suggest that elevated serum Na+ leads to Ca2+ extrusion from muscle via the Na+/Ca2+ exchanger causing a decrease in intracellular Ca2+ levels and an increase in blood Ca2+ levels. Extracellular Ca2+ and, in addition, Zn2+ and Mg2+ might also contribute to the stabilization of the cell membrane. Other possibilities explaining the surprisingly efficacious beneficial effect of dietary sodium exist and are discussed.

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