Searching for therapeutic approaches to skeletal muscle repair and cell therapy

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Duchenne muscular dystrophy (DMD) is characterized by absence of the protein dystrophin, muscle wasting and fibrosis. We have evaluated the role of the profibrotic connective tissue growth factor (CTGF/CCN2) in a genetically model (mdx-CTGF+/- mice) and in a decreasing CTGF activity model, by blocking CTGF antibodies in mdx mice. In both models, we observed a decrease of fibrosis together with a significant less muscle damage compared to mdx mice. The decrease or blocking of CTGF caused an improvement of muscle strength and improvement of wild type cell graft. Thus, CTGF could be novel therapeutic agents to treat muscle fibrosis associated to DMD and improve cell therapeutic approaches.

Biography
Enrique Brandan is a Professor and Chairman of Department of Cell and Molecular Biology, Faculty of Biological Sciences at the Pontificia Universidad Católica de Chile, Santiago, Chile. He has earned his BSc in Biology at Universidad de Chile and PhD in Cell Biology at Universidad Católica de Chile. He was an International Scholar of the Howard Hughes Medical Institute, USA for 10 years. He is a Member of the National Academy of Sciences, Chile. He has been interested in the signaling pathways that control skeletal muscle differentiation and in the mechanisms associated to fibrosis present in several skeletal muscular dystrophies. He has been concentrating in to develop new strategies to fight fibrosis improving skeletal muscle strength and cell therapeutic approaches.

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