Chuanxi Cai

Current Position: Senior Research Associate in the laboratory of Dr. Jianjie Ma in the Department of Physiology & Biophysics at UMDNJ-Robert Wood Johnson Medical School in Piscataway, New Jersey

Education: Ph.D. in Molecular Biology & Biochemistry (2003) from the Institute of Biophysics at the Chinese Academy of Sciences

Non-scientific Interests: Nature, travel, basketball

I received my B.S. degree in Chemistry from Wuhan University in China. My interests in chemistry and biology led me to doctoral studies in the molecular biology and biochemistry program under the supervision of Prof. Jianwen Chen at the Institute of Biophysics in the Chinese Academy of Sciences. During this period, I became increasingly interested in the central role of calcium as a signal for cancer cell apoptosis, which led me to the laboratory of Dr. Jianjie Ma where I began my postdoctoral studies in 2004. During the first two years, my research focused mainly on the role of presenilin-2 in calcium signaling of apoptosis of cancer cells. After finishing that project, my enthusiasm and interest in muscle physiology motivated me to initiate the MG53 related project in Dr. Ma's lab in collaboration with Dr. Hiroshi Takeshima at Kyoto University. We first discovered that a novel muscle-specific protein named Mitsuguimin 53 (MG53) contributes to vesicle trafficking and myogenesis in the course of normal cellular physiology. Recently, we discovered that MG53 acts as a sensor of oxidation to nucleate recruitment of intracellular vesicles to the injury site for membrane repair patch formation. Remarkably, our subsequent studies showed that MG53 can interact with dysferlin to facilitate its membrane repair function, and the membrane trafficking function of MG53 can be modulated through a functional interaction with caveolin-3 (Cav3)—indicating that a molecular complex formed by MG53, dysferlin and Cav3 is essential for repair of muscle membrane damage. This also provides a therapeutic target for treatment of muscular and cardiovascular diseases that are linked to compromised membrane repair. The details of our findings are reported in this paper.

Noah Weisleder

Current Position: Assistant Professor in the Department of Physiology and Biophysics at UMDNJ-Robert Wood Johnson Medical School in Piscataway, New Jersey

Education: Ph.D. in Molecular and Cellular Biology (2003) from Baylor College of Medicine in Houston, TX

Non-scientific Interests: Fencing, baseball, theater

I received my bachelor's degree with distinction from Worcester Polytechnic Institute and my Ph.D. in Molecular and Cellular Biology from Baylor College of Medicine. During my doctoral studies, I became interested in the cellular mechanisms that contribute to the pathophysiology of muscular dystrophy. That led me to study the processes controlling calcium handling in skeletal muscle and now to the mechanisms involved in resealing of muscle cell membranes following injury. The cellular pathways and molecular components of the cell membrane repair machinery are becoming clearer through recent studies, including our findings presented in this paper. We found that MG53 can interact with caveolin-3 and dysferlin to facilitate their function in resealing the sarcolemma of skeletal muscle. Mutations in caveolin-3 that result in muscular dystrophy can alter MG53 function in membrane repair. As these mechanisms continue to come into focus, modulating cell membrane resealing as a therapy for muscular dystrophy and other diseases involving compromised membrane repair is an approach with great translational potential.

Read Dr. Cai and Weisleder's article entitled: Membrane Repair Defects in Muscular Dystrophy Are Linked to Altered Interaction between MG53, Caveolin-3, and Dysferlin … http://www.jbc.org/cgi/content/full/284/23/15894