



Participation of Myoferlin in a Variety of Membrane Trafficking Events

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DESCRIPTION

The six Ferlin proteins are Dysferlin, Misfire, Otoferlin, Myoferlin (MYOF), and Sea Urchin Ferlin. Within this group, MYOF resides. There have been reports of Fer-1, Sea urchin Ferlin, and Misfire in *C. elegans*, Sea urchin, and *Drosophila*. Infertility or poor exocytosis is linked to these proteins' dysfunction. The majority of research on Otoferlin, MYOF, and dysferlin occurs in human tissues. The inner ear's hair cells contain Otoferlin. Its gene mutation results in a nonsyndromic prelingual deafness. Human muscle tissues have significant levels of dysferlin expression, which facilitates membrane healing in a Ca^{2+} dependent ways. Miyoshi myopathy and limb girdle muscular dystrophy are two types of human muscular dystrophy that are linked to dysferlin loss. MYOF is extremely abundant in human muscle tissue as well. It is crucial for myoblast fusion and membrane repair. Myoblasts without MYOF can engage in the first phase of fusion events, but they are unable to mature into big myofibers or to regenerate. MYOF and dysferlin appear to play similar functions in skeletal muscles and may work in concert. Mice lacking both myoferlin and dysferlin have more severe muscular dystrophy than dysferlin-null animals, and transgenic overexpression of MYOF reduces membrane fusion abnormalities in the muscle cells of dysferlin-deficient mice. Whereas dysferlin is abundant in mature myotubes, MYOF is mostly found in immature "prefusion" myoblasts. There is no change in muscle MYOF protein level between individuals with mild and severe limb-girdle muscular dystrophy type 2B induced by a dysferlin mutation. Myoferlin is not overexpressed in compensatory fashion in dysferlinopathy-affected muscles.

MYOF participates in a variety of membrane trafficking events

MYOF is expressed in many different membrane structures, including the plasma membrane, perinuclear vesicular puncta,

Rab7-positive endosomes, as well as the cell periphery, indicating that it is involved in a variety of membrane trafficking activities. According to Doherty et al., MYOF interacts with protein 2 that has an EH domain to take part in endocytic trafficking (EHD2). The internalization and recycling of cell surface receptors are two examples of endocytic transport processes that are mediated by EHD proteins, which include EHD1-4.

MYOF depletion lowers EHD2 levels and slows the recycling of foreign proteins like transferrin, which causes transferrin to accumulate inside cells after it has been internalized and undergoes delayed recycling.

EHD1 and MYOF co-localize in myoblasts and form a prefusion complex that is guided to the surface membrane by the Rho-GAP, GRAF1 (GTPase regulator associated with focal adhesion kinase-1).

MYOF also takes involved in the recycling of the IGF receptor, which starts a crucial signalling process for healthy myogenesis. IGF receptor recycling to the plasma membrane is halted by MYOF depletion and is instead diverted towards lysosomal degradation, which impairs IGF receptor-related pathways and renders cells insensitive to IGF1 stimulation both *in vitro* and *in vivo*. MYOF joins forces with Vascular Endothelial Growth Factor Receptor 2 (VEGFR-2) to mediate the membrane-oriented expression of Endothelial Cells' (ECs) vascular endothelial growth factor receptor 2. Both polyubiquitination and proteasomal degradation of VEGFR-2 are inhibited by MYOF.

The activation of important intracellular signalling cascades including ERK-1/2, JNK, and PLC by VEGF is decreased when MYOF is lost. MYOF is also required for the proper membrane localization of angiopoietin-1, another EC-specific angiogenic receptor.

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