



Mitochondrial fusion is frequent in skeletal muscle and supports excitation-contraction coupling

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Résumé en anglais Genetic targeting experiments indicate a fundamental role for mitochondrial fusion proteins in mammalian physiology. However, owing to the multiple functions of fusion proteins, their related phenotypes are not necessarily caused by altered mitochondrial fusion. Perhaps the biggest mystery is presented by skeletal muscle, where mostly globular-shaped mitochondria are densely packed into the narrow intermyofibrillar space, limiting the interorganellar interactions. We show here that mitochondria form local networks and regularly undergo fusion events to share matrix content in skeletal muscle fibers. However, fusion events are less frequent and more stable in the fibers than in nondifferentiated myoblasts. Complementation among muscle mitochondria was suppressed by both in vivo genetic perturbations and chronic alcohol consumption that cause myopathy. An Mfn1-dependent pathway is revealed whereby fusion inhibition weakens the metabolic reserve of mitochondria to cause dysregulation of calcium oscillations during prolonged stimulation. Thus, fusion dynamically connects skeletal muscle mitochondria and its prolonged loss jeopardizes bioenergetics and excitation-contraction coupling, providing a potential pathomechanism contributing to myopathies.

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