

Publication

A role for septin 2 in Drp1-mediated mitochondrial fission

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Mitochondria are essential eukaryotic organelles often forming intricate networks. The overall network morphology is determined by mitochondrial fusion and fission. Among the multiple mechanisms that appear to regulate mitochondrial fission, the ER and actin have recently been shown to play an important role by mediating mitochondrial constriction and promoting the action of a key fission factor, the dynamin-like protein Drp1. Here, we report that the cytoskeletal component septin 2 is involved in Drp1-dependent mitochondrial fission in mammalian cells. Septin 2 localizes to a subset of mitochondrial constrictions and directly binds Drp1, as shown by immunoprecipitation of the endogenous proteins and by pulldown assays with recombinant proteins. Depletion of septin 2 reduces Drp1 recruitment to mitochondria and results in hyperfused mitochondria and delayed FCCP-induced fission. Strikingly, septin depletion also affects mitochondrial morphology in *Caenorhabditis elegans*, strongly suggesting that the role of septins in mitochondrial dynamics is evolutionarily conserved.

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