～結城賞～

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研究論文内容要旨

Mitochondria are dynamic organelles that frequently move, divide, and fuse with one another to maintain their architecture and functions. However, the signaling mechanisms involved in these processes are still not well characterized. In this study, we analyze mitochondrial dynamics and morphology in neurons. Ca^{2+} influx through voltage-dependent Ca^{2+} channels (VDCCs) was found to cause a rapid halt in mitochondrial movement and induce mitochondrial fission. VDCC-associated Ca^{2+} signaling stimulates phosphorylation of Drp1 at serine 600 via activation of CaMKIα. In neurons, phosphorylation of Drp1 at serine 600 is associated with an increase in Drp1 translocation to mitochondria, whereas in vitro, phosphorylation of Drp1 results in an increase in its affinity for Fis1. It suggests that Ca^{2+} is likely to be functionally important in the control of mitochondrial dynamics through regulaion of Drp1 phosphorylation.