Nonmuscle Myosin II participates in controlling the fission of cellular organelles

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The fission events of various organelles require the merging of two lipid bilayers, but how the two lipid bilayers can come close together during the fission is not well explored. Here, we show that knocking down the actin-binding motor proteins, nonmuscle myosin IIs (NM II) by siRNA or inhibition of their activities by blebbistatin causes the formation of a ring-like assembly of early endosomes (raEE). Inhibition of NM II assembly by an inhibitor of myosin light chain kinase results in the formation of raEE whereas inhibition of NM II disassembly by inhibitors of heavy chain kinases, PKC and CK2 causes the dispersion of early endosomes. The raEEs retain EEA1, Rab7 and LAMP2 markers. Overexpression of an assembly incompetent form, RLC-AA and disassembly incompetent form, NMHC IIB-S6A or NMHC IIA-1916A, induces such defects, respectively.. In parallel, we find the fission of mitochondria is similarly regulated by NM II assembly and disassembly dynamics. Altogether, these data support a model in which NM II activity provides force in regulating the fission events to maintain the size of various organelles in cells.