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Securin and separase modulate membrane traffic by affecting endosomal acidification

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Abstract

Securin and separase play a key role in sister chromatid separation during anaphase. However, a growing body of evidence suggests that in addition to regulating chromosome segregation, securin and separase display functions implicated in membrane traffic in Caenorhabditis elegans and Drosophila. Here we show that in mammalian cells both securin and separase associate with membranes and that depletion of either protein causes robust swelling of the trans-Golgi network (TGN) along with the appearance of large endocytic vesicles in the perinuclear region. These changes are accompanied by diminished constitutive protein secretion as well as impaired receptor recycling and degradation. Unexpectedly, cells depleted of securin or separase display defective acidification of early endosomes and increased membrane recruitment of vacuolar (V-) ATPase complexes, mimicking the effect of the specific V-ATPase inhibitor Bafilomycin A1. Taken together, our findings identify a new functional role of securin and separase in the modulation of membrane traffic and protein secretion that implicates regulation of V-ATPase assembly and function.