

P022 SAND-1 is an effector of RAB-7 and is required for endocytosis in *Caenorhabditis elegans*.

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We describe a temperature-sensitive mutation, which leads to profound defects in late endocytic events in many tissues of *C. elegans*. The mutated gene codes for a protein homologous to yeast Mon1p. In yeast, Mon1p is required in nearly all membrane-traffic pathways, where the vacuole represents the terminal acceptor compartment. The *C. elegans sand-1(or552)* mutation is embryonic lethal at restrictive temperature and causes multiple morphological defects at permissive temperature. In embryos, the blastomeres are filled with extremely large membrane-bound granules, which result from defective endocytic transport of yolk protein. These defects were phenocopied by RNAi of RAB-7, but not of RAB-5 or RAB-11, which indicates that SAND-1 is involved in early to late endosome traffic. In *C. elegans*, coelomocytes are scavenger cells in the body cavity that continuously endocytose fluid. In coelomocytes the *sand-1(or552)* mutant displayed no defects in early endocytosis, but a decreased degradation of endocytosed material and an accumulation of large vacuoles were observed. These vacuoles contain both early and late endosomal markers. In coelomocytes RAB-7::GFP is found predominantly on late endosomal membranes. However, SAND-1 loss of function caused RAB-7 to be mostly cytoplasmic. SAND-1 belongs to a conserved family of proteins present in many eukaryotic species, which, with the exception of yeast Mon1p, has not been characterized before. Our results indicate an important role of SAND-1 in endocytosis.