Mucolipidosis type IV (MLIV) is a neurodegenerative lysosomal storage disorder caused due to MCOLN1 mutations, a gene that encodes mucolipin-1 (TRPML1) - a member of the transient receptor potential cations channels. Two additional homologs are TRPML2 and TRPML3 comprising the TRPML subgroup in the TRP superfamily. The 3 proteins play apparently key roles along the endocytosis process, and thus their cellular localization varies among the different group members. Thus, TRPML1 is exclusively localized to late endosomes and lysosomes, TRPML2 is primarily located in the recycling clathrin independent, glycosyl-phosphatidylinositol (GPI)-anchored proteins, early endosomes and TRPML3 is primarily located in early endosomes. Apparently all the 3 proteins' main physiological function underlies calcium channeling, regulating the endocytosis process. Recent data also indicates that the 3 TRPML proteins form heteromeric complexes at least in some of their cellular content. The physiological function of these complexes in the lysosomal function remains to be elucidated as well as their affect on the pathophysiology of MLIV. Nevertheless, our data indicates ineractions between the TRPMLs regarding their function. Another open question is whether any one of the TRPML bears additional function to the channel activity.