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Implication of Annexin A2 in Exocytotic Site Formation

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The neuroendocrine system depends on elaborate cellular communication provided by intense membrane trafficking. Calcium-regulated exocytosis results in the release of molecules such as neurotransmitters and hormones contained in secretory granules. In neuroendocrine cells, the recruitment and subsequent fusion of secretory granules at the plasma membrane occur at specific sites dedicated to exocytosis. Annexin A2 was the first protein identified at these exocytotic sites in chromaffin cells. Annexin A2 is a calcium- and lipid-binding protein, present in cells either as a 36-kD monomer or as a 90-kD tetramer containing two copies of annexin A2 and two copies of S100A10. It binds two major actors of exocytosis, actin and phospholipids and mediates the formation of lipid microdomains required for the spatial organisation of fusion sites at the plasma membrane. In resting chromaffin cells, annexin A2 is cytosolic, and S100A10 present at the plasma membrane. Following stimulation of exocytosis, annexin A2 translocates to the plasma membrane to form the tetramer.

In this lecture, I will first focus on the mechanisms by which annexin A2 tetramer is recruited to SNARE proteins. In the second part of my lecture, I will present our recent exciting data that demontrates the involvement of cortical actin filaments in lipid domain organisation and indicates how annexin A2 promotes membrane remodelling.

Our results reveal that annexin A2 and the actin cytoskeleton are essential partners in the formation of lipid platforms for granule docking and fusion. This challenges the classical passive role depicted for the cortical actin cytoskeleton in calcium-dependent exocytosis and represents a major advance in our understanding of neuroendocrine secretion.

学生・教職員の多数の来聴を歓迎します。問い合わせ先：相川京子 aikawa.kyoko@ocha.ac.jp