Family ties: annexins, copine, and sorcin

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Annexin A1 and Copine I. Atomic force microscopy was used to monitor the assemblies formed in vitro on lipid bilayers by annexin A1 and copine I. When applied to supported bilayers composed of 25% brain phosphatidylserine and 75% dioleyl phosphatidylcholine in the presence of 1 mM Ca^{2+} both human annexin A1 and human copine I bound only to specialized domains that appeared to be 0.5 to 1.0 nm lower than the rest of the bilayer. These domains may be enriched in phosphatidylserine and have a more disordered structure allowing probe penetration. The binding of the annexin promoted the growth of the domains and created additional binding space for the copine. This may reflect a general ability of annexins to alter membrane structure in such a way that C2 domain-containing proteins like copine can bind.

Novel protein ligands for the annexin A7 N-terminal region. The N-terminal regions of annexins A7 (synexin) and A11 consist of an extended series of short sequence repeats rich in tyrosine, proline, and glycine that provide binding sites for other proteins, including the EF-hand protein sorcin. We recently identified two new potential ligands for the annexin A7 N terminal region by yeast two hybrid screening: the TNFα receptor regulatory protein SODD (Suppressor Of Death Domains) and KIAA0280, a protein of unknown function. Strikingly, the sites of interaction of these proteins with the annexin also contain sequence repeats similar to those present in the N-termini of annexins A7 and A11, and in the annexin A7-binding domain of sorcin. It was also found that the annexin A7 N-terminal region interacts with itself in the two hybrid assay. These results suggest that sequence repeats of this nature form novel structures, called YP pro-β helices, that are characterized by an ability to interact with one another. Specificity of interactions between the pro-β helices in different proteins may be encoded by the variations of residues and lengths of the sequence repeats.