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How some proteins tubulate membranes¹

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Endocytosis, exocytosis, membrane transport between intracellular compartments, virus or toxin entry or exit out of the cell, all imply to deform membrane. Membrane deformation mechanisms of cell membranes by proteins are currently actively studied. Giant vesicles (GUV) are interesting model membrane systems because they are composed of a very limited number of components compared to cellular membranes. The deformations induced by the interaction with a specific protein or any other additional components to the system, can then be directly monitored and the deformation mechanism eventually understood. In this talk, we will focus on different tubular structures induced by proteins. We will show that the B-subunits of Shiga toxin or Cholera Toxin, binding to their lipid receptors, Gb3 or GM1 respectively, incorporated in GUV membrane, induce negative membrane curvature and form tubular invaginations, in absence of any other cellular machinery. Tubular structures can also be obtained when molecular motors walking along microtubules exert a pulling force on the membrane of GUV. The helicoidal assembly of dynamin, a protein involved *in vivo* in membrane fission can also produce tubular structures. This assembly has been reconstituted around membrane nanotubes of controlled diameter; we will show that the initial tube diameter strongly influences dynamin polymerisation. In each case, a physical framework for understanding deformation mechanism will be presented

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