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Lectin-driven and glycosphingolipid-dependent construction of endocytic pits

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Several endocytic processes do not require the activity of clathrin and it has been a major question in membrane biology to know how the plasma membrane is bent and cargo proteins are sorted in these cases. Our previous studies have allowed us to propose the GL-Lect hypothesis: nanodomain construction by glycosphingolipid-binding cellular or pathological lectins induces membrane curvature changes and drives the formation of endocytic pits for the cellular uptake of glycosylated membrane proteins with critical roles in cell migration (CD44, alpha5beta1 integrin) of pathogens (polyoma viruses, norovirus) or pathogenic factors (Shiga and cholera toxins). We are now analyzing how cortical actin dynamics contributes to the clustering of glycosphingolipid-lectin complexes on active membranes, thereby facilitating the nucleation of endocytic tubules exploiting fluctuation forces that had not been linked before to endocytosis. Furthermore, we are identifying mechanisms by which the GL-Lect mechanism is acutely controlled at the plasma membrane. Finally, we study how GL-Lect domain construction at the plasma membrane programs the intracellular distribution of cargo molecules, notably via the retrograde transport route.

Biography

Ludger Johannes has completed his PhD within 30 months at Pierre and Marie Curie University in Paris, and postdoctoral studies from Institut Curie. He currently directs the Chemical Cell Biology unit at Institut Curie. He has published more than 150 papers in reputed journals such as *Cell, Nature,* and *Nature Cell Biology,* and has been serving as an editorial board member of repute. He also aims at exploiting discoveries in fundamental membrane biology research for the development of innovative cancer therapy strategies. He is EMBO member since 2012, and currently holds an ERC advanced grant.

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