BIOENGINEERING SEMINAR

“Lectin-driven and Glycosphingolipid-dependent Construction of Endocytic Pits“

Tuesday, September 5, 2017 – 12h30
EPFL – room AI 1 153

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host: Prof. Bruno Correia

Abstract

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 a novel hypothesis, termed 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.