**Hydraulic fracture of membrane adhesion contacts**

Adhering cells can reduce their volume, either by actively pumping water out through ionic channels or by passive membrane osmosis. It has been shown that this leads to the formation of water filled pockets between the cell membrane and its adhesion substrate [1-2]. Very recently, such pockets have been linked to the formation and positioning of the lumen in embryos [3].

I developed an artificial model system to mimic and elucidate the mechanisms of this ubiquitous phenomenon. I use giant unilamellar vesicles (GUV’s) adhering to a supported lipid bilayer or, to another GUV via the strong biotin-neutravidin links, DNA links or via much weaker and biologically relevant cadherin links. I induce the formation of water pockets between adhered membranes by applying hyperosmotic shock. In this talk, I will discuss what leads to the formation of pockets, what determines their stability and coarsening dynamics and, what is the fate of the membrane linkers - do they break or reorganize in the plane?

By comparing the resulting in vitro findings to the observations on cells I am able to provide hints to the important role of the plasma membrane during the remodeling of cell adhesion contacts.

[1] C.E.Morris et al. ‘The invagination of excess surface area by shrinking neurons’. Biophysical Journal, 85(1), 223-225, (2003).

[2] AJ. Kosmalaska et al. ‘Physical principle of membrane remodelling during cell mechanoadaptation’. Nat Communication 15, 6 : 7292, (2015).

[3] J.G. Dumortier et al. ‘Hydraulic fracturing and active coarsening position the lumen of the mouse blastocyst’. Science 365, 465-468, (2019).