Cell adhesion is a crucial process that takes place through the interaction between a ligand and its receptor at the cell interface. The experimental studies of this interface are challenging due to the lack of control of its chemical and physical properties. For this, I use biomimetic approaches (artificial substrates or model membranes) to quantitatively control the biochemical and physical properties underlying the adhesion process. In the first part of my talk, I will focus on the mechanosensitivity of T-cells, a process at the heart of immune recognition. It takes place through bonds formed by their special receptor called the T-cell receptor or TCR. While the mechanobiology of TCR is well known, its link to cell scale response is poorly understood. By following the spreading response of T-cell on substrate of different rigidity, we aim to study T-cell adhesion and, to which degree substrate rigidity has an influence. We show that T-cell response may be either monotonous or biphasic. For the second part, I will present results on the remodelling of the adhesion contact as cells expel water. This is important in the context of formation of lumen in embryos. We use a biomimetic model system of cell-to-cell adhesion comprised of giant unilamellar vesicles (GUVs) adhering to a supported lipid bilayer or to other GUVs via specific biotin-neutravidin or cadherins bonds. Upon osmotically induced vesicle shrinking, we observe the deformation of the membrane and the formation of water-filled pockets, also seen in cells. We show that the adhesion links are sheared away from the sites of pockets formation than recover with time. The size, distribution and recovery dynamics depends on the magnitude of the osmotic shock and the adhesion strength and density.