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Physico-chemical and biological considerations for membrane wound evolution and repair in animal cells

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Abstract
Membrane damage is a daily threat to the life of a cell, especially cells from muscles, gut, epidermis and vasculature, tissues that are particularly subjected to mechanical stress. Damages can come from different sources and give rise to different holes in terms of size and nature. For example, while some holes are simply scratches in the lipid bilayer, others are delimited by pore forming proteins. It is thus expectable that these wounds will not evolve similarly in a cellular context, and that repair mechanisms will differ to a certain extent. It would therefore be misleading to fully generalize cell membrane damage and repair, and consider it as one universal phenomenon. Indeed, damage has been observed in cells ranging from the rather small mammalian cells (~30µm) to the very big Urchin egg (~100µm). Moreover, the wounds observed or artificially induced in eukaryotic cells range from some nanometers to several micrometers, and can be delimited by particular molecules as mentioned before. This chapter aims at reviewing the different physico-chemical and biological parameters that can influence wound evolution in a cells and to conciliate the different repair mechanisms that have been described by evaluating them in their cellular and wound type context.

1. Physicochemical notions to understand wound repair in simple systems.

1.1. Membrane tension

The size of wounds in plasma membrane can range from a few nanometers to a few microns. From a physical point of view, a hole in a lipid bilayer creates an energetically unfavorable situation and therefore the membrane will have the tendency to self-reseal. Two forces will oppose each other during the healing process. A first force, the "membrane tension", will oppose to wound closure, a second, the "line tension" (see below), will promote reseal.

The **membrane tension** derives from the force defined as **surface tension**, which appears at the interface between two liquid phases. It appears from the coalescence forces between similar molecules within each liquid, which tend to stay together and minimize the surface in contact with the other liquid. It is expressed as an energy per unit area. In the case of a lipid bound vesicle, there are three phases that interplay: the inner phase, the outer phase and the lipid bilayer itself. The tension within the lipid membrane counterbalances the difference between inner and outer pressure, according to Laplace law so that an excess of external pressure will trigger vesicle collapse and an excess of inner pressure not compensated by the outer pressure and by the surface tension will trigger vesicle burst.