To Burst or Not To Burst: Osmotic Regulation in a Protocell Model Through Precursor Mechano-Sensitive Channels

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Extended Abstract

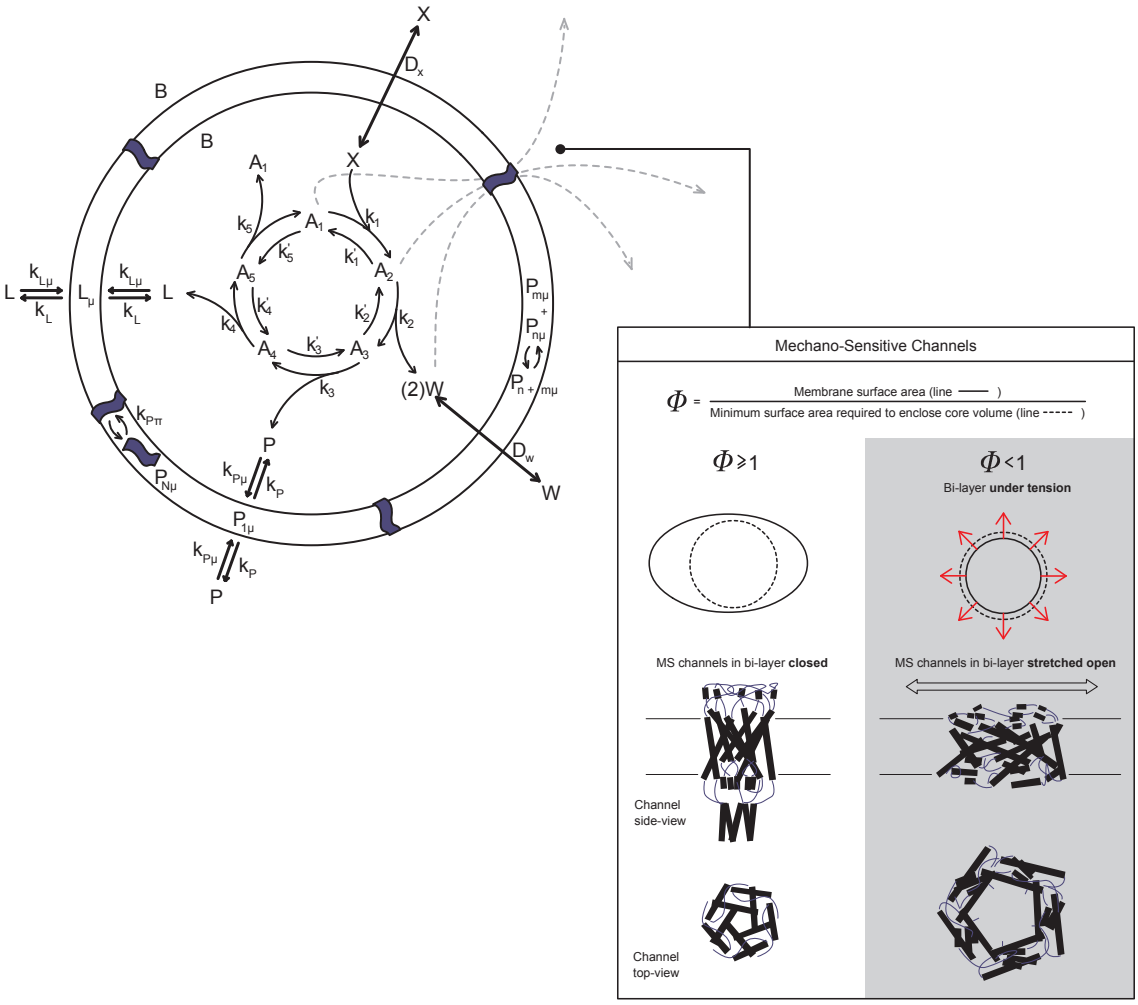

Figure 1: Protocell model with rudimentary Mechano-Sensitive (MS) membrane channels. In osmotic crisis, internal turgor causes tension in the membrane, opening the MS channels and allowing internal solutes to disperse, re-stabilising the system.

We are interested in exploring plausible mechanisms which could enable a simple lipid bi-layer protocell system for more robust and possibly richer self-maintenance dynamics in variable environmental conditions.

One fundamental problem faced by all compartments with a selectively semi-permeable membrane is the ever present threat of *osmotic burst*. For various and sometimes unexpected reasons, internal or external conditions for a cellular system can suddenly change (e.g. an *E. coli* bacterium caught in a rain shower), resulting in the appearance of a large osmotic potential across the membrane. This potential drives a 'shock' flow of water into the cellular compartment, quickly expanding the internal volume and possibly rupturing the membrane. Mechano-Sensitive (MS) channels are one prudent mechanism of increasing interest (Kung (2005)) by which a cell can detect and respond to forces in it's lipid bi-layer. These intricate structures (composed of folded protein helixes) span the membrane, and open a water-filled pore like an iris (see box on Fig. 1) in response to increasing local membrane tension. In the case of the unlucky *E. coli* bacterium caught in the rain shower, the MS channels act as 'emergency valves', releasing internal solutes until osmotic equilibrium is restored again. More generally, MS channels can be thought of as a tranducer mechanism, converting mechanical fluctuations in the membrane (local tensions) into a chemical signal (by way of modulating compartment solute permeability).

This work aims to explore more fully some ideas seeded at ECAL 2007 (Ruiz-Mirazo and Mavelli (2007)) as to how a protein channel feedback system could be useful for cellular stability at a very early stage in the origin of life i.e. in a protocell scenario. In the previous work, one case considered was protein channels becoming aligned and active in the protocell membrane only when the system was in osmotic crisis conditions ($\Phi < 1$, Fig. 1). When open, these channels accelerated the diffusion of an internal waste product out of the protocell compartment, at a rate dependent on a diffusion constant, the number of proteins channels in the membrane and the concentration gradient of the waste.

This study seeks to model the protein channels above as slightly more realistic MS channels. Instead of channels opening indiscriminately whenever there is *some* membrane tension (as in the previous case), now channels open in proportion to the *relative* membrane tension ($1 - \Phi$, when $\Phi < 1$), and each channel has a more realistic binary switching behaviour, remaining effectively closed until a tension transition barrier is crossed, after which it snaps to a fully open conformation. A second objective of this work is to investigate the dynamic implications of the MS channels facilitating not only the diffusion of waste out of the compartment, but also the diffusion of the molecules involved in the internal Ganti (Ganti (2002)) reaction cycle. This direct negative feedback on the growth of the internal cycle presents an interesting dynamical scenario not tested before with the protocell model. Simulations are again being carried out with the ENVIRONMENT (Mavelli et al. (2008)) platform. Results are to be presented at the conference.

References

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