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Fluctuation driven active molecular transport in passive channel proteins

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Living cells interact with their extracellular environment through the cell membrane, which acts as a protective permeability barrier for preserving the internal integrity of the cell. However, cell metabolism requires controlled molecular transport across the cell membrane, a function that is fulfilled by a wide variety of transmembrane proteins, acting as either passive or active transporters. In this talk it is argued that, contrary to the general belief, in active cell membranes passive and spatially asymmetric channel proteins can act as active transporters by consuming energy from nonequilibrium fluctuations fueled by cell metabolism. This assertion is demonstrated in the case of the E. coli aquaglyceroporin GlpF channel protein, whose high resolution crystal structure is manifestly asymmetric. By calculating the glycerol flux through GlpF within the framework of a stochastic model, it is found that, as a result of channel asymmetry, glycerol uptake driven by a concentration gradient is enhanced significantly in the presence of non-equilibrium fluctuations. Furthermore, the enhancement caused by a ratchet-like mechanism is larger for the outward, i.e., from the cytoplasm to the periplasm, flux than for the inward one, suggesting that the same non-equilibrium fluctuations also play an important role in protecting the interior of the cell against poisoning by excess uptake of glycerol. Preliminary data on water and sugar transport through aquaporin and maltoporin channels, respectively, are indicative of the universality of the proposed nonequilibrium-fluctuation-driven active transport mechanism.

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