MECHANOSENSITIVE CHANNELS

Too much pressure

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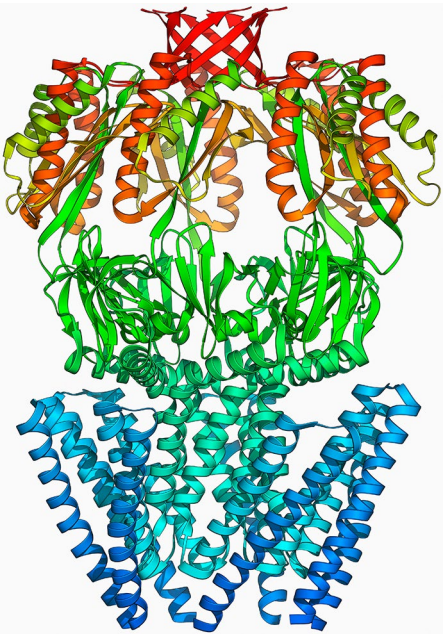

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Mechanosensitive channels do not perceive molecules but physical forces, to which plant cells are constantly exposed. Examples include gravity, wind, elastic energy accumulation after anisotropic growth in twisted and bent tissues, and cell swelling due to osmotic pressure changes during rehydration or flood. How can these mechanical forces be transformed into biochemical signals? A few decades of work have identified transmembrane channels that can sense the lateral tension in the bilayer membrane and generate an ion flux across the membrane to initiate a signalling pathway. These proteins are evolutionary ancient and widely conserved in the tree of life, including in unicellular organisms for which quick adaptation to external osmolarity changes is a vital function.

The exact role of plant stretch-activated channels in initiating multiple downstream outputs and physiological responses to cell swelling has been unclear. To answer this question and move the field beyond electrophysiology studies, Basu and Haswell, in a recent study in *Current Biology*, focused once again on the ubiquitously expressed *Arabidopsis* mechanosensitive ion channel MscS-like 10 (MSL10). They first developed a seedling assay to help nature a little and impose cell swelling by both softening the cell wall and increasing turgor with a hypo-osmotic medium. Then they systematically tested the role of MSL10 during induced cell swelling. The null mutants were defective in practically all the responses: programmed cell death, cytoplasmic calcium transient peak, reactive oxygen species accumulation, mechano-inducible gene expression and cytoplasmic acidification. MSL10-dependent cell death was abolished when a handful of N-terminal residues were replaced by their phosphomimic versions, hinting at a probable post-translational control of the activity.

This study firmly places the MSL10 channel at the heart of the mechanism converting mechanical forces originating during cell swelling into downstream physiological effects. The question now is to know what are the partners of MSL10 directly responsible for effects such as calcium and reactive oxygen species burst. Another interesting question discussed is about the adaptive advantage of killing cells that are swelling too much. Hopefully the future will tell us more about these fascinating pressure-sensing proteins.

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