

New understanding of gating mechanism of CFTR chloride channel

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New research advances our understanding of the gating mechanism of the CFTR, the chloride channel mutated in cystic fibrosis patients. The study by Tzyh-Chang Hwang and colleagues (University of Missouri), and accompanying Commentary by László Csanády (Semmelweis University) appear in the May issue of the *Journal of General Physiology*.

CFTR is a member of the superfamily of ABC proteins found in all organisms, from bacteria to human. The 48 human ABC proteins mostly mediate transmembrane export of substrates at the expense of ATP hydrolysis. They are involved in a wide variety of physiological processes, ranging from insulin secretion to drug detoxification.

Like other ABC proteins, CFTR encompasses two nucleotide binding domains (NBD1 and NBD2), which form a dimer. It is generally accepted that CFTR's opening-closing cycles, each completed within one second, are driven by rapid ATP binding and hydrolysis events in NBD2. Now, using real-time recording, Hwang and colleagues tackle the fundamental question of whether the NBD dimer fully dissociates in each gating cycle, and they provide strong evidence that it does not. The authors propose a gating model for CFTR with a "partial" separation of the NBD dimer, with two distinct cycles.

More information:

Tsai, M.-F., M. Li, and T.-C. Hwang. 2010. *J. Gen. Physiol.*
[doi:10.1085/jgp.201010399](https://doi.org/10.1085/jgp.201010399)

Csanády, L. 2010. J. Gen. Physiol. [doi:10.1085/jgp.201010443](https://doi.org/10.1085/jgp.201010443)

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