Chaperonins are ubiquitous ATP-dependent macromolecular complexes that promote the folding of proteins into thermodynamically stable native conformations. Here, I would like to introduce a bacteriophage EL encoded chaperonin, a drastically different form of chaperonin phylogenetically related to group I and functions in a novel way suggesting the emergence of a different form of chaperonin mediated protein-folding mechanism.

The striking feature of this chaperonin is that the primary sequence exhibits a naturally occurred point mutation (A to T) in the highly conserved ATP-binding region that is conserved in all the known chaperonins. This change in the residue leads to co-chaperonin independent way of protein-folding mechanism with radically different nucleotide-binding conformations. The structural, biochemical and functional investigations reveal that ATP binds co-operatively to both rings and that a misfolded substrate functions as a trigger for progression along the different conformational states. The protein-folding cycle begins with substrate binding followed by ATP hydrolysis and expansion of the internal chamber resulting in ring separation and ring closure. Formation of a single-ring structure with an expanded internal chamber allows $\phi$EL to fold $\beta$-galactosidase, a 116-kDa protein that is not folded by the $E. coli$ GroEL chaperonin.

Collectively, the architecture and nucleotide-binding cycle of the $\phi$EL chaperonin showcases an excellent model for evaluating the already existing bioinformatics tools and software. Furthermore, the data also can be used in building new bioinformatics software for homology modeling, building a database with valuable information about the interaction between the subunits, point mutation studies, etc.

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