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AtpF and membrane proliferation: the role of dimerization

The overproduction of the b subunit of the FoF1-ATP synthase, AtpF, induces massive intracellular membrane (ICM) proliferation in C43(DE3) λ 1 λ . We identified shorter AtpF variants that lead either to ICM accumulation or vesicle formation (AtpF 1_42 FAsH). Partial deletions of the AtpF gene lead to intermediate phenotypes. Here, we investigate the role of the dimerization of the overexpressed AtpF proteins on membrane proliferation. We designed AtpF variants to abolish the dimer interaction either by disruption of the coiled-coiled region in AtpF or by removing the cysteine residues in the FAsH tag, which are known to trigger dimerization via cysteine bond formation. Using TEM analyses, we analyzed the formation of ICM and vesicle formation upon overexpression of AtpF variants. We show that putative loss of dimerization is an important factor involved in the ICM conversion to vesicle formation.

Session

Interaction lipids/polymers/membrane proteins

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