How to determine the number and localization of polar flagellum in *Vibrio* cells?

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Many of the moving bacteria have flagellum, which is generated on the surface of the cell and functions like a screw to swim in the liquid by rotating it. The number and position of flagellar generation vary in bacterial species. *Escherichia coli* and *Salmonella* have multiple flagella around the cells (peritrichous flagella), *Campylobacter* has flagella at both poles of cell, *Pseudomonas aeruginosa* and *Vibrio* have a single flagellum in one pole. FlhF and FlhG are known as proteins that control the formation of flagella at the cell poles. FlhF is also involved in the position determination of flagella formation.

In *Vibrio alginolyticus*, the cell loses flagellum when FlhF is deficient. The cell has many flagella when FlhG is deficient. FlhF and FlhG interact directly. FlhF and FlhG share high homology with the GTP-binding signal recognition particle (SRP) receptor FtsY and cell division inhibitor MinD having ATPase activity, respectively. FlhF is a soluble protein and diffuses into the cytoplasm, and it localizes to the poles of cells with flagella. The polar localization of FlhF increases when FlhG is deleted, so that polar localization of FlhF is inhibited by binding to FlhG. From the ΔflhFG mutant, we isolated a motile suppressor mutant which has peritrichous flagella. We identified the suppressor mutation on a gene that had been uncharacterized, and we named sflA. The gene encodes a putative transmembrane protein containing a J-domain (also referred as the DnaJ-domain) at its cytoplasmic C-terminal region. Recently, we have shown that the deletion of hubP, which encodes a polar landmark protein to anchor three ParA-like proteins (ParA1, ParC, and FlhG) to the cell pole, increases flagellar number at the cell pole, and disrupts the polar localization of FlhG as well as SflA. We think that cytoplasmic FlhG works as a quantitative regulator, controlling the amount of the FlhF localized at the pole, and polar FlhG, anchored by HubP, works as qualitative regulator, directly inhibiting the activity of polar FlhF, in order to achieve the optimal flagellar biogenesis at the cell pole.

In this talk, I'd like to introduce our study about how to control the number and position of flagellar formation in *Vibrio alginolyticus* and I also want to present the recent research on the roles of the ATPase activity of FlhG and the GTPase motif of FlhF.

![Diagram](image.png)