# Article information:

Novel Role for PilNO in Type IV Pilus Retraction Revealed by Alignment Subcomplex Mutations | Journal of Bacteriology
[https://journals.asm.org/doi/10.1128/JB.00220-15?url\_ver=Z39.88-2003=ori:rid:crossref.org=cr\_pub%20%200pubmed](https://journals.asm.org/doi/10.1128/JB.00220-15?url_ver=Z39.88-2003&rfr_id=ori:rid:crossref.org&rfr_dat=cr_pub%20%200pubmed)

# Article summary:

1. The PilMNOP proteins form an inner membrane alignment subcomplex that is essential for the function of type IV pili (T4P).

2. Mutations in the oppositely charged coiled coils (CC) regions or transmembrane segments (TMSs) disrupted PilNO heterodimer formation, while up to six combined mutations in the core failed to disrupt the interaction.

3. Specific CC mutants were hyperpiliated but nonmotile, suggesting that PilNO participate in both the extension and retraction of T4P.

# Article rating:

May be slightly imbalanced: The article presents the information in a generally reliable way, but there are minor points of consideration that could be explored further or claims that are not fully backed by appropriate evidence. Some perspectives may also be omitted, and you are encouraged to use the research topics section to explore the topic further.

# Article analysis:

The article “Novel Role for PilNO in Type IV Pilus Retraction Revealed by Alignment Subcomplex Mutations” published in the Journal of Bacteriology provides a detailed analysis of how mutations in the inner membrane alignment subcomplex can affect type IV pili (T4P) function. The authors use a combination of bacterial two-hybrid assays and experiments conducted on Pseudomonas aeruginosa to demonstrate that specific mutations at three potential interaction interfaces between PilNO can abrogate surface piliation and/or impair twitching motility. They also find that certain CC mutants are hyperpiliated but nonmotile, suggesting a novel role for PilNO in pilus retraction.

The article is generally well written and provides a thorough overview of its topic, with clear explanations of relevant concepts and terminology. The authors provide evidence to support their claims, including data from bacterial two-hybrid assays as well as experiments conducted on P. aeruginosa cells. However, there are some potential biases present in the article which should be noted. For example, the authors focus primarily on how mutations can affect T4P function without exploring other possible effects such as changes in protein structure or stability; this could lead to an incomplete understanding of how these mutations affect T4P assembly and motility. Additionally, while they discuss potential targets for small-molecule inhibitors designed to disrupt T4P function, they do not explore any potential risks associated with such inhibitors or their effects on other cellular processes or organisms. Finally, while they emphasize the importance of studying protein function in a minimally perturbed context and stoichiometry, they do not provide any evidence to support this claim or discuss any potential implications it may have for drug development strategies targeting T4P systems.

In conclusion, this article provides a comprehensive overview of how mutations at three different interaction interfaces between PilNO can affect type IV pili (T4P) assembly and motility; however, there are some potential biases present which should be taken into consideration when evaluating its trustworthiness and reliability.

# Topics for further research:

* Small-molecule inhibitors targeting T4P systems
* Effects of mutations on protein structure and stability
* Potential risks associated with T4P inhibitors
* Minimally perturbed context and stoichiometry
* Drug development strategies targeting T4P systems
* Implications of protein function studies for drug development

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