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# Molecular characterization of a novel T4p component in Neisseria gonorrhoeae

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Background

Neisseria gonorrhoeae (Gc) is the causative agent of the sexually transmitted infection gonorrhea. During pathogenesis, N. gonorrhoeae uses Type IV pili (T4p) to adhere to host cells and promote colonization. T4p are dynamic structures made of pilin fibers that extend and retract from the bacterial cell. Beyond host cell adhesion, T4p are involved in a variety of cellular processes including DNA uptake, twitching motility, and resistance to antimicrobial agents. We previously identified a novel component of the N. gonorrhoeae T4p, which we named TfpC. Our published data illustrated that TfpC is required for full piliation as well as stabilization of a subset of T4p, suggesting it is part of the T4p anti-retraction complex. Moreover, our previously published work showed that deletion of the tfpC gene results in a reduction in levels of pilin protein.

Aim/Methods

Here, we use genetic, biochemical, and biophysical techniques to define how TfpC contributes to T4p dynamics and architecture.

Results

We show that while TfpC is required for complete piliation of Gc, remaining pilus structures undergo extension and retraction at rates similar to strains containing TfpC. We define the subcellular localization of TfpC and investigate the relationship between TfpC and T4p components that promote pilus stabilization. At the molecular level, we propose a model for the interaction of TfpC with the N. gonorrhoeae pilus using structure-function analyses coupled with nuclear magnetic resonance (NMR) and molecular docking studies.

Conclusions

These studies expand our current understanding of T4p architecture as well as the components governing mechanisms of extension and retraction.