ArgR regulates motility and virulence through positive control of flagellar genes and inhibition of diguanylate cyclase expression in *Aeromonas veronii*

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Presenter: Yu-Sheng Lin **Date/Time:** 2025/11/13, 15:20 -16:10

Commentator: Ching-Hao Teng, PhD **Location:** Room 601, Med College Building

Background: *Aeromonas veronii* is a Gram-negative, rod-shaped bacterium that lives mainly in aquatic environments and moves using flagella. It is a zoonotic pathogen infecting both fish and humans. In China, *Aeromonas veronii* infections are found not only in aquaculture but also in humans as an opportunistic pathogen, and in immunocompromised patients, they may progress to severe sepsis. ArgR (arginine repressor) is mainly involved in arginine biosynthesis. Previous studies showed that *argR* knockout strains display motility defects, but the detailed mechanism remains unclear. Moreover, ArgR also affects the level of cyclic di-GMP (c-di-GMP), another key regulator of bacterial motility, which in turn leads to reduced motility.

Objective: *Aeromonas veronii* poses a significant threat to aquaculture and public health in China. Therefore, a deeper understanding of the motility and pathogenic mechanisms of *A. veronii* is essential for developing effective strategies to prevent and control its spread.

Results: ArgR promotes the expression of *flrBC*, which activates the downstream Class III flagellar structural genes and enhances flagellar assembly, thereby increasing bacterial motility. In bacterial physiology, a high intracellular concentration of c-di-GMP typically indicates a transition toward reduced motility and the formation of biofilms or microcolonies. During this stage, bacteria also express adhesion factors to attach to and infect host cells.

This study also found that ArgR represses the expression of diguanylate cyclase, leading to a decrease in intracellular c-di-GMP levels. As a result, it reverses the motility reduction caused by high c-di-GMP, diminishes biofilm formation, and reduces bacterial adhesion and virulence in cell-based assays.

The above observations were made under conditions without exogenous arginine supplementation. When bacteria were cultured with exogenous arginine, ArgR expression increased; however, bacterial motility was reduced. This suggests that, in addition to ArgR, other factors are also involved in regulating bacterial motility, which needs further investigation.

Conclusion: In conclusion, ArgR enhances *flrBC* expression and flagellar assembly while repressing diguanylate cyclase, thereby increasing motility and reducing biofilm formation. However, with exogenous arginine, ArgR expression rises but motility decreases, suggesting other factors also regulate motility and warrant further study.