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Aeromonas hydrophila virulence

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Aeromonas hydrophila is a rod-shaped, gram-negative bacterium typical of the aquatic environment that is also found in drinking water, wastewater, sewage, and food.[1,2] It is considered as an emerging pathogen responsible not only for gastroenteritis and skin infections, but also for more systemic conditions such as peritonitis, bacteremia, meningitis, cholera-like illness, hemolytic uremic syndrome, and necrotizing fasciitis.[3-6] *A. hydrophila* strains isolated in areas hit by natural disasters such as hurricanes and tsunamis have been associated with skin and soft-tissue infections.[7] *A. hydrophila* is also a well-established fish pathogen involved in a range of diseases, including motile aeromonad septicemia in carp, tilapia, perch, catfish, and salmon; red sore disease in bass and carp; and ulcerative infections like epizootic ulcerative syndrome in catfish, cod, carp, and goby.[2] The pathogenesis of Aeromonas infection is complex and multifactorial; it includes O-antigens, capsules, lipopolysaccharide,[8,9] the S-layer,[10] a single, polar flagellum expressed constitutively, lateral inducible flagella, and iron-binding systems;[11] with regard to extracellular products it encompasses hemolysins and enterotoxins,[12] including the cytotoxic enterotoxin (Act), which is a substrate for type 2 secretion system (T2SS),[13] and enzymes such as proteases, amylases, lipases, ADP-ribosyltransferases, and DNases.[14] *A. hydrophila* also possesses a functional T3SS that delivers 4 effector proteins into target host cells, and a T6SS-associated virulence factors that is both an injectisome, like T3SS, and a secretory apparatus promoting bacterial virulence.[15]

Despite the demonstration of its high pathogenic potential, *A. hydrophila* survival and multiplication strategies against the host's immune system have not been fully elucidated. It has been shown that neutrophils, mast cells, eosinophils, and macrophages can release extracellular traps (ETs) consisting of a DNA backbone stabilized by histones, antimicrobial molecules, and proteases. The main function of ETs is to block and to kill the pathogen at the site of infection, thus limiting its spread in the infected organism.[16-18] Such ability of entrapping and killing bacteria may be conferred by an electrostatic interaction between their positive charge and the negative charge of the bacterial surface[19] and by proteins from granules such elastase, myeloperoxidase, cathepsin G, lactoferrin and gelatinase.[17]

However, some pathogens escape ETs by producing DNases that destroy the DNA backbone.[20,21] Despite reports of ET formation in fish,[22] little is known about the ability of *A. hydrophila* to evade the host's immune defense. In the current issue of Virulence, Yachan Ji and colleagues[23] reported the involvement of nucleases in *A. hydrophila* pathogenesis in both murine and fish models using an *A. hydrophila* mutant carrying a nuclease (ahn) deletion. Their results show that Ahn inactivation significantly impairs the ability of *A. hydrophila* to escape killing by the host's immune system, demonstrating the involvement of the nuclease in systemic dissemination.

These findings provide an important contribution to the current knowledge of *A. hydrophila* pathogenicity linked to ET production by immune system cells, and suggest that this could be a general mechanism of invasion that may be not be confined to fish, but also involve different hosts including humans.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

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