Vibrio vulnificus biotype 2 and the eel: a close host-pathogen interaction

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Vibrio vulnificus biotype 2 is the etiological agent of a hemorrhagic septicemia that affects different species of aquatic animals, especially eels under farmed conditions. Biotype 2 isolates can be grouped into three O-serovars, one of which is zoonotic (serovar E). The pathogen can survive in water under starvation conditions for years maintaining its infectivity for eels (both by intraperitoneal and immersion challenge) and mice. Its survival in water is dependent on temperature and salinity, as well as on protist grazing and competition with other bacteria, including the biotype 1 of the species. Water is the prime vehicle for vibriosis transmission and the gills (serovar E) or the anus (non-serovar E) are the main portals of entry into fish. The bacterium multiplies on the gills/anus following saturation dynamics, subsequently invading the blood stream and spreading to the internal organs where it reaches population sizes that are notably lower than those associated to other types of vibriosis. Parallel to bacterial spreading, extensive haemorrhages are produced in all the organs being the head kidney the most damaged organ. A recently performed phylogenetic study has revealed that the biotype 2 is polyphyletic, which would support its reclassification within the species as a pathovar. All fish-virulent isolates of this pathovar harbor a virulence plasmid of around 70 Kb (pVvbt2), which can be transmitted between strains aided by a conjugative plasmid of 52-56 kb This finding supports the hypothesis that this biotype has appeared within the species by acquisition of this plasmid via horizontal gene transfer by *V. vulnificus* strains on fish farms. The only virulence gene/s identified in pVvbt2 is a whole *rtx* (repeat in toxin) gene cluster, which is also present in the chromosome. This protein is involved in virulence since mutations in both genes, the plasmidic and the chromosomal ones, significantly reduce virulence for mice and eels. The rest of plasmidic genes are genes coding for either hypothetical proteins or proteins without homology to any known protein. Among them, two new iron-regulated genes (*vep07* and *vep20*), which code for hypothetical outer membrane proteins, have been selected for virulence studies. Additional chromosomal virulence genes are *gne*, which codes for a sugar epimerase essential for O-antigen biosynthesis and for resistance to microcidal peptides and to the innate immunity of fish and mammals, and *vvp*, which codes for a protease essential for colonization of mucosal surfaces present in aquatic environments and for fish virulence through water.

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