Yersiniosis

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Yersinia enterocolitica is the etiological agent of a gastroenteritis, known as yersiniosis. Occasionally this can give rise to a pseudoappendicitis, which is more severe than that caused by *Y.pseudotuberculosis* and can involve an acute terminal ileitis and mesenteric lymphadenitis. Yersiniosis often results in an immune disregulation which is manifest as a reactive arthritis. Unlike the other pathogenic Yersinia, *Y.enterocolitica* is not thought to be a true zoonosis, being transmitted by the ingestion of infected animal products or by contact. Person-to-person transmission is also possible.

Y.enterocolitica is a small Gram-negative coccobacillus which may be capsulated *in vivo* and which is classified into 34 different serotypes. However, the majority of human disease is caused by serotypes 03 and 08/09. In common with the other pathogenic Yersinia, *Y.enterocolitica* possesses a pyV plasmid which encodes a **type-three secretion system** (T3SS) and the secretion of a virulence (V) antigen and other Yersinia outer proteins (Yops). The V antigen secreted by the *Y.enterocolitica* 03 serotype is homologous to that of the *Y.pestis* and *Y.pseudotuberculosis* V antigens. However the V antigen from the 08 serotype of *Y.enterocolitica* incorporates an additional hypervariable region, which differs from the V03 sequence. These differences are significant since the V antigen from *Y.pestis* is an important protective antigen and so some cross-protection between Yersinia strains with homologous V antigens may be possible.

The V antigen regulates T3S and the **V antigen is also anti-inflammatory**, **inducing IL10 secretion and down-regulating the pro-inflammatory cytokines TNFα and IFNγ**. There is evidence that the N-terminal region of the V antigen stimulates TLR2 receptors to induce this anti-inflammatory effect in host cells. The T3SS is induced on close contact with a host cell *in vivo*, when *Y.enterocolitica* produces a hollow needle-like projection through which effector proteins are translocated directly into the host cell. These effectors are variously cytotoxic, anti-phagocytic or anti-inflammatory and generally promote the apoptosis of host cells and thus the survival of bacteria in the host. By all these means, *Y.enterocolitica* counters innate immune defences and achieves dissemination in the host to establish a bacteremia. A wide range of antibiotics, except for penicillin, are effective for the therapy of yersiniosis. *Y.enterocolitica* is susceptible to sulphadiazine, streptomycin, tetracycline, chloramphenicol and cotrimoxazole. .

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