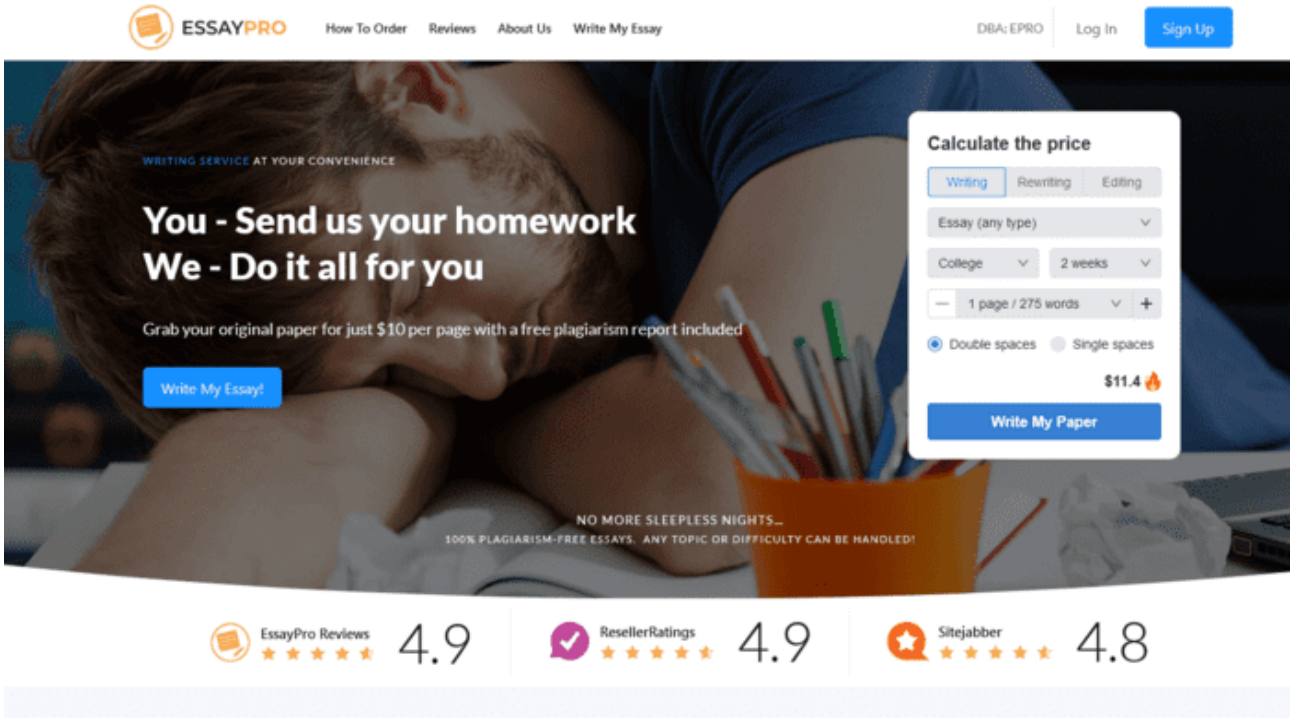
Shigella flexneri



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Shigella flexneri

Shigella flexneri, a facultative anaerobe belonging to the family Enterobacteriaceae, is a Gram-negative rod that is the causative agent of diarrhea and dysentery in humans. Potentially life-threatening, S. flexneri's effects include bacteremia, hemolytic uremic syndrome (HUS) and toxic megacolon (4). The principle disease of diarrhea and dysentery caused by this pathogen is known as shigellosis. 10-100 organisms are sufficient to cause disease, and transmission is generally from person-to-person by way of fecal-oral (2). Shigellosis can be characterized as a disease with over 60% incidence in children ages 1-5 (6).

Life cycle:

S. flexneri causes infection via bacterial penetration of the mucous membrane in the human colon. Humans are the only known reservoir to this pathogen (7). Following invasion of M cells and upon contact with the epithelial cells of the colon, S. flexneri releases Ipa proteins through a type three secretion system. Once inside the host cell, Ipa proteins activate small GTPases in the Rho family as well as c-src, a protooncogene,

leading to cytoskeletal rearrangements. This alteration to the cytoskeleton allows the bacteria to be macropinocytosed by the host cell. Once inside the host, the pathogen colonizes the cytoplasm. IcsA, a bacterial surface protein, activates the host protein N-WASP and, in turn, stimulates actin assembly by host Arp 2/3. Thus, S. flexneri develops actin-based motility enabling the pathogen to become efficient at cell-to-cell spread and host cell cytoplasmic colonization. Infected cells become highly proinflammatory and secrete IL-8. IL-8 attracts neutrophils to the site of infection. The influx of neutrophils, chemokines and cytokines to the area damages the epithelial layer permeability and, in turn, advocates further S. flexneri invasion (6). Once initial invasion of S. flexneri occurs, the targeted epithelial cells require 45 minutes to 4 hours to mount an inflammatory response (8).

A diagram outlining S. flexneri's type three secretion system can be viewed at: http://www.grad.ucl.ac.uk/comp/2003/jointposter/gallery/index.pht?entryID=29

Virulence:

A 214-kb virulence plasmid encodes for S. flexneri's entry into human epithelial cells and intra-intercellular movement (6). Once in contact with the target cell, Ipa proteins (IpaB, IpaC and Ipa D) encode for a specific type III secretion system, thus, enabling entry of the pathogen into the target cell and characterizing S. flexneri with an invasive phenotype (1). One 30-kb block of the plasmid contains genes at the ipa/mxi-spa locus.

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