Genus shigella

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Genus shigella

The 4 species in genus shigella; *S.boydii, S. dysenteriae, S. flexneri,* & *S. sonni* are usually associated with disease in human & non-human primates. *S. sonni* is the most common cause of shigellosis in developed countries, whereas, *S.flexneri* is more common in developing countries.

Gastroenteritis of children is the most common manifestation of disease. *S.dysenteriae* causes severe watery or dysentery. The organism produce disease through production of shiga toxin, which disrupts cellular protein synthesis & cause endothelial damage.

Humans are the sole reservoir of shigellae, & symptomatic carriers are not uncommon. Disease is spread by the fecal-oral route and is highly infectious in that the infection dose is only a few organism

Shigellosis

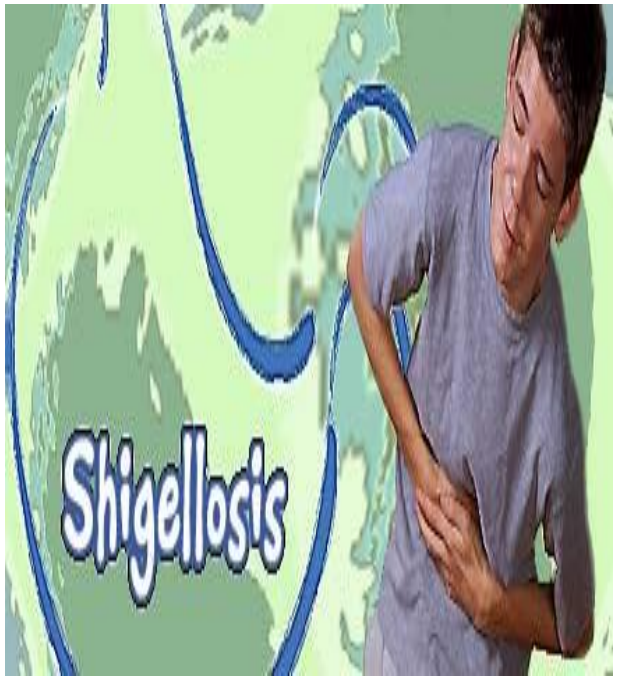
Bacillary dysentery

The natural habitat of shigellae is limited to the intestinal tract of humans & other primates, where they produce **Bacillary dysentery**

Shigellae are G- rods, non-motile, facultative anaerobes, but grow better aerobically. Non-lactose fermentor, all ferments glucose (except *Sh. Sonnie*) with the production of acid but no gas.

Antigenic structure:

Shigellae have complex antigenic patterns. The O Ags of shigellae are LPS, there are more than 40 serotypes. The classification depend on biochemical & antigenic characteristics. The pathogenic species are; *Sh. Dysenteriae, Sh. Flexneri. Sh. Boydii, and Sh. Sonnei.*



Pathogenesis :

Shigella infections are almost always limited to GIT & invasion of bloodstream is very rare. Shigellae are highly communicable, the infecting dose is 10^3 organisms (10^5-10^8 organisms for salmonella & vibrio). The essential pathological process is invasion of the mucosal epithelial cells (e.g. M cells) by induced phagocytosis, escape from the phagocytic vacuoles, multiply & spread within the epithelial cell cytoplasm, & pass to adjacent cells. Microabscesses in the wall of large intestine & terminal ileum lead to necrosis, superficial ulceration & formation of pseudomembrane.

Shigellosis

Bacillary dysentery

Toxins:

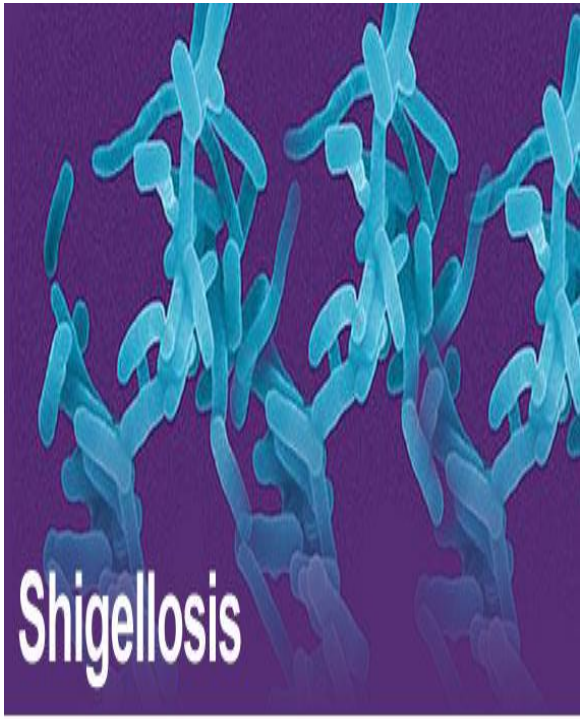
Upon autolysis, all shigellae releases toxic LPS which may contribute to irritation of the bowel wall.

***Sh.dysenteriae* exotoxin:**

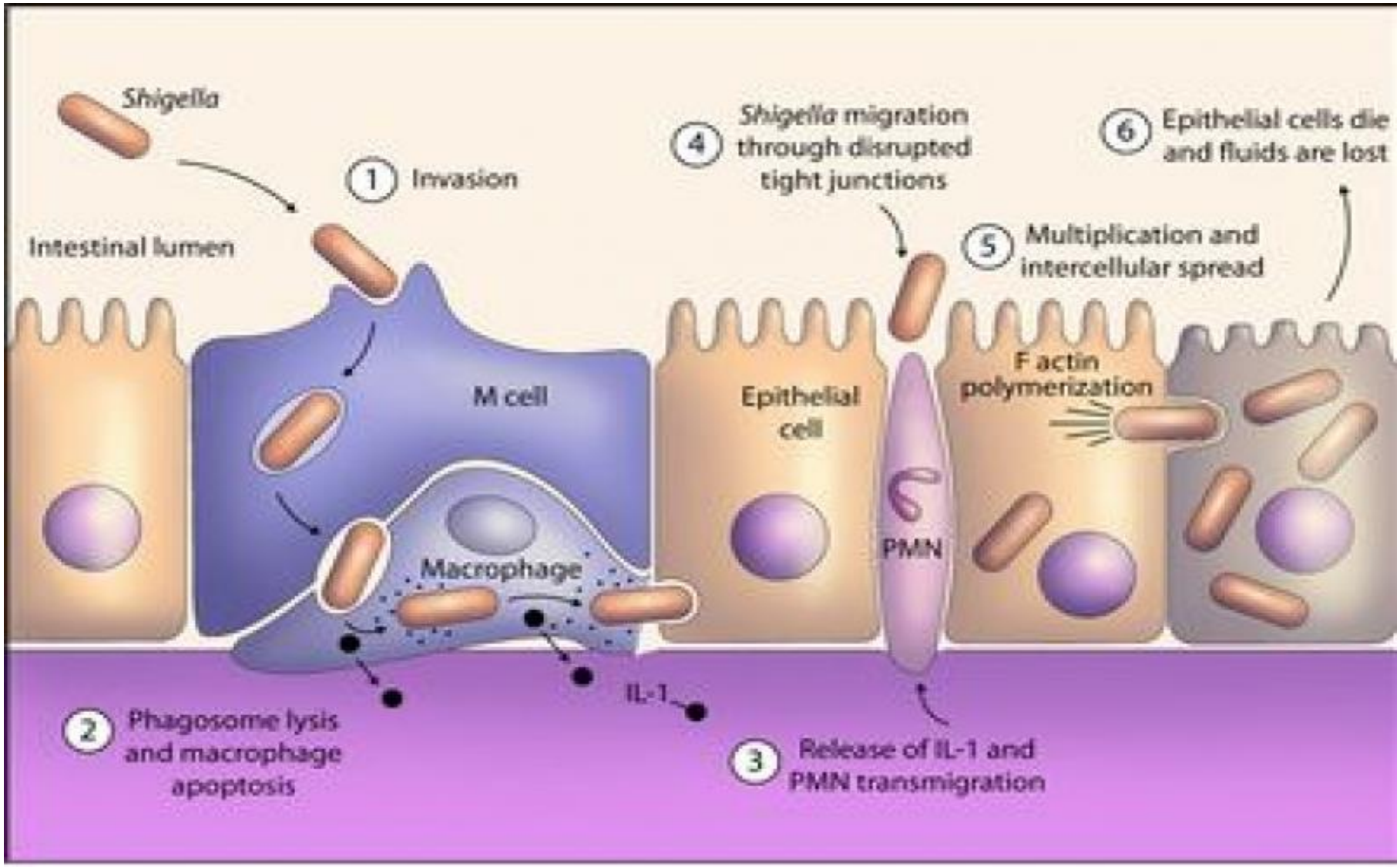
Sh.dysenteriae produce a heat-labile exotoxin that affects both the gut & the CNS. Acting as enterotoxin, it produces diarrhea as does the *E. coli* verotoxin & by the same mechanism. In humans, the exotoxin also inhibit sugars & amino acids absorption in the small intestine. Acting as neurotoxin, may contribute to the extreme severity & the fatal nature of *Sh.dysenteriae* infection and to the CNS reactions. The toxic activity is distinct from the invasive property of shigellae in dysentery; both may act in sequence, the toxin produce an early non-bloody diarrhea & the invasion of the large intestine results in later dysentery with blood & pus in stool.

Clinical findings:

After a short IP (1-2 days), there is a sudden onset of abdominal pain, fever & watery diarrhea. Later, as the infection involves the ileum & colon, the # of stools increases, less liquid, but often contain mucous & blood. Each bowel movement is accompanied by straining & **tenesmus** (rectal spasm), with resulting lower abd. Pain. In children & elderly, loss of water & electrolytes may lead to dehydration, acidosis & even death. On recovery, most persons shed dysentery bacilli for a short period, but a few remain chronic carriers. Upon recovery from infection, most persons develop circulating Abs, but these do not protect against reinfection.



Pathogenesis of shigellosis



Shigellosis
Bacillary dysentery

Lab. Diagnosis:

Specimens: it include fresh stool, mucous flecks & rectal swabs for DSE & culture. Large # of fecal leukocytes & RBCs are seen microscopically. Serum to demonstrate rising titers of agglutinating Abs.

Culture: on differential media e.g. MacConkey or EMB agar & on selective media e.g. Hektone enteric agar or SS agar, which suppress other enterobacteriaceae & G+ bacteria. Colonies are convex, circular, transparent. Shigella produce acid, but not gas (H2S) on TSI agar (Alk. slant/acid. butt).

Serology: normal persons have rise in specific Abs. serology do not use in the diagnosis of shigellosis.

Immunity: Shigella infection followed by specific Ab response. Circulating Abs fails to protect against reinfection. IgA in the gut may be important in limiting reinfection.