Epidemiology of shigella infection and evaluation of metabolic activity.

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Abstract

Diarrhoea is a major global health problem, and recent studies have identified Shigella as a major contributor to this burden. Here we review recent advances in Shigella research. We focus on their epidemiology, etiology, antimicrobial resistance, and the role of the gut microbiota during infection. A combination of improved epidemiological data and a new generation of diagnostics have revealed that the Shigella burden is greater than previously thought. This is not limited to vulnerable populations in low- and middle-income countries. As we gain more and more detailed insight into the orchestrated mechanisms utilized by Shigella to induce infections, we can also begin to understand the complex role of the gut micro biota in preventing and inducing such infections. Using genomics in combination with epidemiological data and laboratory studies has elucidated the evolution and distribution of different species.

Keywords: Diarrhoeal disease, Epidemiology, Genomics Pathogenesis, Shigella.

Introduction

Shigella is a common cause of bacterial diarrhoea worldwide, especially in resource-limited countries. Shigella is less susceptible to acid than other bacteria, allowing it to survive through the stomach. For this reason, as few as 10-100 organisms can cause disease. Ingested bacteria enter the small intestine where they multiply. A large number of bacteria then enter the colon, where they invade the colon cells. Given the relatively low infectious dose, Shigella transmission can occur both through direct person-to-person transmission and through contaminated food or water. Humans are the only natural hosts of disease [1].

Here, we describe the epidemiology, microbiology, and etiology of Shigella infection. Clinical manifestations, diagnosis, and treatment are discussed separately. See Shigella Infection Clinical Manifestations and Diagnosis and Shigella Infection treatment and prevention in adults. Gram-negative facultative anaerobes of the genus Shigella are the major causative agents of shigellosis. This disease differs from the profuse watery diarrhoea commonly seen in biliary or enter toxigenic E. coli diarrhoea, with dysentery scanty and containing blood, mucus, and inflammatory cells [2].

However, in some people with dysentery, moderate diarrhea is the precursor or only sign of infection. Shigellosis accounts for a significant proportion of acute bowel disease in children in developing countries, and this infection is the leading cause of developmental delay in these children. Shigella also poses a significant risk to travelers from developed countries visiting endemic areas, where sporadic food- and water-borne outbreaks occur. The pathogenesis of shigellosis is complex and includes diarrheal prodrome with possible enterotoxin and/or cytotoxicity, cytokine-mediated inflammation of the colon, and colonic epithelial necrosis [3].

The underlying physiological injury that triggers this inflammatory cascade is invasion of the colonic epithelium and lamina propria by Shigella. The resulting colitis and mucosal ulcers lead to bloody, mucous and/or febrile diarrhea [4].

Epidemiology

Shigella, caused by Shigella spp is a major cause of morbidity and mortality. Worldwide, 188 million cases of Shigella diarrhea or dysentery occur each year, resulting in 164,000 deaths. Among her children under 5 years of age in low- and middle-income countries, Shigella spp. is the most common cause of dysentery and her second most common cause of diarrhea overall. Shigella infection can occur through direct person-to-person transmission or through contaminated food or water. Since no intermediate bacterial replication is required to reach a low infectious dose, the minimal infectious dose can be transmitted directly from contaminated fingers. In resource-rich countries, most cases are transmitted by fecal-oral transmission from symptomatic infected persons. Outbreaks in the United States occur primarily in settings such as day care centers and are less commonly caused by general contamination of food and drinking water. Outbreaks have also been associated with untreated recreational water [5].

Conclusion

A combination of large-scale epidemiological studies, more sophisticated in vitro techniques, and genomics has provided unprecedented insight into the success of Shigella. These

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***Received:** 26-Oct-2022, Manuscript No. AAJIDMM-22-82047; **Editor assigned:** 28-Oct-2022, PreQC No. AAJIDMM-22-82047 (PQ); **Reviewed:** 11-Nov-2022, QC No AAJIDMM-22-82047; **Revised:** 15-Nov-2022, Manuscript No. AAJIDMM-22-82047; **Published:** 22-Nov-2022, DOI:10.35841/aajidmm-6.6.129*

***Citation:** Victor D, Epidemiology of shigella infection and evaluation of metabolic activity. J Infect Dis Med Microbiol. 2022;6(6):129*

could be invaluable for the development of future vaccines and alternative therapies. A Shigella vaccine must take into account the many tricks pathogens use to manipulate immune responses and the rapidly changing epidemiology. New therapies may benefit from a detailed description of Shigella etiology and its interactions with the gut microbiota. These tools must be accelerated to stem the tide of increasingly antibiotic-resistant Shigella clones. Shigella research has reached a critical stage and knowledge, technology and experience must be applied to reduce the disease burden of this bacterial pathogen.

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Citation: *Victor D, Epidemiology of shigella infection and evaluation of metabolic activity. J Infect Dis Med Microbiol. 2022;6(6):129*