Pathogenesis of Leptospirosis: Important Issues
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Abstract
The leptospirosis is a re-emerging anthropozoonosis with worldwide distribution. The immunopathogenesis of the disease is extremely complex. Which one the role of inflammatory mediators, cytokines, outer membrane proteins, apoptosis and others factors related with the virulence of the pathogen during the infection.

Summary
Leptospirosis is a re-emerging tropical infectious disease [1], is an important zooanthroponic disease spread-worldwide [2]. The spirochetes of the genus Leptospira is responsible of human and animal leptospirosis characterized as mild febrile illness to severe multiorgan failure, especially pulmonary hemorrhage and renal failure [3]. Pathogenic leptospires are highly motile and invasive spirochetes that have the capacity to survive and grow in tissue by escaping natural defense mechanism [4]. The disease is transmitted to humans by direct or indirect exposure to contaminated urine from mammalian reservoir hosts as rodents but also farm, wild, and domestic mammals [5]. Asymptomatic form of leptospirosis with fever, headache, and myalgia that can spontaneously resolve is one of clinical presentations [3]. The most cases are probably inapparent and associated with host-adapted serovars such as Canicola in dogs, Bratislava in horses and pigs, Hardjo in cattle and Australis and Pomona in pigs [6-9].

In humans can vary in severity according to the infection serovar of Leptospiro, and the age, health and immunological competence of the patient [2]. However, more severe cases, with sepsis and multiple organ failure, including hepatic and renal dysfunctions associated to pulmonary hemorrhage, are also reported [3]. Leptospires enter the body through small cuts or abrasions, via mucous membranes such as the conjunctiva or thorough wet skin. They circulate in the blood stream, with the bacteremic phase lasting for up to 7 days [2]. The second stage of acute leptospirosis is also referred to as the immune phase, in which the disappearance of the organism from the bloodstream coincides with the appearance of antibodies [5]. The mechanisms by which leptospirosis cause disease are not well understood. The presence of the virulence factors has been suggested. The involvement of toxins or toxic factors in the pathogenesis of leptospirosis has long been contemplated, since the absence of the microorganism at the site of tissue injury is a factor that strengthens this hypothesis [10,11].

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on the pathogen’s ability to attach to host tissues during infection. However, additional research is essential to understanding how, the mechanisms by which leptospirosis induces the tissue injury and what role that virulent factors on the pathophysiology of leptospirosis.

References