

## **Pathophysiology of Sepsis**

Sepsis defined as a dysregulated host response to infection leading to organ failure is a global health crisis. Worldwide approximately 49 million incidents of sepsis and 11 million deaths related to sepsis were reported in 2017. Sepsis is not disappearing; in fact, the number of patients with sepsis is increasing worldwide, due to the development of antibiotic resistant bacteria, emergence of novel infectious diseases e.g. COVID-19 and increased longevity these numbers are dramatically increasing each year. Thus, a coordinated global effort is urgently needed to tackle this crisis.

The pathophysiology of sepsis is complex. Previously, it was believed that sepsis was simply an exaggerated, hyperinflammatory response with patients dying from inflammation-induced organ injury. However, data over the last two decades has shown that the manifestation of sepsis can no longer be attributed only to the infectious agent and the immune response it engenders, but is also due to dysregulated coagulation, cellular dysfunction, immunosuppression, altered metabolism and organ dysfunction. Despite these advances in our understanding we still lack a full understanding of the pathogenesis of sepsis, and many questions remain. For instance, what is the mechanism for the initiation, maintenance, and termination of sepsis? What is the underlying mechanism of sepsis causing cellular and subcellular dysfunction? How does cellular dysfunction cause organ failure? What are the mechanisms contributing to dysregulated coagulation and metabolism? Are septic patients hyperinflammatory or immunocompromised?

In this special issue we aim to bring together Original Research, Reviews, Mini-Reviews, Perspective, Protocols, Case Report articles to enhance our knowledge of the pathophysiology of sepsis. In doing so we hope to identify new avenues for healthcare intervention and to accelerate improved diagnosis and treatment for sepsis.

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