

# Immunological Consequences of Trauma and Shock

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## Abstract

*The immune system is a powerful, complex entity composed of numerous cell types and regulated by autocrine, paracrine, and hormonal mechanisms. Trauma and haemorrhagic shock induce numerous changes within this system which are ultimately deleterious and contribute to the high incidence of organ dysfunction and infectious complications seen following injury. Regional hypoxia and depletion of intracellular energy stores occur in response to diminished microcirculatory blood flow, and these changes alter cellular signalling and result in the release of pro-inflammatory cytokines and prostanoids which mediate further suppression of immune function. Neutrophil priming serves to induce tissue damage in critical organ systems such as the lungs, heart, liver, and gut, further insulting the injured organism. Depression of antigen presentation and cytokine elaboration by macrophages and other antigen presenting cells effectively prevents a normal response from the acquired immune system, and lymphocyte-monocyte interactions are squelched. The resulting depression in cell mediated and humoral immunity renders the organism susceptible to microbial infection and contributes to the morbidity and mortality associated with nosocomial infections. Hormonal modulation of the immune response is highly evident following trauma and haemorrhage, and the preponderance of male morbidity associated with sepsis can be explained by the depression in immune function seen in males, but not females in the pro-oestrous state. Despite the multitude of changes induced by trauma and haemorrhage, experimental studies have revealed several promising pharmacologic interventions which may serve to blunt the effect of injury on the immune system, and render the host competent to withstand the bacterial and viral challenges responsible for so much of the late mortality following severe injury.*

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