During polymicrobial sepsis, microbial pathogens and their products activate the innate immune system through signaling receptors of the Toll-like receptor (TLR) family, resulting in hyperinflammation and organ injury. The analysis of preclinical mouse models has shown that inactivation of the common TLR signaling adaptor protein MyD88 prevents the hyperinflammatory response and improves survival. Importantly, MyD88 deficiency does not impair antibacterial defense mechanisms. Thus, TLRs and proteins involved in TLR signaling may represent interesting targets for the development of new drugs for reprogramming pathophysiological immune responses during sepsis.
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TUM Einrichtung:
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