

DHEA and sepsis

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DEHYDROEPIANDROSTERONE MODULATES TOLL-LIKE RECEPTOR EXPRESSION ON SPLENIC MACROPHAGES OF MICE AFTER SEVERE POLYMICROBIAL SEPSIS.

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Toll-like receptors (TLRs) play a pivotal role in the induction of innate immunity after the transactivation of proinflammatory cytokine genes. However, the responses of TLRs during severe polymicrobial sepsis have not been thoroughly examined. Although dehydroepiandrosterone (DHEA), a steroid hormone, is reported to have an immunomodulatory effect after sepsis, the mechanism responsible for its salutary is not known. To investigate this, male ICR/Jcl mice (5-8 weeks old) were subjected to sepsis by cecal ligation and puncture (CLP) or sham operation. The mice received vehicle or DHEA (40 mg/kg body weight) subcutaneously immediately after the surgery. Plasma IL-10 levels and splenic macrophage TNF-alpha production, as well as the expression levels of CD14, TLR2, and TLR4 mRNAs on splenic macrophages, were assessed 6 h after the surgery. The results indicate that mice with sepsis show a marked increase in the plasma IL-10 levels and a decrease in TNF-alpha production by splenic macrophages. TLR2 and TLR4 mRNA expression levels after CLP were significantly lower compared with those after the sham operation. TNF-alpha production and TLR2 and TLR4 mRNA expression on splenic macrophages are restored with DHEA administration. Furthermore, administration of DHEA after CLP delayed the mortality of animals. These results indicate that the anti-inflammatory phase of sepsis induces a marked down-regulation of TLR expression on splenic macrophages; however, administration of DHEA resulted in the restoration of TLR2 and TLR4 mRNA expression.

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