



Fighting the stranger-antioxidant protection against endotoxin toxicity.

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Septic shock is a serious problem in critically ill and surgical patients throughout the world. It is a systemic inflammatory response caused by excessive secretion of proinflammatory mediators, such as tumor necrosis factor-alpha, mainly induced by endotoxin, a major component of the Gram-negative bacterial outer membrane. Experimental evidence suggests that reactive oxygen species (ROS) may be important mediators of cellular injury during endotoxemia, either as a result of macromolecular damage or by interfering with extracellular and intracellular regulatory processes. In addition, nitric oxide is thought to play a key role in the pathogenesis of sepsis. This review begins with a brief overview of the toxic effects of endotoxin at organism level, paying particular attention to cardiovascular damage. It continues by analysing the mechanism by which endotoxin is recognized by specific cells of the immune system, which then respond to bacterial infection and the pathway leading to nuclear factor-kappaB activation and proinflammatory gene transcription. With regard to this process, the review focuses on the involvement of reactive oxygen and nitrogen species. Lastly, the protective role of antioxidants against endotoxin toxicity and their potential clinical use is discussed.

Publication Types:

- Review
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The physiological role of dehydroascorbic acid.

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Dehydroascorbic acid (DHA) is abundant in the human diet and also is generated from

vitamin C (ascorbic acid, AA) in the lumen of the gastrointestinal tract. DHA is absorbed from the lumen of the small intestine and reduced to AA, which subsequently circulates in the blood. Utilization of AA as an antioxidant and enzyme cofactor causes its oxidation to DHA in extracellular fluid and cells. DHA has an important role in many cell types because it can be used to regenerate AA. Both physiological (e.g. insulin, insulin-like growth factor I, cyclic AMP) and pathological (e.g. oxidative stress, diabetes, sepsis) factors alter the transport and metabolic mechanisms responsible for this DHA recycling.

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