Injections of lipopolysaccharide (LPS) have been used to produce the signs of sepsis and study their underlying mechanisms. Intravenous (IV) injections of LPS in anesthetized cats induce tachypnea, tachycardia and hypotension, but ventilatory changes are suppressed after sectioning carotid and aortic nerves. Otherwise, LPS increases the basal frequency of carotid chemosensory discharges, but reduces ventilatory and chemosensory responses to hypoxia and nicotine injections. Increases in cytokines (IL-1 beta, IL-6 and TNF-alpha) are observed in plasma and tissues after injecting LPS. In carotid bodies perfused in vitro, TNF-alpha reduces chemosensory discharges induced by hypoxia. The rat carotid body and its sensory ganglion constitutively express LPS canonical receptor, TLR4, as well as TNF-alpha and its receptors (TNF-R1 and TNF-R2). Increases of TNF-alpha and TNF-R2 expression occur after LPS administration. The activation of peripheral and central autonomic pathways induced by LPS or IL’s is partly dependent on intact vagus nerves. Thus, the carotid and vagus nerves provide routes between the immune system and CNS structures involved in systemic inflammatory responses. (C) 2011 Elsevier B.V. All rights reserved.

**Palabras clave**

**Palabras clave de autor:** Arterial chemoreceptors; Autonomic sensory pathways; Carotid body; Immune receptors; Inflammation; Lipopolysaccharide; Tumor necrosis factor alpha

**KeyWords Plus:** TUMOR-NECROSIS-FACTOR; DORSAL VAGAL COMPLEX; RECEPTOR-TYPE-I; VAGUS NERVE; GLOMUS CELLS; INTRAPERITONEAL INJECTION; FACTOR-ALPHA; RAT; LIPOPOLYSACCHARIDE; ACTIVATION