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## *Injury, Inflammation, and Sepsis: Laboratory and Clinical Approaches*

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**COVER:** Schematic of selected changes in inflammation and permeability after LC. The diagram depicts the onset of selected aspects of acute inflammation and permeability injury associated with blunt trauma-induced LC. Direct traumatic insult to the lungs generates an innate inflammatory response that includes the recruitment and activation of blood leukocytes, the activation of lung tissue macrophages, and the production of multiple mediators such as cytokines and chemokines. Neutrophils contribute significantly to the severity of inflammatory LC injury and are activated at least in part via TLRs such as TLR-2 and TLR-4 in the epithelium. Inflammatory mediators in LC are also released by alveolar type II cells and other resident pulmonary cells. This inflammatory response, in conjunction with direct LC-induced tissue injury, damages the barrier integrity of the alveolocapillary membrane and increases epithelial cell apoptosis/necrosis. Plasma proteins and other substances in permeability edema enter the alveoli and inactivate (inhibit) lung surfactant, exacerbating respiratory deficits. Lung contusion injury can also induce fibroblast activation and proliferation, although the mechanistic contributions of these cells to the progression of acute inflammatory injury are unclear. See text and subsequent sections for details. See Raghavendran et al., pages 122–130, 2009.