Injury, Inflammation, and Sepsis: Laboratory and Clinical Approaches


Volume 32, No. 2 August 2009

Commentary

What’s New in Shock, August 2009?

Review Article

Lung Contusion: Inflammatory Mechanisms and Interaction with Other Injuries

Basic Science Aspects

Targeted Delivery of siRNA to Cell Death Proteins in Sepsis

A Large-Bolus Injection, But Not Continuous Infusion of Sodium Selenite Improves Outcome in Peritonitis

Experimental Polymicrobial Peritonitis–Associated Transcriptional Regulation of Murine Endogenous Retroviruses

Simvastatin Reduces Endotoxin-Induced Nuclear Factor κB Activation and Mortality in Guinea Pigs Despite Lowering Circulating Low-Density Lipoprotein Cholesterol

Retinoid X Receptor α Participation in Dexamethasone-Induced Rat Bile Acid Coenzyme A–Amino Acid N-Acyltransferase Expression in Septic Liver

Mathematical Modeling of Posthemorrhage Inflammation in Mice: Studies Using a Novel, Computer-Controlled, Closed-Loop Hemorrhage Apparatus

Endothelin-1 Contributes to Hemoglobin Glutamer-200–Mediated Hepatocellular Dysfunction After Hemorrhagic Shock

Afferent Pathways Involved in Cardiovascular Adjustments Induced by Hypertonic Saline Resuscitation in Rats Submitted to Hemorrhagic Shock

Inverse Thermodilution with Conventional Pulmonary Artery Catheters for the Assessment of Cerebral, Hepatic, Renal, and Femoral Blood Flow
Volume 32, No. 2  August 2009

Xiang-Shun Xu, Zhi-Zhong Ma, Fang Wang, Bai-He Hu, Chuan-She Wang, Yu-Xing Liu, Xin-Rong Zhao, Li-Hua An, Xin Chang, Fu-Long Liao, Jing-Yu Fan, Hideyuki Niimi, and Jing-Yan Han  203  The Antioxidant Cerebralcare Granule Attenuates Cerebral Microcirculatory Disturbance During Ischemia-Reperfusion Injury

Chiung-Yuan Hsu, Chien-Hua Huang, Wei-Tien Chang, Huer-Wen Chen, Hsiao-Ju Cheng, Min-Shan Tsai, Tsung-Dau Wang, Zui-Shen Yen, Chien-Chung Lee, Shyr-Chyr Chen, and Wen-Jone Chen  212  Cardioprotective Effect of Therapeutic Hypothermia for Postresuscitation Myocardial Dysfunction

Tiziana Genovese, Emanuela Esposito, Emanuela Mazzon, Rosanna Di Paola, Rosaria Meli, Rocco Caminiti, Placido Bramanti, Mitchell P. Fink, and Salvatore Cuzzocrea  219  Beneficial Effects of Ethyl Pyruvate in a Mouse Model of Spinal Cord Injury

Chien-Hsing Ho, Su-Ping Hsu, Chih-Chin Yang, Yi-Huey Lee, and Chiang-Ting Chien  230  Sialic Acid Reduces Acute Endotoxemia-Induced Liver Dysfunction in the Rat

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Current Impact Factor 3.325

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 alveolo-capillary 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.