Commentary

What’s New in Shock, September 2008?

Basic Science Aspects

Effect of Thalidomide on Signal Transduction Pathways and Secondary Damage in Experimental Spinal Cord Trauma

Nitric Oxide Triggers Delayed Anesthetic Preconditioning-Induced Cardiac Protection via Activation of Nuclear Factor-κB and Upregulation of Inducible Nitric Oxide Synthase

The Peritoneum as a Novel Oxygenation Organ: Revitalization of Intraperitoneal Oxygenation

LFA-1 and MAC-1 Mediate Pulmonary Recruitment of Neutrophils and Tissue Damage in Abdominal Sepsis

KLF4 Promotes the Expression, Translocation and Release of HMGB1 in RAW264.7 Macrophages in Response to LPS

Induction of Endotoxin Tolerance Enhances Bacterial Clearance and Survival in Murine Polymicrobial Sepsis

YC-1 Induces Heat Shock Protein 70 Expression and Prevents Oxidized LDL-Mediated Apoptosis in Vascular Smooth Muscle Cells

Effect of Enteral Versus Parenteral Nutrition on Inflammation and Cardiac Function in a Rat Model of Endotoxin-Induced Sepsis

The Effect of Dobutamine on Platelet Aggregatory Function in Newborn Piglets with Hypoxia and Reoxygenation

Treatment with the Glycogen Synthase Kinase-3β Inhibitor, TDZD-8, Affects Transient Cerebral Ischemia/Reperfusion Injury in the Rat Hippocampus

Reduced Cytokine Production by Glycogen-Elicited Peritoneal Cells from Diabetic Rats
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**Cover:** Immunohistochemical expression of Bax and Bcl-2. No positive staining for Bax (A) and for Bcl-2 (E) was found in the spinal cord tissues from sham-operated mice. On the contrary, SCI caused, at 24 h, an increase in Bax expression (B). Thalidomide treatment reduced the degree of positive staining for Bax in the spinal cord (C). On the contrary, positive staining for Bcl-2 was observed in the spinal cord tissues of sham-operated mice (E). At 24 h after SCI, significantly less staining for Bcl-2 was observed (F). Thalidomide treatment attenuated the loss of positive staining for Bcl-2 in the spinal cord from SCI-subjected mice (G). See Genovese, et al., pages 231–240, 2008.