evolving history[J]. Hepatology, 2010, 52(5): 1829-1835.
[2] Margenthaler J A, Landeros K, Kataoka M, *et al*. Effects of endotoxin tolerance on Propionibacterium acnes-primed lipopolysaccharide hepatic injury[J]. J Surg Res, 2003, 112(1): 102-110.
[3] Dugo L, Collin M, Allen D A, *et al*. Insulin reduces the multiple organ injury and dysfunction caused by coadministration of lipopolysaccharide and peptidoglycan independently of blood glucose: role of glycogen synthase kinase-3beta inhibition[J]. Crit Care Med, 2006, 34(5): 1489-1496.
[4] 陈太忠, 张艳, 熊弢, 等. 脂多糖预处理对GSK-3功能活性的影响[J]. 第三军医大学学报, 2012, 34(13): 1304-1309.
[5] Amar S, Belmaker R H, Agam G. The possible involvement of glycogen synthase kinase-3 (GSK-3) in diabetes, cancer and central nervous system diseases[J]. Curr Pharm Des, 2011, 17(22): 2264-2277.
[6] Teng S, Kurata S, Katoh I, *et al*. Cytokine mRNA expression in unilateral ischemic-reperfused rat lung with salt solution supplemented with low-endotoxin or standard bovine serum albumin[J]. Am J Physiol Lung Cell Mol Physiol, 2004, 286(1): L137-L142.
[7] Valatas V, Xidakis C, Roumpaki H, *et al*. Isolation of rat Kupffer cells: a combined methodology for highly purified primary cultures[J]. Cell Biol Int, 2003, 27(1): 67-73.
[8] Filipov N M, Seegal R F, Lawrence D A. Manganese potentiates in vitro production of proinflammatory cytokines and nitric oxide by microglia through a nuclear factor kappa B-dependent mechanism[J]. Toxicol Sci, 2005, 84(1): 139-148.
[9] Reville K, Crean J K, Vivers S, *et al*. Lipoxin A4 redistributes myosin ⅡA and Cdc42 in macrophages: implications for phagocytosis of apoptotic leukocytes[J]. J Immunol, 2006, 176(3): 1878-1888.
[10] Decker K. The response of liver macrophages to inflammatory stimulation[J]. Keio J Med, 1998, 47(1): 1-9.
[11] Deng X, Luyendyk J P, Zou W, *et al*. Neutrophil interaction with the hemostatic system contributes to liver injury in rats cotreated with lipopolysaccharide and ranitidine[J]. J Pharmacol Exp Ther, 2007, 322(2): 852-861.
[12] Yee S B, Hanumegowda U M, Hotchkiss J A, *et al*. Role of neutrophils in the synergistic liver injury from monocrotaline and bacterial lipopolysaccharide exposure[J]. Toxicol Sci, 2003, 72(1): 43-56.
[13] Jirillo E, Caccavo D, Magrone T, *et al*. The role of the liver in the response to LPS: experimental and clinical findings[J]. J Endotoxin Res, 2002, 8(5): 319-327.
[14] Louis H, Le-Moine O, Goldman M, *et al*. Modulation of liver injury by interleukin-10[J]. Acta Gastroenterol Belg, 2003, 66(1): 7-14.
[15] Neyrinck A M, Alexiou H, Delzenne N M. Kupffer cell activity is involved in the hepatoprotective effect of dietary oligofructose in rats with endotoxic shock[J]. J Nutr, 2004, 134(5): 1124-1129.
[16] Deng T, Feng X, Liu P, *et al*. Toll-like receptor 3 activation differentially regulates phagocytosis of bacteria and apoptotic neutrophils by mouse peritoneal macrophages[J]. Immunol Cell Biol, 2013, 91(1): 52-59.
[17] Blackwell T S, Blackwell T R, Christman J W. Induction of endotoxin tolerance depletes nuclear factor-kappaB and suppresses its activation in rat alveolar macrophages[J]. J Leukoc Biol, 1997, 62(6): 885-891.
[18] Fujihara M, Wakamoto S, Ito T, *et al*. Lipopolysaccharide-triggered desensitization of TNF-alpha mRNA expression involves lack of phosphorylation of IkappaBalpha in a murine macrophage-like cell line, P388D1[J]. J Leukoc Biol, 2000, 68(2): 267-276.
[19] Ziegler-Heitbrock H W, Wedel A, Schraut W, *et al*. Tolerance to lipopolysaccharide involves mobilization of nuclear factor kappa B with predominance of p50 homodimers[J]. J Biol Chem, 1994, 269(25): 17001-17004.
[20] Gotschel F, Kern C, Lang S, *et al*. Inhibition of GSK3 differentially modulates NF-kappaB, CREB, AP-1 and beta-catenin signaling in hepatocytes, but fails to promote TNF-alpha-induced apoptosis[J]. Exp Cell Res, 2008, 314(6): 1351-1366.

(**收稿**:2013-01-29;**修回**:2013-03-12)
(编辑 王 红)

“第二届全国病毒感染与器官功能衰竭学术会议”暨“医学病毒学系列学术论坛——病毒感染与器官功能衰竭”征文通知

由中华医学会医学病毒学分会、解放军传染病专业委员会、中华医学会热带病与寄生虫病学分会、解放军微生物专业委员会主办,第三军医大学西南医院、解放军302医院承办的“第二届全国病毒感染与器官功能衰竭学术会议”暨“医学病毒学系列学术论坛——病毒感染与器官功能衰竭”将于2013年11月下旬在美丽的山城——重庆召开。大会将邀请国内外专家和同行就病毒感染与器官功能衰竭领域的基础与临床研究进展进行学术交流,促进本领域基础与临床的密切结合和成果转化。会议将设专题报告、大会发言和书面交流三种形式,热烈欢迎相关领域各级临床医师和基础研究人员踊跃投稿并参加会议交流。

征文范围:(1)病毒感染与器官衰竭的基础与临床研究;(2)病毒感染病毒学、免疫学、流行病学、临床研究与病案报道;(3)新发病毒传染病防治研究。

征文要求:中、英文全文或大摘要一份(800~1 000字)。内容包括题目、作者、单位及邮政编码、联系方式(电话和E-mail)、目的和背景、材料与方法、结果、讨论与结论。

截稿日期:2013年10月15日
投稿要求:论文请以电子邮件发至E-mail:opliuming@126.com
联系人:刘 明
电 话:(023)68765218,传真:(023)68754858

(中华医学会医学病毒学分会)
2013年4月12日