

CORRIGENDUM

DOI: 10.3892/ijmm.2016.2707

TLR4-mediated NF- κ B signaling pathway mediates HMGB1-induced pancreatic injury in mice with severe acute pancreatitis

GANG LI, XUEJUN WU, LE YANG, YUXIANG HE, YANG LIU, XING JIN and HAI YUAN

Int J Mol Med 37: 99-107, 2016; DOI: 10.3892/ijmm.2015.2410

Following the publication of the article, the authors noted that there were certain errors in Fig. 5. In particular, the published Fig. 5A-a contains an image of H&E-stained micrographs from wild-type mice after the administration of a low dose of rhHMGB1, and not after the administration of a high dose of rhHMGB1, as should be shown. In addition, the data in the published graph of Fig. 5C-a is not correct and in Fig. 5C-b, the significance marker is merged with the error bars and is not evident. Shown below is the new version of Fig. 5 with the correct images:

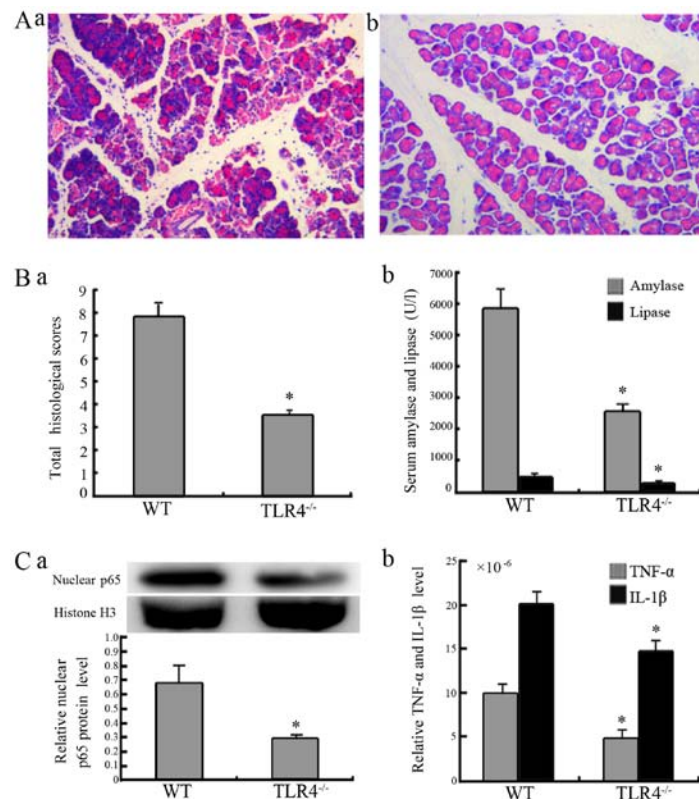


Figure 5. Response of Toll-like receptor 4 (TLR4)-deficient mice to recombinant human high-mobility group box 1 (rhHMGB1) administration. (A) Pancreatic histopathological changes in wild-type (WT) and TLR4-deficient mice 48 h after the administration of a high dose of rhHMGB1. Representative H&E-stained micrographs (original magnification, $\times 200$) from the (a) WT and (b) TLR4^{-/-} groups. The pancreatic tissues in the wild-type group exhibited marked edema, infiltration of inflammatory cells, a mass of necrotic acinar cells and the disappearance of the normal lobe structure in the pancreas; there was a significant reduction in the TLR4-deficient group. (B-a) Histological scores of the pancreas and (b) serum amylase and lipase changes in the WT and TLR4^{-/-} groups. (C) The activation of nuclear factor- κ B (NF- κ B) was assessed from the nuclear p65 [(a) western blot analysis] and its downstream, relative levels of tumor necrosis factor- α (TNF- α) and interleukin-1 β (IL-1 β) [(b) ELISA] in the pancreatic tissue from the WT and TLR4^{-/-} groups. * $P < 0.01$ compared to the WT group. The relative levels of nuclear p65 protein compared to the control group are normalized to histone 3 (H3). The relative levels of TNF- α and IL-1 β compared to the control group are normalized to the total protein concentration of each sample. Data are expressed as the means \pm SD ($n = 6$ /group). Blots shown are from a representative experiment that was repeated 3 times with similar results ($n = 6$ /group). WT, WT mice given a high dose of rhHMGB1; TLR4^{-/-}, TLR4-deficient mice administered a high dose of rhHMGB1.