

## Erratum

# Mannose-capped lipoarabinomannan from *Mycobacterium tuberculosis* induces IL-37 production via upregulating ERK1/2 and p38 in human type II alveolar epithelial cells: Int J Clin Exp Med 2015; 8: 7279-7287

Zhen Huang<sup>1,2</sup>, Gao Wei Zhao<sup>3</sup>, Chun Hai Gao<sup>4</sup>, Xiu Wen Chi<sup>5</sup>, Tao Zeng<sup>6</sup>, Yan Wei Hu<sup>1</sup>, Lei Zheng<sup>1</sup>, Qian Wang<sup>1</sup>

<sup>1</sup>Laboratory Medicine Center, Nanfang Hospital, Southern Medical University, Guangzhou 510515, Guangdong, P. R. China; <sup>2</sup>Department of Laboratory Medicine, Longgang Central District Hospital, Shenzhen 518116, Guangdong, P. R. China; <sup>3</sup>Linyi Blood Center, Linyi 276000, Shandong, P. R. China; <sup>4</sup>Department of Laboratory Medicine, People's Hospital of Linyi, Linyi 276003, Shandong, P. R. China; <sup>5</sup>School of Nursing, Guangdong Medical College, Dongguan 523808, Guangdong, P. R. China; <sup>6</sup>School of Laboratory Medicine, Guangdong Medical College, Dongguan 523808, Guangdong, P. R. China

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**Abstract:** The major surface lipoglycan of *Mycobacterium tuberculosis* (M. tb), mannose-capped lipoarabinomannan (ManLAM), is an immunosuppressive epitope of M. tb. Interleukin (IL)-37, is a newly identified anti-inflammatory cytokine, which reduces systemic and local inflammation. However, the correlation between ManLAM and IL-37 remains unknown. Therefore, in this study, we investigate the possible role and relative molecular mechanism of ManLAM in IL-37 production of human type II alveolar epithelial cells by using A549 cell line. Here, we report that M. tb induced IL-37 mRNA and protein expression in a time-dependent manner. We next fractionated components of M. tb using chloroform:methanol (C:M) and water. In sharp contrast to the C:M phase, water phase was mainly responsible for the production of IL-37. Since ManLAM is the major component of water phase, we found that ManLAM induced IL-37 mRNA and protein expression in a time and dose-dependent manner, while this activity was almost totally abolished by the ERK1/2 (U0126) and p38 (SB203580) inhibitor. ManLAM stimulation significantly induced ERK1/2 and p38 phosphorylation in A549 cells, as well as cell surface TLR2 expression. After interfering TLR2 expression, ERK1/2 and p38 phosphorylation levels were markedly decreased, and also IL-37 production. Though ManLAM also promoted TLR4 expression on A549 cells, TLR4 interference showed no influence on ManLAM-induced IL-37 production. Our results indicate that ManLAM induces IL-37 production in human type II alveolar epithelial cells via up-regulating TLR2/p38 or ERK1/2 pathway, and this provide an important evidence to explain the pathological role of ManLAM that contribute to the persistence of M. tb.

**Keywords:** ManLAM, tuberculosis, IL-37, TLR2, ERK1/2, p38

We apologize for missing addressing the corresponding author of the Letter. The corresponding author's information is "Correspondence to: Qian Wang, Laboratory Medicine Center, Nanfang Hospital, Southern Medical University, Guangzhou, Guangdong 510515, China. E-mail: 1505700787@qq.com".

**Address correspondence to:** Dr. Qian Wang, Laboratory Medicine Center, Nanfang Hospital, Southern Medical University, 1023 Shatai South Road, Guangzhou 510515, Guangdong, P. R. China. E-mail: 1505700787@qq.com