SCIENTIFIC REPORTS

Published online: 19 November 2018

OPEN Author Correction: Discovery of small molecule inhibitors of MyD88dependent signaling pathways using a computational screen

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Correction to: Scientific Reports https://doi.org/10.1038/srep14246, published online 18 September 2015

This Article contains errors.

In Figure 5b, for the experiments with increasing dosage of TIR the labels were listed in the reverse order. The x-axis labels of Figure 5b should read as follows; lane 10 -13 will be LPS + T6167923 + TIR ($12.5 \mu g$), LPS + T6167923 + TIR (25 µg), LPS + T6167923 + TIR (50 µg), LPS + T6167923 + TIR (100 µg), lane 16 -19 will be LPS + T5996207 + TIR (12.5 μg), LPS + T5996207 + TIR(25 μg), LPS + T5996207 + TIR (50 μg), LPS + T5996207 + TIR(100 µg) and lane 22-25 LPS + T59910047 + TIR (12.5 µg), LPS + T59910047 + TIR $(25 \mu g)$, LPS + T59910047 + TIR $(50 \mu g)$, LPS + T59910047 + TIR $(100 \mu g)$, respectively. The corrected Figure 5b is published below.

This change does not affect the conclusions of the Article.

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Figure 5. Dose-dependent reduction of secreted alkaline phosphatase response (SEAP) via inhibition of specific MyD88-mediated signaling after LPS stimulation: comparison of original hit and 2^{nd} generation compounds. Compounds were tested by monitoring LPS-induced SEAP activity via a MyD88-mediated NF-kB driven signaling pathway. HEK 293 stable transfected cell line (TLR4-MD2-NF-kB-SEAP) was activated with LPS (TLR4 ligand) and treated with varying concentrations of compounds (500 μ M to 10 μ M). Culture supernatants were tested for SEAP activity and compared to levels in the absence of compounds. (a) Data are presented as SEAP response units. To determine the compounds inhibit MyD88-signaling by direct binding to TIR domain, the compounds T5910047, T6167923 and T5996207 were pre-incubated at room temperature with varying concentration 100 μ g, 50 μ g, 25 μ g and 12.5 μ g and compounds 100 μ M). Culture supernatants were tested for SEAP activity and compared to levels of compounds.

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