

CORRECTION

Correction: Sirtuin-2 Regulates Sepsis Inflammation in *ob/ob* Mice

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Bar graphs are missing from Fig 3A of the published article. Please see the correct [Fig 3](#) here.



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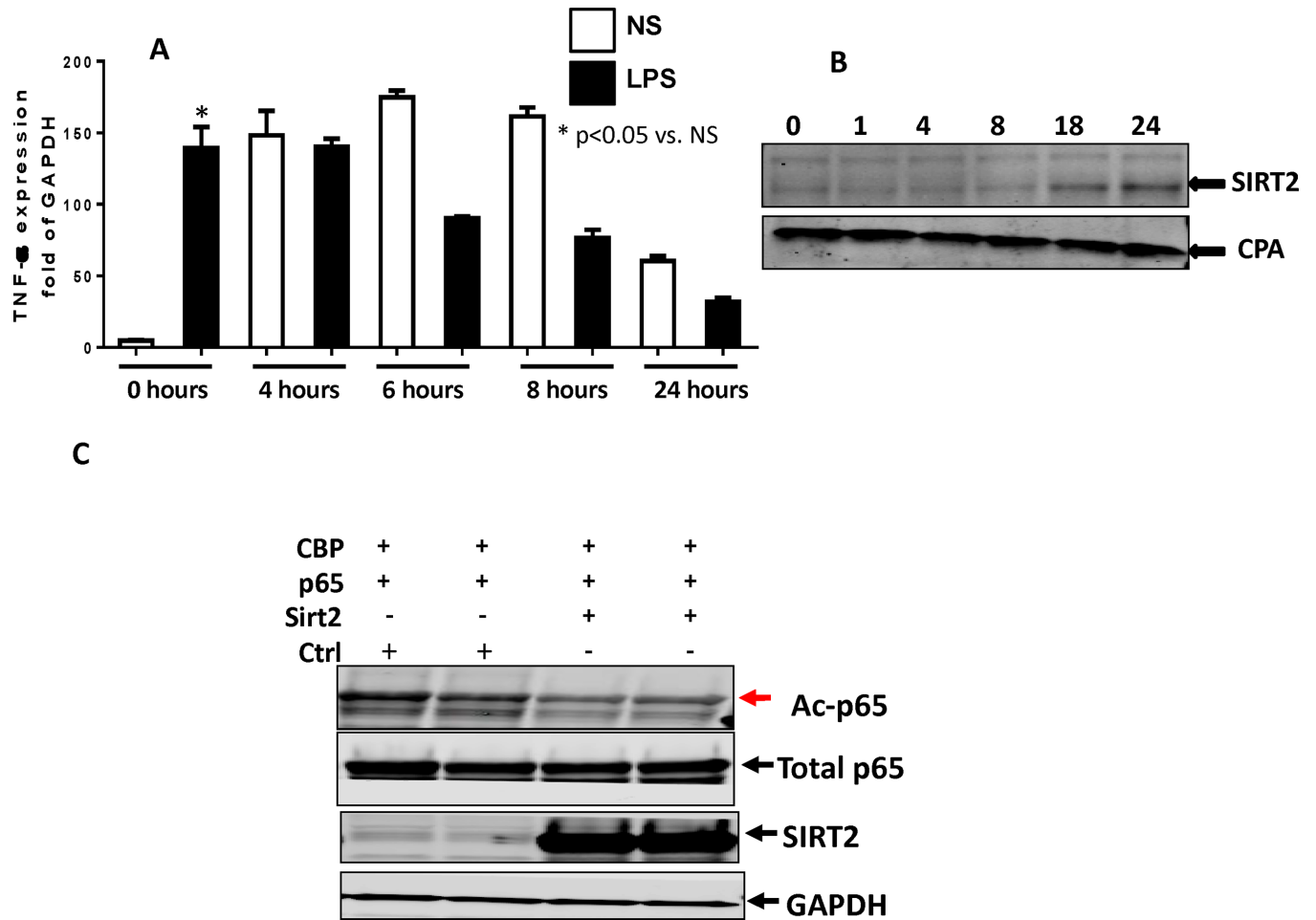


Fig 3. Role of SIRT-2 in the endotoxin tolerant RAW cells. A: RAW cells undergo endotoxin tolerance as early as 4h after LPS stimulation: To study endotoxin tolerance in RAW cells, we stimulated cells with LPS (100ng/ml) and re-stimulated with another LPS challenge (100ng/ml) at 0, 4, 6, 8, and 24 hour time points for four hour. We studied TNF- α mRNA expression. The cells increased TNF- α mRNA expression in response to LPS re-stimulation only at 0h time point. RAW cells were unable to increase TNF- α mRNA further to LPS re-stimulation at 4, 6, 8 and 24h time points, indicating endotoxin tolerance. * $p < 0.05$ vs. respective NS group Tukey's post-hoc analysis; error bars: s.e.m. B: SIRT-2 protein expression increased during endotoxin tolerant phase in RAW cells: RAW cells were treated with LPS (100ng/ml) for 0, 1, 4, 8, 18, and 24 h. Whole cell lysates were collected for western blotting of proteins SIRT-2 and CPA (housekeeping gene). Representative image out of three experiments shows that was increased in SIRT-2 expression in 18 and 24h after LPS stimulation. C SIRT-2 deacetylates NF κ B p65: We studied the effect of SIRT-2 expression on NF κ B p65 acetylation using HEK293 cells. SIRT-2 plasmid was co-transfected with p65 or/and CBP plasmids into HEK293 cells (to increase baseline p65 acetylation) and blotted for antibodies against Ac-p65, total p65, SIRT-2 and GAPDH. NF κ B p65 acetylation (Ac-p65) increased in cells with transfection with p65+CBP while it decreased in cells transfected with p65+CBP+SIRT-2, indicating SIRT-2 directly deacetylates NF κ B p65.

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Reference

1. Wang X, Buechler NL, Martin A, Wells J, Yoza B, McCall CE, et al. (2016) Sirtuin-2 Regulates Sepsis Inflammation in *ob/ob* Mice. PLoS ONE 11(8): e0160431. doi: [10.1371/journal.pone.0160431](https://doi.org/10.1371/journal.pone.0160431) PMID: [27500833](https://pubmed.ncbi.nlm.nih.gov/27500833/)