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Erratum

TLR4-mediated inflammation promotes foam cell formation of vascular smooth muscle cell by upregulating ACAT1 expression

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Since the publication of this paper, it has been noted that the labelling WT and TLR4^{-/-} was missing from the bottom of Figure 7g in the pdf file.

The corrected article appears online together with this erratum. The publishers apologize for any inconvenience this may have caused.

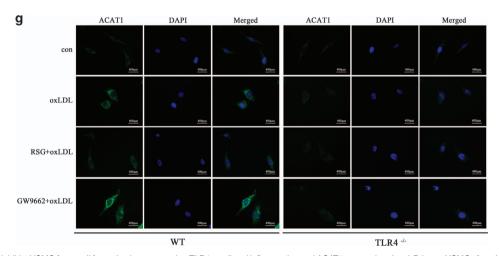


Figure 7 PPARy inhibits VSMC foam cell formation by suppressing TLR4-mediated inflammation and ACAT1 expression. (a-g) Primary VSMCs from WT and TLR4--mice were treated with oxLDL (80 μg/ml) for 24 h in the presence of RSG (50 μM) or GW9662 (10 μM). RSG significantly inhibited the oxLDL-induced lipid droplet accumulation (a) and intracellular cholesterol elevation (b) in VSMCs from WT mice, whereas GW9662 exposure exerted the opposite effect. In contrast, oxLDL failed to increase lipid droplet accumulation (a) and intracellular cholesterol level (b) in VSMCs from TLR4^{-/-}mice. Neither RSG nor GW9662 exerted detectable impact on lipid droplet accumulation (a) and intracellular cholesterol level (b) in VSMCs from TLR4-/- mice. RSG significantly inhibited, whereas GW9662 further promoted, the oxLDL-induced TLR4 (c) and proinflammatory cytokine (d) expressions. The same effect of PPAR γ was also observed in MyD88, NF- κ B p65 (nuclei) and p- $l\kappa$ B α expressions in oxLDL-loaded VSMCs (e). In contrast, RSG or GW9662 exposure exerted no effect on the expressions of MyD88, NF-kB p65 (nuclei), p-lkB\alpha and proinflammatory cytokines in VSMCs from TLR4^{-/-} mice. RSG and GW9662, respectively, suppressed and promoted the oxLDL-induced ACAT1 expression in VSMCs from WT mice, but it was not the case in VSMCs from TLR4-/mice (f and g) (*P<0.05 versus control WT-VSMCs; *P<0.05 versus WT-VSMCs with oxLDL challenge). Results were presented as mean ± S.D. (error bars) of three independent experiments