

Electronic Supplementary Information

Agonistic and antagonistic properties of a *Rhizobium sin-1* lipid A modified by an ether-linked lipid

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This pdf file includes:

Figs. S1 and S2

NMR spectra synthetic compounds

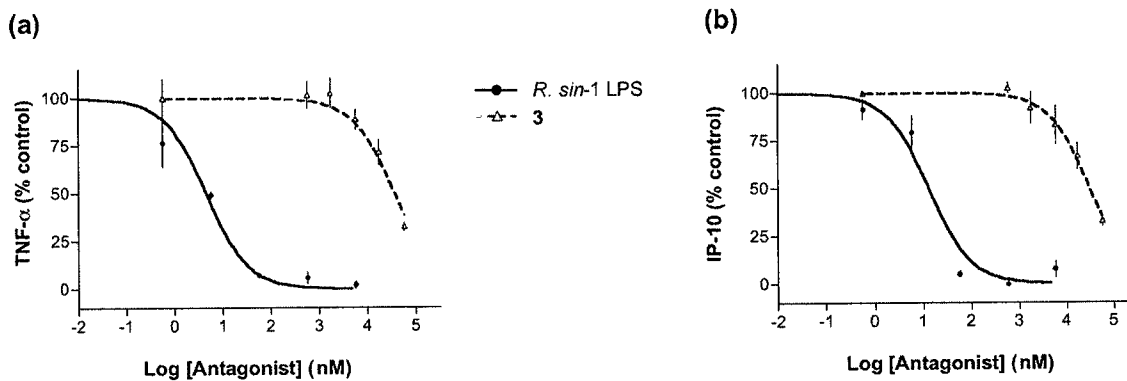


Fig. S1 Antagonism of synthetic *E. coli* lipid A by *R. sin-1* LPS and synthetic compound 3 in human monocytic cells. TNF- α (a) and IP-10 (b) concentrations were measured after preincubation of MM6 cells with increasing concentrations of *R. sin-1* LPS or 3 as indicated for 1 h at 37°C, followed by 5.5 h of incubation with *E. coli* lipid A (100 ng mL⁻¹). Results are expressed as percentage of cytokine concentration of control cells, which are incubated only with *E. coli* lipid A.

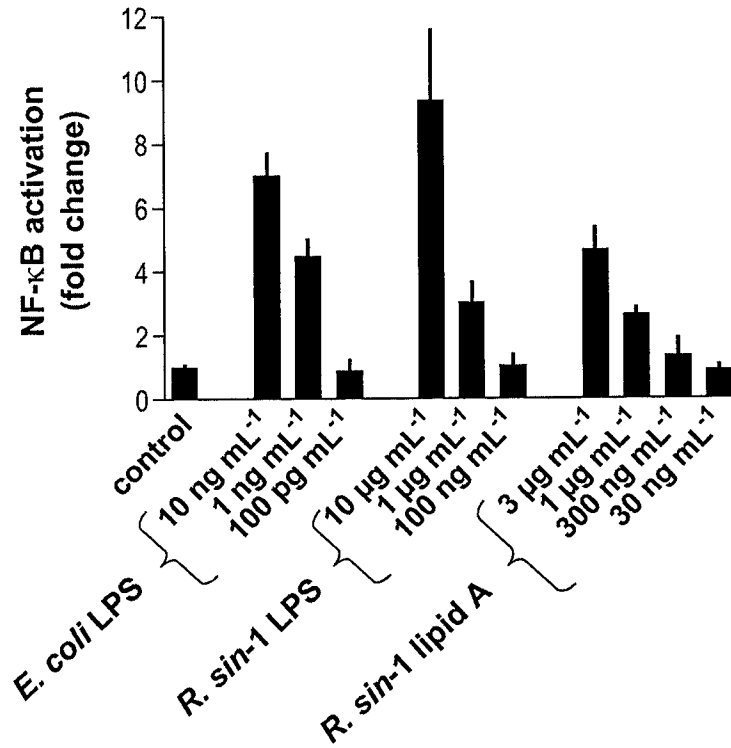


Fig. S2 Response of HEK 293T cells expressing murine TLR4/MD2 to *E. coli* LPS, *R. sin-1* LPS and *R. sin-1* lipid A. Induction of NF-κB activation was determined in triplicate cultures of HEK 293T cells stably transfected with murine TLR4/MD2 and transiently transfected with pELAM-Luc, pRL-TK and pcDNA3 plasmids. Forty-four h post-transfection, cells were treated with *E. coli* LPS, *R. sin-1* LPS and *R. sin-1* lipid A at the indicated concentrations or were left untreated (control). Forty-eight h post-transfection, NF-κB activation was determined by firefly luciferase activity relative to *Renilla* luciferase activity. In the transfection experiment shown, human TNF-α (10 ng mL⁻¹) induced 24.5 ± 0.6-fold activation of NF-κB.

