Tyrosine kinase inhibitors of Ripk2 attenuate bacterial cell wallmediated lipolysis, inflammation and dysglycemia

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Supplementary Figure 1. Time course of glycerol release in 3T3-L1 adipocytes pre-incubated with gefitinib and stimulated with the NOD1 ligand FK565 ($10\mu g/mL$), n=4-12 (A). Changes in gene expression of molecular markers of lipid oxidation, transport and synthesis 24 h (B) and 48 h (C) after stimulation with the NOD1 ligand FK565 ($10\mu g/mL$) in 3T3-L1 adipocytes pre-incubated with gefitinib, n=7-8. Values are mean + SEM. * denotes statistically different from Control condition, # denotes statistically different from FK565 condition (p<0.05).



Supplementary Figure 2. Levels of II-6 secreted from Bone marrow-derived macrophages (BMDMs) are below the detectable level 48 hours after stimulation with the NOD1 ligand FK565 ($10\mu g/mL$). Levels of IL-6 secreted from BMDMs are stimulation with the Tlr4 ligand LPS (0.5 $\mu g/mL$) pre-incubated for 1h with gefitinib (A), n=5. Time course of FK565-stimulated or LPS-stimulated II-6 secretion (B), n=5. Values are mean mean + SEM. Different letters assigned to each condition (a, b, c) denote statistical differences between groups (p<0.05).



Supplementary Figure 3. Immunoblots of basal (ie. no insulin) and 100nM insulin-stimulated pAKT (Ser473; left panel), and total AKT (right panel) from 4 different blots (A-D). For each condition, n=2 basal and n=2 insulin-stimulated samples were included on each gel. 'Con' = Control, 'FK' = FK565 (10µg/mL) and 'gef +FK' = gefitinib (5µM) + FK565 (10µg/mL) Quantitative comparison was conducted between samples from all 4 blots, derived from the experiment and processed in parallel.