Supporting Information

Zhou et al. 10.1073/pnas.0904412106



Fig. S1. Effect of DIO on TLRs expression. (*A*) Total RNA was purified from untreated BMM Φ from lean (*LN*) or obese (*OB*) mice; the mRNA for *TLR4*, *TLR6*, or *TLR7* was measured by real-time RT-PCR. (*B*) BMM Φ from lean and obese mice were infected with *P. gingivalis* for 4 h, and the mRNA for *TLR4*, *TLR6*, or *TLR7* was detected by real-time RT-PCR. Data are expressed as mean \pm SEM (n = 3; *P < 0.05).



Fig. S2. Bio-Plex phosphorylation protein array. BMM Φ from lean (LN) and obese (OB) mice were stimulated with *P. gingivalis* for 15 min, 4 and 24 h; The phosphorylated proteins were quantitated in the cell lysates by using Bio-Plex phosphorylation protein array. Data are expressed as mean \pm SEM (n = 3; *P < 0.05).



Fig. S3. Cell death induced by TNF- α . BMM Φ were treated with TNF- α for 4 h. The effect of TNF- α on survival of BMM Φ was determined by trypan blue dye exclusion assay. Survival rate (%) of the treated BMM Φ was calculated by dividing the surviving number of treated BMM Φ by number of total BMM Φ at indicated dosages of TNF- α . Data are expressed as mean \pm SEM (n = 3; *P < 0.05).



Fig. S4. Model of TLR2 pathway by which DIO and FFAs modulates *P. gingivalis*-induced innate immune responses. DIO elevates serum TNF- α level and down-regulates TLR2 expression. FFAs and TNF- α induce CTMP via TLR2 and TNF- α receptor (*TNFR*), respectively, and the increased intracellular CTMP further inhibits Akt phosphorylation by binding to Akt. The reduction of TLR2 together with the inhibition of Akt in DIO subject results in attenuated innate immune responses by diminishing NF- κ B activation when DIO individual is challenged with *P. gingivalis*. TLR2 antagonizes TNF- α -induced CTMP, possibly through Akt-dependent inhibition of p38 or JNK. Arrow = activation; line ending in bar = inhibition.

AS PNAS