SUPPLEMENTAY FIGURE LEGENDS

Fig S1. Immunofluorescence staining of wild-type and ED1 strains with anti-S-layer antibody

Plate grown cells were fixed onto poly-l-lysine coated coverslips and fixed onto using 4% paraformaldehyde before blocking with PBS (50 mM $NaPO4/Na2PO4$, pH 7.4, 150 mM NaCl) plus 3% (w/v) BSA for 1 h at 25 ^{0}C. Primary anti- S-layer antibody (1/3000, v/v, in PBS) was added for 2 h before washing (2 x10 min, PBS), incubation with AlexaFluor-488/594- conjugated anti-rabbit secondary antibody, seen in green (1/1000, v/v, in PBS; Molecular Probes) (2 h, 25 ^{0}C), and further washing (3 x10 min, PBS). Coverslips were mounted using ProLong Gold DAPI Anti-fade reagent to highlight cellular DNA in blue (Molecular Probes), and visualized using a fluorescence microscope (Zeiss). Images were captured by a digital camera and processed using Axiovison LE software (Zeiss).

Fig. S2. *T. forsythia* surface glycosylation deficient ED1 mutant is more potent than the wild-type strain in inducing NF-kB activity. THP-1-Blue cells were stimulated in triplicate with 10 m.o.i each of either *T. forsythia* wild-type (Tf WT) or ED1 bacteria and the culture supernatants were assayed after 16 h for SEAP activity to assess NF-kB activation. *E. coli* LPS (EcLPS) was used as a positive agonist. Error bars indicate standard deviations. Data shown are representative of three independent experiments with similar results; statistically significant differences between the groups are indicated by asterisks (***, p<0.001; **, p<0.01; *, p< 0.05)

Fig. S3. PCR analysis of oral microbial samples from mice sham-infected, infected with wild-type or ED1 strains of *T. forsythia*. Agarose (1.5%) gel lanes show PCR reaction products with PCR primers specific to 16s rDNA from eubacteria or *T. forsythia*. Lanes M, 1 Kb Plus DNA ladder; +, positive control using *T. forsythia* genomic DNA as template; -, negative

control without bacterial DNA; 1 to 8, oral microbial samples from individual infected mice. The1.4-kb and 620-bp bands (indicated with arrows) represents universal eubacterial and *T. forsythia*-specific rDNA amplicons, respectively.

Fig. S4. *T. forsythia* surface glycans modulate TLR2-dpndendent cytokine expression in dendritic cells. Inflammatory cytokine levels (A. IL-1 β, IL-6 and IL-10; B. IL12p40, IL12p70-, IL-23) were examined by ELISA in supernatants of BMDC's from BALB/c or TLR2 deficient mice following challenge with either the wild-type (Tf WT) or the glycosylation deficient mutant (ED1) of *T. forsythia*. *E. coli* LPS (EcLPS) and Pam_3CSK_4 were used as agonists. The data show the means ± standard deviations of triplicate determinations in one of 3 independent sets of experiments that yielded similar results; *, $p < 0.05$; n. s; not significant