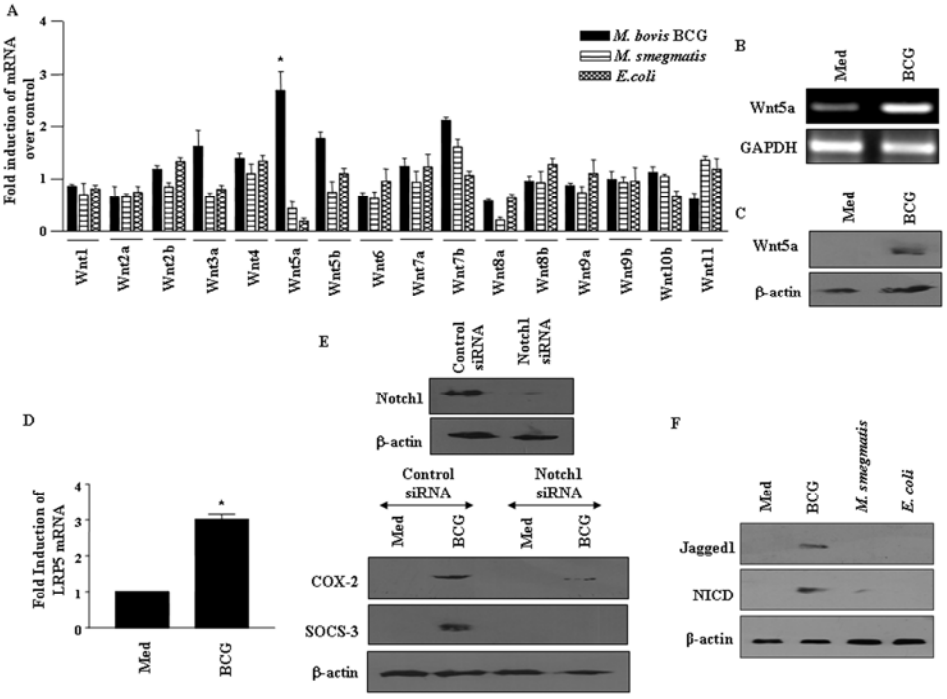
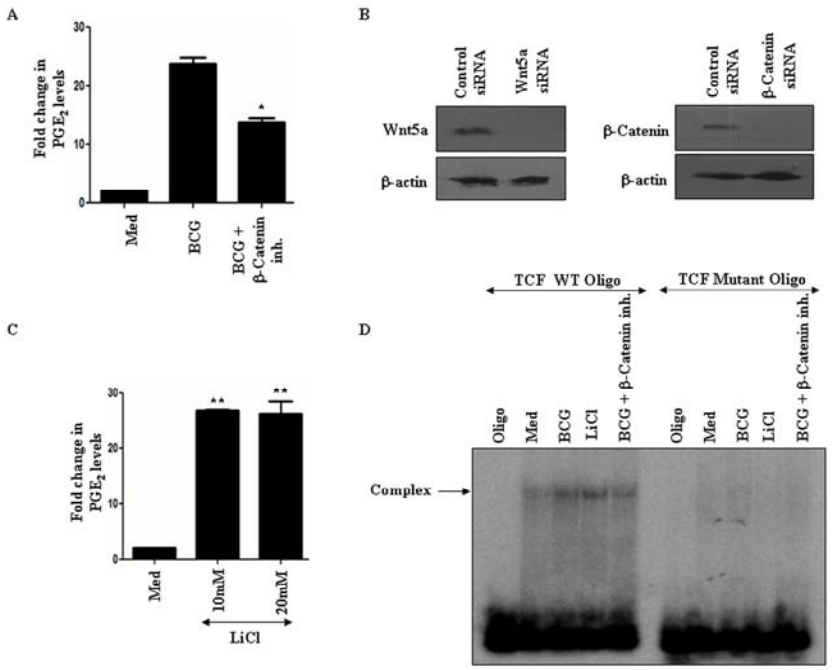
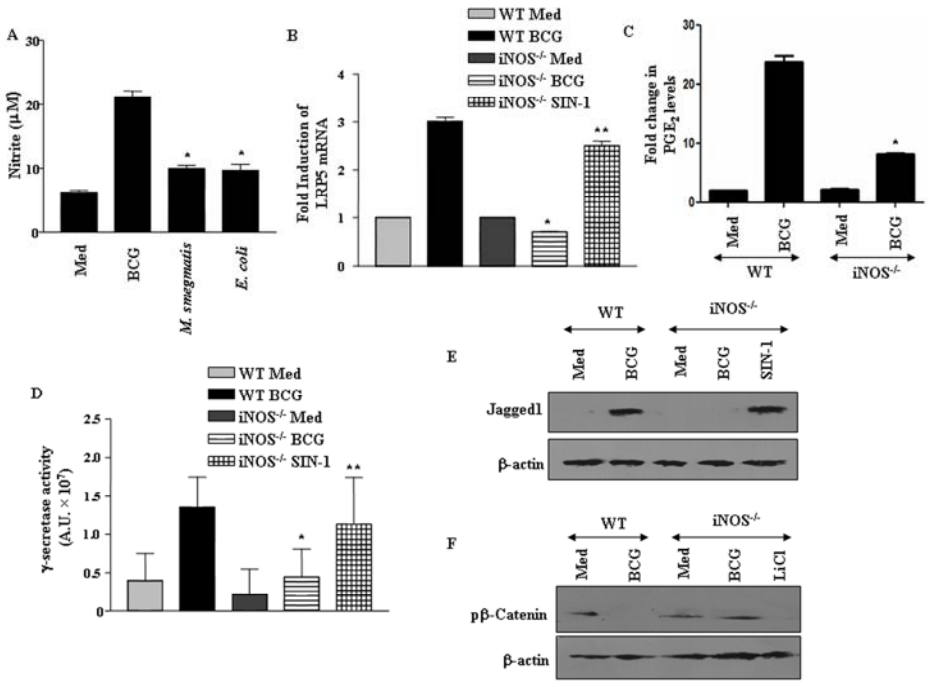


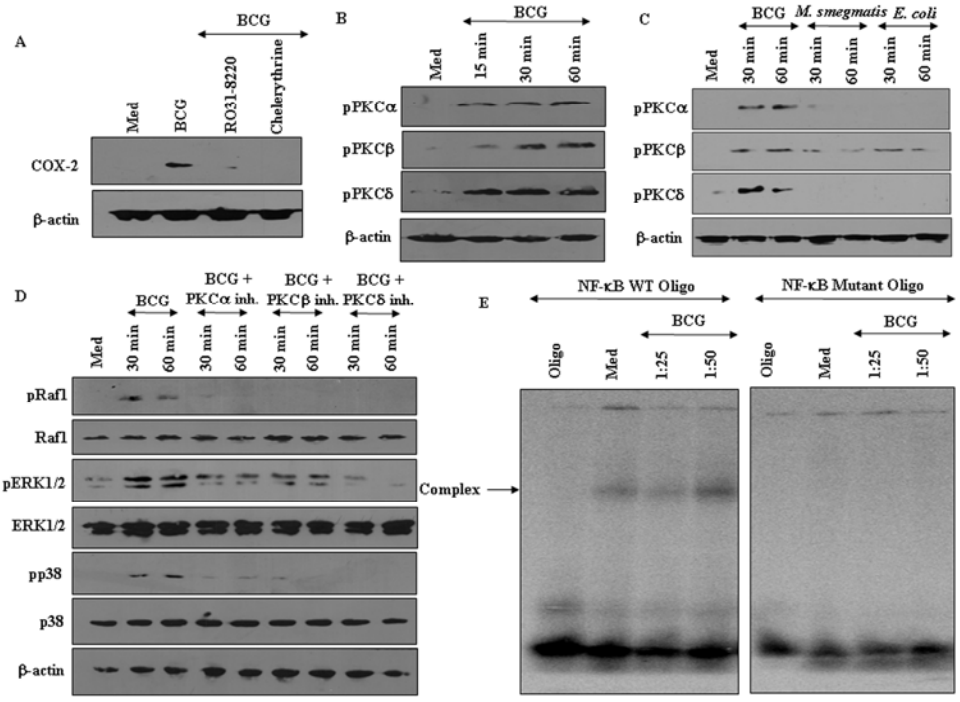
Supplemental FIGURE S1**.** *Mycobacterium* activates Wnt-β-Catenin and Notch1 Pathway in macrophages**.** ***A*****,** Expression analysis of Wnt family members in *M. bovis* BCG, *M. smegmatis* or *E. coli* infected macrophages**.** *B-D,* Infection with *M. bovis* BCG triggers expression of (*B* and *C*) Wnt5a and (*D*) LRP5 as analyzed by (*B*) semi-quantitative PCR, (*C*) immunoblotting and (*D*) quantitative real-time PCR respectively. *E*, siRNA-mediated knock down of Notch1 abrogates *M. bovis* BCG induced expression of COX-2 and SOCS-3. *F*, Compromised ability of *M. smegmatis* and *E. coli* to activate Notch1 signaling in macrophages. The error bars are representing mean ± SE. The data represents three independent experiments. *Med*, Medium. *, $P < 0.05$ *versus* Medium or Control.



Supplemental FIGURE S2. Wnt-β-Catenin signaling controls activation of Notch1 cascade during mycobacterial infection. *A*, Inhibition of β-Catenin abrogates secretion of PGE_2 in *M. bovis* BCG infected macrophages. *B*, siRNA-mediated knock down of Wnt5a and β-Catenin. *C*, LiCl treatment augments PGE_2 secretion. *D*, Treatment of macrophages with β-Catenin inhibitor diminishes *M. bovis* BCG triggered binding of TCF at β-Catenin/TCF binding site at mouse Jagged promoter. LiCl promotes binding of TCF at β-Catenin/TCF consensus. The error bars are representing mean ± SE. Data are representative of three independent experiments. *Med*, Medium. *, $P < 0.05$ *versus* BCG; **, $P < 0.05$ *versus* Medium.



Supplemental FIGURE S3. iNOS/NO is essential for TLR2 mediated regulation of Wnt-β-Catenin signaling. *A*, Infection with *M. bovis* BCG in comparison to *M. smegmatis* or *E. coli* promotes significant increase in NO levels in macrophages. *B*, Expression analysis of LRP5 transcript levels in WT and iNOS$^{-/-}$ macrophages infected with *M. bovis* BCG or iNOS$^{-/-}$ macrophages treated with SIN-1. *C*, Compromised ability of iNOS$^{-/-}$ macrophages to trigger secretion of PGE_2 upon infection with *M. bovis* BCG. *D* and *E*, (*D*) Assessment of γ-secretase activity or (*E*) Jagged1 expression in WT macrophages infected with *M. bovis* BCG and iNOS$^{-/-}$ macrophages infected with *M. bovis* BCG or treated with SIN-1. *F*, Phosphorylation status of β-Catenin in WT and iNOS$^{-/-}$ macrophages infected with *M. bovis* BCG or iNOS$^{-/-}$ macrophages treated with LiCl. The error bars are representing mean ± SE. The data are representing three independent experiments. *Med*, Medium; *WT*, Wild type. *, $P < 0.05$ *versus* WT BCG; **, $P < 0.05$ *versus* iNOS$^{-/-}$ BCG.



Supplemental FIGURE S4. Essential role for PKC-MAPK-NF-κB axis in execution of pathogen-specific TLR2 responses. *A*, Inhibition of PKC activity by Chelerythrine or RO31-8220 abolishes *M. bovis* BCG triggered COX-2 expression. *B*, Activation of PKCα, PKCβ and PKCδ by *M. bovis* BCG. *C*, Infection with *M. smegmatis* and *E. coli* fails to induce activation of PKCα, PKCβ and PKCδ. *D*, Pharmacological inhibition of PKCα, PKCβ and PKCδ abrogates *M. bovis* BCG induced activation of Raf1, ERK1/2 and p38 MAPK. *E*, *M. bovis* BCG promotes binding of NF-κB to its consensus as analyzed by gel-shift assay. The data represent two independent experiments. *Med*, Medium.