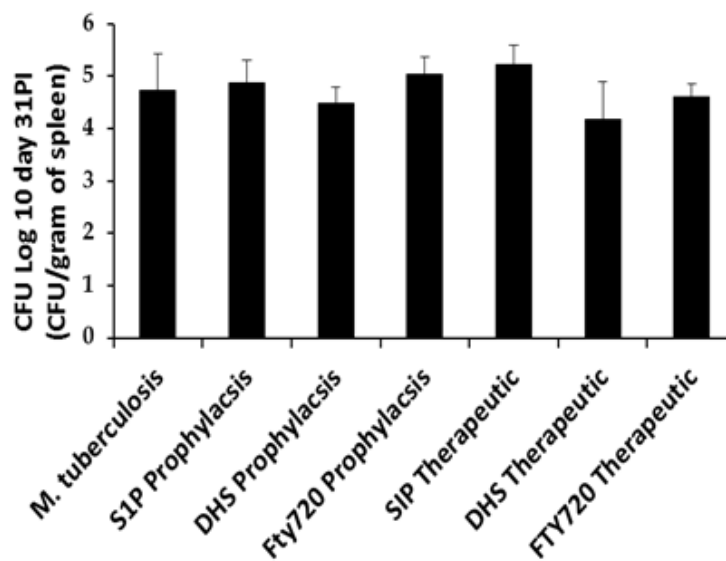


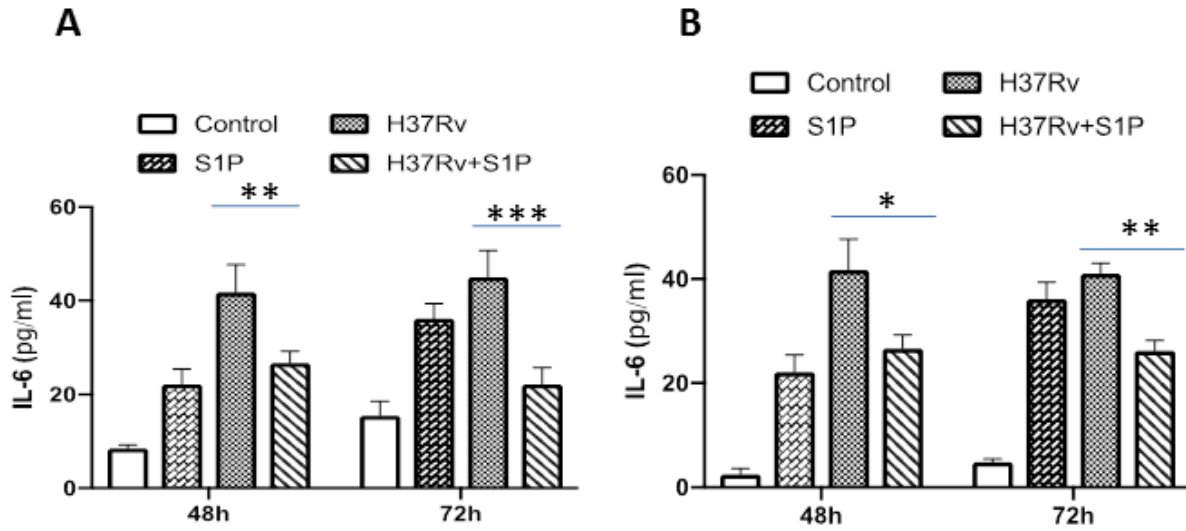
*Supplementary Material*

**Suppl Fig. 1**



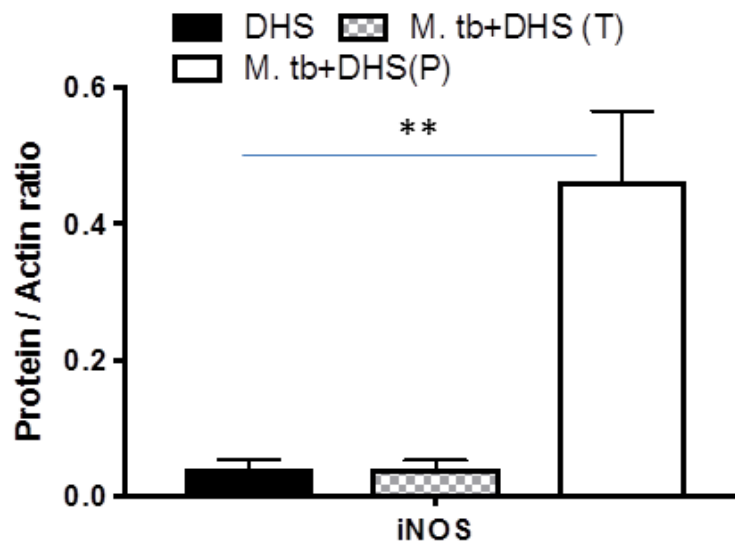
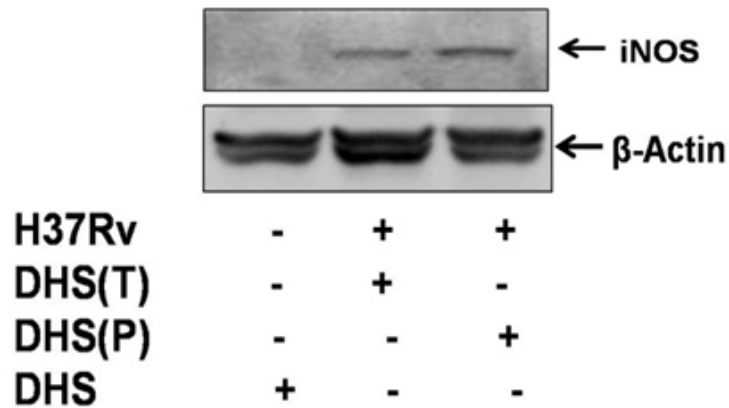
**Suppl. Figure1.** Spleens from mice challenged with *M. tb* in Figure 3A were excised and the numbers of bacterial colonies were analyzed using plate-based method. Shown here is CFU  $\pm$  SEM from several mice used in various indicated groups.

## Suppl Fig. 2



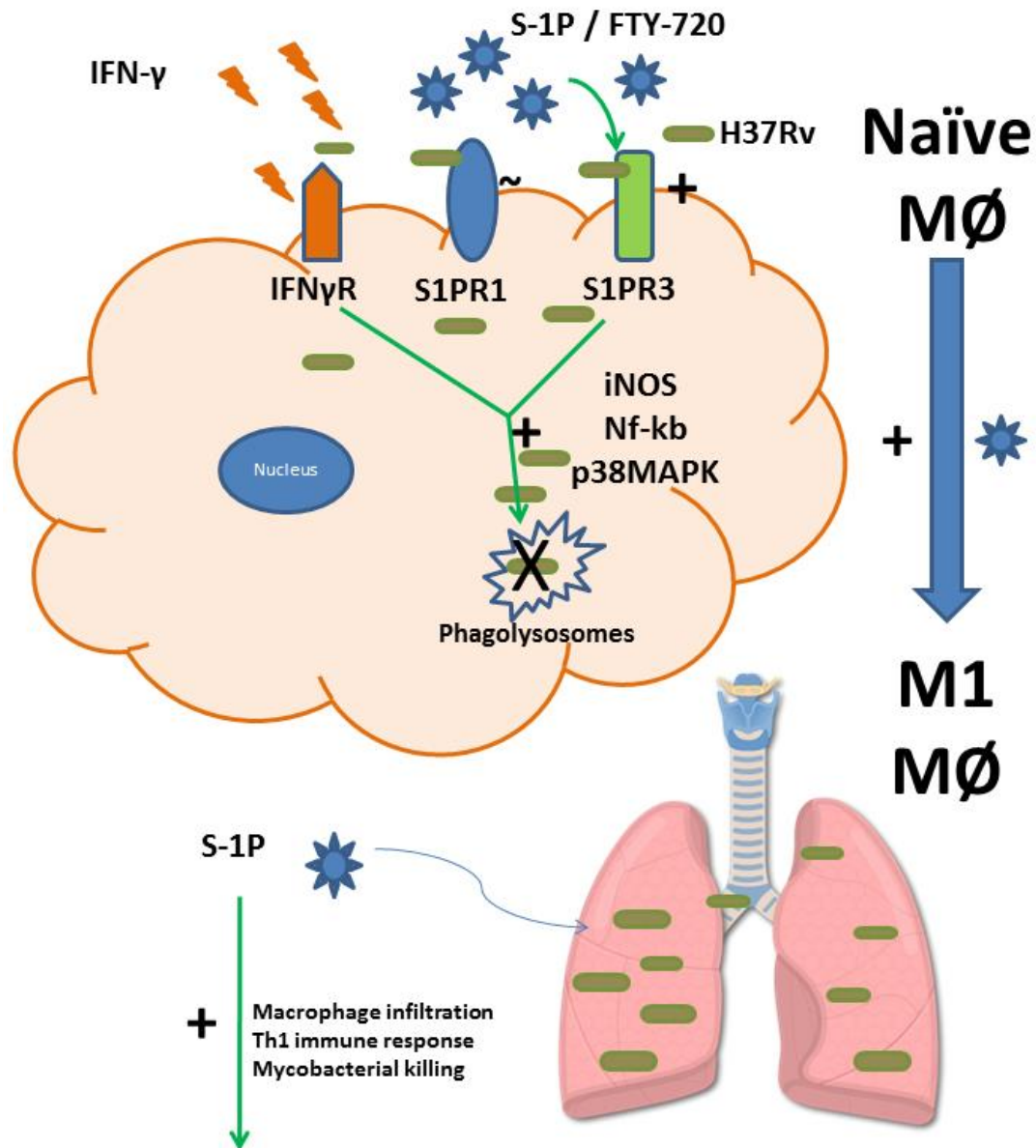
**Suppl. Figure 2.** RAW 264.7 macrophages and BMDM were infected with *M. tb* in presence of S-1P and IL-6 titre was quantified in their culture supernatants. Shown here is the pg/ml of IL-6  $\pm$  SEM released during the course of infection from three independent experiment. Statistical analysis were conducted using two-way Anova followed by Bonferroni post-test (\* $P < 0.05$ , \*\* $P < 0.01$ , \*\*\* $P < 0.001$ )

## Suppl Fig. 3



**Suppl. Figure 3.** Whole lung lysates from DHS treated mice were purified and analysed for M1 effector protein (iNOS) by western blot. Shown here are the representative blots from each groups.  $\beta$ -actin was used as a loading control. Densitometric analysis of the blot shown was quantified by ImageJ software and the values were plotted in terms of relative protein expression. Statistical analysis were conducted using two-way Anova followed by Bonferroni post-test (\* $P < 0.05$ , \*\* $P < 0.01$ , \*\*\* $P < 0.001$ ).

## Suppl Fig. 4



**Suppl. Figure 4.** Schematic representation of possible intra as well as extracellular S-1P receptor signalling components in macrophages and lung tissue for controlling infection, with special focus on S-1PR2 and S-1PR3 signaling for optimum defence against pathogenic mycobacteria.