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Fig. 1. Growth curves of 4 different *M. tuberculosis* strains in Middlebrook 7H9 broth

The 4 *M. tb* strains were inoculated into middlebrook 7H9 at $O.D_{600}$ 0.02 and cultures were shaked at 37°C incubator for 6 days. OD was monitored during the incubation time.

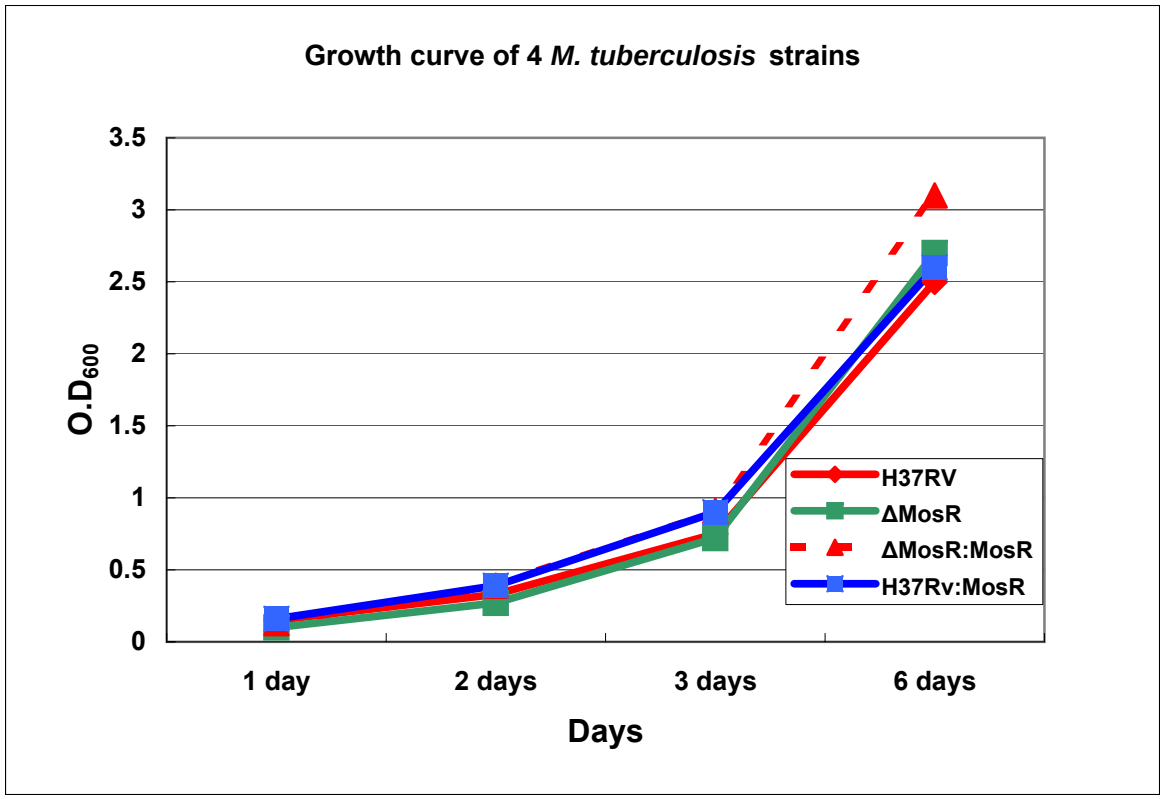


Fig. 2. Colony counts of 4 different *M. tuberculosis* strains subjected to differents stressors

Different *M. tb* strains were grown in Middlebrook 7H9 liquid medium to early log phase (OD_{600}=0.5) and their counts were determined. Aliquots of each strains (10 ml) were subjected to different stressor (SDS 0.05%, for 4 hrs) or to heat shock at 45°C for 24hrs and thier CFU were determined for all strains by plating on Middlebrook 7H10 plates at 37°C.

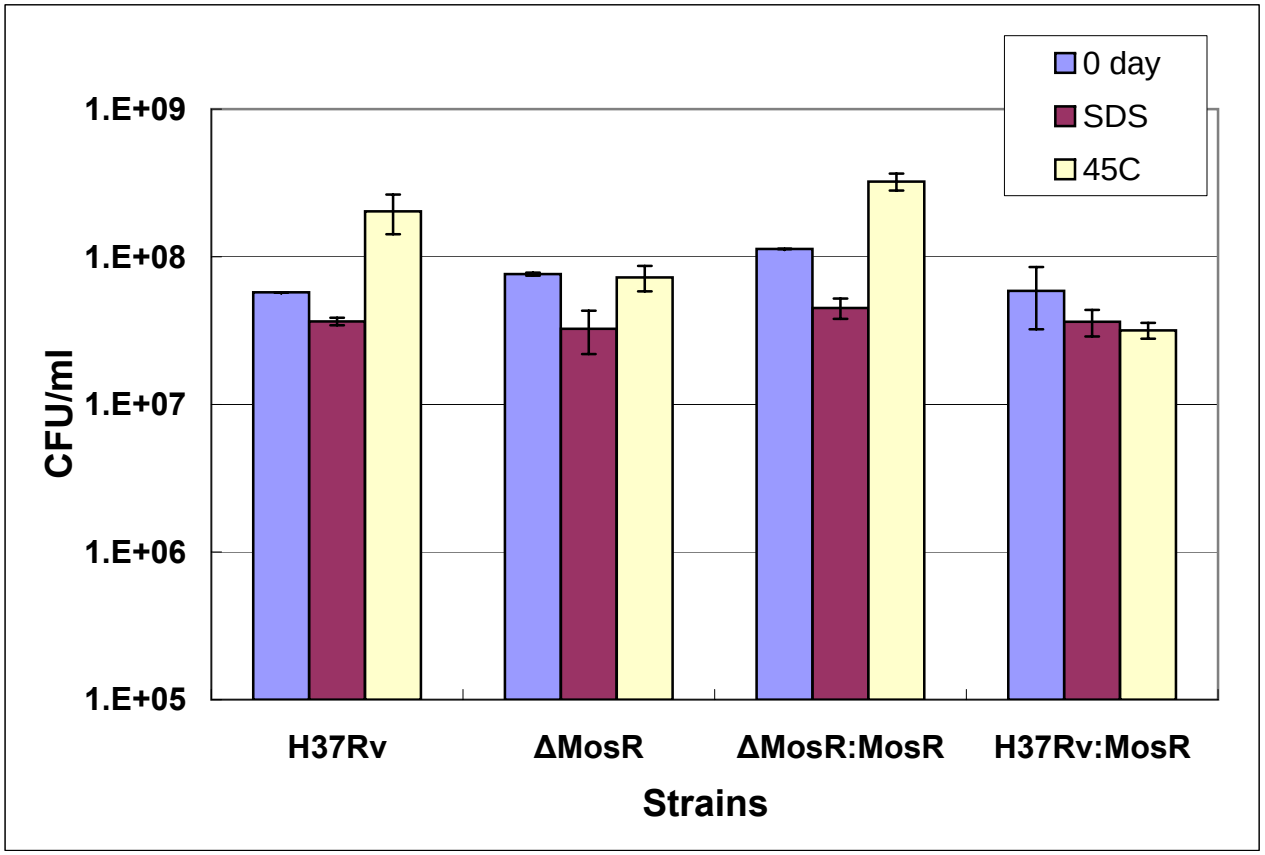
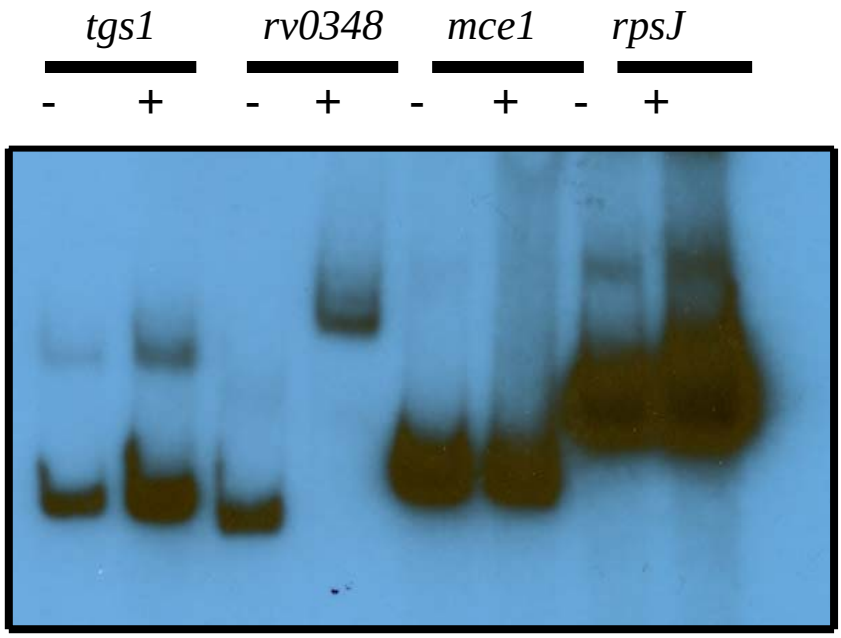


Fig. 3. EMSA assay with different promoter regions of upregulated and downregulated genes.

EMSA assay was performed as described by Talaat et al., 2007. Examples of some genes are shown below. Potential promoter regions of several selected genes were amplified by PCR and end-labelled by radioactive P32. The different probes were allowed to bind to recombinant MBP-Rv0348 and subsequently run on 4% native polyacrylamide gel. The gel was then dried and exposed to Kodak film.



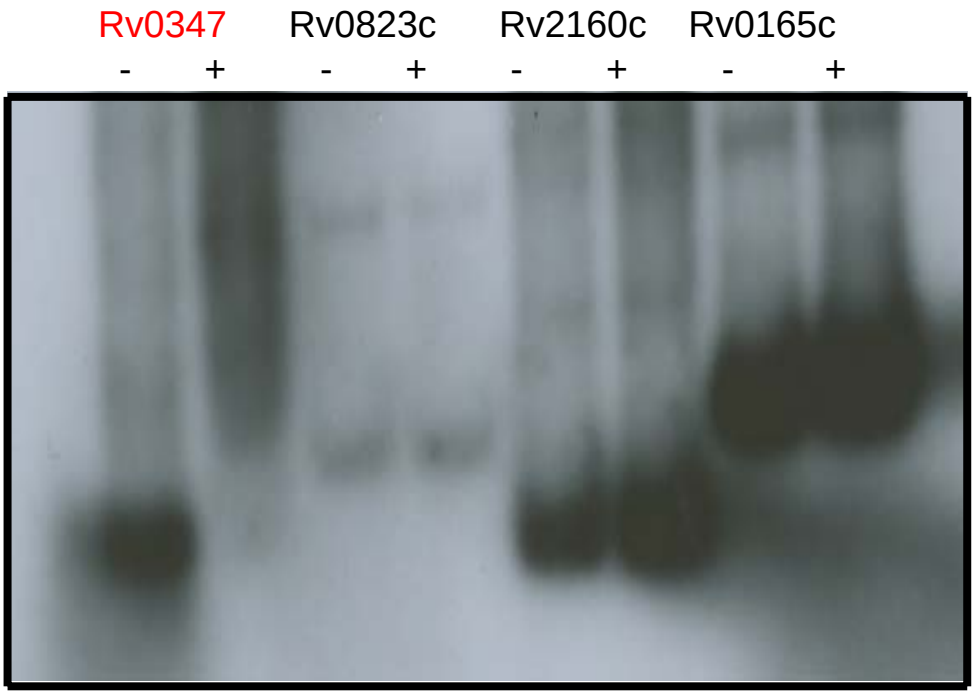

Rv0347
Rv0823c
Rv2160c
Rv0165c
- + - + - + - +

Fig. 4. Recombinant colonies of *M. smegmatis* without (left) or with (right) promoters for the target genes

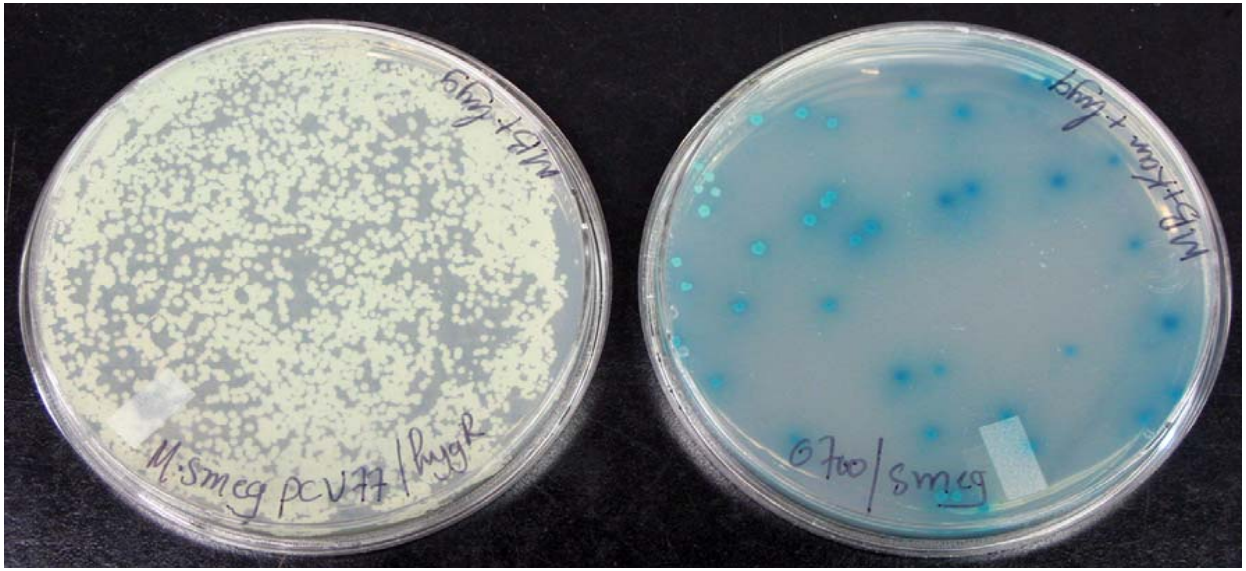


Fig. 5. β-galactosidase activity of aerobic cultures of recombinant M. smegmatis harboring pML28 with or without mosR operon.

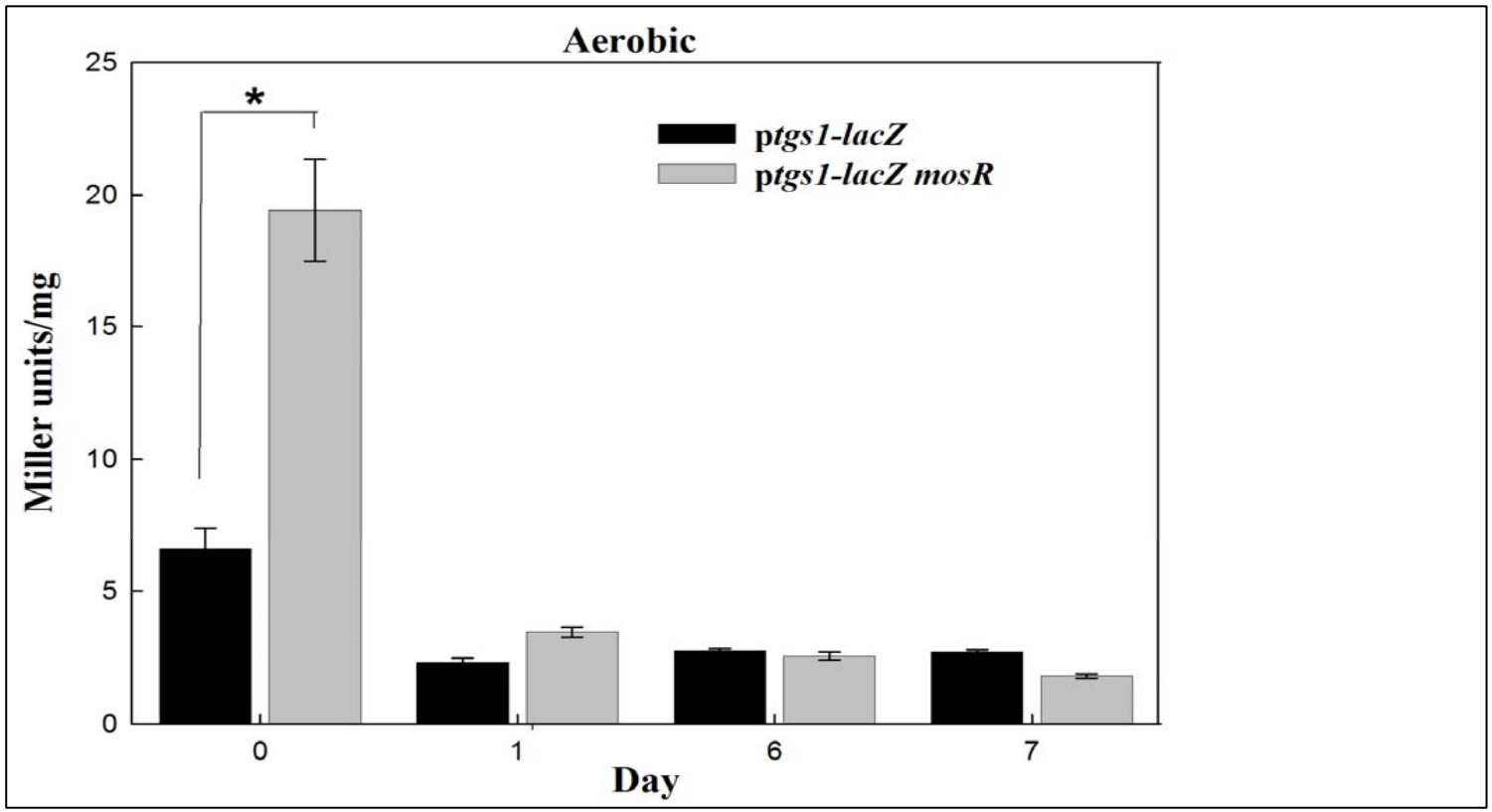


Fig. 6. Histological analysis of lung sections of mice lungs at 62 weeks post infection with ΔmosR. Note the level of accumulation of inflammatory cells (black arrows) compared to areas clear of inflammatory cells (blue arrows). Sections are stained with H&E.

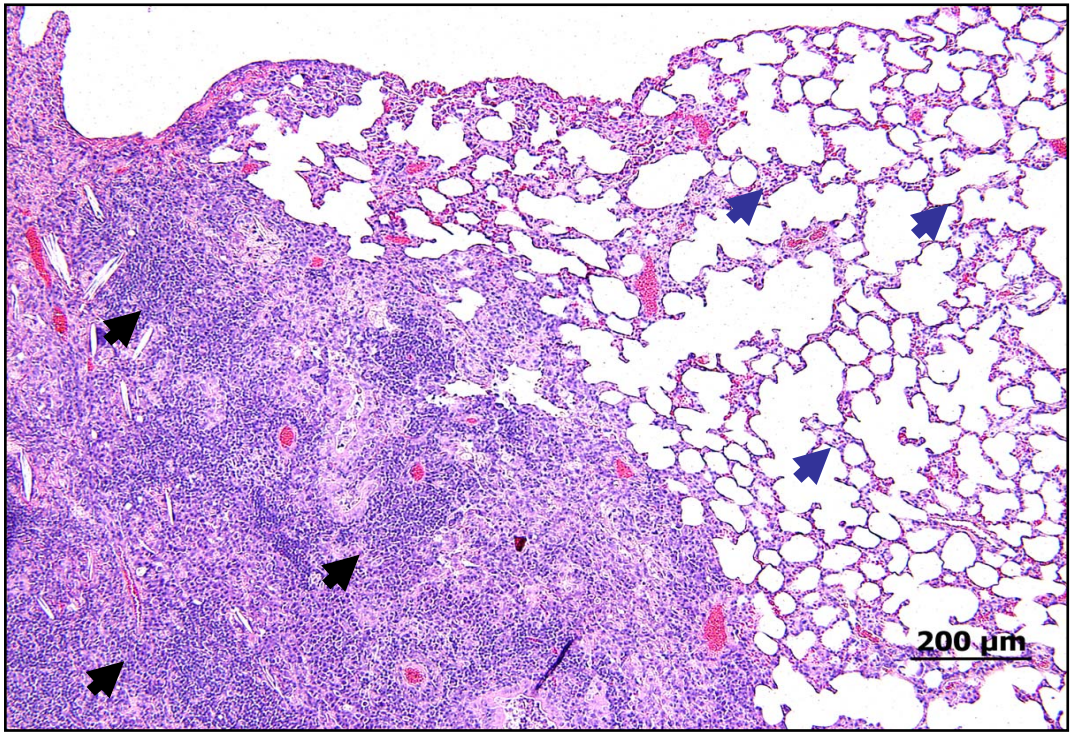
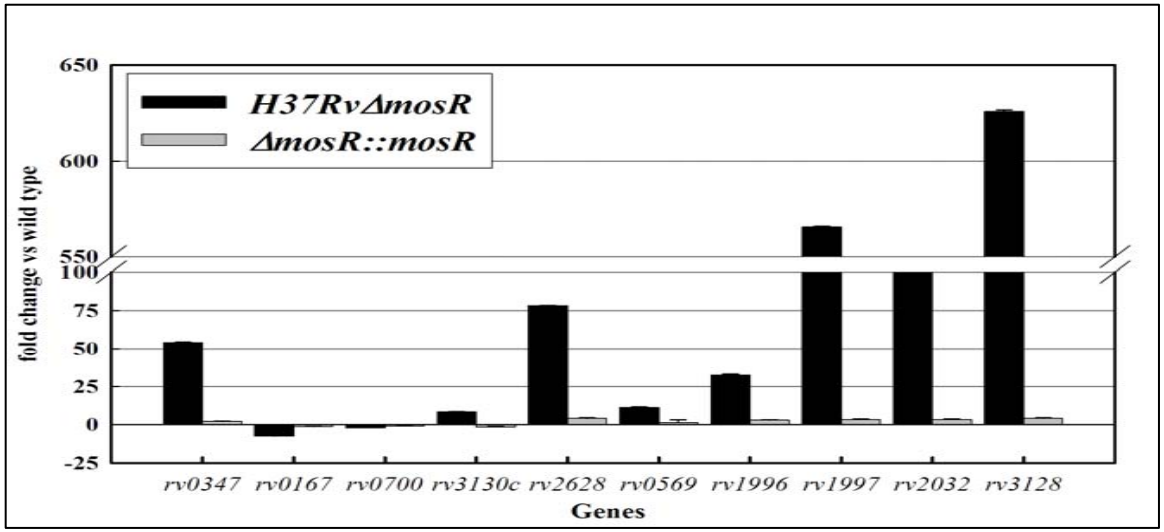


Fig.7. Quantitative, real-time PCR analysis of MosR-dependent genes.

A) Fold changes of 10 genes utilizing RNA from both mutant Δ*mosR* and complemented *Δ mosR::mosR* strains relative to H37Rv wild type strain. Both the Δ*mosR* and *Δ mosR::mosR* are represented by black and grey bars, respectively. Error bars represent ± standard deviations from the means of 3 replicates.



B) Fold changes of *fad22-fad29* gene cluster utilizing RNA from both mutant Δ*mosR* and complemented *Δ mosR::mosR* strains relative to H37Rv wild type strain. Both the Δ*mosR* and *Δ mosR::mosR* are represented by black and grey bars, respectively. Error bars represent ± standard deviations from the means of 3 replicates.

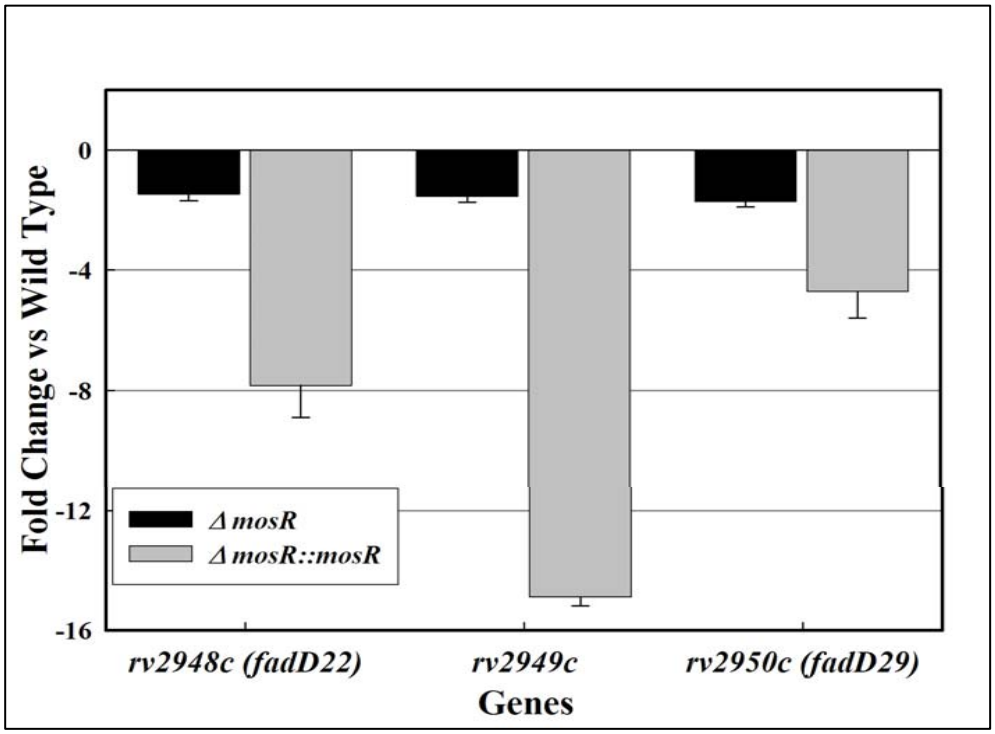


Fig 8. Thin-layer chromatography of the native and alkali-treated lipid samples of *M. tuberculosis* constructs run in petroleum ether/diethyl ether (9:1, v/v).

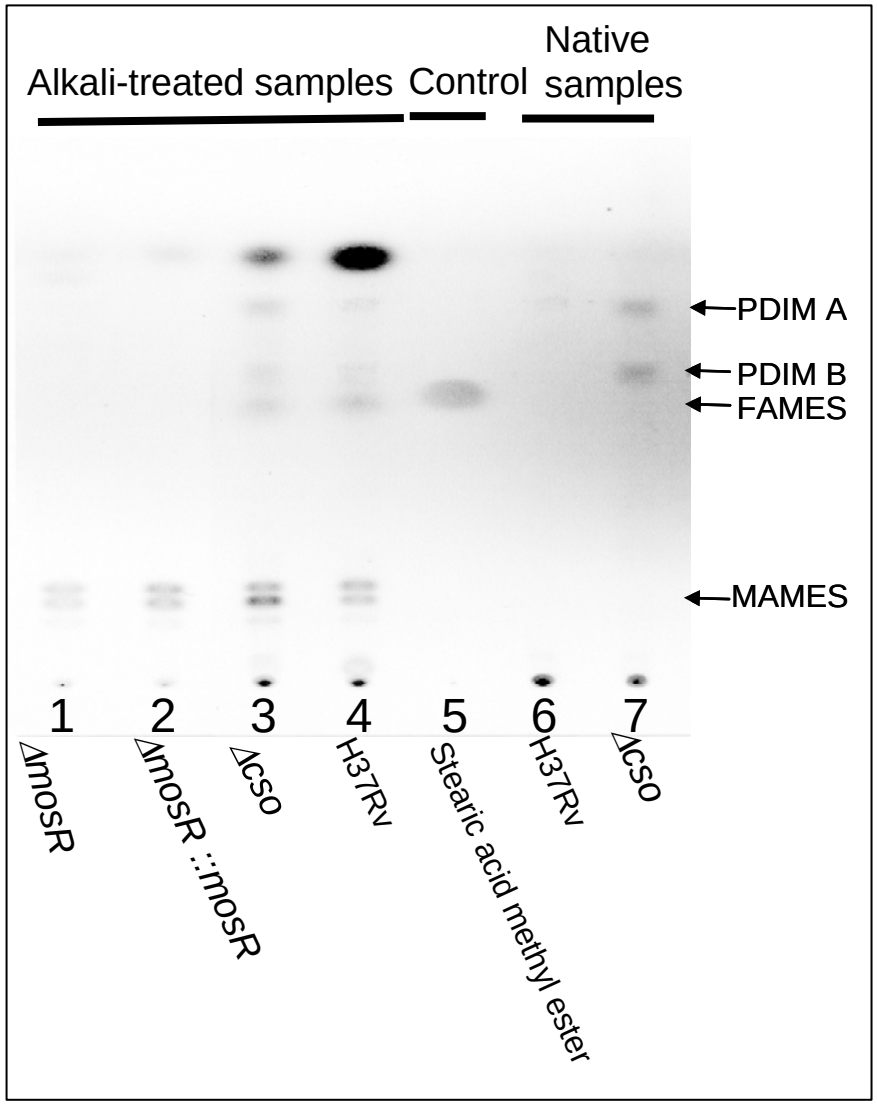


Fig. 9. MALDI-TOF mass spectra of alkali-treated lipids from *M. tuberculosis* H37Rv at different growth phases.

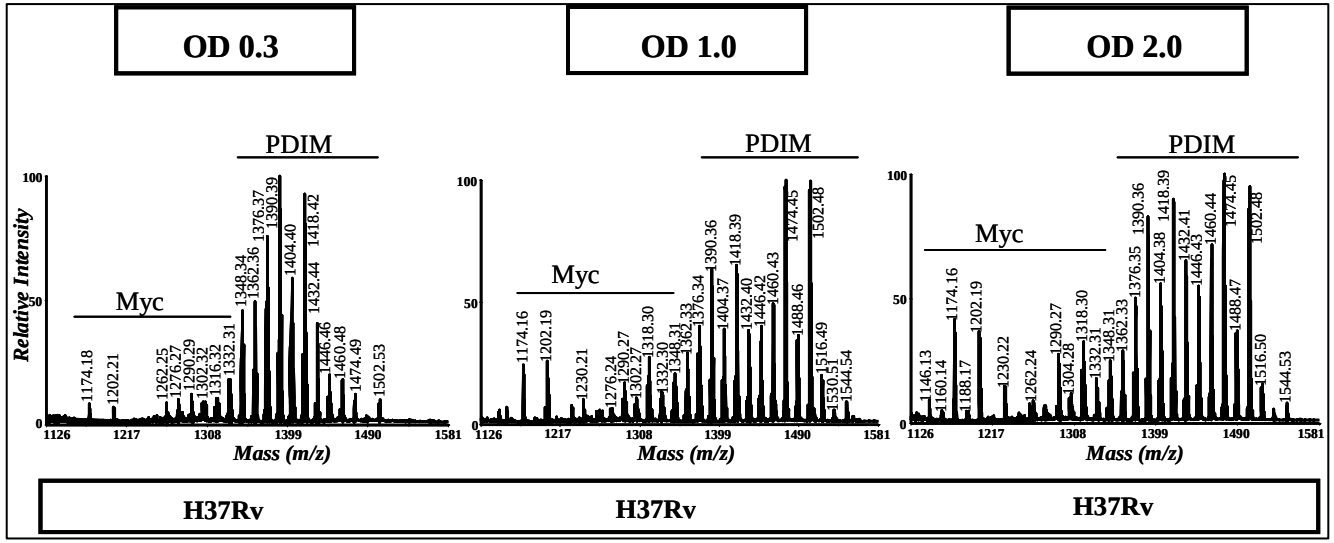


Fig. 10. A diagram depicting several scenarios where MosR can play a role in *M. tuberculosis* survival strategies.

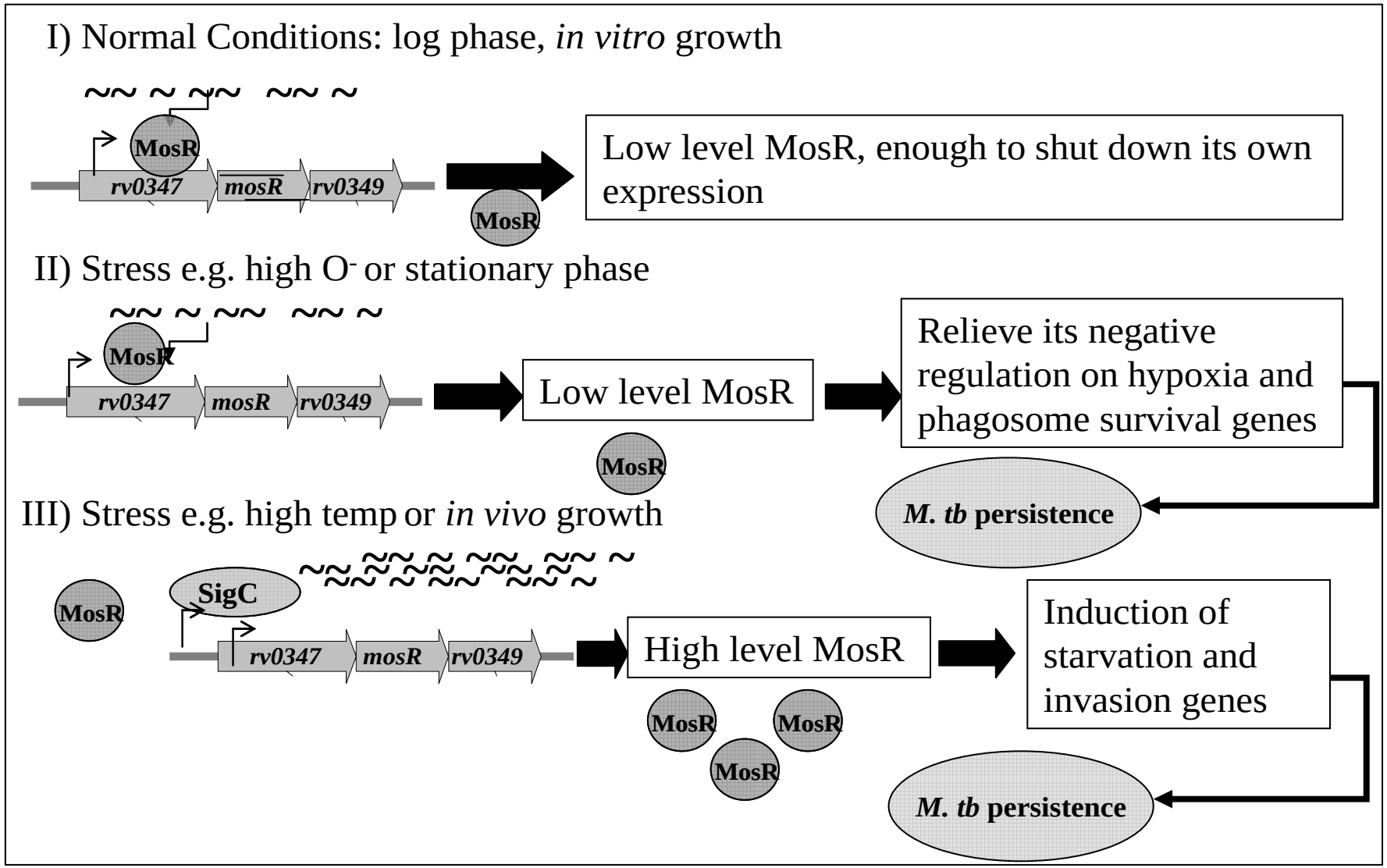


Fig. 10. A diagram depicting several scenarios where MosR can play a role in M. tuberculosis survival strategies. In the first scenario (I), the level of *mosR* transcripts (red wiggly mark) remain unchanged with the potential that MosR binds to its own promoter to repress the expression of *mosR* operon. During certain stress conditions (II, e.g. high O^- level), *mosR* transcript levels go even lower (compared to normal levels) which results in the de-repression of several gene groups responsible for hypoxia and survival in the phagosome. During some other conditions (III, e.g. *in vivo* growth), high levels of MosR are produced (using alternative regulator and possibly different promoter sequences) leading to the induction of genes responsible for starvation and invasion.