Figure S1. Schematic structure of phosphatidyl inositol mannose (PIM) in *M. smegmatis* mutant. PIM structure of wild-type *M. smegmatis* (WT; right) and pimE-deficient *M. smegmatis* (ΔpimE; left) are shown. ΔpimE lacks α1,2-mannose moiety of PIM.

Figure S2. Schematic representation of hypothetical evolutional struggle between mycobacterium and host immunity. Naive mycobacteria possess abundant trehalose dimycolate (TDM) on the cell wall when they first invade into our body (Matsunaga, I., T. Naka, R.S. Talekar, M.J. McConnell, K. Katoh, H. Nakao, A. Otsuka, S.M. Behar, I. Yano, D.B. Moody, and M. Sugita. 2008. *J. Biol. Chem.* 283:28835–28841; 1). Mincle recognizes TDM and induces the production of inflammatory cytokines and NO to elicit innate immunity against mycobacterium (2). Upon infection, mycobacterium is proposed to change the cell wall TDM into glucose monomycolate (GMM) which is no longer recognized by Mincle (3). GMM can be present on CD1b (4) to evoke T cell–mediated acquired immunity against mycobacterium (5).
Figure S3. Lack of TDM-induced pulmonary inflammation in Mincle$^{-/-}$ mice. Mincle$^{+/+}$ and Mincle$^{-/-}$ mice were left untreated (Control) or injected intravenously with an oil-in-water emulsion containing TDM. Lungs were subjected to hematoxylin-eosin staining at day 7 after injection. Bar, 1 mm.
Figure S4. Impaired TDM–induced thymic atrophy in Mincle\textsuperscript{−/−} mice. Mincle\textsuperscript{+/+} and Mincle\textsuperscript{−/−} mice were injected intravenously with an oil-in-water emulsion containing TDM (150 μg). (A) The total thymocyte number was analyzed at day 7 after injection. (B) Thymocytes were analyzed with anti-CD4–allophycocyanin and anti-CD8–phycoerythrin by flow cytometry. The percentages of CD4\textsuperscript{+}CD8\textsuperscript{+} DP thymocytes are shown.