

Supplemental Figure S3. Loss of pPplA impairs the ability of *L. monocytogenes* to form plaques in cell monolayers. (A) The ability of the various *L. monocytogenes* strains to invade, multiply, and spread from cell-to-cell was determined by assessing plaque formation within monolayers of fibroblast tissue culture cells. Cells were infected with an MOI of 10:1 and at 1 hour post-infection were washed, gentamicin was added, and plaques were visualized three days post-infection by staining with Neutral Red. Zones of clearing that did not stain indicate plaque formation. (B) Quantification of the diameter of plaques formed compared to wild-type (set to 100%). At least 20 plaques from three independent experiments were counted and measured for each strain. Data shown for panels A and B are representative of three independent experiments done in duplicate. Loss of *pplA* but not the lipoprotein (*pplA*-G72_{STOP} mutant) impaired the ability of *L. monocytogenes* to reach the host cytosol and spread efficiently from cell-to-cell.