In this study, PhoP from *Salmonella* has been purified to homogeneity and is characterized using spectroscopic approaches. We first characterized that PhoP is monomer in solution and phosphorylation does not change the monomeric state. Equilibrium and kinetic studies were employed to study promoter binding mechanism of PhoP using 13 different promoter DNAs. First, we investigated the thermodynamic and kinetic signatures of PhoP binding to its own promoter, PhoP box DNA. Our results indicate that two PhoP monomers dimerize on the PhoP box. However, the second monomer binds with weaker affinity, possibly due to negative cooperativity between the two binding sites on the DNA. Results of our kinetic experiments showed that binding of the second monomer is indeed the rate limiting step during $[PhoP]_2$-PhoP box active complex formation. Kinetic studies using phosphorylated PhoP-p showed that two monomers of PhoP can bind with similar kinetics and phosphorylation has altered the binding kinetics to overcome the rate limiting second monomer binding. Analyses revealed that PhoP-p dimerizes on the PhoP box with positive cooperativity, suggesting a mechanistic role for phosphorylation. To further understand the differential gene regulation mechanism(s) of PhoP, multiple PhoP boxes from various target promoters have been used to study PhoP binding kinetics. Our results show that two PhoP monomers bind to all PhoP boxes or promoters used in the study. However, in the case of pgtE promoter, it may be possible that only one PhoP may bind under our solution conditions. In most of these cases, the second monomer binding to the PhoP box is found to be the rate limiting step, suggesting that the role of phosphorylation can be generalized. We propose that most PhoP boxes, if not all, evolved with architectural or structural features that allow first monomer of the PhoP to bind with high affinity and second monomer with lower affinity. Phosphorylation switches this binding mode by conferring higher affinity to the second monomer binding. The exact mechanism by which phosphorylation may alter PhoP-PhoP box interaction may need structural information of PhoP-DNA complexes. However, this study provides first mechanistic insight into the PhoP mediated transcription regulation of genes.