Figure 6. FleQ binds c-di-GMP *in vitro*. A) Binding of radiolabeled c-di-GMP by FleQ truncated FleQ, WspR (positive control), QscR (negative control), and no protein control. All reactions contained 20 μM [^{32}P]–c-di-GMP. Data are presented as cpm retained on the nitrocellulose filter. B) Concentration dependent binding of [^{32}P]–c-di-GMP by FleQ (squares) and truncated FleQ (triangles). All data are the average of at least three independent binding reactions. Error bars represent the standard deviations between replicates.

Figure 7. Model for the regulation of gene expression by FleQ, FleN, and c-di-GMP. A) FleQ in the absence of FleN or c-di-GMP maximally represses *pel* transcription. B) The situation in wild-type cells; FleQ binding at the *pelA* promoter is reduced by FleN and ATP/ADP, resulting in less *pel* repression than the situation in panel A. C) C-di-GMP binds to FleQ to cause it to dissociate from DNA thereby causing derepression of transcription from the *pel* promoter.

Supplementary Figure 1. Binding of FleQ, FleN, and truncated FleQ to the *fleSR* promoter in the presence and absence of c-di-GMP. A) Binding of FleQ and FleN to the *fleSR* promoter in the presence of ATP, B) Binding of FleQ and FleN to the *fleSR* promoter in the presence and absence of c-di-GMP, C) Binding of truncated FleQ to the *fleSR* promoter. All reactions contained 10 μM ATP.