Figure S7. A thiamin synthesis hypomorph mutant leads to reciprocal suppression. Growth of WT (A) and pgpA deletion mutant (B) in two-dimensional chloramphenicol-trimethoprim concentration gradient (Materials and Methods). The antagonistic interaction in the WT becomes reciprocally suppressive in the pgpA mutant; at the same time, the pgpA mutant has greatly increased sensitivity to trimethoprim. This effect is due to truncation of the neighboring thiamin synthesis gene thiL which is essential, has overlapping open reading frame with pgpA (Keseler et al, 2005) and thus likely becomes hypomorphic as a result of pgpA deletion. (C) As B but supplemented with thiamin pyrophosphate at 80ng/mL (Materials and Methods): reciprocal suppression in pgpA mutant reverts to an antagonistic drug interaction similar to WT (A). Experiment was done on a different day and with different concentration sampling than those in A,B, leading to slight differences in MICs. (D) Trimethoprim dose-response curves (OD after 13h) of WT and pgpA mutant with and without supplementation of thiamin as in C; trimethoprim sensitivity of the pgpA mutant is completely rescued by thiamin supplementation.