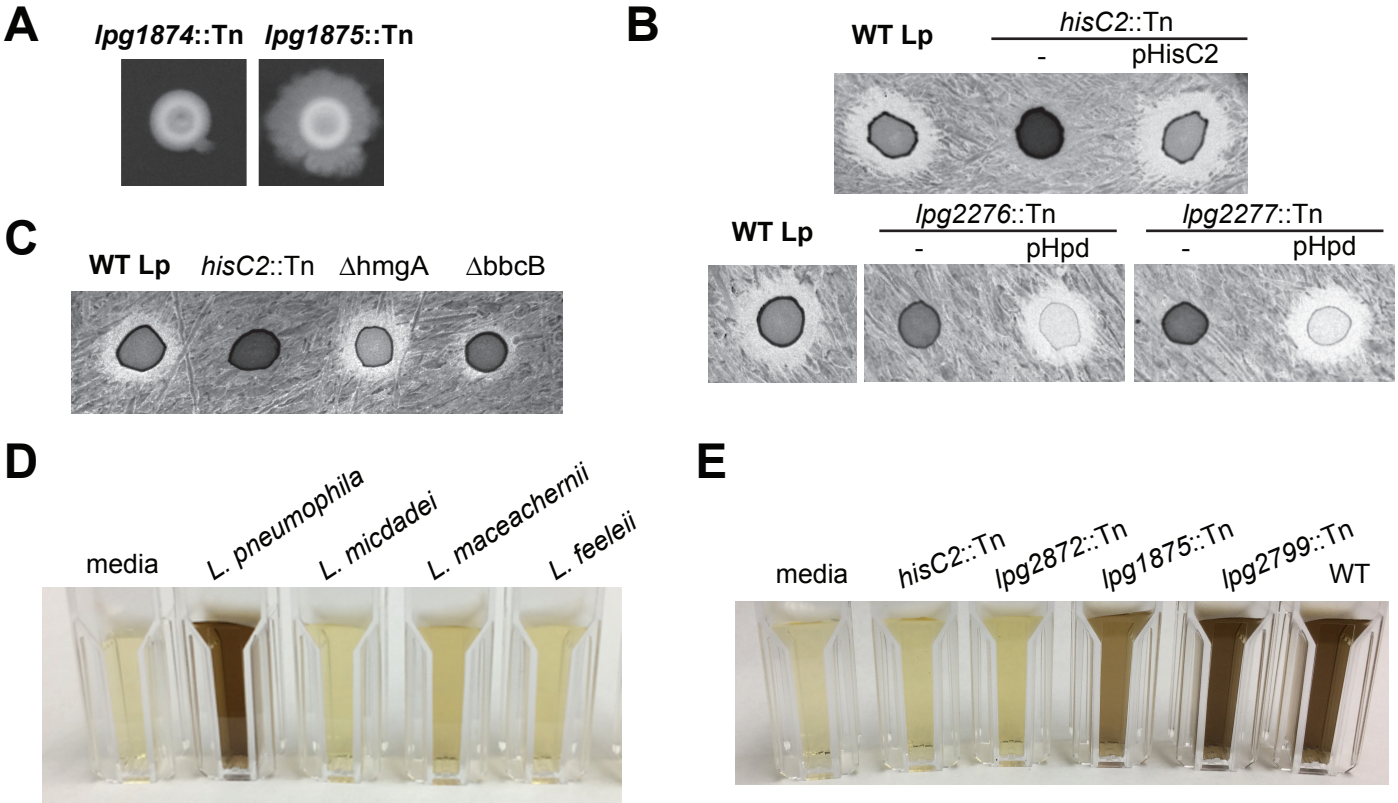

Figure 2-figure supplement 1. Genetic validation linking inhibition-defective mutants to the HGA-melanin pathway. **A)** Inter-bacterial inhibition does not correlate with surface spreading. For example, two mutants with transposon insertions in *lpg1874* (general secretion system protein L) and *lpg1875* (general secretion system protein M) share "small zone" inhibition phenotypes, yet show opposite spreading phenotypes on BCYE. **B)** Overexpression of either *hisC2* or *hpd* from a plasmid was sufficient to complement the "no zone" phenotype in recovered mutants. **C)** Unlike mutations to *hisC2*, deletion of the *hmgA* gene does not disrupt *Lm* inhibition, showing that the intracellular recycling of HGA is not required for inhibitor production. Colors in images from B and C were inverted to facilitate visualization of the zone of inhibition. **D)** Pigmentation of AYE conditioned media following 48 hours growth of various *Legionella* species. While multiple strains produce some pigment, many are less pigmented than *L. pneumophila*. The *L. micdadei* susceptible strain does not secrete detectable pigment. **E)** After 48 hours growth in AYE, none of the "no zone" mutants (represented here by *hisC2*::Tn) produce pigment. A subset of "small zone" mutants also have pigmentation defects (e.g. *lpg2872*::Tn and *lpg1875*::Tn), further implicating the HGA-melanin pathway in inhibition.