EVOLUTION

Bacteria fight it out

Interspecies competition at mucosal surfaces can select for the evolution of invasive traits, according to a recent report in *Current Biology*.

At mucosal surfaces, pathogenic and commensal members of the microbiota must compete with each other. In a mouse model of infection, *Haemophilus influenzae* can out-compete *Streptococcus pneumoniae* in the nasopharynx by stimulating neutrophil-mediated complement-opsonized killing (opsonophagocytosis). *S. pneumoniae* has more than 90 capsular serotypes, which differ in protein structure and have varying responses to complement deposition. In virulent isolates of *S. pneumoniae*, which can cause invasive disease, the capsule confers resistance to opsonophagocytosis.

Elena Lysenko and colleagues were interested in whether *S. pneumoniae* virulence, or capsule type, is affected by the competition with *H. influenzae*. Mathematical modelling of the within-host dynamics of *H. influenzae* co-infection with different strains of *S. pneumoniae* predicted that *H. influenzae* can completely outcompete an opsonophagocytosis-susceptible *S. pneumoniae* (P_s) strain and that the presence of *H. influenzae* drives selection for an invasive *S. pneumoniae* (P_R) strain encoding a capsule that confers resistance

to opsonophagocytosis. In a direct competition experiment comparing two pneumococcal isolates, a P_R isolate (serotype T4) was competitively inferior to a P_s isolate (serotype T23F), indicating that expression of the resistant capsule has a fitness cost. However, in the presence of *H. influenzae*, expression of the more resistant capsule is advantageous — the density of the P_s strain declined steeply within 24 hours of inoculation, whereas there was no decline in the density of the P_R strain. These effects could be reversed by switching the capsule type. Finally, a three-way competition experiment between *H. influenzae* and the two *S. pneumoniae* strains showed that the presence of a sufficient burden of

H. influenzae enhances colonization by the P_R strain and inhibits colonization by the P_s strain, as predicted by the theoretical model.

As pneumococcal transmission occurs during the asymptomatic phase of infection, the factors driving the evolution of costly capsule serotypes that function during the symptomatic phase of infection have been unclear. These results indicate that within-host competition between *H. influenzae* and *S. pneumoniae* could be a contributing factor.

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ORIGINAL RESEARCH PAPER Lysenko, E. S. et al. Within-host competition drives selection for the capsule virulence determinant of *Streptococcus pneumoniae*. *Curr. Biol.* 17 Jun 2010 (doi: 10.1016/j.cub.2010.05.051)