Supporting Information

Wikramaratna et al. 10.1073/pnas.1401849111

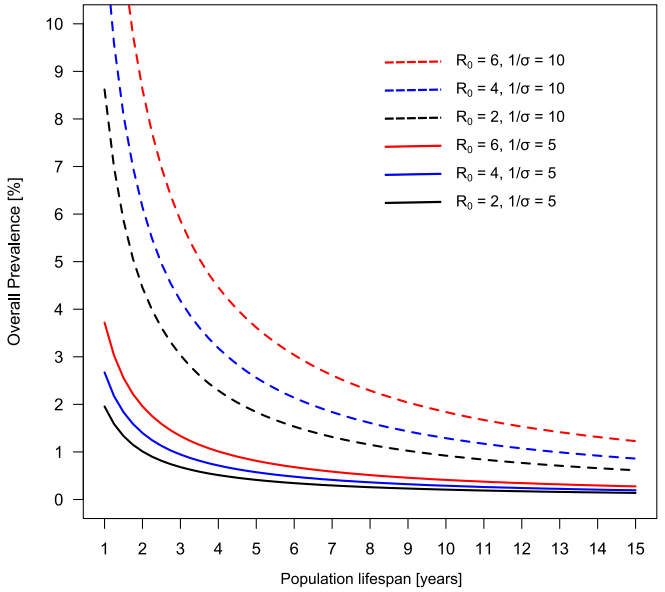

Fig. S1. Dependence of overall prevalence on R_0, the infectious period ($1/\sigma$), and population lifespan. Overall prevalence is calculated as the mean fraction of the host population that is infected with at least one strain of influenza at any one time. (Other parameters used were $\gamma = 0.70$.)

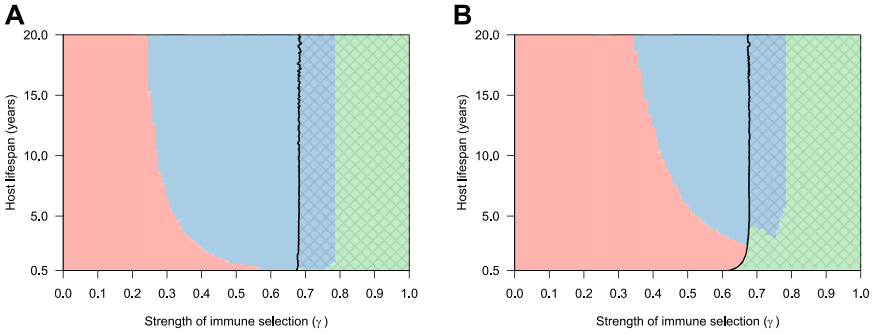

Fig. S2. (*A* and *B*) Dependence of model behavior on the strength of immune selection (γ), host lifespan ($1/\mu$) for two different host infectious periods, (*A*) 5 d and (*B*) 30 d, within a {2, 3, 5} antigenic structure of $R_0 = 2$. Three distinct behaviors can be observed: (*i*) no strain structure (red) where all antigenic types cocirculate at a stable, equal prevalence; (*ii*) cyclical or chaotic strain structure (blue), where individual antigenic types oscillate in prevalence; and (*iii*) discrete strain structure (green), where the prevalence of antigenic types is stable but unequal (more information in refs. 1–3). The cross-hatching indicates the region of parameter space where immune selection acts to permanently suppress a subset of antigenic types (defined here as never acquiring prevalence greater than 10^{-4}).

1. Gupta S, Ferguson N, Anderson R (1998) Chaos, persistence, and evolution of strain structure in antigenically diverse infectious agents. *Science* 280(5365):912–915.
2. Recker M, Pybus OG, Nee S, Gupta S (2007) The generation of influenza outbreaks by a network of host immune responses against a limited set of antigenic types. *Proc Natl Acad Sci USA* 104(18):7711–7716.
3. Wikramaratna PS, Sandeman M, Recker M, Gupta S (2013) The antigenic evolution of influenza: Drift or thrift? *Philos Trans R Soc Lond B Biol Sci* 368(1614):20120200.

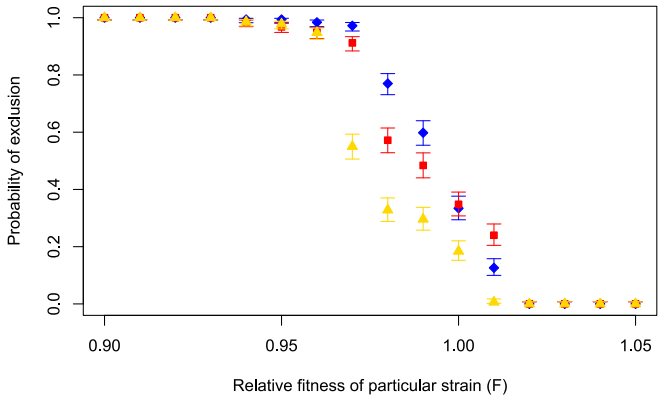

Fig. S3. Effect of fitness (*F*: defined as proportionate reduction/increase in β) of an antigenic type on the probability of being permanently suppressed by immune selection in species with lifespans of 1 y (blue diamonds), 2 y (red squares), and 15 y (yellow triangles). Five hundred simulations were performed with different random initial conditions and binomial confidence intervals were calculated by the Wilson method. (Other parameters used were $1/\sigma = 5$ d, $\gamma = 0.70$, $\beta = 146$.)

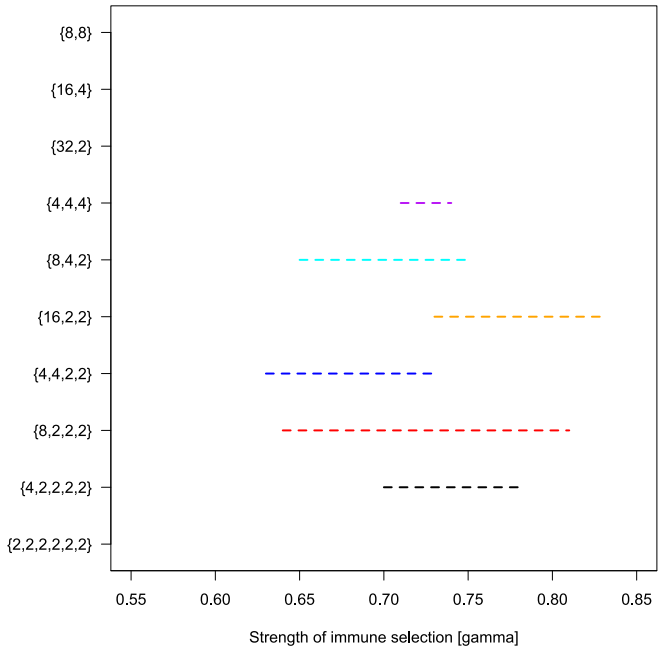

Fig. S4. Effect of multilocus structure and strength of immune selection on regions of parameter space in which individual strains can be suppressed for long periods of time even though the system otherwise exhibits oscillatory behavior. This is shown for each structure that corresponds to 64 strains; note that the behavior does not occur for {2,2,2,2,2,2}, {32,2}, {16,4}, or {8,8}. (Other parameters used were $1/\sigma = 5$ d, $1/\mu = 2$, $\beta = 146$.)

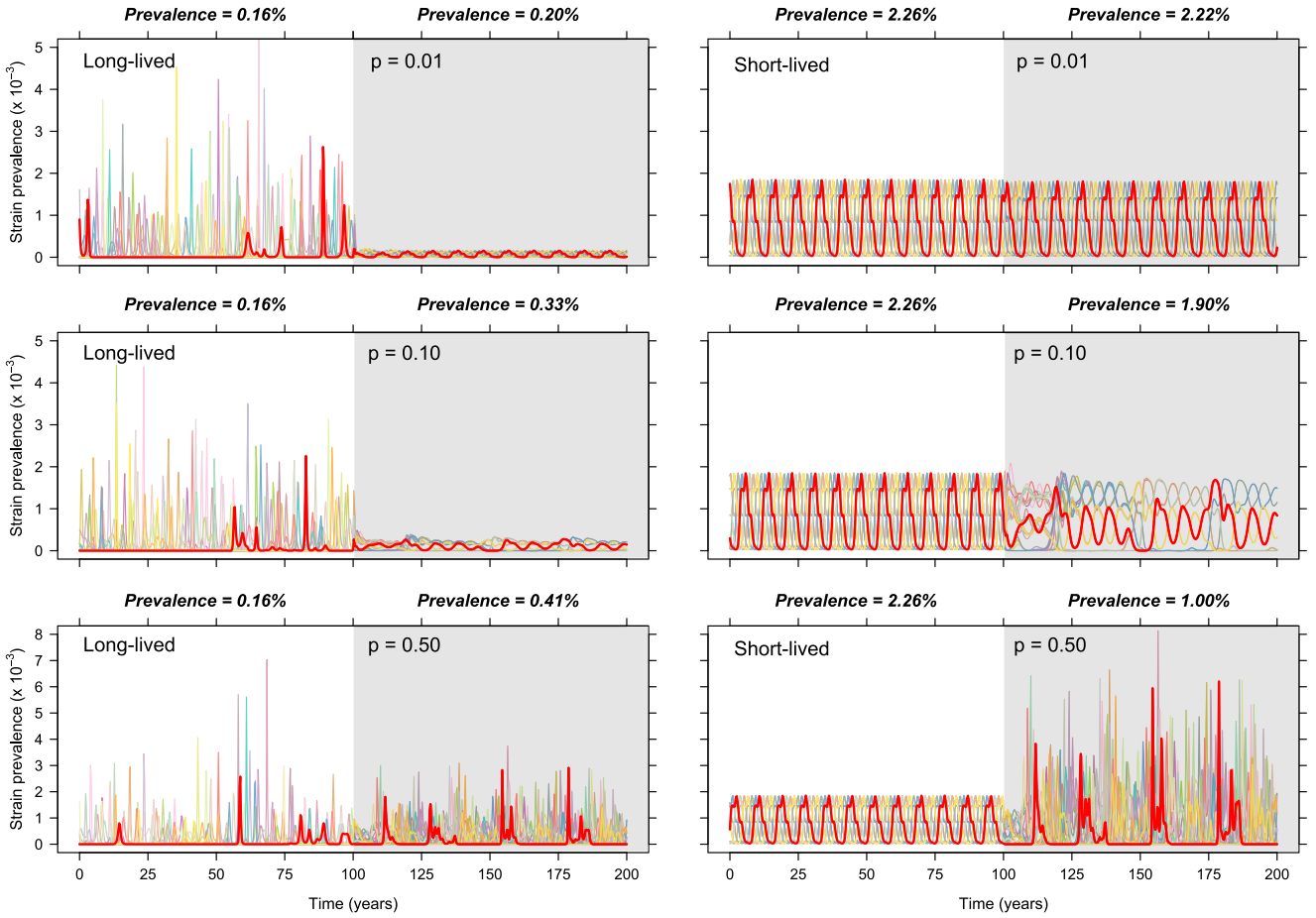

Fig. S5. Effects of increasing proportion of cross-species transmission, p, on antigenic dynamics within long-lived (life expectancy = 15 y) and short-lived (life expectancy = 1 y) species. Each color corresponds to a different strain within a {2, 3, 5} antigenic system (with one highlighted with a thick red line) before (unshaded background) and after (shaded background) the two host species are linked. The change in mean overall prevalence caused by the interaction is shown above each panel. (Other parameters: $\beta = 146$, $1/\sigma = 5$ d, $\gamma = 0.65$.)

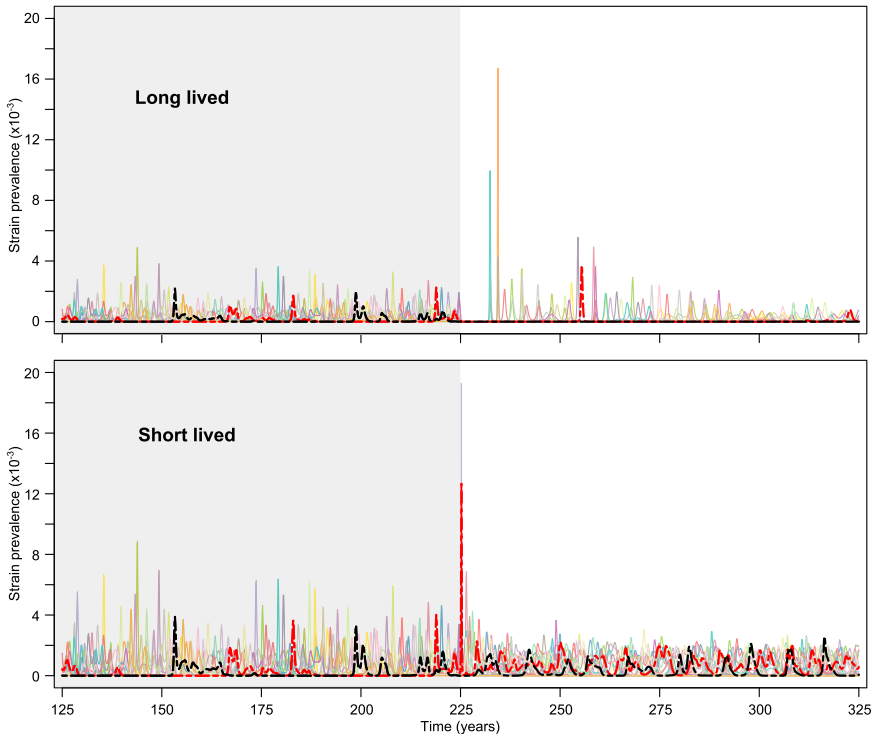

Fig. S6. Continuation of the time series from Fig. 1, illustrating that a previously suppressed strain may continue to circulate after the link between the two populations is terminated at $T = 225$. The shaded background corresponds to the period that the two populations are linked (i.e., transmit to each other). During the period of contact, overall prevalence increases in the long-lived population but decreases in the short-lived population. Consequently, the background level of immunity is too high to allow for epidemic-like behavior in the long-lived population when contact is initially withdrawn. Similarly, the level of immunity in the short-lived population is initially lower than would be expected in the absence of contact, which results in some very large outbreaks. In both cases, the dynamics eventually settle into the general qualitative patterns observed in the absence of contact, but precisely which antigenic variants end up suppressed may differ, as illustrated by the continued presence of the red variant in the short-lived population. Each color corresponds to individual strains within a {2, 3, 5} antigenic system; population life expectancies are 15 y and 2 y (other parameter values: $\beta_{1'} < \beta_1 = 146$ for all strains, except for the red and black strains where this is reversed; $1/\sigma = 5$ d, $p = 0.5$, $\gamma = 0.68$.)

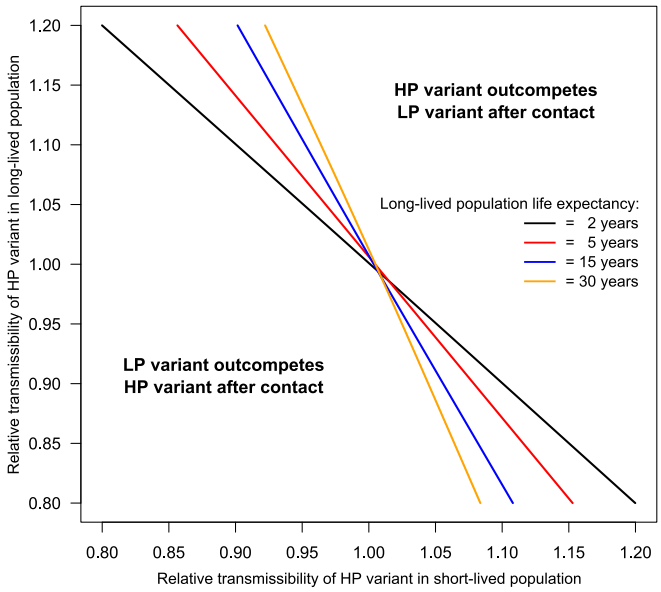

Fig. S7. The relative transmissibility of the highly pathogenic (HP) variant of an antigenic type within each population affects whether it will outcompete its low pathogenicity (LP) counterpart when the two populations are connected. Here, the LP variants of all antigenic types have the same transmissibility (β_l = 146) and the HP variants have zero transmissibility for all but one antigenic type ($\beta_{l'}$ = 0). For the remaining antigenic type, we vary the relative transmissibility of the HP variant in both populations and determine which variant of this type circulates in the long term following contact between the two populations. Of course, if one variant is more transmissible than the other in both populations, then it continues to circulate following contact. If, however, the HP variant is more transmissible in one and less so in the other, then the critical values for relative transmissibility are dependent on the life expectancies of the two populations. In particular, there is increasing emphasis on transmissibility in the short-lived population (life expectancy = 2 y) as the difference in life expectancy increases (shown by the different colored lines). Other parameters: a {2, 3, 5} antigenic system, $1/\sigma$ = 5 d, γ = 0.65, and P = 0.5.

Table S1. Bird species that tested positive for influenza A by RT-PCR in The Netherlands (1), together with adult per-capita mortality rate data from the British Trust for Ornithology website (www.bto.org/about-birds/birdfacts)

Species	Adult per capita mortality rate, /y	Samples	No. positive	%
Mallard	0.373	5,325	282	5.3
Eurasian wigeon	0.470	2,519	76	3.0
Common teal	0.470	856	50	5.8
Northern pintail	0.337	431	12	2.8
Gadwall	0.280	298	8	2.7
White-fronted goose	0.276	3,821	80	2.1
Barnacle goose	0.090	811	8	1.0
Greylag goose	0.170	455	11	2.4
Brent goose	0.100	401	4	1.0
Bean goose	0.230	315	2	0.6
Pink-footed goose	0.171	285	6	2.1
Black-headed gull	0.100	684	5	0.7
Common gull	0.140	224	2	0.9
Herring gull	0.120	735	5	0.7
Common coot	0.299	235	1	0.4

1. Munster VJ, et al. (2007) Spatial, temporal, and species variation in prevalence of influenza A viruses in wild migratory birds. *PLoS Pathog* 3(5):e61.