Appendix I

Sensitivity analysis

The existence of two locally stable equilibria was studied by perturbing each parameter at a time and recording the corresponding equilibria by numerical simulations. The parameters $\sigma$, $\nu$, $\delta$, $\beta$, $\lambda$, $\rho$, $c_{bp}$, $r_h$, and $K_h$ were multiplied by a coefficient ranging on $[0.2, 4]$, and the parameters $ID_{50}$, $r_P$, $r_{b}$, $K$, $c_{pb}$, and $\kappa$ by a coefficient ranging on $[0.2, 1.5]$ (Fig. A1). The initial values for the simulations were $S = K_h$, $I = 0$, $R = 0$, $B = 800$. To obtain upper and lower equilibria we used 0.1 and 1000 as initial values for P.

The existence of bistability is most sensitive to parameters $\kappa$, $ID_{50}$, $K$, and $c_{pb}$.

![Figure A1. Sensitivity analysis of the alternative equilibria with respect to changes in the parameter values. The parameter values are multiplied by a coefficient ranging from 0.0 to 1.5 (first six panels) or from 0.0 to 4.0 (last nine panels). The y-axis shows $I / (S + R)$ at the stable points. $I / (S + R)$ was chosen because it gives a good indication of the disease incidence.](image-url)
Linear infectivity response

For comparison with the sigmoidal infectivity function, the simulations with stochastic environmental variation were repeated using a classical linear infectivity response \( f(P) = P \). The infectivity rate parameter \( \beta \) was set to \( 4 \times 10^{-5} \), otherwise parameters were set as listed in Table 1 (main text). In this scenario, a similar pattern with the number of outbreaks is produced (Fig. A2b/c), as compared to that with sigmoidal infectivity (Fig. 3 in main text), but the mean number of infected hosts is much less affected by the amplitude of environmental variation (Fig. A2d). Longer outbreaks and persistent epidemics are only possible when the variation itself is slow thus providing longer favourable periods for environmental pathogen growth (Fig. A2a).

![Image](image_url)

**Figure A2.** Effect of the strength of stochastic variation (x-axis) and the spectral exponent \( \gamma \) (y-axis) using a linear infectivity response on (A) mean length of an infection peak (see Methods in main text for the definition of infection peaks), (B) mean number of infection peaks, (C) mean time hosts are healthy, and (D) mean infected host density. In all figures values increase from white to black. Parameter values are set as in Fig. 3 in main text.

Correlation between environmental responses

In the model (Eq. 1) used in the main text the environmental variation term \( \theta(t) \) is the same for both
pathogenic and non-pathogenic (competitor) strains, i.e. their responses to the environment are fully correlated. For comparison, the model was simulated with the environmental correlation between strain responses ranging from $\rho = 0$ to $\rho = 1$. Durations of epidemics decrease and numbers of pathogen outbreaks increase when the correlation is reduced (Fig. A3a/b/c). The mean number of infected hosts increases if variation is fast (white) and decreases if variation is slow (brown) (Fig. A3d). This is not surprising, as decreasing environmental correlation increases the likelihood that the pathogen is being favoured, while the non-pathogenic strain is being pressed by the environment.

![Figure A3](image)

Figure A3. Effect of the correlation between strain responses on environmental noise to (a) mean length of an infection peak (see Methods in main text for the definition of infection peaks), (b) mean number of infection peaks, (c) mean time hosts are healthy, and (d) mean infected host density. Three different environmental colours were used: white ($\gamma = 0$), pink ($\gamma = 1$) and brown ($\gamma = 3$). Values are averages over environmental amplitudes from 0 to 1.

**Mutation rates**

The effect of different mutation rates $\alpha_{bp}$ and $\alpha_{pb}$ from benign to pathogenic and pathogenic to benign, respectively, was studied by varying both at the same time (Fig. A4), and holding one fixed
and varying the other (Fig. A5). The effect of overall mutation rate is dependent on pathogen growth rate $r_{p}$. When the pathogen growth rate is small, mutation rate has very little effect. The overall mutation rate will have an effect only if the pathogen growth rate is high enough to increase equilibrium pathogen density above the level required for a permanent endemic infection. The effect of relative mutation rate is also rather small. There is, however, a slight trend for more infections when the rate from benign to pathogenic ($\alpha_{bp}$) is higher and less infections when the rate from pathogenic to benign ($\alpha_{pb}$) is higher.

Figure A4. The effect of overall mutation rate (both $\alpha_{bp}$ and $\alpha_{bp}$) and pathogen growth rate ($r_{p}$) on mean density of infected hosts under pink ($\gamma = 1$) stochastic variation with amplitude $A = 0.15$. Other parameter values are set as in Table 1 in main text.

Figure A5. The effect of the ratio of mutation rates $\alpha_{bp}$ and $\alpha_{bp}$ on mean density of infected hosts under pink ($\gamma = 1$) stochastic variation with amplitude $A = 0.15$. For all ratios, the larger mutation rate was fixed to $\alpha = 0.001$. Other parameter values are set as in Table 1 in main text. The magenta line shows a smoothing spline fit to data points.
Example time-series

Example time-series with three different dynamical outcomes, low incidence, sporadic outbreaks, and persistent epidemic, are shown in Fig. A6.

Figure A6. Example time-series from simulations with stochastic environmental forcing ($\gamma = -1.0$, $A = 0.05$ (top), 0.25 (middle), 0.15 (bottom)). The top row shows typical dynamics in the low disease incidence attractor. The bottom row shows shift from initial low incidence attractor to high incidence attractor, i.e. persistent epidemic. The middle row shows switching between the two attractors, i.e. sporadic outbreaks.