

¹
² Susceptible host dynamics explain pathogen resilience to
³ perturbations

⁴

⁵ **Abstract**

⁶ A major priority for epidemiological research in the time of anthropogenic change
⁷ is understanding how infectious disease dynamics respond to perturbations. Inter-
⁸ ventions to slow the spread of SARS-CoV-2 significantly disrupted the transmission
⁹ of other human pathogens. As interventions lifted, whether and when respiratory
¹⁰ pathogens would eventually return to their pre-pandemic dynamics remains to be
¹¹ answered. Here, we present a framework for estimating pathogen resilience based
¹² on how fast epidemic patterns approach their pre-pandemic, endemic dynamics and
¹³ analyze time series data from Hong Kong, Canada, Korea, and the US. By quanti-
¹⁴ fying the resilience of common respiratory pathogens, we are able to predict when
¹⁵ each pathogen will eventually return to pre-pandemic dynamics. Our predictions
¹⁶ closely match the observed deviations (or lack thereof) from its pre-COVID dynam-
¹⁷ ics. Discrepancies between predicted and observed dynamics indicate the long-term
¹⁸ impacts of pandemic perturbations, suggesting that some pathogens may be con-
¹⁹ verging to a different endemic cycle. Finally, we show that the replenishment rate
²⁰ of the susceptible pool is a key determinant of pathogen resilience, which in turn
²¹ determines the sensitivity of a system to stochastic perturbations. Overall, our anal-
²² ysis highlights the persistent nature of common respiratory pathogens compared to
²³ vaccine-preventable infections, such as measles.

24 Introduction

25 Non-pharmaceutical interventions to slow the spread of SARS-CoV-2 disrupted the
 26 transmission of other human respiratory pathogens, adding uncertainties to their future
 27 epidemic dynamics and their public health burden [1]. As interventions lifted,
 28 large heterogeneities in outbreak dynamics were observed across different pathogens
 29 in different countries, with some pathogens exhibiting earlier and faster resurgences
 30 than others [2, 3, 4]. Heterogeneities in the overall reduction in transmission and the
 31 timing of re-emergence likely reflect differences in intervention patterns, pathogen
 32 characteristics, immigration/importation from other countries, and pre-pandemic
 33 pathogen dynamics [5]. Therefore, comparing the differential impact of the pandemic
 34 perturbations across pathogens can provide unique opportunities to learn
 35 about underlying pathogen characteristics, such as their transmissibility or duration
 36 of immunity, from heterogeneities in re-emergence patterns [6].

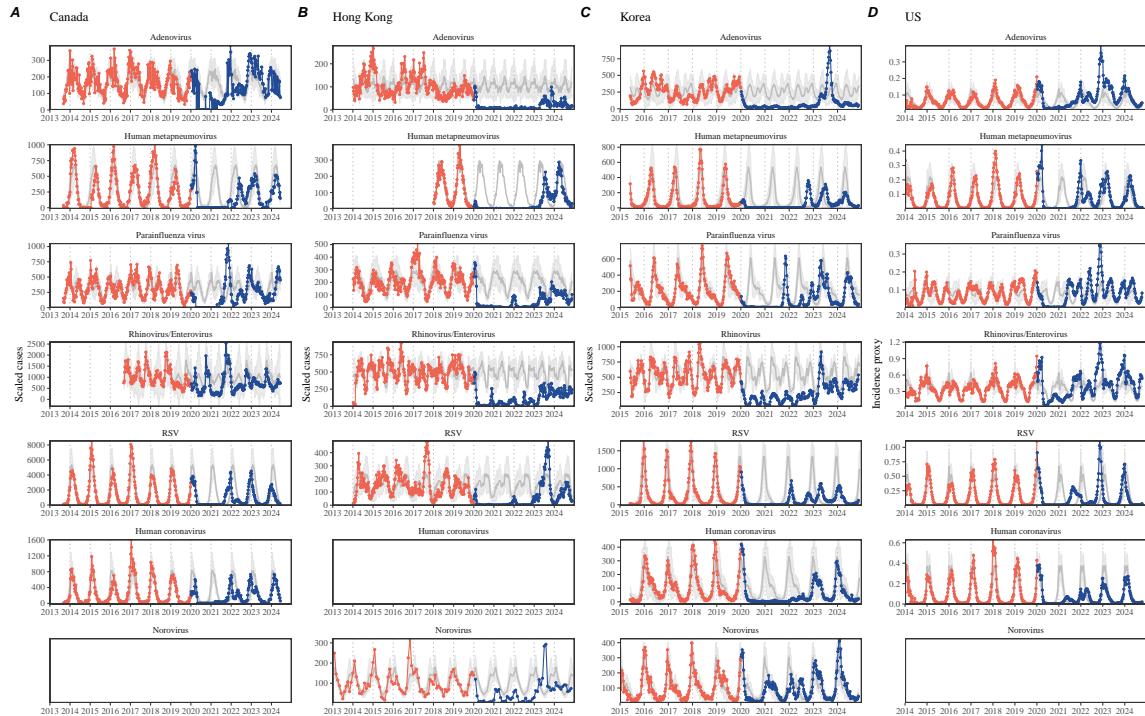


Figure 1: **Observed heterogeneity in responses to pandemic perturbations across respiratory pathogens and norovirus in (A) Canada, (B) Hong Kong, (C) Korea, and (D) US.** Red points and lines represent data before 2020. Blue points and lines represent data since 2020. Gray lines and shaded regions represent the mean seasonal patterns and corresponding 95% confidence intervals around the mean. Mean seasonal patterns were calculated by aggregating cases before 2020 into 52 weekly bins and taking the average in each week. Cases were scaled to account for changes in testing patterns (Materials and Methods).

37 Even though more than five years have passed since the emergence of SARS-CoV-
38 2, we still observe persistent changes in pathogen dynamics following the pandemic
39 perturbations. For example, compared to pre-pandemic, seasonal patterns, human
40 metapneumovirus in Korea seems to circulate at lower levels, whereas RSV in Korea
41 seems to exhibit different seasonality (Figure 1). These observations suggest a fun-
42 damental change in pathogen dynamics following the pandemic perturbations, which
43 might be driven by a long-term shift in either human behavior or population-level
44 immunity [7, 8]. For example, the emergence of SARS-CoV-2 could have caused
45 a long-term shift in population-level immunity through its interactions with other
46 pathogens [9], especially with seasonal coronaviruses [7, 10, 11]. The possibility of a
47 long-lasting impact of the pandemic perturbations poses an important question for
48 future infectious disease dynamics: can we predict whether and when other pathogens
49 will eventually return to their pre-pandemic dynamics?

50 So far, most analyses of respiratory pathogens after pandemic perturbations have
51 focused on characterizing the timing of rebound [1, 12, 5]. Instead, we seek to char-
52 acterize how fast (and whether) a pathogen returns to its pre-pandemic dynamics.
53 These two concepts have a subtle but important difference. For example, it took
54 more than 3 years for human metapneumovirus to rebound in Hong Kong, but the
55 observed epidemic patterns in 2024 appear similar to pre-pandemic seasonal means,
56 suggesting a possible return to pre-pandemic dynamics, though confirmation may
57 require multiple seasons (Figure 1). Measuring this rate of return is useful because it
58 allows us to quantify the ecological resilience of a host-pathogen system, which can
59 inform responses to future interventions [13, 14, 15, 16].

60 In this study, we lay out theoretical and statistical approaches to characterizing
61 the resilience of a host-pathogen system based on how fast the system recovers from
62 perturbation. We begin by laying out a few representative scenarios that capture
63 the potential impact of pandemic perturbations on endemic pathogen dynamics and
64 illustrate how resilience can be measured by comparing the pre- and post-pandemic
65 dynamics of susceptible and infected hosts. In practice, information on suscepti-
66 ble hosts is often unavailable, making this theoretical approach infeasible. Instead,
67 we utilize a mathematical technique to reconstruct empirical attractors from the
68 data [17], which allows us to measure the rate at which the host-pathogen system
69 approaches this empirical attractor after a perturbation; we define this rate to be
70 the empirical resilience of the host-pathogen system. We use this method to ana-
71 lyze pathogen surveillance data for respiratory and non-respiratory pathogens from
72 Canada, Hong Kong, Korea, and the US. Finally, we show that susceptible host dy-
73 namics explain variation in pathogen resilience and further demonstrate that more
74 resilient pathogens will be less sensitive to perturbations caused by demographic
75 stochasticity, thereby providing a direct link between pathogen resilience and persis-
76 tence.

⁷⁷ Conceptual introduction to pathogen resilience

⁷⁸ In classical ecological literature, the resilience of an ecological system is measured by
⁷⁹ the rate at which the system returns to its reference state following a perturbation
⁸⁰ [13, 14, 15, 16]. This rate corresponds to the largest real part of the eigenvalues
⁸¹ of the linearized system near equilibrium—here, we refer to this value as the *in-*
⁸² *trinsic* resilience of the system, which represents the expected rate of return from
⁸³ perturbed states. In practice, we rarely know the true model describing population-
⁸⁴ level dynamics of common respiratory pathogens, limiting our ability to infer the
⁸⁵ intrinsic resilience of a system. Instead, we can measure the *empirical* resilience
⁸⁶ of a host-pathogen system by looking at how fast the system returns to the pre-
⁸⁷ perturbation, endemic dynamics after the perturbation has ended. The COVID-19
⁸⁸ pandemic provides a particularly useful example of a major perturbation, providing
⁸⁹ unique opportunities to measure the resilience of a host-pathogen system.

⁹⁰ As an example, we begin with a simple Susceptible-Infected-Recovered-Susceptible
⁹¹ (SIRS) model with seasonally forced transmission and demography (i.e., birth and
⁹² death). The SIRS model is the simplest model that allows for the waning of im-
⁹³ munity and is commonly used for modeling the dynamics of respiratory pathogens
⁹⁴ [18]. First, consider a pandemic perturbation that reduces transmission by 50% for 6
⁹⁵ months starting in 2020, which causes epidemic patterns to deviate from their origi-
⁹⁶ nal stable annual cycle for a short period of time and eventually come back (Figure
⁹⁷ 2A). To measure the resilience of this system empirically, we first need to be able to
⁹⁸ measure the distance from its pre-pandemic attractor, which is defined as a set of
⁹⁹ points in state space or phase plane that the system is pulled towards [19]. There
¹⁰⁰ are many ways we can measure the distance from the attractor, but for illustrative
¹⁰¹ purposes, we choose one of the most parsimonious approaches: that is, we look at
¹⁰² how the susceptible (S) and infected (I) populations change over time and measure
¹⁰³ the Euclidean distance on the SI phase plane, using the counterfactual unperturbed
¹⁰⁴ phase plane as a reference (Figure 2B; Materials and Methods). In this simple case,
¹⁰⁵ the locally estimated scatterplot smoothing (LOESS) fit indicates that the distance
¹⁰⁶ from the attractor decreases exponentially (linearly on a log scale) on average (Figure
¹⁰⁷ 2C). Furthermore, the overall rate of return approximates the intrinsic resilience of
¹⁰⁸ the seasonally unforced system (Figure 2C).

¹⁰⁹ Alternatively, pandemic perturbations can have a lasting impact on the forces
¹¹⁰ driving pathogen dynamics through a long-term reduction in transmission or per-
¹¹¹ manent change in immunity. As an example, we consider a scenario in which a 10%
¹¹² reduction in transmission persists even after the major pandemic perturbations are
¹¹³ lifted (Figure 2D–F). In such cases, we cannot know whether the pathogen will re-
¹¹⁴ turn to its original cycle or a different cycle until many years have passed, and we
¹¹⁵ cannot measure the distance to the new unknown attractor that the system might
¹¹⁶ eventually approach. Nonetheless, we can still measure the distance from the pre-
¹¹⁷ pandemic attractor and ask how the distance changes over time (Figure 2E). The
¹¹⁸ LOESS fit suggests that the distance from the pre-pandemic attractor will initially

119 decrease exponentially on average (equivalently, linearly on a log scale) and even-
120 tually plateau (Figure 2F). Here, a permanent 10% reduction in transmission rate
121 slows the system, which causes the distance from the pre-pandemic attractor initially
122 to decrease at a slower rate (Figure 2F) than it would have otherwise (Figure 2C)
123 before plateauing at a fixed distance between the two attractors. This example shows
124 that resilience is not necessarily an intrinsic property of a specific pathogen. Instead,
125 pathogen resilience is a property of a specific attractor that a host-pathogen system
126 approaches, which depends on both pathogen and host characteristics.

127 Finally, transient phenomena can further complicate the picture (Figure 2G–I).
128 For example, a stage-structured model, which accounts for reduction in secondary
129 susceptibility, initially exhibits a stable annual cycle, but perturbations from a 10%
130 reduction in transmission for 6 months cause the epidemic to shift to biennial cycles
131 (Figure 2G). The system eventually approaches the original pre-pandemic attractor
132 (Figure 2H), suggesting that this biennial cycle is a transient phenomenon. The
133 LOESS fit indicates that the distance from the attractor initially decreases expo-
134 nentially at a rate that is consistent with the intrinsic resilience of the seasonally
135 unforced stage-structured system, but the approach to the attractor slows down
136 with the damped oscillations (Figure 2I). This behavior is also referred to as a ghost
137 attractor, which causes long transient dynamics and slow transitions [20]. Strong
138 seasonal forcing in transmission can also lead to transient phenomena for a simple
139 SIRS model, causing a slow return to pre-perturbation dynamics (Supplementary
140 Figure S1).

141 This empirical approach allows us to measure the resilience of a two-strain host-
142 pathogen system as well even when we have incomplete observation of the infection
143 dynamics. Simulations from a simple two-strain competition system illustrate that
144 separate analyses of individual strain dynamics (e.g., RSV A vs B) and a joint anal-
145 ysis of total infections (e.g., total RSV infections) yield identical resilience estimates
146 (Supplementary Figure S2, 3). This is expected because eigenvalues determine the
147 dynamics of the entire system around the equilibrium, meaning that both strains
148 should exhibit identical rates of return following a perturbation. Analogous to a
149 single-strain system, strong seasonal forcing in transmission can cause the two-strain
150 system to slow down through transient phenomena (Supplementary Figure S4).

151 These observations yield three insights. First, we can directly estimate the empircal
152 resilience of a host-pathogen system by measuring the rate at which the system
153 approaches an attractor, provided that we have a way to quantify the distance from
154 the attractor—as we discuss later, the attractor of a system can be reconstructed
155 from data from mathematical theory without making assumptions about the under-
156 lying model. The empirical approach to estimating pathogen resilience is particularly
157 convenient because it does not require us to know the true underlying model; esti-
158 mating the intrinsic resilience from fitting misspecified models can lead to biased
159 estimates (Supplementary Figure S5). Second, resilience estimates allow us to make
160 phenomenological predictions about the dynamics of a host-pathogen system follow-
161 ing a perturbation. Assuming that an attractor has not changed and the distance

from the attractor will decrease exponentially over time, we can estimate when the system should reach an attractor. Finally, a change in the (exponential) rate of approach provide information about whether the system has reached an alternative attractor, or a ghost attractor, that is different from the original, pre-pandemic attractor. These alternative attractors may reflect permanent changes in transmission patterns as well as changes in immune landscapes. There will be periods of time when it is difficult to tell whether pathogen dynamics are still diverging from the original attractor due to a long-term perturbation, or have entered the basin of attraction of a new attractor. Now that several years have passed since major interventions have been lifted, many respiratory pathogens may have had sufficient time to begin returning to their post-intervention attractors—empirical comparisons between current cases and pre-pandemic cases are consistent with this hypothesis (Figure 1). With recent data, we can start to evaluate whether we see early signs of convergence to the former attractor or a new one.

Inferring pathogen resilience from real data

Based on these observations, we now lay out our approach to estimating pathogen resilience from real data (Figure 3). We first tested this approach against simulations and applied it to real data. Specifically, we analyzed case time series of respiratory pathogens from four countries: Canada, Hong Kong, Korea, and the US.

So far, we have focused on simple examples that assume a constant transmission reduction during the pandemic. However, in practice, the impact of pandemic perturbations on pathogen transmission was likely more complex (Figure 3A), reflecting introduction and relaxation of various intervention strategies. In some cases, strong perturbations likely caused local fadeouts, requiring immigration/importation from another location for epidemic rebound. Such complexities could lead to longer delays between the introduction of pandemic perturbations and pathogen rebound as well as temporal variation in outbreak sizes (Figure 3B); in this example, continued transmission reduction from interventions limits the size of the first outbreak in 2021 following the rebound, allowing for a larger outbreak in 2022 when interventions are further relaxed.

Previously, we relied on the dynamics of susceptible and infected hosts to compute the distance from the attractor (Figure 2), but information on susceptible hosts is rarely available in practice. In addition, uncertainties in case counts due to observation error, strain evolution, and multiannual cycles in the observed epidemic dynamics (e.g., adenovirus circulation patterns in Hong Kong and Korea) add challenges to defining pre-pandemic attractors, which limits our ability to measure the distance from the attractor. To address these challenges, we can reconstruct an empirical attractor by utilizing Takens' theorem [17], which states that an attractor of a nonlinear multidimensional system can be mapped onto a delayed embedding (Materials and Methods). For example, we can use delayed logged values of pre-pandemic

202 cases $C(t)$ (Figure 3C) to reconstruct the attractor:

$$\langle \log(C(t) + 1), \log(C(t - \tau) + 1), \dots, \log(C(t - (M - 1)\tau) + 1) \rangle, \quad (1)$$

203 where the delay τ and embedding dimension M are determined based on autocor-
204 relations and false nearest neighbors, respectively [21, 22]. This allows us to define
205 the pre-pandemic attractor as a points on an M dimensional space. We can then
206 apply the same delay and embedding dimensions to the entire time series to deter-
207 mine the position in multi-dimensional state space (Figure 3D), which allows us to
208 measure the nearest neighbor distance between the current state of the system and
209 the empirical pre-pandemic attractor (Figure 3E). Specifically, the nearest neighbor
210 distance is calculated by computing the distance between the current position on
211 the M dimensional space and all points in the empirical attractor set and taking the
212 minimum value. In theory, we can now quantify how fast this distance decreases by
213 fitting a linear regression on a log scale, where the slope of the linear regression em-
214 pirically measures pathogen resilience with a steeper slope corresponding to a higher
215 resilience estimate (Figure 3E). However, resulting estimates of pathogen resilience
216 can be sensitive to choices about embedding delays and dimensions. For example,
217 using longer delays and higher dimensions tends to smooth out temporal variations
218 in the distance from the attractor (Supplementary Figure S6).

219 Complex changes in the distance from the attractor suggest that estimating
220 pathogen resilience from linear regression will be particularly sensitive to our choice
221 of fitting windows for the regression (Figure 3E). Therefore, before we tried estimat-
222 ing resilience from real data, we explored an automated window selection criterion
223 for linear regression and tested it against randomized, stochastic simulations across
224 a range of realistic pandemic perturbation shapes. In doing so, we also explored
225 optimal choices for embedding dimensions and evaluated our choices of fitting win-
226 dow parameters and embedding dimensions by quantifying correlation coefficients
227 between the estimated resilience and the intrinsic resilience of a seasonally unforced
228 system (Materials and Methods). Overall, we found large variation in estimation
229 performances with correlation coefficient ranging from 0.21 to 0.61 (Supplementary
230 Figure S7). In almost all cases, the automated window selection approach outper-
231 formed a naive approach, which performs regression between the peak distance and
232 current distance (Supplementary Figure S7).

233 Based on the best performing window selection criteria and embedding dimen-
234 sion, we applied this approach to pathogen surveillance data presented in Figure
235 1 (Materials and Methods). For each time series, we applied Takens' theorem in-
236 dependently to reconstruct the empirical attractor and obtained the corresponding
237 time series of distances from attractors (Supplementary Figure S8). Then, we used
238 the automated window selection criterion to fit a linear regression and estimated the
239 empirical resilience for each pathogen in each country (Supplementary Figure S8);
240 the window selection criterion gave poor regression window for three cases (norovirus
241 in Hong Kong and Korea and rhinovirus/enterovirus in the US), leading to unreal-
242 istically low resilience estimates, and so we used ad-hoc regression windows instead

243 (Supplementary Figure S9; Materials and Methods).

244 For all pathogens we considered, resilience estimates fell between 0.4/year and
245 1.8/year (Figure 4A). We estimated the mean resilience of common respiratory
246 pathogens to be 0.99/year (95% CI: 0.81/year–1.18/year). For reference, this is
247 \approx 7.5 times higher than the intrinsic resilience of pre-vaccination measles in England
248 and Wales (\approx 0.13/year). Finally, resilience estimates for norovirus were comparable
249 to those of common respiratory pathogens: 1.44/year (95%CI: 1.01/year–1.87/year)
250 for Hong Kong and 1.07/year (95%CI: 0.86/year–1.29/year) for Korea. Based on
251 a simple ANOVA test, we did not find significant differences in resilience estimates
252 across countries ($p = 0.25$) or pathogens ($p = 0.67$).

253 Using resilience estimates, we predicted when each pathogen would hypothetically
254 return to their pre-pandemic dynamics, assuming no long-term change in the attrac-
255 tor. Specifically, we extended our linear regression fits to distance-from-attractor
256 time series and ask when the predicted regression line will cross a threshold value;
257 since we relied on nearest neighbor distances, pre-pandemic distances are always
258 greater than zero (Figure 3E), meaning that we can use the mean of pre-pandemic
259 distances as our threshold.

260 We predicted that a return to pre-pandemic cycles has occurred or would be
261 imminent for most pathogens (Figure 4B). In particular, we predicted that 12 out
262 of 23 pathogen-country pairs should have already returned before the end of 2024.
263 For almost all pathogens that were predicted to have returned already, the observed
264 epidemic dynamics showed clear convergence towards their pre-pandemic seasonal
265 averages, confirming our predictions (Figure 4C). However, there were a few ex-
266 ceptions, including norovirus in Hong Kong and rhinovirus/enterovirus in the US,
267 where the observed epidemic dynamics in 2024 exhibit clear deviation from their pre-
268 pandemic seasonal averages (Figure 4C; Figure S9). These observations suggest a
269 possibility that some common respiratory pathogens may have converged to different
270 attractors or are still exhibiting non-equilibrium dynamics. In contrast, pathogens
271 that were predicted to have not returned yet also showed clear differences from their
272 pre-pandemic seasonal averages; as many of these pathogens are predicted to return
273 in 2025–2026, we may be able to test these predictions in near future (Supplementary
274 Figure S10). Our reconstructions of distance time series and estimates of pathogen
275 resilience and expected return time were generally robust to choices of embedding
276 dimensions (Supplementary Figure S11–12).

277 Susceptible host dynamics explain variation in pathogen 278 resilience

279 So far, we have focused on quantifying pathogen resilience from the observed pat-
280 terns of pathogen re-emergence following pandemic perturbations. But what factors
281 determine how resilient a host-pathogen system is? To address this question, we used
282 the SIRS model to explore how changes in susceptible host dynamics affect pathogen

resilience. To do so, we varied the basic reproduction number \mathcal{R}_0 , which represents the average number of secondary infections caused by a newly infected individual in a fully susceptible population, and the duration of immunity and computed intrinsic resilience for each parameter.

We found that an increase in \mathcal{R}_0 and a decrease in the duration of immunity correspond to an increase in pathogen resilience (Figure 5A). Similarly, an increase in \mathcal{R}_0 and a decrease in the duration of immunity corresponds to a faster per-capita rate of susceptible replenishment, which is defined as the ratio between absolute rate at which new susceptibles enter the population and the equilibrium number of susceptible individuals in the population, S^* (Figure 5B). We note that a higher \mathcal{R}_0 drives a faster per-capita susceptible replenishment rate by decreasing the susceptible proportion at equilibrium, S^* . For a simple SIR model that assumes a life-long immunity, we can show analytically that pathogen resilience is proportional to the per-capita rate of susceptible replenishment (Materials and Methods). Overall, these observations suggest that a faster per-capita susceptible replenishment rate causes the system to be more resilient.

By taking the average values of empirical resilience values for each pathogen, we were able to map each pathogen onto a set of parameters of the SIRS model that are consistent with corresponding resilience estimates (Figure 5A). Across all pathogens we considered, we estimated that the average duration of immunity is likely to be short (< 4 years) across a plausible range of \mathcal{R}_0 (< 6). We were also able to obtain a plausible range of susceptible replenishment rates for each pathogen (Figure 5B), but there was a large uncertainty in the estimates for susceptible replenishment rates due to a lack of one-to-one correspondence between susceptible replenishment rates and pathogen resilience.

Pathogen resilience determines sensitivity to stochastic perturbations

Even in the absence of major pandemic perturbations, host-pathogen systems are expected to experience continued perturbations of varying degrees from changes in epidemiological conditions, such as human behavior, climate, and viral evolution. These perturbations can also arise from demographic stochasticity, which is inherent to any ecological systems. Here, we used a seasonally unforced SIRS model with birth/death to explore how resilience of a host-pathogen system determines the sensitivity to perturbations caused by demographic stochasticity (Materials and Methods).

We found that resilience of a host-pathogen system determines the amount of deviation from the deterministic trajectory caused by demographic stochasticity, with less resilient systems experiencing larger deviations (Figure 6). Notably, less resilient systems also exhibited slower epidemic cycles (Figure 6A–C). The periodicity of this epidemic cycle matched those predicted by the intrinsic periodicity of the system

323 (Supplementary Figure S13) where the intrinsic resilience of the system is inversely
324 proportional to its intrinsic periodicity (Supplementary Figure S14). However, we
325 note that the interplay between seasonal transmission and intrinsic periodicity can
326 also lead to complex cycles, as illustrated by a recent analysis of *Mycoplasma pneumoniae*
327 dynamics [23].

328 We also note that the intrinsic resilience is not the sole determinant for how sen-
329 sitive the system is to stochastic perturbations. For example, the population size
330 and average duration of infection also affect the amount of deviation from the deter-
331 ministic trajectory caused by demographic stochasticity, even though these quantities
332 have little to no impact on the intrinsic resilience (Supplementary Figure S15). These
333 conclusions were robust for the seasonally forced SIRS model (Supplementary Figure
334 S16).

335 Discussion

336 COVID-19 pandemic interventions caused major disruptions to circulation patterns
337 of both respiratory and non-respiratory pathogens, adding challenges to predicting
338 their future dynamics [1, 2, 3, 4]. However, these perturbations offer large-scale natu-
339 ral experiments for understanding how different pathogens respond to perturbations.
340 In this study, we showed that pathogen re-emergence patterns following pandemic
341 perturbations can be characterized through the lens of ecological resilience. We
342 showed that variation in pathogen resilience can be explained by the differences in
343 susceptible host dynamics, where faster replenishment of the susceptible pool corre-
344 sponds to a more resilient host-pathogen system. Finally, we showed that pathogen
345 resilience also determines the sensitivity to stochastic perturbations.

346 We analyzed case time series of common respiratory infections and norovirus
347 infections from Canada, Hong Kong, Korea, and the US to estimate their resilience.
348 Overall, we estimated the resilience of these pathogens to range from 0.4/year to
349 1.8/year, which is 3–14 times more resilient than prevaccination measles. These
350 resilience estimates indicate that common respiratory pathogens and norovirus likely
351 exhibit faster susceptible replenishment and are therefore more persistent, indicating
352 potential challenges in controlling these pathogens.

353 Based on our resilience estimates, we made phenomenological predictions about
354 when each pathogen will return to their endemic cycles. For the most part, we
355 accurately predicted which pathogens should have already returned before the end
356 of 2024. However, there were two main exceptions (i.e., norovirus in Hong Kong
357 and rhinovirus/enterovirus in the US), suggesting that these pathogens may be con-
358 verging to new endemic cycles or experiencing long-term transient behavior. These
359 changes may reflect changes in surveillance or actual shift in the dynamics, caused
360 by permanent changes in behavior or population-level immunity. While it may seem
361 unlikely that permanent changes in behavior would only affect a few pathogens and
362 not others, we cannot rule out this possibility given differences in the observed mean

363 age of infections and therefore the differences in age groups that primarily drive
364 transmission [24, 25]. Differences in the mode of transmission between respiratory
365 vs gastrointestinal pathogens may also contribute to the differences in responses to
366 pandemic perturbations.

367 For almost half of the pathogens we considered, we predicted that their return
368 to original epidemic patterns is imminent. We will need a few more years of data
369 to test whether these pathogens will eventually return to their original dynamics or
370 eventually converge to a different attractor. We also cannot rule out the possibility
371 that some pathogens may exhibit long-term transient behaviors following pandemic
372 perturbations. Overall, these observations echo earlier studies that highlighted the
373 long-lasting impact of pandemic perturbations [8, 26, 27, 4, 23].

374 We showed that susceptible host dynamics shape pathogen resilience, where faster
375 replenishment of the susceptible population causes the pathogen to be more resilient.
376 For simplicity, we focused on waning immunity and birth as the main drivers of the
377 susceptible host dynamics but other mechanisms can also contribute to the replenish-
378 ment of the susceptible population. In particular, pathogen evolution, especially the
379 emergence of antigenically novel strains, can cause effective waning of immunity in
380 the population; therefore, we hypothesize that the rate of antigenic evolution is likely
381 a key feature of pathogen resilience. Future studies should explore the relationship
382 between the rate of evolution and resilience for antigenically evolving pathogens.

383 Quantifying pathogen resilience also offers novel approaches to validating population-
384 level epidemiological models. So far, most model validation in infectious disease ecol-
385 ogy is based on the ability of a model to reproduce the observed epidemic dynamics
386 and to predict future dynamics [28, 29, 30, 31, 32]. However, many models can
387 perform similarly under these criteria. For example, two major RSV models have
388 been proposed to explain biennial epidemic patterns: (1) a stage- and age-structured
389 model that allows disease severity to vary with number of past infections and age of
390 infection [30] and (2) a pathogen-interaction model that accounts for cross immunity
391 between RSV and human metapneumovirus [29]. Since both models can accurately
392 reproduce the observed epidemic patterns, standard criteria for model validation
393 do not allow us to distinguish between these two models from population-level data
394 alone. Instead, it would be possible to measure the empirical resilience of each model
395 by simulating various perturbations and comparing the simulations to estimates of
396 empirical resilience from data, using pandemic perturbations as a reference.

397 There are several limitations to our work. First, we did not extensively explore
398 other approaches to reconstructing the attractor. Recent studies showed that more
399 sophisticated approaches, such as using non-uniform embedding, can provide more
400 robust reconstruction for noisy data [22]. In the context of causal inference, choices
401 about embedding can have major impact on the resulting inference [33]. Our re-
402 silience estimates are likely overly confident given a lack of uncertainties in attractor
403 reconstruction as well as the simplicity of our statistical framework. Nonetheless,
404 as illustrated in our sensitivity analyses, inferences about pathogen resilience in our
405 SIRS model appear to be robust to decisions about embedding lags and dimensions—

406 this is because tracking the rate at which the system approaches the attractor is likely
407 a much simpler problem than making inferences about causal directionality. Short
408 pre-pandemic time series also limit our ability to accurately reconstruct the attrac-
409 tor and contribute to the crudeness of our resilience estimates; although this is less
410 likely a problem for respiratory pathogens that are strongly annual, our attractor
411 reconstruction may be inaccurate for those exhibiting multi-annual cycles, such as
412 adenovirus in Hong Kong and Korea. Our framework also do not allow us to distin-
413 guish whether a system has settled to a new attractor or is experiencing long-term
414 transient behavior. Uncertainties in pathogen dynamics due to changes in testing
415 patterns further contribute to the crudeness of our resilience estimates. Despite these
416 limitations, our qualitative prediction that common respiratory pathogens are more
417 resilient than prevaccination measles is also likely to be robust, given how rapidly
418 many respiratory pathogens returned to their original cycles following pandemic per-
419 turbations.

420 Predicting the impact of anthropogenic changes on infectious disease dynamics
421 is a fundamental aim of infectious disease research in a rapidly changing world. Our
422 study illustrates that how a host-pathogen system responds to both small and large
423 perturbations is largely predictable through the lens of ecological resilience. In par-
424 ticular, quantifying the resilience of a host-pathogen system offers a unique insight
425 into questions about endemic pathogens' responses to pandemic perturbations, in-
426 cluding whether some pathogens will exhibit long-lasting impact from the pandemic
427 perturbation or not. More broadly, a detailed understanding of the determinants of
428 pathogen resilience can provide deeper understanding of pathogen persistence.

429 Materials and Methods

430 Data

431 We gathered time series on respiratory infections from Canada, Hong Kong, Korea,
432 and United States (US). As a reference, we also included time series data on norovirus
433 infections when available. In contrast to respiratory pathogens, we hypothesized
434 gastrointestinal viruses, such as norovirus, to be differently affected by pandemic
435 perturbations.

436 Weekly time series of respiratory infection cases in Canada came from a publicly
437 available website by the Respiratory Virus Detection Surveillance System, which
438 collects data from select laboratories across Canada [34]. Weekly time series of
439 respiratory infection cases in Hong Kong came from a publicly available website by
440 the Centre for Health Protection, Department of Health [35, 36]. Weekly time series
441 of acute respiratory infection cases in Korea came from a publicly available website
442 by the Korea Disease Control and Prevention Agency [37]. Finally, weekly time series
443 of respiratory infection cases in the US were obtained from the National Respiratory
444 and Enteric Virus Surveillance System. Time series on number of tests were also
445 available in Canada, Hong Kong, and the US, but not in Korea.

446 **Data processing**

447 For all time series, we rounded every year to 52 weeks by taking the average number
448 of cases and tests between the 52nd and 53rd week. We also rescale all time series to
449 account for changes in testing patterns, which were then used for the actual analysis.

450 For Canada, an increase in testing was observed from 2013 to 2024 (Supplemental
451 Figure S17). To account for this increase, we calculated a 2 year moving average
452 for the number of tests for each pathogen, which we used as a proxy for testing effort.
453 Then, we divided the smoothed testing patterns by the smoothed value at the final
454 week such that the testing effort has a maximum of 1. We then divided weekly cases
455 by the testing effort to obtain a scaled case time series. A similar approach was used
456 earlier for an analysis of RSV time series in the US to account for changes in testing
457 patterns [30].

458 For Hong Kong, we applied the same scaling procedure to the time series as we
459 did for Canada. In this case, we only adjusted for testing efforts up to the end of 2019
460 because there was a major reduction in testing for common respiratory pathogens
461 between 2020 and 2023 (Supplementary Figure S18).

462 For Korea, while we did not have information on testing, the reported number
463 of respiratory infections consistently increased from 2013 to the end of 2019, which
464 we interpreted as changes in testing patterns (Supplementary Figure S19). Since
465 we did not have testing numbers, we used the weekly sum of all acute respiratory
466 viral infection cases as a proxy for testing, which were further smoothed with moving
467 average and scaled to have a maximum of 1. For Korea, we also only adjusted for
468 testing efforts up to the end of 2019.

469 In the US, there has been a large increase in testing for some respiratory pathogens,
470 especially RSV, which could not be corrected by simple scaling (Supplementary Fig-
471 ure S20). Instead, we derived an incidence proxy by multiplying the test positivity
472 with influenza-like illness positivity, which was taken from [https://gis.cdc.gov/](https://gis.cdc.gov/grasp/fluvie)
473 [grasp/fluvie/fluportaldashboard.html](https://gis.cdc.gov/grasp/fluvie/fluportaldashboard.html). This method of estimating an inci-
474 dence proxy has been recently applied in the analysis of seasonal coronaviruses [7]
475 and *Mycoplasma pneumoniae* infections [4]. Detailed assumptions and justifications
476 are provided in [38].

477 **Data summary**

478 To make qualitative comparisons between pre- and post-perturbation dynamics of
479 respiratory pathogen circulation patterns, we calculated the mean seasonal patterns
480 using time series of either rescaled cases or incidence proxy estimates before 2020. We
481 did so by taking the mean value in each week across all years before 2020. Confidence
482 intervals around the means were calculated using a simple t test.

483 Estimating pathogen resilience

484 In order to measure pathogen resilience from surveillance data, we first reconstructed
 485 the empirical pre-pandemic attractor of the system using Takens' embedding theorem
 486 [17]. Specifically, for a given pathogen, we took the pre-pandemic (before 2020) case
 487 time series $C(t)$ and reconstructed the attractor using delayed embedding with a
 488 uniform delay of τ and dimension M :

$$X_{\tau,m}(t) = \langle \log(C(t) + 1), \log(C(t - \tau) + 1), \dots, \log(C(t - (M-1)\tau) + 1) \rangle. \quad (2)$$

489 Here, the delay τ was determined by calculating the autocorrelation of the logged
 490 pre-pandemic time series and asking when the autocorrelation crosses 0 for the first
 491 time [22]; a typical delay for an annual outbreak is around 13 weeks.

492 Then, for a given delay τ , we determined the embedding dimension M using the
 493 false nearest neighbors approach [21, 22]. To do so, we started with an embedding
 494 dimension e and constructed a set of points $A_{\tau,e} = \{X_{\tau,e}(t) | t < 2020\}$. Then, for
 495 each point $X_{\tau,e}(t)$, we determined the nearest neighbor from the set $A_{\tau,e}$, which we
 496 denote $X_{\tau,e}(t_{nn})$ for $t \neq t_{nn}$. Then, if the distance between these two points in the
 497 $e+1$ dimension, $D_{\tau,e+1}(t) = \|X_{\tau,e+1}(t_{nn}) - X_{\tau,e+1}(t)\|_2$, is larger than their distance
 498 in the e dimension, $D_{\tau,e}(t) = \|X_{\tau,e}(t_{nn}) - X_{\tau,e}(t)\|_2$, these two points are deemed to
 499 be false nearest neighbors; specifically, we used a threshold R for the ratio between
 500 two distances $D_{\tau,e+1}(t)/D_{\tau,e}(t)$ to determine false nearest neighbors. For the main
 501 analysis, we used $R = 10$, which was chosen from a sensitivity analysis against
 502 simulated data (Supplementary Text). Once we determined the embedding lag τ
 503 and dimension M , we apply the embedding to the entire time series and calculate
 504 the nearest neighbor distance against the attractor $A_{\tau,M}$ to obtain a time series of
 505 distance from the attractor $D_{\tau,M}(t)$.

506 From a time series of distances from the attractor, we estimated pathogen re-
 507 silience by fitting a linear regression to an appropriate window. To automatically se-
 508 lect the fitting window, we began by smoothing the distance time series using locally
 509 estimated scatterplot smoothing (LOESS) to obtain $\hat{D}_{\tau,M}(t)$, where the smoothing
 510 is performed on a log scale and exponentiated afterwards. Then, we determined
 511 threshold values (T_{start} and T_{end}) for the smoothed distances and choose the fitting
 512 window based on when $\hat{D}_{\tau,M}(t)$ crosses these threshold values for the first time.
 513 These thresholds were determined by first calculating the maximum distance,

$$\max \hat{D} = \max \hat{D}_{\tau,M}(t), \quad (3)$$

514 and the mean pre-pandemic distance,

$$\hat{D}_{\text{mean}} = \frac{1}{N_{t<2020}} \sum_{t<2020} \hat{D}_{\tau,M}(t), \quad (4)$$

515 as a reference, and then dividing their ratios into K equal bins,

$$T_{\text{start}} = \hat{D}_{\text{mean}} \times \left(\frac{\max \hat{D}}{\hat{D}_{\text{mean}}} \right)^{(K-a)/K}, \quad (5)$$

$$T_{\text{end}} = \hat{D}_{\text{mean}} \times \left(\frac{\max \hat{D}}{\hat{D}_{\text{mean}}} \right)^{a/K}, \quad (6)$$

516 where a represents the truncation threshold. This allows us to discard the initial
 517 period during which the distance increases (from the introduction of intervention
 518 measures) and the final period during which the distance plateaus (as the system
 519 reaches an attractor). The fitting window is determined based on when the smoothed
 520 distance $\hat{D}_{\tau,M}(t)$ crosses these threshold values for the first time; then, we fit a
 521 linear regression to logged (unsmoothed) distances $\log D_{\tau,M}(t)$ using that window.
 522 Alongside the threshold R for the false nearest neighbors approach, we tested optimal
 523 choices for K and a values using simulations (Supplementary Text). We used $K = 19$
 524 and $a = 2$ throughout the paper based on the simulation results.

525 Mathematical modeling

526 Throughout the paper, we use a series of mathematical models to illustrate the
 527 concept of pathogen resilience and to understand the determinants of pathogen re-
 528 silience. In general, the intrinsic resilience of a given system is given by the largest
 529 real part of the eigenvalues of the linearized system at endemic equilibrium. Here, we
 530 focus on the SIRS model with demography (birth and death) and present the details
 531 of other models in Supplementary Text. The SIRS (Susceptible-Infected-Recovered-
 532 Susceptible) model is the simplest model that allows for waning of immunity, where
 533 recovered (immune) individuals are assumed to become fully susceptible after an
 534 average of $1/\delta$ time period. The dynamics of the SIRS model is described by the
 535 following set of differential equations:

$$\frac{dS}{dt} = \mu - \beta(t)SI - \mu S + \delta R \quad (7)$$

$$\frac{dI}{dt} = \beta(t)SI - (\gamma + \mu)I \quad (8)$$

$$\frac{dR}{dt} = \gamma I - (\delta + \mu)R \quad (9)$$

536 where μ represents the birth and death rates, $\beta(t)$ represents the time-varying trans-
 537 mission rate, and γ represents the recovery rate. The basic reproduction number
 538 $\mathcal{R}_0(t) = \beta(t)/(\gamma + \mu)$ is defined as the average number of secondary infections that
 539 a single infected individual would cause in a fully susceptible population at time t
 540 and measures the intrinsic transmissibility of a pathogen.

541 When we introduced the idea of pathogen resilience (Figure 2), we imposed sinu-
 542 soidal changes to the transmission rate to account for seasonal transmission,

$$\beta(t) = b_1(1 + \theta \cos(2\pi(t - \phi)))\alpha(t), \quad (10)$$

543 where b_1 represents the baseline transmission rate, θ represents the seasonal ampli-
 544 tude, and ϕ represents the seasonal offset term. Here, we also introduced an extra

545 multiplicative term $\alpha(t)$ to account for the impact of pandemic perturbations, where
 546 $\alpha(t) < 1$ indicates transmission reduction. Figure 2A and 2B were generated assum-
 547 ing $b_1 = 3 \times (365/7 + 1/50)/\text{years}$, $\theta = 0.2$, $\phi = 0$, $\mu = 1/50/\text{years}$, $\gamma = 365/7/\text{years}$,
 548 and $\delta = 1/2/\text{years}$. Specifically, $b_1 = 3 \times (365/7 + 1/50)/\text{years}$ implies $\mathcal{R}_0 = 3$, where
 549 $(365/7 + 1/50)/\text{years}$ represent the rate of recovery. In Figure 2A, we imposed a 50%
 550 transmission reduction for 6 months from 2020:

$$\alpha(t) = \begin{cases} 0.5 & 2020 \leq t < 2020.5 \\ 1 & \text{otherwise} \end{cases} \quad (11)$$

551 In Figure 2B, we imposed a 50% transmission reduction for 6 months from 2020 and
 552 a permanent 10% reduction onward:

$$\alpha(t) = \begin{cases} 1 & t < 2020 \\ 0.5 & 2020 \leq t < 2020.5 \\ 0.9 & 2020.5 \leq t \end{cases} \quad (12)$$

553 In both scenarios, we simulated the SIRS model from the same initial conditions
 554 ($S(0) = 1/\mathcal{R}_0$, $I(0) = 1 \times 10^{-6}$, and $R(0) = 1 - S(0) - I(0)$) from 1900 until 2030.

555 To measure the empirical resilience of the SIRS model (Figure 2C and 2F), we
 556 computed the normalized distance between post-intervention susceptible and logged
 557 infected proportions and their corresponding unperturbed values at the same time:

$$\sqrt{\left(\frac{S(t) - S_{\text{unperturbed}}(t)}{\sigma_S}\right)^2 + \left(\frac{\log I(t) - \log I_{\text{unperturbed}}(t)}{\sigma_{\log I}}\right)^2}, \quad (13)$$

558 where σ_S and $\sigma_{\log I}$ represent the standard deviation in the unperturbed susceptible
 559 and logged infected proportions. The unperturbed values were obtained by simulat-
 560 ing the same SIRS model without pandemic perturbations ($\alpha = 1$). We normalized
 561 the differences in susceptible and logged infected proportions to allow both quantities
 562 to equally contribute to the changes in distance from the attractor. We used logged
 563 prevalence, instead of absolute prevalence, in order to capture epidemic dynamics
 564 in deep troughs during the intervention period. In Supplementary Materials, we
 565 also compared how the degree of seasonal transmission affects empirical resilience
 566 by varying θ from 0 to 0.4; when we assumed no seasonality ($\theta = 0$), we did not
 567 normalize the distance because the standard deviation of pre-intervention dynamics
 568 are zero.

569 We used the SIRS model to understand how underlying epidemiological param-
 570 eters affect pathogen resilience and determine the relationship to underlying sus-
 571 ceptible host dynamics. For the simple SIRS model without seasonal transmission
 572 ($\theta = 0$), the intrinsic resilience equals

$$-\frac{\text{Re}(\lambda)}{2} = \frac{\delta + \beta I^* + \mu}{2}. \quad (14)$$

573 Here, I^* represents the prevalence at endemic equilibrium:

$$I^* = \frac{(\delta + \mu)(\beta - (\gamma + \mu))}{\beta(\delta + \gamma + \mu)}. \quad (15)$$

574 The susceptible replenishment rate is given by

$$S_{\text{replenish}} = \frac{\mu + \delta R}{S^*}, \quad (16)$$

575 where $S^* = 1/\mathcal{R}_0$ represents the equilibrium proportion of susceptible individuals.
576 We varied the basic reproduction number \mathcal{R}_0 between 1.1 to 6 and the average
577 duration of immunity $1/\delta$ between 2 to 4 years, and computed these two quantities.
578 In doing so, we fixed all other parameters: $\mu = 1/80/\text{years}$ and $\gamma = 365/7/\text{years}$.
579 When infection provides a life-long immunity ($\delta = 0$), the intrinsic resilience is
580 inversely proportional to the susceptible replenishment rate:

$$-\frac{\text{Re}(\lambda)}{2} = \frac{\mu}{2S^*} = \frac{S_{\text{replenish}}}{2}. \quad (17)$$

581 Finally, we used a seasonally unforced stochastic SIRS model without demog-
582 raphy to understand how pathogen resilience affects sensitivity of the system to
583 demographic stochasticity (see Supplementary Text for the details of the stochas-
584 tic SIRS model). By varying the basic reproduction number \mathcal{R}_0 between 2 to 20
585 and the average duration of immunity $1/\delta$ between 1 to 40 years, we ran the SIRS
586 model for 100 years and computed the epidemic amplitude, which we defined as
587 $(\max I - \min I)/(2\bar{I})$. Each simulation began from the equilibrium, and we trun-
588 cated the initial 25 years before computing the epidemic amplitude. In doing so,
589 we assumed $\gamma = 365/7/\text{years}$ and fixed the population size to 1 billion to prevent
590 any fadeouts. We also considered a seasonally forced stochastic SIRS model without
591 demography, assuming an amplitude of seasonal forcing of 0.04; in this case, we com-
592 puted the relative epidemic amplitude by comparing the deterministic and stochastic
593 trajectories (Supplementary Materials).

594 Data availability

595 All data and code are stored in a publicly available GitHub repository (<https://github.com/cobeylab/return-time>).

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604 **Supplementary Text**

605 **Resilience of a stage-structured system.**

606 In Figure 2G–I, we used a more realistic, stage-structured model to illustrate how
 607 transient phenomena can cause the system to slow down. Specifically, we used the
 608 stage-structured RSV model proposed by [30], which assumes that subsequent rein-
 609 fections cause an individual to become less susceptible and transmissible than previ-
 610 ous infections. In contrast to a standard SIRS model, this model does not include a
 611 recovered compartment, which allow for temporary protection against new infections,
 612 and assumes that recovered individuals are immediately susceptible to new infections.
 613 The model dynamics can be described as follows:

$$\frac{dM}{dt} = \mu - (\omega + \mu)M \quad (S1)$$

$$\frac{dS_0}{dt} = \omega M - (\lambda(t) + \mu)S_0 \quad (S2)$$

$$\frac{dI_1}{dt} = \lambda(t)S_0 - (\gamma_1 + \mu)I_1 \quad (S3)$$

$$\frac{dS_1}{dt} = \gamma_1 I_1 - (\sigma_1 \lambda(t) + \mu)S_1 \quad (S4)$$

$$\frac{dI_2}{dt} = \sigma_1 \lambda(t)S_1 - (\gamma_2 + \mu)I_2 \quad (S5)$$

$$\frac{dS_2}{dt} = \gamma_2 I_2 - (\sigma_2 \lambda(t) + \mu)S_2 \quad (S6)$$

$$\frac{dI_3}{dt} = \sigma_2 \lambda(t)S_2 - (\gamma_3 + \mu)I_3 \quad (S7)$$

$$\frac{dS_3}{dt} = \gamma_3 I_3 - (\sigma_3 \lambda(t) + \mu)S_3 + \gamma_4 I_4 \quad (S8)$$

$$\frac{dI_4}{dt} = \sigma_3 \lambda(t)S_3 - (\gamma_4 + \mu)I_4 \quad (S9)$$

614 where M represents the proportion of individuals who are maternally immune; S_i
 615 represents the proportion of individuals who are susceptible after i prior infections; I_i
 616 represents the proportion of individuals who are currently (re)-infected with their i -th
 617 infection; μ represents the birth and death rates; $1/\omega$ represents the mean duration
 618 of maternal immunity; $1/\gamma_i$ represents the mean duration of infection; $\lambda(t)$ represents
 619 the force of infection; and σ_i represents the reduction in susceptibility for the i -th
 620 reinfection. The force of infection is modeled using a sinusoidal function:

$$\beta(t) = b_1(1 + \theta \cos(2\pi(t - \phi)))\alpha(t) \quad (S10)$$

$$\lambda(t) = \beta(I_1 + \rho_1 I_2 + \rho_2(I_3 + I_4)), \quad (S11)$$

621 where b_1 represents the baseline transmission rate; θ represents the seasonal ampli-
 622 tude; ϕ represents the seasonal offset term; $\alpha(t)$ represents the intervention effect;

and ρ_i represents the impact of immunity on transmission reduction. We used the following parameters to simulate the impact of interventions on epidemic dynamics [30]: $b_1 = 9 \times (365/10 + 1/80)/\text{years}$, $\theta = 0.2$, $\phi = -0.1$, $\omega = 365/112/\text{years}$, $\gamma_1 = 365/10/\text{years}$, $\gamma_2 = 365/7/\text{years}$, $\gamma_3 = 365/5/\text{years}$, $\sigma_1 = 0.76$, $\sigma_2 = 0.6$, $\sigma_3 = 0.4$, $\rho_1 = 0.75$, $\rho_2 = 0.51$, and $\mu = 1/80/\text{years}$. We assumed a 50% transmission reduction for 6 months from 2020:

$$\alpha(t) = \begin{cases} 0.5 & 2020 \leq t < 2020.5 \\ 1 & \text{otherwise} \end{cases} \quad (\text{S12})$$

The model was simulated from 1900 to 2030 using the following initial conditions: $M = 0$, $S_0 = 1/\mathcal{R}_0 - I_1$, $I_1 = 1 \times 10^{-6}$, $S_1 = 1 - 1/\mathcal{R}_0$, $I_2 = 0$, $S_2 = 0$, $I_3 = 0$, $S_3 = 0$, and $I_4 = 0$. For the phase plane analysis (Figure 2H) and distance analysis (Figure 2I), we relied on the average susceptibility,

$$\bar{S} = S_0 + \sigma_1 S_1 + \sigma_2 S_2 + \sigma_3 S_3, \quad (\text{S13})$$

and total prevalence,

$$I_{\text{total}} = I_1 + I_2 + I_3 + I_4. \quad (\text{S14})$$

These quantities were used to compute the normalized distance from the attractor, as described in the main text.

Resilience of a multistain system.

We used a simple two-strain model to show that a multistain host-pathogen system that is coupled through cross immunity can be described by a single resilience value. The model dynamics can be described as follows [29]:

$$\frac{dS}{dt} = \mu - \mu S - (\lambda_1 + \lambda_2)S + \delta_1 R_1 + \delta_2 R_2 \quad (\text{S15})$$

$$\frac{dI_1}{dt} = \lambda_1 S - (\gamma_1 + \mu)I_1 \quad (\text{S16})$$

$$\frac{dI_2}{dt} = \lambda_2 S - (\gamma_2 + \mu)I_2 \quad (\text{S17})$$

$$\frac{dR_1}{dt} = \gamma_1 I_1 - \lambda_2 \epsilon_{21} R_1 - \delta_1 R_1 + \delta_2 R - \mu R_1 \quad (\text{S18})$$

$$\frac{dR_2}{dt} = \gamma_2 I_2 - \lambda_1 \epsilon_{12} R_2 - \delta_2 R_2 + \delta_1 R - \mu R_2 \quad (\text{S19})$$

$$\frac{dJ_1}{dt} = \lambda_1 \epsilon_{12} R_2 - (\gamma_1 + \mu) J_1 \quad (\text{S20})$$

$$\frac{dJ_2}{dt} = \lambda_2 \epsilon_{21} R_1 - (\gamma_2 + \mu) J_2 \quad (\text{S21})$$

$$\frac{dR}{dt} = \gamma_1 J_1 + \gamma_2 J_2 - \delta_1 R - \delta_2 R - \mu R \quad (\text{S22})$$

where S represents the proportion of individuals who are fully susceptible to infections by both strains; I_1 represents the proportion of individuals who are infected with strain 1 without prior immunity; I_2 represents the proportion of individuals who are infected with strain 2 without prior immunity; R_1 represents the proportion of individuals who are fully immune against strain 1 and partially susceptible to reinfection by strain 2; R_2 represents the proportion of individuals who are fully immune against strain 2 and partially susceptible to reinfection by strain 1; J_1 represents the proportion of individuals who are infected with strain 1 with prior immunity against strain 2; J_2 represents the proportion of individuals who are infected with strain 2 with prior immunity against strain 1; R represents the proportion of individuals who are immune to infections from both strains; μ represents the birth/death rate; λ_1 and λ_2 represent the force of infection from strains 1 and 2, respectively; δ_1 and δ_2 represent the waning immunity rate; γ_1 and γ_2 represent the recovery rate; ϵ_{12} and ϵ_{21} represent the susceptibility to reinfection with strains 2 and 1, respectively, given prior immunity from infection with strains 1 and 2, respectively. The force of infection is modeled as follows:

$$\beta_1 = b_1(1 + \theta_1 \sin(2\pi(t - \phi_1)))\alpha(t) \quad (\text{S23})$$

$$\beta_2 = b_2(1 + \theta_2 \sin(2\pi(t - \phi_2)))\alpha(t) \quad (\text{S24})$$

$$\lambda_1 = \beta_1(I_1 + J_1) \quad (\text{S25})$$

$$\lambda_2 = \beta_2(I_2 + J_2) \quad (\text{S26})$$

In Supplementary Figures S2–S4, we assumed the following parameters: $b_1 = 2 \times 52/\text{years}$, $b_2 = 4 \times 52/\text{years}$, $\phi_1 = \phi_2 = 0$, $\epsilon_{12} = 0.9$, $\epsilon_{21} = 0.5$, $\gamma_1 = \gamma_2 = 52/\text{years}$, $\delta_1 = \delta_2 = 1/\text{years}$, and $\mu = 1/70/\text{years}$. For all simulations, we assumed a 50% transmission reduction for 6 months from 2020:

$$\alpha(t) = \begin{cases} 0.5 & 2020 \leq t < 2020.5 \\ 1 & \text{otherwise} \end{cases} \quad (\text{S27})$$

The seasonal amplitude θ is varied from 0 to 0.4. All simulations were run from 1900 to 2030 with following initial conditions: $S(0) = 1 - 2 \times 10^{-6}$, $I_1(0) = 1 \times 10^{-6}$, $I_2(0) = 1 \times 10^{-6}$, $R_1(0) = 0$, $R_2(0) = 0$, $J_1(0) = 0$, $J_2(0) = 0$, and $R(0) = 0$.

We considered three scenarios for measuring pathogen resilience: (1) we only have information about strain 1, (2) we only have information about strain 2, and (3) we are unable to distinguish between strains. In the first two scenarios (see panels A–C for strain 1 and panels D–F for strain 2), we considered the dynamics of average susceptibility for each strain and total prevalence:

$$\bar{S}_1 = S + \epsilon_{12}R_2 \quad (\text{S28})$$

$$\hat{I}_1 = I_1 + J_1 \quad (\text{S29})$$

$$\bar{S}_2 = S + \epsilon_{21}R_1 \quad (\text{S30})$$

$$\hat{I}_2 = I_2 + J_2 \quad (\text{S31})$$

668 In the third scenario (panels G–I), we considered the dynamics of total susceptible
669 and infected populations:

$$\hat{S} = S + R_2 + R_1 \quad (\text{S32})$$

$$\hat{I} = I_1 + J_1 + I_2 + J_2 \quad (\text{S33})$$

670 These quantities were used to compute the normalized distance from the attractor,
671 as described in the main text.

672 Estimating intrinsic resilience using a mechanistic model

673 We tested whether we can reliably estimate the intrinsic resilience of a system by fit-
674 ting a mechanistic model. Specifically, we simulated case time series from stochastic
675 SIRS and two-strain models and fitted a simple, deterministic SIRS model using a
676 Bayesian framework [4, 23, 39].

677 We simulated the models in discrete time with a daily time step ($\Delta t = 1$),
678 incorporating demographic stochasticity:

$$\beta(t) = \mathcal{R}_0 \left(1 + \theta \cos \left(\frac{2\pi t}{364} \right) \right) \alpha(t)(1 - \exp(-\gamma)) \quad (\text{S34})$$

$$\text{FOI}(t) = \beta(t)I(t - \Delta t)/N \quad (\text{S35})$$

$$B(t) \sim \text{Poisson}(\mu N) \quad (\text{S36})$$

$$\Delta S(t) \sim \text{Binom}(S(t - \Delta t), 1 - \exp(-(\text{FOI}(t) + \mu)\Delta t)) \quad (\text{S37})$$

$$N_{SI}(t) \sim \text{Binom}\left(\Delta S(t), \frac{\text{FOI}(t)}{\text{FOI}(t) + \mu}\right) \quad (\text{S38})$$

$$\Delta I(t) \sim \text{Binom}(I(t - \Delta t), 1 - \exp(-(\gamma + \mu)\Delta t)) \quad (\text{S39})$$

$$N_{IR}(t) \sim \text{Binom}\left(\Delta I(t), \frac{\gamma}{\gamma + \mu}\right) \quad (\text{S40})$$

$$\Delta R(t) \sim \text{Binom}(R(t - \Delta t), 1 - \exp(-(\delta + \mu)\Delta t)) \quad (\text{S41})$$

$$N_{RS}(t) \sim \text{Binom}\left(\Delta R(t), \frac{\delta}{\delta + \mu}\right) \quad (\text{S42})$$

$$S(t) = S(t - \Delta t) + N_{RS}(t) + B(t) - \Delta S(t) \quad (\text{S43})$$

$$I(t) = I(t - \Delta t) + N_{SI}(t) - \Delta I(t) \quad (\text{S44})$$

$$R(t) = R(t - \Delta t) + N_{IR}(t) - \Delta R(t) \quad (\text{S45})$$

679 where FOI represents the force of infection; N_{ij} represents the number of individuals
680 moving from compartment i to j on a given day; and $B(t)$ represents the number
681 of new births. All other parameters definitions can be found in the description of
682 the deterministic version of the model. We simulated the model on a daily scale—
683 assuming 364 days in a year so that it can be evenly grouped into 52 weeks—with
684 the following parameters: $\mathcal{R}_0 = 3$, $\theta = 0.1$, $\gamma = 1/7/\text{days}$, $\delta = 1/(364 \times 2)/\text{days}$,

685 $\mu = 1/(364 \times 50)/\text{days}$, and $N = 1 \times 10^8$. The model was simulated from 1900 to
686 2030 assuming $S(0) = N/3$, $I(0) = 100$, and $R(0) = N - S(0) - I(0)$. The observed
687 incidence from the model was then simulated as follows:

$$C(t) = \text{Beta-Binom}(N_{SI}(t), \rho, k), \quad (\text{S46})$$

688 where ρ represents the reporting probability and k represents the overdispersion
689 parameter of beta-binomial distribution. Here, we used the beta-binomial distribu-
690 tion to account for overdispersion in reporting. We assumed $\rho = 0.002$ (i.e., 0.2%
691 probability) and $k = 1000$.

692 We used an analogous approach for the two-strain model:

$$\beta_1(t) = b_1 \left(1 + \theta_1 \cos \left(\frac{2\pi(t - \phi_1)}{364} \right) \right) \alpha(t) \quad (\text{S47})$$

$$\beta_2(t) = b_2 \left(1 + \theta_2 \cos \left(\frac{2\pi(t - \phi_2)}{364} \right) \right) \alpha(t) \quad (\text{S48})$$

$$\text{FOI}_1(t) = \beta_1(t)(I_1(t - \Delta t) + J_1(t - \Delta t))/N \quad (\text{S49})$$

$$\text{FOI}_2(t) = \beta_2(t)(I_2(t - \Delta t) + J_2(t - \Delta t))/N \quad (\text{S50})$$

$$B(t) \sim \text{Poisson}(\mu N) \quad (\text{S51})$$

$$\Delta S(t) \sim \text{Binom}(S(t - \Delta t), 1 - \exp(-(\text{FOI}_1(t) + \text{FOI}_2(t) + \mu)\Delta t)) \quad (\text{S52})$$

$$N_{SI_1}(t) \sim \text{Binom}\left(\Delta S(t), \frac{\text{FOI}_1(t)}{\text{FOI}_1(t) + \text{FOI}_2(t) + \mu}\right) \quad (\text{S53})$$

$$N_{SI_2}(t) \sim \text{Binom}\left(\Delta S(t), \frac{\text{FOI}_2(t)}{\text{FOI}_1(t) + \text{FOI}_2(t) + \mu}\right) \quad (\text{S54})$$

$$\Delta I_1(t) \sim \text{Binom}(I_1(t - \Delta t), 1 - \exp(-(\gamma_1 + \mu)\Delta t)) \quad (\text{S55})$$

$$N_{I_1 R_1}(t) \sim \text{Binom}\left(\Delta I_1(t), \frac{\gamma_1}{\gamma_1 + \mu}\right) \quad (\text{S56})$$

$$\Delta I_2(t) \sim \text{Binom}(I_2(t - \Delta t), 1 - \exp(-(\gamma_2 + \mu)\Delta t)) \quad (\text{S57})$$

$$N_{I_2 R_2}(t) \sim \text{Binom}\left(\Delta I_2(t), \frac{\gamma_2}{\gamma_2 + \mu}\right) \quad (\text{S58})$$

$$\Delta R_1(t) \sim \text{Binom}(R_1(t - \Delta t), 1 - \exp(-(\epsilon_{21}\text{FOI}_2(t) + \rho_1 + \mu)\Delta t)) \quad (\text{S59})$$

$$N_{R_1 S}(t) \sim \text{Binom}\left(\Delta R_1(t), \frac{\rho_1}{\epsilon_{21}\text{FOI}_2(t) + \rho_1 + \mu}\right) \quad (\text{S60})$$

$$N_{R_1 J_2}(t) \sim \text{Binom}\left(\Delta R_1(t), \frac{\epsilon_{21}\text{FOI}_2(t)}{\epsilon_{21}\text{FOI}_2(t) + \rho_1 + \mu}\right) \quad (\text{S61})$$

$$\Delta R_2(t) \sim \text{Binom}(R_2(t - \Delta t), 1 - \exp(-(\epsilon_{12}\text{FOI}_1(t) + \rho_2 + \mu)\Delta t)) \quad (\text{S62})$$

$$N_{R_2 S}(t) \sim \text{Binom}\left(\Delta R_2(t), \frac{\rho_2}{\epsilon_{12}\text{FOI}_1(t) + \rho_2 + \mu}\right) \quad (\text{S63})$$

$$N_{R_2 J_1}(t) \sim \text{Binom}\left(\Delta R_2(t), \frac{\epsilon_{12}\text{FOI}_1(t)}{\epsilon_{12}\text{FOI}_1(t) + \rho_2 + \mu}\right) \quad (\text{S64})$$

$$\Delta J_1(t) \sim \text{Binom}(J_1(t - \Delta t), 1 - \exp(-(\gamma_1 + \mu)\Delta t)) \quad (\text{S65})$$

$$N_{J_1R}(t) \sim \text{Binom}\left(\Delta J_1(t), \frac{\gamma_1}{\gamma_1 + \mu}\right) \quad (\text{S66})$$

$$\Delta J_2(t) \sim \text{Binom}(J_2(t - \Delta t), 1 - \exp(-(\gamma_2 + \mu)\Delta t)) \quad (\text{S67})$$

$$N_{J_2R}(t) \sim \text{Binom}\left(\Delta J_2(t), \frac{\gamma_2}{\gamma_2 + \mu}\right) \quad (\text{S68})$$

$$\Delta R(t) \sim \text{Binom}(R(t - \Delta t), 1 - \exp(-(\rho_1 + \rho_2 + \mu)\Delta t)) \quad (\text{S69})$$

$$N_{RR_1}(t) \sim \text{Binom}\left(\Delta R(t), \frac{\rho_1}{\rho_1 + \rho_2 + \mu}\right) \quad (\text{S70})$$

$$N_{RR_2}(t) \sim \text{Binom}\left(\Delta R(t), \frac{\rho_2}{\rho_1 + \rho_2 + \mu}\right) \quad (\text{S71})$$

$$S(t) = S(t - \Delta t) - \Delta S(t) + B(t) + N_{R_1S}(t) + N_{R_2S}(t) \quad (\text{S72})$$

$$I_1(t) = I_1(t - \Delta t) - \Delta I_1(t) + N_{SI_1}(t) \quad (\text{S73})$$

$$I_2(t) = I_2(t - \Delta t) - \Delta I_2(t) + N_{SI_2}(t) \quad (\text{S74})$$

$$R_1(t) = R_1(t - \Delta t) - \Delta R_1(t) + N_{I_1R_1}(t) + N_{RR_1}(t) \quad (\text{S75})$$

$$R_2(t) = R_2(t - \Delta t) - \Delta R_2(t) + N_{I_2R_2}(t) + N_{RR_2}(t) \quad (\text{S76})$$

$$J_1(t) = J_1(t - \Delta t) - \Delta J_1(t) + N_{R_2J_1}(t) \quad (\text{S77})$$

$$J_2(t) = J_2(t - \Delta t) - \Delta J_2(t) + N_{R_1J_2}(t) \quad (\text{S78})$$

$$R(t) = R(t - \Delta t) - \Delta R(t) + N_{J_1R}(t) + N_{J_2R}(t) \quad (\text{S79})$$

We simulated the model on a daily scale with previously estimated parameters for the RSV-HMPV interaction [29]: $b_1 = 1.7/\text{weeks}$, $b_2 = 1.95/\text{weeks}$, $\theta_1 = 0.4$, $\theta_2 = 0.3$, $\phi_1 = 0.005 \times 7/364$, $\phi_2 = 4.99 \times 7/364$, $\epsilon_{12} = 0.92$, $\epsilon_{21} = 0.45$, $\gamma_1 = 1/10/\text{days}$, $\gamma_2 = 1/10/\text{days}$, $\rho_1 = 1/364/\text{days}$, $\rho_2 = 1/364/\text{days}$, $\mu = 1/(70 \times 364)/\text{days}$, and $N = 1 \times 10^8$. The model was simulated from 1900 to 2030 assuming $S(0) = N - 200$, $I_1(0) = 100$, $I_2(0) = 100$, $R_1(0) = 0$, $R_2(0) = 0$, $J_1(0) = 0$, $J_2(0) = 0$, and $R(0) = 0$. The observed incidence for each strain is then simulated as follows:

$$C_1(t) = \text{Beta-Binom}(N_{SI_1}(t) + N_{R_2J_1}, \rho, k), \quad (\text{S80})$$

$$C_2(t) = \text{Beta-Binom}(N_{SI_2}(t) + N_{R_1J_2}, \rho, k), \quad (\text{S81})$$

where ρ represents the reporting probability and k represents the overdispersion parameter of beta-binomial distribution. We assumed $\rho = 0.002$ (i.e., 0.2% probability) and $k = 500$. We also considered the total incidence: $C_{\text{total}}(t) = C_1(t) + C_2(t)$.

For both models, we considered a more realistically shaped pandemic perturbation $\alpha(t)$ to challenge our ability to estimate the intervention effects. Thus, we assumed a 40% transmission reduction for 3 months from March 2020, followed by a 10% transmission reduction for 6 months, 20% transmission reduction for 3 months,

707 and a final return to normal levels:

$$\alpha(t) = \begin{cases} 1 & t < 2020.25 \\ 0.6 & 2020.25 \leq t < 2020.5 \\ 0.9 & 2020.5 \leq t < 2021 \\ 0.8 & 2021 \leq t < 2021.25 \\ 1 & 2021.25 \leq t \end{cases}. \quad (\text{S82})$$

708 For all simulations, we truncated the time series from the beginning of 2014 to the
709 end of 2023 and aggregated them into weekly cases.

710 To infer intrinsic resilience from time series, we fitted a simple discrete time,
711 deterministic SIRS model in a Bayesian framework [4]:

$$\Delta t = 1 \text{ week} \quad (\text{S83})$$

$$\text{FOI}(t) = \beta(t)(I(t - \Delta t) + \omega)/N \quad (\text{S84})$$

$$\Delta S(t) = [1 - \exp(-(\text{FOI}(t) + \mu)\Delta t)] S(t - \Delta t) \quad (\text{S85})$$

$$N_{SI}(t) = \frac{\text{FOI}(t)\Delta S(t)}{\text{FOI}(t) + \mu} \quad (\text{S86})$$

$$\Delta I(t) = [1 - \exp(-(\gamma + \mu)\Delta t)] I(t - \Delta t) \quad (\text{S87})$$

$$N_{IR}(t) = \frac{\gamma \Delta I(t)}{\gamma + \mu} \quad (\text{S88})$$

$$\Delta R(t) = [1 - \exp(-(\nu + \mu)\Delta t)] R(t - \Delta t) \quad (\text{S89})$$

$$N_{RS}(t) = \frac{\nu \Delta R(t)}{\nu + \mu} \quad (\text{S90})$$

$$S(t) = S(t - \Delta t) + \mu S - \Delta S(t) + N_{RS}(t) \quad (\text{S91})$$

$$I(t) = I(t - \Delta t) - \Delta I(t) + N_{SI}(t) \quad (\text{S92})$$

$$R(t) = R(t - \Delta t) - \Delta R(t) + N_{IR}(t) \quad (\text{S93})$$

712 where we include an extra term ω to account for importation. Although actual
713 simulations did not include any importation, we had found that including this term
714 generally helped with model convergence in previous analyses [4]. The transmission
715 rate was divided into a seasonal term $\beta_{\text{seas}}(t)$ (repeated every year) and intervention
716 term $\alpha(t)$, which were estimated jointly:

$$\beta(t) = \beta_{\text{seas}}(t)\alpha(t), \quad (\text{S94})$$

717 where $\alpha < 1$ corresponds to reduction in transmission due to intervention effects. To
718 constrain the smoothness of $\beta_{\text{seas}}(t)$, we imposed cyclic, random-walk priors:

$$\beta_{\text{seas}}(t) \sim \text{Normal}(\beta_{\text{seas}}(t - 1), \sigma) \quad t = 2 \dots 52 \quad (\text{S95})$$

$$\beta_{\text{seas}}(1) \sim \text{Normal}(\beta_{\text{seas}}(52), \sigma) \quad (\text{S96})$$

⁷¹⁹ We fixed $\alpha(t) = 1$ for all $t < 2020$ and estimate α assuming a normal prior:

$$\alpha \sim \text{Normal}(1, 0.25). \quad (\text{S97})$$

⁷²⁰ We assumed weakly informative priors on ω and ν :

$$\omega \sim \text{Normal}(0, 200) \quad (\text{S98})$$

$$\nu \sim \text{Normal}(104, 26) \quad (\text{S99})$$

⁷²¹ We assumed that the true birth/death rates, population sizes, and recovery rates
⁷²² are known. We note, however, that assuming $\gamma = 1/\text{week}$ actually corresponds to
⁷²³ a mean simulated infectious period of 1.6 weeks due to a time discretization, which
⁷²⁴ is much longer than the true value; this approximation allows us to test whether we
⁷²⁵ can still robustly estimate the intrinsic resilience given parameter mis-specification.
⁷²⁶ Initial conditions were estimated with following priors:

$$S(0) = Ns(0) \quad (\text{S100})$$

$$I(0) = Ni(0) \quad (\text{S101})$$

$$s(0) \sim \text{Uniform}(0, 1) \quad (\text{S102})$$

$$i(0) \sim \text{Half-Normal}(0, 0.001) \quad (\text{S103})$$

⁷²⁷ Finally, the observation model was specified as follows:

$$\text{Cases}(t) \sim \text{Negative-Binomial}(\rho N_{SI}(t), \phi) \quad (\text{S104})$$

$$\rho \sim \text{Half-Normal}(0, 0.02) \quad (\text{S105})$$

$$\phi \sim \text{Half-Normal}(0, 10) \quad (\text{S106})$$

⁷²⁸ where ρ represents the reporting probability and ϕ represents the negative binomial
⁷²⁹ overdispersion parameter.

⁷³⁰ The model was fitted to four separate time series: (1) incidence time series from
⁷³¹ the SIRS model, (2) incidence time series for strain 1 from the two-strain model,
⁷³² (3) incidence time series for strain 2 from the two-strain model, and (4) combined
⁷³³ incidence time series for strains 1 and 2 from the two-strain model. The model was
⁷³⁴ fitted using rstan [40, 41]. The resulting posterior distribution was used to calculate
⁷³⁵ the intrinsic resilience of the seasonally unforced system with the same parameters;
⁷³⁶ eigenvalues of the discrete-time SIR model were computed by numerically finding
⁷³⁷ the equilibrium and calculating the Jacobian matrix.

⁷³⁸ **Validations for window-selection criteria**

⁷³⁹ We used stochastic SIRS simulations to identify optimal parameters for the window-
⁷⁴⁰ selection criteria that we used for the linear regression for estimating empirical re-
⁷⁴¹ silience. For each simulation, we began by generating a random perturbation $\alpha(t)$
⁷⁴² from a random set of parameters. First, we drew the duration of perturbation τ_{np}^i

743 from a uniform distribution between 1 and 2 years. Then, we drew independent
 744 normal variables z_i of length $\lfloor 364\tau_{\text{np}} \rfloor$ with a standard deviation of 0.02 and took a
 745 reverse cumulative sum to obtain a realistic shape for the perturbation:

$$x_n = 1 + \sum_{i=n}^{\lfloor 364\tau_{\text{np}} \rfloor} z_i, \quad n = 1, \dots, \lfloor 364\tau_{\text{np}} \rfloor. \quad (\text{S107})$$

746 In contrast to simple perturbations that assume a constant reduction in transmission,
 747 this approach allows us to model transmission reduction that varies over time
 748 smoothly. We repeated this random generation process until less than 10% of x_n
 749 exceeds 1—this was done to ensure the perturbation term $\alpha(t)$ stays below 1 (and
 750 therefore reduce transmission) for the most part. Then, we set any values that are
 751 above 1 or below 0 to 1 and 0, respectively. Then, we randomly drew the minimum
 752 transmission during perturbation α_{\min} from a uniform distribution between 0.5 and
 753 0.7 and scale x_n to have a minimum of α_{\min} :

$$x_{\text{scale},n} = \alpha_{\min} + (1 - \alpha_{\min}) \times \frac{x_n - \min x_n}{1 - \min x_n}. \quad (\text{S108})$$

754 This allowed us to simulate a realistically shaped perturbation:

$$\alpha(t) = \begin{cases} 1 & t < 2020 \\ x_{\text{scale},364(t-2020)} & 2020 \leq t < 2020 + \tau_{\text{np}} \\ 1 & \tau_{\text{np}} \leq t \end{cases}. \quad (\text{S109})$$

755 Given this perturbation function, we draw \mathcal{R}_0 from a uniform distribution between
 756 1.5 and 4 and the mean duration of immunity $1/\delta$ from a uniform distribution be-
 757 tween 1 and 4. Then, we simulate the stochastic SIRS model from $S(0) = 10^8/\mathcal{R}_0$
 758 and $I(0) = 100$ from 1990 to 2025 and truncate the time series to 2014–2025; if the
 759 epidemic becomes extinct before the end of simulation, we discard that simulation
 760 and start over from the perturbation generation step.

761 For each epidemic simulation, we computed the empirical resilience by varying
 762 the threshold R for the nearest neighbor approach from 4 to 14 with increments of
 763 2, the number of divisions K for the window selection between 8 and 25, and the
 764 truncation threshold a for the window selection between 1 to 3; this was done for all
 765 possible combinations of R , K , and a . We also compared this with the naive approach
 766 that uses the entire distance-from-attractor time series, starting from the maximum
 767 distance to the end of the time series. We repeated this procedure 500 times and
 768 quantified the correlation between empirical and intrinsic resilience estimates across
 769 two approaches.

⁷⁷⁰ **Supplementary Figures**

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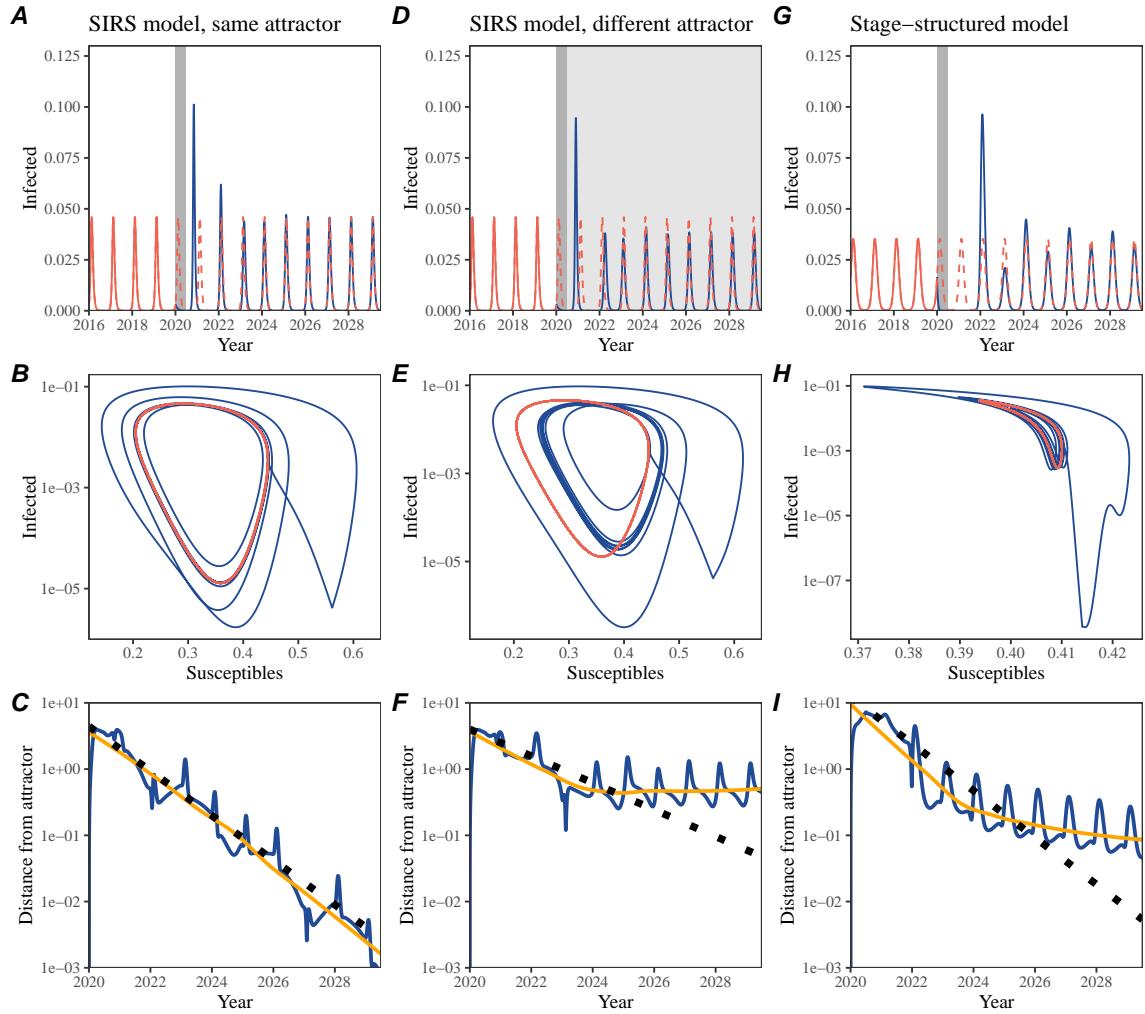


Figure 2: A simple method to measure pathogen resilience following pandemic perturbations across different scenarios. (A, D, G) Simulated epidemic trajectories across various models. Red and blue solid lines represent epidemic dynamics before and after pandemic perturbations are introduced, respectively. Red dashed lines represent counterfactual epidemic dynamics in the absence of perturbations. Gray regions indicate the duration of perturbations with the dark gray region representing a 50% transmission reduction and the light gray region representing a 10% transmission reduction. (B, E, H) Phase plane representation of the time series in panels A, D, and G alongside the corresponding susceptible host dynamics. Red and blue solid lines represent epidemic trajectories on an SI phase plane before and after perturbations are introduced, respectively. (C, F, I) Changes in distance from the attractor over time on a log scale. Blue lines represent the distance from the attractor. Orange lines represent the locally estimated scatterplot smoothing (LOESS) fits to the logged distance from the attractor. Dotted lines show the intrinsic resilience of the seasonally unforced system.

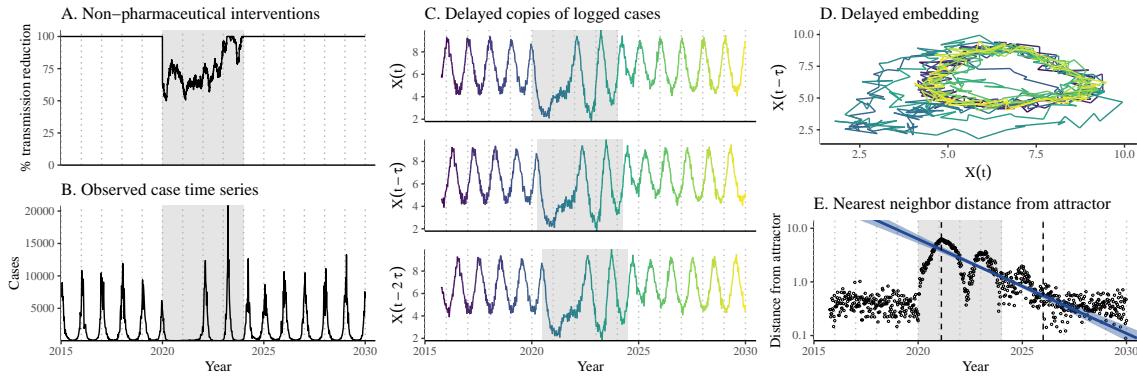


Figure 3: A schematic diagram explaining the inference of pathogen resilience from synthetic data. (A) A realistic example of simulated pandemic perturbations, represented by a relative reduction in transmission. (B) The impact of the synthetic pandemic perturbation on epidemic dynamics simulated using a SIRS model with demographic stochasticity. (C) Generating delayed copies of the logged time series allows us to obtain an embedding. (D) Two dimensional representation of an embedding. (E) Delayed embedding allows us to calculate the nearest neighbor distance from the empirical attractor, which is determined based on the pre-pandemic time series. Blue lines and dashed regions represent the linear regression fit and the associated 95% confidence interval. This distance time series can be used to infer pathogen resilience after choosing an appropriate window for linear regression. For illustration purposes, an arbitrary fitting window was chosen.

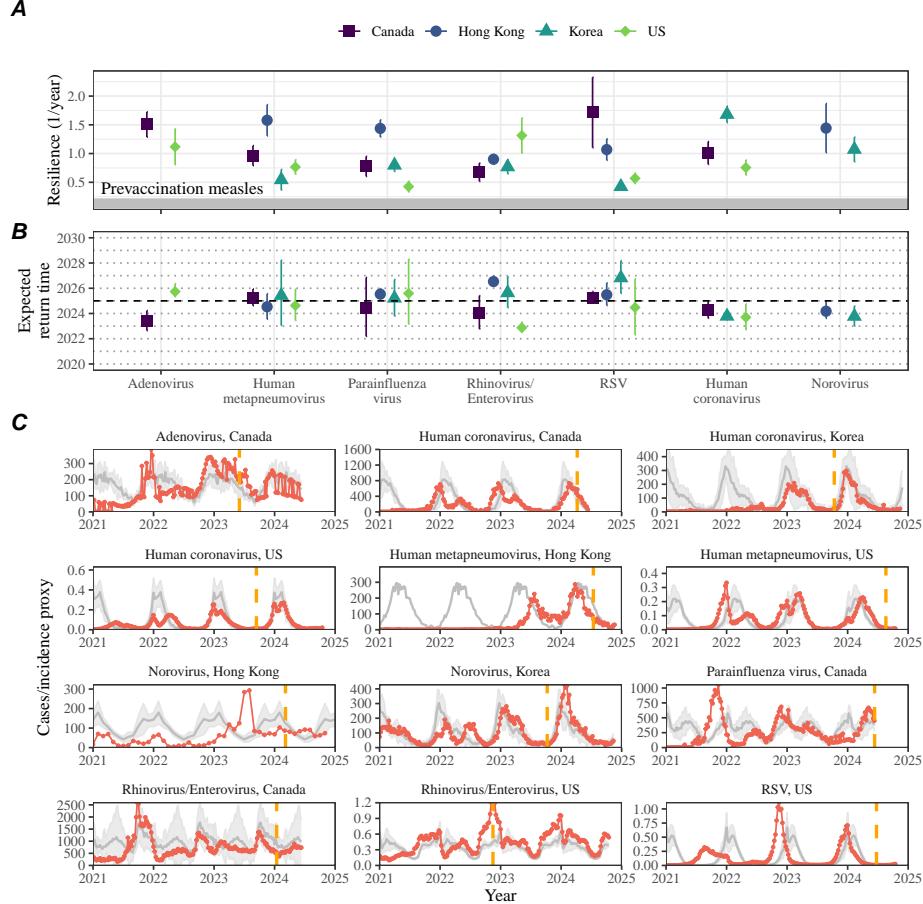


Figure 4: Summary of resilience estimates and predictions for return time. (A) Estimated pathogen resilience. The gray horizontal line represents the intrinsic resilience of pre-vaccination measles dynamics. (B) Predicted timing of when each pathogen will return to their pre-pandemic cycles. The dashed line in panel B indicates the end of 2024. Error bars represent 95% confidence intervals. (C) Observed dynamics for pathogen that are predicted to have returned before the end of 2024. Red points and lines represent data before 2020. Gray lines and shaded regions represent the mean seasonal patterns and corresponding 95% confidence intervals around the mean, previously shown in Figure 1. Orange vertical lines represent the predicted timing of return.

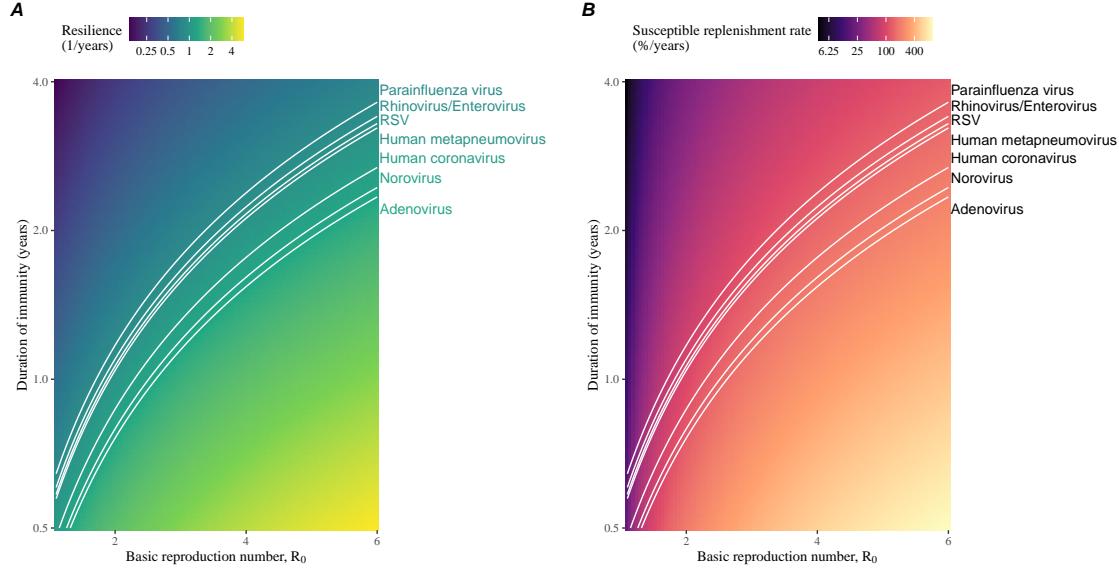


Figure 5: Linking pathogen resilience to epidemiological parameters and susceptible host dynamics. (A) Intrinsic resilience as a function of the basic reproduction number \mathcal{R}_0 and the duration of immunity. (B) Per-capita susceptible replenishment rate as a function of the basic reproduction number \mathcal{R}_0 and the duration of immunity. The standard SIRS model without seasonal forcing is used to compute intrinsic resilience and the per-capita susceptible replenishment rate. Lines correspond to a set of parameters that are consistent with mean resilience estimates for each pathogen, averaged across different countries.

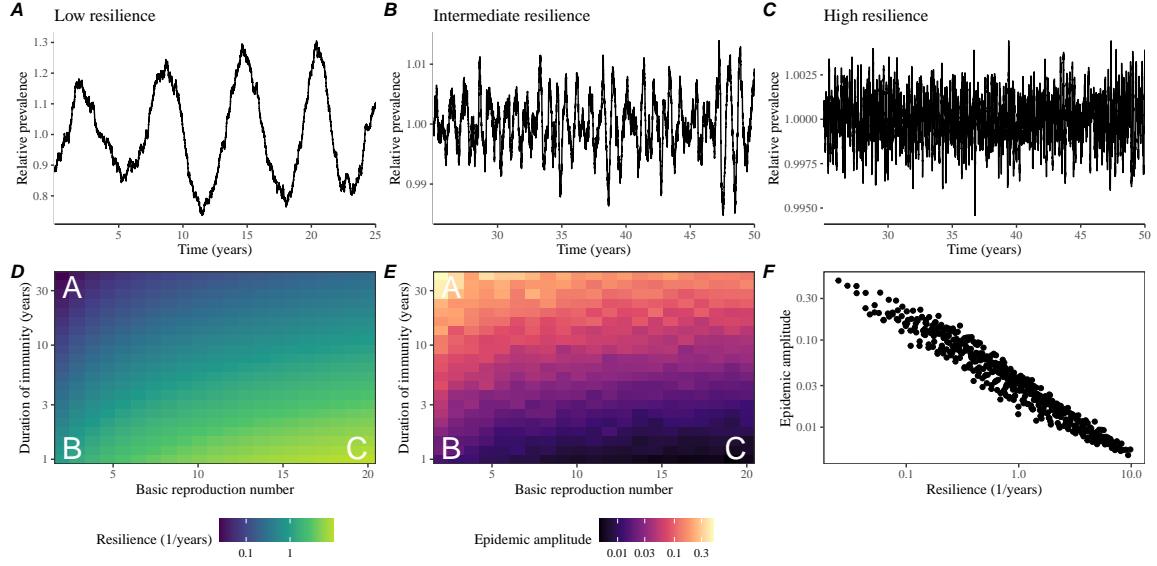


Figure 6: Linking resilience of a host-pathogen system to its sensitivity to stochastic perturbations. (A–C) Epidemic trajectories of a stochastic SIRS model across three different resilience values: low, intermediate, and high. The relative prevalence was calculated by dividing infection prevalence by its mean value. (D) Intrinsic resilience of a system as a function of the basic reproduction number \mathcal{R}_0 and the duration of immunity. (E) Epidemic amplitude as a function of the basic reproduction number \mathcal{R}_0 and the duration of immunity. The epidemic amplitude corresponds to $(\max I - \min I)/(2\bar{I})$, where \bar{I} represents the mean prevalence. Labels A–C in panels D and E correspond to scenarios shown in panels A–C. (F) The relationship between pathogen resilience and epidemic amplitude.

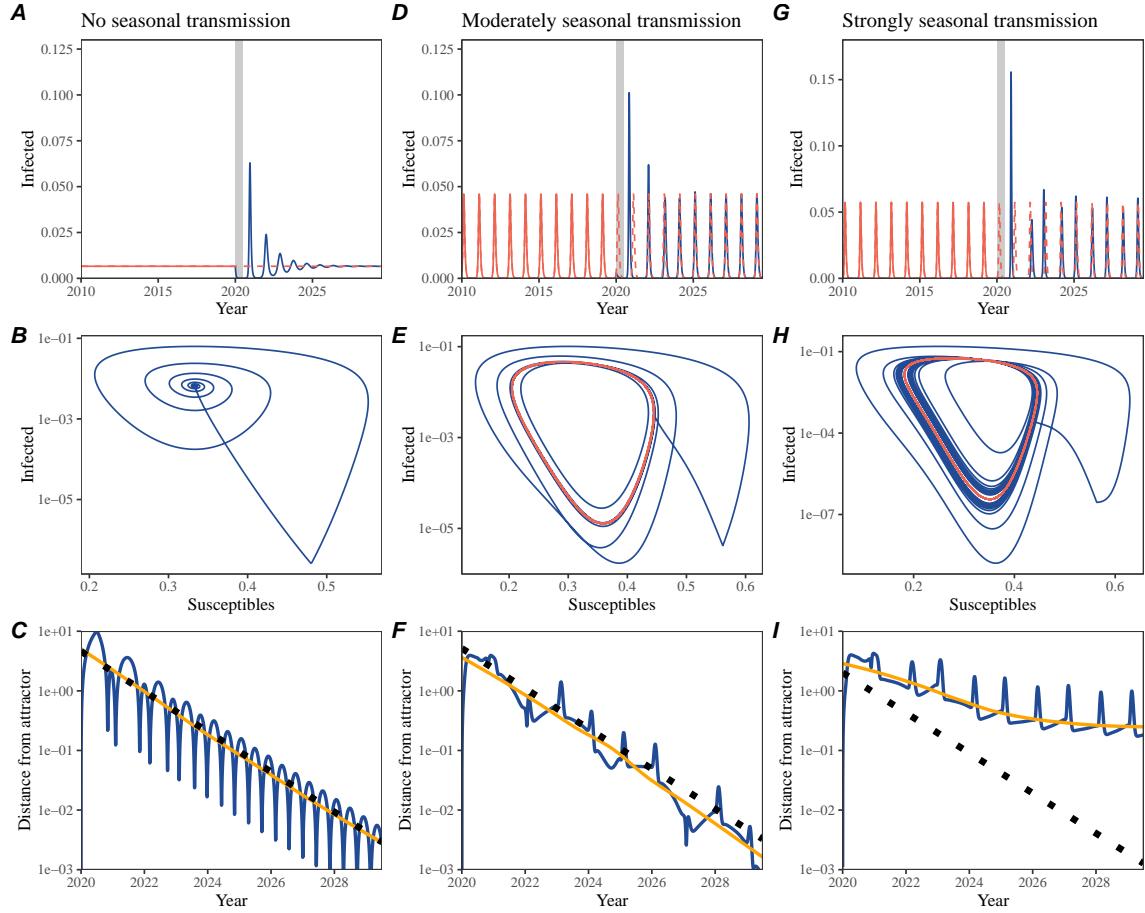


Figure S1: Impact of seasonal transmission on pathogen resilience. (A, D, G) Simulated epidemic trajectories using the SIRS model without seasonal forcing (A), with seasonal forcing of amplitude of 0.2 (D), and with seasonal forcing of amplitude of 0.4 (G). Red and blue solid lines represent epidemic dynamics before and after interventions are introduced, respectively. Red dashed lines represent counterfactual epidemic dynamics in the absence of interventions. Gray regions indicate the duration of interventions. (B, E, H) Phase plane representation of the time series in panels A, D, and G alongside the corresponding susceptible host dynamics. Red and blue solid lines represent epidemic trajectories on an SI phase plane before and after interventions are introduced, respectively. (C, F, I) Changes in logged distance from the attractor over time. Blue lines represent the logged distance from the attractor. Orange lines represent the locally estimated scatterplot smoothing (LOESS) fits to the logged distance from the attractor. Dotted lines show the intrinsic resilience of the seasonally unforced system.

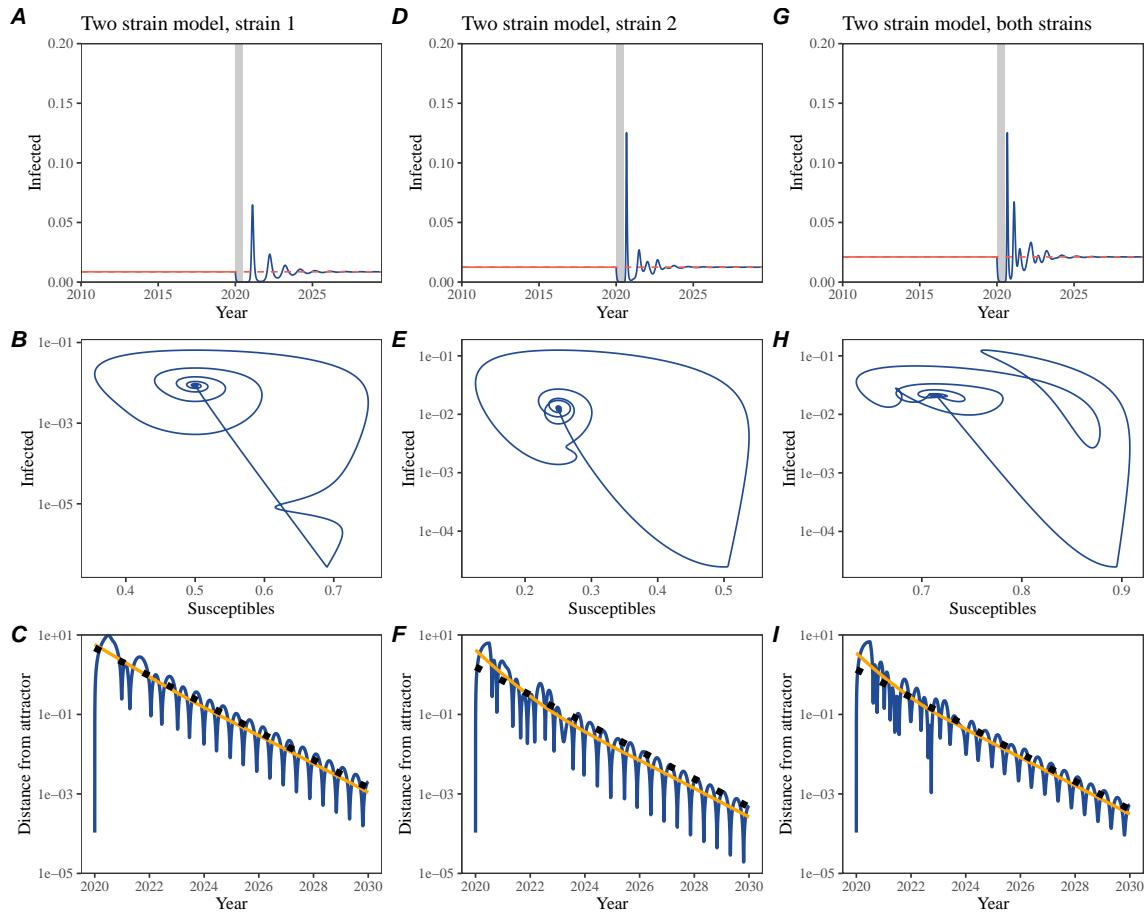


Figure S2: A simple method to measure pathogen resilience following pandemic perturbations for a two-strain competition system without seasonal forcing. (A, D, G) Simulated epidemic trajectories using a two-strain system without seasonal forcing. In this model, two strains compete through cross immunity. Red and blue solid lines represent epidemic dynamics before and after interventions are introduced, respectively. Red dashed lines represent counterfactual epidemic dynamics in the absence of interventions. Gray regions indicate the duration of interventions. (B, E, H) Phase plane representation of the time series in panels A, D, and G alongside the corresponding susceptible host dynamics. Blue solid lines represent epidemic trajectories on an SI phase plane before and after interventions are introduced, respectively. Blue dashed lines represent the corresponding susceptible host dynamics. (C, F, I) Changes in logged distance from the attractor over time. Blue lines represent the logged distance from the attractor. Orange lines represent the locally estimated scatterplot smoothing (LOESS) fits to the logged distance from the attractor. Dotted lines show the intrinsic resilience of the seasonally unforced system.

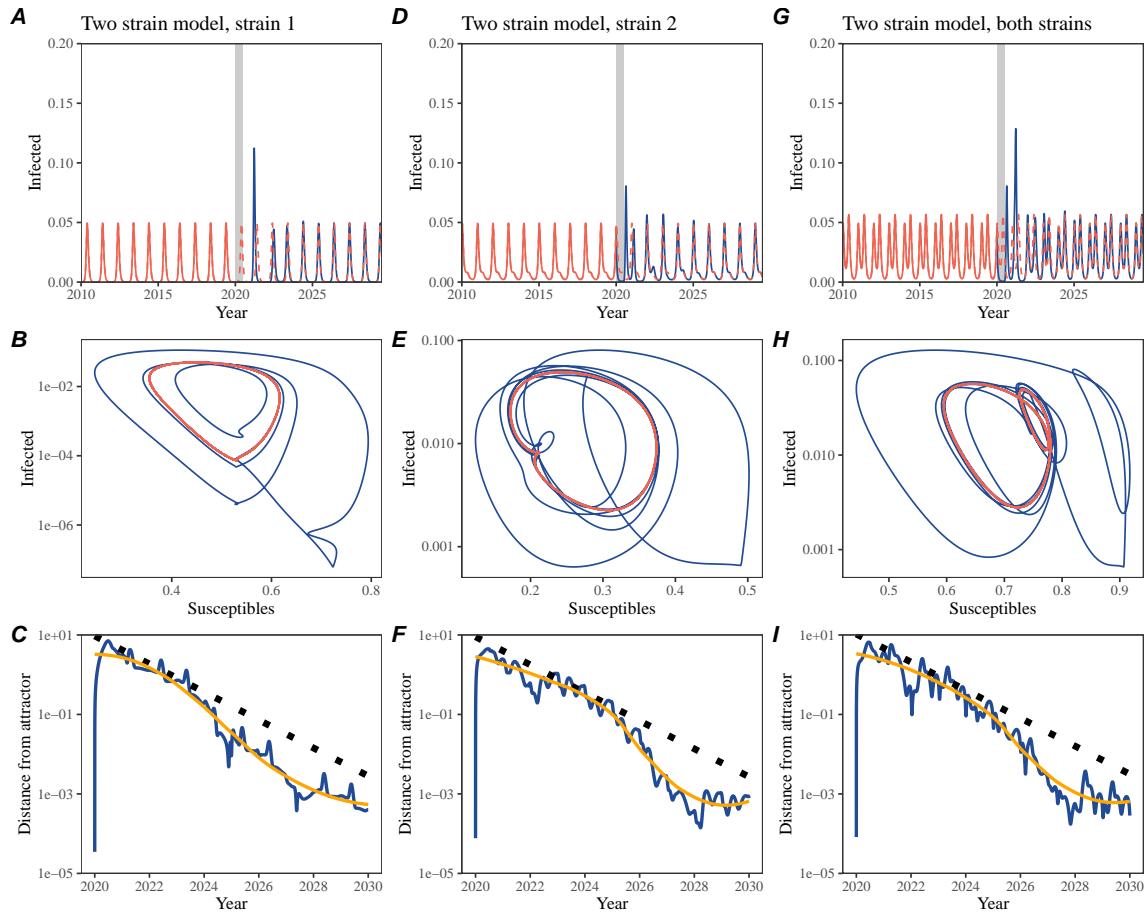


Figure S3: A simple method to measure pathogen resilience following pandemic perturbations for a two-strain competition system with seasonal forcing. (A, D, G) Simulated epidemic trajectories using a two-strain system with seasonal forcing (amplitude of 0.2). In this model, two strains compete through cross immunity. Red and blue solid lines represent epidemic dynamics before and after interventions are introduced, respectively. Red dashed lines represent counterfactual epidemic dynamics in the absence of interventions. Gray regions indicate the duration of interventions. (B, E, H) Phase plane representation of the time series in panels A, D, and G alongside the corresponding susceptible host dynamics. Red and blue solid lines represent epidemic trajectories on an SI phase plane before and after interventions are introduced, respectively. (C, F, I) Changes in logged distance over time. Blue lines represent the logged distance from the attractor. Orange lines represent the locally estimated scatterplot smoothing (LOESS) fits to the logged distance from the attractor. Dotted lines show the intrinsic resilience of the seasonally unforced system.

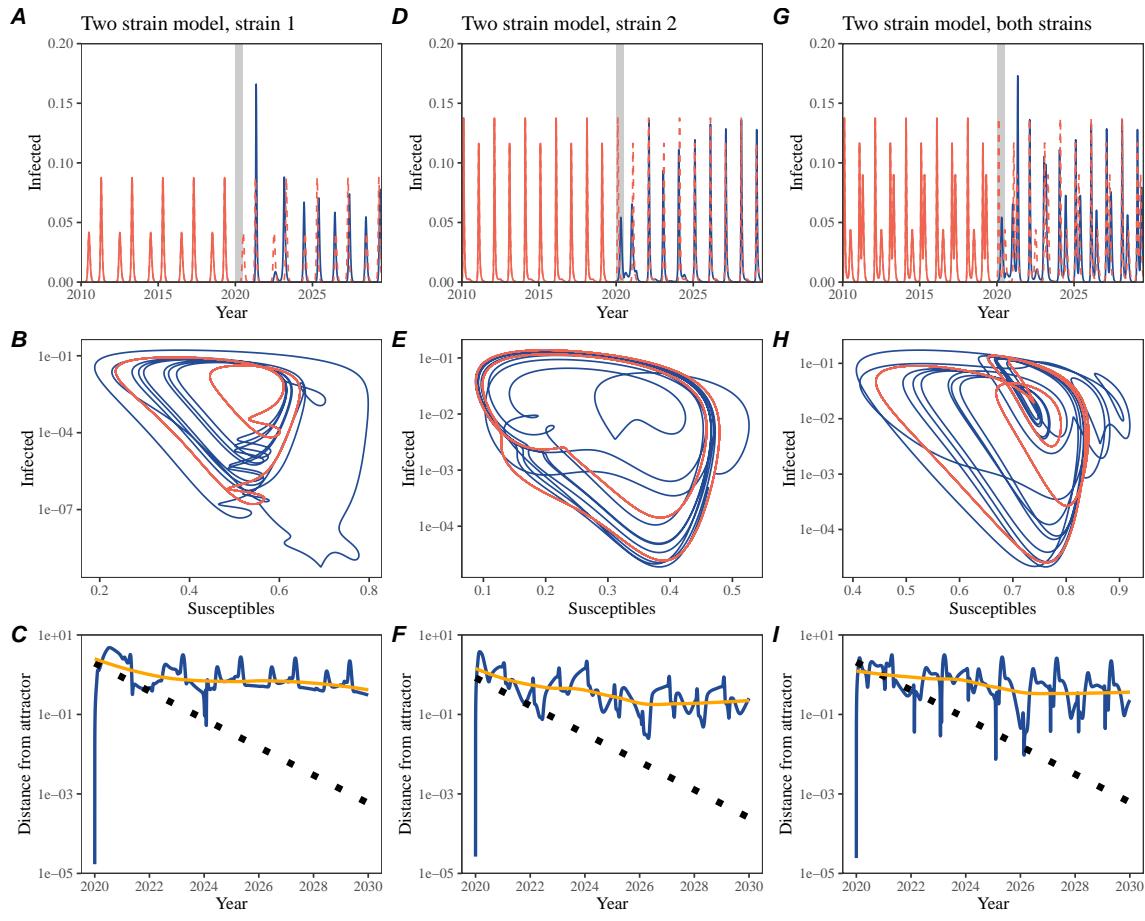


Figure S4: A simple method to measure pathogen resilience following pandemic perturbations for a multi-strain competition system with strong seasonal forcing. (A, D, G) Simulated epidemic trajectories using a two-strain system with seasonal forcing (amplitude of 0.2). In this model, two strains compete through cross immunity. Red and blue solid lines represent epidemic dynamics before and after interventions are introduced, respectively. Red dashed lines represent counterfactual epidemic dynamics in the absence of interventions. Gray regions indicate the duration of interventions. (B, E, H) Phase plane representation of the time series in panels A, D, and G alongside the corresponding susceptible host dynamics. Red and blue solid lines represent epidemic trajectories on an SI phase plane before and after interventions are introduced, respectively. (C, F, I) Changes in logged distance over time. Blue lines represent the logged distance from the attractor. Orange lines represent the locally estimated scatterplot smoothing (LOESS) fits to the logged distance from the attractor. Dotted lines show the intrinsic resilience of the seasonally unforced system.

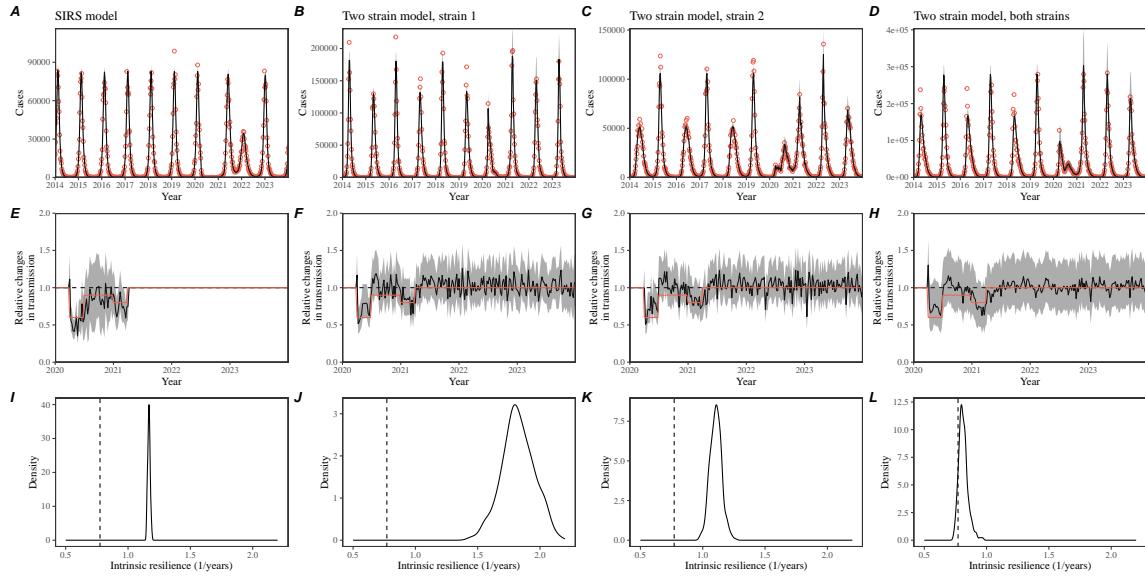


Figure S5: Mechanistic model fits to simulated data and inferred intrinsic resilience. We simulated discrete time, stochastic epidemic trajectories using a seasonally forced SIRS model (A,E,I) and a seasonally forced two-strain model (B–D, F–H, and J–L) and tested whether we can estimate the intrinsic resilience of the seasonally unforced versions of the model by fitting a mechanistic model (Supplementary Text). We fitted the same discrete time, one strain, deterministic SIRS model in each scenario. (A–D) Simulated case time series from corresponding models shown in the title (red) and SIRS model fits (black). (E–H) Assumed changes in transmission due to pandemic perturbations (red) and estimated time-varying relative transmission rates for the perturbation impact from the SIRS model fits (black). Solid lines and shaded regions represent fitted posterior median and 95% credible intervals. (I–L) Comparisons between the true and estimated intrinsic resilience of the seasonally unforced system. Vertical lines represent the true intrinsic resilience of the seasonally unforced system. Density plots represent the posterior distribution of the inferred intrinsic resilience of the seasonally unforced SIRS model using fitted parameters.

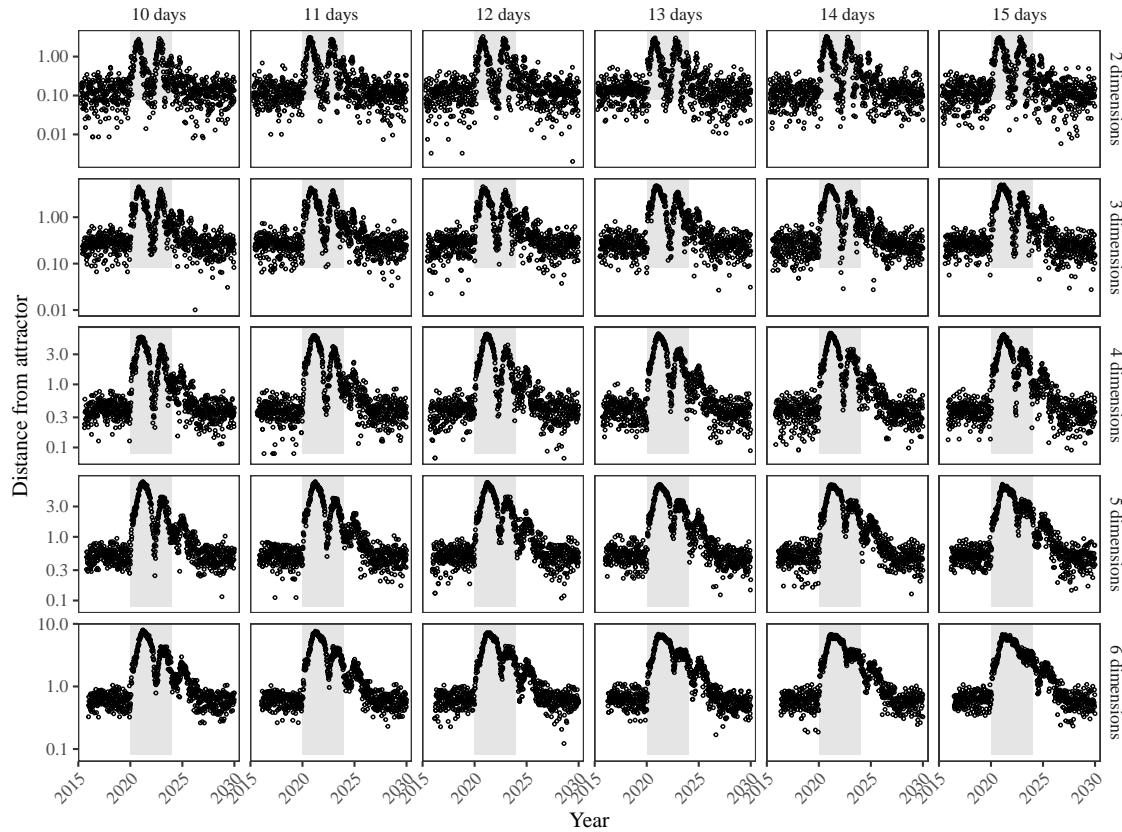


Figure S6: Sensitivity of the distance from the attractor to choice of embedding lags and dimensions. Sensitivity analysis for the distance-from-attractor time series shown in Figure 3E in the main text by varying the embedding lag between 10–15 days and embedding dimensions between 2–6 dimensions. The gray region represents the assumed period of pandemic perturbation.

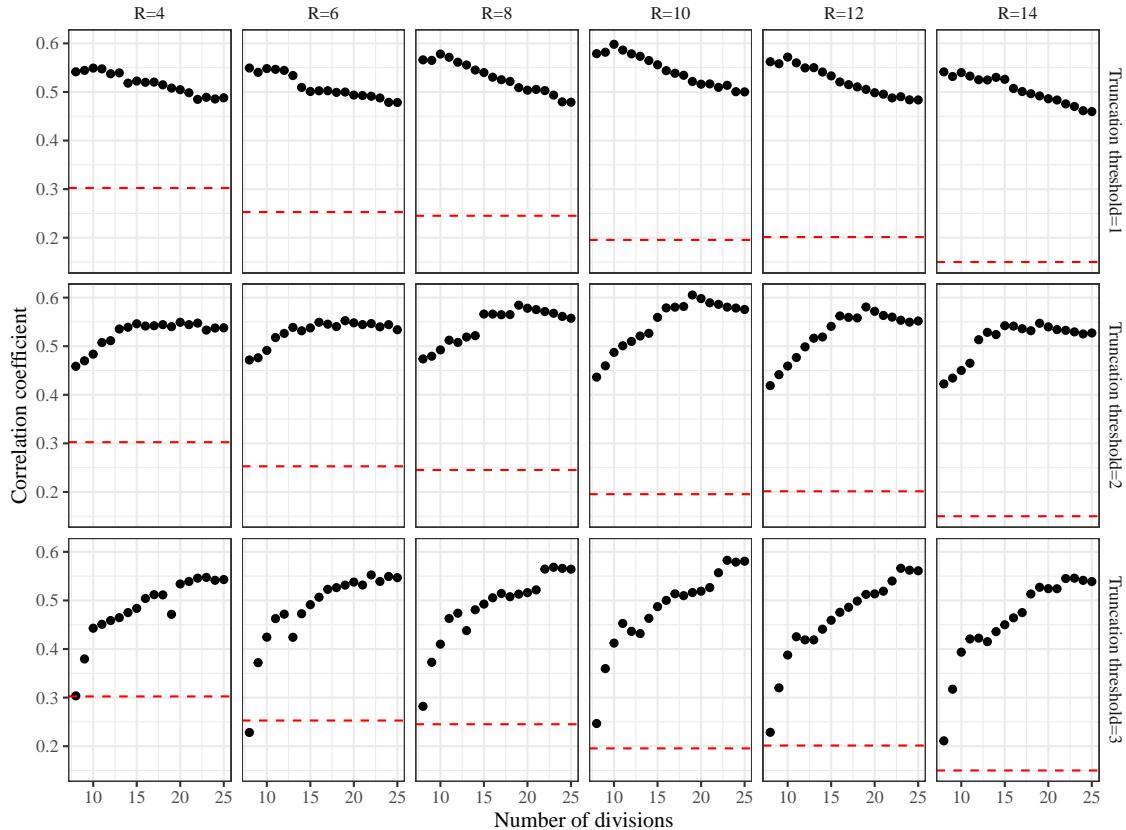


Figure S7: Impact of fitting window selection on the estimation of empirical resilience. We simulated 500 epidemics of a stochastic SIRS model with randomly drawn parameters and randomly generated pandemic perturbations (Supplementary Text). For each simulation, we varied the false nearest neighbor threshold R for reconstructing the empirical attractor as well as the parameters for regression window selection (i.e., the truncation threshold a and the number of divisions K). Each point represents the resulting correlation coefficient between empirical and intrinsic resilience estimates for a given scenario. Dashed lines represent the correlation coefficient between empirical and intrinsic resilience estimates for the naive approach that fits a linear regression on a log scale, starting from the maximum distance until the end of the time series.

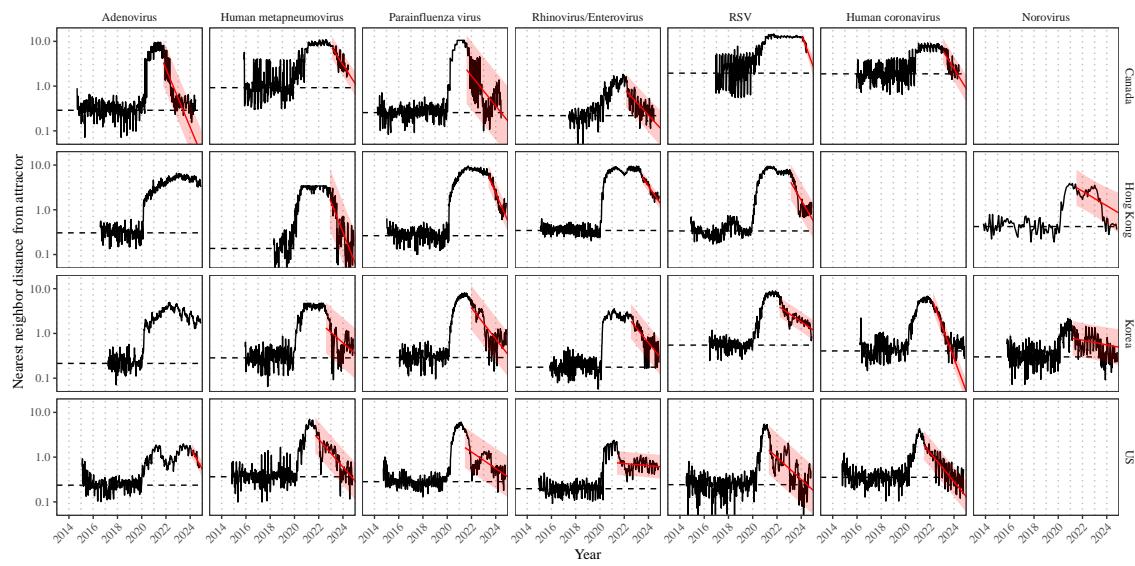


Figure S8: Estimated time series of distance from the attractor for each pathogen and corresponding linear regression fits using automated window selection criterion across Canada, Hong Kong, Korea, and the US. Black lines represent the estimated distance from the attractor. Red lines and shaded regions represent the linear regression fits and corresponding 95% confidence intervals. Dashed lines represent the average of the pre-pandemic nearest neighbor distances.

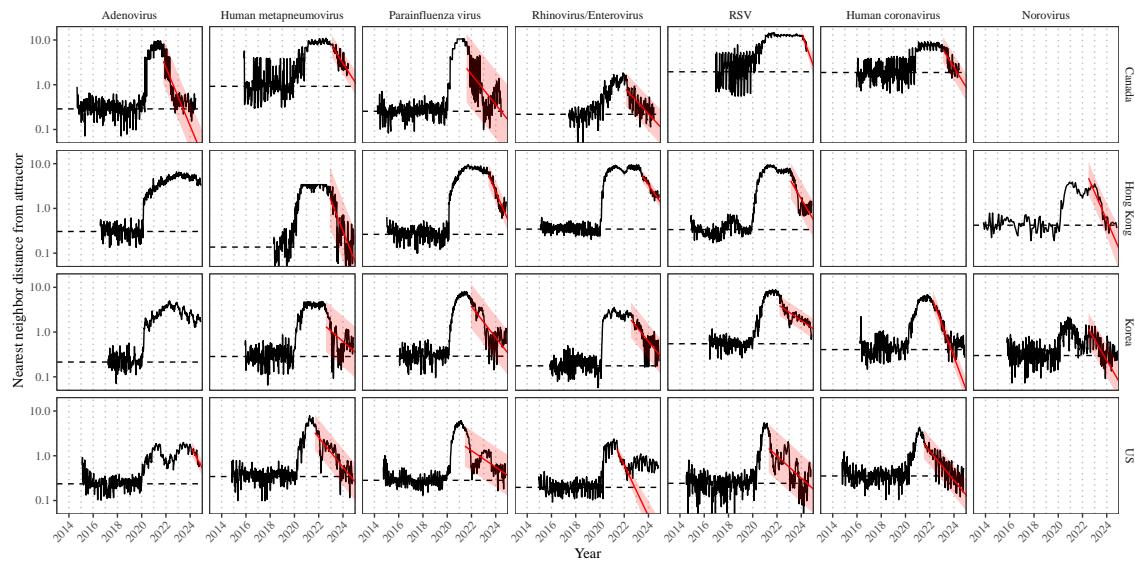


Figure S9: Estimated time series of distance from the attractor for each pathogen and corresponding linear regression fits across Canada, Hong Kong, Korea, and the US, including ad-hoc regression window selection. We used ad-hoc regression windows for norovirus in Hong Kong and Korea and rhinovirus/enterovirus in the US. Black lines represent the estimated distance from the attractor. Red lines and shaded regions represent the linear regression fits and corresponding 95% confidence intervals. Dashed lines represent the average of the pre-pandemic nearest neighbor distances.

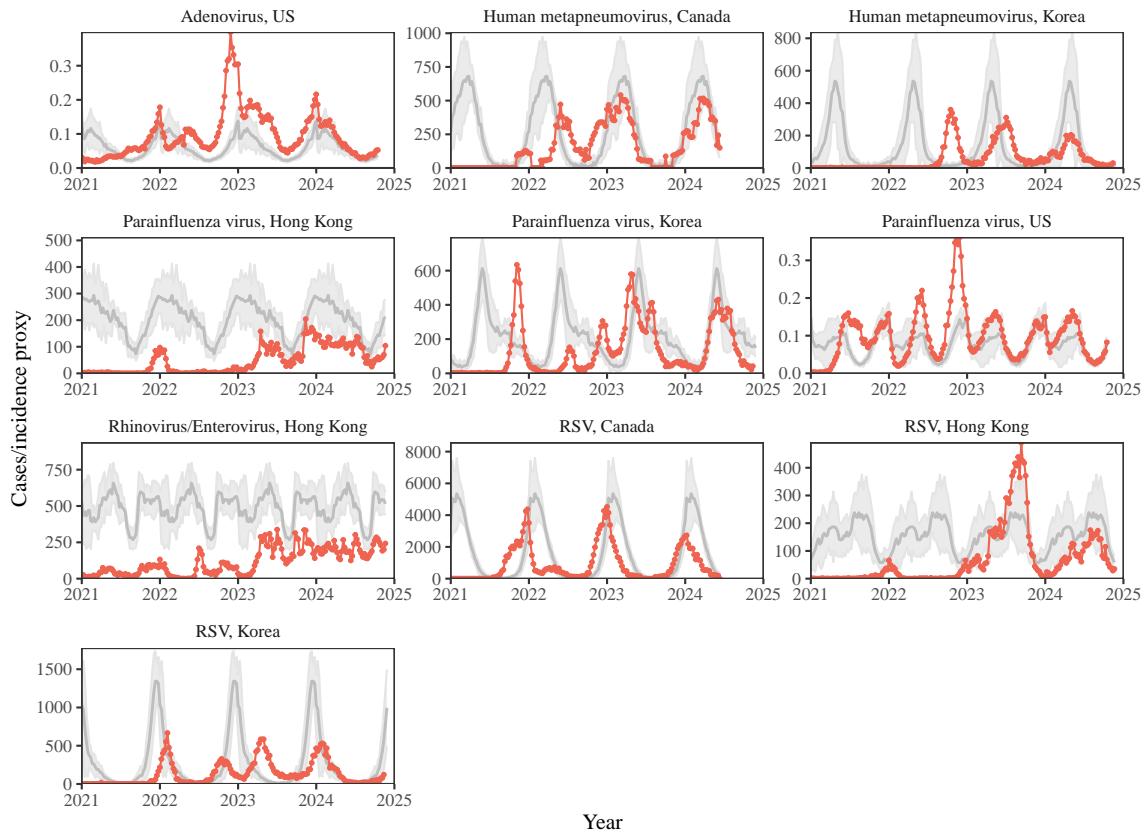


Figure S10: Observed dynamics for pathogen that are predicted to return after the end of 2024. Red points and lines represent data before 2020. Gray lines and shaded regions represent the mean seasonal patterns and corresponding 95% confidence intervals around the mean, previously shown in Figure 1.

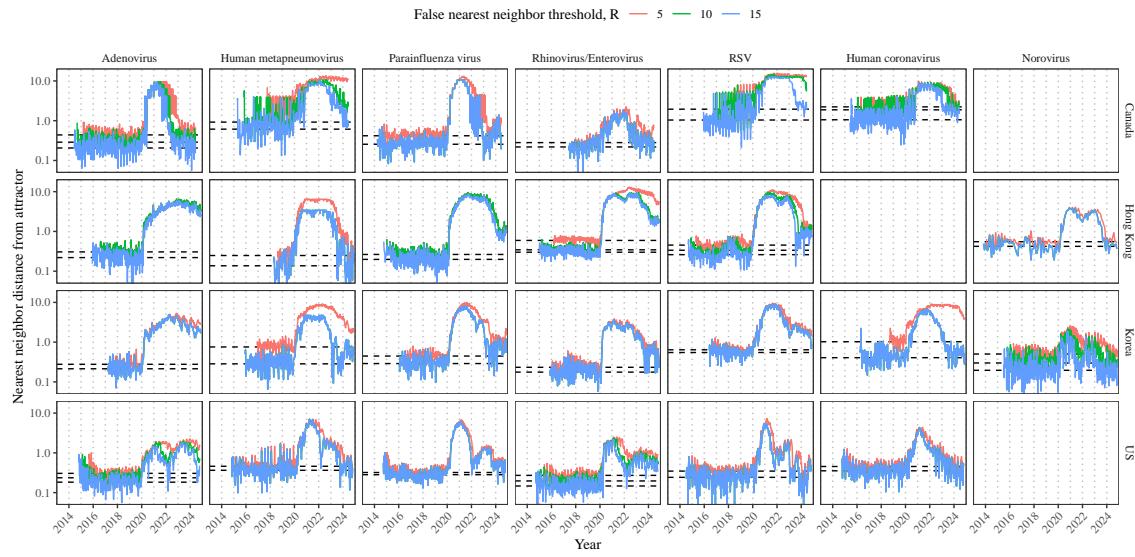


Figure S11: Estimated time series of distance from the attractor for each pathogen across different choices about false nearest neighbor threshold values. Using higher threshold values for the false nearest neighbor approach gives lower embedding dimensions. Colored lines represent the estimated distance from the attractor for different threshold values.

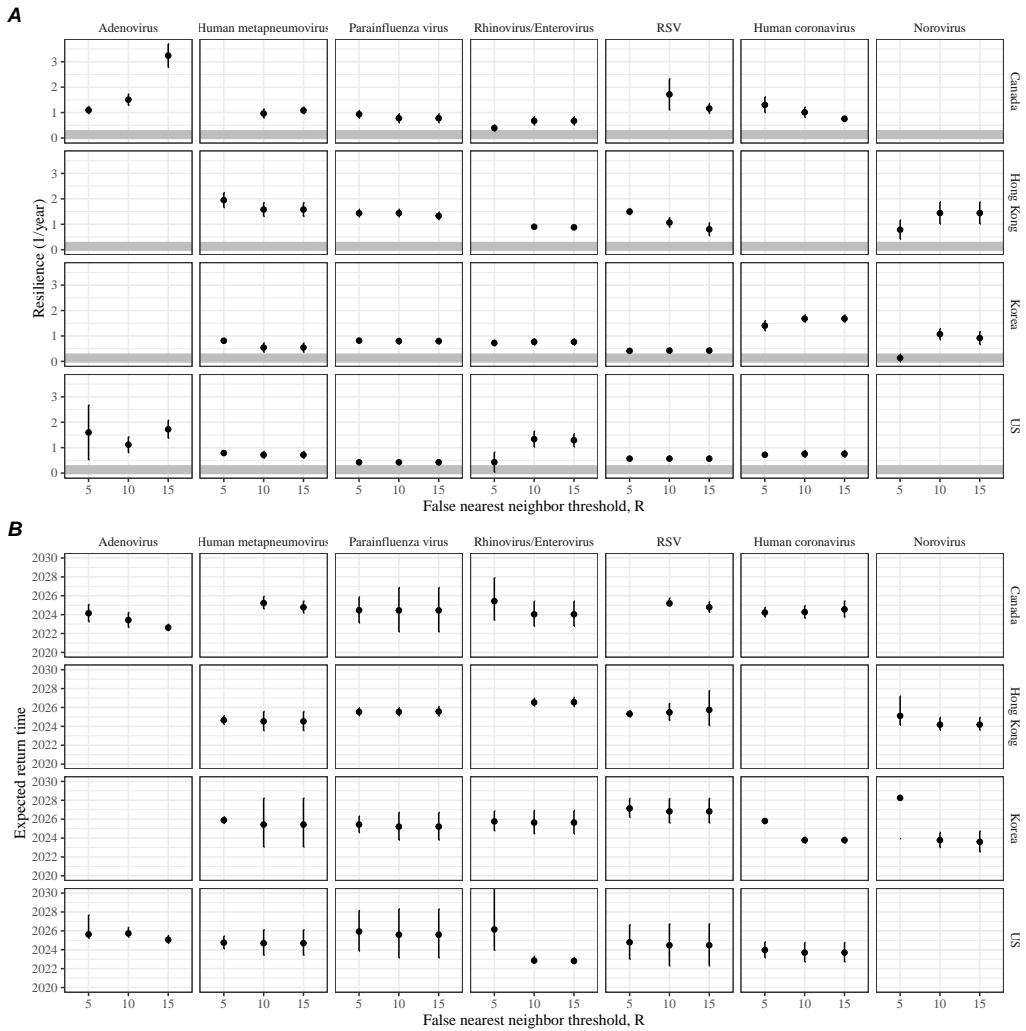


Figure S12: Summary of resilience estimates and predictions for return time across different choices about false nearest neighbor threshold values. The automated window selection method was used for all scenarios to estimate pathogen resilience, except for norovirus in Hong Kong and Korea and rhinovirus/enterovirus in the US. We used the same ad-hoc regression windows for norovirus in Hong Kong and Korea and rhinovirus/enterovirus in the US as we did in the main analysis. (A) Estimated pathogen resilience. The gray horizontal line represents the intrinsic resilience of pre-vaccination measles dynamics. (B) Predicted timing of when each pathogen will return to their pre-pandemic cycles. The dashed line in panel B indicates the end of 2024. Error bars represent 95% confidence intervals.

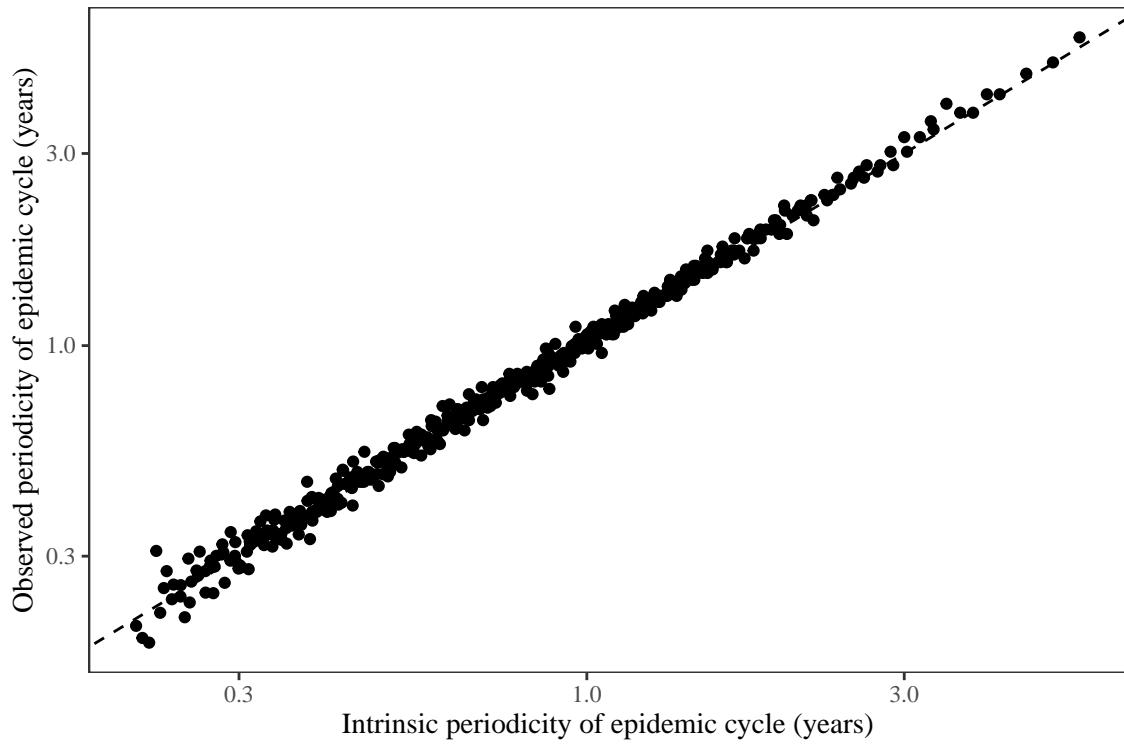


Figure S13: **Comparison between the observed and intrinsic periodicity of the epidemic cycle of the seasonally unforced SIRS model.** The observed periodicity of the epidemic corresponds to the periodicity at which maximum spectral density occurs. The intrinsic periodicity of the epidemic corresponds to $2\pi/\text{Im}(\lambda)$, where $\text{Im}(\lambda)$ is the imaginary part of the eigenvalue.

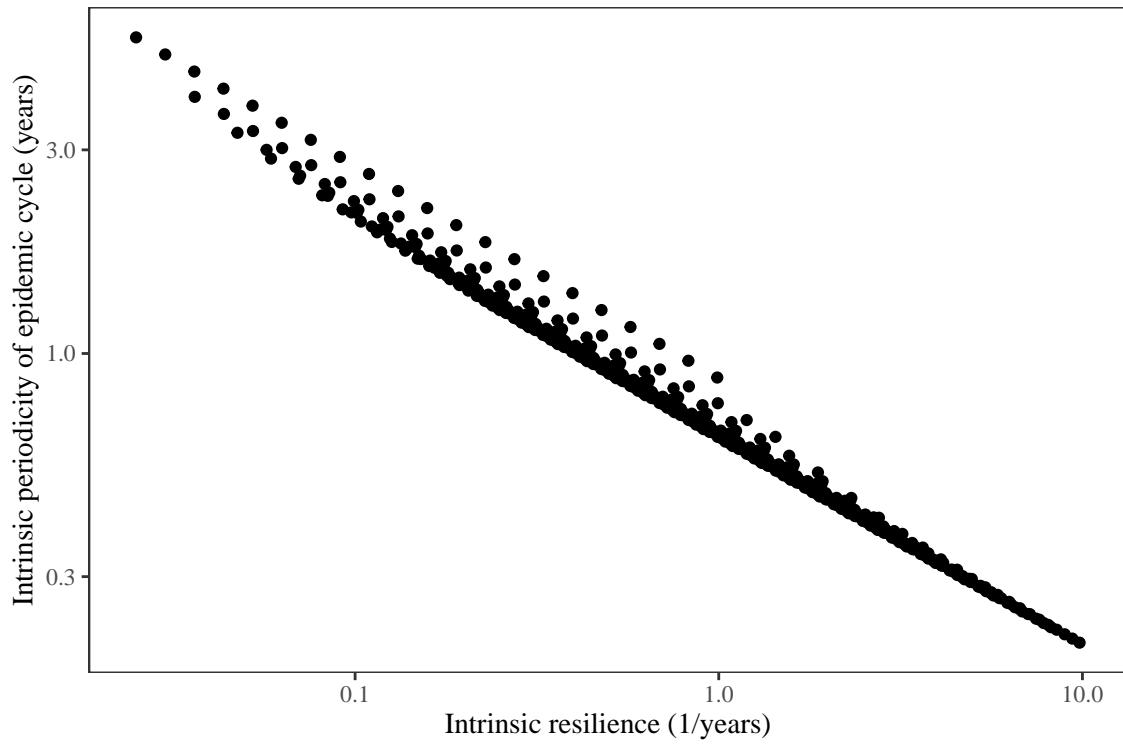


Figure S14: Comparison between the intrinsic resilience and periodicity of the epidemic cycle of the seasonally unforced SIRS model. The intrinsic resilience of the epidemic corresponds to the negative of the real value of the eigenvalue $-\text{Re}(\lambda)$. The intrinsic periodicity of the epidemic corresponds to $2\pi/\text{Im}(\lambda)$, where $\text{Im}(\lambda)$ is the imaginary part of the eigenvalue.

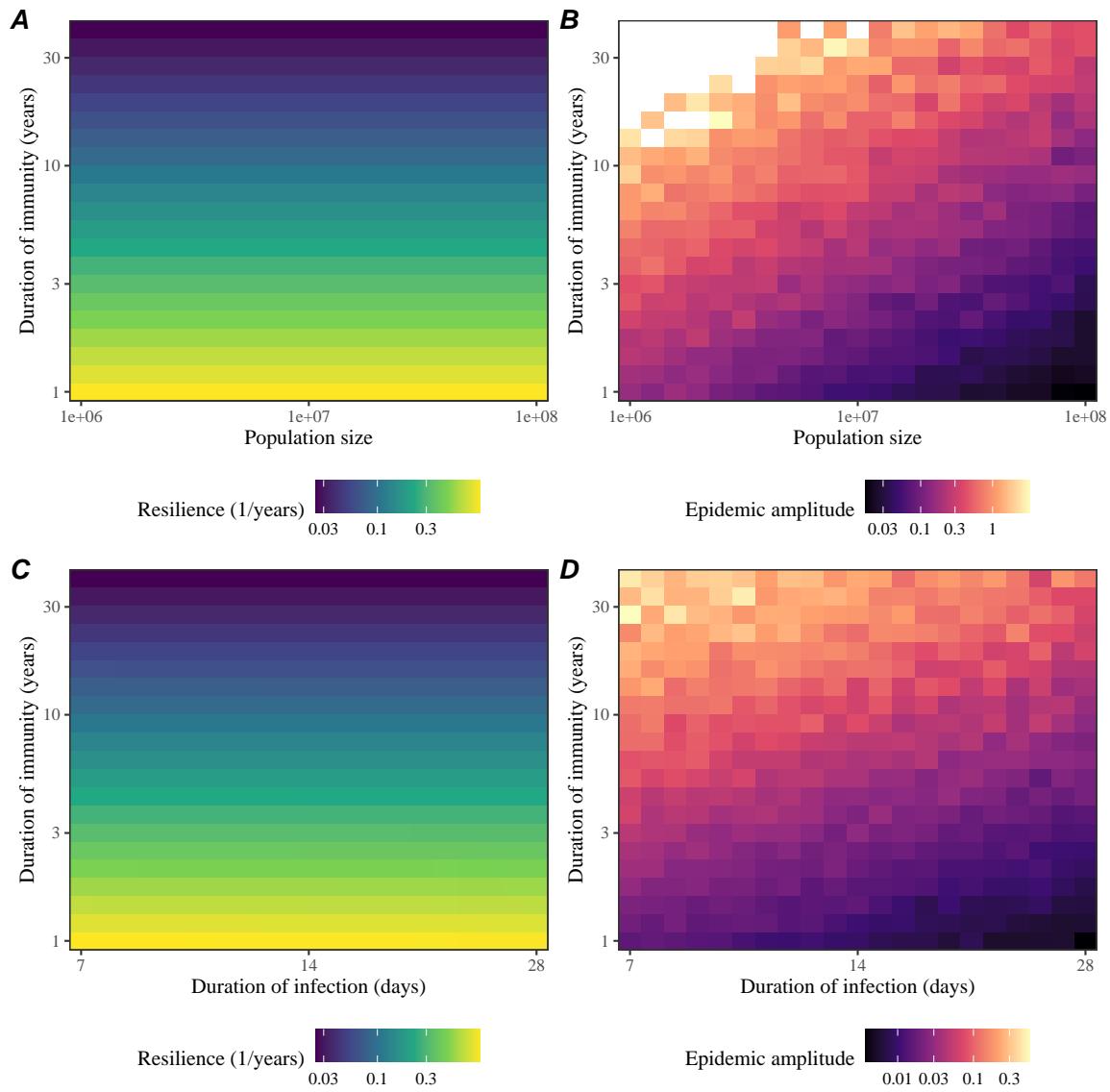


Figure S15: Impact of population size and the average duration of infection of a host-pathogen system on its sensitivity to stochastic perturbations. (A) Intrinsic resilience of a system as a function of population size and the average duration of immunity. (B) Epidemic amplitude as a function of population size and the average duration of immunity. (C) Intrinsic resilience of a system as a function of the average duration of infection and immunity. (D) Epidemic amplitude as a function of the average duration of infection and immunity. The epidemic amplitude corresponds to $(\max I - \min I)/(2\bar{I})$, where \bar{I} represents the mean prevalence.

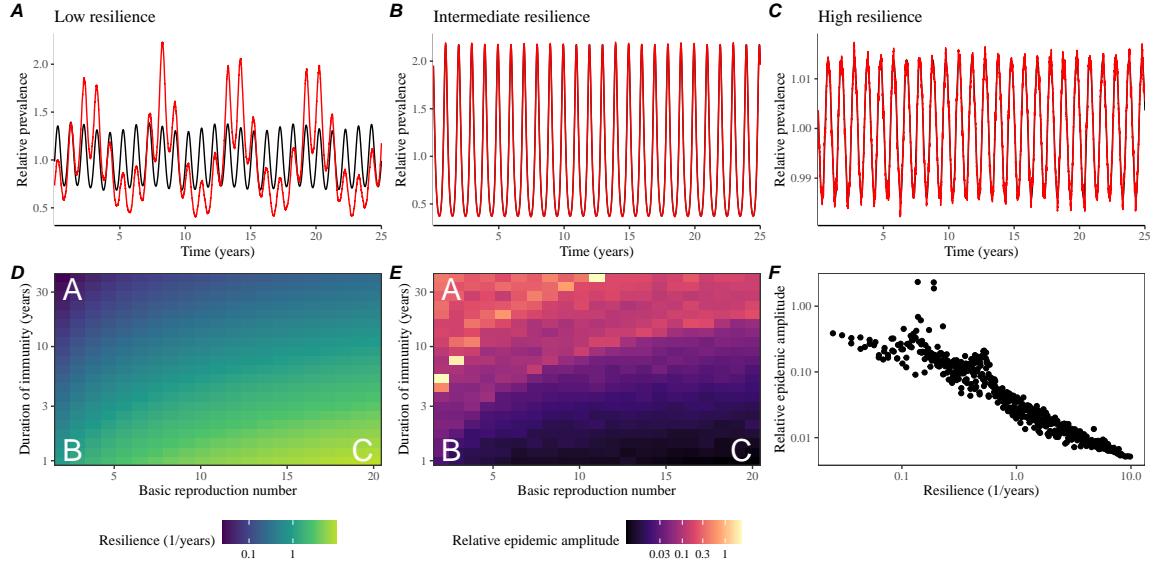


Figure S16: Linking resilience of a host-pathogen system to its sensitivity to stochastic perturbations for the seasonally forced SIRS model. (A–C) Epidemic trajectories of a stochastic SIRS model with demographic stochasticity across three different resilience values: low, intermediate, and high. The relative prevalence was calculated by dividing infection prevalence by its mean value. Black lines represent deterministic epidemic trajectories. Red lines represent stochastic epidemic trajectories. (D) The intrinsic resilience of the seasonally unforced system as a function of the basic reproduction number \mathcal{R}_0 and the duration of immunity. (E) The relative epidemic amplitude, a measure of sensitivity to stochastic perturbations, as a function of the basic reproduction number \mathcal{R}_0 and the duration of immunity. To calculate the relative epidemic amplitude, we first take the relative difference in infection prevalence between stochastic and deterministic trajectories: $\epsilon = (I_{\text{stoch}} - I_{\text{det}})/I_{\text{det}}$. Then, we calculate the difference between maximum and minimum of the relative difference and divide by half: $(\max \epsilon - \min \epsilon)/2$. Labels A–C in panels D and E correspond to scenarios shown in panels A–C. (F) The relationship between pathogen resilience and relative epidemic amplitude.

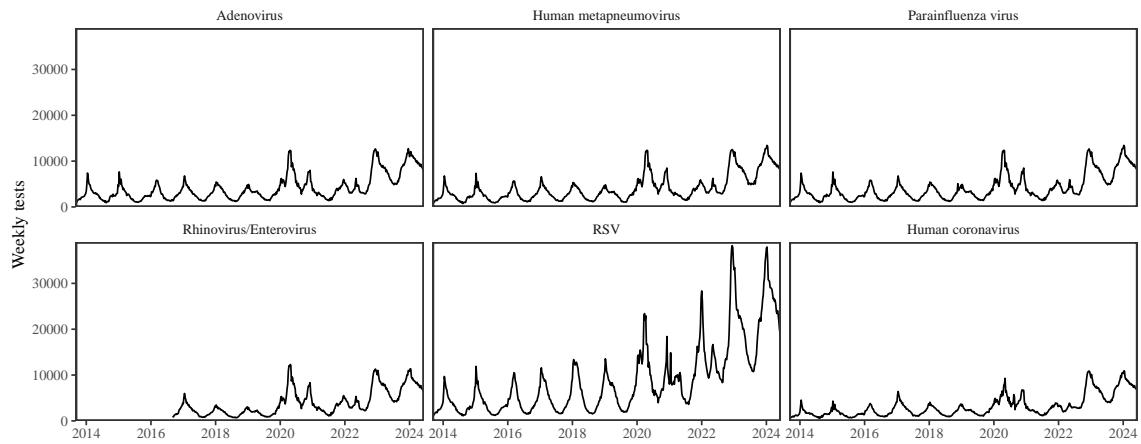


Figure S17: Testing patterns for respiratory pathogens in Canada.

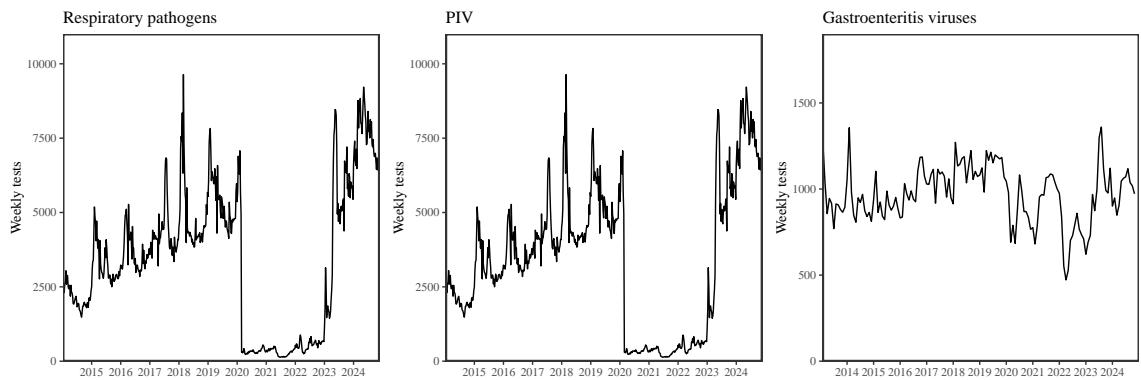


Figure S18: Testing patterns for respiratory pathogens, PIV, and gastroenteritis viruses in Hong Kong.

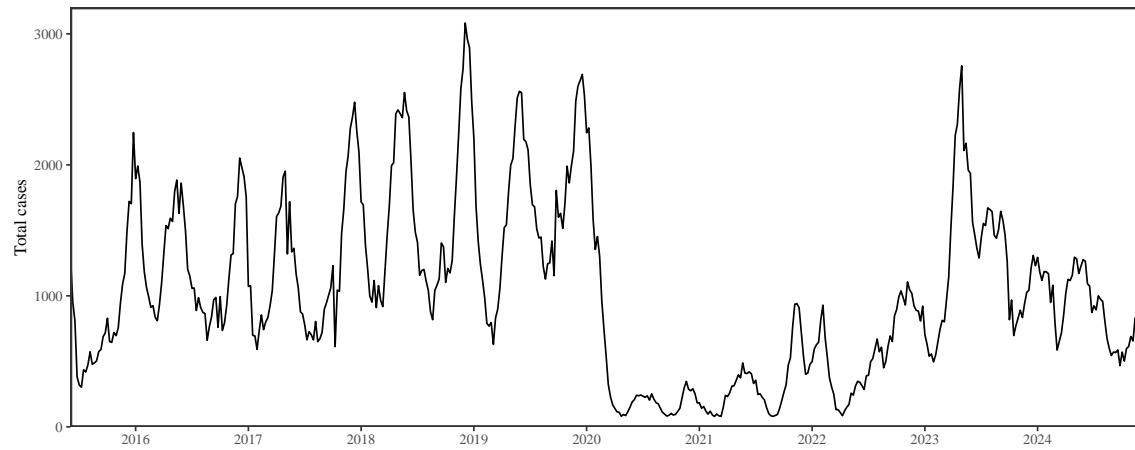


Figure S19: Total number of reported respiratory infection cases in Korea.

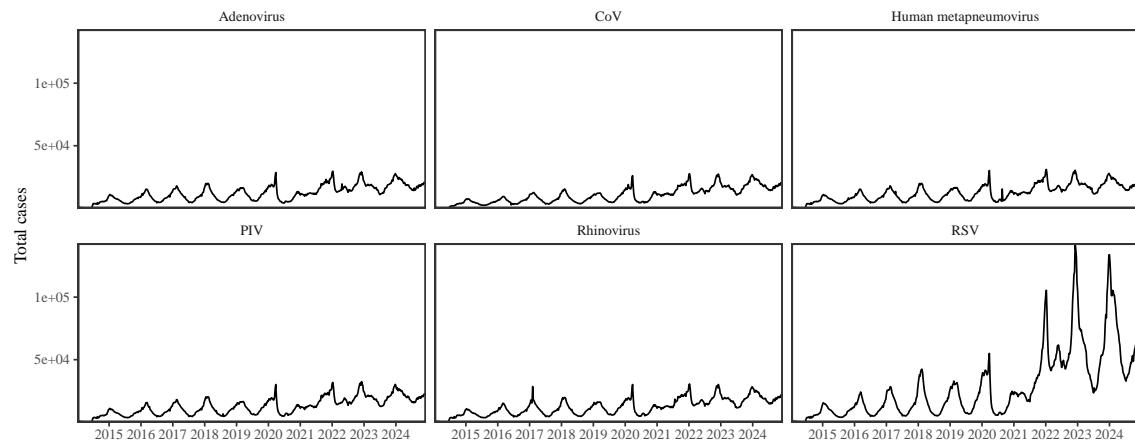


Figure S20: Testing patterns for respiratory pathogens in the US reported through the NREVSS.