

1 Susceptible host dynamics explain pathogen resilience  
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4

## 5 **Abstract**

6 Major priority for epidemiological research in the time of anthropogenic change is  
7 understanding how infectious disease dynamics respond to perturbations. Interven-  
8 tions to slow the spread of COVID-19 significantly disrupted the transmission of other  
9 human pathogens, providing unique opportunities to learn about pathogen charac-  
10 teristics from spatiotemporal variation in re-emergence patterns. As interventions  
11 lifted, a key question of whether and when respiratory pathogens would eventually  
12 return to their pre-pandemic dynamics remains to be answered. To address this  
13 gap, we develop a framework for estimating pathogen resilience based on how fast  
14 epidemic patterns return to their pre-pandemic, endemic cycles. Our analysis re-  
15 veals a possibility that some pathogens may have settled to endemic cycles that are  
16 different from their pre-pandemic patterns. Finally, we show that heterogeneity in  
17 pathogen resilience can be understood in terms of how fast a susceptible host popula-  
18 tion becomes replenished. Our framework offers a novel perspective to characterizing  
19 epidemic dynamics of endemic pathogens and measuring epidemic time scales.

Understanding how ecological systems respond to perturbations is a fundamental challenge in predicting species persistence and extinction [1, 2, 3]. These responses can be characterized in terms of resilience, which often measures how fast a system returns to its stable, reference state following a perturbation [4, 5, 6, 7]. Both theoretical and empirical efforts to quantify resilience of ecological systems have provided key insights for understanding the dynamics of complex systems and linking these findings to actionable strategies for species conservation [8]. However, despite rich literature on ecological resilience, there have been limited applications to measuring the resilience of host-pathogen systems, especially for human pathogens.

Non-pharmaceutical interventions (NPIs) to slow the spread of COVID-19 disrupted the transmission of other human pathogens, providing large-scale natural experiments for understanding how various host-pathogen systems respond to perturbations [9, 10, 11, 12]. In particular, as interventions lifted, large heterogeneities in outbreak dynamics were observed across different pathogens in different countries (Figure 1), likely reflecting differences in NPI patterns, pathogen characteristics, immigration/importation from other countries, and pre-pandemic pathogen dynamics [13]. Even though more than four years have already passed since the emergence of COVID-19, current circulation patterns for many respiratory pathogens appear to be different from their pre-pandemic, seasonal patterns, especially in Hong Kong and Korea: some pathogens, such as human metapneumovirus and bocavirus in Korea, are circulating at lower levels, whereas other pathogens, such as RSV in Korea, seem to exhibit different seasonality (Figure 1). These observations pose two fundamental questions for current and future infectious disease dynamics: (1) can we learn about underlying pathogen characteristics, such as their transmissibility or duration of immunity, from re-emergence patterns? and (2) can we predict whether and when other respiratory pathogens will eventually return to their pre-pandemic dynamics?

To address this question, we propose a framework for characterizing the resilience of a host-pathogen system based on how fast the system recovers from perturbation. We begin by laying out a few example scenarios that represent the expected outbreak dynamics following COVID-19 interventions and illustrating how resilience can be measured by comparing the post- and pre-pandemic dynamics of susceptible and infected hosts. In practice, the dynamics of susceptible hosts are often unavailable, and traditional susceptible reconstruction approaches often require long-term endemic time series, which cannot be applied due to disruptions in epidemic patterns caused by COVID-19 NPIs. Instead, we utilize Takens' embedding theorem to reconstruct empirical attractors from data and further measure the distance from this empirical attractor, which allows us to characterize the rate at which this distance decreases. Analyses of pathogen surveillance data for a wide array of respiratory pathogens in Canada, Hong Kong, and Korea suggest ... Finally, we show that variation in resilience estimates can be understood in terms of susceptible host dynamics.

[SWP: *Revisit.*]

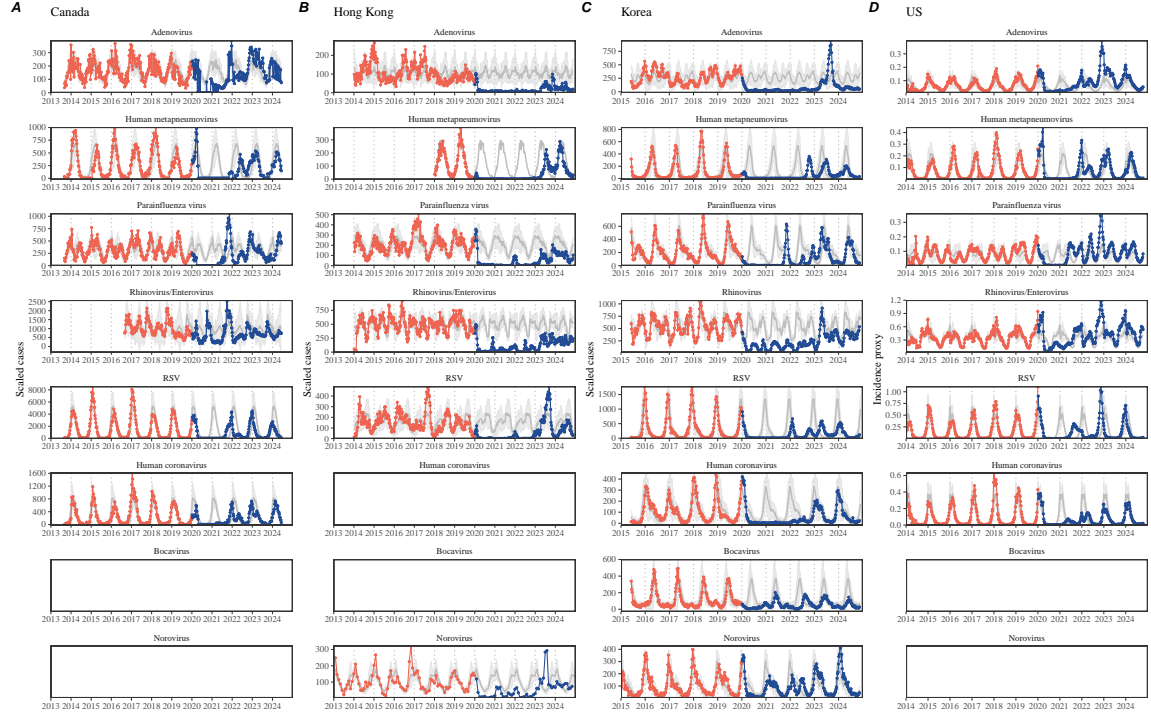


Figure 1: **Observed heterogeneity in responses to COVID-19 pandemic across respiratory pathogens and norovirus in (A) Canada, (B) Hong Kong, (C) Korea, and (D) US.** Red points and lines represent data before 2020. Blue points and lines represent data since 2020. Gray lines and shaded regions represent the mean seasonal patterns and corresponding 95% confidence intervals based on the observed outbreak patterns before 2020.

## Conceptual introduction to pathogen resilience

In classical ecological literature, resilience of an ecological system is measured by the rate at which the system returns to its reference state following a perturbation. This rate corresponds to the largest real part of the eigenvalues of the linearized system near equilibrium—here, we refer to this value as the *intrinsic* resilience of the system, which represents the expected rate of return from perturbed states. However, respiratory pathogens often exhibit seasonal variation in transmission, meaning that the intrinsic resilience of a host-pathogen system varies across season. Nonetheless, we can still measure the *empirical* resilience of a host-pathogen system by looking at how fast the system returns to the pre-pandemic, endemic dynamics after interventions are lifted.

As an example, consider an intervention that reduce transmission by 50% for 6 months starting in 2020, which causes epidemic patterns to deviate from its original stable annual cycle for a short period of time and eventually come back (Figure 2A). To measure the empirical resilience of this system, we first need to be able to measure

the distance from its pre-pandemic attractor. There are many different ways we can measure the distance from attractor, but for illustrative purposes, we choose one of the most parsimonious approach: that is, we look at how the susceptible (S) and infected (I) populations change over time and measure the distance on the SI phase plane (Figure 2B). In this simple case, the locally estimated scatterplot smoothing (LOESS) fit indicates that the distance from attractor decreases linearly on average (Figure 2C). Furthermore, the overall rate of return matches the intrinsic resilience of the seasonally unforced system (Figure 2C).

Alternatively, NPIs can permanently change our behavior and have persisting impact on the pathogen dynamics; as an example, we consider a scenario in which a 10% reduction in transmission persists even after the NPIs are lifted (Figure 2D–F). In such cases, we cannot know whether the pathogen will return to its original cycle or a different cycle until many years have passed after the NPIs are lifted, meaning that we cannot measure the distance against the new attractor that the system will eventually approach. Nonetheless, we can still measure the distance against the original, pre-pandemic attractor and ask how the distance changes over time (Figure 2E). The LOESS fit suggests that the distance from the attractor will initially decrease exponentially on average (equivalently, linearly on a log scale) and eventually plateau (Figure 2F). Here, a permanent 10% reduction in transmission rate slows down the system, which causes the distance from the attractor to decrease at a slower rate (Figure 2F) than it would have otherwise in the absence of permanent transmission reduction (Figure 2C). This example shows that resilience is not necessarily an intrinsic property of a specific pathogen. Instead, pathogen resilience is a property of a specific attractor that a host-pathogen system approaches, which depends on both pathogen and host characteristics. **[SWP: Add discussion about observation error, e.g., under-reporting.]**

Finally, transient phenomena can also complicate the picture (Figure 2G–I). For example, a stage-structured model for RSV initially exhibits a stable annual cycle, but perturbations from NPIs cause the epidemic to exhibit biennial cycles (Figure 2G). Despite this biennial cycle, we see that the system eventually approaches the original pre-pandemic attractor (Figure 2H), suggesting that this biennial cycle is a transient phenomenon. The LOESS fit indicates that the distance from the attractor will initially decrease exponentially at a rate that is consistent with the intrinsic resilience of the seasonally unforced system, but the rate of decrease slows down as the epidemic exhibits a biennial cycle (Figure 2I). In classical ecological theory, this behavior is also referred to as a ghost attractor, which causes long transient dynamics and slow transitions.

These observations indicate three possibilities. First, we can directly estimate the empirical resilience of a host-pathogen system by looking at how fast the system approaches a pre-pandemic attractor, provided that we can measure the distance from attractor. The empirical approach to estimating pathogen resilience is particularly convenient because it does not require us to know the true underlying model. As we show in Supplementary Materials, estimating the intrinsic resilience from fit-

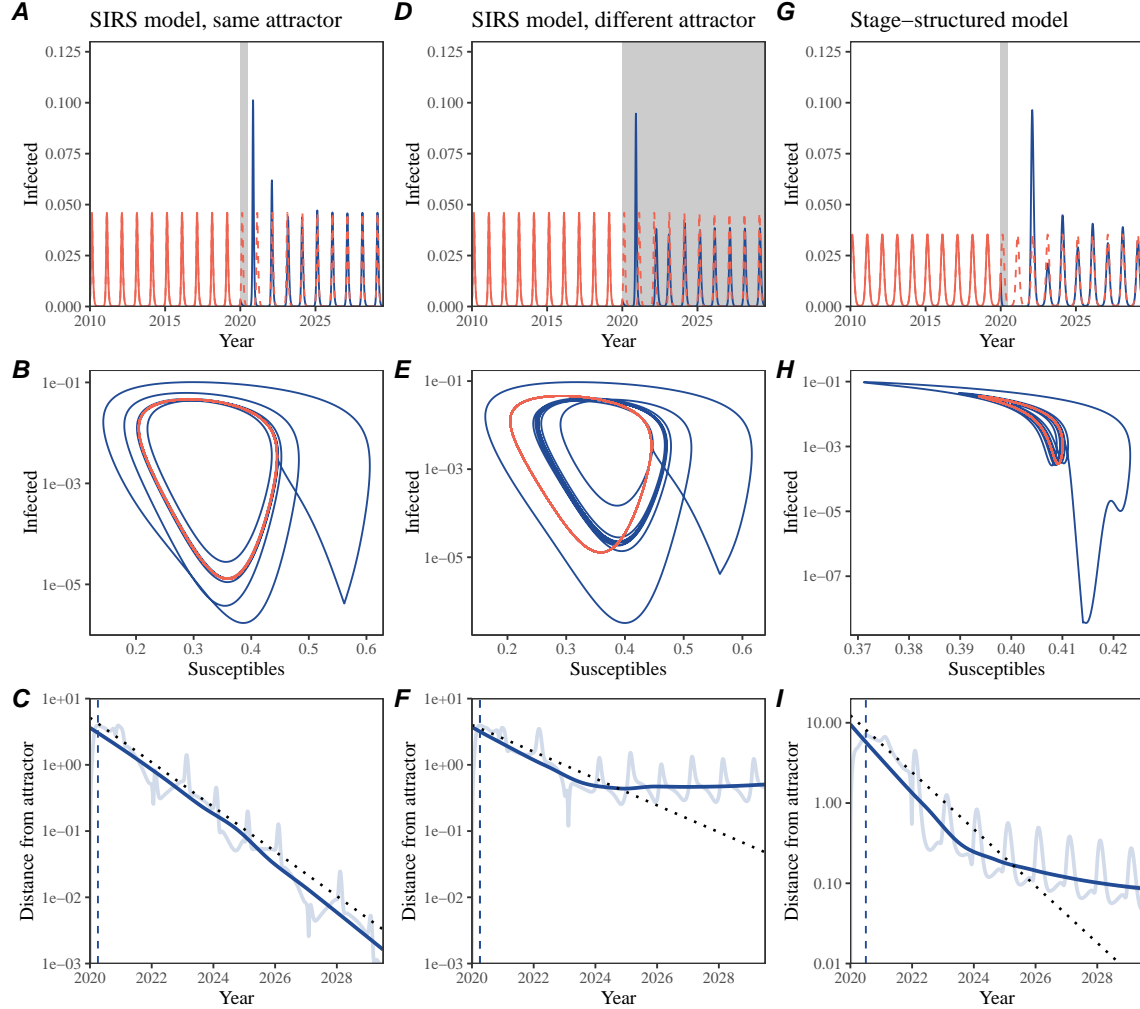


Figure 2: **Conceptual framework for measuring pathogen resilience following NPIs across different scenarios.** (A, D, G) Simulated epidemic trajectories across various models. Red and blue solid lines represent epidemic dynamics before and after interventions are introduced, respectively. Red dashed 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 corresponding model. 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 attractor over time. Transparent solid lines represent the logged distance from attractor. Non-transparent solid lines represent the locally estimated scatterplot smoothing (LOESS) fits to the logged distance from attractor. Dotted lines are superimposed as a comparison to have the same slope as the intrinsic resilience of the system.

119 ting standard compartmental models can lead to biased estimates, especially under

120 model misspecification ([SWP: *TODO*]). Second, resilience estimates allow us to  
 121 make phenomenological predictions about the dynamics of a host-pathogen system  
 122 following a perturbation: assuming that the distance from the attractor will decrease  
 123 exponentially over time, we can obtain a ballpark estimate for when the system will  
 124 reach an attractor. Finally, deviation from an exponential decrease in the distance  
 125 from attractor can provide information about whether the system has reached an  
 126 alternative attractor, or a ghost attractor, that is different from the original, pre-  
 127 pandemic attractor. These alternative attractors may reflect continued perturbations  
 128 from permanent changes in transmission patterns as well as changes in immune land-  
 129 scapes.

130 *[SWP: Multi-strain system to be discussed in the supp after some more investi-*  
 131 *gation.]*

## 132 Inferring pathogen resilience from real data

133 Based on these observations, we now set out to infer pathogen resilience from real  
 134 data. Here, we briefly lay out our approach to estimating pathogen resilience from  
 135 real data (Figure 3). We then test this approach against simulations and apply it to  
 136 real data.

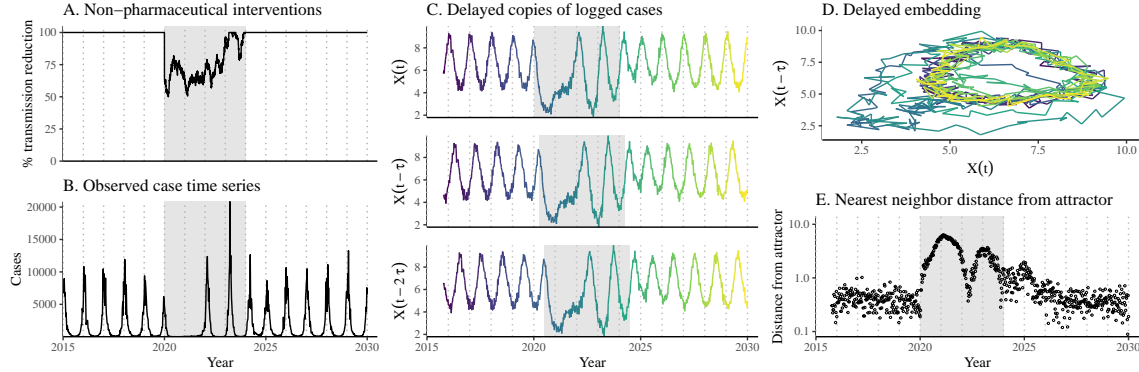
137 So far, we focused on simple examples that assume a constant transmission re-  
 138 duction. However, in practice, the impact of NPIs on pathogen transmission is  
 139 likely more complex (Figure 3A), reflecting introduction and relaxation of various  
 140 intervention strategies. These complexities can lead to longer delays between the  
 141 introduction of NPIs and pathogen re-emergence as well as temporal variation in  
 142 outbreak sizes (Figure 3B): in this example, continued transmission reduction from  
 143 NPIs limits the size of the first outbreak in 2021 following the emergence, allowing  
 144 for a larger outbreak in 2022 when NPIs are further relaxed.

145 Previously, we relied on the dynamics of susceptible and infected hosts to compute  
 146 the distance from attractor (Figure 2), but information on susceptible hosts are  
 147 often not available in practice. In addition, uncertainties in case counts due to  
 148 observation error as well as the possibility of complex, multiannual attractor adds  
 149 challenges to measuring the distance from attractor. To address these challenges, we  
 150 first reconstruct an empirical attractor by utilizing Takens’ theorem, which states  
 151 that an attractor of a nonlinear multidimensional system can be mapped onto a  
 152 delayed embedding. Here, we use delayed copies of logged values of pre-pandemic  
 153 cases  $C(t)$  (Figure 3C) to reconstruct the attractor:

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

154 where the delay  $\tau$  and embedding dimension  $M$  are determined based on autocor-  
 155 relations and false nearest neighbors, respectively. We then apply the same delay  
 156 and embedding dimensions to the entire time series to determine the position on a  
 157 multi-dimensional state space (Figure 3D), which allows us to measure the nearest

neighbor distance between the current state of the system and the empirical attractor (Figure 3E). In principle, we can quantify how fast this distance decreases by fitting a linear regression on a log scale, where the slope of the linear regression corresponds to pathogen resilience.



**Figure 3: A schematic diagram explaining how pathogen resilience can be inferred from real data.** (A) A realistic example of a synthetic NPI, represented by a relative reduction in transmission. (B) The impact of the synthetic NPI on epidemic dynamics simulated using a stochastic SIRS model. (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. This distance time series can be used to infer pathogen resilience by fitting a linear regression after choosing an appropriate window for regression.

Complex changes in the distance from attractor suggest that estimating pathogen resilience from linear regression will likely be sensitive to our choice of fitting windows for the regression. In Supplementary Materials, we explore an automated window selection criteria for linear regression and test it against randomized, stochastic simulations across a wide range of realistic NPI shapes. We find that resilience estimates based on the automated window selection criteria are moderately correlated (0.54) with the intrinsic resilience of the post-NPI attractor. In contrast, a naive approach that uses the entire time series, starting from the peak distance, only gives a correlation of 0.21 and consistently underestimates the intrinsic resilience.

Now, we apply this approach to pathogen surveillance data presented in Figure 1. For each time series, we apply Takens' theorem independently to reconstruct the empirical attractor and obtain the corresponding time series of distance from attractors ([SWP: Supp]). Then, we use the automated window selection criteria to fit a linear regression and estimate the empirical resilience for each pathogen in each country. For most respiratory pathogens, resilience estimates 0.5/year and 2/year (Figure 4A), with the exception of Rhinovirus in the US (0.066/year; 95% CI: 0.018/year–0.113/year) and Bocavirus in Korea (0.087/year; 95% CI: 0.023/year–



0.151/year). Excluding these exceptions, the mean resilience of common respiratory pathogens is 0.974/year (95% CI: 0.784/year–1.16/year). As a reference, this is  $\approx 7$  times higher than the intrinsic resilience of pre-vaccination measles dynamics ( $\approx 0.13$ /year). Finally, resilience estimates for norovirus appears to be comparable to the intrinsic resilience of measles: 0.119/year (95%CI: 0.004/year–0.233/year) for Korea and 0.385/year (95% CI: 0.167/year–0.603/year). A simple ANOVA shows that there are significant differences in resilience estimates across countries ( $p < 0.036$ ) and pathogens ( $p < 0.030$ ).

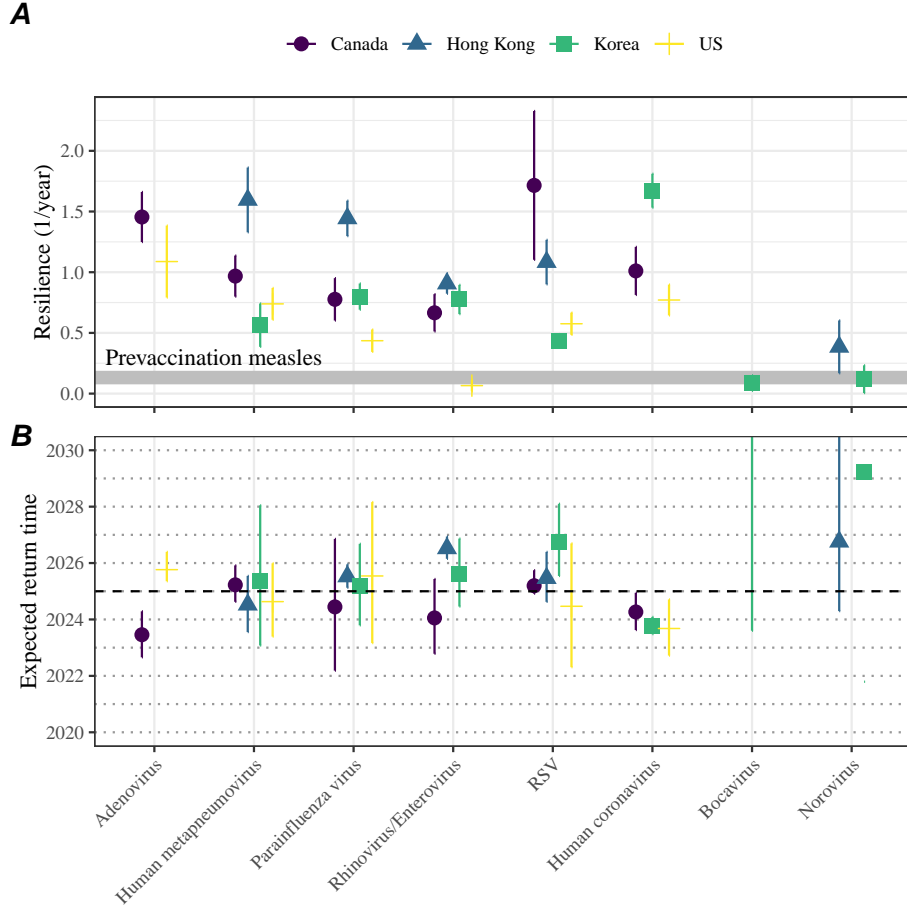


Figure 4: **Summary of resilience estimates.** (A) Estimated pathogen resilience. The gray horizontal line represents the intrinsic resilience of pre-vaccination measles dynamics. (B) Predicted timing of when each pathogen will return to their pre-pandemic cycles. The dashed line in panel B indicates the end of 2024 (current observation time). Error bars represent 95% confidence intervals.

Using resilience estimates, we now predict when each pathogen will return to their original pre-pandemic cycles. Specifically, we extend our linear regression fits to distance-from-attractor time series and ask when the predicted regression line



will cross a threshold value, which we set to a mean of pre-pandemic distances. We predict that a return to pre-pandemic cycles would be imminent for most pathogens (Figure 4B). In addition, we also predict that many pathogens should have already returned to their pre-pandemic dynamics by the end of 2024; but these predictions contradict some of the observed pathogen dynamics. For example, we predict that both human metapneumovirus and RSV in Korea should have returned to their attractors by now, but the magnitude and timing of recent epidemics are different from pre-pandemic patterns (Figure 1). These observations suggest the possibility that some common respiratory pathogens may have converged to different attractors.

## Susceptible host dynamics explain variation in pathogen resilience

So far, we focused on quantifying pathogen resilience from the observed patterns of pathogen re-emergence following COVID-19 interventions. But what factors determine how resilient a host-pathogen system is? Here, we use a standard Susceptible-Infected-Recovered-Susceptible (SIRS) model to show that susceptible host dynamics are the key determinants of pathogen resilience. To do so, we vary 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 compute intrinsic resilience for each parameter.

We find an increase in  $\mathcal{R}_0$  and a decrease in duration of immunity correspond to an increase in pathogen resilience (Figure 5A). These variations can be understood in terms of the susceptible host dynamics, where faster per-capita susceptible replenishment rate causes the system to be more resilient (Figure 5B). This rate can be expressed as a ratio between absolute rate at which new susceptibles enter the population and the equilibrium number of susceptible individuals in the population,  $\bar{S}$ . Therefore, both higher  $\mathcal{R}_0$  and shorter duration of immunity can drive faster per-capita susceptible replenishment rate (Figure 5B), especially because higher  $\mathcal{R}_0$  leads to lower  $\bar{S}$ .

Finally, we can now rank different pathogens based on the average values of empirical resilience, which allows us to determine a set of parameters that are consistent with the estimated resilience (Figure 5A). Across all pathogens we consider, except for bocavirus and norovirus, we estimate that the average duration of immunity is likely to be short ( $< 6$  years) across a plausible range of  $\mathcal{R}_0$ . These rankings further allow us to map each pathogen onto a set of parameters that are consistent with its empirical resilience (Figure 5A) and obtain a plausible range of susceptible replenishment rates for each pathogen (Figure 5B). However, we note that there is no one-to-one correspondance between susceptible replenishment rates and pathogen resilience, leading to a wide uncertainty in the estimates for susceptible replenishment rates (Figure 5B).

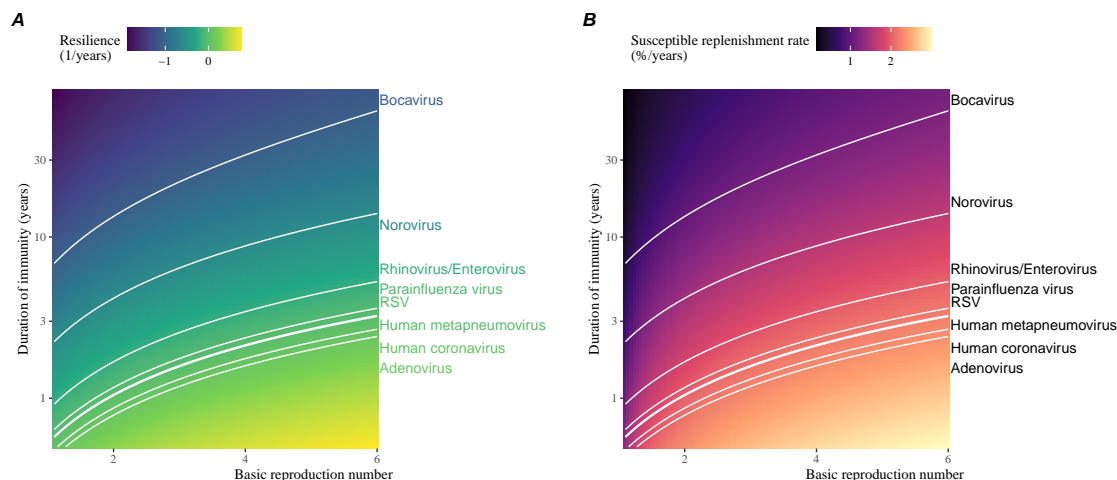


Figure 5: **Linking pathogen resilience to epidemiological parameters and susceptible host dynamics.** (A) The heat map represents intrinsic resilience as a function of the basic reproduction number  $\mathcal{R}_0$  and the duration of immunity. (B) The heat map represents per-capita susceptible replenishment rate as a function of the basic reproduction number  $\mathcal{R}_0$  and the duration of immunity. The standard SIRS model is used to compute intrinsic resilience and per-capita susceptible replenishment rate. Lines correspond to a set of parameters that are consistent with mean resilience estimates for each pathogen. Pathogens are ranked based on their mean resilience estimates, averaged across different countries.

## Discussion

The COVID-19 interventions have caused major disruptions to circulation patterns of both respiratory and non-respiratory pathogens, adding challenges to predicting their future dynamics. On the other hand, these interventions offer large-scale natural experiments for understanding how different pathogens respond to perturbations. In this study, we show that pathogen re-emergence patterns following COVID-19 interventions can be characterized through the lens of ecological resilience. Traditionally, ecological resilience measures how fast a system returns to a reference state following a perturbation. In the context of respiratory pathogens, resilience measures how fast epidemics return to their endemic cycles after interventions are lifted.

We use an attractor reconstruction approach to quantify how distance from attractor changes over time for each pathogen. By fitting a linear regression to log distances, we can estimate pathogen resilience and further predict when each pathogen will return to their endemic cycles. Consistency in resilience estimates across countries is particularly surprising given that each country imposed different intervention measures; this consistency provides robustness to our estimates. The ability to predict future epidemic patterns from resilience estimates also offers a new paradigm for epidemic forecasting. While this approach cannot predict the exact timing of out-

breaks or epidemic patterns, it is nonetheless useful for predicting when epidemics will settle down to regular cycles after a large perturbation, such as COVID-19 interventions.

Our analyses suggest a possibility that several pathogens may have converged to different endemic cycles compared to their pre-pandemic epidemic patterns. Key examples include human metapneumovirus, RSV, and bocavirus in Korea as well as RSV in Hong Kong. These changes may reflect permanent changes in behavior since 2020 or a shift in population-level immunity. However, it seems unlikely that permanent changes in behavior would only affect a few pathogens and not others. A shift in population-level immunity is plausible, as the emergence of SARS-CoV-2 and extinction of influenza B/Yamagata likely caused major changes in immune landscapes; however, we currently do not know how immunity, or lack thereof, from these pathogens would affect infection from other pathogens. Future studies should use detailed mechanistic models, coupled with behavioral and immunological data, to test these hypotheses and better understand post-pandemic dynamics of endemic pathogens.

We show that susceptible host dynamics shape pathogen resilience, where faster replenishment of the susceptible population causes the pathogen to be more resilient. For simplicity, we focus on waning immunity and birth as a main driver of the susceptible host dynamics but other mechanisms can also contribute to the replenishment of the susceptible population. In particular, pathogen evolution, especially the emergence of antigenically novel strains, can cause effective waning of immunity in the population; therefore, we tentatively hypothesize that faster rates of antigenic evolution can also cause a pathogen to be more resilient. Future studies should explore the relationship between the rate of evolution and resilience for antigenically evolving pathogens.

Quantifying pathogen resilience also offers novel approaches to validating epidemiological models. So far, the majority of model validation in epidemiology is based on the ability of a model to reproduce the observed epidemic dynamics and to predict future dynamics. However, there can be plethora of models that meet these criteria. For example, two major RSV models have been proposed so far to explain biennial epidemic patterns: (1) a stage- and age-structured model that allows for disease severity to vary with number of past infections and age of infection and (2) a pathogen-interaction model that accounts for cross immunity between RSV and human metapneumovirus. Since both models can accurately reproduce the observed epidemic patterns, standard criteria for model validation do not allow us to distinguish between these two models. Instead, we can measure the empirical resilience of each model by simulating various perturbations and compare them to estimates of empirical resilience from data, using COVID-19 interventions as an opportunity. Future studies should further investigate using pathogen resilience for validating epidemic models.

There are several limitations to our work. In particular, our estimates of pathogen resilience and the associated ranking are necessarily crude. **[SWP: *Limitation TBD.*]**

290 Nonetheless, our study illustrates the utility of quantifying pathogen resilience for  
291 understanding how different pathogens respond to perturbations.  
292 [SWP: Conclusion paragraph TBD.]

## 293 Materials and Methods

### 294 Data

295 We gathered time series on respiratory infections from four different countries: Canada,  
296 Hong Kong, Korea, and United States (US). As a reference, we also included time  
297 series data on norovirus infections for available countries—in contrast to respiratory  
298 pathogens, we expect gastrointestinal viruses, such as norovirus, to be less affected  
299 by COVID-19 intervention measures.

300 Weekly time series of respiratory infection cases in Canada come from the Res-  
301 piratory Virus Detection Surveillance System, which collect data from select labo-  
302 ratories across Canada. We extracted the data from [https://www.canada.ca/en/  
303 public-health/services/surveillance/respiratory-virus-detections-canada.  
304 html](https://www.canada.ca/en/public-health/services/surveillance/respiratory-virus-detections-canada.html).

305 Weekly time series of respiratory infection cases in Hong Kong came from the  
306 Centre for Health Protection, Department of Health. We extracted the data from  
307 <https://www.chp.gov.hk/en/statistics/data/10/641/642/2274.html>.

308 Weekly time series of respiratory infection cases in Korea came from Korea Dis-  
309 ease Control and Prevention Agency. We extracted the data from [https://dportal.  
310 kdca.go.kr/pot/is/st/ari.do](https://dportal.kdca.go.kr/pot/is/st/ari.do).

311 Finally, weekly time series of respiratory infection cases in the US comes from  
312 the National Respiratory and Enteric Virus Surveillance System.

### 313 Empirical attractor reconstruction

### 314 Linear regression

### 315 Mathematical modeling

316 Throughout the paper, we use a series of mathematical models to illustrate the con-  
317 cept of pathogen resilience and to understand the determinants of pathogen resilience.  
318 In general, the intrinsic resilience for a given system is given by the largest real part  
319 of the eigenvalues of the linearized system at endemic equilibrium. Here, we focus on  
320 the SIRS model and present the details of other models in Supplementary Materials.  
321 The SIRS (Susceptible-Infected-Recovered-Susceptible) model is the simplest model  
322 that allows for waning of immunity, where recovered (immune) individuals are as-  
323 summed to become fully susceptible after an average of  $1/\delta$  time period. The dynamics

324 of the SIRS model is described by the following set of differential equations:

$$\frac{dS}{dt} = \mu - \beta(t)SI - \mu S + \delta R \quad (2)$$

$$\frac{dI}{dt} = \beta(t)SI - (\gamma + \mu)I \quad (3)$$

$$\frac{dR}{dt} = \gamma I - (\delta + \mu)R \quad (4)$$

$$(5)$$

325 where  $\mu$  represents the birth/death rate,  $\beta(t)$  represents the time-varying trans-  
 326 mission rate, and  $\gamma$  represents the recovery rate. The basic reproduction number  
 327  $\mathcal{R}_0 = \beta(t)/(\gamma + \mu)$  is defined as the average number of secondary infections caused  
 328 by a single infected individual in a fully susceptible population and measures the  
 329 intrinsic transmissibility of a pathogen.

330 For this model, the intrinsic resilience corresponds to:

$$-\frac{\text{Re}(\lambda)}{2} = \frac{\delta + \beta I^* + \mu}{2}. \quad (6)$$

331 Here,  $I^*$  represents the prevalence values at endemic equilibrium:

$$I^* = \frac{(\delta + \mu)(\beta - (\gamma + \mu))}{\beta(\delta + \gamma + \mu)}. \quad (7)$$

332 The susceptible replenishment rate is given by:

$$S_{\text{replenish}} = \frac{\mu + \delta R}{S^*}, \quad (8)$$

333 where  $S^* = 1/\mathcal{R}_0$  represents the equilibrium proportion of susceptible individuals.

334 When infection provides life-long immunity ( $\delta \rightarrow 0$ ), the SIRS model becomes  
 335 the SIR model. In this case, the intrinsic resilience is inversely proportional to the

336 In illustrating the impact of

<sup>337</sup> **Supplementary Text**

<sup>338</sup> **Supplementary Figures**

## References

- [1] Edward A Bender, Ted J Case, and Michael E Gilpin. Perturbation experiments in community ecology: theory and practice. *Ecology*, 65(1):1–13, 1984.
- [2] Anthony R Ives and Stephen R Carpenter. Stability and diversity of ecosystems. *science*, 317(5834):58–62, 2007.
- [3] Marten Scheffer, Jordi Bascompte, William A Brock, Victor Brovkin, Stephen R Carpenter, Vasilis Dakos, Hermann Held, Egbert H Van Nes, Max Rietkerk, and George Sugihara. Early-warning signals for critical transitions. *Nature*, 461(7260):53–59, 2009.
- [4] Stuart L Pimm. The structure of food webs. *Theoretical population biology*, 16(2):144–158, 1979.
- [5] Michael G Neubert and Hal Caswell. Alternatives to resilience for measuring the responses of ecological systems to perturbations. *Ecology*, 78(3):653–665, 1997.
- [6] Lance H Gunderson. Ecological resilience—in theory and application. *Annual review of ecology and systematics*, 31(1):425–439, 2000.
- [7] Vasilis Dakos and Sonia Kéfi. Ecological resilience: what to measure and how. *Environmental Research Letters*, 17(4):043003, 2022.
- [8] Jeanne C Chambers, Craig R Allen, and Samuel A Cushman. Operationalizing ecological resilience concepts for managing species and ecosystems at risk. *Frontiers in Ecology and Evolution*, 7:241, 2019.
- [9] Rachel E Baker, Sang Woo Park, Wenchang Yang, Gabriel A Vecchi, C Jessica E Metcalf, and Bryan T Grenfell. The impact of COVID-19 nonpharmaceutical interventions on the future dynamics of endemic infections. *Proceedings of the National Academy of Sciences*, 117(48):30547–30553, 2020.
- [10] Gabriela B Gomez, Cedric Mahé, and Sandra S Chaves. Uncertain effects of the pandemic on respiratory viruses. *Science*, 372(6546):1043–1044, 2021.
- [11] Mihaly Koltai, Fabienne Krauer, David Hodgson, Edwin van Leeuwen, Marina Treskova-Schwarzbach, Mark Jit, and Stefan Flasche. Determinants of RSV epidemiology following suppression through pandemic contact restrictions. *Epidemics*, 40:100614, 2022.
- [12] Sang Woo Park, Brooklyn Noble, Emily Howerton, Bjarke F Nielsen, Sarah Lentz, Lilliam Ambroggio, Samuel Dominguez, Kevin Messacar, and Bryan T Grenfell. Predicting the impact of non-pharmaceutical interventions against COVID-19 on *Mycoplasma pneumoniae* in the United States. *Epidemics*, 49:100808, 2024.



- 375 [13] Eric J Chow, Timothy M Uyeki, and Helen Y Chu. The effects of the COVID-19  
376 pandemic on community respiratory virus activity. *Nature Reviews Microbiol-*  
377 *ogy*, 21(3):195–210, 2023.