The Cohort Net Reproductive Rate: an empirically grounded approach to R_0 for structured populations and epidemics

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Abstract

The net reproductive rate, or per-generation population growth rate R_0, is a key concept for population and epidemic dynamics, because persistence versus extinction is often determined by the sign of R_0-1. Because it is defined as generation-to-generation growth, it is hard to assess empirically for structured populations or epidemics where generations overlap, and is usually estimated by parameterising dynamic models. Here we introduce the Cohort Net Reproductive Rate $R_0^{(c)}$ – the average per-capita number of offspring or secondary infections produced by individuals born or infected at the same time. We derive a general formula for $R_0^{(c)}$, show that it obeys the same invasion threshold as R_0, and analyze when the two coincide or diverge. $R_0^{(c)}$ can be estimated from observations on easily identified groups of individuals, is typically easier to calculate, and provides an intuitive meaning for R_0 when they coincide.

Running title: Cohort R_0

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1 Introduction

The net reproductive rate (or basic reproduction number), R_0, is a cornerstone of ecology, demography and epidemiology. It was introduced nearly a century ago by demographers as the "ratio of total births in two successive generations" (Dublin & Lotka, 1925), that is, the per-generation population growth rate (Lotka, 1925). In infectious disease epidemiology, this corresponds to the ratio of new infections in successive generations when disease prevalence is low, where the generation of an infected individual is one plus that of its infector. Via the *threshold criterion*, R_0 informs whether a population or epidemic grows ($R_0 > 1$), remains stable ($R_0 = 1$), or declines ($R_0 < 1$) (Diekmann *et al.*, 1990).

This criterion is central in epidemiology, where R_0 guides control strategies (Anderson & May, 1982; Heesterbeek & Dietz, 1996; Smith? *et al.*, 2011) and evolutionary analyses (Cortez, 2013; Mylius & Diekmann, 1995). Few quantities in biology have proved so influential, spanning theory, applied management, and even public discourse. Some epidemic models can exhibit *backward bifurcations*, allowing endemic disease persistence (far from extinction) even when $R_0 < 1$, but R_0 still generally determines the fate of an outbreak that begins with few infectives (Diekmann *et al.*, 2010) – that is, it determines local stability versus instability of the disease-free equilibrium (DFE).

Models incorporating individual heterogeneity (in age, size, disease stage, genotype, contagiousness, etc.) have long been essential for understanding how diversity and inheritance shape invasion and persistence of populations and diseases, and for linking population dynamics with evolution and genetics (Lotka, 1925; Ross, 1916). In general this heterogeneity is incorporated via discrete traits (which can be discretizations of continuous traits) leading to matrix projection models, but modelling of time differs. Epidemiologists often use continuous time differential equation models, ecologists discrete time matrix projection models.

Although defined at the population level, R_0 can also be interpreted for individuals. For epidemiology, R_0 corresponds to "the expected number of secondary cases produced by a typical infected individual during its entire period of infectiousness in a completely susceptible population" (Diekmann *et al.*, 1990). In demography, R_0 corresponds to the "mean Lifetime Reproductive Success" (LRS, Caswell, 2011), the expected number of offspring of a *typical* individual over its lifetime. These definitions are parallel: infected, infection and recovery rates in epidemiology correspond to individuals, fertility and mortality rates in demography. Because R_0 provides a simple link between individual and population properties, it is a central measure in demography and population ecology (e.g., Snyder *et al.*, 2021; Tuljapurkar *et al.*, 2020; Waples, 2022). However, in such models, if newborn/newly infected individuals can vary in state, that is, in their *type* (also known as "newborn type" (sensu Caswell, 2019; Cochran & Ellner, 1992; Ellner, 2018) or "birth state" (sensu Rueffler & Metz, 2013)), the individual-level interpretation of R_0 is complicated by the question: what exactly is a *typical* individual?

Moreover, when generations overlap it is difficult to track reproductive/infective output by

generation, and often impossible to assign individuals/infected to a specific generation. This complicates comparisons between the theoretical value of R_0, obtained from a generation-to-generation dynamical model (the "next generation matrix"), and empirical estimates of population growth per generation. As a result, R_0 is often treated as an abstract theoretical quantity, with no clear empirical analogue in heterogeneous natural populations or emerging epidemics (Delamater *et al.*, 2019).

To address these issues, we revisit the foundations of reproduction numbers from an individual-based perspective. We show formally that in a model with several *types* (a population model with several birth states, e.g. small and large newborn or an epidemic model with multiple kinds of infective individuals, e.g., mild and severe infections), the notion of a *typical* individual, used in the individual-based interpretation of the net reproductive rate (see, e.g., Diekmann *et al.*, 1990), means a typical member of a generation, which is in general not the same as a typical member of a cohort of newborns or newly infected individuals. Building on this characterization of R_0, we introduce the *Cohort Net Reproductive Rate*, $R_0^{(c)}$, which instead considers individuals drawn at random from a *cohort* in chronological time, that is, from the set of all individuals born/infected at one time. This renders $R_0^{(c)}$ more directly observable from data, specifically data on the lifetime reproductive or infectious success of a set of individuals born or infected contemporaneously.

$R_0^{(c)}$ is new as an explicitly defined and named measure of lifetime reproduction. Related expressions have appeared in special cases in the literature, but R_0 and $R_0^{(c)}$ have often been conflated, and their difference in the general case has gone unrecognized. We derive a general formula for $R_0^{(c)}$ applicable to any structured or compartmental model and more importantly, we show that $R_0^{(c)}$ obeys the same threshold criterion as R_0: the sign of $R_0^{(c)}-1$ predicts whether populations or epidemics grow or decline. It can therefore be used equivalently for evolutionary/invasion analyses. We further show that the values of $R_0^{(c)}$ and R_0 coincide in some situations that are common in both theoretical and data-driven models, in particular when there is only one newborn type in the model or when the offspring/infection distribution is the same for all parents/infectors. More broadly, $R_0^{(c)}$ illustrates the value of metrics that are not only theoretically consistent but also empirically grounded, ensuring that fundamental concepts retain relevance for the analysis of heterogeneous populations. Our notation and some essential formulas are summarized in Table 1.

2 Models and Assumptions

We consider deterministic models for population and epidemic dynamics in a constant environment, where individuals are heterogeneous with regard to their current *state*. An individual's state is, in principle, a minimal finite list of current attributes that uniquely determines that individual's probability distribution of future states (Caswell, 2001, sec. 3.1). A matrix projection model assumes that there is a finite number s of possible individual states. Integral projection models (IPM) formally allow a continuous state space, but are typically implemented as high-dimensional matrix models(Ellner & Rees, 2006; Metcalf *et al.*, 2013). As in Ellner (2018), we use matrix notation but the formulas are valid for IPMs when suitably interpreted, e.g., $\boldsymbol{v}^T\boldsymbol{w}$ denotes the

Table 1: Parameters and formulas for structured models in discrete (left column) and continuous time (right column). $\rho()$ denotes the spectral radius, $\mathbf{I}$ the identity matrix, ^{T} matrix transposition and $\mathbf{1}$ a vector of all 1's so that $\mathbf{1}^T\boldsymbol{w}=\sum_i w_i$ which equals the L_1 norm $||\boldsymbol{w}||_1$ if $\boldsymbol{w}\geq 0$.

Population ecology (discrete time)	**Epidemiology (continuous time)**
Parameters	
Fertility matrix: $\mathbf{F}$, where $F(i,j)$ is the expected number of offspring in state i produced by a parent in state j	Infection matrix: $\mathbf{F}$, where $F(i,j)$ is the instantaneous rate of appearance of state i infected produced by an infected individual in state j.
Survival matrix: $\mathbf{S}$, where $S(i,j)$ is the probability that an individual in state j survives and transitions to state i and $1-\sum_i S(i,j)$ is the death rate in state j	Transition matrix: $\mathbf{V}$, where $-V(i,j)$, for $i\neq j$ is the instantaneous transition rate from state j to state i and $\sum_i V(i,j)$ is the instantaneous rate of recovery or death for state j.
Population projection over chronological time t	
$\boldsymbol{n}_{t+1}=(\mathbf{F}+\mathbf{S})\boldsymbol{n}_t$ (1a)	$\frac{d\boldsymbol{n}}{dt}=(\mathbf{F}-\mathbf{V})\boldsymbol{n}_t$ (1b)
Asymptotic growth rate	
$\lambda=\rho(\mathbf{F}+\mathbf{S})$ (2a)	$r=\rho(\mathbf{F}-\mathbf{V})$ (2b)
Stationary vector of relative state abundances, $\boldsymbol{w}$	
$(\mathbf{F}+\mathbf{S})\boldsymbol{w}=\lambda\boldsymbol{w}$, with $\mathbf{1}^T\boldsymbol{w}=1$ (3a)	$(\mathbf{F}-\mathbf{V})\boldsymbol{w}=r\boldsymbol{w}$, with $\mathbf{1}^T\boldsymbol{w}=1$ (3b)
Next-generation matrix	
$\mathbf{R}=\mathbf{F}(\mathbf{I}-\mathbf{S})^{-1}$ (4a)	$\mathbf{R}=\mathbf{F}\mathbf{V}^{-1}$ (4b)
Population projection over generation time T	
$\boldsymbol{g}_{T+1}=\mathbf{R}\boldsymbol{g}_T$ (6)	
Per-generation population growth rate	
$R_0=\rho(\mathbf{R})$ (7)	
Stationary newborn/new infective distribution over generation time, $\boldsymbol{w}_g$	
$\mathbf{R}\boldsymbol{w}_g=R_0\boldsymbol{w}_g$, with $\mathbf{1}^T\boldsymbol{w}_g=1$ (8)	
Individual-based interpretation of R_0	
$R_0=\mathbf{1}^T\mathbf{R}\boldsymbol{w}_g$ (9)	
Stationary newborn/new infective distribution over chronological time, $\boldsymbol{w}^*$	
$\boldsymbol{w}^*=\frac{\mathbf{F}\boldsymbol{w}}{\mathbf{1}^T\mathbf{F}\boldsymbol{w}}$ (11)	
Cohort-net reproductive rate $R_0^{(c)}$	
$R_0^{(c)}=\mathbf{1}^T\mathbf{R}\boldsymbol{w}^*$ (12)	

inner product $\int v(z)w(z)dz$.

For both population and epidemic dynamics, the demographic rates that determine the temporal dynamics of the population are summarized in two $s\times s$ projection matrices. For

structured population models, the projection matrices are $\mathbf{F}$, the fertility matrix, and $\mathbf{S}$, the survival matrix (see table 1). Here $F(i,j)$ is the expected number of newborns in state i present in the population at time $t+1$, per individual in state j at time t, and $S(i,j)$ is the probability that an individual in state j survives one time step and transitions to state i (Caswell, 2001). The population projection in chronological time is then

$$\boldsymbol{n}_{t+1} = (\mathbf{F}+\mathbf{S})\boldsymbol{n}_t. \tag{1a}$$

Note that the projection matrices are assumed to be constant and, in particular, density-independent. However, the theory we develop here also applies to the linearization of a density-dependent model at the extinction equilibrium $\boldsymbol{n}_t = \mathbf{0}$. The projection matrices in eqn. (1a) are then simply the density-dependent matrices evaluated at $\boldsymbol{n}_t = \mathbf{0}$ (Caswell, 2001, eq. 16.47). Persistence versus extinction in eqn. (1a) corresponds to local stability versus instability of the extinction equilibrium in the density-dependent model.

In epidemiology (continuous-time, discrete-traits, ordinary differential equation models), the corresponding matrices determining the value of R_0 are $\mathbf{F}$, the infection or transmission matrix, and $\mathbf{V}$, the compartment transition matrix (in these models, disease states are generally called disease compartments). For epidemic models, $\mathbf{F}$ and $\mathbf{V}$ are the result of linearizing a typically nonlinear model at the disease-free equilibrium (DFE) (Diekmann *et al.*, 2010; van den Driessche & Watmough, 2002) and isolating the linearized equations for infected states; see SM I for details. The linearized dynamics for the infected compartments $\boldsymbol{n}(t)$ when $||\boldsymbol{n}||_1$ is small then have the form

$$\frac{d\boldsymbol{n}}{dt} = (\mathbf{F}-\mathbf{V})\boldsymbol{n}(t). \tag{1b}$$

In both cases, if i is not a possible newborn/newly-infected state (i.e., i is not a *type*), then $F(i,j) = 0$ for all j.

Projection over generations is only meaningful if there *is* a next generation, so in both cases we assume that $\mathbf{F} > 0$, meaning that all entries are non-negative and at least one is positive. We also require that $\mathbf{I}-\mathbf{S}$ and $\mathbf{V}$ are invertible. For discrete time, this will be true if any individual dies eventually (i.e., the maximum state-dependent survival probability is <1). For continuous time, the equivalent condition that $\mathbf{V}^{-1}$ exists and is finite corresponds to each individual spending a finite total time in all compartments of the model (van den Driessche & Watmough, 2002). In both cases we also require some assumptions of primitivity (irreducibility and aperiodicity), which we summarise in SM II, together with a further, mild condition ensuring convergence of generation-structured abundances to a unique stable distribution.

At the population stable state distribution – assuming the environment is constant – abundances grow or decline exponentially at a rate given by the dominant eigenvalue, denoted

ρ, of the projection matrix:

$$\textit{Population ecology (discrete time):} \qquad \boldsymbol{n}_t = \lambda^t \boldsymbol{n}_0, \qquad \text{with } \lambda = \rho(\mathbf{F}+\mathbf{S}) \tag{2a}$$

$$\textit{Epidemiology (continuous time):} \qquad \boldsymbol{n}_t = e^{rt} \boldsymbol{n}_0, \qquad \text{with } r = \rho(\mathbf{F}-\mathbf{V}) \tag{2b}$$

At the stable state, the relative abundance vector in chronological time, $\boldsymbol{w}$, is constant; it is the right eigenvector, scaled so that $\mathbf{1}^T\boldsymbol{w} = 1$, associated with the dominant eigenvalue λ or r (where $\mathbf{1}$ is the vector of ones and ^{T} denotes transposition):

$$\textit{Population ecology (discrete time):} \qquad \lambda\boldsymbol{w} = (\mathbf{F}+\mathbf{S})\boldsymbol{w} \tag{3a}$$

$$\textit{Epidemiology (continuous time):} \qquad r\boldsymbol{w} = (\mathbf{F}-\mathbf{V})\boldsymbol{w} \tag{3b}$$

3 Background: the Net Reproductive Rate, R_0

From the chronological-time matrices we can derive the next-generation matrix, $\mathbf{R}$ (Caswell, 2001; van den Driessche & Watmough, 2002):

$$\textit{Population ecology (discrete time):} \qquad \mathbf{R} = \mathbf{F}(\mathbf{I}-\mathbf{S})^{-1} \tag{4a}$$

$$\textit{Epidemiology (continuous time):} \qquad \mathbf{R} = \mathbf{F}\mathbf{V}^{-1} \tag{4b}$$

For *types* i and j, $R(i,j)$ is the expected number of offspring/cases of *type* i produced by a newborn/newly-infected of *type* j over its entire lifetime/infectious period. From $\mathbf{R}$, one can directly compute $r(j)$, the expected number of offspring/secondary infection that a newborn/newly-infected of *type* j will produce during its lifetime/infectious time (regardless of the types of the offspring/cases produced):

$$r(j) = \sum_i R(i,j) \quad \text{or equivalently} \quad \boldsymbol{r}^T = \mathbf{1}^T\mathbf{R}, \tag{5}$$

The next-generation matrix $\mathbf{R}$ flattens the differences in generation time across types. It considers that all newborns (or newly-infected) arise from parents (or infectors) of the same "age" measured in the synthetic generation time over which $\mathbf{R}$ projects abundances:

$$\boldsymbol{g}_{T+1} = \mathbf{R}\boldsymbol{g}_T, \tag{6}$$

where $\boldsymbol{g}_T$ is the vector of abundances of generation T structured by types; for every $T \geq 1$ (regardless of $\boldsymbol{g}_0$), $\boldsymbol{g}_T$ has value 0 for states that are not types (see SM II). At the stable state, $\boldsymbol{g}_T$, of length s, has value 0 for states that are not *types*. As in chronological-time projection (eqs. 1a, 2a), abundances change exponentially over time at rate $\rho(\mathbf{R})$ per generation. This is the per-generation growth rate of Dublin and Lotka (Dublin & Lotka, 1925), i.e. R_0, as derived for general structured

models by Diekmann *et al.* (1990) for epidemiology and Cushing & Zhou (1994) for demography[1] :

$$R_0 = \rho(\mathbf{R}). \tag{7}$$

Public health monitoring of ongoing epidemics uses several related definitions of a time-varying "effective reproduction number" R_t that takes account of time-varying conditions (e.g. Fraser, 2007). Goldwasser *et al.* (2026) have recently shown that two of those (the "case" and "instantaneous" reproduction numbers) are identical to the "mechanistic" $R_0 = \beta N/\gamma$ for SIR and SEIR models with one infectious class and infection rate proportional to SI.

3.1 Individual level interpretation of R_0

Let $\boldsymbol{w}_g$ be the stable *type* proportions within a generation. It is the right-eigenvector of $\mathbf{R}$, scaled so that $\mathbf{1}^t \boldsymbol{w}_g = 1$, associated with dominant value R_0:

$$\mathbf{R}\boldsymbol{w}_g = R_0 \boldsymbol{w}_g, \tag{8}$$

Summing the elements on both sides of equation 8 leads to (via eq.5)

$$\boxed{R_0 = \sum_j r(j) w_g(j), \quad \text{or equivalently} \quad R_0 = \mathbf{1}^T \mathbf{R} \boldsymbol{w}_g = \boldsymbol{r}^T \boldsymbol{w}_g}. \tag{9}$$

The net reproductive rate corresponds therefore to the average of the $r(j)$ values weighted by the $w_g(j)$ (the probability that an individual taken at random in a generation is of type j). This clarifies the notion of a *typical* individual in the individual-based definition of R_0 (Diekmann *et al.*, 1990) and provides an unambiguous, equivalent definition of the per-generation growth rate:

> *R_0 is the expected number of offspring/secondary cases, produced over its life-time/infectious period by a newborn/newly-infected individual chosen at random from the stable distribution $\boldsymbol{w}_g$ in a given generation.*

3.2 The threshold criterion

The threshold criterion relates the position of R_0 relative to 1 with that of the Malthusian parameter ($r = \ln\lambda$) relative to 0. It has been established by Diekmann *et al.* (1990, p.368) for

[1] Note that Cushing & Zhou (1994) write $R_0 = \rho\Big((\mathbf{I}-\mathbf{S})^{-1}\mathbf{F}\Big)$, which is equivalent because $\mathbf{R}$ and $(\mathbf{I}-\mathbf{S})^{-1}\mathbf{F}$ are similar and therefore have the same eigenvalues; two square matrices $\mathbf{A}$,$\mathbf{B}$ are similar if $\mathbf{A} = \mathbf{V}\mathbf{B}\mathbf{V}^{-1}$ for some matrix $\mathbf{V}$.

epidemiology and Cushing & Zhou (1994, Theorem 3, p.306) for demography:

$$\begin{cases} \lambda > 1 \iff r > 0 \iff R_0 > 1 \\ \lambda = 1 \iff r = 0 \iff R_0 = 1 \\ \lambda < 1 \iff r < 0 \iff R_0 < 1 \end{cases} \tag{10}$$

For epidemiological models, the threshold criterion requires some technical assumptions about $\mathbf{F}$ and $\mathbf{V}$ that are generally satisfied in practice (Diekmann *et al.*, 2010, Appendix A), which we summarize in SM II.

The threshold criterion (10) makes it possible to use R_0 to predict whether a population or epidemic will grow, remain stable, or go extinct, thereby justifying its central role in control strategies (Smith? *et al.*, 2011). In a constant environment – the usual framework for R_0 studies – evolution maximizes the long-term growth rate λ (or r), not R_0 (Charlesworth, 1980; Demetrius, 1981; Murray, 1992; Stenseth, 1984), but via the threshold criterion, R_0 can be used in evolutionary invasion analyses (Cortez, 2013; Mylius & Diekmann, 1995).

4 The Cohort Net Reproductive Rate, $R_0^{(c)}$

4.1 Definition

Because R_0 is defined as a per-generation growth rate (eq.7) or, equivalently, the average LRS weighted by the type probability distribution *in a generation* (eq.9), it is difficult to compare directly with observed data because researchers almost never know which generation an observed individual belongs to. Pedigrees or infection chains are sometimes available, but they usually cover only a small fraction of the population. This motivates an empirically grounded alternative: the Cohort Net Reproductive Rate, $R_0^{(c)}$:

> *$R_0^{(c)}$ is the expected number of offspring/secondary cases, produced over its lifetime/infectious period, by a newborn/newly infected individual chosen at random from a cohort (the set of individuals born or newly infected at the same chronological time) when the population is at its stable distribution $\boldsymbol{w}$.*

At the stable state, all cohorts have the same type distribution (see eq. 11 below), so $R_0^{(c)}$ is equivalently the expected number of offspring/secondary cases produced by a newborn/newly-infected individual drawn from *any* cohort. In this sense, the essential contrast between R_0 and $R_0^{(c)}$ is not really *cohort* versus *generation*, but *chronological time* versus *generation time*: $R_0^{(c)}$ describes reproduction or infection as it unfolds in real time, while R_0 describes it along the synthetic generation-time axis used by the next-generation matrix.

4.2 Formulas

The Cohort Net Reproductive Rate is therefore the average LRS weighted by the type probability distribution *in a cohort* or time-step. At the stable state, all cohorts have the same type distribution, represented by the vector of relative abundances of newborn/newly-infected individuals, $\boldsymbol{w}^*$:

$$w^*(i) = \frac{\sum_j F(i,j)w(j)}{\sum_{i,j} F(i,j)w(j)}, \quad \text{that is,} \quad \boldsymbol{w}^* = \frac{\mathbf{F}\boldsymbol{w}}{\mathbf{1}^T\mathbf{F}\boldsymbol{w}}, \tag{11}$$

Like $\boldsymbol{w}_g$, vector $\boldsymbol{w}^*$ is defined on the s states of the population structure and has value 0 for states that are not types. From the law of total expectation, and paralleling eqn. (9), we have:

$$\boxed{R_0^{(c)} = \sum_j r(j)w^*(j) = \sum_{i,j} R(i,j)w^*(j), \quad \text{that is,} \quad R_0^{(c)} = \boldsymbol{r}^T\boldsymbol{w}^* = \mathbf{1}^T\mathbf{R}\boldsymbol{w}^*} \tag{12}$$

Both R_0 and $R_0^{(c)}$ represent the expected LRS of a typical individual. The difference lies in the sampling: for R_0, the typical individual is drawn from a generation; for $R_0^{(c)}$, it is drawn from a cohort. Substituting eqn. (11) into eqn. (12), we have a general expression of the Cohort Net Reproductive Rate for any structured model,

$$\boxed{R_0^{(c)} = \frac{\mathbf{1}^T\mathbf{R}\mathbf{F}\boldsymbol{w}}{\mathbf{1}^T\mathbf{F}\boldsymbol{w}}.} \tag{13}$$

where $\mathbf{R}$ is the next-generation matrix for the model.

5 Properties of the Cohort Net Reproductive Rate

5.1 Threshold criterion for the Cohort Net Reproductive Rate

Like the classical R_0, the Cohort Net Reproductive Rate satisfies the threshold criterion: whether the population or epidemic grows or declines depends only on the sign of $R_0^{(c)} - 1$:

$$\boxed{\begin{cases} \lambda > 1 \iff r > 0 \iff R_0^{(c)} > 1 \\ \lambda = 1 \iff r = 0 \iff R_0^{(c)} = 1 \\ \lambda < 1 \iff r < 0 \iff R_0^{(c)} < 1 \end{cases}} \tag{14}$$

The Cohort Net Reproductive Rate can therefore be used in the same way as R_0 for evolutionary analyses (Cortez, 2013; Mylius & Diekmann, 1995) and control strategies (Anderson & May, 1982; Heesterbeek & Dietz, 1996; Smith? *et al.*, 2011).

Proof:. In discrete time, from eq. (3a) we have $(\mathbf{F}+\mathbf{S})\boldsymbol{w} = \lambda\boldsymbol{w}$ therefore

$$\mathbf{F}\boldsymbol{w} = (\lambda\mathbf{I} - \mathbf{S})\boldsymbol{w} = (\mathbf{I} - \mathbf{S})\boldsymbol{w} + (\lambda - 1)\boldsymbol{w}. \tag{15}$$

Substituting this expression for $\mathbf{F}\boldsymbol{w}$ into the numerator of eq.(13), we obtain

$$R_0^{(c)} = 1 + (\lambda - 1)\frac{\mathbf{1}^T\mathbf{R}\boldsymbol{w}}{\mathbf{1}^T\mathbf{F}\boldsymbol{w}}. \tag{16}$$

The Perron-Frobenius Theorem tells us that all entries in $\boldsymbol{w}$ are positive; so as $\mathbf{F}$ is non-negative with at least one positive entry, the denominator in the fraction in eq.(16) is necessarily strictly positive. The numerator is always positive, therefore the fraction is always finite and positive, hence $R_0^{(c)} - 1$ is a positive multiple of $(\lambda - 1)$.

In the same way, for the continuous time (infectious disease) model we have

$$R_0^{(c)} = 1 + r\frac{\mathbf{1}^T\mathbf{R}\boldsymbol{w}}{\mathbf{1}^T\mathbf{F}\boldsymbol{w}} \tag{17}$$

implying that $R_0^{(c)} - 1$ is a positive multiple of r. ■

R_0 for infectious diseases was introduced by Diekmann *et al.* (1990) in a very general continuous-time model where individuals are cross-classified by infection status and by a "heterogeneity" state h that can be discrete or continuous. The low-prevalence (linearized) spread of infections is governed by an infection kernel $A(\tau,\xi,\eta)$ defined to be the expected infectivity of an individual which was infected τ units of time ago, while having h-state η, towards a susceptible which has h-state ξ. In SM V we give the definitions of R_0 and $R_0^{(c)}$ for that model, and show that under the Diekmann *et al.* (1990) assumptions about the infection kernel, $R_0^{(c)}$ also satisfies the Threshold Criterion for that model: its value relative to 1 determines whether the disease decreases or increases exponentially over time.

5.2 When does $R_0^{(c)} = R_0$?

One newborn type If there is only one newborn type – say all newborn/newly-infected are in state 1 – then the only non-zero entry in $\boldsymbol{w}_g$ (respectively $\boldsymbol{w}^*$) is $w_g(1) = 1$ (respectively $w^*(1) = 1$). Then (by eqs. 12 and 9), $R_0^{(c)} = R_0 = R(1,1)$. Age structured models such as Leslie matrices are a demographic example, and epidemic models with a single newly-infected (entry) compartment, such as the classic SIR model, are an epidemiological example. So for the majority of historical models in population ecology and epidemiology, R_0 *is* the expected lifetime reproduction (or number of secondary infections) produced by an individual (or rare infective) taken at random from a cohort.

Equal reproduction or generation-time characteristics across types

Equal expected lifetime reproductive success If there are multiple offspring types but $r(j)$ has the same value $\tilde{r}$ for all types j (that is, expected LRS is independent of type), then $\boldsymbol{r}^T = \tilde{r}\mathbf{1}^T$ and therefore $\boldsymbol{r}^T\boldsymbol{w}^* = \boldsymbol{r}^T\boldsymbol{w}_g = \tilde{r}$, so from (9) and (12) we have $R_0^{(c)} = R_0 = \tilde{r}$ – the common LRS $\tilde{r}$ is the population's R_0.

Equal distribution of parental age at birth-giving Another sufficient condition is that the distribution of parental age at reproduction does not vary across offspring types. We show in SM IV, building on Ellner (2018), that this condition implies $R_0^{(c)} = R_0$. Generation time is the expectation of parental age at reproduction, therefore this condition implies that all types have the same generation time (but the reverse is not true: equal distributions of parental age require equal moments of every order, not just the average age).

Differences between types are not heritable $R_0^{(c)} = R_0$ whenever offspring type is independent of parent state, meaning that $\mathbf{F} = \boldsymbol{c}\boldsymbol{b}^T$ for some non-negative vectors $\boldsymbol{c}$ and $\boldsymbol{b}$ with $\mathbf{1}^T\boldsymbol{c} = 1$. Mathematically, this says that all columns of $\mathbf{F}$ are multiples of $\boldsymbol{c}$ and that therefore $\boldsymbol{w}^* = \boldsymbol{c}$. Equivalently, the matrix $\mathbf{F}$ has rank equal to one. The biological interpretation is that the distribution of offspring/new infection types is the same (equal to $\boldsymbol{w}^*$) for all potential parents/infectors. From an evolutionary perspective, $\mathbf{F}$ having rank one implies that there is no type inheritance between parent and offspring: independence from parent state implies independence in particular from the parent's type (i.e., their state at birth), so parent and offspring type are completely uncorrelated. In the demographic literature, $\mathbf{F}$ having rank one has been called "exact mixing at birth" (Ellner *et al.*, 2016b). In the mathematical epidemiology literature, the very common assumption of proportionate mixing (which includes the special case of random mixing) results also in $\mathbf{F}$ having rank one (see, for example, eqn. 6.4 in Hethcote & Van Ark (1987)).

Proof: In the population ecology (discrete time) case the next generation matrix is

$$\mathbf{R} = \mathbf{F}(\mathbf{I}-\mathbf{S})^{-1} = \boldsymbol{c}\boldsymbol{b}^T(\mathbf{I}-\mathbf{S})^{-1}, \tag{18}$$

implying that $\mathbf{R}\boldsymbol{u}$ is a multiple of $\boldsymbol{c}$ for any vector $\boldsymbol{u}$. In particular, $\boldsymbol{w_g} = (1/R_0)\mathbf{R}\boldsymbol{w_g}$ is a multiple of $\boldsymbol{c}$. As $\mathbf{1}^T\boldsymbol{w_g} = \mathbf{1}^T\boldsymbol{c} = 1$, we have $\boldsymbol{w_g} = \boldsymbol{c}$. For the same reasons we also have $\boldsymbol{w}^* = \boldsymbol{c}$. From eqns. (9) and (12) we then have $R_0 = R_0^{(c)} = \mathbf{1}^T\mathbf{R}\boldsymbol{c}$. The epidemiology case (continuous time) is identical except that the formula for the next generation matrix has $\mathbf{V}^{-1}$ in place of $(\mathbf{I}-\mathbf{S})^{-1}$. ■.

This result was anticipated by Diekmann *et al.* (1990). They presented the formula for $R_0^{(c)}$ for models where the next generation operator has one-dimensional range, but only as a way to compute R_0 for that case, without naming it or explaining its biological meaning. See SM V for the details and formulas. Similar formulas were derived by Fraser (2007) for the instantaneous and case "effective reproductive number" R_t in Kermack-McKendrick models with exact mixing at birth and homogeneous mixing.

Only one reproductive or infective state A particular case of rank-one $\mathbf{F}$ arises when only one state has positive fecundity/infectivity, for example stage-structured matrix models with a single "flowering plants" or "reproductive adults" stage. In that case, $\boldsymbol{w}^*$ and $\boldsymbol{w}_g$ are both equal to the one nonzero column of $\mathbf{F}$ rescaled so the entries sum to 1. For example, Diekmann *et al.* (2010) consider a compartmental epidemic model with two latent types E_1,E_2 that merge into a single infective state I. Because there is only one infective compartment, $\mathbf{F}$ has a single nonzero column and therefore

rank one. Consequently $R_0^{(c)}$, which is easily derived for that model, exactly matching the Diekmann *et al.* (2010, eqn. 2.10) formula for R_0. See SM III for the model and the resulting formula.

Prevalence of rank-one $\mathbf{F}$ *in empirical models* Rank-one $\mathbf{F}$ matrices are also very common in data-driven matrix population models. The COMADRE data base of animal matrix projection models (Salguero-Gómez *et al.*, 2016) contained 490 empirically fitted matrix projection models for 79 different species, after quality-control screening as in Hernández *et al.* (2024) of version 4.21.8.0. All 490 $\mathbf{F}$ matrices had rank one. For the COMPADRE plant matrix data base (1505 matrices for 202 species, after screening of version 6.22.5.0), 86% of fitted models (88% of species) had $\mathbf{F}$ matrices with rank one. In the PADRINO plant and animal IPM data base (Levin *et al.*, 2022) the fecundity kernels for 10 of 13 species (77%) had rank one.

6 Case Study and Empirical Estimation of R_0 and $R_0^{(c)}$

We now study as an illustration a simple model with two states, both of which are possible offspring types.

Epidemiology interpretation Consider a virus affecting differently female (type 1) and male (type 2) susceptibles. We assume that the disease-free equilibrium is a population of 200 susceptibles with a 50:50 sex ratio, $n(1)=n(2)=100$. The recovery rate is $v_1=0.5$ per week for females, $v_2=0.9$ for males.

The rate at which infectious individuals of type (that is, sex) j transmit the disease to type i susceptibles results from contact rates $c_{i,j}$ and the probability of infection per contact p_j (assumed to depend only on infector type): $\beta_{i,j}=c_{i,j}p_j$. Males are assumed to be twice as infectious as females: $p_1=2p_2=\frac{1}{100}$. The virus is mostly (but not entirely) transmitted via intimate contact, so contact rates are higher between the sexes than within them: $c_{1,2}=c_{2,1}=0.9$ and $c_{1,1}=c_{2,2}=0.1$ per week. Linearizing this model at the disease-free equilibrium leads to (for details, see SM I)

$$\mathbf{F}=\begin{bmatrix} N_1\beta_{1,1} & N_1\beta_{1,2} \\ N_2\beta_{2,1} & N_2\beta_{2,2} \end{bmatrix}=\begin{bmatrix} 0.1 & 0.45 \\ 0.9 & 0.05 \end{bmatrix} \quad \text{and} \quad \mathbf{V}=\begin{bmatrix} v_1 & 0 \\ 0 & v_2 \end{bmatrix}=\begin{bmatrix} 0.5 & 0 \\ 0 & 0.9 \end{bmatrix} \tag{a1}$$

The dominant eigenvalue and associated right eigenvector of $\mathbf{F}-\mathbf{V}$ are

$$r=0.05 \quad \text{and} \quad \boldsymbol{w}=\begin{bmatrix} 0.5 \\ 0.5 \end{bmatrix} \tag{b1}$$

As $r>0$, the virus spreads in the population. The value of $\boldsymbol{w}$ implies that at steady state for the (linearized) low-prevalence disease dynamics, there are equally many male and female infectives at any given time. Note that this need not remain true once the disease is too prevalent for the linearized dynamics to remain accurate.

Population ecology interpretation Consider a population categorised by two types (i.e., two different birth states, e.g., two different plumage colours). Within each time-step of one year, type 1 individuals produce on average $f_1=1$ offspring of which a proportion $g_1=0.1$ are of type 1 (the rest of type 2) and type 2 produce $f_2=0.5$ offspring of which a proportion $g_2=0.9$ are of type 1. Type 1 individuals (respectively type 2) have a probability $s_1=0.5$ to survive the time-step (respectively $s_2=0.1$). Individual type is preserved by survival, therefore in this model individuals are classified only by type, and age is irrelevant. This yields the following fertility and survival matrices:

$$\mathbf{F}=\begin{bmatrix} f_1 g_1 & f_2 g_2 \\ f_1(1-g_1) & f_2(1-g_2) \end{bmatrix}=\begin{bmatrix} 0.1 & 0.45 \\ 0.9 & 0.05 \end{bmatrix} \quad \text{and} \quad \mathbf{S}=\begin{bmatrix} s_1 & 0 \\ 0 & s_2 \end{bmatrix}=\begin{bmatrix} 0.5 & 0 \\ 0 & 0.1 \end{bmatrix} \tag{a2}$$

The dominant eigenvalue and associated right-eigenvector of the chronological projection matrix $\mathbf{F}+\mathbf{S}$ are

$$\lambda=1.05 \quad \text{and} \quad \boldsymbol{w}=\begin{bmatrix} 0.5 \\ 0.5 \end{bmatrix} \tag{b2}$$

As $\lambda>1$ the population is asymptotically increasing with time, and $\boldsymbol{w}$ indicates that at steady state there are equally many type 1 and type 2 individuals in the population.

Common interpretation (epidemiology and population ecology) From the chronological time projection matrices, we obtain the next-generation matrix (eqn.4), $\mathbf{R}=\mathbf{F}\mathbf{V}^{-1}=\mathbf{F}(\mathbf{I}-\mathbf{S})^{-1}$:

$$\mathbf{R}=\begin{bmatrix} 0.2 & 0.5 \\ 1.8 & 0.056 \end{bmatrix}. \tag{d}$$

From $\mathbf{R}$ we can read off the reproductive numbers of the two types (the column sums of $\mathbf{R}$, eqn. 5):

$$r(1)=2 \quad \text{and} \quad r(2)=0.556. \tag{e}$$

Because $\boldsymbol{w}$ and $\boldsymbol{F}$ are the same for the population biology and epidemiology interpretations of the model, the two have the same relative abundances of newborn/newly-infected at any given time (eq.11):

$$\boldsymbol{w}^*=\begin{bmatrix} 0.3667 \\ 0.6333 \end{bmatrix} \tag{f}$$

There are fewer type 1 than type 2 newborn/newly-infected in any cohort at birth ($\boldsymbol{w}^*$, eq.f), but equally many type 1 and type 2 in the steady-state total populations ($\boldsymbol{w}$, eq.b1 or eq.b2) because the latter recover/die faster than the former.

From these ingredients we can now compute the population Cohort Net Reproductive Rate

(eq.12):

$$R_0^{(c)} = \mathbf{1}^T \mathbf{R} \boldsymbol{w}^* = 1.0852 \tag{g}$$

As there is not exact mixing at birth ($\beta_{1,1} \neq \beta_{1,2}$ and $g_1 \neq g_2$), we expect the Cohort Net Reproductive Rate to differ from R_0. Indeed, the per-generation growth rate (eq.7) is

$$R_0 = \rho(\mathbf{R}) = 1.0792 \tag{h}$$

The difference can be understood by considering the stationary vector of newborn type abundances in a generation:

$$\boldsymbol{w}_g = \begin{bmatrix} 0.3625 \\ 0.6375 \end{bmatrix}. \tag{i}$$

There are relatively more type 1 (and less type 2) newborn in a cohort (eqn. f) than in a generation (eqn. i). This is because type 1 newborn/newly infected have "younger" mothers/infectors: the mothers/infectors are predominantly of type 2, and type 2 individuals survive/remain infectious for less time (see eq.a1 or eq.a2). This difference in generation time, which is not accounted for in $\boldsymbol{w}_g$, implies there is a higher production rate and turnover of type 1 newborn/newly infected in chronological time. In turn, as type 1 individuals have a higher net reproductive rate than type 2 (eq.e), we have $R_0^{(c)} > R_0$.

6.1 Empirical estimation of the classic and the Cohort Net Reproductive Rates from individual trajectories

We simulated, for 50 years, a population corresponding to the population ecology model above, where we assume that reproduction is Bernoulli, i.e. one offspring produced at most per time-step, with initial condition $\boldsymbol{n}_0 = (100,100)^T$, 200 individuals distributed across types according to the stationary distribution $\boldsymbol{w}$ (eqn. (b)). We treat the simulation as a monitored population, where each newborn is captured and marked. Individuals are then re-captured throughout their lifetime so that their fertility trajectories and age at death are known (or can be estimated). In this simulation, a total of 29,683 individuals were tagged, 2,245 of which were still alive by the end of the 50yr study. Cohort number 1 consisted of all individuals born in year $t = 1$, and generation number 1 consisted of all offspring produced by the original population ($\boldsymbol{n}_0$). The maximum individual lifespan was 13 years, with the individual reproducing during its final year (cohort 19, generation 10). We call a cohort or generation *complete* if all members have died by the end of the study.

Estimation of cohort mean lifetime reproduction $R_0^{(c)}$ can only use complete cohorts. Near the end of the simulation unusually short-duration cohorts would be more likely to be complete, which (because of the correlation between age at death and LRS) would induce a negative bias in the estimate of $R_0^{(c)}$. To reduce this bias, all cohorts following the first incomplete cohort were discarded. For this simulation, this implies that cohorts 1 to 41 can be used (blue in fig.1a).

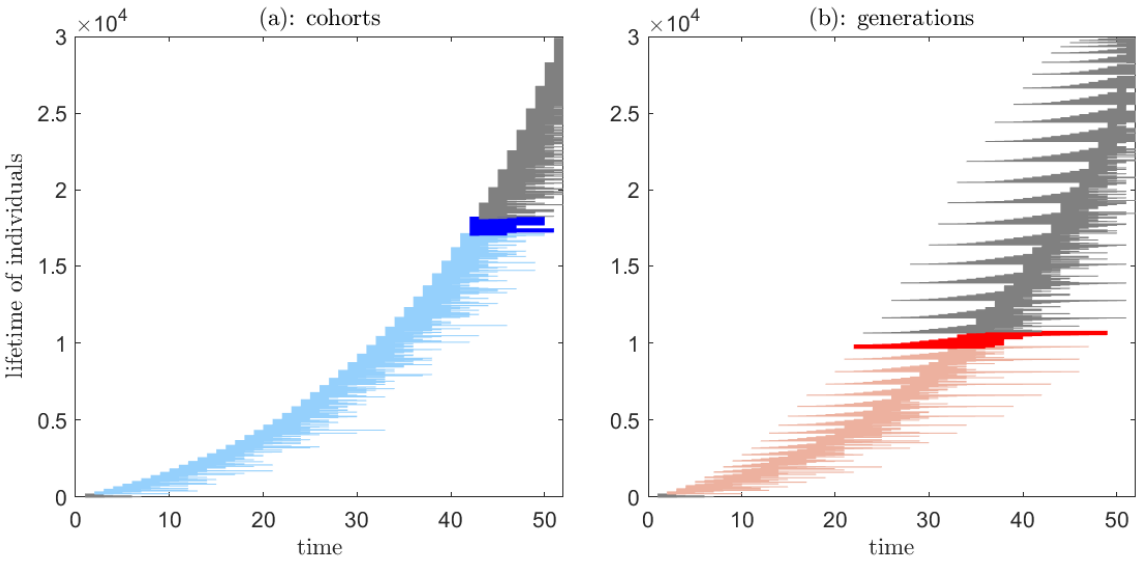

Figure 1: Lifespans (segments from birth until death or until the end of the study) of the 29,683 tagged individuals of the simulated study population, sorted by (a) cohorts and (b) generations. The last complete cohort (resp. generation) is highlighted in dark blue (resp. red) and its predecessors in light blue (resp. red). Lifetime reproductive output of the individuals in these cohorts (resp. generations) can be used to assess empirically $R_0^{(c)}$ (resp., R_0).

Cohort 41 is highlighted in dark blue. Cohorts 1 to 41 altogether included 17,864 individuals, with mean LRS calculated as 1.0832 which is quite close to the true $R_0^{(c)} = 1.0852$.

Estimating R_0 empirically requires knowing the generation of each individual. Provided the investigators can identify the mother of each newborn, they can empirically estimate R_0 by considering all successive completed generations. In this simulation, generation 20 was the last complete generation (dark red on fig.1b). Because individuals in later generations are born across wider and wider spans of time (fig.1b), the complete generations only included 10,451 individuals. This yields a mean LRS across complete generations of 1.0707, not far from the true value ($R_0 = 1.0792$). The last complete generation (generation 20) contains individuals from cohorts 21 to 48, many with non-overlapping lifespans.

Thus, one important difference between the two empirical estimations is sample size: from exactly the same data set, the sample size for estimating R_0 was 41% smaller. The births comprising each successive generation occur over a wider and wider span of time, with the variance in birth dates increasing approximately linearly as a function of generation number, whereas the births in every cohort occur in a single year. Therefore, many more generations than cohorts are incomplete at the end of the study. Note, in this example the organisms have very short reproductive lifespans and therefore low variance in the age at which they have offspring; the broadening range of birth years for successive generations would be much more pronounced in organisms with a longer reproductive life span.

The second difference is, that if the population has existed for some time, all observed

cohorts are an approximation to $\boldsymbol{w}^*$, while the first "generation" is defined arbitrarily. Successive generations come to approximate $\boldsymbol{w}_g$ (under the mild extra condition, beyond the irreducibility already assumed, given in SM II) but the time this takes depends on the damping ratio of the next generation matrix, so arguably data from the first few generations should be discarded, along with data from the many generations that are not complete at the end of the study.

Discussion

The concept of net reproductive rate has long united demography, ecology, and epidemiology. By introducing the Cohort Net Reproductive Rate $R_0^{(c)}$, we resolve a long-standing disconnect between theoretical formulations and empirically directly measurable individual trajectory quantities. $R_0^{(c)}$ is an expectation over individuals born or infected at the same time, while R_0 considers newborn or newly infected individuals of the same generation. More fundamentally, the two differ in the time axis along which reproduction or infection is tracked: chronological time for $R_0^{(c)}$, generation time for R_0. While $R_0^{(c)}$ is relatively easy to assess empirically, doing the same for R_0 requires the (rare) knowledge of the pedigree/transmission tree of the population/epidemic, together with an arbitrary definition of an initial generation which implies discarding much hard-won data from the period before the generation structure has converged to its stable distribution $\boldsymbol{w}_g$. In that sense, the Cohort Net Reproductive Rate $R_0^{(c)}$ improves empirical tractability without losing theoretical rigor.

Our results show that $R_0^{(c)}$ retains the same invasion threshold property as R_0 – populations and epidemics expand when $R_0^{(c)} > 1$ and decrease when $R_0^{(c)} < 1$. In multi-type systems where offspring type is not independent of parent state (so $\mathbf{F}$ has rank > 1) – that is, where there may be type inheritance (the correlation between parental and offspring type may not be zero) – the difference between generation-based and cohort-based weighting encapsulates the effects of variation, across types, in expected lifetime reproductive success and in the distribution of parental age at birth-giving (whose expectation is generation time). The introduction of the Cohort Net Reproductive Rate will allow users of the ever-increasing proportion of such models, both in ecology and epidemiology (see, e.g., Laha & Majumdar, 2023; Pellis *et al.*, 2012; Pigeon *et al.*, 2021; Plard *et al.*, 2018; Zhukova *et al.*, 2023) to produce estimates for the mean LRS observed in the population across complete cohorts.

Conversely, we have shown that a large fraction of models, both theoretical and data-driven, in population ecology and epidemiology, have $R_0 = R_0^{(c)}$ as a result of easily identified structural properties of the model. This frequent equivalence may explain the late introduction of $R_0^{(c)}$ via this manuscript (though we note that previous studies have included formulations similar to $R_0^{(c)}$ (see SM V) or even identical (as a side remark in Cochran & Ellner (1992)). The equivalence has several benefits for users of models where $R_0 = R_0^{(c)}$. First, it provides an alternative and in many cases simpler way to calculate R_0, as it does not require eigenvalue computation. For example, Poulton & Ellner (2025) found that only one of two theoretically permissible definitions of $\mathbf{F}$ and $\mathbf{V}$ (depending on whether shedding into the environment is considered a new infection or a transfer)

produced a next generation matrix from which R_0 was easy to calculate. But the (identical) formula for $R_0^{(c)}$ is easily derived without recourse even to pencil and paper, as the appropriately weighted sum of $r(j)$ values. For matrix population models, the methods and software now available to calculate $r(j)$ values (i.e., mean LRO conditional on state at birth) for arbitrary transition matrix structures Snyder *et al.* (2026); van Daalen & Caswell (2017) become a tool for calculating R_0 for high-dimensional matrices (iteration matrices for "megamatrix" Integral Projection Models, or matrix models with multiply cross-classified individuals) where matrix inversion may be numerically challenging on a personal computer or laptop, while matrix multiplication is still feasible.

Second, it clarifies the biological meaning of R_0. In cases of equality, R_0 does not only represent the (abstract) notion of generation-to-generation population growth rate, or the average reproductive success across the (abstract) founders of a generation. It also has the much clearer interpretation as the average lifetime reproductive success of the individuals born at one time, once the population has reached stable population structure.

Conceptually, $R_0^{(c)}$ extends the reach of reproductive-number theory. It provides a common language for linking individual life trajectories, population and epidemic dynamics, and evolutionary demography using quantities that can be assessed in the field from individual life-history data. Practically, it offers a route for integrating demographic monitoring and epidemiological surveillance within a unified framework grounded in observables. Beyond its immediate analytical advantages, the Cohort Net Reproductive Rate encourages a re-examination of how we define "typical individuals" and how heterogeneity shapes persistence and invasion. It invites a synthesis between theoretical demography and empirical ecology similar to that achieved between epidemiological theory and public-health data during the past decades. We expect this framework to stimulate new applications – from quantifying individual and population fitness in structured populations to estimating transmission potential in emerging pathogens – thereby reaffirming reproductive numbers as both theoretical cornerstones and empirically grounded measures of biological success.

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Supplementary Materials

I Linearisation at the disease free equilibrium

We follow closely van den Driessche & Watmough (2008), considering epidemic models that can be written in the form

$$\begin{aligned} \frac{dn}{dt} &= \mathcal{F}(n,y) - \mathcal{V}(n,y) \\ \frac{dy}{dt} &= \mathcal{G}(n,y) \end{aligned} \tag{19}$$

where n is the state vector for infected states, and y is the state vector of uninfected states. $\mathcal{F}$ contains the rates at which each infected compartment is increased by secondary infections, and $\mathcal{V}$ contains the rates of disease progression (transfer to another infected compartment), recovery, and death. There is often some flexibility about which processes go into $\mathcal{F}$ versus $\mathcal{V}$ — for example, in a model for an environmentally transmitted disease, shedding of pathogens into the environment can be classified as a new infection, or as progression. These choices result in different definitions of R_0, but (subject to satisfying the necessary general assumptions) all will have the threshold property.

We assume that the disease-free manifold $n = 0$ is invariant, and that the infection-free subsystem $\frac{dy}{dt} = \mathcal{G}(0,y)$ has a globally stable disease-free equilibrium (DFE) y^*. The transmission matrices $\mathbf{F}$ and $\mathbf{V}$ in the definition of R_0 are the Jacobian matrices of $\mathcal{F}(n,y^*)$ and $\mathcal{V}(n,y^*)$ with respect to the infected states n. These define the *linearized* dynamics of the infected compartments near the DFE, given as eqn. (1b) in the main text,

$$\frac{dn}{dt} = (\mathbf{F} - \mathbf{V})n(t). \tag{20}$$

For the model of an initial disease outbreak presented verbally in main text section 6, the

simplest complete model following the biological description is the SIS model

$$\begin{aligned}\frac{dS_i}{dt} &= -S_i\sum_{j=1}^{2}\beta_{ij}I_j + v_iI_i \quad i=1,2\\ \frac{dI_i}{dt} &= S_i\sum_{j=1}^{2}\beta_{ij}I_j - v_iI_i \quad i=1,2\end{aligned} \tag{21}$$

where $S_i(t)+I_i(t)\equiv N_i$, a constant total population size for each kind of susceptible/infective. On each line, the first term is infection and the second is transition. The Jacobian of the infectious subsystem (I_1,I_2) at the DFE can be written as $\mathbf{F}-\mathbf{V}$ for

$$\mathbf{F}=\begin{bmatrix}N_1\beta_{1,1} & N_1\beta_{1,2}\\ N_2\beta_{2,1} & N_2\beta_{2,2}\end{bmatrix} \quad \text{and } \mathbf{V}=\begin{bmatrix}v_1 & 0\\ 0 & v_2\end{bmatrix}. \tag{22}$$

II Irreducibility and aperiodicity considerations

Discrete time. We assume that the projection matrix $\mathbf{A}=\mathbf{F}+\mathbf{S}$ is primitive (irreducible, aperiodic) and some power of $\mathbf{A}$ has all positive entries. The Perron-Frobenius Theorem then guarantees the existence of a unique dominant eigenvalue λ that is real and positive, and corresponding real and positive left and right eigenvectors v and w, respectively. The theory also applies to IPMs under analogous irreducibility and positivity assumptions (Ellner *et al.*, 2016a).

Continuous time. The threshold condition for R_0 in compartmental epidemic models requires some technical assumptions about $\mathbf{F}$ and $\mathbf{V}$ (Diekmann *et al.*, 2010, Appendix A). $\mathbf{F}$ must be non-negative (as we have assumed in the main text). $-\mathbf{V}$ (which is denoted $\mathbf{\Sigma}$ in Diekmann *et al.* (2010)) must be *positive off-diagonal*, meaning (confusingly) that all off-diagonal elements of $-\mathbf{V}$ must be non-negative. Equivalently, all off-diagonal elements of $\mathbf{V}$ must be non-positive. If the Jacobian at the DFE has been partitioned properly into infections (in $\mathbf{F}$) and transitions (in $\mathbf{V}$) then the off-diagonal elements of $-\mathbf{V}$ represent rates at which individuals are transferring into a particular compartment (from another particular compartment) and therefore will be non-negative. Thus the positive off diagonal assumption is a requirement that $\mathbf{F}$ and $\mathbf{V}$ have their desired biological meanings. Additionally, $\mathbf{V}^{-1}$ must be positive, meaning that all entries are non-negative (recall, we have already assumed that $\mathbf{V}$ is invertible). Under our assumption that $-\mathbf{V}$ is positive off-diagonal, the two additional properties that we require (invertibility, positivity) will hold so long as all eigenvalues of $-\mathbf{V}$ have negative real part (Diekmann *et al.*, 2010, Appendix A). $-\mathbf{V}$ describes the epidemic dynamics near the DFE in the absence of any transmission (if $\mathbf{F}$ and $\mathbf{V}$ have been properly specified). In any biologically reasonable model, eliminating transmission will invariably lead to exponentially fast decline of disease prevalence, implying that $-\mathbf{V}$ cannot have an eigenvalue with zero

or positive real part. So although the necessary technical assumptions on $\mathbf{V}$ may seem formidable and abstract, they actually correspond to requiring that the model is biologically sensible and the DFE Jacobian has been split into two pieces that both have their correct biological meanings.

These assumptions on $\mathbf{V}$ also have important implications for the eigenvalues and eigenvectors of $\mathbf{F}-\mathbf{V}$. Under our assumptions, all off-diagonal entries of $\mathbf{F}-\mathbf{V}$ are non-negative. We assume that adding a sufficiently large multiple of the identity matrix to $\mathbf{F}-\mathbf{V}$ produces a matrix satisfying the assumptions of the Perron-Frobenius Theorem, and therefore having a positive real eigenvalue such that all eigenvalues have smaller absolute value and therefore smaller real part (note, this is equivalent to assuming that replacing all diagonal elements of $\mathbf{F}-\mathbf{V}$ with 1's yields a matrix satisfying the assumptions of the Perron-Frobenius Theorem). As addition of $k\mathbf{I}$ does not alter eigenvectors, and shifts all eigenvalues horizontally in the complex plane, $\mathbf{F}-\mathbf{V}$ thus has a unique dominant real eigenvalue such that no other eigenvalue has a larger real part. Corresponding to the dominant eigenvalue is a strictly positive eigenvector, and there are no other positive eigenvectors. These properties correspond to the Perron-Frobenius properties of $\mathbf{F}+\mathbf{S}$ in the discrete time case, except that the dominant eigenvalue may be negative (i.e., $r<0$ for an epidemic that does not spread).

Irreducibility of the type-restricted next-generation matrix. Recall that a state i is a *type* if row i of $\mathbf{F}$ is not identically zero, and let $\mathcal{T}$ denote the set of type states. Let $\mathbf{R}^\circ$ denote the principal submatrix of $\mathbf{R}$ (discrete time: $\mathbf{R}=\mathbf{F}(\mathbf{I}-\mathbf{S})^{-1}$; continuous time: $\mathbf{R}=\mathbf{F}\mathbf{V}^{-1}$) with both rows and columns restricted to $\mathcal{T}$. Since $F(i,j)=0$ for every $i\notin\mathcal{T}$ and every j, every row of $\mathbf{R}$ indexed by a non-type state is identically zero as well; hence $\mathbf{R}$ itself is reducible whenever $\mathcal{T}$ is a strict subset of all states, and irreducibility can only be a property of the restriction $\mathbf{R}^\circ$. After permuting states so that types come first, $\mathbf{R}$ therefore takes the block form

$$\mathbf{R}=\begin{bmatrix}\mathbf{R}^\circ & * \\ 0 & 0\end{bmatrix}, \tag{23}$$

so its eigenvalues are exactly those of $\mathbf{R}^\circ$ together with a set of zero eigenvalues. Hence $\rho(\mathbf{R})=\rho(\mathbf{R}^\circ)$, and since $\mathbf{R}^\circ$ is irreducible and non-negative (below), $R_0=\rho(\mathbf{R}^\circ)>0$: irreducibility of $\mathbf{R}^\circ$ is what guarantees R_0 is not merely the spectral radius of $\mathbf{R}$ in the abstract, but its simple, positive, uniquely-attained dominant eigenvalue.

Discrete time. Under our standing assumption that $\mathbf{A}=\mathbf{F}+\mathbf{S}$ is irreducible, $\mathbf{R}^\circ$ is itself irreducible. This is a special case of a general decomposition result for $\mathbf{F}(\mathbf{I}-\mathbf{S})^{-1}$ due to Li & Schneider (2002, Prop. 4.1 and Thm. 4.2): after a suitable permutation of states, $\mathbf{R}$ takes the block form (23) where $\mathbf{R}^\circ$ is irreducible and the zero rows correspond exactly to the non-type states.

Continuous time. We are not aware of a published analogue of this result for $\mathbf{F}\mathbf{V}^{-1}$, so we give a direct proof: Since adding $k\mathbf{I}$ for any k changes only the diagonal entries of $\mathbf{F}-\mathbf{V}$, leaving its off-diagonal zero/nonzero pattern – and hence its reducibility or irreducibility – unchanged, the assumption above that $(\mathbf{F}-\mathbf{V})+k\mathbf{I}$ is irreducible for sufficiently large k is equivalent to $\mathbf{F}-\mathbf{V}$ itself

being irreducible. That is, the associated digraph (directed graph) – with an edge $j' \to k'$ whenever either $F(k',j') > 0$ or the off-diagonal entry of $-\mathbf{V}$ at (k',j') is positive – is strongly connected. Let $i,j \in \mathcal{T}$. Since i is a type, there is some state k with $F(i,k) > 0$. Since $\mathbf{F}-\mathbf{V}$ is irreducible, there is a path from j to k in its digraph. Append the edge $k \to i$ (via $F(i,k) > 0$). The resulting path from j to i is a mix of edges in the digraph of $-\mathbf{V}$, and edges in the digraph of $\mathbf{F}$. Cut this path at the end of every F-edge. Each resulting segment consists of some number of transition ($-\mathbf{V}$ off-diagonal) edges followed by one F-edge. Because $\mathbf{V}^{-1} = \int_0^\infty e^{-\mathbf{V}t} dt$, and for a matrix with non-negative off-diagonal entries $e^{-\mathbf{V}t}(k',j') > 0$ for some $t > 0$ if and only if k' is reachable from j' in the off-diagonal digraph of $-\mathbf{V}$, each such segment corresponds to a positive entry of $\mathbf{R} = \mathbf{F}\mathbf{V}^{-1}$.

Chaining these positive entries across segments, exactly as in the discrete-time case, shows $(\mathbf{R}^\circ)^m(i,j) > 0$ for some finite m, establishing irreducibility of $\mathbf{R}^\circ$■.

Primitivity of the type-restricted next-generation matrix. Irreducibility of $\mathbf{R}^\circ$ guarantees a unique dominant eigenvalue R_0 with a positive eigenvector, but does not by itself guarantee that successive generations *converge* to this eigenvector (rather than merely remaining bounded relative to it), since an irreducible matrix may still be periodic – as an explicit example, if $\mathbf{S} = s\mathbf{I}$ and $\mathbf{F} = \left[\begin{smallmatrix} 0 & f \\ f & 0 \end{smallmatrix}\right]$, then $\mathbf{A} = \mathbf{F}+\mathbf{S}$ is primitive but $\mathbf{R} = \mathbf{F}(\mathbf{I}-\mathbf{S})^{-1}$ is irreducible yet has two eigenvalues of equal modulus, and generation-structured abundances oscillate indefinitely rather than converging to $\boldsymbol{w}_g$. Since $\mathbf{R}^\circ$ is non-negative, primitivity follows from irreducibility together with one further condition on the model ingredients: that some type i have positive probability or rate of eventually giving rise to a new individual of its own type again, whether directly or via some chain of survival/progression –

There is a type i, and a state k with $F(i,k) > 0$, such that k is reachable from i by survival alone, (24)

where "reachable by survival alone" means via $\mathbf{S}$ in discrete time (allowing $k = i$, so that $S^a(k,i) > 0$ for some $a \geq 0$) or via the off-diagonal entries of $-\mathbf{V}$ in continuous time (again allowing $k = i$). Equivalently, condition (24) says $R^\circ(i,i) > 0$ for some type i, since $R^\circ(i,i) = \sum_{a \geq 0}(\mathbf{F}\mathbf{S}^a)(i,i)$ (discrete) or $R^\circ(i,i) = [\mathbf{F}\mathbf{V}^{-1}](i,i)$ (continuous) is exactly the sum, over all such reachable k, of the corresponding contributions. Under condition (24), $\mathbf{R}^\circ$ is primitive, since an irreducible non-negative matrix with at least one positive diagonal entry is automatically primitive (a self-loop, combined with strong connectivity, forces every cycle length's greatest common divisor to be 1), and generation-structured abundances converge to $\boldsymbol{w}_g$ as claimed in sec. 6, at a rate governed by the subdominant eigenvalue of $\mathbf{R}^\circ$ (the damping ratio). Because every row of $\mathbf{R}$ indexed by a non-type state is zero, $\boldsymbol{g}_T$ has no support on non-type states for any $T \geq 1$ regardless of $\boldsymbol{g}_0$; consequently the dynamics of $\boldsymbol{g}_T$ for $T \geq 1$ are governed entirely by $\mathbf{R}^\circ$, and primitivity of $\mathbf{R}^\circ$ is therefore sufficient (not merely necessary) for the full generation-structured vector $\boldsymbol{g}_T$ – not just its restriction to types – to converge to $\boldsymbol{w}_g$.

Consequence for $\boldsymbol{w}_g$. Since $\mathbf{R}^\circ$ is irreducible, the Perron-Frobenius Theorem gives it a

strictly positive eigenvector on $\mathcal{T}$, unique up to scaling; extended by zero to the non-type states, this is exactly the vector $\boldsymbol{w}_g$ of the main text (eq. 8), which is accordingly zero on non-type states and strictly positive on every type state.

III A two-latent-type epidemic model with a single infective compartment

Diekmann *et al.* (2010) consider a compartmental epidemic model with two latent types E_1,E_2 (their equations 2.1–2.5), for which the linearized infection subsystem is

$$\begin{bmatrix} \dfrac{dE_1}{dt} \\ \dfrac{dE_2}{dt} \\ \dfrac{dI}{dt} \end{bmatrix} = \begin{bmatrix} p\beta I - (\nu_1+\mu)E_1 \\ (1-p)\beta I - (\nu_2+\mu)E_2 \\ \nu_1 E_1 + \nu_2 E_2 - (\gamma+\mu)I \end{bmatrix} = (\mathbf{F}-\mathbf{V}) \begin{bmatrix} E_1 \\ E_2 \\ I \end{bmatrix} \tag{25}$$

$$\text{where} \qquad \mathbf{F} = \begin{bmatrix} 0 & 0 & p\beta \\ 0 & 0 & (1-p)\beta \\ 0 & 0 & 0 \end{bmatrix} \qquad \mathbf{V} = \begin{bmatrix} \nu_1+\mu & 0 & 0 \\ 0 & \nu_2+\mu & 0 \\ -\nu_1 & -\nu_2 & \gamma+\mu \end{bmatrix}. \tag{26}$$

Diekmann *et al.* (2010) show how several related "next generation" matrices can be constructed, having dimensions three, two, and one, with R_0 as their dominant eigenvalue. New infections are produced in proportions $\boldsymbol{w}^* = (p, 1-p, 0)^T$, and from the usual considerations for compartmental epidemic models (mean residence times and infectivities for each compartment),

$$r(j) = \left(\frac{\nu_j}{\nu_j+\mu}\right)\frac{\beta}{\gamma+\mu}, \qquad j = 1,2.$$

Consequently

$$R_0 = R_0^{(c)} = \left(\frac{p\nu_1}{\nu_1+\mu} + \frac{(1-p)\nu_2}{\nu_2+\mu}\right)\frac{\beta}{\gamma+\mu}, \tag{27}$$

exactly as in Diekmann *et al.* (2010, eqn. 2.10).

IV No variation in parental age distribution across newborn types implies $R_0^{(c)} = R_0$

Define $p_{O,age}(a \mid i)$ as the probability that the mother of a randomly chosen type-i newborn (in any cohort of a stationary population) was aged a, and $p_{age}(a)$ as the probability that the mother of a newborn chosen at random (in a cohort) was aged a. By the arguments used to

derive eqn. (11) in Ellner (2018), but considering only type-i offspring, we get

$$\begin{cases} p_{O,age}(a\,|\,i) = \frac{1}{\lambda^a w^*(i)} \boldsymbol{e}_i^T \mathbf{F}\mathbf{S}^{a-1}\boldsymbol{w}^* \\ p_{age}(a) = \sum_i w^*(i) p_{O,age}(a\,|\,i) = \frac{1}{\lambda^a}\mathbf{1}^T\mathbf{F}\mathbf{S}^{a-1}\boldsymbol{w}^* \end{cases} \tag{28}$$

where $\boldsymbol{e}_i$ is the i^{th} canonical basis vector in which the i^{th} coordinate equals one and all others are zero. The generation time at the population level is the expectation of this distribution:

$$T = \sum_{a\geq 1} a p_{age}(a) = \frac{1}{\lambda}\mathbf{1}^T\mathbf{F}\left(\mathbf{I}-\frac{\mathbf{S}}{\lambda}\right)^{-2}\boldsymbol{w}^*, \tag{29}$$

which corresponds to eq.(12) in Ellner (2018). Similarly, the generation time of type-i offspring is $T_O(i) = \sum_a a p_{O,age}(a\,|\,i)$.
If the distribution of parental age does not depend on the type of the newborn, that is if

$$\forall i,a: \quad p_{O,age}(a\,|\,i) = p_{age}(a), \tag{30}$$

we claim that $R_0^{(c)} = R_0$. (Note, the $\forall$ symbol is read as "for all").
Note first that condition (30) implies $T_O(i) = T$ for every type i, that is, that there is no variation in generation time across types, but the converse is not true: equality of two distributions requires equality of all moments, not merely their means.
Proof: Condition (30), written out using (28), is

$$\forall i,a: \quad \frac{1}{\lambda^a w^*(i)}\boldsymbol{e}_i^T\mathbf{F}\mathbf{S}^{a-1}\boldsymbol{w}^* = \frac{1}{\lambda^a}\mathbf{1}^T\mathbf{F}\mathbf{S}^{a-1}\boldsymbol{w}^*, \tag{31}$$

and since the factor λ^{-a} cancels, this is equivalent to

$$\forall i,a: \quad \boldsymbol{e}_i^T\mathbf{F}\mathbf{S}^{a-1}\boldsymbol{w}^* = w^*(i)\mathbf{1}^T\mathbf{F}\mathbf{S}^{a-1}\boldsymbol{w}^*. \tag{32}$$

Summing over $a \geq 1$ and using $\sum_{a\geq 1}\mathbf{S}^{a-1} = (\mathbf{I}-\mathbf{S})^{-1}$,

$$\forall i: \quad \boldsymbol{e}_i^T\mathbf{F}(\mathbf{I}-\mathbf{S})^{-1}\boldsymbol{w}^* = w^*(i)\mathbf{1}^T\mathbf{F}(\mathbf{I}-\mathbf{S})^{-1}\boldsymbol{w}^*, \tag{33}$$

that is, recalling $\mathbf{R} = \mathbf{F}(\mathbf{I}-\mathbf{S})^{-1}$ (eq. 4a),

$$\forall i: \quad w^*(i) = \frac{\boldsymbol{e}_i^T\mathbf{R}\boldsymbol{w}^*}{\mathbf{1}^T\mathbf{R}\boldsymbol{w}^*} = \frac{\boldsymbol{e}_i^T\mathbf{R}\boldsymbol{w}^*}{R_0^{(c)}}. \tag{34}$$

implying that

$$\boldsymbol{e}_i^T\mathbf{R}\boldsymbol{w}^* = R_0^{(c)} w^*(i). \tag{35}$$

This last equation says, entry by entry, that $\mathbf{R}\boldsymbol{w}^* = R_0^{(c)}\boldsymbol{w}^*$, so $\boldsymbol{w}^*$ is an eigenvector of $\mathbf{R}$ with eigenvalue $R_0^{(c)}$. Since row i of $\mathbf{F}$ (and hence of $\mathbf{R}$) is identically zero for every non-type state i, $w^*(i) = 0$ for every non-type i; and since $\boldsymbol{w} > 0$ and every type i has some j with $F(i,j) > 0$, $w^*(i) > 0$ for every type i. Restricted to type states, the eigenvector equation becomes $\mathbf{R}^\circ\left(\boldsymbol{w}^*|_\mathcal{T}\right) = R_0^{(c)}\left(\boldsymbol{w}^*|_\mathcal{T}\right)$ (SM II), with $\boldsymbol{w}^*|_\mathcal{T}$ strictly positive. Since $\mathbf{R}^\circ$ is irreducible (SM II), the Perron-Frobenius Theorem guarantees its positive eigenvector is unique up to scaling and belongs to the dominant eigenvalue R_0. Hence $\boldsymbol{w}^* = \boldsymbol{w}_g$ and $R_0^{(c)} = R_0$. ■

V Historical antecedents of $R_0^{(c)}$

The Cohort Net Reproductive Rate $R_0^{(c)}$, has been "hiding in plain sight" for many years. Specifically, a special case occurs in passing in the paper by Diekmann *et al.* (1990) where the Generation Net Reproductive Rate R_0 was first introduced in full generality for epidemic models, but it was not given a name, nor were its general properties identified. Please be patient, as it takes a while to describe the Diekmann *et al.* (1990) general model. Having done that, we explain the special case considered by Diekmann *et al.* (1990), then present the general formula for $R_0^{(c)}$ in their model, and show that it satisfies the Threshold Criterion.

Diekmann *et al.* (1990) assumed that individuals are classified by *state* $\xi \in \Omega$ (called the h state variable, for "heterogeneity") and by time since infection, τ. $S(\xi)$ denotes the steady-state h state distribution of susceptibles in the absence of disease, i.e. $\int_X S(\xi)d\xi$ is the number of susceptibles with $\xi \in X$. $A(\tau,\xi,\eta)$ is defined to be the expected infectivity of an individual which was infected τ units of time ago, while having h-state η, towards a susceptible which has h-state ξ. The expected number of new infections produced by an individual in an otherwise all-susceptible population, during its infective lifetime, for an individual that was itself infected in h-state η is then

$$\int_\Omega S(\xi)\int_0^\infty A(\tau,\xi,\eta)d\tau d\xi. \tag{36}$$

The nature of the space Ω and the meaning of $d\xi$ are not specified precisely by Diekmann *et al.* (1990). For the definition and properties of R_0 it is sufficient for Ω to be a compact metric space equipped with a finite measure on the Borel $\sigma-$field, with $d\xi, d\eta$ denoting integration with respect to that measure. A is assumed to be continuous and bounded on $[0,\infty)\times\Omega\times\Omega$, and the integral operator with kernel $A_0(\xi,\eta) = \int_0^\infty A(t,\xi,\eta)dt$ satisfies the assumptions in Appendix C of Ellner & Rees (2006) that guarantee stable population growth in general Integral Projection Models.

The next generation operator $K(S)$ maps the h-state distribution ϕ of infectives in one generation to the h-state distribution of infectives in the next, assuming that the (tacitly linearized) dynamics (36) of a rare infection continues to hold:

$$K(S)\phi = S(\xi)\int_\Omega\int_0^\infty A(\tau,\xi,\eta)d\tau\phi(\eta)d\eta. \tag{37}$$

R_0 is defined to be the spectral radius of $K(S)$, $\lim_{m\to\infty}||K(S)^m||^{1/m}$ Under appropriate (but unstated) compactness, positivity, and irreducibility conditions, R_0 equals the dominant eigenvalue of $K(S)$, and is shown to satisfy the Threshold Criterion.

Diekmann *et al.* (1990) then consider how R_0 can be computed explicitly in some special cases, beginning with the case where $K(S)$ has a one-dimensional range, that is, "the distribution (over the h-state space Ω) of the "offspring" (i.e. the ones who become infected) is independent of the state of the "parent" (i.e. the one who transmits the infection)" (Diekmann *et al.*, 1990, p.369) Mathematically, it is expressed as

$$\int_0^\infty A(\tau,\xi,\eta)d\tau = a(\xi)b(\eta). \tag{38}$$

The definition of a and b is ambiguous, as we can multiply a by any constant and divide b by the same, so we impose the scaling that $\int_\Omega S(\xi)a(\xi)=1$. $S(\xi)a(\xi)$ is then the probability distribution of h-state at infection, for all infectees of one infector, and $b(\eta)$ is the total number of infectees for an individual infected in h-state η. Regardless of the h-state composition of new infectees in generation n, Sa is the state distribution of new infectees in generation $n+1$ and therefore the only eigenvector of $K(S)$, with eigenvalue

$$R_0 = \int_\Omega S(\eta)a(\eta)b(\eta)d\eta. \tag{39}$$

Assuming that F has one-dimensional range (i.e., rank one) is stronger than assuming that $K(S)$ has one-dimensional range, because (38) is about the total number of new infections over one infective lifetime, rather than the new infections produced at any one time by an infective. However, Diekmann *et al.* (1990) also mention the stronger assumption

$$A(\tau,\xi,\eta) = a(\xi)B(\tau,\eta), \tag{40}$$

which results in eqn. (38) holding with $b(\eta) = \int_0^\infty B(\tau,\eta)d\tau$. Assumption (40) is the analog, for the Diekmann *et al.* (1990) model, of assuming in our models that the $\mathbf{F}$ matrix has rank one. When (40) holds, Sa is also the state distribution of new infectees at any time, as all infectors have a as their frequency distribution of infectees at any time. Consequently, the formula above for R_0 is also the formula for the cohort Net Reproductive Rate $R_0^{(c)}$ in this model. In summary, if the transmission function A has rank one at all times (i.e., eqn. (40)), then $R_0 = R_0^{(c)}$ for the Diekmann *et al.* (1990) model.

It is also possible to define $R_0^{(c)}$ in general for the Diekmann *et al.* (1990) model, and once that is accomplished, it is easy to show that $R_0^{(c)}$ satisfies the Threshold Criterion. To simplify

notation slightly, define

$$A_r(\xi,\eta)=\int_0^\infty e^{-r\tau}A(\tau,\xi,\eta)d\tau \tag{41}$$

so that the next generation operator K (omitting (S) for simplicity) is

$$K\phi(\eta)=\int_\Omega S(\xi)A_0(\xi,\eta)\phi(\eta)d\eta. \tag{42}$$

As in the main text here, let $\boldsymbol{w}_g$ denote the eigenfunction corresponding to the dominant eigenvalue R_0, normalized so $\int_\Omega \boldsymbol{w}_g(\eta)d\eta=1$,

$$R_0\boldsymbol{w}_g(\eta)=\int_\Omega S(\xi)A_0(\xi,\eta)\boldsymbol{w}_g(\eta)d\eta. \tag{43}$$

The infection dynamics in chronological time are given by eqn.(2.4) in Diekmann *et al.* (1990),

$$i(t,\xi)=\int_\Omega\int_0^\infty S(\xi)A(\tau,\xi,\eta)i(t-\tau,\eta)d\tau d\eta \tag{44}$$

where $i(t,\xi)$ is the rate of new state-ξ cases at time t (individuals/time) — not the number of infectives. The low-density (linearized) exponential increase of the population is characterized by the dominant eigenvalue r and corresponding eigenvector ψ, such that $\psi(\xi)e^{rt}$ is a solution of eqn. (44); existence and uniqueness of this pair is asserted by Diekmann *et al.* (1990) under positivity and irreducibility assumptions for which they refer the reader to *Banach Lattices and Positive Operators* by H.H. Schaeffer (Springer, 1960). That is,

$$\psi(\xi)=\int_\Omega S(\xi)A_r(\xi,\eta)\psi(\eta)d\eta, \tag{45}$$

with ψ normalized to integrate to 1 over Ω (like $\boldsymbol{w}_g$). ψ is the steady-state distribution of new infections, and therefore corresponds to $\boldsymbol{w}^*$ in the main text, not to $\boldsymbol{w}$. Consequently (using the fact that ψ is an eigenfunction)

$$\begin{aligned}
R_0^{(c)} &= \iint_{\Omega\times\Omega} S(\xi)A_0(\xi,\eta)\psi(\eta)d\eta d\xi \\
&= \iint_{\Omega\times\Omega} S(\xi)A_r(\xi,\eta)\psi(\eta)d\eta d\xi + \iint_{\Omega\times\Omega} S(\xi)(A_0(\xi,\eta)-A_r(\xi,\eta))\psi(\eta)d\eta d\xi \\
&= \int_\Omega \psi(\xi)d\xi + \iint_{\Omega\times\Omega} S(\xi)(A_0(\xi,\eta)-A_r(\xi,\eta))\psi(\eta)d\eta d\xi \\
&= 1+\iint_{\Omega\times\Omega} S(\xi)(A_0(\xi,\eta)-A_r(\xi,\eta))\psi(\eta)d\eta d\xi.
\end{aligned} \tag{46}$$

As the integrand in the last line of eqn. (46) has the same sign as r wherever it is not zero, this

shows that $R_0^{(c)}$ satisfies the Threshold Criterion in the Diekmann *et al.* (1990) general model.

The next near-miss was scored by Cochran & Ellner (1992), attempting to generalize R_0 from age-based models where the formula $R_0 = \int_0^\infty l(a)m(a)da$ holds, to general stage-based matrix projection models. Work on that paper was not completed until after Diekmann *et al.* (1990) was published, but because the title of Diekmann *et al.* (1990) referred strictly to infectious diseases, its relevance for matrix projection models was not recognized.

Cochran & Ellner (1992) suggested several definitions for R_0 for models with multiple newborn types (if there is only one, the classical formula applies with $l(a)$ and $m(a)$ being the age-dependent average vital rates implied by the projection model). With multiple newborn types, they asserted that R_0 should not count newborns directly, but instead weight them with the reproductive value of the state they are born into. As we now know (and readers of Diekmann *et al.* (1990) knew then) this claim is incorrect in that the resulting R_0 would not satisfy the Threshold Criterion. If the reproductive value weighting is omitted, then "Eq. 17 gives $R_O(j)$ in terms of the total number of offspring, without any weighting for relative offspring quality" (Cochran & Ellner, 1992, p. 351), and the resulting formula is the same as our $r(j) = \sum_i R(i,j)$ as per eqn. (5). The following section then deals with the "population average" (Table 2, page 352): $R_0 = \sum_j R_0(j)b_j$, where their definition of $b(j)$, eqn. (19) in Cochran & Ellner (1992), corresponds exactly to the definition of $\boldsymbol{w}^*$ in this paper, eqn. (11). Combining the (deprecated) definition of $R_0(j)$ with the "population average" defined in Table 2, produced $R_0^{(c)}$ exactly as defined in eqn. (12) of this paper.

The current definition of R_0 for matrix population models was established by Cushing & Zhou (1994). They defined $R_0 = \rho((\mathbf{I}-\mathbf{S})^{-1}\mathbf{F})$, which as mentioned in the main text gives exactly the same value of R_0 as the now more common definition $R_0 = \rho(\mathbf{F}(\mathbf{I}-\mathbf{S})^{-1})$. They wrote that this "definition of net reproductive value is, of course, purely mathematical. A natural question to ask is whether in fact it has the correct biological interpretation. That is to say, is $[R_0]$ equal to the expected number of offspring produced by an individual over its life time?". They correctly noted that in a "general structured model, newborns can lie in any of the classes", which renders the individual-level interpretation of R_0 more complicated. They then consider the particular newborn cohort that in our notation corresponds to $\boldsymbol{w}_g$, the steady-state type distribution of a generation. They find that "the average number of offspring over the expected lifetime of a cohort" equals R_0, that is $\mathbf{1}^T\mathbf{R}\boldsymbol{w}_g = R_0$. However, that case is misleading in that $\boldsymbol{w}_g$ does not correspond to the common meaning of "cohort" (i.e., a set of individuals born at one time), because in general $\boldsymbol{w}^* \neq \boldsymbol{w}_g$. Therefore, the expected number of offspring produced by a cohort, R_0^c, is generally not equal to R_0.

The swerve away from $R_0^{(c)}$ and towards the Diekmann *et al.* (1990) and Cushing & Zhou (1994) definition was solidified by Caswell (2001). Caswell noted that for age-structured models R_0 is "the mean number of offspring by which a newborn individual will be replaced by the end of its life, and thus the rate by which the population increases from one generation to the next". For general stage-structured models the "and" no longer holds — those two properties no longer align in general,

so one can have one or the other but not both. Caswell (2001) considered the latter property to be more important, writing (sec. 5.3.4) that the stage-classified net reproductive rate should "give the per-generation growth rate" as well as "relate to" lifetime reproductive output, and should satisfy the Threshold Criterion. He therefore favored the now-standard definition of R_0 via the next-generation matrix. Regarding the suggestions by Cochran & Ellner (1992) based on "adding the different types of offspring", Caswell wrote that "there is no guarantee that such a sum will satisfy either of our desires for what R_0 should do." This is perhaps correct, given that Cochran & Ellner (1992) emphasized "population average" measures in which offspring are weighted by type-specific reproductive value, and that sum does not satisfy the Threshold Criterion (it is also somewhat incoherent, as parents were not weighted by their reproductive values). However, our results show that (once again) absence of evidence is not evidence of absence, and the appropriately weighted (by $\boldsymbol{w}^*$) sum of type-specific expected total offspring production does satisfy the Threshold Criterion.

Caswell (2011) provides a method to compute the type-specific mean and higher moments of lifetime offspring production and also discusses the relationship between R_0 and the vector of type-specific means (denoted ρ_1 there and $\boldsymbol{r}$ here). The discussion in (Caswell, 2011) reflects the belief, only corrected here, that type-specific means do not share with R_0 the ability of "serving as an indicator variable for population growth". For example, (Caswell, 2011), argues that R_0 "is linked to, and calculated from, a model of population dynamics", whereas $\boldsymbol{r}=\rho_1$ "is calculated for a cohort, not a population, and so it has no such linkage". This is confusing, as a population is made up of cohorts, and moreover R_0 and ρ_1 are calculated from exactly the same population model. In view of the results here about $R_0^{(c)}$, Caswell (2011) was attempting to find an explanation for a nonexistent difference, so it is not surprising that this proved to be difficult. Regarding Caswell's closing comment – "Developing connections to population dynamics is an interesting unsolved problem" (Caswell, 2011, p.e20809) – we of course fully agree.