DENSITY-DEPENDENT DISCRETETIME S-I-S EPIDEMIC MODELS

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Infectious Disease Models

- R. M. Anderson and R. M. May (1996)
- N. T. J. Bailey (1975)
- W. O. Kermack and A. G. McKendrick (1932)
- R. Ross (1911)
- etc

Questions

What are the consequences of the interactions between density-dependent birth or recruitment processes and disease-induced mortality in seasonal environments?

• (L. Allen [1994, 2000], Hwang and Kuang [2001, 2003], Castillo-Chavez et al [2005], etc).

Demographic Equation (Constant Environment)

$$N(t+1) = f(N(t)) + \gamma S(t) + \gamma I(t)$$
 (1)

where at generation t,

S(t) = susceptible population;

I(t) = infected population (assumed infectious);

N(t) = S(t) + I(t) = total population;

 $f \in C^1([0,\infty),[0,\infty)$ models birth or recruitment process;

Disease Induced Mortality: $\gamma \geq \gamma$

When $\gamma = \gamma = \gamma$, then (1) becomes

$$N(t+1) = f(N(t)) + \gamma N(t)$$
 (2)

Examples Of Recruitment Functions

1. Constant recruitment function

$$f(N(t)) = \Lambda$$

2. Geometric recruitment function

$$f(N(t)) = \mu N(t)$$

3. Beverton - Holt recruitment function

$$f(N(t)) = \frac{\mu k N(t)}{k + (\mu - 1)N(t)}$$

4. Ricker recruitment function

$$f(N(t)) = N(t)e^{r(1-\frac{N(t)}{k})}$$

Demographic Equation In Seasonal Environments

$$N(t+1) = f(t,N(t)) + \gamma S(t) + \gamma I(t)$$

$$1t \qquad 2t$$

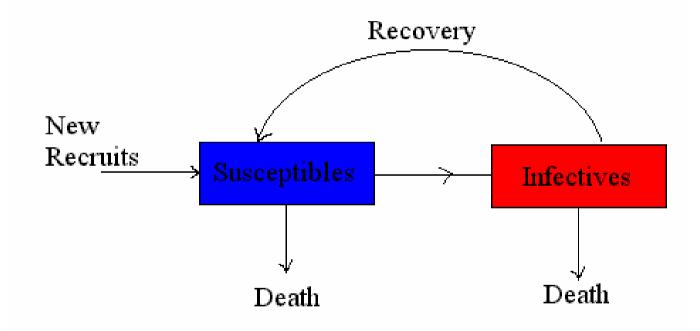
$$where$$

$$f(t+T,N(t)) = f(t,N(t))$$

$$\gamma = \gamma$$

$$i(t+T) \qquad it$$

S-I-S Model



SIS Epidemic Model With Disease-Induced Death

$$S(t+1) = f(N(t)) + \gamma_1 \phi(\alpha \frac{I(t)}{N(t)}) S(t) + \gamma_2 (1-\sigma) I(t)$$

$$I(t+1) = \gamma_1 \left(1 - \phi(\alpha \frac{I(t)}{N(t)})\right) S(t) + \gamma_2 \sigma I(t)$$
(3)

where $0 < \gamma_2 < \gamma_1 < 1, 0 < \sigma < 1 \text{ and } N(t) > 0$.

The escape function $\phi:[0,\infty) \to [0,1]$ is a monotone convex probability function with $\phi(0) = 1$ and $\phi' \le 0$.

Model Assumptions

- Disease increases mortality but does not affect fecundity;
- No acquired immunity;
- No latent period (or latent period is very short);
- Transmission is frequency dependent rather than density dependent.

Deterministic SIS Model

- Our model is a deterministic SIS epidemic model and has no "probability" of transmission. The assumption of deterministic dynamics is valid in a large population, where stochasticity is unimportant.
- This assumption places a constraint on the applicability of our model. For example, stochastic transmission (including a Poisson process) in a small population (close to extinction) would not be described by our model.

Disease Extinction or Persistence

Let
$$R_0 = \frac{-\gamma_1 \alpha \phi'(0)}{1 - \gamma_2 \sigma}$$
.

No disease induced death: Castillo – Chavez and Yakubu [2001]

Theorem (Franke and Yakubu, 2008):

Let
$$N(0) \ge I(0) > 0$$
.

1. If $R_0 < 1$, then $\lim_{t\to\infty} I(t) = 0$. That is, the

disease goes extinct.

2. If $R_0 > 1$ and the total population is uniformly

persistent, then there exists $\eta > 0$

such that $\underline{\lim}_{t\to\infty} I(t) \ge \eta > 0$. That is,

the disease is uniformly persistent.

R_0

- Without disease-induced mortality, it is known that $R_0>1$ implies disease persistence.
- With disease-induced mortality, independent of initial population size of healthy individuals, a tiny number of infectious individuals can drive the total population to extinction.

Auxiliary Functions

$$1. D_i(N) = f(N) + \gamma_i N$$

The total population of new births and survivors;

2.
$$F_N(I) = \gamma_1 (1 - \phi(\alpha \frac{I}{N}))(N - I) + \gamma_2 \sigma I$$

Infective population in the next generation;

3.
$$G_N(I) = f(N) + \gamma_1(N-I) + \gamma_2 I$$

Total population in the next generation;

4.
$$H(N, I) = (G_N(I), F_N(I))$$

Vector of the total and infective populations.

Disease-Free State

If I(t) = 0, then the demographic equation

$$N(t+1) = f(N(t)) + \gamma_1 S(t) + \gamma_2 I(t)$$

becomes

$$S(t+1) = f(S(t)) + \gamma_1 S(t).$$

This reduced equation describes the disease - free state dynamics.

Demographic Basic Reproduction Number

$$R_{D_i} = \frac{f'(0)}{1 - \gamma_i}$$
 whenever $f(0) = 0$.

- 1. Let f(0) = 0. If $R_{D_1} > 1$, then the disease free susceptible population is persistent.
- 2. Let f(0) = 0. If $R_{D_1} < 1$, then $\{(0,0)\}$ is locally asymptotically stable. That is, both the susceptible and infected populations go extinct at low population sizes.
- 3. R_{D_1} is the disease free state demographic basic reproduction number.
- 4. If either f(0) > 0 or f(0) = 0 and $R_{D_2} > 1$ then the total population is uniformly persistent.

Dramatic Population Extinction

Theorem : Let $R_0 > 1$, f(0) = 0 and $f(N) \le f'(0)N$ for all N > 0. Then there is a function $\zeta = \zeta(\gamma_1, \gamma_2, \phi, \alpha, \sigma, F_1) > 1 \text{ such that if } 1 < R_{D_1} < \zeta$ then the total population goes extinct under H iterations.

Illustrative Example

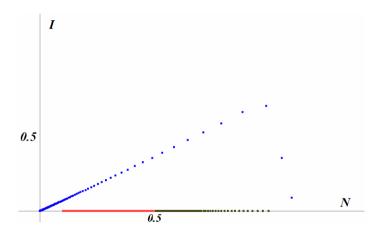
Let
$$f(N) = \frac{aN}{1 + bN}$$
 and $\phi(N) = e^{-\frac{\alpha d}{N}}$
where
 $0.1 < a < 0.2$, $b = 1$, $\alpha = 5$, $\gamma_1 = 0.9$,
 $\gamma_2 = 0.8$ and $\sigma = 0.9$.

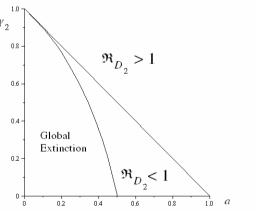
$$R_{D_1} = \frac{a}{1 - \gamma_1} > \frac{0.1}{1 - 0.9} = 1$$
 implies the

persistence of the susceptible population in the absence of the disease.

$$R_{D_2} = \frac{a}{1 - \gamma_2} < \frac{0.2}{1 - 0.8} = 1.$$

As predicted by the theorem, 0.1 < a < 0.177 gives the extinction of the total population.



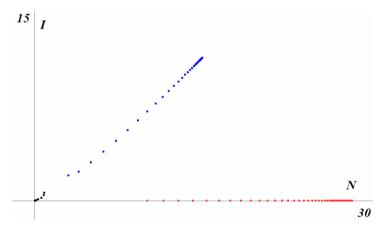


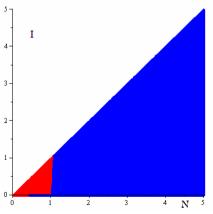
Multiple Attractors

Theorem: Let $\overline{\lim}_{N \to \infty} \frac{f(N) + \gamma_1 N}{N} < 1$ and $R_{D_2} > 1$. Then H has multiple fixed points when G_N "decreases" at low population sizes while it "increases" at high population values.

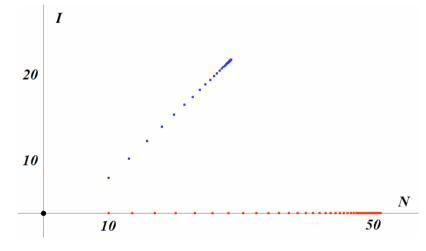
 $Corollary: Let \, \overline{\lim}_{N \to \infty} \, \frac{f(N) + \gamma_1 N}{N} < 1.$

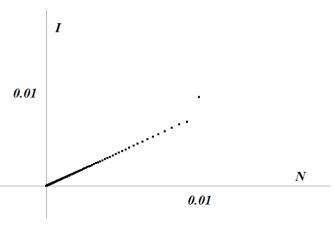
If $R_{D_1} > 1$ and there is $0 < N_0$ with $G_{N_0}(I_1N_0) > N_0$, then the origin is not a global attractor and H has at least two positive fixed points.

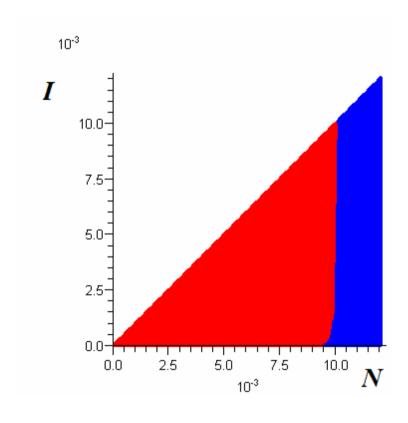




Origin an Attractor







Impact of Seasonality

Consider Model (5) with the Beverton-Holt recruitment function

$$f(t,N) = \frac{aN}{1 + b_t N},$$

and

$$\phi\left(rac{lpha I}{N}
ight)=e^{-rac{lpha J}{N}},$$

where

0.11 <
$$a < 0.15$$
, $b_t = 1.2 + (-1)^t * 0.1$, $\alpha = 5$, $\gamma_{1t} = 0.9 + (-1)^t * 0.0$
 $\gamma_{2t} = 0.8 + (-1)^t * 0.05$, and $\sigma = 0.9$.

In this example,
$$\mathcal{R}_{D_i} = \prod_{t=0}^{p-1} (f'(t,0) + \gamma_{it})$$
.

$$\mathcal{R}_{D_1} = (a + \gamma_{1,0})(a + \gamma_{1,1}) > (0.11 + 0.95)(.11 + 0.85) > 1.01 > 1$$

implies the persistence of the susceptible population in the absence of the disease (Lemma (8)), where

$$\mathcal{R}_{D_2} = (a + \gamma_{2.0})(a + \gamma_{2.1}) < (0.15 + 0.85)(.15 + 0.75) = 0.9 < 1.$$

With our choice of parameters, the disease-free dynamics are governed by the Beverton-Holt model and the susceptible population persists.

$$\begin{array}{lcl} a & = & 2, & b_t = 1.3 + (-1)^t * 1.2995, & \alpha \in [5,300], & \gamma_{1t} = 0.45 + (-1)^t * 0.03, \\ \gamma_{2t} & = & 0.4 + (-1)^t * 0.02, \text{ and } \sigma = 0.0002. \end{array}$$

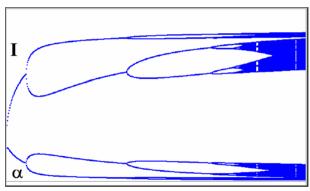


Figure 2: Infective population undergoes period-doubling bifurcation route to chaos as α varies between 5 and 400. On the x-axis, $\alpha \in [5,300]$ and on the y-axis, $I \in [0,400]$.

SIS MODEL IN SEASONAL ENVIRONMENTS

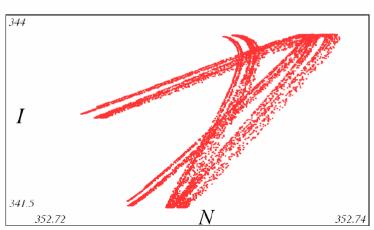


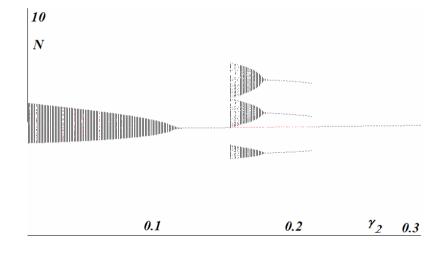
Figure 3: A chaotic attrator in the (I, N) -space.

Question

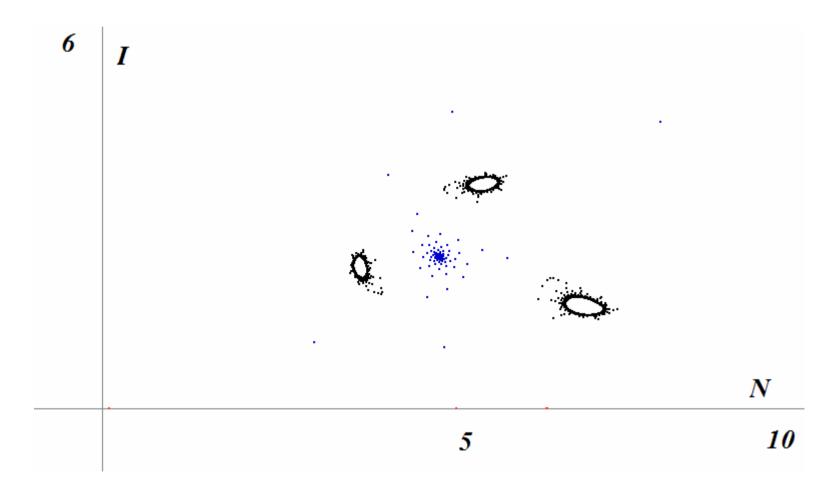
What empirical evidences do we have regarding the potential roles of seasonal fluctuations in cycling (host or pathogen) populations?

Complex Disease Dynamics

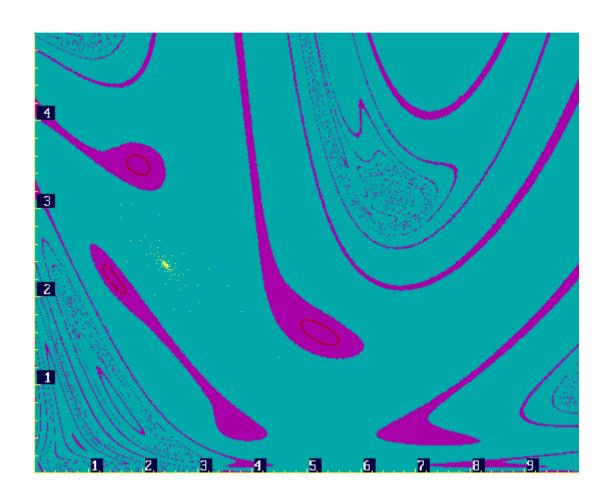
Let f(N) = Nexp(r - N) and $\phi(\frac{\alpha I}{N}) = e^{-\frac{\alpha I}{N}}$ where $\alpha = 5$, $\gamma_1 = 0.9$, $\gamma_2 \in (0, 0.9)$, r = 4 and $\sigma = 0.9$. In the absence of the disease, the susceptible population is on a globally attracting positive fixed point at $S_{\infty} = 6.303$.



Overcompensatory Dynamics



Fractal Basin Boundaries



Geometric Growth

Let $f(N) = \mu N$. In the absence of the disease, the suceptible (disease - free state) equation becomes

$$S(t+1) = \mu S(t) + \gamma_1 S(t) = (\mu + \gamma_1)S(t).$$
 Hence,

$$S(t) = (\mu + \gamma_1)^t S(0) \text{ and } R_{D_1} = \frac{\mu}{1 - \gamma_1}.$$

SIS Model With Geometric Growth

Let
$$i = \frac{I}{N}$$
 and $s = \frac{S}{N}$.

Then i(t) + s(t) = 1 and our SIS model becomes

$$i(t+1) = \frac{F_1(i(t))}{\mu + \gamma_1 + (\gamma_2 - \gamma_1)i(t)}$$
 (5)

R_0

Under geometric growth,

$$R_{0} = \frac{-\gamma_{1}\alpha\varphi'(0)}{(1-\gamma_{1})(R_{D_{1}}-1)+1-\gamma_{2}\sigma}.$$

Theorem: If $R_0 \le 1$, then $\lim_{t\to\infty} i(t) = 0$. That is, the proportion of the infected eventually decreases to zero.

If $R_0 > 1$, then the proportion of the infected population is uniformly persistent.

Envelopes on Compact Intervals [Cull, 1986]

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Let F:[0,1] \rightarrow [0,1] have a unique critical point, i_c, and a unique positive fixed point, i_\infty, where 0 < i_c < i_\infty < 1.
 Also, let \{0\} be an unstable fixed point of F.
 A function E:[0,1] \rightarrow [0,1] envelopes the function F if and only if E(i) \ge F(i) on [0,i_\infty] and E(i) \le F(i) on [i_\infty,1].
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Globally Stable Positive Fixed Point

Theorem (Cull[1986]): If E envelopes F on [0,1] and E(E(i)) > i for all i in $[i_c, i_\infty)$, then i_∞ is a globally asymptotically stable positive fixed point of F on (0,1].

Theorem [F-Y, 2008]: If $R_0 > 1$, our SIS epidemic model with geometric growth has a unique positive globally asymptotically stable equlibrium.

Conclusion

- Our model framework allows the population dynamics and disease transmission to be fairly general.
- We highlighted the role of disease-induced mortality, seasonality and the complexity of the interaction between infectives and susceptible in discrete-time models.
- Disease-induced death can force the extinction of a population with $R_0 > 1$, where the population persists without disease-induced death.
- Disease-induced death can generate multiple attractors with complicated basin structures.
- In epidemic models with disease-induced death, the disease-free dynamics do not drive the disease dynamics.
- Seasonal environments can generate complex bifurcations where none existed in constant environments.