Modeling Epidemics with Seasonal Components Gives Rise to Chaos

Connor Leipelt

Department of Mathematics and Statistics

University of Massachusetts Amherst

Amherst, Massachusetts

December 2023

Contents

1 Introduction **3**

2 Establishing the SEIR Model **3**

2.1 System of ODEs and Parameters 3

2.2 Reproduction Rate of Infection and more Parameters 4

2.3 Introduction of Seasonal Component 5

2.4 Equilibrium States 5

3 Numerics **7**

3.1 Relation between Exposed and Infectives 7

3.2 Parameters for Simulations 8

3.3 Simulations 9

4 Conclusions and Thoughts **16**

4.1 Findings 16

4.2 Meaning and Uses 16

4.3 Future Goals 16

List of Figures

1 Fig. 1.(a) $-log(I)$ vs. T, $\beta_1 = 0.05$ 9

2 Fig. 1.(b) $-log(I)$ vs. $-log(S)$, $\beta_1 = 0.05$ 9

3 Fig. 2.(a) $-log(I)$ vs. T, $\beta_1 = 0.2$ 10

4 Fig. 2.(b) $-log(I)$ vs. $-log(S)$, $\beta_1 = 0.2$ 10

5 Fig. 3.(a) $-log(I)$ vs. T, $\beta_1 = 0.26$ 11

6 Fig. 3.(b) $-log(I)$ vs. $-log(S)$, $\beta_1 = 0.26$ 11

7 Fig. 4.(a) $-log(I)$ vs. T, $\beta_1 = 0.266$ 12

8 Fig. 4.(b) $-log(I)$ vs. $-log(S)$, $\beta_1 = 0.266$ 12

9 Fig. 5.(a) $-log(I)$ vs. T, $\beta_1 = 0.268$ 13

10 Fig. 5.(b) $-log(I)$ vs. $-log(S)$, $\beta_1 = 0.268$ 13

11 Fig. 6.(a) $-log(I)$ vs. T, $\beta_1 = 0.2685$ 14

12 Fig. 6.(b) $-log(I)$ vs. $-log(S)$, $\beta_1 = 0.2685$ 14

13 Fig. 7 Onset of bifurcation vs. Contact rate (β_0 vs. β_1), $Q \approx \beta_0/100$. 15

1 Introduction

Annual rates of endemic diseases vary largely from steady values to high oscillatory behavior creating cycles of repeated endemics. There has been previous work on this subject by Bartlett, Anderson, and May who have worked on small and large communities of endemics using stochastic perturbations [4] [2]. This cyclic behavior leads to the question of seasonal variation affecting the dynamics of the endemic. Analysis of the biennial pattern in measles in NYC agrees that large seasonal variation can generate a biennial cycle [8]. This project goes through the analysis of the model made in *Seasonality and Period-doubling Bifurcations in an Epidemic Model* [3].

2 Establishing the SEIR Model

2.1 System of ODEs and Parameters

We are working with a given population. The population can be divided into 4 groups:

- **Susceptibles**, those able to contract the disease
- **Exposed**, those who have been infected but are not yet infectious
- **Infectives**, those capable of transmitting the disease
- **Recovered**, those who have become immune to the disease

These groups are denoted as S, E, I, R respectively.

This model is made to assume that total population, P, is equal to these four groups and to the value 1. $S + E + I + R = P = 1$.

We suppose that the 4 groups are smooth functions of time with the following parameters:

- μ = birth rate and death rate.
- An exposed individual's probability of becoming infectious in a specified time interval is independent of time after initial contact, meaning that the probability of remaining in the exposed class at time τ after initial contact is $e^{-\alpha\tau}$, where $1/\alpha$ is the mean latent period or the average time between being exposed to infective.

- The probability that an infective recovers after time τ is $e^{-\gamma\tau}$, where $1/\gamma$ is the mean infectious period or the average time between being infective and recovered.
- Recovered individuals are permanently immune.
- An exposure of a susceptible by an infective transmits the infection.
- Contact Rate, $\beta(t)$, is the average number of susceptibles in the given population contacted per infective per unit time.
- $\beta(t)S(t)$ is the rate of total number of susceptibles infected by one infective.
- $\beta(t)S(t)I(t)$ represents the rate of infection of susceptibles by all infectives.

From all of this we can now write down our system of ODEs:

$$S'(t) = \mu - \beta(t)S(t)I(t) - \mu S(t)$$

$$E'(t) = \beta(t)S(t)I(t) - (\mu + \alpha)E(t)$$

$$I'(t) = \alpha E(t) - (\mu + \gamma)I(t)$$

Note: The model is well understood when $\beta(t) = \beta_0$, a constant.

2.2 Reproduction Rate of Infection and more Parameters

Another important piece is the basic reproductive rate of infection, $Q = \beta_0\alpha/[(\mu+\alpha)(\mu+\gamma)]$, given by Anderson [1]. If $Q > 1$, the model has an endemic equilibrium state and a trivial equilibrium point. This is due to the fact that each injection is producing itself plus more. This means that the trivial equilibrium point must be unstable. It has also been shown that the endemic steady state is asymptotically stable. Interestingly, regardless of the value of Q as long as it is nonzero, it will reproduce itself [3].

We next define the *effective infectee number* (EIN) to be the average number of cases produced per average infective in one infectious period. We find that the infectee number approaches unity

(value 1) if the system approaches equilibrium.

Q can be difficult to measure on its own, but it can be indirectly approximated because the susceptible fraction of the population is equal to $1/Q$ at equilibrium. The result is that Q is approximately $1 + L/A$ where L is the mean lifespan and A is the mean age of acquiring the infection [6] [2] [3]. It is key to note that the basic reproductive rate of diseases varies considerably. Two examples are $Q = 12 - 18$ for measles in Western countries and $Q = 6 - 8$ for rubella [7].

2.3 Introduction of Seasonal Component

We are now investigating the relationship between Q and inter-epidemic periods, by changing β_1 in the equation below for $\beta(t)$ and keeping the other parameters constant; this action is in contrast to having $\beta(t) = \beta_0$ where β_0 is a constant value for all t. This is now where we start to look into the seasonal component. We will be using the function of $\beta(t)$ found in [3].

$$\beta(t) = \beta_0(1 + \beta_1 cos(2\pi t))$$

where $0 < \beta_1 < 1$ and $\beta_0 > 0$. It is important to note that London and Yorke have shown that for diseases which confer permanent immunity it is necessary for seasonality in order to have recurrent epidemic [8].

2.4 Equilibrium States

We now look at concepts from the equilibrium case that can be applied to recurrent epidemics with seasonal contact rates. To do this we first set up more parameters:

- $\eta = 1/\gamma$
- $\lambda = 1/\alpha$
- We define the following:

$$C[a, d] = \hat{\eta} \frac{\int_a^d \beta(t) S(t) I(t)\, dt}{\int_a^d I(t)\, dt}$$

  with $\hat{\eta} = \eta/[(1 + \mu\eta)(1 + \mu\lambda)]$ seeing that $\hat{\eta} < \eta$.

From this we can see that $C[a,d]/\hat{\eta}$ is the ratio of the average number of susceptibles becoming infected(i.e. becoming exposed) per unit time to the average number of infectives in the time interval $[a,d]$.

If $(S(t),E(t),I(t)) = (S(t+p),E(t+p),I(t+p))$ for $t \geq 0$, where p is an integer of greater than or equal to unity(1), then $C[0,p]$ is the effective infectee number along a periodic orbit having period p. We can simplify our $C[0,p]$ by substituting in pieces from our ODE system.

$$C[0,p] = \hat{\eta}\frac{\int_0^p \beta(t)S(t)I(t)\,dt}{\int_0^p I(t)\,dt}$$

$$\text{Note: } E'(t) = \beta(t)S(t)I(t) - (\mu+\alpha)E \rightarrow \beta(t)S(t)I(t) = E' + (\mu+\alpha)E$$

$$\text{and } I'(t) = \alpha E(t) - (\mu+\gamma)I(t) \rightarrow I(t) = \frac{\alpha E(t) - I'(t)}{(\mu+\gamma)}$$

and $\hat{\eta} = \eta/[(1+\mu\eta)(1+\mu\lambda)]$ and $\gamma = \frac{1}{\eta}$ and $\alpha = \frac{1}{\lambda}$

Giving us:

$$C[0,p] = \frac{\eta}{(1+\mu\eta)(1+\mu\lambda)}\frac{\int_0^p E'(t) + (\mu+\alpha)E(t)\,dt}{(\frac{1}{\mu+\gamma})\int_0^p \alpha E(t) - I'(t)\,dt}$$

Note: $\int_0^p E'(t)\,dt = \int_0^p I'(t)\,dt = 0$ since we get $E(p) - E(0) = 0 = I(p) - I(0)$.

$$\text{So we get } C[0,p] = \frac{\eta(\mu+\frac{1}{\eta})(\mu+\frac{1}{\lambda})\int_0^p E(t)\,dt}{(1+\mu\eta)(1+\mu\lambda)\frac{1}{\lambda}\int_0^p E(t)\,dt}$$

$$\text{Giving us } C[0,p] = 1$$

From this we see that if:

$$\lim_{t\to\infty}[S(t),E(t),I(t)] = \lim_{t\to\infty}[S(t+p),E(t+p),I(t+p)]$$

then we have that $\lim_{t\to\infty} C[t,t+p] = 1$.

Therefore, if a periodic orbit having period p is asymptotically stable, the EIN approaches 1(unity). Since the EIN measures the average number of cases produced per infective we can infer some scenarios. If EIN < 1 then the disease will be less prevalent. On the other hand, if EIN > 1 then the

disease will become more prevalent. Lastly, if a recurrent epidemic has a high level of incidence a certain year, then there is a high probability that a low level of incidence will happen the following year.

Through this analysis, we see that there is a direct relationship between the susceptible fraction at equilibrium and the basic reproductive rate for general periodic solutions.
Since $C[0,p] = 1$ we have that:

$$\frac{\eta}{(1+\mu\eta)(1+\mu\lambda)} \int_0^p \beta(t)S(t)I(t)\,dt = \int_0^p I(t)\,dt$$

along a periodic orbit.
By using the Mean Value Theorem we see that we can say that there are two positive numbers, $\hat{\beta}\hat{S}$, such that:

$$\hat{\beta}\hat{S} = \frac{\int_0^p \beta(t)S(t)I(t)\,dt}{\int_0^p I(t)\,dt}$$

Giving us:

$$\hat{\eta}\hat{\beta}\hat{S} = 1 \text{ or } \hat{S} = \frac{1}{\hat{\eta}\hat{\beta}}$$

This means that there exist mean values of the susceptible fraction and the basic reproductive rate which are inversely related [3].

3 Numerics

3.1 Relation between Exposed and Infectives

A section of the Seasonal paper goes through a simplification process of reducing the number of ODEs that we wish to analyze down from 3 to 2. This is done by showing that infective and exposed groups are approximately linearly related to first-order.
To do this we let $\epsilon = \mu(Q-1)$ be the force of the injection, and assume that $\epsilon > 0$ and $\epsilon \ll 1$. We

also assume that there exists two positive constants Δ_2 and Δ_3 such that:

$$(\mu + \alpha) = \frac{\Delta_2}{\epsilon} \text{ and } (\mu + \gamma) = \frac{\Delta_3}{\epsilon}$$

Next we do a change of variables by setting the following equal:

$$S = S_0(1 + x),\ E = E_0(1 + y),\ I = I_0(1 + z)$$

where

$$(S_0, E0, I0) = \left(\frac{1}{Q}, \frac{\mu + \gamma}{\alpha} I_0, \frac{\epsilon}{\beta_0}\right)$$

is the endemic equilibrium point, then from this one can show that:

$$y - z = \frac{\epsilon}{\Delta_3} z' = O(\epsilon),\ y = z + O(\epsilon)$$

which implies that:

$$\frac{E}{E_0} = \frac{I}{I_0} + O(\epsilon)$$

Giving us the ratio of infective to exposed individual to be γ/α to order ϵ, which allows us to restrict to the examination of susceptibles and infectives only [3].

3.2 Parameters for Simulations

For these models the variable changing from figure to figure is β_1. The following parameters are kept constant and at these values [3]:

- $\mu = 0.02$/year
- $\alpha = 35.84$/year
- $\gamma = 100$/year
- $\beta_0 = 1800$/year
- Giving $Q \approx 18$

3.3 Simulations

The seasonal paper decided to plot two graphs for each chosen β_1: $-log(I)$ vs. T and a $-log(I)$ vs. $-log(S)$. For the first plot in the paper $\beta_1 = 0.05$. For β_1 very small, a stable periodic orbit having period 1 emerges from the endemic equilibrium point As shown in the following two graphs.

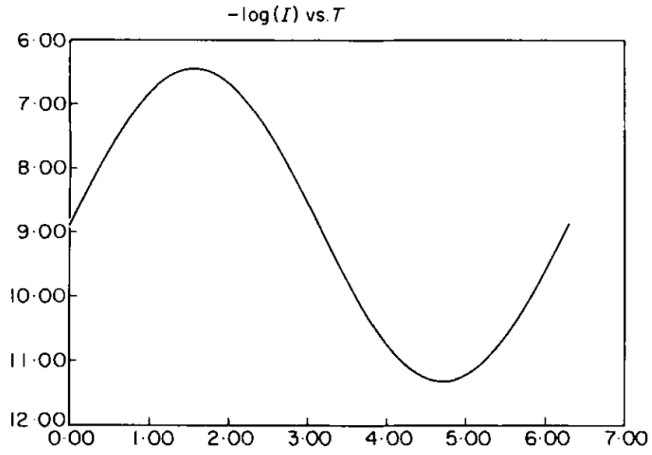

Figure 1: Fig. 1.(a) $-log(I)$ vs. T, $\beta_1 = 0.05$

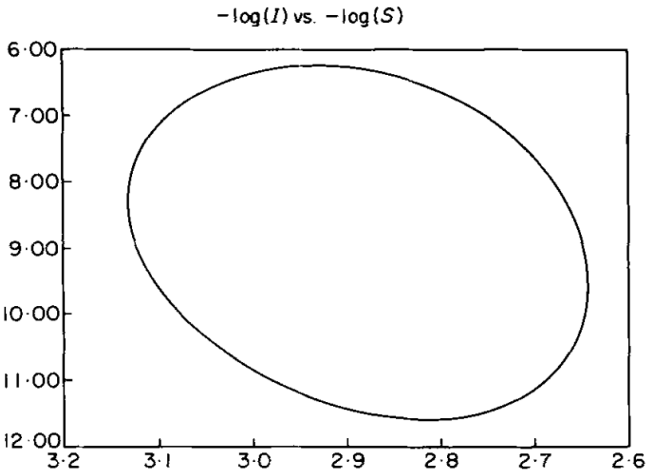

Figure 2: Fig. 1.(b) $-log(I)$ vs. $-log(S)$, $\beta_1 = 0.05$

As β_1 increases, past a critical value of $\beta_{1,c}$, the period 1 orbit becomes unstable and a stable biennial orbit appears. For measles, the period 2 orbit has been used in modelling biennial outbreaks in NYC and England and Wales [3]. This 2 period shows that alternating years have highs and

lows of the epidemic.

Further increments in β_1 yield orbits whose periods double at critical values of β_1. The bifurcations of period doublings follow the Feigenbaum sequence [5] where an accumulation point is reached at some value of β_1 where every orbit is unstable. At this accumulation point, the epidemic is said to be chaotic [3]. This specific period doubling values for β_1 are used in the following Fig 2 - Fig 6.

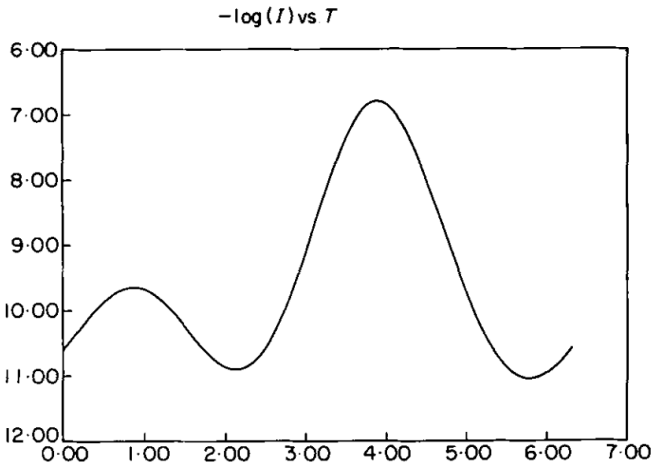

Figure 3: Fig. 2.(a) $-log(I)$ vs. T, $\beta_1 = 0.2$

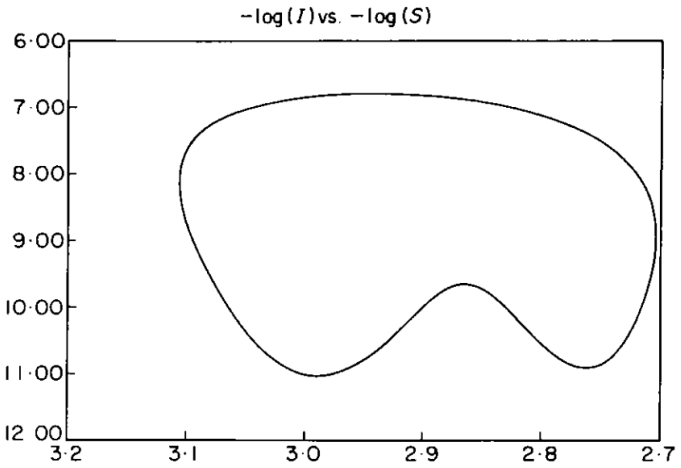

Figure 4: Fig. 2.(b) $-log(I)$ vs. $-log(S)$, $\beta_1 = 0.2$

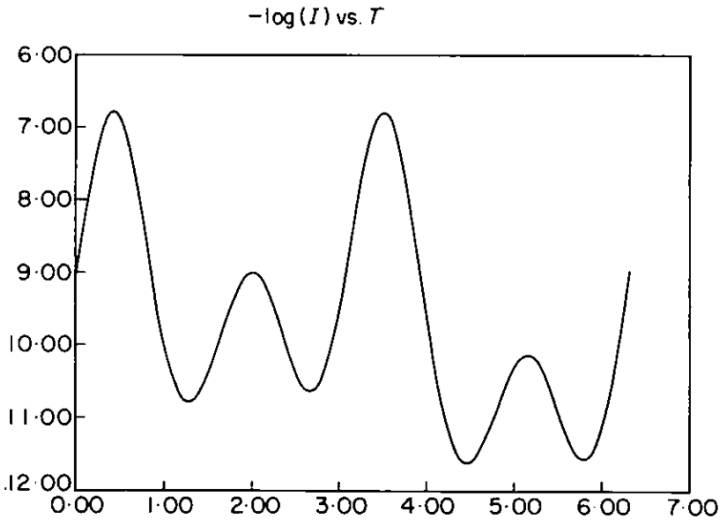

Figure 5: Fig. 3.(a) $-log(I)$ vs. T, $\beta_1 = 0.26$

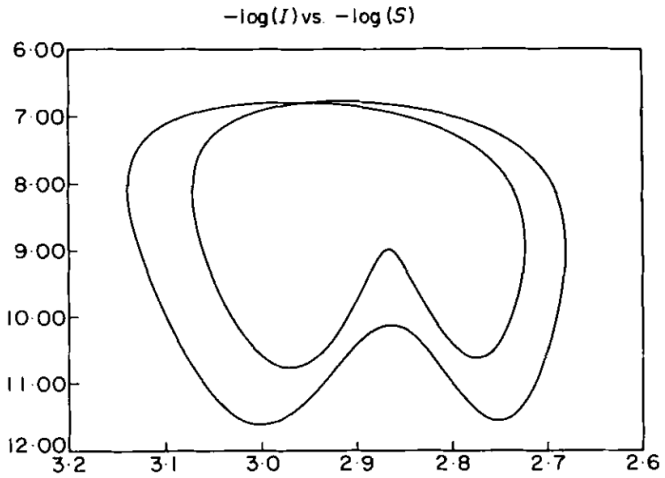

Figure 6: Fig. 3.(b) $-log(I)$ vs. $-\ log(S)$, $\beta_1 = 0.26$

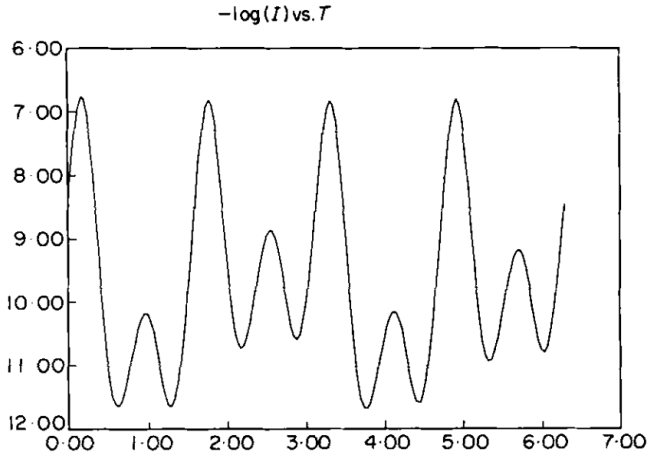

Figure 7: Fig. 4.(a) $-log(I)$ vs. T, $\beta_1 = 0.266$

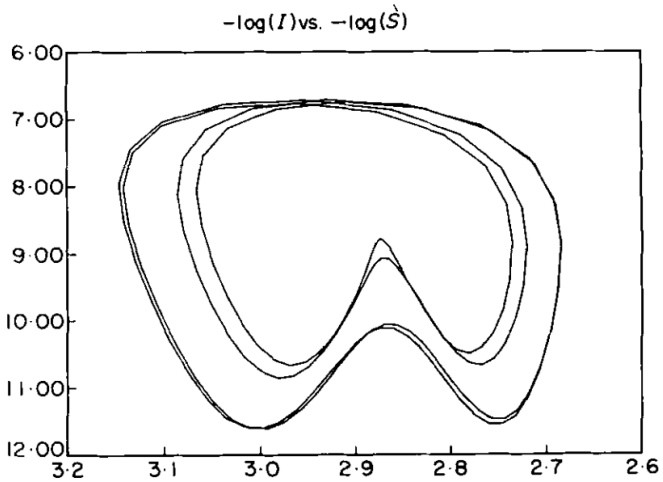

Figure 8: Fig. 4.(b) $-log(I)$ vs. $-log(S)$, $\beta_1 = 0.266$

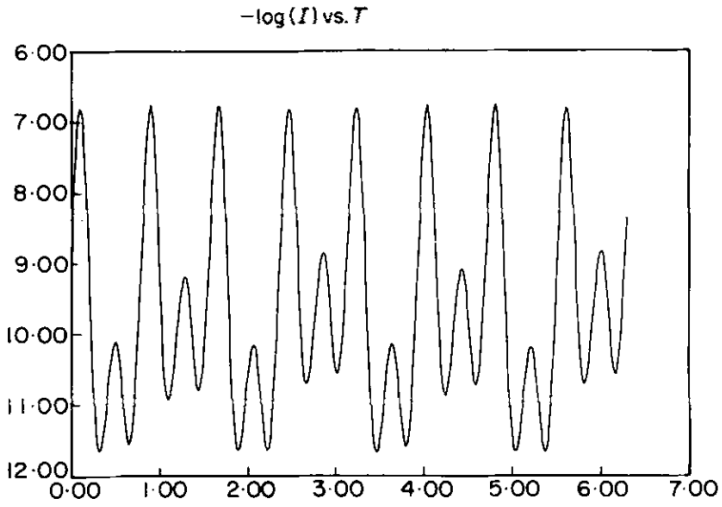

Figure 9: Fig. 5.(a) $-log(I)$ vs. T, $\beta_1 = 0.268$

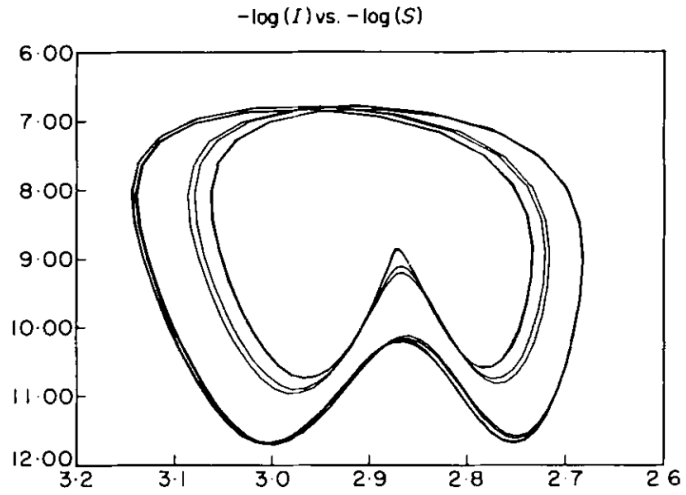

Figure 10: Fig. 5.(b) $-log(I)$ vs. $-\ log(S)$, $\beta_1 = 0.268$

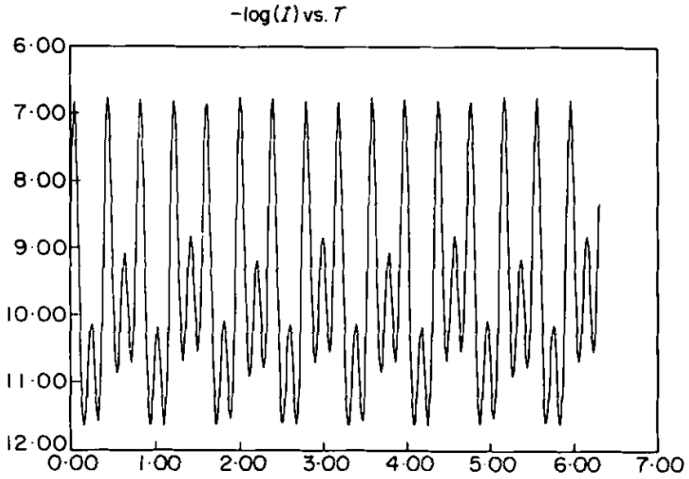

Figure 11: Fig. 6.(a) $-log(I)$ vs. T, $\beta_1 = 0.2685$

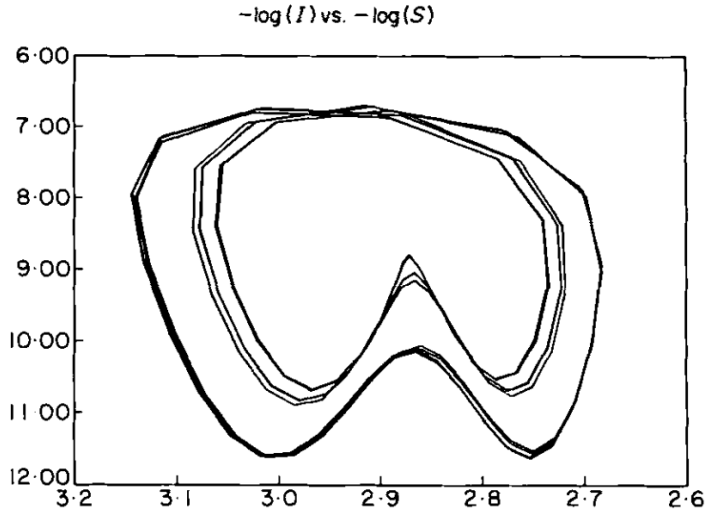

Figure 12: Fig. 6.(b) $-log(I)$ vs. $-log(S)$, $\beta_1 = 0.2685$

Note: We see that there is very little variation in the amplitude of the biennial peaks of the infectives over a wide range of β_1. The peaks mark the years of high amounts of infective and one can see that the following year the amount is considerably lower and spikes back up the year after that, creating our biennial outbreak.

An advantage of the SEIR model is that with new information being introduced as low level noise, the parameter β_1 is robust [3]. This means, if one models a disease in which the outbreaks are

biennial, for a wide range of parameters of β_1, the location of the predicted peaks does not change. A key factor to understanding when biennial outbreaks do occur is the basic reproductive rate, which is directly proportional to the contact rate.

In the following graph we see the onset of bifurcations at $\beta_{1,c}$ as a function of average contact rate, β_0 aka $(Q/\hat{\eta})$.

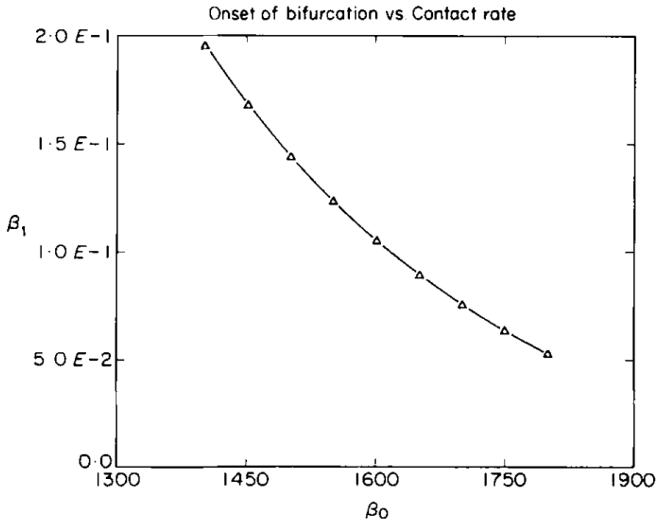

Figure 13: Fig. 7 Onset of bifurcation vs. Contact rate (β_0 vs. β_1), $Q \approx \beta_0/100$.

Low values of β_0 require large values of seasonal forcing (β_1) to produce a biennial cycle and high values of β_0, the contact rate, have biennial outbreaks occurring much more frequently. From this we can conclude that large populations are more susceptible to biennial outbreaks than small populations [3]. It is important to note that for extremely low values of the basic reproductive rate, it is unlikely that a period-doubling bifurcation occurs for a reasonable amplitude of seasonal variation. In the case of rubella $Q = 6 - 8$, it produces only period 1 (annual) epidemics in this model.

4 Conclusions and Thoughts

4.1 Findings

Through [3] it's been numerically shown that small-amplitude periodic solutions arising from the seasonally-forced SEIR epidemic model form a sequence of period-doubling bifurcations.

4.2 Meaning and Uses

Interpreting this tells us that as β_1 increases, the period will effectively start to double, going from period 1, to period 2, to period 4, etc. and will lead to chaotic behavior. It's also been found that the patterns of high and low infective amounts persists through the presence of small-amplitude noise. Because of the inability to construct small-amplitude orbits of arbitrary periodicity it is difficult to create a unifying theory of recurrent epidemics.

4.3 Future Goals

Not all evidence suggests that seasonality is the driving force in all situations. If we wish to create a more structured understanding of recurrent epidemics, then we must look into studying epidemics themselves more and look into the possibility of adding in other factors such as the existence of a climactic effect or multiple infectious agents [3].

References

[1] ANDERSON, R. M. (1982a). In: Population Dynamics of Infectious Diseases (Anderson, R. M., ed.), pp. 1-37, London and New York: Chapman and Hall.

[2] ANDERSON, R. M. (1982b). In: Population Biology of Infectious Diseases (Anderson, R. M. and May, R. M., eds), pp. 149-176, Berlin, Heidelberg and New York: Springer-Verlag.

[3] Aron, Joan L., and Ira B. Schwartz. "Seasonality and period-doubling bifurcations in an epidemic model." Journal of Theoretical Biology, vol. 110, no. 4, 1984, pp. 665–679, https://doi.org/10.1016/s0022-5193(84)80150-2

[4] BARTLETY, M. S. (1960). Stochastic Population Models in Ecology and Epidemiology. London: Methuen.

[5] COLLET, P. and ECKMANN, J.-P. (1980). Iterated Maps on the Interval as Dynamical Systems. Boston: Birkhauser

[6] DIETZ, K. (1975). In: Proceedings of the SIMS Conference on Epidemiology in Alta, Utah, July 8-12, 1974 (Ludwig, D. and Cooke, K. L., eds), pp. 104-121, Philadelphia: SIAM.

[7] HETHCOTE, H. W. (1983). Am. J. Epidem. 117, 2

[8] LONDON, WAYNE P., and JAMES A. YORKE. "Recurrent outbreaks of measles, chickenpox and mumps." American Journal of Epidemiology, vol. 98, no. 6, 1973, pp. 453–468, https://doi.org/10.1093/oxfordjournals.aje.a121575