Abstract

For at least the past ten years, eating disorders have had a major impact in the physical and mental health of women, particularly young women. Anorexia and bulimia nervosa are closely linked eating disorders. Anorexia often precedes bulimia. However, there are about 2 million women in college that have been exclusively bulimic. In this article, we focus on the role of college-peer pressure on the dynamics of anorexia-free bulimia. The model looks at bulimia as a progressive disease and explores the impact of intervention (treatment) at two stages of disease progression. The impact of relapse (a common occurrence among bulimics) is taken into account. Analysis indicates that the disorder cannot be wiped out in this population without a shift in cultural pressures; control strategies should include early detection and treatment, as well as preventative education campaigns.

Keywords: bulimia nervosa, eating disorders, dynamical systems, peer pressure, reproductive number
1 Introduction

Like AIDS, eating disorders suddenly became widely known to the North American public in the mid 1980s. The catalyst was the death of popular singer Karen Carpenter from cardiac arrest, associated with the abuse of a drug used to induce vomiting in case of poisoning (Gordon, 1990). Public attention focused for the first time on the self-destructive aspects of anorexia nervosa, and the related illness bulimia nervosa. Both of these disorders revolve around a fear of obesity, obsession with being thin, and a struggle between societal pressures to remain thin and the biological necessity of consuming food. An increase in incidence of these and related disorders has been observed, primarily in young women, over the roughly twenty years since concerted efforts to keep data on them began. In the segment of the population most deeply impacted, there is a rising epidemic of both anorexia (which causes emaciation from excessive dieting) and bulimia (which changes body chemistry through repeated purging), driven by pressures to conform to cultural ideals. This paper uses techniques developed for the modeling of infectious diseases to study the spread of bulimia as a peer-pressure-driven phenomenon.

Because of the psychological aspects of these disorders, prevalence data are based in large part on estimates gleaned from self-reporting on questionnaires and records of those individuals actually clinically diagnosed. It is estimated that eight million people in the U.S. have been diagnosed with anorexia or bulimia, seven million of whom are females (ANRED, 2001), and that 0.5 to 3.7 percent of females suffer from anorexia nervosa in their lifetime, and 1.1 to 4.2 percent of females from bulimia nervosa (APA, 2000). The National Center for Health Statistics estimated that about 9,000 people admitted to hospitals were diagnosed with bulimia in 1994, and about 8,000 were diagnosed with anorexia (NCHS, n.d.). These disorders can be fatal: according to the American Anorexia and Bulimia Association, an estimated 1000 women die of anorexia each year (AABA, n.d.). Both of these eating disorders seem to have the most impact on young women between the ages of 15–25 in the U.S. (Steiner & Lock, 1998). Because the media portray thinner models as how women should look — the average North American model weighs 23 percent less than the average North American woman (Wooley, 1991) — young women, struggling to develop their identities, are especially vulnerable to developing these illnesses in response to such pressures (Murray et al., 1995; Steiner & Lock, 1998). The McKnight investigators wrote (2003) that “thin body preoccupation and social pressure is the strongest proximal indicator of the onset of eating disorders.” Early studies found young women in their early teens with anorexia, and a history of bulimic behavior by their first year of college. Once anorexic there is a 50% chance that an individual will become bulimic (Drewnowski, Yee & Krahn, 1988). Bulimia nervosa has increased in epidemic proportions among the college female population (Gordon, 1990), currently approximately 8 million, since it was first clinically identified in the late 1970s (Halmi, Falk & Schwartz, 1981). The incidence of bulimia in college-age females has been estimated to be anywhere from 4 to 20 percent (Halmi et al., 1981; Drewnowski et al., 1988).

The distinction between anorexia and bulimia lies in the type of behavior which arises in response to the compulsion to be thin. Most anorexies, perceiving their bodies to be fatter than they really are, thereby develop a desire to maintain an unhealthily low body weight which manifests itself through excessive dieting (to the point of food avoidance) and exercise (Brumberg, 1988; Schlundt & Johnson, 1990). In contrast, bulimics are not
always able to maintain severe dietary restraint. These individuals turn to purging to try to reduce caloric intake, primarily self-induced vomiting and/or abuse of laxatives or other medication. Despite these extreme measures, bulimics often remain near their normal body weight (because of limited dietary restraint), making the disorder easier to conceal. Although there is some overlap between sufferers of the two disorders, the differences in behavior keep the diagnoses distinct. Anorexics, in fact, may even be proud of the thinness they achieve, while bulimics, feeling shameful, become adept at hiding the disorder (Gordon, 1990). While the clinical symptoms of anorexia nervosa therefore arise from an excessively low body mass, bulimia presents hazards that follow from purging and resulting changes in body chemistry. Repeated vomiting causes acid from the stomach to damage dental enamel, salivary glands, and other parts of the digestive tract, and can even break blood vessels in the eyes. Disturbances of these acids disrupts body chemistry, as does abuse of laxatives (Mitchell, 1986, 1990), to the point that in extreme cases death can occur (Keel & Mitchell, 1987). More commonly, the menstrual cycle becomes irregular or stops altogether. It is also common for young women to develop relatively mild initial symptoms (e.g., infrequent or without purging episodes) triggered by a period of marked unhappiness or restrictive dieting (Mitchell, 1990): for a time the bulimic episodes may occur periodically rather than continuously, and remain easy to conceal (“closet bulimia”). As the severity of the disorder increases, the individuals’ lives become more chaotic, provoking them in some multi-impulsive cases to scavenge leftovers from a dustbin or steal in order to feed the compulsion (Mitchell, 1990). One review found (Bilich, 1989) that the onset of purging behavior lags behind the onset of bingeing by an average of a year or so. There is then a corresponding feeling of shame and desire for secrecy on the part of the bulimic. It is generally only at this point that symptoms become severe enough to force the sufferer into treatment.

Despite the difficulties with gathering accurate prevalence data for these disorders, one statistic which has remained relatively consistent across studies is the mean age at onset of bulimia: 18 years (Bilich, 1989; Mitchell, 1990). The coincidence of this age with the point in many young women’s lives when they leave a family environment for the first time to enter a largely peer-defined university culture motivates the present study. Furthermore, Gordon (1989, 1990) observed that there is a significant social component to bulimic behavior on college campuses: its incidence has often been observed in clusters — for example, among women living in the same dormitory or sorority house — even to the extent in extreme cases of purging in groups. Studies of the factors which contribute to the development of eating disorders consistently cite peer sociocultural pressure as a leading risk factor. Lieberman (2001) found, “At the level of the individual, involvement in a close friendship, high opposite-sex relational esteem, severe weight and body-shape teasing, peer pressure about weight and appearance, externalized self-perceptions, and peer attributions about the importance of weight and appearance for popularity and dating were important predictors of problematic eating behaviors.” A recent study of risk factors for eating disorders identified seven separate factors, of which the strongest (and in some cases the only factor significantly related to outcome) was thin body preoccupation and social pressure (McKnight Investigators, 2003). It is clear, therefore, that in addition to the environment produced by cultural norms, peer pressure plays a significant role in the rising prevalence of bulimia in this population.

As the prevalence of eating disorders rises, their treatment has become an important research topic in its own right. Without treatment, up to 20 percent of people with serious
eating disorders die. With treatment, that number falls to 2–3 percent (ANRED, 2001). However, treatment is not always successful, and, as with other behavior disorders, there is a significant relapse rate. A survey (Keel & Mitchell, 1997) found that five to ten years after presentation, about 50% of bulimics had made complete recoveries, 20% still met all clinical criteria for bulimia despite treatment, and about 30% experienced relapse. However, there are indications that many bulimics who relapse do so within six months of entering treatment (Mitchell, 1990).

Variability in success rates stems in part from the need for individualized treatment plans. Because each individual’s situation involves a different combination of the many contributing factors, treatment must be customized for each individual. A typical treatment plan used might include hospitalization, medication to relieve depression and anxiety, nutrition counseling, individual, group and family counseling, and support groups to break down isolation and alienation (Mitchell, 1990). Treatment is often along behavioral lines at first and gradually focuses more on emotional problems. Estimates of the time required for full recovery are on the order of years, so this phase is a significant one in the course of the disorder.

Treatment rates are limited by two significant factors: the secretive nature of the disorder, discussed above, and cost. The cost of treatment may vary from US $3800 up to US $24,000 per month or more for intensive treatment (e.g., Mirasol, n.d.; Westwind, 1999). The healthcare industry has no standardized measures of “need for treatment” for eating disorders, and consequently many people cannot seek treatment even if they have medical insurance.

This article examines the influence of peer pressure on the prevalence of bulimia in a population of college females, by considering a theoretical model in which both the incidence rate and the treatment rate are influenced by peer-pressure terms akin to the mass action terms used in models for the spread of infectious diseases. Sociologists have observed (Crane, 1991; Gladwell, 2000) that some social phenomena, such as juvenile crime, are “contagious” in the sense that the rate at which they occur depends on the number of individuals involved, as well as on the number of individuals who might become involved, and where “tipping points” determine whether or not the phenomenon dies out or persists in a population. Our model makes this epidemiological analogy quantitative, and tries to account for the population dynamics behind observed data. Our approach borrows from the analysis of dynamical systems in mathematical epidemiology, in order to address the following questions: What implications does a peer-pressure-driven progression have for the control of bulimia nervosa in a university population? and what are the relative effects of treatment and education at different (early or advanced) stages of the disorder? An education campaign might serve to reduce the rate of “infection” (e.g., Winzelberg et al., 2000), as well as to encourage bulimics to seek treatment early. The following section describes our theoretical model and its underlying assumptions; later sections present its analysis and draw some conclusions.

2 The model

A general model for anorexia and bulimia in the population consisting of young women ages 12 to 22 might look as follows (Figure 1). The at-risk population (S) can develop either
anorexia (A) or bulimia (B). Once anorexic, an individual may become bulimic. There is treatment available specifically for anorexics (TA) or for bulimics (TB). Some individuals in treatment suffer relapses; others eventually recover (R). There is also a given rate at which new individuals age into the [S] population and others age out (downward diagonal arrows).

![Anorexia and bulimia model](image)

Figure 1: Anorexia and bulimia model

If we now restrict our attention to bulimia in college-age females, we can simplify the above model while differentiating between the early and advanced stages of the disorder, since treatment is typically only sought once an individual is in the advanced stage. In order to keep the model as simple as possible, we shall consider the effect of public education campaigns by reducing the rate at which susceptible individuals develop bulimia, rather than adding an educated class. This new model, illustrated in Figure 2, eliminates a number of the classes of the first model, and identifies individuals only by stage of bulimia nervosa. Members of the at-risk (susceptible) population S may develop the first stage of bulimia nervosa (B₁) either directly or by progression from anorexia. The individuals in state B₁ are closet bulimics, who are not receiving treatment (commonly out of embarrassment or shame) nor are their symptoms severe enough to make discovery likely. When episodes of bingeing and purging occur with greater frequency and severity, they move to the advanced state, B₂, where the symptoms are harder to hide. At this state, bulimic individuals may enter treatment, either because of pressure from peers already in treatment or because they are so sick that they need medical help. Some individuals in treatment (T) relapse to the B₂ class, but many will progress to a full recovery in time. For simplicity, and because recovery usually requires some years spent in treatment — and we here assume the average time a female stays in college to be approximately three years (lower than the nominal four years due to effects of transfers, dropouts, and other family and personal situations) — this more focused model does not include the recovered stage R of the previous model. We use \( P = S + B₁ + B₂ + T \) to represent the total constant population. (Note all transition rates in Figure 2 are given as per capita except the recruitment rate, which is given as an absolute, cf. the equation below for \( dS/dt \).)

To specify the rates at which individuals move from one class to another, we consider the factors underlying each transition. In our model, susceptibles become bulimics due to peer pressure. Therefore, the rate at which susceptible women develop bulimia depends on how much interaction a woman has with her bulimic friends. We assume that the rate depends upon two factors: cultural pressure to conform to unrealistic ideals, measured by a parameter
\( \alpha \), and the environmental peer pressure which acts as a catalyst, measured in direct relation to the proportion of bulimics already present among the peer set, \((B_1 + B_2)/P\). This gives a per capita rate of \( \alpha(B_1 + B_2)/P \). It is important to note that peer pressure is considered here as a collective force, rather than simply as a sum of individual pressures, and we use the bulimia incidence within the population under study as an estimate of the strength of this force in pressing upon its members the unrealistic ideal suggested by the larger culture. At this stage it is very rare that women will look for treatment, but we can model the effects of an education campaign \( \sigma \) by considering \( \alpha \) as a function of \( \sigma \), \( \alpha(\sigma) \). In contrast, the progression to the more severe stage of bulimia depends more on the disorder itself than on peer pressure, and is modeled here by a constant per capita rate \( \gamma \).

With regard to treatment rate, we make the assumption that friends and family play different roles in pressuring bulimics to get treatment. One factor is that of parents, or occasionally healthcare professionals, discovering an individual’s symptoms and obliging her to enter treatment; we model this with a constant per capita rate \( \rho \). The other factor is peer pressure, measured in terms of the proportion of peers currently receiving treatment, \( T/P \), multiplied by a constant of proportionality \( \delta \), so that the overall per capita treatment rate is \( \rho + \delta T/P \). Finally, we assume that the relapse rate depends on the individual more than peer pressure, so we model this with a constant per capita rate \( \phi \). Since the model considers only the college female population, we do not model the slower transition to full recovery, but instead incorporate the turnover in the population with a demographic parameter \( \mu \), the per capita rate at which individuals enter and leave the population.

A note about mixing and population size is in order here. In each case above where the strength of peer pressure has been estimated by proportions of the at-risk population in various stages of bulimia, an underlying assumption is that proportional, homogeneous mixing occurs among students — that is, students mix reasonably uniformly with each other regardless of bulimic status (the peer group is the entire population). This simplifying assumption is valid for a small to moderate-sized college population, but not for large universities where individuals are likely to mix preferentially in partially isolated subgroups. To incorporate the latter case we shall restrict our attention to a single such subgroup within which mixing is uniform. If, instead, mixing were heterogeneous, it would be more appropriate to measure peer pressure using proportions of the preferred subgroup (in this case, bulimics).
We can now write a set of four differential equations for this model:

\[
\begin{align*}
\frac{dS}{dt} &= \mu P - \alpha(\sigma)S \frac{B_1 + B_2}{P} - \mu S, \\
\frac{dB_1}{dt} &= \alpha(\sigma)S \frac{B_1 + B_2}{P} - \gamma B_1 - \mu B_1, \\
\frac{dB_2}{dt} &= \gamma B_1 - \mu B_2 - \rho B_2 - \delta B_2 \frac{T}{P} + \phi T, \\
\frac{dT}{dt} &= \rho B_2 + \delta B_2 \frac{T}{P} - \phi T - \mu T.
\end{align*}
\]

The model can be reduced to three equations since we have a constant total population, \( P = S + B_1 + B_2 + T \). For the moment, however, we will merely rescale it with the dimensionless variables \( x = \frac{S}{P}, \ y_1 = \frac{B_1}{P}, \ y_2 = \frac{B_2}{P}, \) and \( z = \frac{T}{P} \), to eliminate dependence on the population size \( P \) and obtain the model

\[
\begin{align*}
\frac{dx}{dt} &= \mu - \alpha(\sigma)x(y_1 + y_2) - \mu x \\
\frac{dy_1}{dt} &= \alpha(\sigma)x(y_1 + y_2) - \gamma y_1 - \mu y_1 \\
\frac{dy_2}{dt} &= \gamma y_1 - \mu y_2 - \rho y_2 - \delta y_2 z + \phi z \\
\frac{dz}{dt} &= \rho y_2 + \delta y_2 z - \phi z - \mu z
\end{align*}
\]

where \( 1 = x + y_1 + y_2 + z \).

The model parameters are summarized in Table 1. Recall that we are considering the “infective” force \( \alpha \) as a decreasing function of the effort \( \sigma \) expended in public education about eating disorders.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \mu )</td>
<td>Per capita “mortality” rate (aging in or out)</td>
</tr>
<tr>
<td>( \alpha(\sigma) )</td>
<td>Per capita peer pressure rate at which people develop bulimia (( B_1 ))</td>
</tr>
<tr>
<td>( \sigma )</td>
<td>Effort expended in education to reduce the prevalence of bulimia</td>
</tr>
<tr>
<td>( \gamma )</td>
<td>Per capita rate at which bulimics progress from ( B_1 ) to ( B_2 )</td>
</tr>
<tr>
<td>( \delta )</td>
<td>Per capita rate at which advanced bulimics (( B_2 )) get treatment due to peer pressure from people currently in treatment</td>
</tr>
<tr>
<td>( \rho )</td>
<td>Per capita rate at which advanced bulimics (( B_2 )) get treatment due to needed medical care or family intervention</td>
</tr>
<tr>
<td>( \phi )</td>
<td>Per capita relapse rate at which people in treatment return to ( B_2 )</td>
</tr>
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</table>

Table 1: Parameter list
3 Analysis

3.1 The bulimia-free equilibrium and $R_0$

One possible end state for this model is the \textit{bulimia-free equilibrium} $(x, y_1, y_2, z) = (1, 0, 0, 0)$. It can be quickly seen that if, on average, women in the college population leave the university environment faster than they develop bulimia, then no bulimic state will ever develop in this population, as stated formally below in terms of \textit{global stability} (all solutions approaching a given point).

\textbf{Proposition 1}. The bulimia-free equilibrium $(1, 0, 0, 0)$ is globally stable if $\alpha < \mu$.

\textbf{Proof}. Use the Lyapunov function $V = y_1 + y_2 + z$.

\[ \frac{dV}{dt} = \alpha x(y_1 + y_2) - \mu (y_1 + y_2 + z) \leq (\alpha x - \mu)(y_1 + y_2) \leq (\alpha - \mu)(y_1 + y_2) \]

since $x \leq 1$. If $\alpha < \mu$, then $dV/dt < 0$. $\square$

Thus one goal of an education campaign might be to reduce $\alpha(\sigma)$ to this extent if possible. However, if the mean age at onset is 18 years (Mitchell, 1990), then this is not currently the case, so we shall consider $\alpha > \mu$.

We can calculate the basic reproductive number $R_0$ of the disorder, interpreted in epidemiological models as the average number of secondary cases caused by an infected individual. Since the transmission here is collective rather than individual, we should interpret $R_0$ as an indicator of how conducive the environment is to developing this eating disorder, a ratio of how quickly individuals become bulimic relative to how fast bulimics recover or leave the population. It will be seen below that this quantity plays an important role in determining the possible limiting behaviors of the model, i.e., to what extent bulimia becomes (or remains) endemic in the population. To calculate it, we used the next generation operator method given in Diekmann, Heesterbeek & Metz (1990) and Castillo-Chavez, Feng & Huang (2002). Here we took $y_1$ and $y_2$ as the infective compartments and $x$ as the susceptible compartment. Details are given in the appendix. The computation yields the following expression:

\[ R_0 = \frac{1}{2} \left[ \frac{\alpha}{\mu + \gamma} + \frac{\phi \rho}{(\mu + \phi)(\mu + \rho)} + \sqrt{\left( \frac{\alpha}{\mu + \gamma} - \frac{\phi \rho}{(\mu + \phi)(\mu + \rho)} \right)^2 + \frac{4 \gamma \alpha}{(\mu + \rho)(\mu + \gamma)}} \right]. \quad (6) \]

Despite the complexity of the formula, we can make some observations that will facilitate an interpretation. First we observe (by neglecting the last term under the radical) that

\[ R_0 > \max \left( \frac{\alpha}{\mu + \gamma}, \frac{\phi \rho}{(\mu + \phi)(\mu + \rho)} \right). \]

The first of these two quantities, $\alpha/(\mu + \gamma)$, is the basic reproductive number for a single-stage model of bulimia; roughly speaking, individuals enter the $B_1$ class at a rate of $\alpha$ and...
leave at a rate of $\mu + \gamma$, or, equivalently, after an average time of $1/(\mu + \gamma)$. The second quantity, $\frac{\phi}{\mu + \phi} \frac{\rho}{\mu + \rho}$, is the proportion of advanced-stage bulimics who enter treatment $(\frac{\phi}{\mu + \phi})$ and then suffer a relapse $(\frac{\rho}{\mu + \rho})$, before aging out of the college population. This latter cycle is a secondary source of bulimia cases, and cannot by itself satisfy the usual $R_0 > 1$ criterion for an endemic state to arise. $R_0$ exceeds the former term by the societal pressure contributed by the presence advanced bulimics, and the latter term by the initial incidence rate. The two terms are in some sense complementary.

Next we observe (since $\sqrt{m + n} < \sqrt{m + \sqrt{n}}$) that

\[ R_0 < \max \left( \frac{\alpha}{\mu + \gamma} \frac{\phi \rho}{(\mu + \phi)(\mu + \rho)} \right) + \sqrt{\frac{\alpha}{\mu + \gamma} \frac{\gamma}{\mu + \rho}}. \]

If, as we might expect (and as is borne out by the parameter estimates in Section 4), bulimia incidence can be attributed more to initial development of the disorder than to relapses, then we have

\[ R_0 < \frac{\alpha}{\mu + \gamma} + \sqrt{\frac{\alpha}{\mu + \gamma} \frac{\gamma}{\mu + \rho}}. \quad (7) \]

The first of the two terms on the right side of (7) is again the reproductive number for the first infective class, $R_0(S \rightarrow B_1)$. The second term is a reproductive number for the second infective class, $R_0(S \rightarrow B_2)$. It involves a radical because replacement of advanced-stage bulimics is a two-stage process: $S \rightarrow B_1 \rightarrow B_2$. The two factors inside the radical are the reproductive numbers for the two stages involved, $R_0(S \rightarrow B_1)$ and $R_0(B_1 \rightarrow B_2)$. This type of expression for two-stage cycles has been seen before in models for infectious diseases, e.g., Diekmann et al. (1990), May & Anderson (1988, equation (5.7)). The actual value of $R_0$ is less than $R_0(S \rightarrow B_1) + R_0(S \rightarrow B_2)$ because of the $S \rightarrow B_1$ overlap in the two cycles. Expressions for $R_0$ of the form seen in (6) have previously been observed in models for multi-stage diseases, including a three-stage model in Diekmann, Dietz & Heesterbeek (1991) and a four-stage HIV model in Dietz, Heesterbeek & Tudor (1993).

Finally, the fact that $R_0$ is independent of $\delta$, the peer-pressure treatment rate parameter, might lead us to conclude that peer-based encouragement to seek treatment does not affect society’s conduciveness to support an “epidemic” of bulimia. However, we recall that $R_0$ measures response to the first appearances of bulimia in the population, while $\delta$ corresponds to a term whose strength also depends on the presence of peers currently in recovery. As we shall see in the following section, this parameter does have an important role to play in determining the size of the bulimic population in situations where bulimia does arise.

### 3.2 Endemic equilibria

Another possible end state for model (1–5) is one in which bulimia exists in an endemic state within the college female population. If $R_0 > 1$, then such a state certainly exists, although the number of endemic equilibria is difficult to establish analytically, as the following analysis shows.

**Proposition 2.** If $R_0 > 1$, we have either one or three endemic equilibria.

If $R_0 < 1$, we have zero or two endemic equilibrium points.
Proof. We obtain the equilibrium conditions from equations (1–5), and reduce the system to just one equation by expressing the equilibrium values for \( x, y_1, \) and \( z \) in terms of \( y_2 \):

\[
  z = y_2 \frac{\rho}{\mu + \phi - \delta y_2} \quad \text{from (4)},
\]

\[
  y_1 = y_2 \frac{\mu}{\gamma} \left( 1 + \frac{\rho}{\mu + \phi - \delta y_2} \right) \quad \text{from (3), (8)},
\]

\[
  x = \frac{\mu}{\mu + \alpha y_2 \left[ 1 + \frac{\mu}{\gamma} \left( 1 + \frac{\rho}{\mu + \phi - \delta y_2} \right) \right]} \quad \text{from (1), (9)},
\]

and then substituting into (5):

\[
  1 = \frac{\mu}{\mu + \alpha y_2 \left[ 1 + \frac{\mu}{\gamma} \left( 1 + \frac{\rho}{\mu + \phi - \delta y_2} \right) \right]} + y_2 \left[ \frac{\mu}{\gamma} \left( 1 + \frac{\rho}{\mu + \phi - \delta y_2} \right) + 1 + \frac{\rho}{\mu + \phi - \delta y_2} \right].
\]

After multiplying by the various denominators and dividing by \( y_2 \), we will have a cubic equation in \( y_2 \), of the form

\[
  f(y_2) = Ay_2^3 + By_2^2 + Cy_2 + D = 0,
\]

where \( A > 0, B, C \) and \( D \) are functions of model parameters (see appendix for details).

Now, for an endemic equilibrium we need for \( x^*, y_1^*, y_2^*, \) and \( z^* \) to be nonnegative and sum to 1. The use of (5) to get (11) guarantees the latter; we can see from (8) that in order to have \( z \geq 0 \), we need \( \mu + \phi - \delta y_2^* > 0 \). Therefore, we require that

\[
  y_2^* < \frac{\mu + \phi}{\delta}.
\]

With this condition, we can see from (9) and (10) that \( y_1 \) and \( x \) are then also nonnegative. Hence, endemic equilibria are solutions to \( f(y_2) = 0, y_2^* \in (0, \frac{\mu + \phi}{\delta}) \). It is not necessary to worry about whether \( \frac{\mu + \phi}{\delta} > 1 \) or \( \frac{\mu + \phi}{\delta} < 1 \) since equation (5) already guarantees solutions less than 1.

We now consider \( f(0) \) and \( f(\frac{\mu + \phi}{\delta}) \). Calculations show that

\[
  f(0) = D < 0 \iff \alpha > \mu \frac{\mu + \phi + \rho}{\mu + \phi + \rho \frac{\mu}{\mu + \gamma}} \iff R_0 > 1 \quad \text{(12)}
\]

and

\[
  f \left( \frac{\mu + \phi}{\delta} \right) = \left( \frac{\mu + \phi}{\delta} \right) \left( \frac{\mu + \gamma}{\gamma} \right) \frac{\mu}{\gamma} \alpha \rho^{2} > 0.
\]

By continuity of \( f \) at least one solution \( y_2^* \) exists when \( R_0 > 1 \) (since then \( f(0) < 0 < f(\frac{\mu + \phi}{\delta}) \)). More specifically, since \( f \) is cubic, if \( R_0 > 1 \), we have either one or three endemic equilibria. If \( R_0 < 1 \), we have zero or two endemic equilibrium points. \[\square\]

At this point there remains the possibility of having multiple endemic equilibria; numerical investigations, such as the one considered in Section 4, indicate that multiple endemic equilibria do not occur, but the complexity of the model makes it difficult to show in general.
We can, however, make some observations that make multiple endemic equilibria unlikely. First, we note that the bifurcation at \( R_0 = 1 \) is “forward” — that is, the small (in \( y_2 \)) endemic equilibrium which arises for \( R_0 \) close to 1 exists only for \( R_0 > 1 \):

**Proposition 3.** The bifurcation at \( R_0 = 1 \) is forward in \( R_0 \), and endemic equilibria with \( y_2 \) close to zero arise only for \( R_0 > 1 \).

**Proof.** In terms of equation (11), \( R_0 = 1 \iff f(0) = 0 \). That is, there is a solution \( y_2^* \) of (11) which crosses zero as \( R_0 \) crosses 1. Therefore, as a graphical analysis will show, if \( f'(0) > 0 \) for \( R_0 \) in some neighborhood of 1, then that solution \( y_2^* \) will be positive for \( R_0 > 1 \), and negative for \( R_0 < 1 \) (again in some neighborhood of 1). This corresponds to a “forward” bifurcation in \( R_0 \), in that a meaningful (nonnegative) solution will exist only for \( R_0 > 1 \). If instead \( f'(0) < 0 \) in a neighborhood of \( R_0 = 1 \), then the solution which is zero for \( R_0 = 1 \) will be positive for \( R_0 < 1 \) and negative for \( R_0 > 1 \), corresponding to a “backward” bifurcation. Thus we need only show that \( f'(0) > 0 \) for \( R_0 \) sufficiently close to 1.

Calculations show that

\[
f'(0) > 0 \iff \alpha > \mu \frac{2(\mu + \phi) + \rho \sqrt{\mu+\gamma}}{2 + \left(\frac{\mu+\gamma}{\gamma}\right) \frac{\mu+\phi+\rho}{\delta}} (\mu + \phi) + \frac{\gamma}{\mu+\gamma} \delta \rho. \tag{13}
\]

If we define the quantities

\[
\alpha_1 = \mu \frac{\mu + \phi + \rho}{\mu + \phi + \rho \frac{\mu+\gamma}{\mu+\gamma}} \quad \text{and} \quad \alpha_2 = \mu \frac{2(\mu + \phi) + \rho}{2 + \left(\frac{\mu+\gamma}{\gamma}\right) \frac{\mu+\phi+\rho}{\delta}} (\mu + \phi) + \frac{\gamma}{\mu+\gamma} \delta \rho,
\]

then (12) becomes \( R_0 > 1 \iff \alpha > \alpha_1 \), and (13) becomes \( f'(0) > 0 \iff \alpha > \alpha_2 \). Now we can observe that \( \alpha_2 < \mu \frac{2(\mu + \phi) + \rho}{2(\mu + \phi) + \frac{\mu+\gamma}{\mu+\gamma} \rho} \alpha_1 \), so that indeed for \( \alpha \) close enough to \( \alpha_1 \) — or, correspondingly, \( R_0 \) close enough to 1 — we must have \( \alpha > \alpha_2 \), and thus \( f'(0) > 0 \). \( \Box \)

We next observe that when \( R_0 = 1 \) (\( \alpha = \alpha_1 \)) we know one solution (zero) to the equilibrium condition (11), so factoring it out we are left with the quadratic \( Ay_2^2 + By_2 + C |_{\alpha = \alpha_1} = 0 \), which we can easily solve for \( y_2^* \):

\[
y_\pm = \left\{ \frac{(\mu+\gamma)}{(\mu + \phi + \rho)} \left( 2(\mu + \phi) + \left( 1 + \frac{\mu}{\mu+\gamma} \right) \rho \right) \pm \rho \sqrt{K} \right\}, \tag{14}
\]

where

\[
K = (\mu + \phi + \rho)^2 + 2 \frac{2\mu + \gamma}{\gamma} (\mu + \phi + \rho) \frac{\gamma}{\mu + \gamma} \delta \rho + \left( \frac{\gamma}{\mu + \gamma} \right)^2 \delta^2.
\]

It follows from (14) that \( y_+ > y_- > \frac{\mu + \phi}{\delta} \) (by completing the square inside the radical), so both nonzero solutions to (11) are outside the state space. Since we have seen that \( f \left( \frac{\mu + \phi}{\delta} \right) > 0 \), the only way to have multiple endemic equilibria inside the state space (i.e., meaningful) is first to have a bifurcation where these two solutions \( y_+, y_- \) outside the state space vanish, and then to have a second bifurcation in which another pair arises inside the state space. Numerical analysis indicates that the number of solutions to (11) is always three, in which case no meaningful multiple endemic equilibria can occur.

Finally, since \( \delta \) is a bifurcation parameter independent of \( R_0 \), we consider in the subsection below two special cases: the extremes of \( \delta \).
3.3 Special Cases in $\delta$

Since we are interested in the effects of peer pressure, we now consider two special cases in $\delta$, which we recall is independent of $R_0$. If we consider the case $\delta = 0$, we find that $A = B = 0$ in (11). This makes the endemic equilibrium condition linear. Therefore if $\delta = 0$, there is never more than one endemic equilibrium. Our other special case is the opposite extreme, when everybody in $B_2$ gets treated: when $\delta \to \infty$.

Can we have multiple endemic equilibria for arbitrarily large $\delta$? Consider the part of $f(y_2)$ which has $\delta^2$,

$$f(y_2) = \delta^2 \left( \frac{\mu + \gamma}{\gamma} \right) \left[ \left( \frac{\mu + \gamma}{\gamma} \right) \alpha y_2^3 - (\alpha - \mu)y_2^2 \right] + \ldots$$

If $\delta$ is very big then this part must be zero at an endemic equilibrium $y_2^*$. This means either

$$(y_2^*)^2 = O \left( \frac{1}{\delta} \right)$$

or

$$\left( \frac{\mu + \gamma}{\gamma} \right) \alpha y_2^* - (\alpha - \mu) = O \left( \frac{1}{\delta} \right),$$

i.e.,

$$y_2^* = \frac{\alpha - \mu}{\alpha} \frac{\gamma}{\mu + \gamma} + O \left( \frac{1}{\delta} \right) \approx \frac{\alpha - \mu}{\alpha} \frac{\gamma}{\mu + \gamma}.$$

Suppose that $\alpha > \mu$. Then $y_2^* > 0$. In fact, for sufficiently large $\delta$, $y_2^* > \frac{\mu + \phi}{\delta}$. Thus, we have three points where we know $f$: 0, $\frac{\mu + \phi}{\delta}$, and $y_2^*$. Furthermore, we can show $f'(y_2^*) > 0$. Now, since we also know that $f(y_2) \to -\infty$ as $y_2 \to -\infty$ and $f(y_2) \to \infty$ as $y_2 \to \infty$, we can establish where the zeroes of $f$ must lie. If $R_0 < 1$, we have $f(0) > 0$, $f \left( \frac{\mu + \phi}{\delta} \right) > 0$, $f(y_2^*) = 0$, $f'(y_2^*) > 0$, so there must be a zero crossing to the left of 0, another one between $\frac{\mu + \phi}{\delta}$ and $y_2^*$ (since $f'(y_2^*) > 0$), and the third at $y_2^*$, near $\frac{\alpha - \mu}{\alpha} \frac{\gamma}{\mu + \gamma}$. If $R_0 > 1$, we have $f(0) < 0$, $f \left( \frac{\mu + \phi}{\delta} \right) > 0$, $f(y_2^*) = 0$, $f'(y_2^*) > 0$, and the only change is that the first zero crossing must be in $(0, \frac{\mu + \phi}{\delta})$, as promised by Proposition 1. In both cases the first two zeroes are the two $O \left( \frac{1}{\delta} \right)$ solutions promised above. In neither case is there an extra pair of endemic equilibria. Thus, if any ever exist, they must do so for intermediate values of $\delta$.

4 Parameter values and control strategies

We now estimate values for model parameters in order to determine model predictions. As some of the parameters can be estimated only very roughly (see below), our principal objective shall be to see how closely model behavior corresponds to empirical observations, and to suggest directions for control measures and further data-gathering.

In calculating the removal rate, $\mu$, we take into consideration the average time an individual remains in a given college population. This statistic is not readily available but should take into account departures due to other reasons than graduation, including transfers and
dropping out. Bradburn (2002) notes that 32% of the students who began their postsecondary education in the 1995–96 school year had left without a credential, and without returning, by the end of the spring 1998 term. THECB (2003) reports that over 40 percent of students left their Texas postsecondary institutions without graduating (this includes transferring). We will therefore use an estimate of three years, $\mu = 3$, making $\mu = \frac{1}{3}$ years$^{-1}$. This gives us a reference by which to measure other rates through proportions of bulimics undergoing treatment, relapse, etc. below.

In the absence of accurate data on progression of symptoms, we estimated that it takes between half a year and one year to pass to the more advanced stage of bulimia ($B_2$). For the rate $\gamma$ of going from $B_1$ to $B_2$, this gives a value in the range of 1 to 2 years$^{-1}$; we took $1.5$ years$^{-1}$.

The average per capita treatment rate is $\rho + \delta z$, where $z$ is the proportion of the population in treatment, as in (1–5). One non-interventive study ($n = 102$) found that 34% of the subjects had the disorder for at least fifteen months, 15% had it for five years or more, and only 28% ever entered treatment (Fairburn, Cooper, Doll, Norman & O’Connor, 2000). We therefore take for the proportion of bulimics who enter treatment before leaving the population $\rho + \delta z / (\mu + \gamma)$, which, with the above value for $\mu$, gives $\rho + \delta z \approx .13$ years$^{-1}$.

Mitchell (1990) gives the mean time till seeking treatment as 6 to 7 years; inverting this gives an estimate for $\rho + \delta z$ of .14 to .17 years$^{-1}$, which is close to the above value of .13 years$^{-1}$.

We take the above treatment proportion together with the conservative 4% prevalence estimate reported by Halmi et al. (1981), Drewnowski et al. (1988) and others, we obtain an estimate for $z$ of $z = (.28)(.04) \approx .01$. This means that if $\rho$ and $\delta$ are of the same order, then $\rho + \delta z \approx \rho$, and we can take $\frac{\rho}{\mu + \rho} \approx .28$ and $\rho \approx .13$ years$^{-1}$. Since $\delta$ measures the pressure exerted by peers, while $\rho$ measures family or healthcare professionals discovering symptoms and obliging the bulimic to enter treatment, it is difficult to compare their orders: there is no available data measuring peer pressure to seek treatment, and we found no studies which estimated the relative importance of the two factors in leading bulimics to seek treatment. However, we will proceed under the above assumption that the two parameters are of the same order, which will give us a value for $\rho$.

The proportion of bulimics in treatment who relapse before leaving the population is $\phi / (\mu + \phi)$. Keel & Mitchell (1997) found relapse rates ranging from 26% to 43%, and centered around 30%. Olmsted, Kaplan & Rockert (1994) observed a 31.25% relapse rate, so we take $\phi / (\mu + \phi) = .3125$, which gives $\phi = .15$ years$^{-1}$.

The average per capita rate at which women in the population develop bulimia is $\alpha (y_1 + y_2)$, with $y_i$ as in (1–5). Although $\alpha$ is a peer pressure parameter, we can estimate it using prevalence data (ideally longitudinal, as we will discuss below, but in the absence of such data for our population we will use the single figure given earlier). If we define $Y = y_1 + y_2$ and suppose that the susceptible proportion is changing very slowly ($dx/dt \approx 0$, though not necessarily at equilibrium), we can rewrite equation (1) as $\alpha = \mu (1 - x) / x Y$. Using the conservative estimate of 4% (Drewnowski et al., 1988) given earlier for $Y$ and the above estimate $z \approx .01$, this gives $x \approx .95$ and $\alpha = .439$ years$^{-1}$.

These estimates give us $\alpha / (\mu + \gamma) = .239$, which is indeed greater than the corresponding estimate for $\frac{\phi}{\mu + \phi} - \frac{\rho}{\mu + \rho} = .28(.3125) = .0875$, thereby justifying (7) and the associated discus-
sion in the previous section. These estimates also yield values of $R_0 \approx 1.05$, consistent with our initial observation that bulimia is present in an endemic state in the college population.

This leaves us with the role of $\delta$, and the potential effects of $\sigma$, to consider. If we again consider $\delta$ with respect to $\rho$, we can see that the effect of increasing this peer-pressure treatment rate is to reduce the size of the bulimic population by as much as 80%. If $\delta$ is no bigger than $\rho = 0.13$ years$^{-1}$, we find the bulimic population $y_1 + y_2 \approx 0.065$, or 6.5% of the total. If instead $\delta$ is two orders of magnitude greater than $\rho$, that is, $\delta z \approx \rho$, then the resulting $\delta = 13$ years$^{-1}$ yields $y_1 + y_2 \approx 0.014$. (If we set $\delta = 13$ years$^{-1}$ and adjust $\rho$ to zero to keep $\rho + \delta z \approx 0.13$ years$^{-1}$, then $R_0$ rises to 1.16, but $y_1 + y_2$ reduces only to 0.045.) An intermediate value of $\delta$ will yield the estimate used earlier of 0.04; this suggests that the force of peer pressure to seek treatment for bulimia is greater than that of caregivers obliging the bulimic to seek treatment, but its effectiveness is limited by the small size of the support group providing the pressure ($z$).

We see that $\delta$ affects the size of the bulimic population but apparently not whether it persists; as noted earlier, $R_0$ is independent of $\delta$. However, this is not entirely surprising, as $\delta$ measures an avenue of control which reduces only the number of advanced-stage bulimics, and does not address the onset of the disorder. We therefore turn to the notion of a public education campaign $\sigma$ which could deter some potential bulimics from developing the disorder. If we consider $\alpha$ as a decreasing function of $\sigma$, then it might be possible to reduce $\alpha$ below the threshold value $\alpha_1$ where $R_0 = 1$. For the parameter values above, this corresponds to a decrease of only 8% in $\alpha$; such a modest drop seems well within reach — but it is not at all clear what the cost would be for the campaign to achieve such a reduction. Even a small drop in $\alpha$ may require enormous effort, as it involves targeting not only potential bulimics but the entire peer group and its cultural standards, which drive the peer-pressure term.

We might also wish to consider the effects of an education campaign on the rate $\gamma$ at which symptoms advance, but in this model the only way to recovery is through the advanced bulimia stage, so slowing the process down only appears to make the situation worse, by keeping individuals bulimic longer. (An exception to this conclusion is the case where $B_2$ individuals contribute more to the peer pressure than $B_1$ individuals, which would necessitate weighting terms for $y_1$ and $y_2$ in equations (1) and (2).) A more productive approach would be to encourage bulimic individuals to seek help before symptoms become more severe. Incorporating such a change into our model would necessitate an extra term in the equations for $y_1$ and $z$; this term would decrease $R_0$ only if it reflected broad-based cultural pressure (a constant per capita rate) rather than a peer pressure term dependent only on individuals currently in treatment. Otherwise its role would be similar in nature to that of $\delta$.

At this point some discussion is in order regarding the measuring of peer pressure, which has typically been done by means of survey instruments. Early validated measures designed to assess various indices related to the perception of peer pressure tended to be rather long (40–50 items or more), and consequently difficult to administer reliably to some of the adolescent population at whom they are targeted. More recently, shorter tests have been developed (and validated) to measure peer pressure (e.g., Santor et al., 2000; the McKnight investigators, 2003). These measures tend to be more narrowly focused, e.g., on peer pressure to exhibit antisocial behavior, or to change eating behaviors. Survey questions may ask about perceived pressure to exhibit a particular behavior, or about the subject actually having done
it as a result of such pressure. To measure the force of peer pressure in spreading bulimia within a given population, as in our model, it is necessary to target instruments toward the effective peer pressure to conform to cultural body-shape norms via eating behaviors, that is, the average extent to which peer pressure actually influences the decision-making process.

However, in estimating peer-pressure-based parameters for a dynamical system, there is another important distinction. Studies such as the McKnight study examine the relative importance of different factors in the spread of a given behavior. The model described in this paper assumes (based on conclusions of studies such as those cited above) that collective, peer-pressure-based forces are the driving force for initial development of bulimic symptoms (unlike the pressure to seek treatment, which we have modeled as having both a collective component and an environmental component). The dimensionless measures of peer pressure (and other factors) described in the previous paragraph therefore serve primarily to guide the assumptions that shape a model; parameter estimates for a dynamical system involving time must be based on longitudinal incidence data. The parameter $\alpha$ in our model is a rate with per-time units, rather than a dimensionless incidence rate or correlation, and so requires a comparison of the overall per capita rate at which a given population becomes bulimic, to the prevalence rate of bulimia within the population (the quotient of which should determine $\alpha$). For example, using the aggregate data of the McKnight study, 32 new cases of eating disorders developed in a population of 1103 middle-school girls over 3 years, at the beginning of which time there was a 2.5% prevalence of eating disorders in the baseline population. This gives a per-capita rate of growth in incidence of $\frac{32}{1103}/3\text{yr} \approx 0.01\text{yr}^{-1}$ and an estimate $\alpha \approx \frac{0.01}{0.025} = 0.4\text{yr}^{-1}$, not far from the rough estimate of $0.439\text{yr}^{-1}$ obtained above for a college-age population. (The conversion of Likert-scale peer pressure data directly into per capita transition rates will remain an open problem until such time as multiple data sets with measures of both peer pressure and prevalence are available for regression purposes.)

The test of the model comes in the extent to which it can account for observed data and make new predictions, relative to an alternative. In this case, an alternative might be that instead bulimia develops based primarily on factors independent of the current incidence of bulimia in the population. Removal of the $y_1+y_2$ term in equation (1) makes the model linear (except for $\delta$) and eliminates the bulimia-free equilibrium, predicting instead an asymptotic prevalence $y_1 + y_2 + z = \frac{\alpha}{\mu + \alpha}$. In this case our original estimate for $\alpha$ in a college population would be $\mu(1-x)/x \approx 0.0175\text{yr}^{-1}$, while the value estimated from the McKnight study becomes a significantly lower $0.0097\text{yr}^{-1}$.

A further test of the model might compare the respective levels of peer pressure and bulimia in different populations. Some studies have identified subgroups of the same population with significantly different levels of peer pressure (e.g., different social cliques (Lieberman, 2001)) and/or prevalences of eating disorders (e.g., Hispanic vs. white or African-American girls (Striegel-Moore et al., 2000; McKnight investigators, 2003)). Comparing peer pressure and prevalence data from such paired populations can lead to a better understanding of the quantitative relationship between the two measures.
5 Conclusion

The model for bulimia considered in this paper is just the barest sketch of a complex social phenomenon, an initial model for a disorder which has historically presented difficulties in standardizing clinical diagnosis. Nevertheless, its analysis accounts for some of the population dynamics underlying the spread of the disorder and the recommendations to focus control strategies on multilevel interventions and decreasing peer pressure (e.g., Lieberman, 2001). It also provides a quantitative basis for distinguishing the relative contributions of different factors involved. Our conclusions have come to focus through the lens of the disorder’s basic reproductive number, \( R_0 \) — an environment’s conduciveness to collective transmission of the disorder via pressure to conform to unhealthy cultural norms — which appears to determine whether bulimia will persist in the population at risk. The form of \( R_0 \) reflects the structure of the disorder (for example, the two stages of early and advanced symptoms modeled here), and highlights the driving forces behind it. In particular, we have seen that the terms based on peer pressure drive only those transitions which do not involve more environmental or societal forces: the peer-pressure-based “infective” force proves the gateway to control, but it is society’s ability (through family and caregivers) to get bulimics into treatment that appears in \( R_0 \), rather than the peer-pressure-based treatment rate. Instead, pressure from peers for bulimics to seek treatment plays a role in reducing the endemic levels at which the disorder exists. Efforts to prevent or eradicate such endemic levels must therefore address the societal pressures which influence individuals’ entry into the binge-purge cycle (cf. Hotelling, 1989).

Our analysis also found the treatment currently offered to advanced-stage bulimics to be inefficient in eradicating the disorder because it is not well-placed. In the scenario modeled, bulimic individuals need to get worse before any treatment begins. Treatment of individuals at an earlier stage offers a higher potential for population-level control of the disorder by reducing the amount of time that bulimics contribute to unhealthy peer pressure (treatment in early stages may also cost less than treatment of more advanced bulimia). Since females in the early stages of bulimia rarely seek treatment, any efforts to encourage earlier treatment will require an education campaign to make these students realize that they have a problem that needs to be treated. We have seen that peer-based encouragement to seek help could significantly reduce the number of bulimics. It is also important to consider any campaign of education and treatment based on self-referral in the light of expected utility, using diagnostic-efficiency statistics to minimize the cost generated by false positives (e.g., Neufeld, 1977). Education and treatment programs should therefore be targeted carefully to ensure a high base rate of genuine need for treatment. One simple four-question screen for potential eating disorders developed by the McKnight investigators (2003), for example, had a sensitivity of 0.72, a specificity of 0.80, and an efficiency of 0.79.

Even though statistics show that the prevalence of bulimia has risen dramatically in recent years, the amount of public concern does not seem to have undergone a similar increase. However, the analysis in preceding sections suggests that public education campaigns must be systemic in order to succeed: The per capita transmission rate is modeled by the product of the current bulimia incidence (in terms of the state variables) and the strength of cultural norms in influencing at-risk women to become bulimic (given by the parameter \( \alpha \)), and to the extent that \( R_0 \) drives the disorder, \( \alpha \) drives \( R_0 \). Efforts at public education, which commonly
aim to clarify why eating disorders are dangerous, must therefore also make the connection to the cultural norms and pressures that lead many women to develop distorted self-images. Counseling, for example, made not only available but encouraged for all students, could help eliminate the identification of thinness with self-esteem (cf. Stice, 1999) while supporting healthy weight management.

Future work, then, should consider more specifically the effects of a public education campaign on all segments of the population, especially as prevention for those vulnerable but not yet bulimic, and encouragement to seek treatment early for those bulimics whose symptoms have not yet become advanced. Measuring the effects of collective societal forces such as public education and peer pressure remains difficult, but is crucial in order to understand the dynamics of disorders such as bulimia and anorexia which are driven by those forces. Further clinical studies can lead to better estimates of these forces, as well as the rate at which symptoms become severe and the relative importance of the factors which presently lead individuals to seek treatment.

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Appendix

Calculation of $R_0$

$R_0$ for model (1–5) can be computed as follows via the next generation operator approach (Diekmann et al., 1990; Castillo-Chávez et al., 2002). Take $y_1$ and $y_2$ as the infective compartments and $x$ as the susceptible compartment. Solve for the equilibrium value $z^*$ in equation (4) and substitute it in equation (3) to put $dy_2/dt$ in terms of $y_1$ and $y_2$. The Jacobian of $y_1, y_2$ evaluated at the bulimia-free equilibrium is then

$$J = \begin{bmatrix} -\gamma - \mu + \alpha & \frac{\alpha}{\gamma} \\ \frac{\alpha}{\gamma} & \frac{\alpha}{\mu + \phi} - \rho - \mu \end{bmatrix}.$$ 

We then write $J$ in the form $J = M - D$, with $M \geq 0$ elementwise and $D > 0$ diagonal, and compute $MD^{-1}$:

$$MD^{-1} = \begin{bmatrix} \frac{\alpha}{\mu + \gamma} & \frac{\alpha}{\mu + \rho} \\ \frac{\alpha}{\gamma + \mu} & \frac{\alpha}{(\mu + \phi)(\rho + \mu)} \end{bmatrix}.$$
$R_0$ is the dominant eigenvalue of $MD^{-1}$, namely

$$R_0 = \frac{1}{2} \left[ \frac{\alpha}{\mu + \gamma} + \frac{\phi \rho}{(\mu + \phi)(\mu + \rho)} + \sqrt{\left( \frac{\alpha}{\mu + \gamma} - \frac{\phi \rho}{(\mu + \phi)(\mu + \rho)} \right)^2 + \frac{4 \gamma \alpha}{(\mu + \rho)(\mu + \gamma)}} \right].$$

**Coefficients of equation (11)**

$$A = \alpha \left( \frac{\mu + \gamma}{\gamma} \right)^2 \delta^2$$

$$B = \left( \frac{\mu + \gamma}{\gamma} \right) \delta \left[ \alpha \rho - 2 \left( \frac{\mu + \gamma}{\gamma} \right) \alpha(\mu + \phi + \rho) - \delta(\alpha - \mu) \right]$$

$$C = \left( \frac{\mu + \gamma}{\gamma} \right) \delta \left[ 2(\mu + \phi + \rho)(\alpha - \mu) - \delta \alpha \rho \right.$$

$$+ \left( \frac{\mu + \gamma}{\gamma} \right)^2 (\mu + \phi + \rho)^2 \alpha - \left( \frac{\mu + \gamma}{\gamma} \right)(\mu + \phi + \rho) \rho \alpha \left. \right]$$

$$D = (\mu + \phi) \left[ \alpha \rho - \left( \frac{\mu + \gamma}{\gamma} \right)(\mu + \phi + \rho)(\alpha - \mu) \right]$$

**References**


