Abstract

We develop a mathematical model based on the hypothesis that one of the most significant causes of major depressive disorder in young women is the pressure they are under from the media and their peers to attain a stereotypical model of “beauty”. Empirical evidence supports the view that the U.S. media’s archetype of beautiful women has adverse effects on women’s mood and self-esteem. The media’s impact is moderated by individual differences in the level of internalization of the thin-figure archetype. Diminishing the role of the media is impractical. Hence our model focuses on identifying the means by which women become vulnerable to its effects. It is assumed that women enter the system in one of two classes – a high vulnerability and a low vulnerability group. Once in the system, peer pressure will drive women’s movement between these two groups. The impact of the flow between the two vulnerability groups, treatment, and relapse are explored using a nonlinear system of four differential equations.

1 Introduction

There are currently about seven million women in the United States who meet the criteria for clinical depression (National Institute of Mental Health, 2002). There is a highly reliable finding that women are approximately twice as likely as men to suffer from depression, both clinical and subclinical, and that this holds even when gender differences in self-reporting behavior are accounted for (Nolen-Hoeksema, 2001; Nolen-Hoeksema & Girgus, 1994). Why are women so likely to suffer from depression? Firstly, we note that the gender difference in depression emerges quite suddenly at around puberty: girls begin to show significantly higher rates of depression than boys between the ages of thirteen and fifteen (Nolen-Hoeksema, 2001; Nolen-Hoeksema & Girgus, 1994). Furthermore, girls who go through puberty earlier are more likely to suffer from depression (Nolen-Hoeksema, 2001). These effects do not appear to be due to hormonal changes (2001). Since the hormonal changes occurring during puberty do not account for the dramatic increase in rates of depression in girls at this age, the next most probable explanation for the observed increase is that there are social pressures that emerge or that become more salient at this developmental stage.

One possible explanation for the gender difference in depression that emerges at puberty is that girls at this age are undergoing physical changes in their bodies (often including weight gain) at
the same time that they are receiving increased pressure to fulfill their gender role – an important part of which is to be attractive. That is, at this age the pressure on girls to be feminine drastically increases, and an important part of being feminine in this culture is to be physically attractive. Further, being physically attractive in our culture means having an extremely thin figure – one that is unattainable for the vast majority of women. The primary means through which this standard of beauty is propagated is the mass media, which bombards us with images of women who on average weigh significantly less than the average American woman.

It is evident from the literature (reviewed in the next section) that media portrayals of female beauty have a significant negative effect on the mental health of women – in particular, on their mood and self-esteem. There is also evidence that this effect is moderated (or at least mediated) by an individual difference variable, namely the degree of “internalization of societal pressures regarding prevailing standards of attractiveness”\(^1\) (hereafter referred to as “degree of internalization”) (Thompson & Heinberg, 1999, p. 339).

Based on this line of research, we develop a mathematical model of depression in women between the ages of 15 and 24. We assume that the major cause of depression in this population is the dissatisfaction women feel with themselves and their bodies because of the pressure placed on them, by both their immediate community and the mass media, to conform to our society’s unattainable standards of beauty. Women enter the population into one of two classes based on their degree of internalization: the highly vulnerable class (those with high internalization – \(V_1\)), and the less vulnerable class (those with low internalization – \(V_2\)). From the highly vulnerable class, women move at a constant rate to the depressed class (defined as those women meeting the DSM-IV criteria for major depression) due to the negative effects of the media images to which they are exposed. Women move between the two vulnerability classes as function of peer pressure (the highly vulnerable and depressed classes exert peer pressure upon the less vulnerable class, and the less vulnerable class exerts pressure upon the highly vulnerable class). From the depressed class, some women move to the treatment class, and, if treatment is successful, they then move to the less vulnerable class. If treatment is unsuccessful, they are assumed to relapse – that is, they move back to the depressed class.

In analyzing this model, we find that there is always at least one, apparently stable, endemic equilibrium. This value will be either small or large depending on the strength of the two per capita peer pressure terms (\(\varepsilon\) and \(\delta\)) relative to each other. The only other parameter that effects the size of the endemic equilibrium significantly is the treatment parameter. We find that there is a disease-free equilibrium only in the case where no one enters the system in the highly vulnerable class, and we use this equilibrium to find \(R_0\). We interpret \(R_0\) in terms of the model. Implications for reducing prevalence and future directions for research are discussed.

2 Empirical evidence

The question of how current standards of beauty – that emphasize thinness so strongly – affect women’s attitudes towards their own bodies and their general psychological well-being has begun to be addressed in the psychological literature in last two decades. In particular, researchers have tested the hypothesis that increased exposure to media (while female images become increasingly thin) are responsible for increased rates of body dissatisfaction, depression, anxiety, and eating

\(^1\)There are numerous studies that have found such an individual difference variable, and in each, this variable is conceptualized differently. What is clear is that there is such a variable; what is still in need of further empirical study is exactly what this individual difference variable is. In the present study, we choose to use the definition proposed by Thompson and Heinberg in 1999.
disorders in women. “Researchers have concluded that females concern with weight and body image has attained epidemic proportions [in recent years] and is now considered a normal part of the female experience” (Posavac, Posavac, & Weigel, 2001, p. 325). It is of the utmost importance to address this at the moment, given the scope of media exposure in the United States concurrent with the strong disparity between media-portrayed images and real-life female appearance: according to Kilbourne (1999) “Americans are exposed to approximately 3,000 advertisements daily”; At the same time, “The average young woman is 5’4” and weighs 152 pounds (National Center for Health Statistics, 1997) while Caucasian teenage girls have described their “ideal girl” as 5’7”, weighing 110 pounds, and wearing a size 5 (Nichter & Nichter, 1991). Clearly, this ideal does not represent the average American woman – instead it reflects the image of beauty portrayed in the media. Furthermore, it is estimated that one in 40,000 women meet the approximate height, weight, and shape requirements to be a fashion model” (Wolf, 1991)

Social comparison theory has provided a useful framework for the discussion of the influence of media images on women. It has been proposed that women compare their bodies to the thin images presented in advertising and other media and become aware of the discrepancy between their bodies and models’ bodies: as a result of this social comparison process, women become dissatisfied with their own bodies and, after numerous and frequent such experiences, vulnerable women may become depressed and/or develop an eating disorder (Laird and Wilcox, 2000). Posavac and colleagues remark that “because the media’s preferred image of slim feminine attractiveness is so exaggerated, most of our participants were doomed to perceive a discrepancy between their bodies and that of the media standard when they compared their bodies with those of the fashion models” (2001). However, not all women are equally affected by the media. A negative response to media influence is particularly strong for those women who already demonstrate high internalization of the current societal definition of beauty and of the heavy emphasis placed upon being “beautiful”: the results of a 1994 study by Stice et al “support the hypothesis that internalization of sociocultural pressures mediates the relation between media exposure and eating pathology.” (Stice et al, 1994)

In a study performed by Posavac, Posavac, and Weigel, two experiments were done in order to investigate the relation between media exposure and weight concern in young women (2001). In the first experiment, 136 female undergraduates ages 18 to 25 were given a test that measured trait body dissatisfaction2, and were then randomly assigned to be exposed to either slides of fashion models selected from popular women’s magazines (Glamour, Vogue, Cosmopolitan) or neutral slides (car images). A test measuring weight concern was given after exposure to the slides. 3 Women who showed initial body satisfaction (scores between 0 and 5 in the EDI-2) were unaffected by exposure to media images. Women who had initial body dissatisfaction (scores of 6 or higher) demonstrated significantly more weight concern after viewing images of thin women than did the dissatisfied women who were exposed to neutral images. The second experiment replicated the conditions of the first one, but added a condition in which participants were shown slides of women who were attractive but not extremely thin. As predicted, the women who had shown initial body dissatisfaction and were exposed to images of fashion models showed more weight concern than did the dissatisfied women who were exposed to images of attractive women who were not of abnormally thin weight (2001). These two experiments demonstrate that women who show initial body dissatisfaction are vulnerable to adverse effects of media exposure. The study also

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2Trait body dissatisfaction was measured with the EDI-2 (Eating Disorder Inventory, second edition) developed by Garner in 1991. The scale ranges from 0 to 27. A score of 0 indicates no body dissatisfaction, while a score of 27 reveals extreme dissatisfaction. (2001)

3Weight concern following exposure to the slides was measured with the Weight concern subscale of the Body Esteem Scale developed by Franzoi & Shields in 1984. The scale ranges from 10 to 50. Lower scores correspond to higher weight concern. (2001)
reveals that the detrimental effects of media exposure result from the fact that the images show unrealistically thin standards of beauty: images of more realistic beauty do not show the same adverse consequences. 

There is also direct evidence that poor body image may cause depression. In a recent study of low-income, African-American middle school students, Grant and her colleagues found that the girls in their sample (n=165) had significantly higher depression scores than their male counterparts. A similar pattern was observed with respect to body image: mean scores on the body dissatisfaction measure were significantly higher for girls than for boys, with higher scores indicating poorer body image (1999). The most significant finding, however, was that “gender no longer predicted a significant amount of the variance in depressive symptoms once body image was included in the equation.” (1999, p. 306). This means that body image is a moderator of the relationship between gender and depression – that is, it is reasonable to conclude that being female increases one’s risk of having poor body image, and that this in of itself increases one’s risk of suffering depression.

In sum, the media has significant effects on the mood and self-esteem of women, and this effect is either moderated or mediated by an individual difference variable such as degree of internalization or already existing body dissatisfaction.

3 The model

We construct a simple model for the dynamic process through which individuals may become depressed that includes, in a rather simplified way, the impact of exposure to media images of beauty ideals. More importantly, the model includes the role of peer pressure on these dynamics. That is, we focus on how people become more or less vulnerable. We concentrate on the population

\[ \varepsilon \left( \frac{V_1 + D}{N} \right) \]
\[ \delta \left( \frac{V_2}{N} \right) \]
\[ (1-q)\mu N \]
\[ p\beta \]
\[ (1-p)\beta \]

Figure 1: Model

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Figure 1: Model

The authors point out that the converse interpretation is not supported by the data – that is it does not appear that depression is a risk factor for poor body image (1999).
that is most affected, namely women between the ages of 15 and 24. At age 15, it is assumed that 
a woman has a probability $q$ of being highly vulnerable to socio-cultural pressures (such as the 
media), and a probability $(1 - q)$ of being less vulnerable. The highly vulnerable subpopulation 
is denoted $V_1$, while the less vulnerable is denoted $V_2$. The per-capita rate at which women turn 
15 and enter our system is $\mu$. This is also the per-capita rate at which they leave the system. 
Women who are less vulnerable can become more vulnerable as a result of peer pressure from 
those women who show high vulnerability and those who are depressed. This is consistent with 
evidence that family and peer pressure are directly related to the likelihood of being in the high 
vulnerability group (Agras et al, 2001). In our model, $\varepsilon$ is the rate at which less vulnerable women 
are affected by highly vulnerable and depressed women. Women who are very vulnerable can 
become less vulnerable as a result of the influence that less vulnerable individuals have on them. 
The rate at which more vulnerable women are affected by less vulnerable ones is denoted by $\delta$. 
In accordance with evidence found in the literature (see section 2), only highly vulnerable women 
become depressed as a result of media influence. We denote the rate of infection due to exposure to 
media images of ideal female beauty as $\alpha$, the subpopulation of women who are depressed as $D$, and 
the subpopulation undergoing treatment for depression as $T$, which usually consists of medication 
and therapy. Women who are depressed seek treatment at a per-capita rate that is nearly constant 
(NIMH). We denote this rate $\gamma$. Of the women who seek treatment, some recover and become less 
vulnerable, while others relapse. The probability of undergoing a successful treatment and moving 
from $T$ to $V_2$ is designated $p$. (Therefore the probability of relapsing is $1 - p$). $\beta$ is the rate at 
which women leave treatment.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>$N$</td>
<td>Total population of women in the US between the ages of 15 and 24</td>
</tr>
<tr>
<td>$q$</td>
<td>Probability of being highly vulnerable to cultural pressures at age 15</td>
</tr>
<tr>
<td>$\mu$</td>
<td>Per capita rate at which women enter and leave the system</td>
</tr>
<tr>
<td>$\varepsilon$</td>
<td>Per capita peer pressure rate exerted by the high vulnerability and depressed classes upon the low vulnerability class</td>
</tr>
<tr>
<td>$\delta$</td>
<td>Per capita peer pressure rate exerted by the low vulnerability class upon the high vulnerability</td>
</tr>
<tr>
<td>$\alpha$</td>
<td>Per capita rate at which media influence causes women to become clinically depressed</td>
</tr>
<tr>
<td>$\gamma$</td>
<td>Per capita rate at which depressed women seek treatment</td>
</tr>
<tr>
<td>$p$</td>
<td>Probability that treatment is successful</td>
</tr>
<tr>
<td>$\beta$</td>
<td>Per capita rate at which women leave treatment</td>
</tr>
</tbody>
</table>

Table 1: List of Parameters
4 Model Equations

\[
\frac{dV_1}{dt} = q\mu N + \varepsilon \left(\frac{V_1 + D}{N}\right) V_2 - \delta V_2 \frac{V_1}{N} - \alpha V_1 - \mu V_1, \\
\frac{dV_2}{dt} = (1 - q)\mu N + \delta \frac{V_2}{N} V_1 + \beta p T - \varepsilon \left(\frac{V_1 + D}{N}\right) V_2 - \mu V_2, \\
\frac{dD}{dt} = \alpha V_1 + \beta (1 - p) T - \gamma D - \mu D, \\
\frac{dT}{dt} = \gamma D - \beta (1 - p) T - \beta p T - \mu T.
\]

We rescale the variables by letting \( x = \frac{V_1}{N}, \ y = \frac{V_2}{N}, \ z = \frac{D}{N}, \) and \( w = \frac{T}{N} \). We get the equivalent system

\[
\frac{dx}{dt} = \mu q + \varepsilon (1 - x - z - w) (x + z) - \delta (1 - x - z - w) x - (\alpha + \mu) x, \\
\frac{dy}{dt} = (1 - q) \mu + \delta y x + \beta p w - \varepsilon y (x + z) - \mu y, \\
\frac{dz}{dt} = \alpha x + \beta (1 - p) w - (\gamma + \mu) z, \\
\frac{dw}{dt} = \gamma z - \beta w - \mu w,
\]

where \( x + y + z + w = 1 \).

Setting \( y = 1 - x - z - w \) reduces the system to the following three-dimensional system:

\[
\frac{dx}{dt} = \mu q + \varepsilon (1 - x - z - w) (x + z) - \delta (1 - x - z - w) x - (\alpha + \mu) x, \quad (1) \\
\frac{dz}{dt} = \alpha x + \beta (1 - p) w - (\gamma + \mu) z, \quad (2) \\
\frac{dw}{dt} = \gamma z - \beta w - \mu w. \quad (3)
\]

5 Equilibria

From Equations (2) and (3) we solve for \( z \) and \( x \) in terms of \( w \), and get

\[
z = \frac{\beta + \mu}{\gamma} w \\
x = \frac{(\gamma + \mu)(\beta + \mu) - \gamma \beta (1 - p)}{\alpha \gamma} w
\]

Substituting these expressions for \( z \) and \( x \) into Equation (1), we now have:
We look at the special case where $b/a = 1/2$. This is a positive equilibrium point whenever

\[
\frac{(\gamma + \mu)(\beta + \mu) - \gamma \beta (1 - p)}{\alpha \gamma} - \frac{\beta + \mu}{\gamma} + 1)
\]

is always positive. Hence, there is at least one real equilibrium.

The right hand side defines $f(w) = aw^2 + bw + c$, where

\[
a = \frac{\varepsilon}{\gamma}(\gamma + \mu)(\beta + \mu) - \gamma \beta (1 - p) + \beta + \mu)(\gamma + \mu)(\beta + \mu) - \gamma \beta (1 - p) - \beta + \mu - 1)
\]

\[
b = \frac{\varepsilon}{\gamma}(\gamma + \mu)(\beta + \mu) - \gamma \beta (1 - p) + \beta + \mu)(\gamma + \mu)(\beta + \mu) - \gamma \beta (1 - p) - \beta + \mu - 1)
\]

\[
c = \mu q.
\]

The discriminant, $\Delta$, given by

\[
\Delta = \left[\frac{\varepsilon}{\gamma}(\gamma + \mu)(\beta + \mu) - \gamma \beta (1 - p) + \beta + \mu)(\gamma + \mu)(\beta + \mu) - \gamma \beta (1 - p) - \beta + \mu - 1)
\]

\[
+ 4[\frac{\varepsilon}{\gamma}(\gamma + \mu)(\beta + \mu) - \gamma \beta (1 - p)]^2
\]

\[
+ \delta(\gamma + \mu)(\beta + \mu) - \gamma \beta (1 - p) + \beta + \mu)
\]

\[
+ \delta(\gamma + \mu)(\beta + \mu) - \gamma \beta (1 - p) + \beta + \mu)
\]

is always positive. Hence, there is at least one real equilibrium.

6 Bifurcation analysis

6.1 $q = 0$

We look at the special case where $q = 0$. In this case, $w = -b/a$.

\[
w = \frac{\varepsilon(\gamma + \mu)(\beta + \mu) - \gamma \beta (1 - p) + \beta + \mu)}{\alpha \gamma} - \frac{\delta(\gamma + \mu)(\beta + \mu) - \gamma \beta (1 - p)}{\alpha \gamma} - \frac{(\alpha + \mu)(\gamma + \mu)(\beta + \mu) - \gamma \beta (1 - p)}{\alpha \gamma}
\]

This is a positive equilibrium point whenever $a$ and $b$ have opposite signs.
6.1.1 $R_0$

When $q = 0$, there also exists a depression free equilibrium, where $x = 0$, $y = 1$, $z = 0$, and $w = 0$. It is easily shown (Appendix A) that

$$R_0 = \sqrt{\left(\frac{\alpha}{\gamma + \mu}\right)\left(\frac{\varepsilon}{\delta + \alpha + \mu - \varepsilon}\right) + \frac{\gamma \beta (1 - p)}{(\gamma + \mu)(\beta + \mu)}}$$

The equilibrium point $(0, 1, 0, 0)$ is locally asymptotically stable whenever $R_0 < 1$. For $R_0 > 1$, there exists an endemic equilibrium when $a$ and $b$ have opposite signs.

When $R_0 > 1$, there exist two equilibria: one at 0, and one positive endemic equilibrium. As $q$ increases from 0, the positive equilibrium moves right, while the zero equilibrium becomes negative.

When $R_0 < 1$, there exist two equilibria: one at 0, and one negative equilibrium. As $q$ increases from 0, the zero equilibrium moves right (becoming slightly positive), while the negative equilibrium becomes more negative.

Now we look at the interpretation of $R_0$.

1. $\frac{\varepsilon}{\alpha + \mu + \delta - \varepsilon} = \frac{\varepsilon}{\alpha + \mu - (\delta - \varepsilon)}$ is the proportion of people in $V_1$ who move to $D$, that is, the proportion of highly vulnerable individuals who become depressed or the probability of becoming depressed given that you are highly vulnerable. This term gives the rate of conversions from media effects, where $(\varepsilon - \delta)$ represents the “demographic” effect of peer pressure on the pool of most vulnerable people.

2. $\frac{\alpha}{\gamma + \mu}$ is proportion of $V_1$ that goes to $D$ due to peer pressure from $D$, that is, the proportion of highly vulnerable individuals who become depressed as a result of the influence of depressed individuals.

Hence, for the case $q = 0$, $\left(\frac{\varepsilon}{\alpha + \mu + (\delta - \varepsilon)}\right)\left(\frac{\alpha}{\gamma + \mu}\right)$ gives the proportion of people that are driven to depression from $V_1$ by the media. Note that we need $\alpha + \mu + \delta > \varepsilon$, or $\frac{\varepsilon}{\alpha + \mu + \delta} < 1$.

1. $\frac{\gamma}{\beta + \mu}$ is the probability of moving from $D$ to $T$, that is, the probability that one depressed person gets treatment before leaving the system.

2. $\frac{1}{\beta + \mu}$ is the average resident time in $T$, that is, the average length of time that an individual spends in treatment.

3. $\beta (1 - p)$ is the rate from $T$ to $D$, that is, the rate at which individuals in treatment relapse.

Hence, $(\frac{\gamma}{\beta + \mu})(\frac{1}{\beta + \mu})\beta (1 - p)$ is the contribution from $D$ to $T$ to $D$, that is, the number of secondary infections due to relapse.

In total, $R_0$ represents the number of secondary cases of depression caused by the interactions between high and low vulnerability individuals, by the media, and by relapse from treatment.

6.1.2 Case when $\varepsilon > \delta$

Some algebra (Appendix B) shows that, when $R_0 > 1$,

$$b = \frac{(\gamma + \mu)(\beta + \mu)(\alpha + \mu)}{\alpha \gamma}(R_0 - 1) + (\varepsilon - \delta)(\frac{\gamma \mu + \beta \mu + \mu^2 + \gamma \beta p}{\alpha \gamma}) > 0,$$
since $R_0 > 1$ and $\varepsilon > \delta$.
Additional algebra (Appendix C) shows that
\[
a = \varepsilon\left(\frac{\gamma + \mu + \beta + \mu - \gamma\beta(1-p)}{\alpha\gamma} + \frac{\beta + \mu}{\gamma}\right) - \delta\left(\frac{\gamma + \mu + \beta + \mu - \gamma\beta(1-p)}{\alpha\gamma} + \frac{\beta + \mu}{\gamma}\right)
\]
\[
= \left(\frac{\gamma\mu + \beta\mu + \mu^2 + \gamma\beta p}{\alpha\gamma}\right)\left(\frac{\gamma\mu + \beta\mu + \mu^2 + \gamma\beta p + \beta + \mu}{\gamma} + 1\right)(\delta - \varepsilon) - \varepsilon\left(\frac{\beta + \mu}{\gamma}\right) < 0,
\]
since $\varepsilon > \delta$.
Therefore, $b > 0$ and $a < 0$ when $\varepsilon > \delta$ and $R_0 > 1$. That is, there exists a positive endemic equilibrium.

6.1.3 Conclusion: bifurcation when $q = 0$

If $R_0 < 1$, then the depression free equilibrium is asymptotically stable. If $R_0 > 1$, then the depression free equilibrium is unstable, and a positive endemic equilibrium is born.

6.2 $q = 0$

If we let $w_1$ and $w_2$ be the solutions of $f(w)$, we know from the quadratic formula that $w_1w_2 = \frac{c}{a}$. We know that $c$ is always positive. $f(w)$ has one positive root and one negative root for $a < 0$. This means that the system has one positive equilibrium for $a < 0$. When $a > 0$, however, the two solutions have the same sign. This means that the system has two possible positive equilibria for the condition $a = 0$ when
\[
\varepsilon = \frac{\delta(\gamma\mu + \beta\mu + \mu^2 + \gamma\beta p)}{\gamma\mu + \beta\mu + \mu^2 + \gamma\beta p + \alpha\beta + \alpha\mu}
\]
Since $a < 0$ for $\varepsilon \geq \delta$ and $c > 0$, there is always one positive and one negative equilibrium. This case also holds when $\varepsilon$ is just slightly smaller than $\delta$. However, when
\[
\varepsilon < \frac{\delta(\gamma\mu + \beta\mu + \mu^2 + \gamma\beta p)}{\gamma\mu + \beta\mu + \mu^2 + \gamma\beta p + \alpha\beta + \alpha\mu}
\]
then $a > 0$. In this case, there exist two possible positive equilibria.
In conclusion, a bifurcation occurs at $a = 0$, which happens when
\[
\varepsilon = \frac{\delta(\gamma\mu + \beta\mu + \mu^2 + \gamma\beta p)}{\gamma\mu + \beta\mu + \mu^2 + \gamma\beta p + \alpha\beta + \alpha\mu}
\]

7 Parameter estimates

The parameter values used for the simulations are listed in Table 2.

1. $q$ is the probability of being highly vulnerable at age 15. We estimate that about two out or every three women show high internalization of cultural standards at this age, and are therefore classified as highly vulnerable. Therefore, we estimate $q$ to be $\frac{2}{3}$. 9
Table 2: List of Parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$q$</td>
<td>$\frac{2}{3}$</td>
</tr>
<tr>
<td>$\mu$</td>
<td>$(\frac{1}{9})(\frac{1}{365})$</td>
</tr>
<tr>
<td>$\varepsilon$</td>
<td>$\frac{1}{2}$</td>
</tr>
<tr>
<td>$\delta$</td>
<td>$\frac{1}{5}$</td>
</tr>
<tr>
<td>$\alpha$</td>
<td>$\frac{1}{180}$</td>
</tr>
<tr>
<td>$\gamma$</td>
<td>$(\frac{1}{3})(\frac{1}{270})$</td>
</tr>
<tr>
<td>$\beta$</td>
<td>$\frac{1}{365}$</td>
</tr>
<tr>
<td>$p$</td>
<td>$0.8$</td>
</tr>
</tbody>
</table>

2. $\frac{1}{\mu}$ is the average life span in the system. Since we are looking at the female population between the ages of 15 and 24, we take the average life span to be 9 years, or $9(365)$ days. Therefore, $\mu = \frac{1}{9(365)}$.

3. $\varepsilon$ is the product of two terms: the number of contacts that a person in $V_2$ has with individuals in $V_1$ and $D$ and the “infection probability” of these contacts. We define a contact to be a conversation of at least five minutes on the topic of beauty, and assume that there are about five such contacts per day, and that ten of them are required for “infection”. Hence, $\varepsilon = 5(\frac{1}{10}) = \frac{1}{2}$.

4. $\delta$ is also the product of two terms: the number of contacts that a person in $V_1$ has with individuals in $V_2$ and the “infection probability” of these contacts. We estimate that a highly vulnerable individual has about five such contacts per day, and that twenty-five contacts are necessary for “infection”. Accordingly, $\delta = 5(\frac{1}{25}) = \frac{1}{5}$.

5. $\frac{1}{\alpha}$ is the average time that an individual spends in the high vulnerability class before becoming depressed. We estimate this to be about six months, that is, 180 days. Therefore, $\alpha = \frac{1}{180}$.

6. $\gamma$ is the product of two terms. The first one is the probability of seeking treatment, and the second one is the reciprocal of the average time spent in $D$ before going to $T$. According to the National Institute of Mental Health, about one third of women who are depressed seek treatment. Consequently, the first term is $\frac{1}{3}$. We estimate the time that an average woman stays depressed before seeking treatment to be about nine months (i.e., 270 days), so the second term is $\frac{1}{270}$. Therefore, $\gamma = \frac{1}{3}(\frac{1}{270})$.

7. $\frac{1}{\beta}$ is the average time of treatment. This is about one year, or 365 days. Hence, $\beta = \frac{1}{365}$.

8. $p$ is the probability that treatment is successful, that is, the probability that an individual in $T$ will move to $V_2$. According to the National Institute of Mental Health, about 80 percent of treatments for depression result in recovery. Therefore, $p = 0.8$.

According to the 2000 census, the number of women between the ages of 15 and 24 in the United States is 19,936,400. This is the value for $N$ in our model. In order to find the value for $D$, we found the total number of men and women in the age range of interest. This number is 39,183,891. According to the National Institute of Mental Health 6.1 percent of the U.S. population suffers from major depression. Of the total depressed population, two thirds are women. Therefore, $D = 39,183,891(0.061)(\frac{2}{3}) = 1,593,478$. The value for $T$ was found by multiplying $D$ by $\frac{1}{3}$, since about one third of women who are depressed seek treatment (NIMH). $T = 531,159$ The remainder of the population of interest (women between the ages of 15 and 24) corresponds to $V_1 + V_2$. About one third of them are in $V_2$, and two thirds are in $V_1$. Therefore, $V_2 = 5,937,254$ and $V_1 = 11,874,508$.

From the values of $V_1$, $V_2$, $D$, $T$, and $N$, we get the initial values for our rescaled variables.
<table>
<thead>
<tr>
<th>Variable</th>
<th>x</th>
<th>y</th>
<th>z</th>
<th>w</th>
</tr>
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<tbody>
<tr>
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<td>0.59</td>
<td>0.30</td>
<td>0.08</td>
<td>0.03</td>
</tr>
</tbody>
</table>

Table 3: Initial Conditions

8 Simulations and interpretation

8.1 Solutions for the System

Using the estimated parameters and initial values, we numerically solve our model. The results are shown in figure 1.

As can be seen from the graph, the solutions always reach stability. The population in $V_1$ decreases, as the populations in $D$ and $T$ increase.
8.2 Effect of $\varepsilon$

Figure 3 shows the bifurcation that occurs when $\varepsilon \approx 0.0378$. For values of $\varepsilon$ less than the bifurcation value, the depressed population remains close to zero. However, when $\varepsilon$ becomes greater than the bifurcation value, the depressed population grows rapidly and reaches a stable point.

If we plot this graph for various $\gamma$, we see that $\gamma$ is very important (see figure 4): increasing $\gamma$ causes a significant decrease in the value of the larger equilibrium. This means that one way to significantly reduce the number of people in the depressed class is to increase either the rate at which people go into treatment, or more likely, increase the probability that people will seek treatment. That is, encouraging people who are currently depressed to get treatment will not only benefit those people, but it will benefit the entire community because it will result in both a decrease in the pressure to move from $V_2$ to $V_1$ (less to more vulnerable) and an increase in the pressure to move from $V_1$ to $V_2$ (more to less vulnerable).
Note that the highest curve corresponds to increasing our probability of seeking treatment from \( \frac{1}{3} \) to \( \frac{1}{2} \), and the greater equilibrium for this value is approximately .5 (down from .6 for the original probability). The next curve corresponds to increasing the probability of seeking treatment to one, and the next corresponds to decreasing the average time it takes for a depressed person to seek treatment from nine months to three months (with the probability still equal to one). The lowest curve corresponds to \( \gamma = 1 \), which would mean that everyone seeks treatment after one day of being clinically depressed – this is highly unrealistic, but we can see that as we tend toward this situation, the equilibrium tends to zero.

One would expect that changing the probability of entering the system into the high vulnerability class would have a significant effect on the outcome, but it does not: \( q \) is not a sensitive parameter. In fact, \( q \) effects no visible change in the graph at all when its value is varied from .1 to .9. This is interesting because it implies that in our model it is relatively unimportant what happens to you before you are fifteen, which contradicts predominant thought.

In order for alpha to have a significant effect on the behavior of the system, it has to become very small. This effect can best be seen from graphing \( \alpha \) versus \( z \) for fixed \( \epsilon \).

From this graph we can see that small perturbations in \( \alpha \) cause significant change in the equilibrium value for very small \( \alpha \) (say, \( \alpha \) less than .008). For \( \alpha \) larger, small changes in alpha have very little effect on the equilibrium value. This result is unexpected. One would expect, from looking at the model, that \( \alpha \) would be an important parameter value in the behavior of the system but it is not.

The effect of varying \( \delta \) is not discussed here, because it is most easily seen from the graph of \( z \) as a function of both \( \delta \) and \( \epsilon \).
8.3 Peer influence

Figure 6 shows the incidence of depression for varying peer pressure rates. In the graph we can see that the incidence of depression is affected dramatically by the interaction between $\varepsilon$ and $\delta$. When $\varepsilon$ is small compared to $\delta$, the incidence of depression is nearly zero. There is a linear relationship between $\varepsilon$ and $\delta$ that determines the region in which the depressed population remains small, and the region in which it grows rapidly and reaches a constant value of about 60%.

The figure 7 shows the linear relationship between $\varepsilon$ and $\delta$ that determines the two regions of interest. The equation for this line is:

$$\delta = 5.3\varepsilon$$

9 Conclusions

Surprisingly, in our model, the incidence of depression is not as dependent on exposure to the media as to other societal factors such as peer pressure and the rate of going into treatment. Depression rates could be kept at a very low incidence if the effect that highly vulnerable and depressed individuals have on less vulnerable people (i.e., $\varepsilon$) is reduced so that it is kept below one fifth the value of $\delta$, or if the influence of less vulnerable people on highly vulnerable individuals is increased so that it is about five times as strong as its counterpart. This could be done by reducing $\varepsilon$ by either lowering the contact rate or the infection rate of this term. Currently, the contact rate for $\varepsilon$ is high because it is normal in our culture for women to disparage their own appearance to one another.
If this behavior were discouraged, $\varepsilon$ would become smaller. If the current emphasis placed upon physical appearance (and weight in particular) diminished, the contact rate of the $\varepsilon$ term would also be reduced. Another way to lower the value of $\varepsilon$ is by reducing the “infection probability”. This could be accomplished through educational programs that teach people to be more critical of the pressure from their peers to be thin. We could also increase $\delta$ by encouraging women who feel good about their bodies to be vocal about this, instead of labeling them conceited, as we currently do.

Treatment is the other important parameter, and perhaps more practical to manage: if $\gamma$ were increased, the incidence of depression would significantly diminish. There are basically two ways to accomplish this task: one is by increasing the amount of depressed individuals who seek treatment, and the other is by decreasing the time that they spend depressed before seeking treatment. Presently, most people are skeptical about seeking treatment for depression, and this is largely due to a widespread social misunderstanding of depression: it is largely believed that depressed individuals could overcome their problem if they would ”get over it”. This is a grave misunderstanding of the seriousness of depression, and the fact that it is a disease that needs treatment just like any other disease. A public education campaign is recommended as an effective way of teaching people about depression, and breaking the myth that depression is somehow a personal choice.

10 Suggestions for future research

A curious implication of our model is that $q$, the probability of being highly vulnerable at age 15, is not a very sensitive parameter. This is a potential drawback of our model since psychological
theory and research dictate that $q$ should be very important. Future research could further incorporate the significance of this factor. It would also be interesting to analyze a model that includes a path from $T$ to $V_1$, thus accounting for individuals who recover from depression but remain highly vulnerable. Another factor that could be taken into account in the model is the effect on demographics caused by suicide. This was not incorporated in the current model for the purposes of tractability.

Additionally, it would be interesting to analyze the rate of media effect on highly vulnerable people as a product of two terms: the contact rate and a probability of infection. Looking at the media’s negative effects in this way would provide a way of investigating what would happen if only the probability of infection is reduced. This rate could also be viewed as a logarithmic function, given that the existence of a threshold based on the amount of exposure to the media is likely: there may be a significant difference in the effects of being exposed to ten or fifty images, whereas the difference between the effects of exposure to five hundred or one thousand images may not be substantial. A behavior such as this one for $\alpha$ may change the dynamics of the model in a way worth examining.

Finally, it is necessary to further investigate the dynamics between the high and low vulnerability classes. Factors other than peer pressure may be examined, such as the impact of the pressure exerted by individuals outside the age range (e.g., family members) or the effects of traumatic personal incidents that may cause women to become more vulnerable.
11 Acknowledgments

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References


Appendix A: Calculation of $R_0$

When $q = 0$, there also exists a depression free equilibrium, where $x = 0$, $y = 1$, $z = 0$, and $w = 0$. We will study the stability of this equilibrium by the next generation approach. The Jacobian matrix of our system evaluated at the depression free equilibrium $(0, 1, 0, 0)$ is given by either

$$J = \begin{pmatrix} \varepsilon - \delta - (\alpha + \mu) & \varepsilon & 0 \\ \alpha & -(\gamma + \mu) & \beta(1 - p) \\ 0 & \gamma & -(\beta + \mu) \end{pmatrix}$$

or

$$J = \begin{pmatrix} 0 & \varepsilon & 0 \\ \alpha & 0 & \beta(1 - p) \\ 0 & \gamma & 0 \end{pmatrix} - \begin{pmatrix} \delta + \alpha + \mu - \varepsilon & 0 & 0 \\ 0 & \gamma + \mu & 0 \\ 0 & 0 & \beta + \mu \end{pmatrix}$$

where

$$M = \begin{pmatrix} 0 & \varepsilon & 0 \\ \alpha & 0 & \beta(1 - p) \\ 0 & \gamma & 0 \end{pmatrix}$$

and

$$D = \begin{pmatrix} \delta + \alpha + \mu - \varepsilon & 0 & 0 \\ 0 & \gamma + \mu & 0 \\ 0 & 0 & \beta + \mu \end{pmatrix}$$

The number of secondary cases of depression generated by a single case in a depression-free population, $R_0$, is given by the dominant eigenvalue of

$$MD^{-1} = \begin{pmatrix} 0 & \frac{\varepsilon}{\gamma + \mu} & 0 \\ \frac{\alpha}{\delta + \alpha + \mu - \varepsilon} & 0 & \frac{\beta(1 - p)}{\beta + \mu} \\ 0 & \frac{\gamma}{\gamma + \mu} & 0 \end{pmatrix}$$

The eigenvalues of $MD^{-1}$ are

$$\lambda_1 = 0$$

$$\lambda_2 = -\sqrt{\frac{\alpha}{\gamma + \mu}}(\frac{\varepsilon}{\delta + \alpha + \mu - \varepsilon} + \frac{\gamma \beta(1 - p)}{(\gamma + \mu)(\beta + \mu)})$$

$$\lambda_3 = \sqrt{\frac{\alpha}{\gamma + \mu}}(\frac{\varepsilon}{\delta + \alpha + \mu - \varepsilon} + \frac{\gamma \beta(1 - p)}{(\gamma + \mu)(\beta + \mu)})$$

$R_0$ is given by

$$R_0 = \sqrt{\frac{\alpha}{\gamma + \mu}}(\frac{\varepsilon}{\delta + \alpha + \mu - \varepsilon} + \frac{\gamma \beta(1 - p)}{(\gamma + \mu)(\beta + \mu)})$$
Appendix B

\[ b = \varepsilon \left( \frac{(\gamma + \mu)(\beta + \mu) - \gamma \beta(1 - p)}{\alpha \gamma} + \frac{\beta + \mu}{\gamma} \right) - \delta \left( \frac{(\gamma + \mu)(\beta + \mu) - \gamma \beta(1 - p)}{\alpha \gamma} \right) \\
- (\alpha + \mu) \left( \frac{(\gamma + \mu)(\beta + \mu) - \gamma \beta(1 - p)}{\alpha \gamma} \right) \\
= \left( - \frac{(\gamma + \mu)(\beta + \mu) - \gamma \beta(1 - p)}{\alpha \gamma} \right)(\alpha + \mu) + \varepsilon \left( \frac{\beta + \mu}{\gamma} \right) + \frac{(\gamma + \mu)(\beta + \mu) - \gamma \beta(1 - p)}{\alpha \gamma} (\varepsilon - \delta) \\
= - \frac{(\gamma + \mu)(\beta + \mu)(\alpha + \mu)}{\alpha \gamma} + \frac{\gamma \beta(1 - p)(\alpha + \mu)}{\alpha \gamma} \\
+ \varepsilon \left( \frac{\beta + \mu}{\gamma} \right) + \left( \frac{(\gamma + \mu)(\beta + \mu) - \gamma \beta(1 - p)}{\alpha \gamma} \right)(\varepsilon - \delta) \\
= \frac{(\gamma + \mu)(\beta + \mu)(\alpha + \mu)}{\alpha \gamma} \left( -1 + \frac{\alpha \varepsilon}{(\alpha + \mu)(\beta + \mu)} + \frac{\gamma \beta(1 - p)}{(\gamma + \mu)(\beta + \mu)} \right) \\
+ \left( \frac{(\gamma + \mu)(\beta + \mu) - \gamma \beta(1 - p)}{\alpha \gamma} \right)(\varepsilon - \delta) \\
= \frac{(\gamma + \mu)(\beta + \mu)(\alpha + \mu)}{\alpha \gamma}(R_0 - 1) + (\varepsilon - \delta) \left( \frac{\gamma \mu + \beta \mu + \mu^2 + \gamma \beta p}{\alpha \gamma} \right) \\
\]

Appendix C

\[ a = \varepsilon \left( \frac{(\gamma + \mu)(\beta + \mu) - \gamma \beta(1 - p)}{\alpha \gamma} + \frac{\beta + \mu}{\gamma} \right)(\alpha \gamma) - \delta \left( \frac{(\gamma + \mu)(\beta + \mu) - \gamma \beta(1 - p)}{\alpha \gamma} \right)(\alpha \gamma) \\
- (\gamma \mu + \beta \mu + \mu^2 + \gamma \beta p) \left( \frac{\gamma \mu + \beta \mu + \mu^2 + \gamma \beta p}{\alpha \gamma} \right) + \frac{\beta + \mu}{\gamma} + 1(\delta - \varepsilon) - \varepsilon \left( \frac{\beta + \mu}{\gamma} \right) \]

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