U.S.A. the Fast Food Nation: Obesity as an Epidemic

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Abstract

The prevalence of overweight and obesity has increased dramatically in the Unites States. Obesity has become a disease of epidemic proportions. In fact, 1 out of 3 people in the United States are obese. Fast-food accessibility is partly to blame for observed patterns of obesity and overweight. The aim of this project is to study the potential role of peer-pressure in fast-food consumption as well as its effect on an individual's weight. We explore these effects on the dynamics of obesity at the population level using an epidemiological model. In this framework, we can explore the impact of intervention strategies. Statistical data analysis provides insights on the relation between demographic factors and weight.

1   Background

Major world organizations such as the American Obesity Association, National Institutes of Health, World Health Organization, American Heart Association, all agree on one thing: obesity is growing at an alarming rate and is now a serious disease of epidemic proportions. Since 1980, obesity rates in U.S have increased by more than 60% in adults, while rates have doubled in children, and tripled in adolescents [**?**]. According to the Center for Disease Control and Prevention, obesity is defined as "the excessively high amount of body fat or adipose tissue in relation to lean body mass" [**?**]. Some of the "identifiable signs and symptoms"of obesity include: excess accumulation of fat, increased levels of glucose, as well as increased blood pressure, and cholesterol levels [**?**]. Obesity is assessed and measured using the *Body Mass Index* (BMI), which is a number calculated using the individual's height and weight. Individuals are considered *underweight* if their corresponding BMI falls below 18.5, *normal weight* if their BMI falls between 18.5-24.9, *overweight* if their BMI falls between 25.0-29.9, and *obese* if their BMI falls above 30.0 [**?**]. Hence, classification of individual's weight depends on their BMI.

In general, there exists several factors that play a role in body weight, and therefore, in becoming obese. These factors include an individual's environment, behavior, metabolism, culture, genes, and socioeconomic status. The rapidity with which obesity rates have increased in the U.S, and even worldwide can be attributed to the previous factors. In particular, the increased U.S obesity rates can predominantly be explained by changes in the individual's behavior and environment. This is because in the last 20 years, people have modified their calorie intake and energy expenditure as well as reduced their physical activity ([**?**],[**?**],[**?**]). In 1977 the proportions of meals consumed away from home was 16%, by 1987 that proportion rosed to 24%, and by 1995 to 29%. In 1977, Americans got 18% of their total calories intake away from home, and fast food places accounted for 3% of the total calorie intake. By, 1994 total calorie intake away from home rose to approximately 34%, while fast food calorie intake rose to 12% by the year 1997. By 2002, fast food consumption accounted for more than 40% of a family's budget spent on food [**?**]. The reduced physical activity of individuals and increased consumption of energy intake, can be also be attributed to the family's hectic work and schedules. In addition economic growth, urbanization and globalization of food markets are some of the forces that have contributed to the development of obesity as an epidemic.

When considering the world population, the World Health Organizations, also reports that roughly one billion adults worldwide are overweight and around 300 mil-

lions of these are considered clinically obese [**?**]. In addition, 17.6 million children under five years are consider to be overweight worldwide. Hence, this problem is global and it affects both industrialized (developed) and developing countries. The epidemic of obesity is making such a negative impact that is has also been associated with other fatal and non-fatal diseases. For example, nonfatal diseases include: respiratory difficulties, arthritis, infertility and psychological disorders(depression, eating disorders, and low self-esteem). Fatal diseases include: diabetes, heart attacks, blindness, renal failure and certain type of cancers. ([**?**],[**?**]). By 2002, it was estimated that obesity accounted for 300,000 deaths in U.S annually. Furthermore, by this same year, it was estimated that obesity and its complications were already costing the nation about $117 billion annually, of the $1.3 trillion spent on health care each year ([**?**], [**?**]).

2 Introduction

According to the Center for Disease Control (CDC), approximately 64% of U.S adults and 15% of children and adolescents are overweight. In 2002, obesity was the second cause of preventable deaths after smoking [**?**]. Major organizations such as the World Health Organization and National Institute of Health as well as other sources ([**?**],[**?**],[**?**]) indicate that an increase of energy intake, and nutrient poor foods with high levels of saturated facts, and sugars is partly to blame for the increased number of overweight and obese individuals [**?**]. Today, Americans are considered to be the fattest people in the world after the Sea Islanders ([**?**], [**?**], [**?**]). A study on the effects of fast food consumption among children also found that fast food could be one of the factors for the increased prevalence of obesity in children. It was found that children who ate fast food consumed more total and saturated fat, more carbohydrates, sugar and less dietary fiber, milk, fruit and vegetables. Of the 6,212 children and adolescents, 30% ate fast food any given day, and they ate an average of more than 187 calories per day than those children who did not ate fast food. These additional calories per day can account for an extra six pounds per year ([**?**],[**?**]).

The first motivation for this work came from a stronger link observed between the effects of fast food and obesity in a documentary, called "Super Size Me". In this documentary, a typical individual, Morgan Spurlock, films himself eating all 3 meals per day for 30 days at Mc'Donald restaurants. At the end of the 30 days, Spurlock not only gained 20 pounds, but he had high levels of cholesterol and high blood pressure [**?**]. Throughout the years, the food marketing industry has suc-

ceeded in making people consume more and more. Most fast food restaurants can "Size it your way", i.e., you can have a medium, large or king-sized value meal. By ordering items together, and by super sizing their value meal, people save an average of 78 cents, but for this 78 cents people get a 200 to 250 increased calorie intake ([**?**]). Surprisingly, U.S residents spend more money on fast food than they spend on movies, books, magazines videos and records combined. In 1970, Americans were spending $6 billion on fast food, and by the year 2000, Americans were spending $110 billions ([**?**], [**?**]).

Fast food industry has not only transform the American diet, but the landscape, economy, workforce, and popular culture. Fast food is relative "good" in taste, inexpensive and convenient that it has become a "common place that it has acquired an air of inevitability, as though it were somehow unavoidable, a fact of life" [**?**]. Statistics show, that on any given day, 1/4 of the adult population visits a fast food restaurant [**?**]. Millions of people buy fast food everyday, supersizing their value meals thinking they are saving money and time without thinking of the actual cost of super sizing their value meal: gaining weight. Thus, because of the increasing number of overweight and obese individuals (adults, adolescents, and children) and the detrimental non-fatal and fatal consequences of obesity, for this project, we are interested in analyzing the dynamics of fast food consumption and obesity in the U.S population using an epidemiological model. In particular, the aim of this project is to study the potential role of peer pressure in fast food consumption as well as its effect on individual's weight. We developed a mathematical model, with special cases, to analyze the progression rate from normal weight individuals(N) to overweight (O_1) and obese(O_2) individuals. The classification of N, O_1, O_2 individuals is based on their *BMI*. The progression from normal to overweight individuals is measured by incorporating a peer pressure, β, by which individuals start eating at fast food restaurants. People start eating at fast food restaurants not only because other people invite them to come along but because of socio-economic status, accessability and convenience to fast food restaurants.

This paper is divided as follows: background and introduction are given on sections 1 and 2. Section 3 includes the statistical analysis on the demographic factors with the individuals weight. Section 4 introduces the obesity model, while in section 5 and 6 two special cases of this model are presented. Section 7 include sensitivity analysis while section 8 have the parameter estimation. Section 9 support the analysis of the obesity model through numerical simulations. Finally section 10 concludes our work.

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Figure 1: Correlation Matrix

3 Statistical Analysis of Demographic Variables and Sample Weight

3.0.1 Data Description

The data to be studied was collected from the National Health and Nutrition Examination Survey (NHANES) 2001-2002 Sample Person Demographics File, which provided sample weights and demographic variables for 11,039 individuals of all ages. The demographic variables are: gender, age, race/ethnicity, education and income. This data is available at *www.cdc.gov/nchs/nhanes.htm*.

3.1 Analysis of Correlation

The goal of this analysis is to find out if any particular demographic variables have a relation with the sample weight. The correlation matrix was used to study this relation. From the correlation matrix (see Figure (**??**)) we obtained that the demographic variables age, race, education and income are positively correlated to weight and gender is not correlated to weight.

3.2 Exploratory Analysis of Possible Interactions of Demographic Variables and Sample Weight

3.2.1 Methodology

The goal of this analysis is to find out if any particular demographic variables have an individual effect with weight or if its effect depend on the level of other demographic variable. A versatile statistical tool to study this relation is the analysis if variance (ANOVA). The starting model to be analyzed contains factor effects as well as all possible combinations of interaction factor effects. To analyze this model a SAS program was created (see Appendix) to produce the ANOVA table that decomposes the total variation in the data, as measured by the Total Sum of Square (TSS), into component that measure the variation of each factor, a components that measure the variation given each factor interaction and the error sum of square (ESS). The table

Figure 2: Interaction Plot: Existence of Interaction

also gives the F-statistic values and the p-values of these components for testing effects. The SAS program created two tables, Type I SS and Type III SS. Type III SS is used for unbalanced factor sample size. The fact that some demographic factors contain missing values implies that we have an unbalanced factor sample size, then the ANOVA table Type III SS will be used. In order to test the factor and interaction effects, we can use the p-values from the obtained table. Since in this type of study we do not need to be too precise, a level of significance of 5% is used. Any p-value of each factor and interaction effect that falls below this level of significance is considered statistically significant. After concluding, with the significant effects, a ANOVA model is suggested. We emphasize the importance of examining the appropriateness of the ANOVA model under consideration, so any inference made with this model can be valid. This appropriateness of the model can be determined from the residual analysis. This residual analysis is carried out by the normal score plot of the residual to determine normality of the residuals, the residual versus fitted value to determine constancy of error variance and the presence of outliers; and the distance of variance plot (Cook's distance) to determine influential outliers observations. The reason why the normality assumption is that the estimator and testing procedure are based on t-distribution which is sensitive to large departures from normality.

3.2.2 Results

Analyzing the SAS output (see Table (**??**)), we obtained that there is: age (group) main effect with a p-value < 0.0001, race main effect with a p-value < 0.0001, education main effect with a p-value < 0.0001 and income main effect with a p-value of 0.0059. There are two-factor interaction effects between age-race with a p-value of < 0.0001, between age-education with a p-value < 0.0001, between race-education with a p-value of < 0.001 and between race-income with a p-value of 0.0002. There are three-factor interaction effects between age-race-education with a p-value of 0.0022, between age-race-income with a p-value < 0.0001 and between race-education-income with a p-value 0.0008. Also, weak four-factor interaction effect between age-race-education-income is found because the p-value of 0.0500. Some examples of the existence and no existence of interactions are show in Figure (**??**) and Figure (**??**).

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Figure 3: Interaction Plot: No Existence of Interaction

Table 1: ANOVA Table

The five-factor ANOVA model is:
Y = $\mu_{..}$ + α Age + β Race + γ Education + ρ Income + $(\alpha\beta)$ Age*Race + $(\alpha\gamma)$ Age*Education + $(\beta\gamma)$ Race*Education + $(\beta\rho)$ Race*Income + $(\alpha\rho\gamma)$ Age*Race*Education + $(\alpha\beta\rho)$ Age*Race*Income + $(\beta\gamma\rho)$ Race*Education*Income.

Here $\mu_{..}$ means the the overall mean weight. The age main effect implies that for each age level, the mean weight is statistically significant different form the other. The same interpretation applies to the main effects for race, education and income. The two-factor interactions implies that for each two level combination the mean weight is statistically significant different from the other. Finally, the same interpretation can be given to the three-factor interactions for each three level combination.

For the examination of the appropriateness model our results are: normality for the residuals, constancy of error variance and no influential outliers, see Figure (??).

Figure 4: Plot of Analysis of Residuals

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Figure 5: Obesity Model

4 Obesity Model with Nonlinear Quitting

This model focuses on the total population of individuals in the United States, who are divided in several classes or sub-populations. The normal weight individuals $N(t)$, are composed of the susceptible individuals who initially do not eat fast food. These individuals may start eating in fast food restaurants due to peer pressure. This peer pressure not only refers to frequent fast food eaters that invite individuals to go eat at fast food restaurants but also to factors such as time, media and individual's socio-economic status that influenced them to go to fast food restaurants. For this project, we are assuming that eating at fast food restaurants increases the individual's weight. Once they start eating at fast food restaurants they become fast food eaters and start a progression to overweight $O_1(t)$ due to large fast food consumption. If they continue to eat at fast food restaurants they can become obese individuals, $O_2(t)$. Both classes can stop eating fast food, and then move to quitting class, $Q_i(t)$, for $i = 1, 2$ for O_1 and O_2 respectively. The normal individuals, $N(t)$, are individuals who have BMI between 18.5 and 24.5. The overweight class, $O_1(t)$, are individuals who have BMI between 24.5 and 29.9 and the obese class, are individuals who have BMI over 29.9 [?].

Parameters:

β = Peer-pressure rate to start eating fast food (media, economic factor, etc).

μ = Mortality rate.

γ = Rate at which an overweight individual becomes an obese individual by continuing eating at fast food restaurants.

α_i = Rate at which an individual stops eating fast food by family or health care recommendation (quitting rate) for $i = 1, 2$.

α_0 = Maximum quit rate due to obese individuals.

L = Obesity level at which the quit rate due to the obese individuals reaches $\frac{1}{2}\alpha_0$, half of its maximum.

ϕ_i = Relapse rate, for $i = 1, 2$.

The quitting class is consider with a non-linear term, $\alpha_i + \frac{\alpha_0 O_2}{(L+O_2)}$ for $i = 1,2$ that depends on the obese population, O_2. This is a collective influence, like peer pressure. This section is to investigate the effects of this pressure to quit, on the system dynamics.

The non-linear differential equations system is:

$$\frac{dN}{dt} = \mu P - \beta N \frac{(O_1 + O_2)}{P} - \mu N, \tag{1}$$

$$\frac{dO_1}{dt} = \beta N \frac{(O_1 + O_2)}{P} + \phi_1 Q_1 - (\gamma + \mu)O_1 - \left(\alpha_1 + \frac{\alpha_0 O_2}{L + O_2}\right)O_1, \tag{2}$$

$$\frac{dO_2}{dt} = \gamma O_1 + \phi_2 Q_2 - \mu O_2 - \left(\alpha_2 + \frac{\alpha_0 O_2}{L + O_2}\right)O_2, \tag{3}$$

$$\frac{dQ_1}{dt} = \left(\alpha_1 + \frac{\alpha_0 O_2}{L + O_2}\right)O_1 - (\phi_1 + \mu)Q_1, \tag{4}$$

$$\frac{dQ_2}{dt} = \left(\alpha_2 + \frac{\alpha_0 O_2}{L + O_2}\right)O_2 - (\phi_2 + \mu)Q_2, \tag{5}$$

$$P = N + O_1 + O_2 + Q_1 + Q_2. \tag{6}$$

By adding all the equations the total population is constant, i.e $\frac{dP}{dt} = 0$. Then the model can be re-scale by introducing: $x = \frac{N}{P}$, $y_1 = \frac{O_1}{P}$, $y_2 = \frac{O_2}{P}$, $z_1 = \frac{Q_1}{P}$ and $z_1 = \frac{Q_1}{P}$, with a new constant K that comes from re-scaling the nonlinear term, $K = \frac{L}{P}$. Since the total population is constant, the system can be reduced to a four dimensional system.

$$\frac{dx}{dt} = \mu - \beta x(y_1 + y_2) - \mu x, \tag{7}$$

$$\frac{dy_1}{dt} = \beta x(y_1 + y_2) + \phi_1 z_1 - (\gamma + \mu)y_1 - \left(\alpha_1 + \frac{\alpha_0 y_2}{K + y_2}\right)y_1, \tag{8}$$

$$\frac{dy_2}{dt} = \gamma y_1 + \phi_2 z_2 - \mu y_2 - \left(\alpha_2 + \frac{\alpha_0 y_2}{K + y_2}\right)y_2, \tag{9}$$

$$\frac{dz_1}{dt} = \left(\alpha_1 + \frac{\alpha_0 y_2}{K + y_2}\right)y_1 - (\phi_1 + \mu)z_1, \tag{10}$$

$$\frac{dz_2}{dt} = \left(\alpha_2 + \frac{\alpha_0 y_2}{K + y_2}\right)y_2 - (\phi_2 + \mu)z_2, \tag{11}$$

$$1 = x + y_1 + y_2 + z_1 + z_2. \tag{12}$$

4.1 Obesity Free Equilibrium

One equilibrium for this model is the obesity free equilibrium $(x, y_1, y_2, z_1, z_2) = (1, 0, 0, 0, 0)$. It can be seen that if the normal weight individuals do not go to fast food restaurants, then no obesity state will ever develop in the population, as stated formally in terms of global stability (all solutions approach a given point.)

The global stability is established using the Liapunov method [?]. Consider a system $\dot{x} = f(x)$ which has a fixed point x^* when $f(x) = 0$. Suppose that we can find a Liapunov function, i.e. a continuously differentiable, real valued function $V(x)$ with the following properties:

1. $V(x) > 0$ for all $x \neq x^*$, and $V(x^*) = 0$.

2. $\dot{V} < 0$ for all $x \neq x^*$.

Then x^* is globally asymptotically stable: for all initial conditions, $x(t) \to x^*$ as $t \to \infty$ [?].

Theorem 1. Global Stability of Obesity-Free Equilibria
The obesity free equilibria (OFE), (1,0,0,0,0) is globally stable if $\beta < \mu$.

Proof. Consider the Liapunov function $V(y_1, y_2, z) = y_1 + y_2 + z_1 + z_2$.
Since y_1, y_2, z_1 and z_2 are nonnegative then $V(y_1, y_2, z_1, z_2) \geq 0$.

$$\begin{aligned}
\frac{dV}{dt} &= \frac{dy_1}{dt} + \frac{dy_2}{dt} + \frac{dz_1}{dt} + \frac{dz_2}{dt} \\
&= \beta x(y_1 + y_2) - \mu(y_1 + y_2 + z_1 + z_2) \\
&= (\beta x - \mu)(y_1 + y_2) - \mu(z_1 + z_2) \\
&\leq (\beta x - \mu)(y_1 + y_2) \\
&\leq (\beta - \mu)(y_1 + y_2) \qquad since \qquad x \leq 1 \\
&< 0 \qquad since \qquad \beta < \mu
\end{aligned}$$

Therefore since $\frac{dV}{dt} < 0$ the OFE is globally asymptotically stable. $\qquad\square$

Since μ is very small this is a very restrictive assumption, thus, one goal of an education campaign might be to reduced β, the peer pressure to start eating at fast food restaurants. Using the particular map function above if $\beta < \mu$ the population will become entirely normal weight. Therefore, in order to have an obesity endemic equilibria we consider $\beta > \mu$.

Figure 6: Obesity Model without Relapse

5 Obesity Model without Relapse ($\phi_i = 0$)

In this special case of the obesity model the effect of stopping eating at fast food permanently is explore, where the stopping rate is a nonlinear term. Since the individuals do not go back to eat fast food after they quit is enough to have only one quitting class for both, overweight and obese individuals.

5.1 The Basic Reproductive Number, R_0

R_0 is typically a threshold quantity in epidemiological models, defined as the average number of secondary cases produced by a typical infected individual. Since the transmission here is collective rather than individual, we interpreted R_0 as an indicator of how conducive the environment is to developing obesity, a ratio of how quickly individuals become overweight or obese relative to how fast obese or overweight individuals quit eating fast food or leave the population [?] or as a measure of the number of secondary conversions to fast food use from interactions with frequent fast food users in a population of few fast food consumers. Since we have a nonlinear term for quitting, α_0 and K are not going to be present in R_0 because we linearize around the OFE, no terms nonlinear in the infected class variable, obese class.

The basic reproduction number, R_0, is calculated by using the second generator approach as described on Diekmann and Heesterbeek, and van den Driessche and Watmough ([?], [?]). The next generation matrix from F and V to be FV^{-1} where:

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & \beta & \beta & 0 \\ 0 & \gamma & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} and \qquad V = \begin{pmatrix} \mu & \beta & \beta & 0 \\ 0 & \mu + \gamma + \alpha_1 & 0 & 0 \\ 0 & 0 & \alpha_2 + \mu & 0 \\ 0 & -\alpha_1 & -\alpha_2 & 0 \end{pmatrix} \qquad (13)$$

F represents the paths of infection (rate of appearance of new infections in each compartment) and V represents the remaining dynamics (rate of transfer of individuals into a compartment by all other compartments and the rate of transfer of individuals out of a compartment). The four columns and rows correspond to the

four compartments N, O_1, O_2 and Q.

The basic reproduction number is the leading eigenvalue (spectral radius) of FV^{-1}, which is:

$$R_0 = \frac{1}{2}\left(\frac{\beta}{\mu + \gamma + \alpha_1} + \sqrt{\left(\frac{\beta}{\mu + \gamma + \alpha_1}\right)^2 + \frac{4\gamma\beta}{(\mu + \gamma + \alpha_1)(\alpha_2 + \mu)}}\right). \qquad (14)$$

The term $\frac{1}{\mu+\gamma+\alpha_1}$ is the average time that an overweight individual spent in the compartment O_1, i.e. being overweight, this value multiplied by β, which is the rate at which normal weight individuals N enter O_1, gives the first term of R_0, $\frac{\beta}{\mu+\gamma+\alpha_1}$ thus,

$$R_0 > \frac{\beta}{\mu + \gamma + \alpha_1}.$$

Therefore, this term is the reproductive number for the first infective class, $R_0(N \to O_1)$ or the reproductive number when there are no obese individuals (O_2). Obesity incidence can be attributed to initial development, in which case since $(\sqrt{M} + \sqrt{N} \leq \sqrt{M + N})$:

$$R_0 < \frac{\beta}{\mu + \gamma + \alpha_1} + \sqrt{\frac{\beta}{\mu + \gamma + \alpha_1}\frac{\gamma}{\alpha_2 + \mu}}.$$

The term $\frac{1}{\alpha_2+\mu}$ is the average time that an individual spent in the class O_2, obesity. Therefore, the second term is a reproductive number for the second infective class, $R_0(N \to O_2)$, where γ is the progression rate to obesity. It involves a radical because replacement of overweight individuals to obesity occurs via two stage process, progression: $N \to O_1 \to O_2$. Essentially, this type of R_0 has been seen in models with multi stage infections ([?], [?], [?]).

Typically, if $R_0 < 1$ the disease free equilibria of the population is stable, as well as in this model the obesity free equilibrium is globally asymptotically stable and whenever $R_0 > 1$ the disease free equilibria becomes unstable and the endemic equilibria is established in the population and becomes stable. Therefore, in our case when $R_0 > 1$ the obese individuals persist in our population.

5.2 Endemic Equilibria

The previous section shows that if $\beta < \mu$ and $R_0 < 1$ then the obesity free equilibrium is globally asymptotically stable, meaning that neither overweight nor obesity is present in the population.To solve for the endemic equilibria where $R_0 > 1$, the equations in system (??) (applied to this special case) are set equal to and solve for x, y_1, y_2 and z. Since the total population is constant, i.e. $\frac{dP}{dt} = 0$ the fact that $x + y_1 + y_2 + z_1 + z_2$ can be use to simplify the system. Now define:

$$\Omega = \gamma + \alpha_1 + \mu \tag{15}$$
$$\Delta = \alpha_2 + \mu \tag{16}$$
$$G(y_2) = \frac{\alpha_0 y_2}{(K + y_2)} \tag{17}$$

In this case because of the nonlinearity for quitting the number of endemic equilibria is difficult to establish analytically, however we can prove existence of at least an equilibrium solution.

Theorem 2. Existence of Endemic Equilibrium
If $R_0 > 1$, then there exists at least one endemic equilibrium solution.

Proof. The equilibrium conditions are obtained from equations (??)-(??), and reduce the system to just one equation by expressing the equilibrium values for x, y_1 and z in terms of y_2:

$$y_1 = \frac{y_2}{\gamma}(\Delta + G(y_2)) \text{ from (??)}, \tag{18}$$

$$z = \frac{y_2}{\mu}\left(\frac{(\alpha_1 + G(y_2))(\Delta + G(y_2))}{\gamma} + (\alpha_2 + G(y_2))\right) \text{ from (??) and (??)}, \tag{19}$$

$$x = \frac{\mu}{\frac{\beta y_2}{\gamma}(\Delta + \gamma + G(y_2)) + \mu} \text{ from (??) and (??)}, \tag{20}$$

and then substituting into either (??) or (??) gives an expression for y_2:

$$0 = \beta\left[\frac{\mu(\Delta + \gamma + G(y_2))}{\frac{\beta y_2}{\gamma}(\Delta + \gamma + G(y_2)) + \mu}\right] - (\Omega + G(y_2))(\Delta + G(y_2)). \tag{21}$$

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After multiplying the various terms a quartic polynomial in y_2 is obtained, define:

$$
\begin{align}
a &= \mu\gamma(\beta(\Delta+\gamma)-\Delta\Omega) \tag{22}\\
b &= \mu\gamma(\beta-\Delta-\Omega) \tag{23}\\
c &= \beta\Delta\Omega(\Delta+\gamma) \tag{24}\\
d &= \beta(\Delta\Omega+(\Delta+\Omega)(\Delta+\gamma)) \tag{25}\\
e &= \beta(2\Delta+\Omega+\gamma) \tag{26}
\end{align}
$$

Therefore, the 4th degree polynomial will be:

$$
F(y_2) = Ay_2^4 + By_2^3 + Cy_2^2 + Dy_2 + E \tag{27}
$$

where the coefficients are functions of model parameters:

$$
\begin{align}
A &= c + \alpha_0 d + \alpha_0^2 e + \alpha_0^3 \beta \\
B &= (Ke+\mu\gamma)\alpha_0^2 + (2dK-b)\alpha_0 + 3Kc - a \\
C &= \mu\gamma K\alpha_0^2 + (K^2 d - 2Kb)\alpha_0 + 3K^2 c - 3Ka \\
D &= -K^2 b\alpha_0 + K^3 c - 3K^2 a \\
E &= K^3 a
\end{align}
$$

For an endemic equilibrium need the endemic solution x^*, y_1^*, y_2^* and z^* to be non-negative and add up to 1. Looking at the equations (**??**)-(**??**), $x>0, y_1>0, z>0$ and therefore $x+y_1+z>0$. Now, we know that $y_2 = 1-(x+y_1+z)>1$ this implies that $y_2>0$. Now consider $F(0)$ and $F(\infty)$. Calculations show that

$$
\begin{align}
F(0) &= aK^3 = \mu\gamma[\beta(\Delta+\gamma)-\Delta\gamma] \tag{28}\\
F(\infty) &= \alpha_0^3\beta + \alpha_0^2 e + \alpha_0 d + c. \tag{29}
\end{align}
$$

From (**??**) notice that $c>0, d>0$ and $e>0$, therefore $F(\infty)>0$. If $F(0)<0$ which implies that $\left(\frac{1}{\gamma}+\frac{1}{\Delta}\right) < \frac{1}{\beta}$, then by continuity of F at least one solution y_2^* exists when $R_0 > 1$ (since $F(0) < 0 < F(\infty)$). Thus, there exists at least one endemic equilibrium solution.

$\square$

Figure 7: Linear Quitting Obesity Model

6 Obesity Model with Relapse and Linear Quitting $(\alpha_0 = 0)$

Consider a special case of the obesity model where $\alpha_0 = 0$, which implies a linear quitting rate for the overweight individuals α_1 and for the obese individuals α_2. In this case the impact of relapse in our model is explore. Two quitting classes are consider, $Q_1(t)$ and $Q_2(t)$ which represent the overweight and obese individuals that quit, respectively, therefore in this case the quitting is temporarily. We consider this relapse from the quitting classes to be a linear term ϕ_i for $i = 1, 2$ this are rates at which individuals in each quitting class, according to their BMI, go back to start eating at fast food restaurants.

6.1 The Basic Reproductive number, R_0

The calculation of R_0 for the relapse model was performed with the same method as for the nonlinear quitting model. Again, F represents the paths of infection (rate of appearance of new infections in each compartment) and V represents the remaining dynamics (rate of transfer of individuals into a compartment by all other compartments and the rate of transfer of individuals out of a compartment). The four columns and rows correspond to the five compartments N, O_1, O_2, Q_1 and Q_2, respectively.

The basic reproduction number is the leading eigenvalue (spectral radius) of FV^{-1}, which in this case tends out to be:

$$R_0 = \frac{1}{2}\left[\left(\frac{\beta}{\Omega}\left(1 + \frac{\gamma}{\Delta}\right) + p_1 + p_2\right) + \sqrt{\left(\frac{\beta}{\Omega}\left(1 + \frac{\gamma}{\Delta}\right) + p_1 + p_2\right)^2 + \frac{4p_2\beta\gamma}{\Omega\Delta}}\right] \quad (30)$$

where Δ and Ω as in (??) and:

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$$p_1 = \frac{\phi_1 \Theta_1}{\Omega}, \tag{31}$$

$$p_2 = \frac{\phi_2 \Theta_2}{\Delta}, \tag{32}$$

$$\Theta_i = \frac{\alpha_i}{\phi_i + \mu}. \tag{33}$$

$$\tag{34}$$

The term $\frac{1}{\mu+\gamma+\alpha_1}$ is the average time that an overweight individual spent in this compartment O_1, i.e. being overweight, this value multiplied by β yields the rate at which normal weight individuals N enter O_1 and it multiplied by α_1, gives us the rate at which overweight individuals O_1 enter Q_1. Similarly, $\frac{\alpha_2}{\mu+\alpha_2}$ gives the rate at which obese individuals O_2 enter the class Q_2, and $\frac{1}{\phi_i+\mu}$ is the average time an individual spent in the quitting class i. Thus

$$R_0 > \frac{(\beta + \phi_1 \Theta_1)}{\Omega} + \frac{\phi_2 \Theta_2}{\Delta}$$

The second term also involves a radical term similar to the model without relapse because replacement of overweight individuals to obesity is a two stage process, progression: $N \to O_1 \to O_2$.

6.2 Endemic Equilibria

The previous section shows that if $\beta < \mu$ and $R_0 < 1$ then the obesity free equilibria is globally asymptotically stable, meaning that neither overweight nor obesity is present in our population. To solve for the endemic equilibria, where $R_0 > 1$, we set each one of the equations (??)-(??) applied to this special case) equal to zero an solve for x, y_1, y_2, z_1 and z_2.

In order to calculate the endemic equilibrium we use (??) and (??) and introduce two new variables:

$$\Psi = \frac{1}{\gamma} + \frac{1}{\Delta - \phi_2 \Theta_2}, \tag{35}$$

$$\Sigma = \left(\frac{\gamma}{\Omega - \phi_1 \Theta_1} - \frac{1}{\beta \Psi} \right). \tag{36}$$

Therefore, the endemic equilibria for this model with relapsed and linear quitting is:

$$x^* = \frac{1}{\beta\Psi\Sigma(\Delta - \phi_2\Theta_2) + 1}, \tag{37}$$

$$y_1^* = \frac{\mu\Sigma}{\gamma}(\Delta - \phi_2\Theta_2), \tag{38}$$

$$y_2^* = \mu\Sigma, \tag{39}$$

$$z_1^* = \mu\Theta_1\Sigma(\Delta - \phi_2\Theta_2), \tag{40}$$

$$z_2^* = \mu\Theta_2\Sigma. \tag{41}$$

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7 Sensitivity Analysis of R_0

If a small perturbation is made to a parameter $(\delta \rightarrow \epsilon\delta)$ it will also affect R_0 $(R_0 \rightarrow \epsilon R_0)$. The normalized sensitivity index S_δ is define to be the ratio of the corresponding normalized changes:

$$S_\delta = \frac{\epsilon R_0}{R_0} \Big/ \frac{\epsilon\delta}{\delta} = \frac{\delta}{R_0}\frac{\partial R_0}{\partial\delta} \tag{42}$$

The normalized sensitivity indexes for the five most important parameters for the obesity model without relapse $(\phi_i = 0)$ in R_0 are:

$$
\begin{aligned}
S_\gamma &= \frac{\gamma}{R_0}\frac{\partial\gamma}{\partial R_0} = \frac{-\gamma}{R_0}\frac{\beta}{4\Omega^2}\left[2 + \frac{\beta\Delta - 2\Omega(\Omega - \gamma)}{\Omega\Delta\left(R_0 - \frac{\beta}{2\Omega}\right)}\right]\\[2mm]
S_\beta &= \frac{\beta}{R_0}\frac{\partial\beta}{\partial R_0} = \frac{\beta}{R_0}\left[\left(\frac{1}{4\Omega}\right)\left(2 + \frac{\beta\Delta + 2\gamma\Omega}{\Omega\Delta(R_0 - \frac{\beta}{2\Omega})}\right)\right]\\[2mm]
S_{\alpha_1} &= \frac{\alpha_1}{R_0}\frac{\partial\alpha_1}{\partial R_0} = \frac{-\alpha_1}{R_0}\left[\left(\frac{\beta}{4\Omega^2}\right)\left(2 - \frac{(\beta\Delta + 2\gamma\Omega)}{\Omega\Delta(R_0 - \frac{\beta}{2\Omega})}\right)\right]\\[2mm]
S_{\alpha_2} &= \frac{\alpha_2}{R_0}\frac{\partial\alpha_2}{\partial R_0} = \frac{-\alpha_2}{R_0}\left(\frac{\gamma\beta}{2\Omega\Delta^2(R_0 - \frac{\beta}{2\Omega})}\right)\\[2mm]
S_\mu &= \frac{\mu}{R_0}\frac{\partial\mu}{\partial R_0} = \frac{-\mu}{R_0}\left[\left(\frac{\beta}{4\Omega^2}\right)\left(2 + \frac{\beta\Delta^2 + 2\gamma\Omega(\Delta + \Omega)}{\Omega\Delta^2(R_0 - \frac{\beta}{2\Omega})}\right)\right]
\end{aligned}
$$

Substitution of the variables given in (??) and the estimated parameter values (next section), it was found that β is the most sensitive parameter to R_0 followed by α_1, γ, α_2 and μ. The reason for this is that as the peer pressure to start eating fast food increases, the bigger R_0 becomes (proportionality). Then after enough overweight individuals, the best strategy would be to increase α_1 making them stop eating fast food fast enough that they do not go to the obese class, which will reduce R_0. Then we should focus on reduce γ so the flow from overweight individuals that become obese decrease, if there enough obese individuals then the focus to reduce R_0 should be to reduce α_2

For the relapse model we found numerically, that the sensitivity indexes did not change. Therefore, β and α_1 are the most sensitive parameters to R_0.

8 Parameter Estimation

In order to be able to run simulations, firs parameters must be estimated. We estimate the values for model parameters in order to determine model predictions. As some of the parameters can be estimated only very roughly, our principal objective shall be to see how closely model behavior corresponds to pragmatic observations.

This paper focuses on the US population, which consists of approximately 300 million people, of which 33% are normal weight, 34% are overweight and 30% are obese ([?], [?]). Since our model focuses on the progression of gaining weight for a normal weight individual, the initial condition for N is 99 million. In order to calculate the mortality rate, μ, we take into consideration the average life time of an individual which is approximately 70 years (840 months); therefore $\mu = 1/840 = 0.0012$ months $^{-1}$. The time it takes an overweight individual to become obese by continuing to consume fast food is approximately 7.5 months. This approximation is obtained by studying the "Super-Size Me" documentary by Morgan Spurlock, in which he increased his BMI value from normal weight to overweight in a period of one month, 90 meals [?]. His case is an extreme case since all his meals were fast food. On average, an individual consumes 12 fast food meals per month; hence it takes approximately 7.5 month for an average individual to increase his/her weight status [?]. Now, the rate at which an individual progresses from the overweight compartment to obesity is $\gamma = \frac{1}{7.5} = 0.13$ months^{-1}.

The parameters β, α_1, and α_2 depend on peer-pressure; there is no accurate form of quantifying peer-pressur. In the absence of accurate data pertaining relapsed rate, we approximate ϕ_1 and ϕ_2 taking into consideration the fact that 33% of the US population is on a diet, of which 16.5% break the diet ([?], [?]).

Figure 8: Effect of Peer Pressure to Start Eating Fast Food

9   Numerical Simulations

Numerical solutions with respect to our model were considered in order to study the behavior of the obesity epidemic due to fast food consumption as time progresses. A MatLab program was used to test the relevance of the peer-pressure parameters, β, α_1, and α_2 because from the above sensitivity analysis it was concluded that our model are the most sensitive to these. In the special case were we are considering the possibility of relapse, the focus is given to the effect of introducing relapse rates ϕ_1 and ϕ_2. In doing so, the role of peer-pressure on becoming a fast food eater and stopping eating fast food was determined.

9.1   Effect of Peer Pressure to Start Eating Fast-Food (β)

Figure (??) shows the effect of peer-pressure to start eating fast food, β. In Figure (a), $\beta = 0.6$, a peer-pressure that roughly resembles the current situation. This choice of β causes $R_0 = 3.5375 > 1$, hence resulting in an obesity epidemic. We are able to predict in a period of approximately 10 months that 35% of the normal weight individuals will become overweight. Furthermore, in roughly 18 months (1.5 years), 25% of the normal weight individuals will become obese. In Figure (b), we set $\beta = 0.09$, a very low peer-pressure which maintains $R_0 < 1$. Notice that the obesity epidemic is under control, keeping both the percentage of overweight and obese individuals to a minimum. This shows how peer-pressure has a strong influence on becoming a fast food eater.

9.2   Effect of Peer Pressure for Overweight Individuals to Stop Eating Fast-Food (α_1)

Figure (??) shows the effect of peer-pressure for the overweight individuals to stop eating fast food, α_1. In Figure (a), α_1 is given a low value, $\alpha_1 = 0.2$, which results in $R_0 = 2.679 > 1$. From the dynamics of the model, we are able to predict that in 15 months (1.3 years), 22% of the normal weight individuals will become overweight. Consequently, in only 18.5 months (1.6 years), 18% of the normal weight individuals will become obese. Now, in Figure (b), the value of α_1 changes to $\alpha_1 = 0.95$, decreasing R_0 to 0.804, the obesity epidemic is under control. With this choice

Figure 9: Peer Pressure to Stop Eating Fast Food

Figure 10: Replapse Effect

of α_1, the normal weight individuals will progress to the overweight compartment, however since α_1 is high, these individuals will also leave the compartment at a fast rate, hence not advancing to obesity. In conclusion α_1 also has a strong effect in controlling the obesity epidemic.

9.3 Effect of Peer Pressure to Start Eating Fast-Food (β)

In numerical solutions of this model, we focus our attention on the effect of relapse rates, ϕ_1 and ϕ_2, (see Figure (??)). In the Figure(a), the values for ϕ are set to equal the values for α, obtaining $R_0 = 3.06$, hence an epidemic exists. It is reasonable for this to be the outcome because if everyone is quitting at the same rate as they are coming back into the the system by starting to eat fast food again. As a consequence of choosing these parameters, the quitting class will remain constant and there will be an obesity outbreak, since the concentration of individuals will be mainly in the overweight and obese compartment. Therefore, the relapse rate ϕ contributes to the outbreak of the obesity epidemic.

10   Conclusion

From the statistical analysis we concluded with a factor and interaction effects model which explain 61.05% of the total variability in weight. The weight is statistically significant different between the three age's categories, between the five race's categories and between the three education categories, but not statistically significant different between gender.

The models with support of the numerical analysis, showed that peer-pressure, β had a strong influence in becoming a fast-food eater. Furthermore, the rate at which individuals stop eating fast food, α_1 also seemed to be effective in controlling the obesity epidemic. It would appear that in order to reduce the current obesity rates, we should focus on lowering the peer pressure from fast food eaters. However, controlling β is difficult to achieve since β is deduced from the peer-pressure due to frequent fast food eaters, media, social and economic status. Hence, we should gear our attention in incrementing the peer pressure to stop eating fast food, α_1. This is a more realistic approach since it is easier to increment health awareness programs that are accessible to the general public.

11   Future Work

For simplicity purposes, for this project we just considered natural mortality rates. Research indicates that obesity is also associated to other chronic mortality diseases, such as heart attacks, diabetes and certain type of cancers, therefore, for future work we would like to add the mortality rate due to these chronic or fatal diseases, and analyze how this new rate affect our models. Integrating this new rate would make our total population nonconstant. Another possible extension of our model would be to consider a progressive model in which a population of underweight, normal and overweight and obese individuals are considered. Another area to explore would be an age structure model using the possible correlation between age and weight we found from the statistical analysis. Finally, we would like to do a more in depth analysis of our original model.

12   Acknowledgements

References

[1] ABC news, 2004. Obsessed by Fast Food: Will Fast Food be the death of us /? Website:http://abcnews.go.com/sections/GMA/GoodMorningAmerica/ GMA0201Obsessed/with Fast food.html, 8th January.

[2] Advocate Health, 2004. Understanding calories and exercise. www.advocatehealth.com/system/info/library/articles/fitness/foodforthought/fitcalo.html.

[3] American Obesity Association, 2004. Obesity is a Chronic Disease. www.obesity.org/treatment/obesity.shtml.

[4] Bowman, S. Gortmaker, S. Ebbeling, C, Pereira M, and Ludwig, D., 2004. Effects of Fast Food Consumption on Energy Intake and Diet Quality Among Children in a National Household Survey. *Pediatrics.***113**, 112-118.

[5] Calorie Control Council National Consumer Survey, 2004: Trends and statistis, 2004. Website: http://www.caloriecontrol.org/trndstat.html.

[6] Castillo-Chavez, C., Feng, Z., and Huang, W., 2002. On the computation of R_0 and its role on global stability. In C. Castillo-Chavez et al. (Eds.), *Mathematical approaches for emerging and re-emerging infectious diseases*, Part I, IMA Vol, **125**, 224-250.

[7] Census Releases 2003 U.S Population Estimates, 2003. Adults, Older people and children: latest estimates. Website: http://usgovinfo.about.com/cs/censustatistics/a/latestpopcounts.htm.

[8] CDC(Center for Disease Control), 2004. Defining overweight. Website: http://www.cdc.gov/nccdphp/dnpa/obesity/defining.htm.

[9] CDC(Center for Disease Control), 2004. Overweight and obesity: Health consequences. Website: http://www.cdc.gov/nccdphp/dnpa/obesity/consequences.htm

[10] Children's hospital Boston News, 2004. Clear Link between fast food, obesity. Website: http://web1.tch.harvard.edu/chnews/01-2004/obesity.html. January.

[11] Critser, G, 2003. Fat land: How Americans Became the Fattest People in the World. Houghton Mifflin Company, Boston.

[12] Diekmann, O., Dietz, K., and Heesterbeek, J. A.P (1991). The basic reproduction ratio for sexually transmitted diseases, Part 1: Theoretical considerations. *Mathematical Bioscienes*, 107, 325-339.

[13] Diekmann, O., Heesterbeek, J.A.P., 2000. Mathematical Epidemiology of infectious diseases: Model building, Analysis and interpretation. Wiley, NY.

[14] Dietz, K., Heesterbeek, J. A. P., and Metz, J. P., & Tutor, D, W. (1993). The basic reproduction ratio for sexually transmitted diseases, Part 2: Effects of variable HIV-infectivity. *Mathematical Biosciences*, **117**, 35-47.

[15] Driessche, P.V., Watmough, J., 2002. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Journal of Mathematical Bioscince.***20**, 1-21.

[16] Department of Health and Human Services, 2002. CDC's Role in combating Obesity and the Scientific Basis for Diet and Physical Activity. Website: http://www.hhs.gov/asl/testify/t020725a.html, 25th July.

[17] Food and diet News Service, 2004. Treat yourselve better-calories. Website: http://www.foodanddiet.com/NewFiles/calorieburnchart.html.

[18] Gonzales, B. et. al. (2003) Am I too fat /? Bulimia as an epidemic. *Journal of Mathematical Psychology.* **47**, 515-526.

[19] Mangel, M. and Clark, Colin W. (1988). Dynamic Modeling in Behavioral Ecology. *Pricenton University Press, Princeton, N.J.*, pp. 41-81.

[20] Medline Plus, 2004. Reuters Health information. Americans Abandoning Low-carb Diets-Survey. Website: http://nlm.nih.gov/medlineplus/news/fullstory_18977.html, July, 15th.

[21] National Institute of Diabetes and Digestive and Kidney Diseases of the National Institute of Health, 2004. Statistics realted to Overweight and Obesity. Website: http://www.niddk.nih.gov/health/nutrit/pubs/statobes.htm.

[22] Natural Strength news, 2002. Value Meals: The High Price of Fast Foods.Website://www.naturalstrength.com/nutrition//detail.asp/?ArticleID=585, 12th August.

[23] News Service 2think, 2004. Fast Food Nation: The Dark Side of the All-American Meal. Website: http://www.2think.org/fastfood.shtml.

[24] Recent reviews and press, 2004. Supersize Me: A film of epic proportions. Website: http://www.supersizeme.com/home.aspx/?page=archived/06/03/04channel4.

[25] Strogatz, S.H. (1994) Nonlinear Dynamics and Chaos. Perseus Books: Massachussetts.

[26] The cool nurse, 2004. Calories burned per minute for various activities. Website: htpp://www.coolnurse.com/calories.htm.

[27] World Health Organization, 2004. Facts related to chronic diseases. Website: http://www.who.int/dietphysicalactivity/publications/facts/chronic/en.