Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

HHS Public Access

Author manuscript

Obesity (Silver Spring). Author manuscript; available in PMC 2014 August 01.

Published in final edited form as:

Obesity (Silver Spring). 2014 February ; 22(2): 590–597. doi:10.1002/oby.20520.

Dynamic Model Predicting Overweight, Obesity, and Extreme Obesity Prevalence Trends

Diana M. Thomas[1], **Marion Weedermann**[2], **Bernard F. Fuemmeler**[3], **Corby K. Martin**[4], **Nikhil V. Dhurandhar**[4], **Carl Bredlau**[1], **Steven B. Heymsfield**[4], **Eric Ravussin**[4], and **Claude Bouchard**[4]

[1]Center for Quantitative Obesity Research, Montclair State University, Montclair, NJ

[2]Department of Mathematics, Dominican University, River Forest, IL

[3]Duke University Medical Center, NC

[4]Pennington Biomedical Research Center, Louisiana State University System, Baton Rouge, LA

Abstract

Objective—Obesity prevalence in the United States (US) appears to be leveling, but the reasons behind the plateau remain unknown. Mechanistic insights can be provided from a mathematical model. The objective of this study is to model known multiple population parameters associated with changes in body mass index (BMI) classes and to establish conditions under which obesity prevalence will plateau.

Design and Methods—A differential equation system was developed that predicts population-wide obesity prevalence trends. The model considers both social and non-social influences on weight gain, incorporates other known parameters affecting obesity trends, and allows for country specific population growth.

Users may view, print, copy, and download text and data-mine the content in such documents, for the purposes of academic research, subject always to the full Conditions of use:http://www.nature.com/authors/editorial_policies/license.html#terms

Corresponding Author: Diana M. Thomas, Center for Quantitative Obesity Research, Montclair State University, Montclair, NJ 07043, Phone: (973) 655-7262, Fax: (973) 655-7686, thomasdia@mail.montclair.edu.

CONFLICT OF INTEREST

Bouchard: Claude Bouchard is a consultant for Weight Watchers International and a member of the Scientific Advisory Board of Pathway Genomics.

Dhurandhar: The following Patents are granted or have been applied for: Patent number 6,127,113: Viral obesity methods and compositions. Patent number 6,664,050: Viral obesity methods and compositions. Patent number US 8,008,436B2, dated August 30, 2011: Adenovirus 36 E4orf1 gene and protein and their uses. Provisional patent filed: Adenovirus Ad36 E4orf1 protein for prevention and treatment of non-alcoholic fatty liver disease, July 2010. Provisional patent filed: Enhanced glycemic control using Ad36E4orf1 and AKT1 Inhibitor. January 2012.

Thomas: Diana Thomas is a consultant for Jenny Craig.

Author contributions

Study concept and design: Thomas, Bouchard

Drafting of the manuscript: Thomas, Weedermann, Heymsfield, Bouchard

Critical revision of the manuscript for important intellectual content: Thomas, Weedermann, Fuemmeler, Martin, Dhurandhar, Bredlau, Heymsfield, Ravussin, Bouchard

Model development: Thomas, Weedermann, Dhurandhar, Ravussin, Bouchard

Mathematical analysis: Thomas, Weedermann

Mathematical simulations: Bredlau

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Results—The dynamic model predicts that: obesity prevalence is a function of birth rate and the probability of being born in an obesogenic environment; obesity prevalence will plateau independent of current prevention strategies; and the US prevalence of obesity, overweight, and extreme obesity will plateau by about 2030 at 28%, 32%, and 9%, respectively.

Conclusions—The US prevalence of obesity is stabilizing and will plateau, independent of current preventative strategies. This trend has important implications in accurately evaluating the impact of various anti-obesity strategies aimed at reducing obesity prevalence.

Keywords

Mathematical model; differential equation; infectious disease model; obesity prevalence

INTRODUCTION

Why is the prevalence of obesity in the United States (US) appearing to level off(1)? Are prevention and treatment strategies working (2)? Can we expect the plateau to continue or is the apparent stabilization of obesity prevalence a temporary state?

These questions are vigorously debated due to their potential impact on healthcare costs and the desire to credit or discredit various policies to reduce obesity at the national level in many countries of the world. Existing predictions of obesity trends (3, 4) cannot fully answer these questions because their underlying models assume *a priori* that obesity prevalence will either increase without bound or will continue to increase and eventually plateau. Dynamic models based on differential equations circumvent these limitations and can generate a predicted curve based on biological, behavioral and social factors that can potentially raise or lower population size in each BMI class such as the dynamic models developed by Hill et al.(5) and Keisuke et al(6). These models predicted obesity prevalence by segregating normal weight and obese populations into two compartments. However, existing models did not include the progression from normal weight to overweight and finally to the obese classification. Existing models also did not predict the prevalence of extreme obesity and the model did not include important moderators of obesity prevalence such as the impact of childhood obesity, differential population birth rates, and the higher susceptibility to weight regain in individuals who have lost weight.

Here we present a comprehensive differential equation model that overcomes these limitations by incorporating the mechanisms known to increase or decrease the population prevalence within each BMI class. The model was designed to predict obesity prevalence after input of country specific parameters, resulting in a highly flexible model that can be applied to other developed countries or communities. The proposed model is used here to determine whether the US obesity epidemic will plateau and how soon this will occur. These are issues of high national significance due to the medical, fiscal and social consequences imposed by excess population adiposity.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

METHODS

Model Development

Using the well-established susceptible, infected, recovered (SIR) model framework (7–9) from infectious disease modeling, we developed six differential equations that describe interactions and transitions between populations with different body mass index (BMI) classifications. SIR models have been applied to model the characteristics of a variety of infectious disease outbreaks such as HIV(10), tuberculosis (11), and influenza (12). SIR models also have been successfully applied to capture the dynamics of non-communicable conditions such as alcoholism (13), ecstasy use (14), and criminal activity (9). The application of SIR models in these conditions does not assume the mechanisms behind contagion, rather they reflect the overall dynamics, produce predictions of long-term outcomes, and identify which parameters have the most impact on the evolution of the epidemic.

The SIR approach divides a population into compartments of infected and non-infected individuals and model terms are constructed to describe the flow to and from each compartment (Figure 1). Complete details and a step by step model formulation appear in the Appendix. Here we outline the main qualitative properties central to our model for obesity prevalence.

Individuals in a population are deemed susceptible in our model if their BMI is below 25 kg/m^2 and they have never been overweight. In order to incorporate the long time scale necessary for normal weight individuals to become overweight, we introduced a class of individuals who were exposed to either social or non-social influences that lead to weight gain and these individuals will eventually become overweight. Thus, the exposed class can be considered as a latency period for obesity. The ‘infected’ population was comprised of three different classes; an overweight population ($25 \leq BMI < 30$ kg/m^2), an obese population ($30 \leq BMI < 40$ kg/m^2), and an extremely obese population ($BMI \geq 40$ kg/m^2). Spontaneous transition to overweight, obese, and extremely obese independent of social influence was modeled by linear terms, similar to the Hill model (5). Socially influenced transition to overweight, obese, and extremely obese was modeled by a mass action term (5). Overweight individuals who lose enough weight to return back to normal BMI (≤ 25 kg/m^2) are considered recovered. Weight regain in these individuals is assumed to occur at a higher infectivity rate in comparison to normal weight susceptible individuals who progress normally to the overweight category. In addition, the model allows for modest weight loss in the obese and extremely obese classes, which returns the individual to a lower BMI category. For example, obese individuals may lose enough weight to be classified as overweight and as a result move from the obese category to the overweight category. The model time scale also includes the natural population birth and death rates.

The model assumes extremely obese individuals do not have the capacity to socially draw other classes toward obesity. This model criterion is based on recent literature (15–17) demonstrating that individuals do not “imitate” the behavior of the extremely obese. Additionally, individuals exposed to the effects of obesity at birth are considered born in an “obesogenic” environment and are thereby considered more susceptible to becoming

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

overweight and later obese. Finally, because the epidemiological literature indicates that obese individuals die at an earlier age than normal weight people with the years of life lost in the obese population ranging from two to seven years (18, 19), the model included differential death rates.

We acknowledge the possible existence of normal weight individuals who are “immune” to weight gain. Since there is no flow out of the immune class, this population does not impact model dynamics and therefore a model equation is not required for this subpopulation. Finally, we note that only the flow rates between compartments are key factors in the model. Importantly, a population plateau or trend curve shape is *not assumed* as part of model development.

Model parameters

Models were created and simulations performed for the Unites States (US) and United Kingdom (UK). Model birth and death rates were set using population birthrate from published survey data for the US (see Table 1)(20). The model was also simulated based on survey data (Table 1) from the United Kingdom (21). The probability of being born into an obesogenic environment was estimated from the percentage of reproductive age women classified as overweight or obese. All other model parameters were set using initial trends from US based or UK-based obesity prevalence values (Table 1) for their respective simulation (20, 21). Some model parameters such as the rate of effective interaction between overweight and obese individuals are impossible to know, however, the range is fixed by knowledge of total prevalence in each category and thus dynamics can be examined by fluctuating these parameters within their ranges. Specifically, only information from 1988–1998 for the U.S. and 1993–1997 for the U.K. was used to fit parameters. The model was simulated forward and then compared to the actual data past 1998 to test for agreement. Details on specific parameter calculations are provided in Table 1.

To observe the effects of varying birthrates on trajectories, the model was simulated for four different birthrates; one birth per 1000 individuals (0.0010), 14.4 births per 1000 individuals (US birthrate, 0.0144), 20 births per 1000 individuals (0.0200), and 50 births per 1000 individuals (0.0500). All other parameters specific to the US simulation appear in Table 1. Similarly, the effects of varying the probability of being born in an obesogenic environment on future prevalence rates were analyzed by simulating the model with respect to maternal obesity for probability values, 0.0, 0.55, and 0.95, while holding all other parameters fixed. A similar analysis was conducted by raising the death rate of the obese and extremely obese from the uniform death rate value ($D_0 = 0.0144$), to $D_0 = 0.0150$ and finally $D_0 = 0.02$. Similar to the other analysis, other parameters specific to the US simulation appear in Table 1.

Model analysis

Long-term behavior or trends are analyzed by first calculating the equilibria or steady-states. This is achieved by setting the derivatives equal to zero and solving the resulting equations algebraically, as shown in the Appendix. The next step was to determine whether the trajectories defined by the differential equation actually plateau at the calculated steady-state

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

value. If they do, we refer to the steady-state as a plateau. A rigorous mathematical proof of the existence of a plateau relying on well-established differential equation theory (22) is included in the Appendix.

Model simulations

Specific model trajectories were simulated using the default differential equation solver available through Matlab r2012a (2012, MathWorks, MA).

Web-based program

The model was programmed to permit interested users to input parameters and baseline prevalence values and observe the resulting obesity prevalence rates predicted over time by the model with a graphical display of results. The web-based program can be accessed at http://www.pbrc.edu/research-and-faculty/calculators/obesity-prevalence/.

RESULTS

An obesity prevalence plateau

For any parameter choice, trajectories converge to a positive plateau (see Appendix for mathematical analysis). Using US prevalence data from 1988, model simulations reveal that it takes an approximately 40 year period for obesity percentages to plateau at prevalence rates of 28% for overweight, 32% for obesity, and 9% for extreme obesity (Figure 2 **Panel A**).

Applying parameter and baseline conditions to the UK conditions indicates that approximately 21% of the population will be overweight, 27% will be obese, and 5% extremely obese by 2033 (Figure 2 **Panel B**). In contrast to the US simulation, the UK simulation revealed that a plateau will not be reached before 2033. The parameters related to socially influenced weight gain estimated in the UK simulation did not differ from the analogous parameters determined in the US case. However, the parameters related to weight gain from non-social influences in the UK simulation were significantly lower than those in the US case (Table 1). The plateau for the US was directly calculated from the closed form expressions of the equilibrium (see the Appendix) as 26.8% classified overweight, 31.1% classified obese, 9.8% classified extremely obese. For the UK, the plateau was determined to be 25.7% classified overweight, 39.6% classified obese, and 9.4% classified extremely obese.

Model Validation

Since US model parameters were fit to data from 1988–1998, model simulations in this time interval represent calibration and not true prediction. However, as observed in Figure 2 A, past 1998, the model simulations demonstrated good agreement with mean data from 2008. Likewise, since we applied data points from 1993–1997 to fit UK model parameters, we expect good agreement between model simulations and actual data in this time interval. However, as observed in Figure 2 B, there is good agreement between model simulations and reported mean data between 1997 and 2008. To distinguish calibration from validation

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

and forecast, Figure 2 curves were depicted as solid for calibration, dashed for validation, and dotted for forecast.

The dependence of the plateau on population birth rate

The model analysis revealed that the level at which obesity rates plateau in a population depends on birth rate expressed as childbirths per 1000 people per year. Specifically, higher birthrate leads to increased time to plateau and lower obesity prevalence. Figure 3 A illustrates this phenomenon. It shows three different trajectories where all parameters are equal (US parameters in Table 1) except for birthrate. While this result may seem counterintuitive, a large new influx of births into the susceptible category replenishes the system. A higher birthrate yields a larger normal weight category and hence this category requires a longer time to proceed toward obesity and influence the final prevalence plateau.

The dependence of the plateau on the probability of being born into an obesogenic environment

The dependence of the level at which obesity rates will plateau also depends on the probability of being born into an obesogenic environment reflecting risk of childhood obesity. As the probability of being born into an obesogenic environment increases, the value at which obesity plateaus increases, and the time to plateau increases. This is illustrated in Figure 3B for three probability values of being born into an obesogenic environment as indexed herein by maternal weight during or around pregnancy.

The dependence of the plateau on the differential death rate

Similar to the probability of being born into an obesogenic environment, the differential death rate for obese and extremely obese populations also impacts the level at which obesity rates will plateau. It was found that the higher the differential death rate, the lower the plateau value, illustrated in Figure 3C.

DISCUSSION

This study proposes a dynamic model that predicts obesity prevalence (5) by including interactions and transitions between populations of different BMI classes, population wide differential birthrate, differential death rate, probability of being born into an “obesogenic” environment, and the lag time involved in weight gain. Dynamic models such as the one developed in our study capture long-term trends without being dependent on databases or *a priori* determination of the type of curve the trend will follow. Rather, our dynamic model relies on the relationships between segments of the populations and then predicts flow based on these input and output relationships.

Parameters were fit to the newly-developed dynamic model using US prevalence data and birth and death rates from 1988 to 1998. If these parameters remain constant, the model predicts plateaus by the year 2030 at prevalence rates of 28%, 32% and 9% for overweight, obesity, and extreme obesity, respectively. Similarly, we applied model parameters fit to data from the UK and found that approximately 34% of the population will be overweight,

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

32% will be obese, and 5% extremely obese by 2033, though a plateau was not reached by 2033 in the UK simulation.

The model formulation described in this study provides a foundation for the inclusion of additional possible influences on obesity prevalence. There is no need to develop an entirely new model to include additional influences. Only the flow rates or specific model terms would need to be altered.

Many countries do not have a stable birth rate but have either increasing (China) or decreasing (European countries) birth rates. In fact, the birth rates in the US have decreased by 50% from 1950–1970 and held fairly steady at approximately 14 births per 1000 people since then (23). For model tractability, we assume a constant birth rate; however, a time dependent birth rate would provide potential for improved understanding of birth rate impacts on obesity prevalence. Changes in birth rate or other parameters would induce a "jumping plateau" effect as seen in Denmark for example (24) where obesity prevalence plateaus and then increases past this plateau only to plateau at a new value.

Another useful factor for model advancement would be the inclusion of immigration effects. We noted here that the new influx of normal weight individuals through births impacted prevalence rates and time to plateau. It stands to reason that a new influx through immigration would also have an effect on long-term trends, depending in part on the characteristics of the migrants.

The model can be applied to a particular state by inputting birth rates and other parameters for the specific state. However, because the model does not include migration in and out of the region, the predictions would be overly simplified. To capture the full geographic dynamics, a geographic spread model would have to be developed.

Geographical models describing the spread of infectious diseases involve combining models as the one presented here with a conservation law and Fick's law of diffusion (25). These spatial models could be applied to evaluate the effectiveness of obesity control strategies across geographical locations. However, the current models that predict obesity trends do not consider a potential progressive spread of obesity over geographical locations. Rather, they consider solely overall population trends, evolving over time. With careful analysis of regional patterns in obesity trends, our model can be extended to include a geographical diffusion component. Understanding the dynamics of how obesity moves geographically from obesity hotspots and how the borders of these hotspots influence future geographical spread of obesity are important but under-investigated issues.

Model application to developing countries

The model developed in this paper is based on assumptions most applicable to Western countries. Many developing countries have rapidly changing birth rates, infectious disease related deaths, high infant mortality, and rapid changes in food supplies and transportation systems as they undergo rapid nutritional and lifestyle transitions. These and possibly other influences will impact predictions. Some of these factors can be encompassed through variable birthrates and differential death rates. However, specific reviews of these different

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

influences and factors will need to be considered for appropriate model application to developing countries.

Additional potential model extensions

The current model provides a framework for extensions. For example, one can consider assortative mating (26) or differential birth rates by revising these terms within the currently developed model. To include the effects of age structure, the current model can be revised as an age structured model (27). Such inclusions will need to be well thought out and carefully analyzed since they will increase the complexity of the model.

In summary, we have developed a comprehensive and dynamic mathematical model to predict changes in overweight, obesity and extreme obesity prevalence. The model is flexible and can be adapted to specific parameters of a community, region, ethnic subpopulation, or country. The model predicts a slower increase in obesity prevalence and an eventual plateau of the obesity epidemic by about 2030 in the United States. It should be noted that despite the predicted deceleration in obesity rate, the prevalence of obesity remains sufficiently high at all times to warrant new and effective obesity prevention and management strategies. This model provides a baseline to evaluate the efficacy of various obesity prevention and management strategies and policies. To be effective, changes in policies and disease prevention programs will need to produce a change in obesity prevalence that is larger than predicted by our model for the natural course of the epidemic.

Acknowledgments

Funding/Support

This research was supported in part by National Institutes of Health grants R15 DK090739, U01DK094418, HL45670, the John W. Barton Sr. Chair in Genetics and Nutrition, and a NORC Center Grant # 2P30DK072476 entitled “Nutritional Programming: Environmental and Molecular Interactions” sponsored by NIDDK.

References

1. Baskin ML, Ard J, Franklin F, Allison DB. Prevalence of obesity in the United States. Obes Rev. 2005; 6(1):5–7. Epub 2005/01/19 OBR165 [pii]. 10.1111/j.1467-789X.2005.00165.x [PubMed: 15655032]
2. Bray GA, Macdiarmid J. The epidemic of obesity. West J Med. 2000; 172(2):78–9. Epub 2000/02/29. [PubMed: 10693361]
3. Finkelstein EA, Khavjou OA, Thompson H, Trogdon JG, Pan L, Sherry B, et al. Obesity and severe obesity forecasts through 2030. Am J Prev Med. 2012; 42(6):563–70. Epub 2012/05/23 S0749-3797(12)00146-8 [pii]. 10.1016/j.amepre.2011.10.026 [PubMed: 22608371]
4. Wang Y, Beydoun MA, Liang L, Caballero B, Kumanyika SK. Will all Americans become overweight or obese? estimating the progression and cost of the US obesity epidemic. Obesity (Silver Spring). 2008; 16(10):2323–30. Epub 2008/08/23 oby2008351 [pii]. 10.1038/oby.2008.351 [PubMed: 18719634]
5. Hill AL, Rand DG, Nowak MA, Christakis NA. Infectious disease modeling of social contagion in networks. PLoS Comput Biol. 2010; 6(11):e1000968. Epub 2010/11/17. 10.1371/journal.pcbi.1000968 [PubMed: 21079667]
6. Andersen LG, Baker JL, Sorensen TI. Contributions of incidence and persistence to the prevalence of childhood obesity during the emerging epidemic in Denmark. PLoS One. 2012; 7(8):e42521. Epub 2012/08/18. 10.1371/journal.pone.0042521 [PubMed: 22900026]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

7. Kermack W, McKendrick A. A Contribution to the Mathematical Theory of Epidemic. Proc R Soc Lond. 1927; 115:700–21.
8. Castillo-Chavez C, Castillo-Garsow CW, Yakubu AA. MSJAMA. Mathematical models of isolation and quarantine. JAMA. 2003; 290(21):2876–7. Epub 2003/12/06. doi: 10.1001/jama.290.21.2876 290/21/2876. [PubMed: 14657077]
9. Brauer, F.; Castillo-Chávez, C. Mathematical models in population biology and epidemiology. 2. New York: Springer; 2012. p. xxiiip. 508
10. Nowak MA, McLean AR. A mathematical model of vaccination against HIV to prevent the development of AIDS. Proc Biol Sci. 1991; 246(1316):141–6. Epub 1991/11/22. 10.1098/rspb.1991.0136 [PubMed: 1685238]
11. Castillo-Chavez C, Song B. Dynamical models of tuberculosis and their applications. Math Biosci Eng. 2004; 1(2):361–404. Epub 2004/09/01. [PubMed: 20369977]
12. Simon C, Yosinao N. A mathematical model to distinguish sociological and biological susceptibility factors in disease transmission in the context of H1N1/09 influenza. J Theor Biol. 2011; 286(1):50–6. Epub 2011/07/27 S0022-5193(11)00358-4 [pii]. 10.1016/j.jtbi.2011.07.008 [PubMed: 21787790]
13. Gorman DM, Gruenewald PJ, Hanlon PJ, Mezic I, Waller LA, Castillo-Chavez C, et al. Implications of systems dynamic models and control theory for environmental approaches to the prevention of alcohol- and other drug use-related problems. Subst Use Misuse. 2004; 39(10–12):1713–50. Epub 2004/12/14. [PubMed: 15587949]
14. Song B, Castillo-Garsow M, Rios-Soto KR, Mejran M, Henso L, Castillo-Chavez C. Raves, clubs and ecstasy: the impact of peer pressure. Math Biosci Eng. 2006; 3(1):249–66. Epub 2006/01/01. [PubMed: 20361822]
15. Puhl R, Brownell KD. Bias, discrimination, and obesity. Obes Res. 2001; 9(12):788–805. Epub 2001/12/18. 10.1038/oby.2001.108 [PubMed: 11743063]
16. Wardle J, Volz C, Golding C. Social variation in attitudes to obesity in children. Int J Obes Relat Metab Disord. 1995; 19(8):562–9. Epub 1995/08/01. [PubMed: 7489027]
17. Greenberg BS, Eastin M, Hofschire L, Lachlan K, Brownell KD. Portrayals of overweight and obese individuals on commercial television. Am J Public Health. 2003; 93(8):1342–8. Epub 2003/08/02. [PubMed: 12893625]
18. Fontaine KR, Redden DT, Wang C, Westfall AO, Allison DB. Years of life lost due to obesity. JAMA. 2003; 289(2):187–93. Epub 2003/01/09 joc20945 [pii]. [PubMed: 12517229]
19. Peeters A, Barendregt JJ, Willekens F, Mackenbach JP, Al Mamun A, Bonneux L. Obesity in adulthood and its consequences for life expectancy: a life-table analysis. Ann Intern Med. 2003; 138(1):24–32. Epub 2003/01/07 200301070-00008 [pii]. [PubMed: 12513041]
20. Flegal KM, Carroll MD, Ogden CL, Curtin LR. Prevalence and trends in obesity among US adults, 1999–2008. JAMA. 2010; 303(3):235–41. Epub 2010/01/15 2009.2014 [pii]. 10.1001/jama.2009.2014 [PubMed: 20071471]
21. Health Survey for England - 2010: Trend tables
22. Hale, JK. Ordinary differential equations. 2. Huntington, N.Y: R. E. Krieger Pub. Co; 1980. p. xvip. 361
23. United States Census Bureau. The 2012 Statistical Abstract: Births, Deaths, Marriages, & Divorces. 2012.
24. Due P, Heitmann BL, Sorensen TI. The obesity epidemic in Denmark. Ugeskr Laeger. 2006; 168(2):129–32. Epub 2006/01/13 VP46220 [pii]. [PubMed: 16403333]
25. Balcan D, Goncalves B, Hu H, Ramasco JJ, Colizza V, Vespignani A. Modeling the spatial spread of infectious diseases: the GLobal Epidemic and Mobility computational model. J Comput Sci. 2010; 1(3):132–45. Epub 2011/03/19. 10.1016/j.jocs.2010.07.002 [PubMed: 21415939]
26. Jacobson P, Torgerson JS, Sjostrom L, Bouchard C. Spouse resemblance in body mass index: effects on adult obesity prevalence in the offspring generation. Am J Epidemiol. 2007; 165(1):101–8. Epub 2006/10/17. 10.1093/aje/kwj342 [PubMed: 17041131]
27. Oizumi R, Takada T. Optimal life schedule with stochastic growth in age-size structured models: Theory and an application. J Theor Biol. 2013; 323:76–89. Epub 2013/02/09. 10.1016/j.jtbi.2013.01.020 [PubMed: 23391431]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

References

1. Thomas DM, Schoeller DA, Redman LA, Martin CK, Levine JA, Heymsfield SB. A computational model to determine energy intake during weight loss. Am J Clin Nutr. Dec; 2010 92(6):1326–1331. [PubMed: 20962159]
2. Hale, JK. Ordinary differential equations. 2. Huntington, N.Y: R. E. Krieger Pub. Co; 1980.

APPENDIX. DETAILED MODEL DESCRIPTION

The appendix is a self-contained complete description of the obesity prevalence model development and analysis. The appendix is separated into four distinct sections. The first section presents details of model development including model assumptions, definitions of model state variable, description of the model parameters, and the formulation of all model terms. The second section provides step by step numerical parameter calculation details for the United States and the United Kingdom. The third section provides a lay description of the conclusive theoretical model results and their biological meaning. The last section provides rigorous theorem statements and their proofs using traditional mathematical notation and classical mathematical proof structure.

I. MODEL DEVELOPMENT

The Newtonian derivative of a variable represents the instantaneous rate of change for the variable changing with time. An ordinary differential equation is an equation that relates the derivative of a variable to a function of itself. A system of ordinary differential equations represents several equations that relate a set of interconnected variables that influence each other and impact their derivatives. Because individuals cannot transition to obesity without moving from a normal to obese BMI classification, this movement or flow is appropriately described by differential equation.

Modeling this flow requires identification of the mechanisms which increase or decrease the population within each BMI classification. The mechanisms considered in the developed model appear as a flowchart in Figure 1. Each compartment depicted in Figure 1 represents a *state variable* or more importantly, a variable we desire a prediction for over time. Table 1 lists each term of the differential equation model that describes the flow from and to each compartment as depicted in Figure 1.

Model assumptions

Every mathematical model relies on assumptions. Some assumptions are instated to enhance model tractability, for if we included every possible effect, the model will be so complex that we would be unable to analyze it. These are referred to as *simplifying assumptions*. As models advance in complexity, simplifying assumptions are typically disposed over time through careful investigation of how to conduct the ensuing analysis. Non-simplifying model assumptions are based on known facts that influence the phenomena we are modeling, which in this case is the time dependent changes in the prevalence of obesity. The list that follows outlines all assumptions behind our model formulation.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

(A1) The immune population does not contribute to the analysis, and thus can be ignored.

(A2) The population can be compartmentalized as follows:

$S(t)$ Individuals consistently classified with BMI <25 at year t (susceptible).

$E(t)$ Individuals consistently classified with BMI < 25 that have been effectively exposed by year t but are not yet overweight (exposed).

$I_1(t)$ Individuals classified as overweight (25≤BMI < 30) at year t.

$I_2(t)$ Individuals classified as obese (30≤BMI<40) at year t.

$I_3(t)$ Individuals classified as extremely obese (40≤BMI) at year t.

$R(t)$ Individuals who have reduced back to normal weight at year t (susceptible but predisposed to weight regain).

(A3) The extremely obese cannot socially influence population weight gain.

(A4) The death rate D_O for obese and extremely obese populations is higher than the death rate D of susceptible, exposed, overweight, and recovered populations.

(A5) Recovered individuals are susceptible to becoming overweight once again at a different rate than normal weight individuals who have never been overweight. This transition is independent of social influence.

(A6) Social interactions between compartments are governed by the law of mass action and modeled by multiplying the population numbers in each class.

(A7) A fraction of the population is born with a higher risk for becoming obese.

(A8) Individuals can spontaneously gain weight through non-social influences. As a result not all movement through the different BMI classifications arises from social interaction.

We formulate assumptions (A1)–(A8) using a system of six differential equations and begin by denoting the total population in year t, $N(t)$ as the sum of the state variables,

$$S(t), E(t), I_1(t), I_2(t), I_3(t), R(t):$$
$$N(t)=S(t)+E(t)+I_1(t)+I_2(t)+I_3(t)+R(t)$$

to arrive at the obesity prevalence model:

$$S'(t)=(1-p)\mu N(t)-DS(t)-\frac{k_1 I_1(t)S(t)}{N(t)}-\frac{k_2 I_2(t)S(t)}{N(t)}-\alpha S(t)$$
$$E'(t)=p\mu N(t)-DE(t)-aE(t)+\frac{k_1 I_1(t)S(t)}{N(t)}+\frac{k_2 I_2(t)S(t)}{N(t)}+\rho_R R(t)+\alpha S(t)$$
$$I_1'(t)=-DI_1(t)+aE(t)-a_1 I_1(t)-\rho_1 I_1(t)+\beta_2 I_2(t)$$
$$I_2'(t)=-D_o I_2(t)+a_1 I_1(t)-a_2 I_2(t)-\beta_2 I_2(t)+\beta_3 I_3(t)$$
$$I_3'(t)=-D_o I_3(t)+a_2 I_2(t)-\beta_3 I_3(t)$$
$$R'(t)=-DR(t)+\rho_1 I_1(t)-\rho_R R(t)$$

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

The initial values and parameters must be non-negative: $S(0)$, $E(0)$, $I_1(0)0$, $I_2(0)$, $I_3(0)$, $R(0) \geq 0$ and $\mu, p, k_1, k_2, D, \alpha, a, \rho_R, D_0, \rho_1, \rho_2, a_1, a_2, \beta_1, \beta_2, \beta_3 \geq 0$. Since we are interested in studying the time-dependent changes in obesity prevalence within a fixed population, we will impose a further restriction that the total population remains constant.

(A9) The total population shall remain constant for all times t. That is,

$$N(t)=N=S(t)+E(t)+I_1(t)+I_2(t)+I_3(t)+R(t)$$

Assumption (A9) is consistent with the above system of differential equations provided that

$$\frac{d}{dt}\left(S(t)+E(t)+I_1(t)+I_2(t)+I_3(t)+R(t)\right)=0,$$

which holds when the total number of births equals the total number of deaths at all times t: $\mu N(t) = D(S(t) + E(t) + I_1(t) + R(t)) + D_o(I_2(t) + I_3(t)) = DN(t) + (D_o - D)(I_2(t) + I_3(t))$. The inclusion of this assumption further reduces the model to:

$$\begin{aligned}
S'(t)&=(1-p)\left(DN+(D_o-D)(I_2(t)+I_3(t))\right)-DS(t)-\tfrac{k_1 I_1(t)S(t)}{N}-\tfrac{k_2 I_2(t)S(t)}{N}-\alpha S(t)\\
E'(t)&=p\left(DN+(D_o-D)(I_2(t)+I_3(t))\right)-DE(t)-aE(t)+\tfrac{k_1 I_1(t)S(t)}{N}+\tfrac{k_2 I_2(t)S(t)}{N}+\rho_R R(t)+\alpha S(t)\\
I_1'(t)&=-DI_1(t)+aE(t)-a_1 I_1(t)-\rho_1 I_1(t)+\beta_2 I_2(t)\\
I_2'(t)&=-D_o I_2(t)+a_1 I_1(t)-a_2 I_2(t)-\beta_2 I_2(t)+\beta_3 I_3(t)\\
I_3'(t)&=-D_o I_3(t)+a_2 I_2(t)-\beta_3 I_3(t)\\
R'(t)&=-DR(t)+\rho_1 I_1(t)-\rho_R R(t)
\end{aligned}$$

Incorporating assumption (A9) also implies that we can replace the equation of one of the subpopulations. Letting $R(t) = N - S(t) - E(t) - I_1(t) - I_2(t) - I_3(t)$ results in the 5-dimensional model:

$$\begin{aligned}
S'(t)&=(1-p)\left(DN+(D_o-D)(I_2(t)+I_3(t))\right)-DS(t)-\tfrac{k_1 I_1(t)S(t)}{N}-\tfrac{k_2 I_2(t)S(t)}{N}-\alpha S(t)\\
E'(t)&=p\left(DN+(D_o-D)(I_2(t)+I_3(t))\right)-DE(t)-aE(t)+\tfrac{k_1 I_1(t)S(t)}{N}+\tfrac{k_2 I_2(t)S(t)}{N}+\rho_R\left(N-S(t)-E(t)-I_1(t)-I_2(t)-I_3(t)\right)+\alpha S(t)\\
I_1'(t)&=-DI_1(t)+aE(t)-a_1 I_1(t)-\rho_1 I_1(t)+\beta_2 I_2(t)\\
I_2'(t)&=-D_o I_2(t)+a_1 I_1(t)-a_2 I_2(t)-\beta_2 I_2(t)+\beta_3 I_3(t)\\
I_3'(t)&=-D_o I_3(t)+a_2 I_2(t)-\beta_3 I_3(t)
\end{aligned}$$

We refer to the above system of equations as **System 1** and will assume $S(0)$, $E(0)$, $I_1(0)0$, $I_2(0)$, $I_3(0) \geq 0$ in all our rigorous proofs.

The special case of uniform death rates

In the case where $D = D_0$ the constant population assumption reduces to $\mu = D$ and **System 1** can be written in terms of the birth rate:

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

$$S'(t)=(1-p)(\mu N)-\mu S(t)-\frac{k_1 I_1(t)S(t)}{N}-\frac{k_2 I_2(t)S(t)}{N}-\alpha S(t)$$
$$E'(t)=p(\mu N)-\mu E(t)-aE(t)+\frac{k_1 I_1(t)S(t)}{N}+\frac{k_2 I_2(t)S(t)}{N}+\rho_R\,(N-S(t)-E(t)-I_1(t)-I_2(t)-I_3(t))+\alpha S(t)$$
$$I_1{}'(t)=-\mu I_1(t)+aE(t)-a_1 I_1(t)-\rho_1 I_1(t)+\beta_2 I_2(t)$$
$$I_2{}'(t)=-\mu I_2(t)+a_1 I_1(t)-a_2 I_2(t)-\beta_2 I_2(t)+\beta_3 I_3(t)$$
$$I_3{}'(t)=-\mu I_3(t)+a_2 I_2(t)-\beta_3 I_3(t)$$

The advantage of working with this simplified system allows us to easily observe the impact of birth rate on obesity prevalence. All simulations and the web-calculator were performed using this simplified model, while all theoretical results were proved using the general model.

II. DETERMINATION OF MODEL PARAMETERS

Model parameters were determined using the method of shooting [1]. Shooting requires an initial guess of all parameter values that can be chosen by the user (Figure 2 **Panel A**) and is not determined by population data such as birth rate. The resulting trajectories are directly inspected. If the rate of growth is too high, that is the initial increase of prevalence rates rose too quickly past the data points provided from 1988–1998, we adjust the parameters that contribute to growth by reducing them. Similarly, if the initial increase was not high enough, parameters that contribute to growth were raised. This method of feedback testing requires several iterations because adjusting one parameter impacts other compartments. However, once the initial growth rates have been adjusted so that the resulting trajectories move through the data points provided by the 1988–1998 NHANES prevalence data, the parameters are fixed (Figure 2 **Panel B**).

III. LAY DESCRIPTION OF THEORETICAL RESULTS

Good modeling practice requires that is that basic properties of the phenomena are intrinsic to the model itself. For example, in this case since we are modeling obesity prevalence over time, we expect that our model should not give rise to solutions that are negative or unbounded. Indeed, we hope that solutions even exist, since there is no assurance that simply writing down differential equations will guarantee existence of a solution. Thus, the first rigorous result verifies not only the existence of solutions to the model, but that solutions are non-negative and bounded.

Proposition 1

The solution to the model which represents prevalence of obesity in a population is never negative and is capped by the amount of the total population.

Since the solution to System 1 cannot be expressed as a stand-alone formula, the next question is whether we can determine what will happen as a forecast. Perhaps obesity prevalence may oscillate over time, or perhaps it will have random patterns. The initial collected data in most countries do not suggest such patterns and we should hope that the outcome of the model follows the observed trend.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

In fact, we prove in two steps that the solutions plateau over time. We first show the existence of a steady state or plateau value and then we show that any solution that starts near this value will head to this value (plateau) over time.

Proposition 2

The model yields a unique positive value that is a steady state or a plateau. If obesity prevalence arrives at this value, it remains at this value.

Corollary

For the cases of the US and UK described in Table 1, obesity prevalence will plateau. For the US, the plateau will occur at 31.09% for obese (30≤BMI<40) and 9.80% for extremely obese (40≤BMI). For the UK, the plateau will occur at 39.62% for obese and 9.37% for extremely obese.

The method of proving whether obesity prevalence will plateau requires evaluation and analysis of a 5 by 5 matrix called the Jacobian, which cannot be conducted symbolically. Thus, country specific parameter values need to be entered into the matrix. On the other hand, numerical simulation is straightforward and can be achieved using the online calculator accessible at: http://www.pbrc.edu/research-and-faculty/calculators/obesity-prevalence/

IV. MATHEMATICAL PROOFS OF THEORETICAL RESULTS

In this section, we prove that

1. Solutions to our model exist, are bounded, and are nonnegative.
2. That for any choice of parameters and baseline prevalence, all trajectories will plateau.
3. The plateau value will be positive, indicating that obesity cannot be eradicated on its own.

Existence of solutions is guaranteed by Peano's Existence Theorem, see for example[2]. Instead of proving non-negativity and boundedness of solutions directly, we will identify a positively invariant set X of System 1. The set is such that if the initial conditions lie in X, then so do the corresponding solutions, which must then be non-negative and bounded because of the structure of the set X.

Based on assumption (A9), we define the set

$X=\{(S,E,I_1,I_2,I_3)\in R_+^5 : S+E+I_1+I_2+I_3\leq N\}$, where R_+^5 denotes the set of all quintuples with non-negative components. We will see that X is an invariant set of System 1. To see that solutions remain in X for all times $t\geq 0$, note first that it is true for $t=0$ based on the initial conditions and assumption (A9). If $(S(t), E(t), I_1(t), I_2(t), I_3(t))\in X$ for some $t\geq 0$, then the right hand side of $S'(t)$ is non-negative whenever $S(t)=0$ and $I_j(t)\geq 0$ for $j=1, 2$. Similarly, $I_j'(t)\geq 0$ whenever $I_j(t)=0$, $E(t)\geq 0$, $S(t)\geq 0$, and $I_k(t)\geq 0$ for; $j\neq k$. Also $E'(t)\geq 0$ whenever $E(t)=0$ and provided that $(S(t), E(t), I_1(t), I_2(t), I_3(t))\in X$. This shows that

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

$(S(\tilde{t}), E(\tilde{t}), I_1(\tilde{t}), I_2(\tilde{t}), I_3(\tilde{t})) \in R_+^5$ for $t < \tilde{t} < t + \delta$ for some $\delta > 0$. At the same time, $\Sigma(t) = S(t) + E(t) + I_1(t) + I_2(t) + I_3(t)$ gives $\Sigma'(t) = (D + \rho_R)(N - \Sigma(t)) - \rho_1 I_1(t)$. If $(S(t), E(t), I_1(t), I_2(t), I_3(t)) \in X$ and $D \leq D_O$, then $\Sigma'(t) \leq (D + \rho_R) N - (D + \rho_R) \Sigma(t)$. Solving this differential inequality for $t \geq \tilde{t}$ yields $\Sigma(\tilde{t}) \leq \Sigma(t) e^{-(D+\rho_R)(t-t)} + N(1 - e^{-(D+\rho_R)(t-t)})$ From here we can see that $\Sigma(t) \leq N$ implies $\Sigma(\tilde{t}) \leq N$. Thus, $(S(t), E(t), I_1(t), I_2(t), I_3(t)) \in X$ implies that $(S(\tilde{t}), E(\tilde{t}), I_1(\tilde{t}), I_2(\tilde{t}), I_3(\tilde{t})) \in X$ for $t < \tilde{t} < t + \delta$ for some $\delta > 0$. Consequently, X is a positively (also forward) invariant set. We have proved the following.

Proposition 1

Assume $D \leq D_O$ Then all forward solutions to System 1 corresponding to initial conditions in X are non-negative and bounded (and remain in X).

Furthermore $D \leq D_O$ ensures that the set X, attracts R_+^5. This can be seen from $\Sigma(t) \leq \Sigma(0) e^{-(D+\rho_R)t} + N(1 - e^{-(D+\rho_R)t})$, which implies that $limsup_{t\to\infty} \Sigma(t) \leq N$.

Predicted trends plateau (Equilibria and Global Stability)—System 1 has one positive equilibrium in X. Using I_1 to express the equilibrium values for each subpopulation, we obtain:

$$S^* = \frac{(1-p)DN + (D-D_o)(\alpha_{12}+\alpha_{13})}{\alpha + D + kI_1^*} I_1^*$$
$$E^* = \alpha_{1E} I_1^*$$
$$I_2^* = \alpha_{12} I_1^*$$
$$I_3^* = \alpha_{13} I_1^*$$

and I_1^* corresponds to the solutions of the quadratic equation $v_2 Z^2 + v_1 Z + v_o = 0$, where

$$v_2 = k\left((D_o - D)(\alpha_{12}+\alpha_{13}) - (D+a)\alpha_{1E} - \rho_R(1 + \alpha_{1E} + \alpha_{12} + \alpha_{13})\right)$$
$$v_1 = k(\rho_R + D)N + \frac{(\alpha+D)}{k} v_2 - (D+\rho_R)(D_o - D)(\alpha_{12}+\alpha_{13})$$
$$v_o = (\alpha + pD)(D+\rho_R)N > 0$$

In the above expressions,

$\alpha_{12} = \frac{a_1(D_o+\beta_3)}{(D_o+\beta_2)(D_o+\beta_3)+a_2 D_o}$, $\alpha_{13} = \frac{a_2}{D_o+\beta_3}\alpha_{12}$, $\alpha_{1E} = \frac{1}{a}(a_1 + D + \rho_1 - \beta_2\alpha_{12}) > 0$, and $k = \frac{k_1}{N} + \alpha_{12}\frac{k_2}{N}$. Consider the function $f(Z) = v_2 Z^2 + v_1 Z + v_o$. When $D_o = D$, the leading coefficient v_2 reduces to

$$\overline{v}_2 = -k\left((D+a)\alpha_{1E} + \rho_R(1 + \alpha_{1E} + \alpha_{12} + \alpha_{13})\right) < 0$$

and $v_2 < 0$ continues to be true as long as $D_o - D < \frac{-\overline{v}_2}{\alpha_{12}+\alpha_{13}}$. This condition then guarantees that the quadratic equation $f(Z) = 0$ has a unique positive solution I_1^*.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Proposition 2

Assume $0 \leq D_o - D < \frac{-\overline{v}_2}{\alpha_{12}+\alpha_{13}}$. Then System 1 has a unique positive equilibrium (S^*, E^*, I_1^*, I_2^*, I_3^*) in X. The equilibrium can be described in terms of I_1^*, which is given by

$$I_1^* = \frac{-v_1 - \sqrt{v_1^2 - 4v_o v_2}}{2v_2}$$

All other values of the equilibrium are given above.

Proposition 1 implies the existence of a compact attractor in the set X and the positive equilibrium lies in X and must be contained in the attractor. For parameter values given in Table 1, one can verify that the positive equilibrium is locally asymptotically stable by showing that all eigenvalues of the corresponding Jacobian have negative real parts.

The Jacobian is given by

$$\begin{pmatrix} -D-\alpha-\frac{k_1}{N}I_1-\frac{k_2}{N}I_2 & 0 & -\frac{k_1}{N}S & -\frac{k_2}{N}S+(1-p)(D_0-D) & (1-p)(D_0-D) \\ \frac{k_1}{N}I_1+\frac{k_2}{N}I_2+\alpha-\rho R & -D-a-\rho_R & \frac{k_1}{N}S-\rho_R & p(D_o-D)+\frac{k_2}{N}S-\rho_R & p(D_o-D)-\rho_R \\ 0 & a & -D-a_1-\rho_1 & \beta_2 & 0 \\ 0 & 0 & a_1 & -D_0-a_2-\beta_2 & \beta_3 \\ 0 & 0 & 0 & a_2 & -D_0-\beta_3 \end{pmatrix}$$

In the case when $D = D_0$, this matrix simplifies to

$$\begin{pmatrix} -\mu-\alpha-\frac{k_1}{N}I_1-\frac{k_2}{N}I_2 & 0 & -\frac{k_1}{N}S & -\frac{k_2}{N}S & 0 \\ \frac{k_1}{N}I_1+\frac{k_2}{N}I_2+\alpha-\rho_R & -\mu-a-\rho_R & \frac{k_1}{N}S-\rho_R & \frac{k_2}{N}S-\rho_R & -\rho_R \\ 0 & a & -\mu-a_1-\rho_1 & \beta_2 & 0 \\ 0 & 0 & a_1 & -\mu-a_2-\beta_2 & \beta_3 \\ 0 & 0 & 0 & a_2 & -\mu-\beta_3 \end{pmatrix}$$

Corollary

For parameter values given in Table 1, in the case when $D = D_0$, we obtain the following:

- **i.** For the U.S., the equilibrium values are (S^*, E^*, I_1^*, I_2^*, I_3^*) = (2.77%, 13.28%, 26.8%, 31.09%, 9.80%). This equilibrium is locally asymptotically stable.
- **ii.** For the U.K., the equilibrium values are (S^*, E^*, I_1^*, I_2^*, I_3^*) = (4.21%, 19.89%, 25.67%, 39.62%, 9.37%). This equilibrium is locally asymptotically stable.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

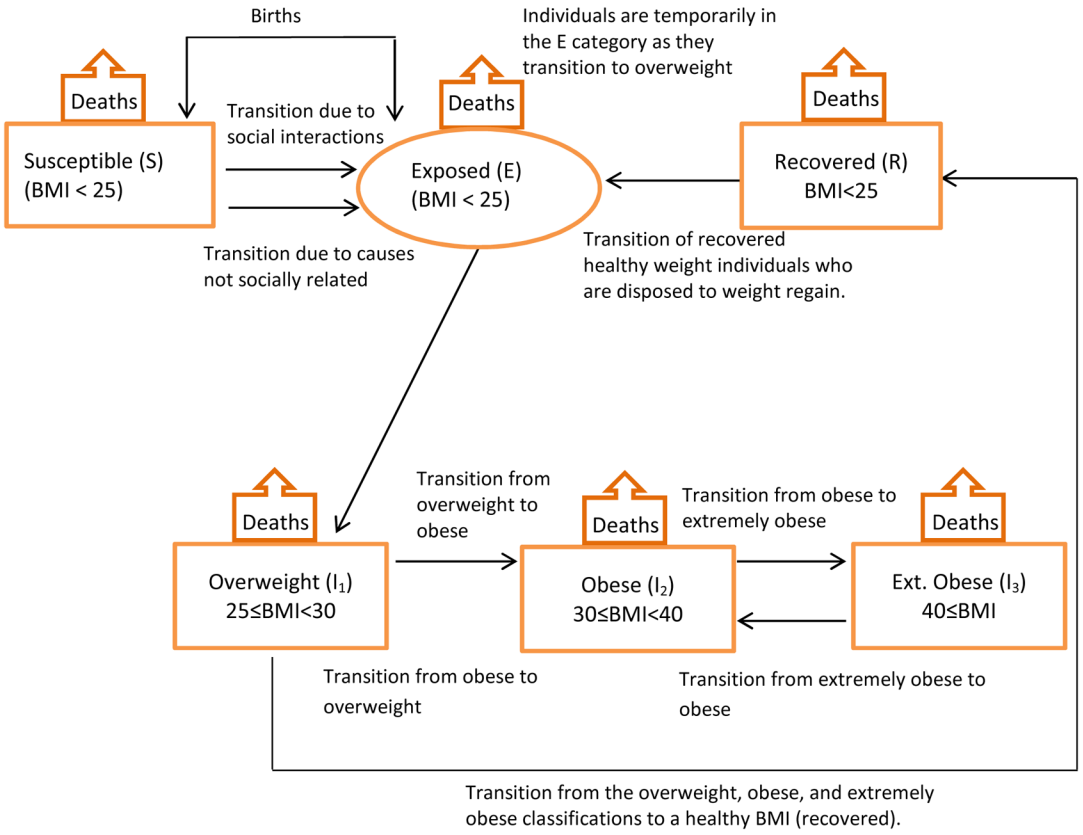

Figure 1.
Diagram describing flow from each compartment formulated in the dynamic model.

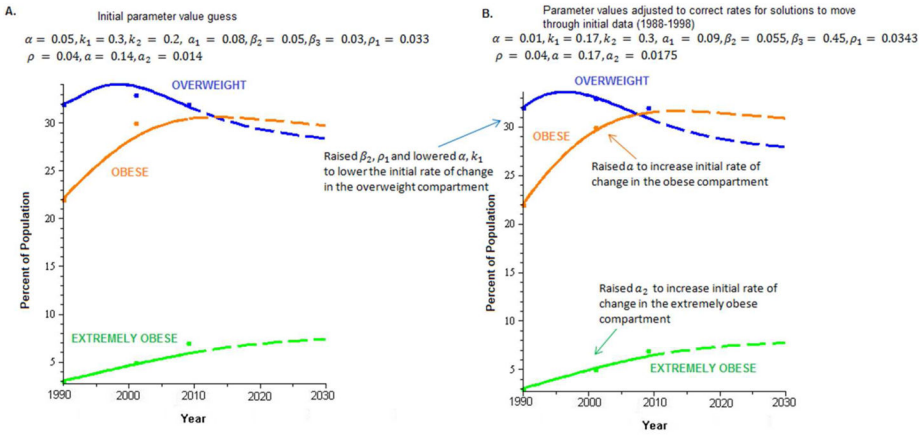

Figure 2.
The method of shooting employed to determine parameter values to force solutions through the initial prevalence data (1988–2000). **Panel A** depicts model solutions for an initial guess of parameter values. This initial guess led to an overestimated rate of increase in the overweight category (blue) and an underestimated rate of increase in the obese and extremely obese category. Parameters that influence these rates were adjusted so that the

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

rate of overweight increase was lowered and the rate of increased prevalence of obese and extremely obese were slightly higher. **Panel B** shows a good fit through prevalence data from 1988–2000. The resulting curves continue to follow prevalence data after 2000 which validates their projections and the parameter estimates.

Table 1

Detailed description of each model term.

Compartment	Assumption	Mathematical Formulation
Susceptible (BMI <25)	Proportion of births entering a nonobesogenic environment BMI < 25.	$\mu N(1-p)$
$S(t)$	A fraction of the population dies (normal death rate)	$-DS$
	A fraction of susceptibles become exposed.	$-\frac{k_1 I_1 S}{N}$
	The rate of transition is dependent on • contact with overweight individuals (25≤BMI<30) • contact with obese individuals (30≤BMI<40) • spontaneous increase in weight, unrelated to social contact	$-\frac{k_2 I_2 S}{N}$ $-\alpha S$
Exposed This compartment models the lengthy time period involved in exposure leading to infection. Individuals in this compartment do not show symptoms and are not infectious.	Proportion of births born into and obesogenic environment.	$p(\mu N)$
	A fraction of the population dies (normal death rate).	$-DE$
	A fraction of the population becomes overweight (25≤BMI<30)	$-\alpha E$
$E(t)$	A fraction of the recovered population becomes susceptible to re-infection.	$\rho_R R$
	A fraction of the susceptibles become exposed. The rate of transition is dependent on: • contact with overweight individuals (25≤BMI<30) • contact with obese individuals (30≤BMI<40) • spontaneous increase in weight, unrelated to social contact.	$\frac{k_1 I_1 S}{N}$ $\frac{k_2 I_2 S}{N}$ αS
Infected (Overweight 25 ≤BMI < 30)	A fraction of the population dies (normal death rate).	$-DI_1$
$I_1(t)$	A fraction of exposed became infected.	αE
	A fraction of infected (overweight) transition to a higher stage of infection (obese).	$-a_1 I_1$
	A constant fraction of overweight individuals recover.	$-\rho_1 I_1$
	A constant fraction of obese transitioned back to the overweight compartment.	$\beta_2 I_2$

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Compartment	Assumption	Mathematical Formulation
Infected (Obese 30 ≤BMI < 40)	A fraction of the population dies (differential death rate).	$-D_0 I_2$
$I_2(t)$	A fraction of overweight individuals became obese.	$a_1 I_1$
	A fraction of infected (obese) transition to a higher stage of infection (extremely obese).	$-a_2 I_2$
	A constant fraction of obese individuals recovered and transitioned back to the overweight compartment.	$-\beta_2 I_2$
	A constant fraction of extremely obese individuals transitioned back to the obese compartment.	$\beta_3 I_3$
Infected (Extremely Obese BMI ≥40)	A fraction of the population dies (differential death rate).	$-D_0 I_3$
$I_3(t)$	A fraction of infected (obese) individuals transitioned to a higher stage of infection (extremely obese).	$a_1 I_1$
	A constant fraction of extremely obese transition back to the obese compartment.	$-\beta_3 I_3$
Recovered (BMI ≤25)	A fraction of the population dies (normal death rate).	$-DR$
$R(t)$	A fraction of infected (overweight) recovered.	$\rho_1 I_1$
	A fraction of the recovered population returns to the exposed class.	$\rho_R R$

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

1. What is already known about this subject?

Future obesity prevalence has been predicted by statistical models and simple dynamic models that predict only the size of the obese population. The simple models do not assume differential death rates or the effect of country specific birth rate and more importantly birth into sub-populations at higher risk for obesity. The models also do not differentiate between individuals who have never been overweight and individuals who have lost weight and are more pre-disposed to weight regain.

2. What this study adds

- The proposed model includes differential death rates, possibility of differentiated births into more susceptible versus less susceptible to obesity, the long time length involved in weight gain, compartmentalization into BMI cutpoints (normal weight, overweight, obese, and extremely obese), and increased susceptibility to weight regain for individuals who have lost weight.
- Based on these inclusions, we demonstrate for the first time that birth rate influences obesity prevalence.
- We predict obesity prevalence trends for both the U.S. and the U.K. using the same model with appropriate parameter changes.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

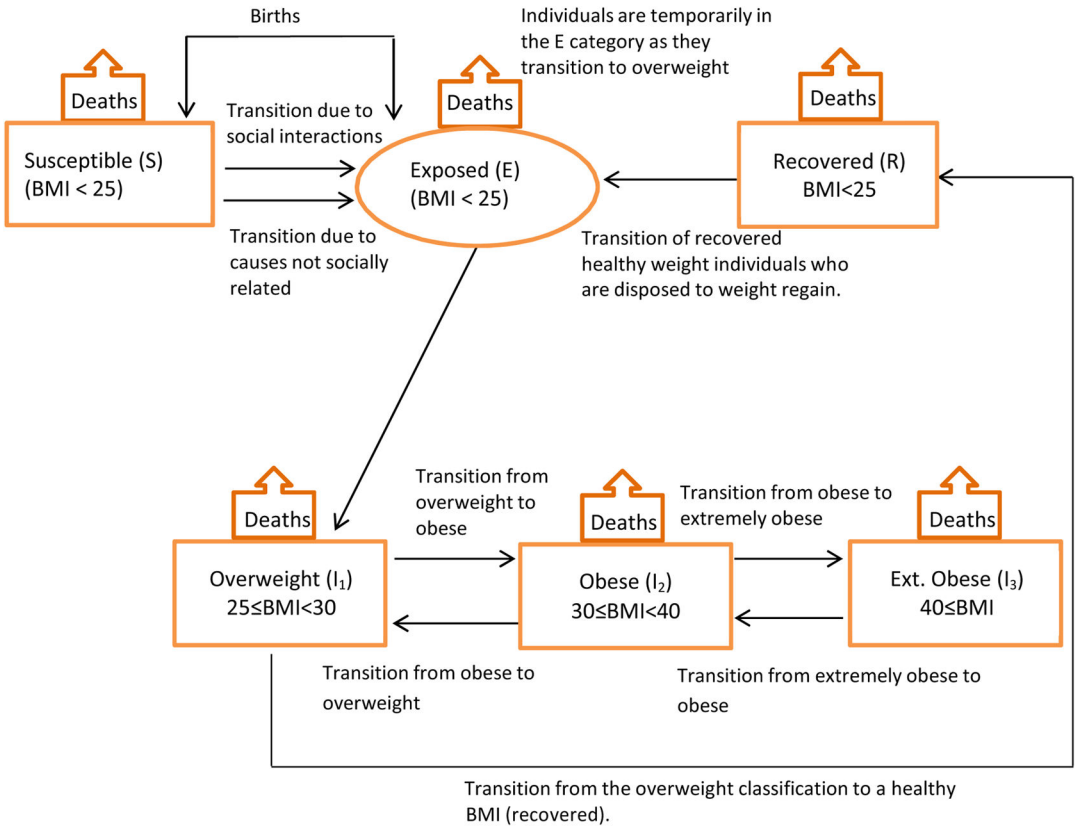

Figure 1.

Diagram describing flow from each compartment formulated in the dynamic model. All compartments include a population wide differential death term.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

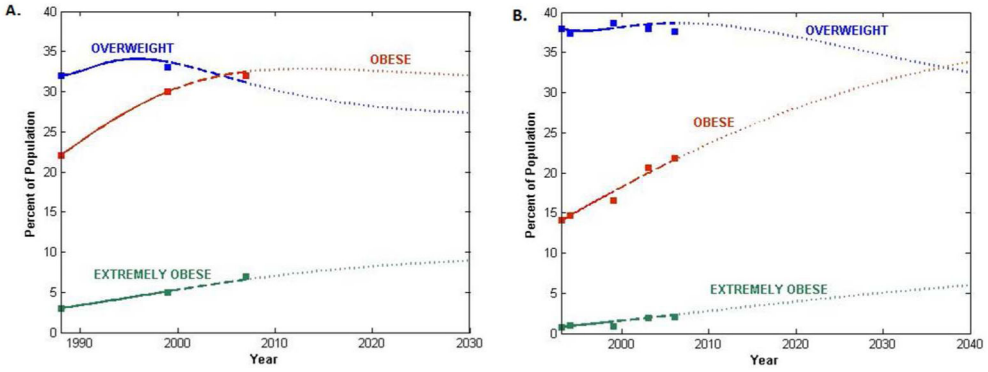

Figure 2.

Comparison of model predictions with actual trends. Parameters and baseline conditions applied in model simulations appear in Table 1. Panel A depicts model predicted trends (solid curves) in overweight, obese, and extremely obese in US adults from years 1988 to 2030. Solid circles depict the Centers for Disease Control reported trends in overweight, obese, and extremely obese in US adults from years 1988–2008 (20). **Panel B** depicts model predicted trends (solid curves) in overweight, obese, and extremely obese in adults in the UK from years 1993 to 2033. Solid circles depict the Health Survey for England reported trends in overweight, obese, and extremely obese in US adults from years 1993–2008. In comparison to the US, parameter values for social influence and recovery rates are almost identical. The spontaneous rate of transition is significantly lower. The portion of the simulations that were fit to data is depicted by solid curves. The dashed curves represents the simulation which did not rely on curve fitting and represents model validation. The dotted portion of the simulations represent the portion of the curve that is a forecast beyond available data.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

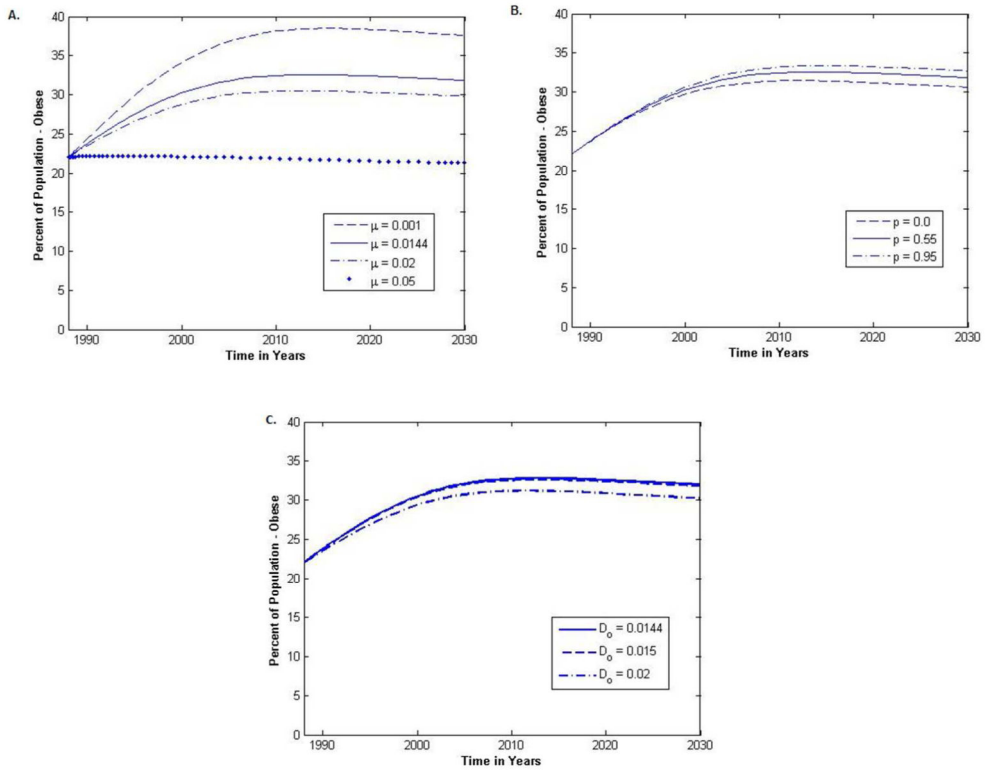

Figure 3.

The dependency of the plateau on birth rate can be observed by varying the birth rate parameter. In **Panel A**, the percent of the obese population was plotted for four birth rates; μ = 0.001, 0.0144, 0.02, and 0.05, which reflect rates of 1, 14.4, 20, and 50 births per 1,000 individuals. The curves show that the percent at which obesity plateaus decreases as a function of increasing birth rate. Similarly, **Panel B** depicts three simulations for different probabilities of being born into obesogenic environment; p = 0.0, p = 0.55, p = 0.95. As p increases, the value at which obesity plateaus increases and the time to plateau increases. **Panel C** depicts three simulations for different obese and extremely obese population death rates D_0 = 0.0144, D_0 = 0.0150, D_0 = 0.02. As D_0 increases the value at which obesity plateaus decreases.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Table 1

List of parameters used in model simulation for the United States obesity prevalence predictions and the United Kingdom obesity prevalence predictions.

United States Simulation		
Parameter		**Method of estimation**
Description	**Value**	
Probability (p) of being born in obesogenic environment.	p=0.55	55% of females of reproductive age are overweight or obese (25).
Birth rate	%=0.0144	Central Intelligence Agency World Factbook (26)
Baseline Prevalence Rates	32% overweight 22% obese 3% strictly obese	1988, CDC prevalence rates (20)
Social influence by overweight and obese	k_1=0.4 k_2=0.2	Fit to initial trends, 1988–1998 using shooting (see Appendix) (20)
Spontaneous rate of weight gain to each class; exposed, overweight, obese, extremely obese	α = 0.05 a=0.14 a_1=0.08 a_2=0.014	Fit to initial trends, 1988–1998 using shooting (see Appendix) (20)
Rate of weight loss to each class; extremely obese to obese, obese to overweight, overweight to normal weight	β_2=0.05 β_3=0.03 ρ_1=0.033	Fit to initial trends, 1988–1998 using shooting (see Appendix) (20)
Rate of weight regainers transitioning from normal weight to overweight	ρ_R=0.04	Fit to initial trends, 1988–1998 using shooting (see Appendix) (20)
Death rate of obese and extremely obese populations	D_0=16.5–22.0	Range reported in (27)
United Kingdom simulation		
Parameter		**Method of estimation**
Description	**Value**	
Probability (p) of being born in obesogenic environment.	p=0.30	30% of females pre-pregnancy BMI are classified overweight or obese (28).
Birth rate	μ=0.01229	Central Intelligence Agency World Factbook (26)
Baseline Prevalence Rates	38% overweight 14 % obese 0.8% extremely obese	1988 Health Survey for England (21)
Social influence by overweight and obese	k_1=0.4 k_2=0.2	Fit to initial trends, 1993–1997 using shooting (see Appendix) (21)
Spontaneous rate of weight gain to each class; exposed, overweight, obese, extremely obese	α = 0.05 a=0.05 a_1=0.025 a_2=0.01	Fit to initial trends, 1993–1997 using shooting (see Appendix) (21)
Rate of weight loss to each class; extremely obese to obese, obese to overweight, overweight to normal weight	β_2=0.001 β_3=0.03 ρ_1=0.003	Fit to initial trends, 1993–1997using shooting (see Appendix) (21)
Rate of weight regainers transitioning from normal weight to overweight	ρ_R=0.05	Fit to initial trends, 1993–1997using shooting (see Appendix) (21)