ESTIMATING ENERGY IMBALANCE GAP EXPLAINING OBESITY PREVALENCE AMONG NEW ZEALAND ADULTS: A SIMULATION APPROACH

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ABSTRACT

Rates of overweight and obesity have increased dramatically around the world. Obesity is the result of complex interaction among various factors namely biological, behavioral, cultural, psychosocial, economic, and environmental factors. In this paper, we use a new system dynamics model that quantifies the energy imbalance gap responsible for the New Zealand adult obesity epidemic. Such result can be used for designing informed sustainable obesity interventions.

INTRODUCTION

Obesity in New Zealand (NZ) has been recognized as a major public health concern in recent years, affecting large numbers of people in every age/gender/ethnic group. Per the 2015/16 national NZ Health Survey, 67% of adults (33% of children aged 2–14 years) in NZ were overweight or obese, 47% of Māori adults (15% of Māori children) and 67% of Pacific adults (30% of Pacific children) were obese. The obesity epidemic has several consequences. It increases the risk for other health problems such as diabetes, heart disease, stroke, and certain types of cancer, some of the leading causes of preventable death. Obesity leads to loss of quality of life and significant cost (in terms of healthcare cost as well as the cost of unhealthy workforce that will burden competitiveness in global economic). Obesity is the result of interplay among various factors namely biological, behavioral, cultural, psychosocial, economic, and environmental factors; these factors operate at multiple levels varying from individual-level health behaviors to family to work, school, community characteristics to local, state, national or international policies [1]. For both public policies and scientific analysis, obesity should be treated as a complex system in which multiple individual and socio-environmental factors affect human behavior. The complexity of the obesity problem, its dynamic nature, and the lack of agreement on the main drivers of obesity have led to an increasing focus on systems approaches to the study of obesity [1-3].

At its core, obesity results from the imbalance of energy intake and energy expenditure in the body. Such imbalance can be quantified by the energy imbalance gap (EIG) and the maintenance energy gap (MEG). The EIG captures the average daily difference between energy intake and expenditure and is the forcing function behind changes in the body mass. The MEG captures the increased energy intake needed to maintain higher average bodyweights compared with an initial (e.g., the late 1980s) distribution of bodyweight. Understanding and quantifying the dynamics of EIG and MEG enable us to explain the magnitude of changes required to reverse the obesity epidemic, provide intervention targets, and estimate the contribution of different drivers of obesity. In this paper we illustrate application of simulation and computational techniques in addressing the obesity problem by presenting a system dynamics simulation model developed by the authors of this paper [4] and applied to the data from national New Zealand Health Survey. In the rest of this paper, the structure of the model as well as the preliminary results from application of the method to national New Zealand Health Survey and corresponding implications will be discussed.

METHODOLOGY

This study was performed in three main steps. In the first step a population level system dynamics model (SDM) which captures BMI distribution and obesity prevalence in adults was developed based on the methodology developed by Fallah-Fini et al [4]. In the second step the energy imbalance gap was modeled. In the third step the SDM was calibrated using the data from New Zealand Health Survey over years 1996 to 2012 to estimate the energy imbalance gap that can explain the prevalence of obesity in adults in the past decade.

Step 1: Developing the Population Level System Dynamics Model of BMI Distribution

Since the prevalence of obesity and overweight varies across different genders and races, we first put the New Zealand adult population into four subpopulations based on their gender (male and female) and race (New Zealand/European and Maori). Consequently the energy imbalance gap will be estimated for each subpopulation.

For each gender/race subpopulation, we used the method developed by Fallah-Fini et al. [4] to efficiently simulate the dynamics of BMI distribution over time based on an established individual-level model of adult body weight dynamics from the literature [5]. The following steps summarize the modeling details that we perform for each gender/race subpopulation:

1. The domain of BMI as the attribute of interest was divided into several distinct ranges (not necessarily equal) represented by distinct stocks. Each stock represents the members of the gender/race subpopulation whose BMI values fall within the BMI range associated with that stock. For example, assume the red curve in Figure 1 shows the distribution of BMI in each gender/race group. The population in each group is disaggregated into $\boldsymbol{M}$ distinct BMI groups. In this study we used 14 BMI groups ([12-15), [15-18), [18-20), [20-23), [23-25), [25-28), [28-30), [30-33), [33-35), [35-38), [38-41), [41-54), [45-50), [50-55)). Let $\boldsymbol{P_k}$ represents frequency of individuals in BMI group *k*, and $\boldsymbol{Xi_k}$ and $\boldsymbol{Xf_k}$ represent the initial and final values of the range of BMI associated with BMI group *k*, respectively. The BMI distribution of each gender/race group was approximated by calculating the height of the vertical bar associated with sub-population group *k* as shown in Equation (1). Those individuals who change their BMI values (e.g., lose or gain weight) will contribute to flows between stocks of population groups (macro population groups).

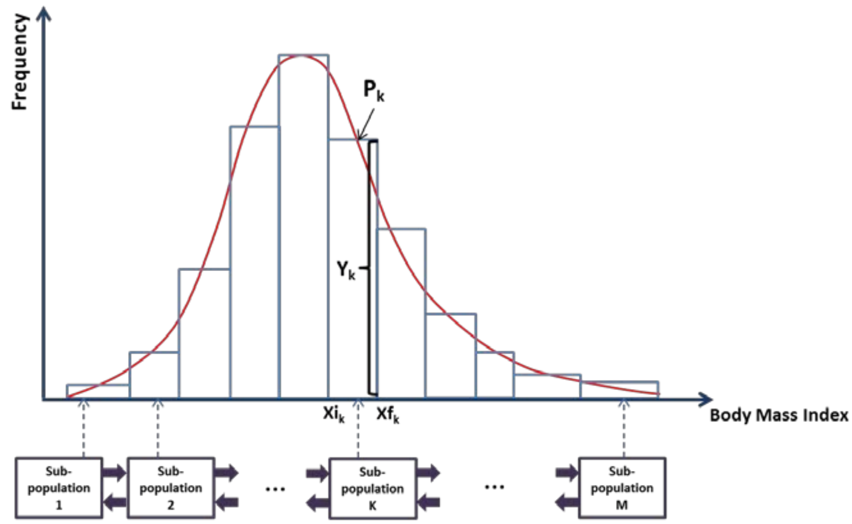

Figure 1: Disaggregation of population in each gender/race group into macro population groups [4]

$$Y_k = \frac{P_k}{Xf_k - Xi_k} \tag{1}$$

2. We assign a representative agent to each BMI group and modeled the weight change dynamics for those representative agents. The representative agent of each BMI group represents average of individuals in that group with respect to their BMI, i.e. an individual with BMI averaging $\boldsymbol{Xi_k}$ and $\boldsymbol{Xf_k}$. Defining representative agents enabled us to connect the individual-level (micro) dynamics of weight gain and loss to the population (macro-level) distribution of BMI. We captured the dynamics of weight gain and loss for representative individuals over time using the Hall's model of adult metabolism and body-weight change [5]. Body weight (BW) in Hall's model is represented by two stocks capturing Fat Mass (FM) and Fat Free Mass (FFM). The change in body weight is then modeled as the result of an imbalance between energy intake (I) and energy expenditure (E) of individuals and is partitioned into/out of FM and FFM.
3. Representative individual's BMI change rate provides the speed by which individuals move from that BMI group to the neighboring BMI groups. Let $\boldsymbol{dBMI_k/dt}$ represent the BMI change rate of representative individual of group $\boldsymbol{k}$ due to an imbalance between energy intake and energy expenditure associated with that representative individual. The rate of population leaving BMI group k is a function of rate of change of the representative individual of group k (i.e., $\boldsymbol{dBMI_k/dt}$) and the height of vertical bar associated with group k (i.e., $\boldsymbol{Y_k}$) and is calculated in Equation (2). Positive values for the rate of change of representative individual of group k imply that some of the population elements in BMI group k will move to BMI group $k+1$. Negative rate of change implies that some of the individuals in BMI group k will move to group $k-1$.

$$\begin{aligned} &Rate\ of\ population\ leaving\ group\ k\ to\ group\ k+1 = Max\ (dAtt_k * Y_k\ , 0) \\ &Rate\ of\ population\ leaving\ group\ k\ to\ group\ k-1 = Max\ (-dAtt_k * Y_k\ , 0) \end{aligned} \tag{2}$$

By calculating the net rate of population change for group k (from/to groups $k+1$ and/or $k-1$ groups) at any simulation time step, we updated the percentage of population in group k in the next time step. New percentages of population in different BMI groups at the start of the next time step provides the new estimate for the distribution of population BMI.

By repeating these steps over time, the dynamics of population BMI over time resulting from the imbalance between energy intake and energy expenditure is captured. To make sure our model is demographically representative of the New Zealand adult population, we also modeled both the rate of transition from childhood into adulthood as well as the deaths.

Step 2: Modeling the Energy Imbalance Gap

The energy imbalance gap associated with representative individual of any BMI class *k* in any gender/race group *j* at any time *t* (represented by $\Delta EI_k^j(t)$) was modeled as a function of the equilibrium energy expenditure $E_k^{j*}(t)$ of the representative individual calculated at time *t* (the energy required for normal activity and maintenance of the body) and an "energy gap multiplier" (represented by $\mu_k^j(t)$) (Equation 3).

$$\Delta EI_k^j(t) = I_k^j(t) - E_k^{j*}(t) = E_k^{j*}(t) * \mu_k^j(t) \tag{3}$$

Energy intake for each representative individual was then calculated by adding the energy gap to the reference energy intake for that individual. A multiplier above zero will lead to BMI growth and one under zero will reduce the BMI for that group as shown in Equation (4).

$$\begin{aligned} &\mu_k^j(\mathrm{t}) = \text{Time effect}^j + \text{BMI effect}_k^j + \text{BMI\&Time Interaction effect}_k^j \\ &\text{where} \quad \text{Time effect}^j = \beta_1 + \beta_2 \text{Time}^j + \beta_3 (\text{Time}^j)^2 + \beta_4 (\text{Time}^j)^3 \\ &\qquad\quad \text{BMI effect}_k^j = \beta_5 BMI_k^j + \beta_6 (BMI_k^j)^{\beta_7} \\ &\qquad\quad \text{BMI\&Time Interaction effect}_k^j = \beta_8 \text{Time}^j BMI_k^j \end{aligned} \tag{4}$$

Step 3: Model Calibration and Parameter Estimation

A good population-level model should be able to closely replicate those distributions observed in the past after taking into account the sampling errors. This basic intuition was used in our study to estimate the parameters of the model. Specifically, the parameters forming the energy gap multiplier $\mu_k^j(t)$ were estimated such that the BMI distributions over years 1996 to 2012 generated by the model for each subpopulation *j* got as close as possible to the subpopulation's BMI distribution per data from national New Zealand health survey. We used a maximum likelihood method for estimating the unknown model parameters. All simulations and optimizations were conducted in Vensim™ [9] software.

RESULTS AND DISCUSSION

We used data form New Zealand Health Survey over years 1996 to 2012 for New males and females, age 20-74. Figure 2 shows the estimated energy imbalance gaps for New Zealand/European males and females across different BMI groups over time.

Our results for both New Zealand/European male and female show no negative energy imbalance gap across different weight groups meaning that New Zealand/European population in the past decade has always eaten more than what they needed. For New Zealand/European male, the magnitude of the gap shows a spike around late 1990s. However, the values start to decrease toward 2003. The drops in magnitudes of EIG are more significant among normal weight and overweight adults as opposed to obese and severely obese population. Moreover, magnitude of energy imbalance gap was higher in overweight and obese New Zealand/European males. Among New Zealand/European females, we see that underweight, normal weight, and over weight people show lower values for the magnitude of EIG across years. The magnitude of EIG among obese and severely obese people drops slightly between 2003 to 2010, but increases afterward. Our results show that obesity continues at different rates across different BMI groups.

Figure 2: Estimated Energy Imbalance Gap (Kcal/day) Across BMI Groups (New Zealand/European Male and Female, age 20-74)

	BMI Class	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012
New Zealand/European Male	[15-18)	17.2	21.3	24.0	25.4	25.7	25.1	23.8	22.1	20.2	18.3	16.6	15.3	14.7	14.9	16.2	18.8	22.9
	[18-20)	13.5	18.2	21.1	22.7	23.0	22.4	21.1	19.3	17.2	15.2	13.3	12.0	11.3	11.6	13.1	15.9	20.4
	[20-23)	10.6	15.7	19.0	20.7	21.1	20.5	19.1	17.2	15.0	12.8	10.9	9.4	8.8	9.1	10.7	13.9	18.9
	[23-25)	8.6	14.1	17.6	19.6	20.1	19.5	18.0	16.0	13.7	11.4	9.3	7.8	7.2	7.6	9.4	12.8	18.2
	[25-28)	7.4	13.3	17.2	19.3	19.9	19.3	17.8	15.7	13.3	10.9	8.7	7.2	6.5	7.0	9.0	12.8	18.6
	[28-30)	7.2	13.6	17.8	20.0	20.7	20.2	18.6	16.4	13.9	11.4	9.2	7.6	7.0	7.6	9.7	13.8	20.0
	[30-33)	8.0	14.8	19.3	21.8	22.6	22.0	20.4	18.2	15.6	13.0	10.7	9.1	8.5	9.2	11.5	15.8	22.5
	[33-35)	9.9	17.1	21.8	24.5	25.4	24.9	23.3	21.0	18.3	15.6	13.3	11.7	11.1	11.9	14.4	19.0	26.1
	[35-38)	12.7	20.4	25.4	28.3	29.3	28.8	27.2	24.8	22.1	19.4	17.0	15.4	14.8	15.7	18.4	23.3	30.8
	[38-40)	16.6	24.7	30.0	33.1	34.2	33.8	32.2	29.8	27.0	24.2	21.8	20.1	19.6	20.6	23.5	28.7	36.6
	[40-46)	25.0	33.7	39.5	42.9	44.2	43.8	42.2	39.8	37.0	34.1	31.7	30.0	29.6	30.8	34.0	39.6	48.2
	≥46	45.8	55.5	62.1	66.0	67.7	67.5	66.0	63.5	60.7	57.8	55.3	53.7	53.5	55.0	58.7	65.1	74.7
New Zealand/European Female	[15-18)	-1.8	2.4	5.4	7.4	8.4	8.8	8.7	8.2	7.6	7.1	6.8	6.9	7.6	9.2	11.7	15.4	20.6
	[18-20)	-0.9	3.6	6.8	8.8	9.9	10.2	10.0	9.4	8.7	8.0	7.6	7.7	8.4	10.0	12.7	16.7	22.2
	[20-23)	0.0	4.8	8.2	10.3	11.4	11.7	11.4	10.6	9.8	9.0	8.5	8.4	9.1	10.8	13.6	17.8	23.7
	[23-25)	1.1	6.2	9.8	12.0	13.0	13.2	12.8	11.9	10.9	10.0	9.3	9.2	9.9	11.6	14.5	18.9	25.2
	[25-28)	2.4	7.8	11.4	13.7	14.7	14.9	14.3	13.3	12.1	11.0	10.2	10.0	10.6	12.3	15.3	20.0	26.5
	[28-30)	3.8	9.4	13.2	15.5	16.5	16.5	15.8	14.6	13.3	12.0	11.0	10.7	11.3	13.0	16.1	21.0	27.8
	[30-33)	5.3	11.1	15.1	17.4	18.4	18.3	17.4	16.1	14.5	13.0	11.9	11.5	11.9	13.6	16.8	21.9	29.0
	[33-35)	7.0	13.0	17.1	19.4	20.3	20.1	19.1	17.5	15.8	14.1	12.8	12.2	12.6	14.3	17.5	22.7	30.1
	[35-38)	8.8	15.0	19.1	21.5	22.3	22.0	20.8	19.0	17.0	15.2	13.7	13.0	13.2	14.9	18.2	23.5	31.2
	[38-40)	10.7	17.1	21.3	23.6	24.4	23.9	22.5	20.5	18.3	16.3	14.6	13.7	13.9	15.4	18.8	24.2	32.1
	[40-46)	14.0	20.7	25.0	27.2	27.8	27.1	25.4	23.1	20.5	18.0	16.0	14.8	14.8	16.3	19.7	25.3	33.5
	≥46	20.5	27.5	31.9	34.0	34.2	33.0	30.7	27.7	24.4	21.2	18.6	16.8	16.4	17.6	20.9	26.7	35.5

Figure 3 shows the average energy gap across different BMI classes weighted by the population in that class for different subpopulations. On average, the magnitude of the energy gap for New Zealand/European male is larger than New Zealand/European female till year 2005. However, after year 2005, the magnitude of EIG among women is larger than men. Both New Zealand/European male and female show an increase in the magnitude of their EIG starting from year 2009.

Figure 3: Estimated Energy Imbalance Gap (Kcal/day) (New Zealand/European Male and Female, age 20-74)

	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012
New Zealand/European Male	8.7	14.5	18.4	20.6	21.3	20.8	19.4	17.4	15.1	12.8	10.7	9.2	8.7	9.3	11.5	15.5	21.8
New Zealand/European Female	2.4	7.7	11.4	13.7	14.8	15.0	14.5	13.6	12.4	11.3	10.5	10.2	10.8	12.5	15.6	20.3	27.1

Our results show the strengths of a multidisciplinary, systems science approach to the study of energy imbalance and suggest several areas for future public health research. As the next step, we can model the magnitude of the estimated trend in energy imbalance gap as a function of the socio-environmental factors over time. Such study enables us to find those socio-environmental factors that have a significant role in prevalence of obesity in New Zealand over the past decades. Another important direction is to assess the impact of public health interventions on subgroups and BMI classes by evaluating the effect of such interventions on the magnitude of the energy imbalance gap of individuals and consequently the shifts in distribution of body mass index (which is translated into change in prevalence of obesity). As a result we can identify those interventions that may have the greatest potential impact on prevalence of obesity.

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