
Abstract
Childhood obesity is a serious public health problem and a major cause of morbidity among children in the United States. At its core, obesity results from the imbalance of energy intake and energy expenditure, i.e., energy imbalance gap (EIG). The EIG captures the average daily difference between energy intake and expenditure. Understanding the dynamics of EIG can help us explain the magnitude of changes required in energy intake and physical activity to reverse the childhood obesity epidemic. In this paper, we use a novel method in system dynamics and quantify the dynamics of EIG over time for US children age 7 and above. Overall, children whose BMI percentile are higher than 85 percent (i.e., overweight, obese, and severely obese children) showed higher estimated EIG compared to underweight or normal weight children. Over time, children across all weight groups showed an increase in their estimated EIG as they aged from 7 to 9 years old and then a drop until age 13 which is followed by a major increase until age 17.

Keywords: Obesity, Energy imbalance gap, System Dynamics, Method of Simulated Moments

1. Introduction
In the past four decades, the prevalence of obesity among American children has increased significantly. Childhood obesity has been a serious public health problem and a major cause of morbidity among children in the US. According to the data from 2016 National Health and Nutrition Examination Survey (NHANES), the obesity prevalence was 18.5%, affecting about 13.7 million children and adolescents aged 2-19 years [1]. Childhood obesity is a major risk factor for health complications such as diabetes, gallbladder disease, and obstructive sleep apnea [2]. Various factors such as biological, psychosocial, cultural, environmental (food, physical and cultural environment), and economic drivers [3] contribute to the prevalence of obesity. The dynamics and mechanisms that influence obesity require a systems approach to analyze the problem and assess interventions [4].
At its core, obesity results from the imbalance of energy intake and energy expenditure in the body [5]. Such imbalance can be quantified by the energy imbalance gap (EIG). The EIG captures the average daily difference between energy intake and expenditure [5]. Understanding the dynamics of EIG can help explain the magnitude of changes required in energy intake and physical activity to reverse the childhood obesity epidemic. It can also help policy makers to define intervention targets and to estimate the contribution of different obesity interventions on future prevalence of childhood obesity. This understanding not only helps in reversing the obesity trend but also in the prioritization and allocation of resources to obesity prevention and control policies. However, except Fallah-Fini et al. [5], most of the studies in the literature have developed the estimates of EIG for the entire population averaged over a long period of time [6, 7]. Moreover, almost all studies in the literature have focused on estimating EIG for adults.

In this paper, we use a novel method in system dynamics [8] and quantify the dynamics of EIG over time for US children age 7 and above across different sex/ethnicity/weight groups. Correct specification of such dynamics across different subpopulations is essential because individuals of different ethnicity/sex/weight group may have different obesity trends and may respond differently to interventions [5].

2. Methods

This study captures the dynamics of childhood obesity among US children over time. The system dynamics model developed in this paper is multi-level in the sense that it builds on the individual-level model of body weight dynamics for children developed by Hall et al. (2013) [9] and it replicates such model to estimate population level trends.

Our modeling framework is accomplished in four main steps. In the first step, we introduce the individual level model of body weight dynamics for children [9] and how such model captures the dynamics of body mass index (BMI) in children over time as they are exposed to some levels of energy intake and physical activity. In the second step, the energy imbalance gap (EIG) is modeled. In the third step, the individual-level model is replicated to develop population level measures associated with the distribution of BMI. Lastly, we use the data from NHANES to calibrate our model and estimate the dynamics of EIG that can explain the changes in the
distributions of BMI among US children across six different subpopulations defined based on their sex (male or female) and ethnicity (Non-Hispanic White, Non-Hispanic Black, Mexican-American). The details of the four steps performed for each subpopulation are below:

**Step 1: Individual-level model of body weight dynamics in children**

We modeled the dynamics of weight gain and loss for children using the Hall et al. (2013)’s model of children metabolism and body-weight change [9]. Body weight (BW) in Hall et al. (2013)’s model is represented by two stocks capturing Fat Mass (FM) and Fat Free Mass (FFM) associated with individuals. The change in body weight (i.e., fat mass and fat free mass) is modeled as the result of an imbalance between energy intake (EI) and energy expenditure (EE). Energy expenditure is composed of several components: 1) the Resting Metabolic Rate (RMR) which is the energy required to perform vital body functions while body is at rest. RMR mainly depends on FM and FFM; 2) the energy needed for physical activity; 3) the energy required for digesting food and nutrients consumed (thermic effect of food); 4) the energy required for developing new mass or digesting existing mass; and 5) the changes in energy expenditure due to perturbation $\Delta I$ away from the reference energy intake associated with normal growth in children.

Equation (1) shows the formula associated with energy expenditure in children adopted from Hall et al. (2013) [9], where $\gamma_L = 22.4$ kcal/(kg.day), $\gamma_F = 4.5$ kcal/(kg.day), $\beta = 0.24$, $\eta_F = 180$ kcal/kg, $\eta_L = 230$ kcal/kg, $\rho_{FM} = 9.4$ kcal/g, and $\rho_{FFM} = 4.3 \times FFM + 837$ kcal/g. $K$ is a constant determined by the initial energy balance condition and takes value of 800 kcal/day for males and 700 kcal/day for females. $\delta$ is a function of time.

$$EE = \frac{K + \gamma_{FFM} FFM + \gamma_F FFM + \delta BW + \beta \Delta I + \left[ \eta_{FFM} \frac{\rho_{FFM}}{\rho_{FM}} p + \eta_F \frac{\rho_{FM}}{\rho_{FM}} (1 - p) \right] EI + g \left[ \frac{\eta_{FFM}}{\rho_{FFM}} - \frac{\eta_F}{\rho_{FM}} \right]}{1 + \frac{\eta_{FFM}}{\rho_{FFM}} p + \frac{\eta_F}{\rho_{FM}} (1 - p)}$$ (1)

$\Delta I$ in Equation (1) captures the perturbation from the reference energy intake ($I_{ref}$) associated with normal growth in children that is calculated as shown in Equation (2), where $EB_{ref}$ is the reference energy imbalance gap during normal growth in males and females, and $FFM_{ref}$ and
$FM_{ref}$ are reference body composition data associated with children who are on trajectory of normal growth.

$$I_{ref} = EB_{ref} + K + \gamma_{FFM}FFM_{ref} + \gamma_{FM}FM_{ref} + \delta BW + \frac{\eta_{FM}}{\hat{\rho}_{FM}}[(1 - p)EB_{ref} - g] + \frac{\eta_{FFM}}{\hat{\rho}_{FFM}}[pEB_{ref} + g]$$  

(2)

At any time step in the simulation model, the imbalance between energy intake and energy expenditure (EI-EE) of individuals is then partitioned into/out of FM and FFM using Forbes’ partitioning equation (3) [10]. The partitioning function $p$ for children is defined as below:

$$p = \frac{c}{c + FM}, \quad C = 10.4 \text{ kg} \times \frac{\hat{\rho}_{FM}}{\rho_{FM}}$$  

(3)

Therefore, the time course of weight loss and gain can be obtained by solving the differential equations related to change in FM and FFM as shown in Equation (4).

$$\rho_{FM} \frac{dFM}{dt} = (1 - p)(EI - EE) - g(t)$$  
$$\hat{\rho}_{FFM} \frac{dFFM}{dt} = p(EI - EE) + g(t)$$  

(4)

where $g(t)$ is a function of time alone and as time increases with progression to adulthood, $g(t)$ approaches zero. The rate of change of FM and FFM are then translated into changes in FM index (FMI) and FFM index (FFMI) using the data of height trajectory of children and finally the rate of change in BMI of individuals is calculated (i.e., $dBMI/dt$).

**Step 2: Modeling the Energy Imbalance Gap (EIG)**

The energy imbalance gap associated with each individual $j$ (represented by $EIG_j(t)$) was modeled as a function of the equilibrium energy expenditure $EEE_j(t)$ of individual $j$ (the energy required for normal activity, maintenance, and normal growth of the body) and an ‘energy gap multiplier’ (represented by $\mu_j(t)$)—see Equation (5).
\[ EIG_j(t) = EI_j(t) - EEE_{j*}(t) = \mu_j(t) * EEE_{j*}(t) \]  

Energy intake for each representative individual \((EI_j(t))\) was then calculated by adding the energy gap to the equilibrium energy expenditure for that individual \((EI_j(t) = EEE_{j*}(t) + EIG_j(t))\). A positive (negative) energy imbalance gap multiplier will lead to an increase (decrease) in BMI for that group on top of the changes in BMI that have already been made due to presence of growth in children.

The energy gap multiplier \(\mu_j(t)\) was defined as a function of BMI of individual \(j\), age of individual \(j\), time, interaction between BMI and age, and interaction between BMI and time, as represented in Equation (6).

\[
\mu_j(t) = \text{Time effect} + \text{BMI effect}_j + \text{Age effect}_j + \text{Age&BMI Interaction Effect}_j + \text{BMI&Time Interaction Effect}_j
\]

where

\[
\text{Time effect} = \beta_1 + \beta_2 Time + \beta_3 (Time)^2 + \beta_4 (Time)^3
\]

\[
\text{BMI effect}_j = \beta_5 BMI_j + \beta_6 (BMI_j)^{\beta_7j}
\]

\[
\text{Age effect}_j = \beta_8 Age_j + \beta_9 (Age_j)^{\beta_{10j}}
\]

\[
\text{BMI&Time Interaction Effect}_j = \beta_{11} Time BMI_j
\]

\[
\text{Age&BMI Interaction Effect}_j = \beta_{12} Age BMI_j
\]

**Step 3: Modeling population**

To develop the population level model, we replicated the individual level model described in Step 1 to develop population level characteristics such as average BMI of the population, standard deviation, and different percentiles. All individuals in our model begin at the same age (i.e., age 7 in this study) but have unique fat mass and fat free mass at the start of the simulation. Given the initial values for fat mass and fat free mass of each individual \(j\) and the trajectory of energy intake of each individual \(j\) (i.e., \(EI_j(t)\)), as estimated in steps 1 and 2, the trajectory of
BMI of each individual $j$ in the simulation model is generated, and consequently the distribution of BMI of the population under analysis is simulated over time.

In this preliminary study, we assume that all children have the same normal level of physical activity over time as specified in the Hall et al. (2013)’s model. Moreover, we assume that initial fat mass and fat free mass as well as energy intake trajectory are the only factors that capture differences between individuals. We do not consider individual differences due to genetics, environmental factors, or how different bodies process nutrients differently. Figure 1 shows a simplified representation of our individual-level model and the replication of N=1000 individuals to develop a population-level model.

![Diagram](image)

**Figure 1:** Simplified stock and flow diagram of body weight dynamics

**Step 4: Model calibration and estimation of Energy Imbalance Gap (EIG)**

and above for six different subpopulations identified by their sex and ethnicities. We used the data of the first wave (i.e., 1999-2000) to obtain the BMI distribution of 7 years old children, which is also the age that simulation starts at. Consequently, we used the data for the next five waves to obtain the BMI distributions at age 9, 11, 13, 15, and 17, respectively. We also chose the initial values for FM and FFM of individuals in the simulation such that the distribution of BMI of individuals in the model is the same as the distribution of BMI for 7 years old children obtained from data of 1999-2000 survey.

We ran the simulation for 10 years and let each individual \(j\) in the model grow and the trajectory of their BMI be generated as they are exposed to energy intake \(EI_j(t)\). Consequently, our model generates the trajectories of BMI distributions from age 7 to 17. We used the Method of Simulated Moments (MSM) [8, 11] to estimate parameters \(\beta_1 - \beta_{12}\) in equation (6) by matching two sets of moments of BMI distributions: simulated moments generated by the model and empirical moments obtained from NHANES. We defined the distributional moments to be mean, standard deviation, and the 5\(^{th}\), 50\(^{th}\), 85\(^{th}\), and 95\(^{th}\) percentiles of the BMI distributions (in total 30 moments; 6 moments per two-year intervals from age 9 to 17). This estimation results in simulating the trajectories of energy imbalance gap for each individual \(j\) (i.e., \(EIG_j(t)\)) and, consequently, the trajectory of energy intake of each individual \(j\) (i.e., \(EI_j(t)\)).

3. Results

The algorithm described in the Methods section was implemented in Vensim software and the estimations and analysis of data were done in MATLAB. Following the procedure described in the calibration section, Table 1 shows the estimated values for the unknown parameters (\(\beta_1\) to \(\beta_{12}\)) and captures the effects and interactions of time, BMI, and age of individuals on the estimated energy imbalance gap for non-Hispanic White male children, as preliminary results. In the next version of this article, we report the results for all subpopulations.
Table 1: Estimated values for parameters associated with energy imbalance gap multiplier

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Estimation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Time Effect</strong></td>
<td></td>
</tr>
<tr>
<td>$\beta_1$</td>
<td>-0.0130</td>
</tr>
<tr>
<td>$\beta_2$</td>
<td>0.0031</td>
</tr>
<tr>
<td>$\beta_3$</td>
<td>-0.0048</td>
</tr>
<tr>
<td>$\beta_4$</td>
<td>0.0005</td>
</tr>
<tr>
<td><strong>BMI Effect</strong></td>
<td></td>
</tr>
<tr>
<td>$\beta_5$</td>
<td>0.0039</td>
</tr>
<tr>
<td>$\beta_6$</td>
<td>-0.0150</td>
</tr>
<tr>
<td>$\beta_7$</td>
<td>0.0195</td>
</tr>
<tr>
<td><strong>Age Effect</strong></td>
<td></td>
</tr>
<tr>
<td>$\beta_8$</td>
<td>-0.0052</td>
</tr>
<tr>
<td>$\beta_9$</td>
<td>-0.0131</td>
</tr>
<tr>
<td>$\beta_{10}$</td>
<td>-0.0045</td>
</tr>
<tr>
<td><strong>Interaction between BMI and Time</strong></td>
<td>$\beta_{11}$</td>
</tr>
<tr>
<td><strong>Interaction between BMI and Age</strong></td>
<td>$\beta_{12}$</td>
</tr>
</tbody>
</table>

Figure 2 shows the estimated EIG (Kcal/day) for male non-Hispanic White children age 7 to 17 for ten different population percentiles (from 5\textsuperscript{th} to 95\textsuperscript{th} percentiles in intervals of 10). For example, a 7 years old non-Hispanic White male whose BMI is in the 85\textsuperscript{th} percentile of the population has eaten about 58 Kcal/day more than what he needed for normal maintenance and growth of his body. Overall, children whose BMI percentile are higher than 85 percent (i.e., overweight, obese, and severely obese children) showed higher estimated EIG compared to underweight (children with BMI percentile less than 5\%) or normal weight (children with BMI percentile between 5\% and 85\%) individuals at each wave.

Over time, children across all weight groups showed an increase in their estimated EIG from 1999-00 to 2001-02 as they aged from 7 to 9 years old and then a drop till year 2005-06 when they got to age 13, followed by an increase till year 2009-10 as they got to age 17. The magnitudes of the EIG at age17 (year 2009-10) proved much higher than other years across all weight groups. Moreover, positive values of EIG across all weight groups at age 17 (year 2009-10) imply prevalence of obesity in the cohort under analysis will increase as children transition to age 18.
To validate our findings, we compared the distributional moments obtained from survey data with the ones obtained from the simulation model across five waves from year 2001-02 to 2009-10 (See Table 2). Overall, our estimated model was able to generate simulated moments that are fairly close to the moments from data. It should be noted that this fit will never be perfect given the limitations and assumptions in modeling as discussed earlier.

Table 2: Comparing distributional moments obtained from NHANES data and simulation model


**Discussion and Limitations**

Our model is the first in the literature that is capable of estimating the energy imbalance gap (average daily difference between energy intake and expenditure) of US children for different weight groups, sex, and ethnicities. No such analysis exists in the public health literature. We adopted the Method of Simulated Moments to replicate the individual-level model of body weight dynamics and develop the population-level distributional moments with respect to their BMI values.

Our preliminary results showed the EIG for non-Hispanic White males age 7 to 17 across six waves from 1999-2000 to 2009-2010. The estimated EIG quantifies the amount of energy (Kcal/day) that children have consumed above (if positive) or below (if negative) of their equilibrium energy expenditure.

Using our estimated model parameters, we can project future trajectories of EIG in children and consequently project future childhood obesity trends. We can also use our estimated model to evaluate the effects of various interventions on future prevalence of obesity as long as we can translate the effects of those interventions to changes in energy intake and/or physical activity of individuals. Finally, we can use the estimated past trajectories of EIG to estimate the contribution of different drivers of obesity.

Our research has several limitations. We have assumed initial body weight and energy intake trajectories as the sole sources of difference among individuals. We do not capture any heterogeneity among individuals in the simulation due to genetics or environment. Moreover, to capture the effect of time, age, and BMI in defining the energy imbalance gap multiplier, we specified a general model that allows very flexible, nonlinear relationships with time, age, and BMI in the model. To enhance this analysis, we can try other functional forms and compare their quality of fit to the NHANES data. Additionally, we can perform required tests to check the robustness of our model to extreme conditions.
Reference:


