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Demographic Obesity Modeling Based on Complex System Dynamics

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Demographic Obesity Modeling Based on Complex System Dynamics Approach

by

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ABSTRACT

The aim of this thesis is development of a systemic obesity model that can track and monitor body weight on individual and population levels.

Considering that obesity is responsible for over 60% of all leading death causes in developed societies and the world-wide spread of that preventable condition, it is critical to understand the mechanisms that may lead to its control and containment. The obesity model presented here was developed using System Dynamics (SD) approach which helps to map complex and dynamic causal relations, containing multiple feedback loops, between key variables and their effects and also considered in the temporal domain.

The model is validated by using the actual data provided by a Windsor Medical Weight Loss Clinic in Windsor, ON, and actual data acquired from online sources. Based on the available data, sensitivity analysis was performed, as well as a variety of scenario analyses.

DEDICATION

I would like to dedicate my thesis with all my heart to my beloved family,

to my mother, for her unconditional support of my studies and giving me a chance to prove and improve myself through all walks my of life, and

to my brother who has never left my side and supported me each step of the way, who is very special.

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LIST OF ACRONYMS

BEE	Basal energy expenditure
BM	Body Mass
BM0	Reference Body Mass
BMss	Steady State Body Mass for constant EI and PAF
BMI	Body Mass Index
BMR	Basal Metabolic Rate
BMR0	Basal Metabolic Rate at BM0
BMss	Steady state body Mass
BW	Body weight
C	Total content of combustible energy in body
cf	Energy stored per kilogram fat
cl	Energy stored per kilogram lean tissue
DES	Discrete Event Simulation
Distr	Distribution
EE	Energy Expenditure
EEc	Energy Expenditure used to convert excess energy into tissue
EI	Energy Intake
EIss	Steady State energy intake for given body mass and FAP
ef	Efficiency in the conversion of energy to fat mass
el	Efficiency in the conversion of energy to lean mass
Fig	Figure
FFM	Fat Free Mass

FM	Fat Mass
fr	Fraction of Fat
GHEX	Government Health Expenditure
kf	Basal metabolic rate per kilogram fat
kl	Basal metabolic rate per kilogram lean tissue
LM	Lean Mass
Max	Maximum value used from upper side of the range
Mean	Average value used for generating the date
Min	Minimum value used from the lower side of the range
NCDs	Non-Communicable Diseases
NPF	Non-Physical Factor
OAREV	Available Own Source Revenue
PA	Physical activity
PAL	Physical Activity Level
PAF	Physical Activity Factor
RMR	Resting Metabolic Rate
RN	Random Normal
SD	System dynamics
TAREV	Total Available Revenue
TEE	total energy Expenditure
Var	Variation
W	Weight
WHO	World Health Organization

CHAPTER 1

INTRODUCTION

1.1. Background & Motivation

In the first decade of the 21st century Canada's population increased almost 5% from 31 million to more than 33 million (see Figure 1-1). At the same time, total personal spending on medical care and healthcare services grew by more than 70% from $29 billion to $38 billion from 2000 to 2004 and then exceeding the $50 billion threshold in 2008 (Statistics Canada, 2010).

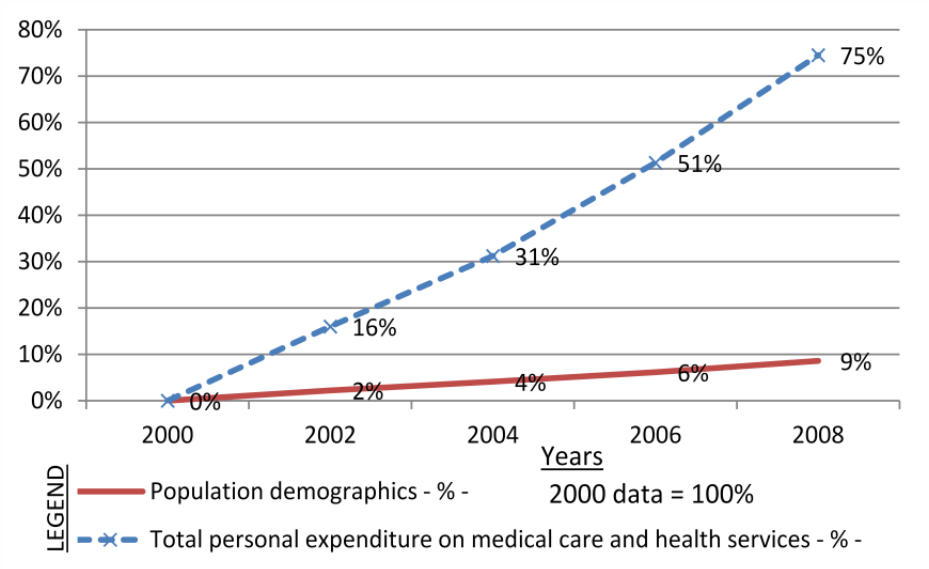

Figure 1-1 – Growth of Canadian population vs. healthcare spending 2000-2008.

If health care costs continue to grow at that rate, healthcare spending will soon overwhelm provincial and federal budgets (Statistics Canada, 2010). Such a predicament is common to many countries, independent of their economic development level. Effective working solutions, however, despite vigorous national discussions, have not yet been found. To prevent this from happening, many governments are implementing hard cost control measures, which effectively use various forms of health care rationing, either by limiting access to the health care system (in the US approximately 17% of population has no health insurance) or selective access to some medical procedures (UK or Canada

have set budget limits for annual health expenditures, which in turn creates waiting lines for elective procedures). The root causes of continuous cost increases in health care are many: demographic trends, expanding longevity and quality of life, new technologies, insurance bureaucracy overhead, etc. While there are many reasons for growing demand for health services, the situation is exacerbated by the corresponding escalating costs; reasonable solutions are unfortunately not yet apparent (Salamati & Pasek, 2013).

The growing demand for health services is also an expression of a global trend in which increasing part of the population is afflicted by various chronic conditions and non-communicable diseases (NCDs), which can be managed and are not necessarily fatal. In fact, according to WHO in 2005 over 60% of deaths worldwide were attributable to NCDs, but by 2020 that number is expected grow by 70%. NCDs are also often referred to as "lifestyle" diseases (or "diseases of the rich") because most of them are caused by smoking, alcohol abuse, poor diets and physical inactivity, etc. and, in principle, are preventable. Two of the leading NCDs, cardiovascular disease and diabetes, have a very strong causal link to obesity (White, 2001).

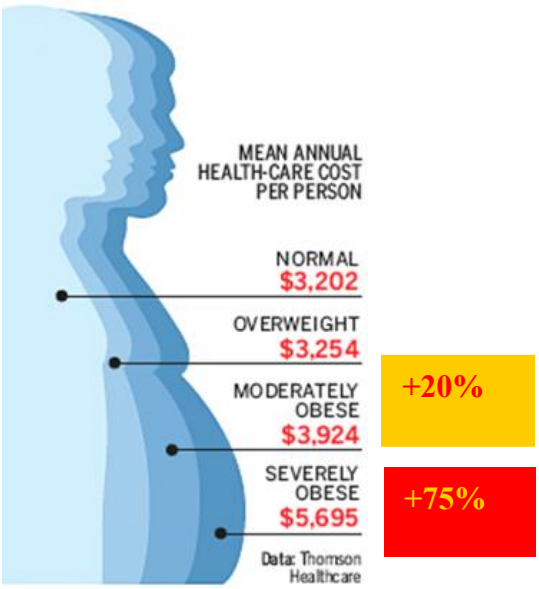

Figure 1-2 – Healthcare costs associated with obesity (BusinessWeek, June 10, 2007)

The spreading prevalence of obesity among workforce impacts employers' health-care costs due to the medical problems associated with obesity (see Figure 1-2). According to

a Thomson Healthcare survey of 54,000 employees, healthcare spending for a severely obese employee can be even 75% higher than that on an employee of normal weight. The occurrence of circulatory and metabolic diseases such as diabetes is three times higher in obese workers, who also exhibit more instances of arthritis, back pain, and are more prone to injuries (Bloomberg Business Week Magazine, June 10, 2007).

Another side effect of overweight/obesity is discrimination against people with weight issues, which is a result of a popular negative stereotype that overweight/obese individuals are inactive, uninspired or lacking strong-mindedness. Such an image causes remarkable injustice in employment, health, health care and education (Canadian Obesity Network).

Figure 1-3 shows predictions of future prevalence of adult obesity in Canada from 2013 to 2019, by weight category. It is estimated that by 2019, 55.4% of the adult population in Canada will be overweight (34.2%) and obese (21.2%). In addition, the prevalence for obese classes I, II and III is expected to increase to 14.8%, 4.4% and 2.0%, respectively (Twells, et al., 2014).

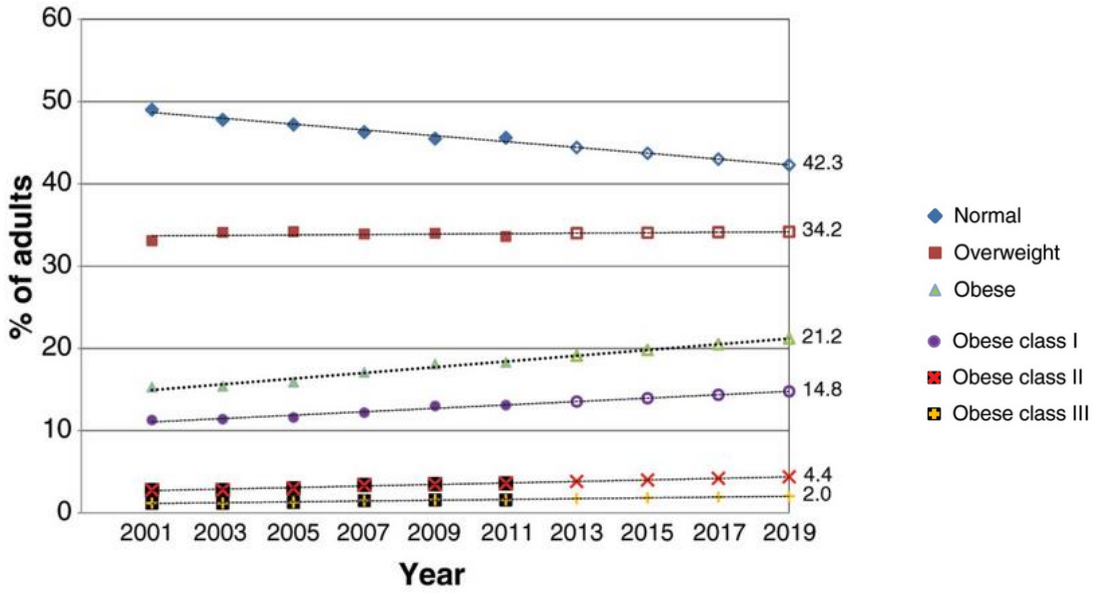

Figure 1-3 - Forecasts of future prevalence of adult obesity in Canada

The World Health Organization (WHO) developed guidelines for evaluating obesity based on (Raine, 2004). In Canada, there are comprehensive guidelines for development

of the practical recommendations for fighting obesity based on (Lau, Douketis, Morrion, Hramiak, & Sharma, 2007). The common way to classify adults is based on calculation of Body Mass Index (BMI) derived from weight and height data (Ko & Tang, 2007), using the following formula:

$$BMI = \frac{weight}{(height)\text{^}2} \left[\frac{kg}{m^2}\right] \quad (1\text{-}1)$$

Table 1-1 - Weight Categories

Category	BMI	Category	BMI
Normal	18.5–24.9	Obese class I	30.0–34.9
Overweight	25.0–29.9	Obese class II	35.0–39.9
Obese	≥ 30.0	Obese class III	≥ 40.0

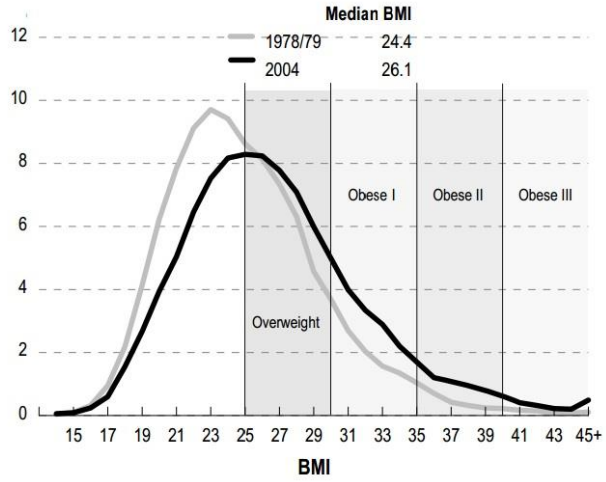

Figure 1-4 - Percentage distribution of household population aged 18 or older, by body mass index (BMI), Canada excluding territories, 1978/79 and 2004 (1978/79 Canada Health Survey; 2004 Canadian Community Health Survey: Nutrition).

It is estimated that direct costs of overweight and obesity stand at $6 billion – 4.1 % of Canada's total current health care budget. However, this estimate only captures health care costs directly related to obesity, and does not represent indirect effects, such as, for example, productivity loss, lost tax revenues or psychosocial price (Anis, et al., 2010).

10-year projection for the three main indicators of Government Health Expenditures (GHEX), Own Source Revenue (OAREV) and Total Available Revenue (TAREV) is shown in Figure 1-5 (Rovere & Skinner, 2010). All show alarming trends. These projections demonstrate that if Ontario does not significantly restructure the way it finances healthcare spending, then these expenditures will devour 75% and 100% of the Province's own-source revenue by 2019 and 2030, respectively. Thus it is imperative that agencies responsible for population's health (e.g., government, health care systems, etc.) introduce right health policies, initiatives and take appropriate actions.

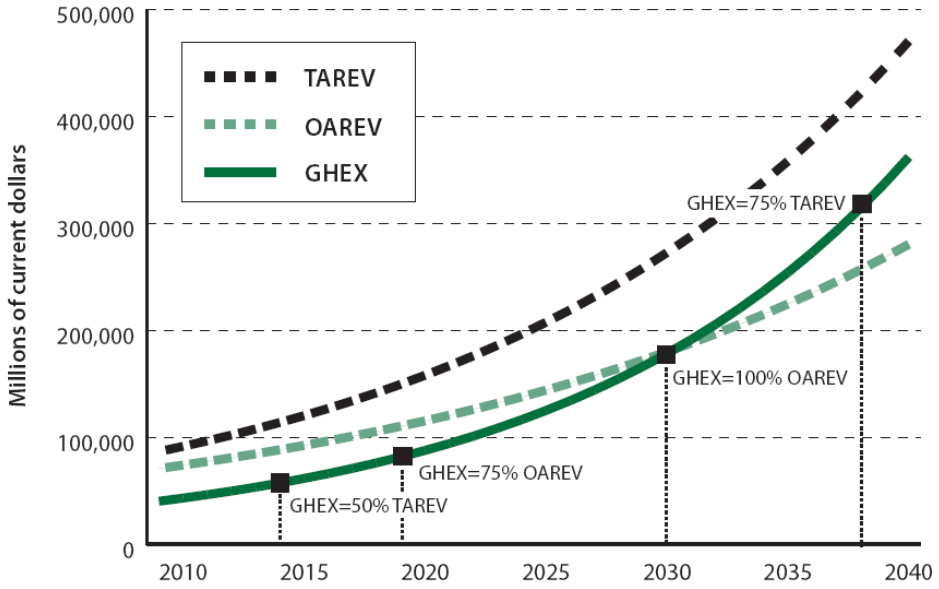

Figure1-5 - Future forecasts for the three main indicators of government health expenditures (Rover & Skinner, 2010)

Obesity is commonly recognized as a condition leading to many other medical conditions. It is responsible (directly or indirectly) for over 60% of all leading death causes in developed societies, not to mention significant contribution to the health care costs (Bhattacharya, 2011).

Table 1-2 shows the impact of preventable diseases in relation to the population mortality rates (Danaei, et al., 2009) and gives all-cause deaths (thousands) attributable to risk factors and the 95% confidence intervals for their sampling uncertainty in Canada.

As can be readily noted, tobacco smoking and high blood pressure are two leading death causes in Canada among both men and women. Overweight – obesity and physical inactivity, together, are the second most frequent death causes after tobacco smoking. By considering indirect impact of obesity (e.g., high blood pressure, high glucose, dietary trans-fatty acids, etc.) the impact can be even greater. Furthermore, overweight and obesity increase the risk of many preventable diseases such as cardiovascular disease, hypertension, type-2 diabetes, arthritis and some types of cancer (World Health Organization, 2003; Wannamethee, 2005).

Table 1-2 – Death causes (thousands) attributable to risk factors in Canada

Risk Factor	**Male**	**Female**	**Both Sexes**
Tobacco smoking	248 (226-269)	219 (169-244)	467 (436-500)
High blood pressure	164 (153-175)	231 (213-249)	395 (372-414)
Overweight-obesity (high BMI)	114 (95-128)	102 (80-119)	216 (188-237)
Physical inactivity	88 (72-105)	103 (80-128)	191 (164-222)
High blood glucose	102 (80-122)	89 (69-108)	190 (163-217)
High LDL cholesterol	60 (42-70)	53 (44-59)	113 (94-124)
High dietary salt (sodium)	49 (46-51)	54 (50-57)	102 (97-107)
Low dietary omega-3 fatty acids (sea food)	45 (37-52)	39 (31-47)	84 (72-96)
High dietary trans fatty acids	46 (33-58)	35 (23-46)	82 (63-97)
Alcohol use	45 (32-49)	20 (17-22)	64 (51-69)
Low intake of fruits and vegetables	33 (23-45)	24 (15-36)	58 (44-74)
Low dietary polyunsaturated fatty acids (PUFA)(in replacement of SFA)	9 (6-12)	6 (3-9)	15 (11-20)

Even though obesity has been well studied from the medical outlook of an individual, there is still a need for understanding the causes and effects from systemic point of view. The system in this case pertains to both individual (human body) level and population level.

The focus of the work presented in this thesis is on development and testing of an obesity system model representing an individual, which is also explored on a range of conditions of a population.

1.1.2. Understanding Obesity

While a multitude obesity related research results is being published every year, these efforts are generally not coordinated and focused, and they typically lead to only fractional models addressing some narrow aspect of the phenomena. Even though the global spread of obesity is being acknowledged, no successful efforts to fight it have been developed. As (Lustig, 2006) pointedly described it: "The Centers for Disease Control and Prevention says obesity results from an energy imbalance, by eating too many calories and not getting enough physical activity. Big Food says it's a lack of activity, the TV industry says it's the diet. The Atkins people say it's too much carbohydrate, the Ornish people say it's too much fat. The juice people say it's the soda, the soda people say it's the juice. The schools say it's the parents, the parents say it's the schools. How are we going to fix this, when no one will accept responsibility?"

One of the key reasons for that is limited understanding of those phenomena. Most of the contemporary obesity mechanism models are based either on glucostatic theory (Mayer 1953) or lipostatic theory (Kennedy 1953). Based on either of those theories the origin of obesity is perceived as a disorder in one of the two feedback systems (e.g., signaling equilibrium of either fat deposition or glucose blood levels). In practice, however, neither of those two approaches could satisfactorily explain the obesity phenomenon (Salamati & Pasek, 2013).

The basic logic for body weight equilibrium can be explained as follows: if the energy intake (EI) from different sources of food (both solid and liquid) exceeds the total energy expenditure (TEE) due to resting metabolic rate (RMR), energy used to digest food, and physical activity (PA), then the individual will gain weight, otherwise individual loses weight or maintains the current weight if EI = TEE.

Several factors complicate dynamic modeling of weight change. First, RMR is a function of the body weight, body composition (fraction of fat mass (FM) and fat free mass

(FFM)), gender, race, and age (Harris & Benedict, 1919; Cunningham, 1980; Bernstein, Thornton, et al., 1983; Schofield, 1985; Cunningham 1991; Bitar, Fellmann, et al., 1999; Frankenfield, Roth-Yousey, et al., 2005). Variation in EI affects RMR as well the adaptive thermogenesis process (Rosenbaum, Leibel et al., 1997; Jequier & Tappy, 1999; Rosenbaum, Hirsch, et al., 2008). Moreover, obesity is traditionally defined by body mass index (BMI), which considered by many researchers not the most reliable indicator..

(Poehlman, 1992) found that "total daily energy expenditure and its components decline with advancing age. However, (Speakman & Westerterp, 2010) discovered that in the first half of life it increases with age, but in the second part of life (>57.8 for men and >39.8 for women), RMR is decreasing with age. However, in many statistical studies, age is treated as an independent variable in explaining RMR (Harris and Benedict 1919; Cunningham 1980; Cunningham 1991; Vaughan, Zurlo et al. 1991; Poehlman 1992; Maffeis, Schutz et al. 1993; Tershakovec, Kuppler et al. 2002; Speakman & Westerterp, 2010). The model distinguishes individuals based on age, gender, height, initial weight and takes physical activity and energy intake as inputs and provides the dynamics of body weight as outputs.

In considering weight, food (solid and liquid) consumption is the most important source of daily energy intake by an individual. Whether individuals' energy intake is provided by various food categories, such as carbohydrates, fats, or proteins, the impact on dynamics of weight is not significantly different as long as the number of caloric intake stays the same. Thus, the main emphasis in this study is on the total EI and there are no differences between different nutrients are considered to have significant effects (Rahmandad & Sabounchi, 2011).

Factors associated with energy expenditure are more varied. These factors include either the resting metabolic rate or basal metabolic rate (RMR: the energy needed to perform essential body tasks while body is at rest) which account for 50-75% of energy expenditure, the energy used for physical activity, and the energy used to digest consumed food and nutrients and creation of new tissue. RMR is dependent on the body composition (FM and FFM have different levels of energy to be maintained) as well as

differences between individuals such as age, and gender. Energy spending associated with physical activity (PA) is largely proportional to the total weight (BW~FM+FFM) and the PA severity (Rahmandad & Sabounchi, 2011).

1.2. Problem Statement

The aim of this research is to develop a dynamic model of individual/population weight change over time which eventually will lead to development of useful rules for body weight management and control (both on individual and population levels). The model can be used at the single individual level and be very useful for tracking and management of the body weight as a function of individual physical and environmental attributes, energy intake, and physical activity levels. Taking into account the same categories and applying them to a population (e.g., by using proper distributions of parameters and variables), model can also provide insights into similar population-wide responses. The population level analysis is of particular importance to institutions responsible for public health (e.g., governments, health care systems, etc.) and their long-term efforts to maintain both healthy populations and control health-related expenditures at the same time.

The model, based on available medical literature and data is intended as a decision support tool assisting decision makers in exploring long-term effects of the policies they may consider developing and implementing. Policies are defined as principles or protocols to guide decisions leading to achieve rational outcomes. In terms of population health advocacy for "healthy" public policies is not enough, it also has to be supported by corresponding preventive measures (in particular when dealing with response to diseases), meaning that stewards of public health must develop and explore variety of options for disease control and prevention. It is also worth noting that obesity was recognized as a disease by American Medical Association (AMA) only last year (2013). On the other hand, obesity is still not well understood, and as a result models to represent how it affects individuals and populations are still lacking.

1.3. Proposed Approach

The target of this research is to study the time-based dynamics of obesity to create a reliable system dynamics model that can be used for obesity policy analysis at population level. The model has two-levels. The presented model is based on individual level energy models which allow to explore the weight change over the individuals' life, and further extends individual level model to the population level. The model captures individual characteristics, such as, gender, height, initial weight, and age and uses physical activity and energy intake as inputs to provide the time-varying body weight as output. In this study the body weight dynamics on individual level model will be introduced first and then the population level model, consisting of multiple replications of individual models and their relationships will be discussed (Rahmandad & Sabounchi, 2011).

According to (Collins, Khoury, Morton, & other, 2008) the most common forms of obesity are dependent on a number of factors or causes such as genes and environment, including diet and physical activity patterns. Genetics plays an indispensable part in energy homeostasis and influences energy intake and spending, and further partitioning of calories, which include tendency to store calories ingested when they exceed the actual energy expenditure (Chung, 2008). Another effect of genetic variations is visible in eating behavior, taste, and fullness (Rankinen, Bouchard, 2006, Wardle, Carnell, Haworth, et al., 2008). Furthermore, "obesogenic" environment provides plenty of opportunities to increase energy intake (e.g., increased availability and access to fast food outlets) and reduce availability of physical activity (e.g., fewer physical activity opportunities due to lack of sidewalks, walking trails, bike lanes, or parks).

The system proposed in this thesis accounts for the differences among individuals. This means that not only the physical but also the non-physical factors (both endogenous and exogenous) differences need to be considered (Collins, Khoury, Morton, & Olster, 2008).

Development of the model is based on the assumption that each individual is an independent entity with different characteristics of age, gender, height, weight, food consumption and physical activity patterns. It is impossible to find even two individuals having identical characteristics (Collins, Khoury, Morton, & Olster, 2008).

1.3.1. System Dynamics

System Dynamics (SD) is a perspective and set of conceptual tools to understanding how complex systems behave over time (Sterman, 2000). This modeling approach is often used to simulate complex systems for policy analysis and design (Michael, Radzicki, Robert, & Taylor, 2008). The main difference between System Dynamics and other approaches is dealing with internal feedback loops, stocks, and flows. These elements shows that even simple systems may behave in a complex nonlinearity way" (System Dynamics Society, 2010).

1.3.2. Some Main Features of Systems Dynamics

- Model the problem, issue, or evaluation questions, not the whole program or real world
- Assume most problems have internal causes
- Assume events follow patterns, it means that they are generated by structures
- Choosing of the problem boundary is an essential step
- Extent in time and space is generally more important than detail
- Ability of applying insights from other models and simulations (e.g. system archetypes, or the behavior of epidemics) (Harris, Williams, 2005)

1.3.3. SD's Essential Differentiators

- The model and the real world are associated together
- The effect of information feedback is the focus
- Models includes both quantitative and qualitative factors
- Simulated model is used to test hypotheses (Harris, Williams, 2005)

(Meadows, 2008) defined three main factors differentiate it from other modeling methods:

- SD explains why a system changes over a period of time, as opposed to why a system is in a particular state at any point in time
- SD takes a broad view of the elements that cause changes, as opposed to a more detailed microscopic view

- SD demonstrate reciprocal feedback relationships between variables, instead of simple one-way causality**,** similar to most statistical methods

1.3.4. Feedback Thinking

A complicated system is conceptualized by internal feedback loops and circular causality which are the core of System Dynamics approach (System Dynamics Society, 2011).

1.3.5. Loop Dominance and Nonlinearity

Although the loop concept underlies feedback and circular causality, it is not enough by itself. The descriptive power and perceptiveness of feedback understandings also depend on the concepts of active structure and loop dominance (System Dynamics Society, 2011).

1.3.6. The Endogenous Point of View

The core for the system dynamics approach is the concept of endogenous change. It sets down features of model establishment: exogenous interventions are seen at most as activators of system behavior; the causes are comprised within the formation of the system itself (System Dynamics Society, 2011).

1.3.7. System Structure

The structure of SD models includes flow (rate) variables, stock (level) variables, and auxiliary variables. Flow variables are the components that determine the variation of stocks (e.g., development of body mass). Stock variables are the accumulations within the system (e.g., body mass). Auxiliary variables are the remaining elements in the model which represent steps to determine flow variables using stock variables (e.g., basal metabolic rate).Causal Diagram is the basic SD objective to understand the structural causes that trigger system performance (Campuzano and Mula, 2011). In SD methodology, causal loop diagram is applied to represent the system. It includes the key factors of the system and the relationships among them based on the causes which have influence on the effects. Causal loop diagrams serve two main purposes. First, they can be applied as conceptual sketches of causal hypothesis during model development and second, they can make a simpler representation of a model. A causal loop diagram

describes the major feedback mechanisms which can be either negative or positive. Negative loops play a role of stabilizing elements that lead the model towards a balanced situation. Positive loops make the system unstable that is, an initial disturbance in the system leads to further change and an instability. The systems usually contain both loop types and the final performance depends on which one is dominant. The relationships among the variables in causal loop diagram are represented by arrows which come with a + or – sign. The + sign means a positive change in the origin variable of the arrow will produce a positive change in the destination variable. The – sign represents that a positive change in the origin variable will result in a negative change in the destination variable (Sterman, 2000). A closed chain of relationships is called a loop, or a feedback loop. When we turn on the tap to fill a glass with water, the amount of water in the glass increases. The amount of water in the glass, however, also has an effect on the speed at which it is filled. We fill it more slowly when it is fuller. Therefore, a loop exists. Delay is the element which simulates the time taken to convey inflows or information to outflows (Sterman, 2000).

1.3.8. Behavior as a Consequence of Structure

The system dynamics approach focuses attention on a continuous view. The continuous view attempts to look beyond events to determine the elemental dynamic patterns of them. Furthermore, the continuous view not only focuses on discrete decisions but also on the policy structure underlying decisions. Events and decisions are considered as surface phenomena that ride on a fundamental tide of system structure and behavior (System Dynamics Society, 2011).

1.3.9. Levels and Rates

(Homer & Hirsch, 2006) provided strong declaration that the system dynamics is suitable to address the dynamic complication characterizing numerous public health issues. Individuals who use system dynamics of chronic disease prevention modeling should look for incorporating all the fundamental components of a modern ecological method, comprising disease outcomes, health and risk behaviors, environmental factors, and health related resources and delivery systems.

Based on (Homer & Hirsch, 2006), obesity is one of the direct applications of System Dynamics. The SD is an approach that addresses dynamically complex health issues. It has already caused powerful contributions in addressing epidemiological issues, as well as issues of healthcare capacity, delivery, and patient flow management.

According to (Brailsford, 2008), SD advances in healthcare modeling due to its use generally at a higher, more aggregated and strategic level than Discrete Event Simulation (DES). SD models can be greatly complicated but they display dynamic complexity of responses rather than detailed complication. The author explains that there are a lot of reasons why SD fits well in modeling healthcare issues, which may justify the increase in new applications using the SD approach. In a healthcare setting it is not frequent for a stakeholder to draw well defined limits around the system, and disregard any interactions with the environment. Since often several healthcare problems are compounded, and many stakeholders with conflicting objectives interact simultaneously, the qualitative aspects of SD are very helpful to understand such matters (Brailsford, 2008).

Availability of data, their quality, and integrity is another issue. Any DES needs an essential and fundamental amount of data for developing the model. Since SD models are usually higher level and more aggregated, the data requirements are less stringent. One crucial advantage of SD is that the models usually process data quickly and do not need numerous repetitions, so they can be operated in real time with decision-makers (Brailsford, 2008).

CHAPTER 2

LITERATURE REVIEW

2.1. Body Weight Modeling

Individuals have an important role in their own weight management while overweight and obesity are considered as problems of the individual. Having a healthy lifestyle requires a long-term weight management approach. Healthy lifestyle is a balance of healthy eating and physical activities to balance energy expenditure and energy intake. To manage the weight is important to develop healthy eating habits that keep us fuller longer. Another key factor related to body weight management is to know what your body needs in order to control overconsumption and under consumption of food (envirocancer, 2010).

According to (Christiansen, Garby, & Sorensen, 2004), obesity develops over time and it is due to the imbalance of energy intake and energy expenditure. It is difficult to measure or control this energy imbalance over short point of time. A mathematical model can effectively explain the relationship between the change in weight, and the amounts of the energy intake and the energy expenditure, controlled by the physical activity factor PAF.

From evolutionary stand point, human survival always depended on physical activity, whether to hunt for food or run away from natural dangers; Food was always scarce. Over time, however, people have developed for themselves a living environment with abundant of food that requires minimal physical activity. The modern living lifestyle has lower energy demands, promotes comfort and well-being; however, this sedentary lifestyle is not well suited for humans. Physical inactivity has become prevalent. Regular physical activity enables self-adjustments within muscular skeletal and the cardiorespiratory system which have positive impact on the prevention and treatment of many metabolic disorders (Pedersen, 2006; Hawley, 2007). Physical inactivity is associated with many health risks and should rather be perceived as abnormal.

Physical activity can be viewed as a strategy to burn calories. Physical activity is an incentive that should be correctly managed to contribute a significant improvement in

energy balance regulation. In other words, having an active lifestyle has a greater impact on energy balance and body fat than is normally recognized by health professionals (Tremblay & Therrien, 2006).

Physical Activity level (PAL) provides information about severity and duration of a set of different activities performed during a 24-hour period (Gerrior, Juan, & Basiotis, 2006). The equation for measuring the PAL is provided below:

$$\Delta PAL = \frac{(METs-1)\left(\frac{\left(\frac{1.15}{0.9}\right)\times Duration[min]}{1440}\right)}{\left(\frac{BEE}{0.0175}\right)\times 1440\times weight[kg]} \quad (2\text{-}1)$$

where the Metabolic Equivalent of Task (MET), is a numerical value which stand for the resting metabolic rate for a particular activity and BEE is basal energy expenditure. After calculating ΔPAL for each physical activity, the physical activity category (PAL: sedentary, low active, or very active) is determined based on the basal activity impact on energy expenditure (a factor of 1.1) and the sum of all activities (sum of ΔPAL). The PAL can be calculated as:

$$PAL = 1.1 + \sum_{i=0}^{n} \Delta PAL_i \quad (2\text{-}2)$$

where ΔPALi is the list of each activity impact on energy expenditure, n – number of activities.

Physical activities are categorized as follows (Gerrior, Juan, & Basiotis, 2006):

Table 2-1- PA categories for men

Category	**PA**	**PAL range**
Sedentary	1.0	1.0 < PAL < 1.4
Low active	1.2	1.4 < PAL < 1.6
Active	1.27	1.6 < PAL < 1.9
Very active	1.54	1.9 < PAL < 2.5

Table 2-2 – PA categories for women

Category	**PA**	**PAL range**
Sedentary	1.0	1.0 < PAL < 1.4
Low active	1.14	1.4 < PAL < 1.6
Active	1.27	1.6 < PAL < 1.9
Very active	1.45	1.9 < PAL < 2.5

There is another category based on (McArdle, et al., 1996):

Table 2-3- Physical activity categories

Activity Factor	**Category**	**Definition**
1.2	Sedentary	Little or no exercise and desk job
1.375	Lightly Active	Light exercise or sports 1-3 days a week
1.55	Moderately Active	Moderate exercise or sports 3-5 days a week
1.725	Very Active	Hard exercise or sports 6-7 days a week4
1.9	Extremely Active	Hard daily exercise or sports and physical job

Sedentary time is the time that we are neither active nor sleeping. Sedentary activities are activities with little or no strong physical activities such as, for example, sitting, reading, watching television, playing video games, and computer use for much of the day. A sedentary lifestyle can lead to many preventable causes of death (Mark, 2008; Elsevier, 2013; Olds, Ridley, & Dollman, 2006).

2.2. Obesity System Modeling

A sequential approach is required for development of the Obesity System Model (OSM). The system design methodology is based on building blocks feeding each other. According to (Box & Draper, 1987), “Essentially, all models are wrong, but some are useful”, the OSM simulation will start by the basic parts of the system (e.g., behaviors related to personal characteristics, physical activity and dieting etc.). In the future, the

model can be extended to consider Education, Physical Environment and Non-physical activities.

A number of equations are used in the model and the causal relationships between variables can be recognized.

The causal relationships are considered as potential factors for being used in the model development. The causal relationships are clustered into different categories such as Body Mass, Physical Activity Level (PAL), Basal Metabolic Rate (BMR), Body Fat, Energy Expenditure Requirements (EER) and Psychology.

Most of the relationships are complex, including multiple factors and variables which may be difficult. Identifying a unified way of measuring the variables or transforming them to maintain unit consistency throughout the model is another challenge.

2.3. Key Modeling Causal Relationships

In this research the aim is to develop a mathematical model to define the relationship between the change in body weight, amounts of energy intake (EI) and the energy expenditure (EE), controlled by physical activity factor (PAF) (Christiansen, Garby, & Sorensen, 2004). A general view of the energy balance is presented in Figure 2-1.

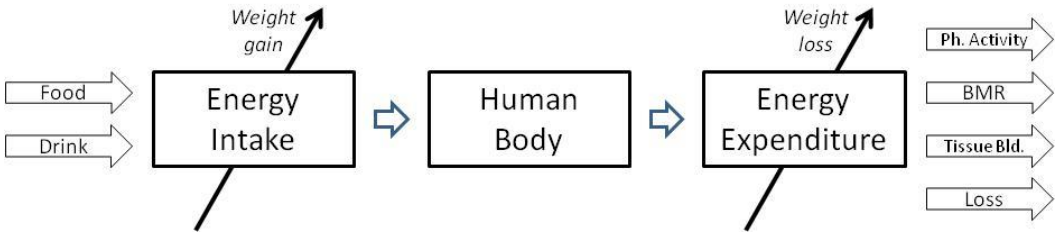

Figure 2-1- Human body energy balance principle

As it can be seen, the two major elements associated with the energy intake are food and drink consumption. On the other side, the two main factors associated with the energy expenditure are physical activity and basal metabolic rate. Additionally, transformation of energy consumed as food in to either fat or lean tissue. It is not as impactful as the other two elements, but it can account for 20% of overall contribution (Christiansen, Garby, & Sorensen, 2004)

Figure 2-1 shows that energy balance is achieved when the energy intake is equal to the energy expenditure. On the other hand, when the energy intake exceeds energy expenditure, the individual may gain weight and when energy expenditure exceeds energy intake, the individual may lose weight.

The energy expenditure necessary to transform matter into fat or lean tissue and the energy expenditure of a larger mass increase with the body mass increase. This kind of energy spending depends on the fraction of fat in added tissue. The fraction of fat in additional tissue and energy required to convert energy into tissue are included in the model (Christiansen, Garby, & Sorensen, 2004).

The ratio between change in EI/PAF and change in Steady State Body Mass (BMss) depends only on fr, kf and kl. The steady-state equation (2-3) is described by (Christiansen, Garby, & Sorensen, 2004):

$$\frac{EI}{PAF} = BMR\,(BMss)BMR0 + \left(Kf \times fr + kl\,(1 - fr)\right) \times (BMss - BM0) \tag{2-3}$$

Steady-state body mass is only dependent on BMR0 and the ratio of EI/PAF:

$$\frac{\Delta BM}{\Delta \frac{EI}{PAF}} = \frac{1}{kf \times fr + kl\,(1 - fr)} \tag{2-4}$$

The general equation for the development of body mass over time is (Christiansen, Garby, & Sorensen, 2004):

$$\frac{dBM}{dt} = \frac{EI - PAF\,(BMR0 + (kf \times fr + kl(1 - fr))(BM - BM0)}{\frac{cf \times fr}{ef} + cf(1 - fr)/el} \tag{2-5}$$

The efficiency in conversion of excess energy into new tissue does not affect steady state, but has a direct proportional influence on the rate of weight increase during periods of positive energy imbalance (Christiansen, Garby, & Sorensen, 2004).

The energy stored per kilogram fat (cf) and lean tissue (cl) are taken from (Garrow, 1978). The energy expenditure of fat (kf) and lean tissue (kl) are taken from (Garby, et al., 1988). The values are as follows:

cf = 30.0 [MJ/kg] $cl = 4.0$ [MJ/kg]

$kf = 0.027$ [MJ/ (kg×d)] $kl = 0.116$ [MJ/ (kg×d)]

The fraction of fat,

$$fr = \Delta FM/\Delta BM; \quad (2\text{-}6)$$

Studies have indicated that fr is constant and independent of body weight, with 0.67 for men and 0.76 for women (Heitmann & Garby, 1998; Heitmann & Garby, 2002). Increase of body mass may also increasefr, but at the moment there is not adequate data to consider this in the model. (Christiansen & Garby, 2002) have found that fr has no influence on steady-state but has a direct proportional influence on the rate of weight increase during periods of positive energy imbalance (Christiansen, Garby, & Sorensen, 2004).

The fat mass fraction in the body, taken from (Heitmann & Garby, 1998), corresponds to the following values of BMR0, used in the presented model:

$men: BMR0 = 7.8$ [MJ/d]

$women: BMR0 = 6.8$ [MJ/d]

The efficiency for the transformation of excess energy into new tissue was studied for pigs in (Thorbek, et al., 1983) and (Noblet, et al., 1999) but it has evidently not been determined for humans. We should apply their values to humans. The values used in the paper are:

$ef = 0.85$ $el = 0.55$

2.4. Change of Steady State

Change in energy intake or physical activity factor causes change in steady-state at the body weight. For example, consider an individual in steady state who instantly changes the amounts of EI and/or PAF to new values, which are subsequently kept as constant. The body weight will move toward a new steady state as a result of homeostasis, and the difference between the old and the new steady state can be calculated (Christiansen, Garby, & Sorensen, 2004).

Using the constant values gives:

$$\Delta BMss/(\Delta\, EI/PAF) = \begin{cases} 17.7\ \left[\frac{kg}{\frac{MJ}{d}}\right] & \text{(men)} \\ 20.7\ \left[\frac{kg}{\frac{MJ}{d}}\right] & \text{(women)} \end{cases} \quad \left[\frac{kg}{\frac{MJ}{d}}\right] \tag{2-7}$$

If, for instance, the ratio EI/PAF went up in a steady state situation by 0.1 MJ/d and remained constant afterwards, then body weight would increase in steady-state by 1.77 kg for men and 2.07 kg for women over a year (Christiansen, Garby, & Sorensen, 2004).

2.5. Asymptotic Approach to Steady State

According to (Christiansen, Garby, & Sorensen, 2004), considering another case where EI and PAF are constant while BMss are generated by Equation (2-2). For example, due to a previous change in life style, the present body weight BM(0) is not in steady state. Then Equation (2-1) may be used to take out BMR0 from Equation (2-2). The result is Equation (2-8):

$$(dBM(t))/dt\ =\ PAF\ (kf \times fr + kl(1-fr))/((cf \times fr)/ef + cl(1-fr)/el)\ \times (BMss - BM(t)) \tag{2-8}$$

If fr does not depend on body mass the solution BM(t) to Equation (9) is a simple exponential asymptotic increase towards the new steady state body mass. The equation would be:

$$BM(t)\ =\ BMss - (BMss - BM(0))\ \times\ e^{-PAF\ \frac{kf \times fr + kl(1-fr)}{\frac{cf \times fr}{ef} + cl(1-fr)/el}t} \tag{2-9}$$

The equation is not explicitly dependent on EI or BMR0; but only implicitly through BMss: The half-time, T1/2; for this asymptotic method, i.e., the time it takes before BM(t) has changed by half of the difference BMss – BM(0) is described by the expression (Christiansen, Garby, & Sorensen, 2004):

$$T1/2 = \frac{ln2}{PAF} \frac{\frac{cf \times fr}{ef} + \frac{cl(1-fr)}{el}}{kf \times fr + kl(1-fr)} = \begin{cases} \frac{320}{PAF} & days\ (men) \\ \frac{409}{PAF} & days\ (women) \end{cases} \quad (2\text{-}10)$$

2.6. Constant Rate of Weight Gain

A constant rate of increase in body weight can be considered. In this situation, the time course of EI is computed, assuming that PAF is constant in order to achieve a constant value for dBM(t)/dt. The calculated value of EI(t) is compared with the value of EIss that corresponds to a steady-state condition with the present value of BM and PAF, and EIss is calculated from Equation (04). In this situation, energy intake surpasses the steady-state value by only a very small amount. Consequently, the difference between EI and EIss would be (Christiansen, Garby, & Sorensen, 2004):

$$EI - EIss = \left(\frac{cf \times fr}{ef} + \frac{Cl(1-fr)}{el}\right)\frac{dBM}{dt} \quad (2\text{-}11)$$

This difference is dependent of BM and proportional to the rate of weight increase.

In what is explained above, it is assumed that PAF is considered constant, so the weight increase is just due to the excess energy intake. The analogous study can be performed for constant energy intake, assuming that the weight increase is just because of inadequate physical activity. For the difference, it can be noted that (Christiansen, Garby, & Sorensen, 2004):

$$PAFss - PAF = \frac{dBM}{dt} \times \frac{\frac{cf \times fr}{ef} + cl(1-fr)/el}{BMR0 + (kf \times fr + kl(1-fr))(BM - BM0)} \quad (2\text{-}12)$$

And the dimensionless difference is:

$$\frac{PAFss - PAF}{PAFss} = \frac{dBM}{dt} \frac{\frac{cf \times fr}{ef} + \frac{cl(1-fr)}{el}}{EI} \quad (2\text{-}13)$$

This difference is independent of body weight.

BMR has been received a lot of attention in the studies on the development and treatment of obesity. BMR can be calculated as the sum of the energy expenditure of tissues and organs in a fasting and resting condition and in thermo-neutral situations. The main factors associated with BMR are mass and metabolic rate of tissues and organs. For example, EE is about 10, 15, 20, 35, and 35 times higher in the digestive tract, liver, brain, heart, and kidney than in resting muscle whereas it is only ~1/3 of in the white adipose tissues. Thus, although organs only constitute ~7% of the body weight, they account for ~60% of BMR. In comparison, skeletal and adipose tissues account for 35–40% of BW but only 18–22% and 3–4% of BMR, respectively. Normally, BMR depends on body composition as indicated by fat-free mass (FFM) and fat mass (FM) and on gender, age, physical activity, and nutritional status (Lazzer, et al., 2009). The main element of BMR is FFM (Wang, et al., 2007), whereas FM is important only in obese individuals (Johnstone, Murison, Duncan, Rance, Speakman., 2005). Gender plays a significant role in BMR, as men have a greater BMR than females after adjustment for body composition. In addition, BMR notably drops with advancing age in sedentary populations at a rate of ~1–2% per decade after the age of 20. Such a decline in EE probably contributes to an impaired ability to regulate energy balance with age. The issue of whether EE decreases with age and whether females have lower EE than males, have been explored in several studies, but the literature is imprecise on this topic concerning obese subjects. The aim of (Lazzer, et al., 2009) was to explore the relationship between BMR, gender, age, anthropometric characteristics, and body composition in a very large sample of severely obese white subjects. Therefore, the Equation for the prediction of BMR in adults is the following:

$$BMR = 46 \times BW - 14 \times Age + 1{,}140 \times gender + 3{,}252 \qquad (2\text{-}14)$$

Where gender = 1 for males and 0 for females, BMR is expressed in kJ, age in years, BW in kg.

(Butte, Chirstiansen, & Sorensen, 2007) developed a model based on empirical data and human energetics to predict the total energy cost of weight gain and obligatory increase

in energy intake and/or decrease in physical activity level associated with weight gain in children and adolescents. Their model includes the following:

$$Body\ mass\ (BM)\ =\ FFM\ +\ FM \tag{2-15}$$

Total energy cost of weight gain is equal to the sum of energy storage, EE associated with increased BM, conversion energy (CE), and diet-induced EE (DIEE).

They assumed that BMR is measured by the expression (Butte, Chirstiansen, & Sorensen, 2007):

$$BMR\ = kf\ \times\ FM\ +\ kff\ \times\ FFM \tag{2-16}$$

In (Christiansen, Garby, & Sorensen, 2004) the basal EE of FM (kf) and basal EE of FFM (kff) are assumed to be constants common to all adults. However, these data make it possible and necessary to estimate values for each sex. In the literature, the physical activity level (PAL) has frequently been defined as the ratio between total EE and basal metabolic rate: PAL = TEE/BMR. However, some part of the EE does not depend on physical activity; therefore, there is a need for more complicated equation for total EE during weight gain accounting for conversion energy (CE) and diet-induced EE (DIEE). CE is the energy used to convert dietary energy intake into combustible energy in new tissue. The authors assumed that DIEE = 0.1 EI. In (Christiansen, Garby, & Sorensen, 2004), this term was disregarded, with the argument that it can be included in the definition of EI (replace EI by 0.9 EI). However, this is not sufficient when the estimation of energy requirements is a key point.

$$TEE\ =\ CE\ +\ DIEE\ +\ PAF \times BMR \tag{2-17}$$

The efficiency of the conversion from EI into new tissue depends on the composition of the tissue. They model this effect by the two efficiency coefficients ef (efficiency in the conversion of energy to FM) (0.85) and eff (efficiency in the conversion of energy to FFM) (0.42) for the formation of FM and FFM, respectively. If fr indicates the fat fraction in new tissue, this is partitioned as (Butte, Chirstiansen, & Sorensen, 2007):

$$\Delta FM\ = fr \times \Delta BM, \Delta FFM\ =\ (1\ -\ fr) \times \Delta BM \tag{2-18}$$

The relationship between energy imbalance and rate of increase in body mass (BM) in the adult model is as follow:

$$\frac{dBM}{dt} = \frac{1}{c}\left(0.9 \times EI - PAL \times BMR\right) \quad (2\text{-}19)$$

Where the constant c is given by:

$$c = cf \times fr/ef + cff \times (1 - fr)/eff \text{ [MJ]} \quad (2\text{-}20)$$

Energy stored per kilogram of FFM (cff) equals 4.48 kJ/g (1.07 kcal/g) based on the assumptions that FFM is comprised, on average, of 0.19 g protein/g FFM, and the heat of combustion of protein is equal to 5.65 kcal/g. Energy stored per kilogram of FM (cf) equals 9.25 kcal/g based on the heat of combustion of fat; eff equals 42% for protein; and ef equals 85% for fat (Butte, Chirstiansen, & Sorensen, 2007).

2.7. Issues and Misconceptions about Obesity

In (Flatt J., 2011), some issues and misconceptions about obesity are discussed as follows:

- Problems in applying the energy balance concept- The energy balance equation explains that

  Energy Balance = Energy Intake – Energy Expenditure

  which supports the statement that obesity is due to a "positive energy balance", it means that energy intake is more than energy expenditure. This view complicated past and present. It is not able to recognize the really important difference between lean and obese individuals is the degree of adiposity at which, on average, their energy intake tends to adjust itself to their energy expenditure.

- Problems with the metabolic efficiency concept- The second misconception based on the energy balance equation is that obesity could happen due to unusually low metabolic rates.

- Misleading expectations about the importance of “adaptive thermogenesis”- This represents the decline in energy expenditure from changes in fat mass or fat-free mass, under condition of repeated physical activity, in response to a decline in energy intake (Major, Doucet, Trayhurn, Astrup, & Tremblay, 2007). This means that human body is able to compensate, somewhat for the prescribed energy deficiency.
- Problem in judging the importance of de novo lipogenesis and of its metabolic costs.
- The inapplicability of the “nutrient-partitioning” concept- This concept has been mostly expanded in concept of meat production. In lean as well as in overweight adults whose weight is moderately firm, necessarily all the nutrients consumed over a period of a few days are oxidized, in spite of diet, physical activity level, and adiposity degree. Furthermore, the large changes in physical activity, food consumption, and energy balance happening under free-living situations cause nutrient balances to differ considerably from day to day. So the nutrient-partitioning concept is a bit meaningless when applied to adult humans.
- Failure to recognize the different impacts of energy intake and energy expenditure on energy balance- This concepts says that the impact of an aggressive approach according to only physical activity where the diet is constant is lower than the impact of an aggressive approach based on diet only when the physical activity factor is constant.
- Problems in understanding energy intake regulation- Daily food intake changes. It can be concluded that energy intake has a tendency to adapt itself incredibly to the energy expenditure over time, even though large daily variations from energy balance happen. Therefore, adjustment of intake to energy expenditure in humans must be happening over periods of several days.
- The “defense of body weight” concept- when a weight intervention occurs, Individual body weights tend to return to their original. It is tendency of mechanism toward “defend” a particular body composition. In this concept

the problem is that it seems to indicate that mechanisms exist to actively drive the fat mass to a particular level, much as one would expect if a set-point mechanism existed. This perception fails to take into consideration that before the intervention, individual's body composition had already developed until a steady state of weight maintenance had become established.

- Confusion about the effect of exercise on the energy balance- Probability of developing obesity will be reduced by regular physical activity. A complete advantage of physical activity can be taken in combination with a dieting strategy.

2.8. Modeling Tools

There are multiple factors involved in creating the obesity problem such as biological, psychosocial, cultural, environmental, economic drivers, and also elements associated with the food, physical and cultural environment which influence human behavior (Huang, Drewnosksi, et al., 2009). Abundance of factors involved and the mechanism affect obesity require a system approach to examine issues and assess interventions. Simulation models play a significant role in explosion response planning. SD applications of public health problems include a wide range of drug abuse (Caulkins & Crawford, et al. 1993; Homer, 1993; Behrens & Caulkins, et al., 1999), bio-terror contingency planning (Kaplan & Craft et al. 2002), individual obesity (Abdel-Hamid, 2002), diabetes (Jones, Homer et al. 2006), polio vaccination strategies (Thompson & Tebbens 2007), chronic disease (Homer & Hirsch, et al.,2004; Homer, Hirsch et al. 2007), smoking (Levy & Hyland, et al., 2007), cardio-vascular health (Homer, Milstein et al. 2008), and hepatitis (Behrens, Rauner et al. 2008), among individuals.

In the proposed work, Vensim software is used for simulation based on SD. There are other software packages that can be used. In Table 2-3, the most software providers for SD are listed.

Table 2-4- Major software Packages for SD Modeling

#	Package name	License type	Free?	Contact
1	Vensim	Commercial	Yes	www.vensim.com/
2	Anylogic	Commercial	No	www.anylogic.com
3	Stella, iThink	Commercial	No	www.iseesystems.com/
4	Consideo	Commercial	No	www.consideo-modeler.de/
5	TRUE	Trial 30 days	Yes	www.true-world.com
6	Simile	Commercial	No	www.simulistics.com/
7	Powersim	Commercial	No	www.powersim.com

2.9. Causal Relationships and Standards

In (Christiansen, Garby, & Sorensen, 2004), the theory indicates that an increase in body mass of 1 kg/year correlate to an energy imbalance of 71 kJ/day for men. 82% of this imbalance is transformed into new tissue, while 18% are used for energy conversion. If a man in steady state changes energy intake by 0.1 MJ/day and the physical activity factor remains constant, then the equivalent growth in steady-state body mass is 1.77 kg/PAF, and it will take 320/PAF days before half the change of body mass occurs. About 20 percent of energy expenditure which is used to convert food energy to tissue leads to modifications in previous estimations of the energy imbalance. Consequently, the authors developed a differential equation that quantifies another component of the change in body mass (dBM/dt) as a function of PAF, BM and a number of constants.

By application of the First Law of Thermodynamics to the rate of accumulation of body energy as the difference in energy intake and expenditure, it has been recognized that if that difference equals 1%, body weight will increase by 1 kg/year (Christiansen, Garby, & Sorensen, 2004).

Studies have linked rising obesity rates to the consumption of an increasingly energy-dense diet (Kant, 2005; Ledikwe, 2006). Energy density is the amount of energy (in kilocalories) per a given weight of food (in grams). Energy-dense foods, often high in

refined grains, added sugars, and added fats (Kant, 2005), are palatable, inexpensive, and convenient. However, they have been associated with increased energy intakes and poor diet quality (World Health Organization, 2003; Ledikwe, 2006).

Accordingly, decreasing dietary energy density is one strategy to stem the global obesity epidemic, and resources to decrease energy density are available for both the lay public (Rolls & Barnet, 2000) and health professionals. However, the links between dietary energy density and obesity rates are poorly understood.

(Lazzer, et al., 2009) aimed to explore the relationship between basal metabolic rate (BMR), age, gender, anthropometric characteristics, and body composition in severely obese Caucasian subjects. BMR can be recognized as the sum of the Energy Expenditures (EEs) of tissues and organs in fasting and resting state, and in thermo-neutral conditions. It depends on the mass and metabolic rate of tissues and organs. Generally, BMR depends on body composition as expressed by fat-free mass (FFM), fat mass (FM), gender, age, physical activity, and nutritional status. The main determinant of BMR is FFM, whereas FM is significant only in obese subjects. Gender is also an important element of BMR, since men have a greater BMR than women. Moreover, BMR declines with advancing age in sedentary populations at a rate of approximately 1 to 2% per decade after the age of 20.

2.10. Social Networks Can Affect Weight

In the pioneering Framingham Heart Study, researcher found that probability of becoming obese increased by 40% if a participant's sibling became obese. Since spouses share meals and may have similar exercise habits, the chance of becoming obese rose by 37% if a spouse became obese but if participants had a friend who became obese, the risk of becoming obese increased by 57%. Of course, friends share some meals, but nothing like most meals (Harvard Health Publications, 2011). Students were more likely to gain weight if they had friends who were heavier than they were. In an opposite manner, students were more likely to lose weight— or gain weight at a slower pace — if their friends were leaner than they were.

Social network also influences how active a student is in sports. (By social networks, researchers mean face-to-face friends, not Facebook friends.)

“These results can help us develop better interventions to prevent obesity,” says David Shoham, PhD. “We should not be treating adolescents in isolation.”

Researchers found that partial reason for obesity clustering in social networks was due to the way students selected friends.

But even after controlling for this friend-selecting procedure, there still was a significant connection between obesity and a student’s circle of friends. For instance, if a marginally overweight student at one high school had thin friends (average BMI 20), there was a 40 percent chance the student’s BMI would decrease in the future and a 27 percent chance it would increase (Nauert, 2012).

However, if a borderline overweight student had obese friends (average BMI 30), there was a 15 percent chance the student’s BMI would decrease and a 56 percent chance it would increase.

Researchers believe this shows that social influence “tends to operate more in detrimental directions, especially for BMI; a focus on weight loss is therefore less likely to be effective than a primary prevention strategy against weight gain” (Nauert, 2012).

Although there is no a fully understanding of obesity spread mechanism, scientists suspect that a social network affects what its fellows perceive as normal and acceptable. If people see their friends and others around them becoming heavier and heavier over time, they may accept weight gain as norm, even inevitable. Instead of exercising more or eating less when their weight begins to increase, they may simply accept this weight gain (Harvard Health Publications, 2011).

Incidentally, social networks can also exert opposite influence, and help people maintain a healthy weight (Harvard Health Publications, 2011).

2.10.1. Obesogenic Environments

Environments that are obesogenic increase obesity spread across the world (Hill, Wyatt, Reed, Peters, 2003). The term obesogenic environment was created by (Swinburn, et al., 1999), and according to whom the majority of industrialized nations encourage positive energy balance in their populations due to their physical, economic, social, and cultural environments. An increased risk of obesity is associated with low levels of physical activity (Erlichman, Kerbey, James, 2002) and obesogenic environments discourage not only physical activity but also promote inactivity during both occupational and leisure time (Brownell, 2002; Hill, Peters, 1998). Occupationally related activity decreased greatly since the turn of the 20th century (Popkin, Duffey, Gorden-Larsen, 2005). In industrialized nations and urban areas of developing countries, jobs requiring heavy manual labor have been largely replaced by jobs in services and high-technology sectors, which require minimal physical effort (French, Story, Jeffrey, 2001). Increasingly, people are spending much more time in front of television, playing electronic games, and computer use has increased sedentary behavior of both adults and children, and the use of automobiles and public transportation systems adds more opportunities for physical inactivity (Brownell, 2002; Hill, Wyatt, 2005). Although these changes make unavoidable spread of obesity in obesogenic environments, not everyone living in an obesogenic environment becomes obese. There are two main explanations as to why this happens. The first is that obese susceptible genotypes vary across and within populations; the second is that individual exposure to obesogenic environments varies within any population.

2.10.2. Environmental Effects on Obesity

Recent research suggests that overweight and obesity is the consequence of interaction of individuals with their environment and both are more than just individual problems (envirocancer, 2010).

The environment in this context does not refer Mother Nature (e.g., natural environment). Rather, these are the circumstances in which we live and eat. Table 2-5 shows some features of such environment.

Table 2-5 - Dimensions of environment

Environment Dimension	Description	Examples
Built/ Structural Environment	• Physical environment • Food choice • Physical activity choices	• Grocery store choices • Workplace vending machine choices • Walking/biking trails
Economic Environment	• Can the community afford healthy choices? • Money spent promoting healthy choices • Money spent making healthy choices available	• Can residents afford the fresh produce available in local stores? • Does the town have money to make sidewalks safe to use?
Social Environment	• Attitudes, perceptions, and values of the individuals in a community • Media and advertising influences on individuals and their choices	• Employees don't think they have time to take a walk on their lunch breaks • It's tough to turn down doughnuts brought to the office by coworkers
Policy Environment	• Laws, regulations, and policies that impact eating and physical activity choices. (The policies may be part of the larger social environment or may reflect the "policies" in a home, workplace, or classroom.)	• Workplace policies for healthy foods at meetings • Lack of policy prohibiting snowmobiles on hiking trails.

Environmental Approach to Obesity Prevention:

We live in an obesogenic environment which can be defined as the sum of influences (including our surroundings, opportunities, or conditions of life) advances obesity in individuals and populations.

Although individuals have the responsibility of choosing healthy food, in an environment where having healthy choices is difficult; individuals are not the only responsible for making the right choice. The variety of food culture in the United States has much to do with the weight problems that are now so prevalent in our population. Changes in food use in the U.S. over the last 30 years are informative (envirocancer, 2010).

- Overall energy intake has increased by 15%.
- Fruit and vegetable consumption has increased, but still remains well below recommended levels.
- Milk consumption per capita declined 22.6%.
- Concurrently soft drink consumption increased 131%
- The percentage of foods prepared and eaten outside the home increased 200%

An environmental approach to prevent obesity considers all individual, physical, economic, social, and policy factors affecting obesity when planning an intervention. This approach takes changeable aspects of both environment and individual into consideration. Environment-based interventions make it easier for people to eat healthy; they don't tell people how to eat. Environmental approach is also useful when it is applied to physical activity and nutrition. Individuals tend to make the easy choice or the choice that feels best. The easy choices for individuals are often those foods with high fat and sugar (which taste and feel good, and are often less expensive), and physical inactivity (envirocancer, 2010).

An environment-based intervention shifts the options to make healthy choices easier and good-feeling.

Table 2-6 - Differences between individual and environmental approaches to obesity prevention (envirocancer, 2010)

	Individual Approach	Environmental Approach
Focus	Changing the individual.	Changing the community environment.
Who/What Changes?	Individual behavior.	Structural, social, economio, or policy structures.
Who is Responsible?	Individuals working with health professionals.	Community leaders. Policy makers and health professionals working with community members.
Who is Affected?	People already interested in changing.	Everyone in the community.
Tools	Individual education.	Community development.
Side Effects	Stigmatizing and victim blaming.	Support individual approaches.

CHAPTER 3

THE INDIVIDUAL-LEVEL MODEL

Building models of obesity using individual-level time series data has been hampered by practical considerations related to following and manipulating an individual's eating, physical activity, and weight change over extended periods of time. Available dynamic models for obesity rely on short-term time series data and small sample sizes (Kozusko, 2001; Flatt, 2004; Christiansen, Garby, et al., 2005; Butte, Christiansen, et al., 2007; Hall, 2010) which decrease their direct applicability for policy analysis at the population level.

3.1. Model Structure and Definition

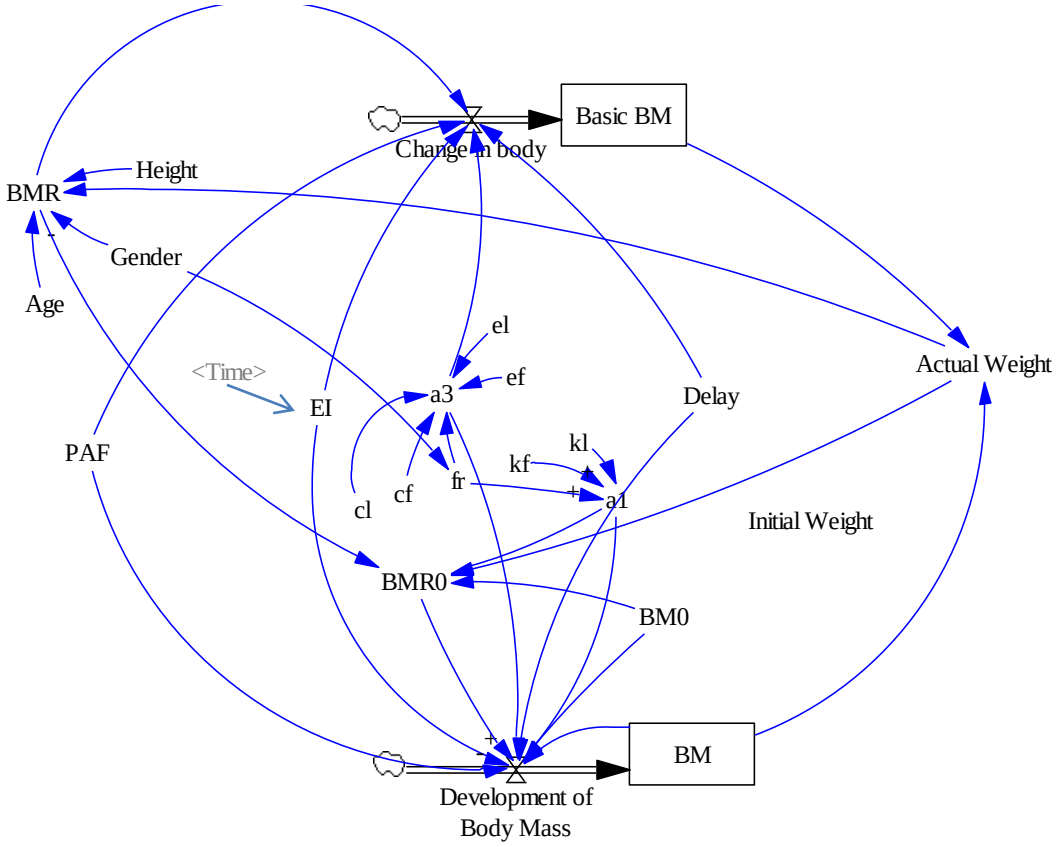

Figure 3-1 - Obesity System Model for an Individual

Overal structure of the individual-level model is presented in Figure 3-1. There are three distinct sections in the model. The first section includes revised Harris Benedict's

equation (Roza & Shizgal, 1984) for calculation the BMR as the main driver. Height, Age, Gender, Weight and Actual weight are the main inputs to this function. It is identifiable that BMR is an input for both of the adjacent main functions that include: Change in body and Development of body mass. The structure of Section I is presented in Figure 3-2.

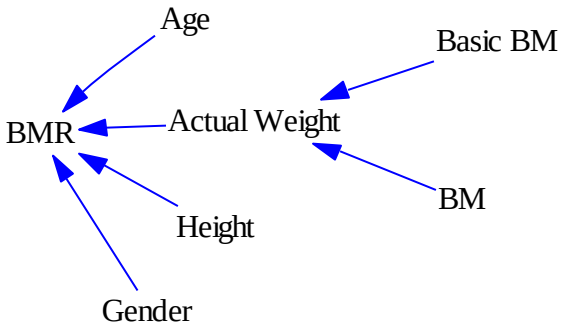

Figure 3-2 – Section I Structure

Central part of section II is based on Butt's transfer function (Butt, Christiansen, & Sorensen, 2007) and introduces Change in the body mass. There are three main elements to this function including BMR, Physical Activity Factor and Energy Intake. There is an additional factor, Delay, which is needed to account for time the body takes to digest and absorb the energy intake. The a3 coefficient is also an input to this transfer function, as introduced earlier. The structure of this section is displayed in Figure 3-3.

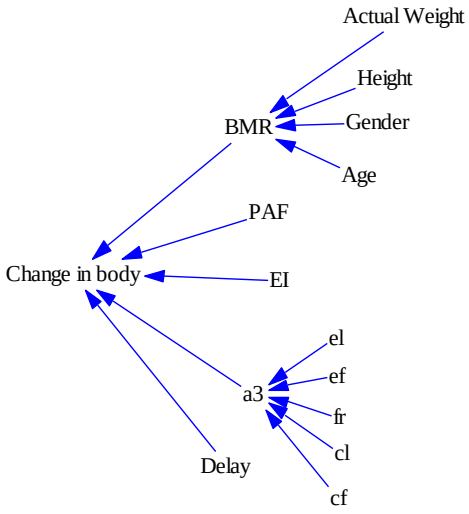

Figure 3-3 – Section II Structure

In Section III, the transfer function developed by (Christian, Garby, & Sorensen, 2004)- Development of Body Mass – has eight inputs including Physical activity Factor, energy Intake, Basal Metabolic Rate at reference body mass, Coefficients a1, a3, Delay, Body Mass, and Reference Body Mass . The structure for this section is presented in Figure 3-4.

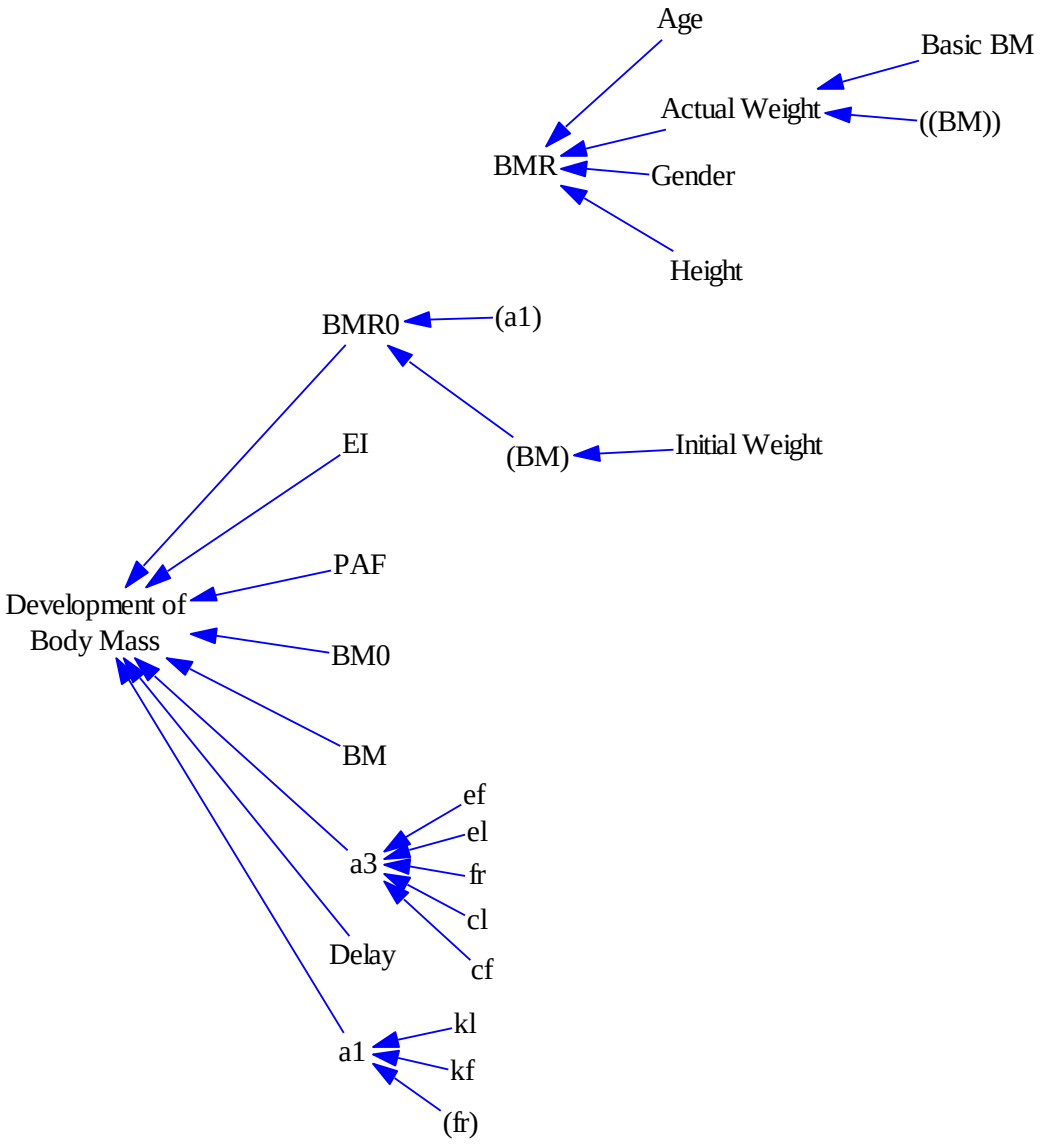

Figure 3-4 – Section III Structure

The Actual Weight is the weight that an individual reaches at each time step. Two main inputs for this function are Body Mass and Basic Body Mass.

There are some inputs in the model which should be introduced by the individual as parameters and include the following:

- The initial weight – The weight of the individual at the beginning [kg]
- The Gender – Male/Female [Unit-less]
- The Age – The physical age of the person [years]
- The Height of an individual [Cm]
- The Physical Activity – How active the individual is [Unit-less]
- The Energy Intake – The amount of energy that the individual is receiving in a day [MJ/d]

3.2. Formulas Used in the Model

Roza & Shizgal, 1984

$$BMRmen = 88.362 + (13.397 \times Actual\ Weight) + (4.799 \times Height) - (5.677 \times Age) \quad \text{[kcal/d]} \tag{3-1}$$

$$BMRwomen = 447.593 + (9.247 \times Actual\ Weight) + (3.098 \times Height) - (4.33 \times Age) \quad \text{[kcal/d]} \tag{3-2}$$

Butt, Christiansen, & Sorensen, 2007

$$Change\ in\ Body = (0.9 \times EI - BMR \times PAF)/(cf \times fr/ef + cl \times (1 - fr)/el) \quad \text{[kg]} \tag{3-3}$$

The equation used in the model:

$$((0.9 \times EI - BMR \times PAF))/a3) \times Delay \tag{3-4}$$

$$Basic\ BM = \int Change\ in\ Body \quad \text{[kg]} \tag{3-5}$$

Initial Value is the "Initial Weight"

Chirstiansen, Garby, Sorensen, 2004

BM_0 = 80 [kg] for both male and female (3-6)

BM_0 is reference body mass which has no influence on results.

$$a1 = kf \times fr + kl \times (1 - fr) \quad [\text{MJ/ (kg×d)}] \tag{3-7}$$

$$a3 = cf \times fr/ef + cl \times (1 - fr)/el \quad [\text{MJ/kg}] \tag{3-8}$$

$$BMR0 = BMR - a1 \times (Actual\ Weight - BM0) \quad [\text{MJ/d}] \tag{3-9}$$

$$Development\ of\ Body\ Mass = \frac{EI - PAF \times (BMR0 + a1 \times (BM - BM0)}{cf \times fr/ef + cl \times (1 - fr)/el} \quad [\text{kg/d}] \tag{3-10}$$

The equation used in the model:

$$(EI - PAF \times (BMR0 + 1 \times (BM - BM0)))/a3) \times Delay \tag{3-11}$$

$$BM = \int Development\ of\ Body\ Mass \quad [\text{kg}] \tag{3-12}$$

The initial Value is the "Initial Weight".

$$Actual\ Weight = IF\ THEN\ ELSE(Basic\ BM > BM, Basic\ BM, BM) \tag{3-13}$$

Vensim is using an unusual way of defining the If/Then/Else condition. The way to read it is:

If (Basic BM > BM) then use Basic BM. Otherwise use BM. All of the If/Then/else functions are the same.

In the model we have "Delay" which means it takes time for the body to stabilize energitecally and adapt a change. In other words, a certain intervention in diet or physical activity at a particular point in time will create a response at a later interval. Considering the delay enables the model to accurately simulate smaller intervals of time.

"The whole thing plays out over a span of roughly 12 hours on average," says Dr. David Katz, and director of the Yale University Prevention Research Center. "By the time the residual waste of food is passing out of us, the useable parts have all been put to use -- for energy expenditure, as heat generation, or placed into storage, as either glycogen (from carbohydrate), or fat" (Huffpost Healthy Living, 2014).

Given this average, we set 12 hours as the time delay for the body to response to the interventions.

3.3. How the Model Can Be Used in Policy Intervention

With the increasing prevalence of overweight and obesity, numerous strategies are used in an effort to control body weight. Dietary treatment regimens and exercise programs are the main options to induce weight loss and they aim to produce a negative energy balance until the targeted body weight is reached (Christiansen & Garby, 2002). Effective strategies for prevention of obesity have been elusive since the recognition of obesity as a major public health issue (Klesges, et al., 2010). Obesity is a result of chronic, quantitative imbalance between energy intake and energy expenditure (Huang, Drewnowski, Kumanyika, 2009).

According to (Rahmandad & Sabounchi, 2011), policy intervention does not often change directly PA or EI, but does it through various indirect approaches such as taxes on sugar sweetened drinks, availability of facilities for physical activity, healthy menus in school cafeteria and other similar engagements to change EI and PA. To assess how the intervention affects obesity, the impact of an intervention on EI and PA for different population members should be evaluated. As a result of these challenges, simulating population level weight change, and determining potential effective interventions in a new population group, needs dynamic models that has following features:

1. Capture the individual-level body weight changes realistically, building on biological processes that adjust energy balance in the body.
2. Link individual and population level dynamics in a practical and standard way.
3. Demonstrate how interventions on energy intake and physical activity affect both individual and population (Rahmandad & Sabounchi, 2011).

CHAPTER 4

POPULATION-LEVEL MODEL

4.1. Model Structure for the Population

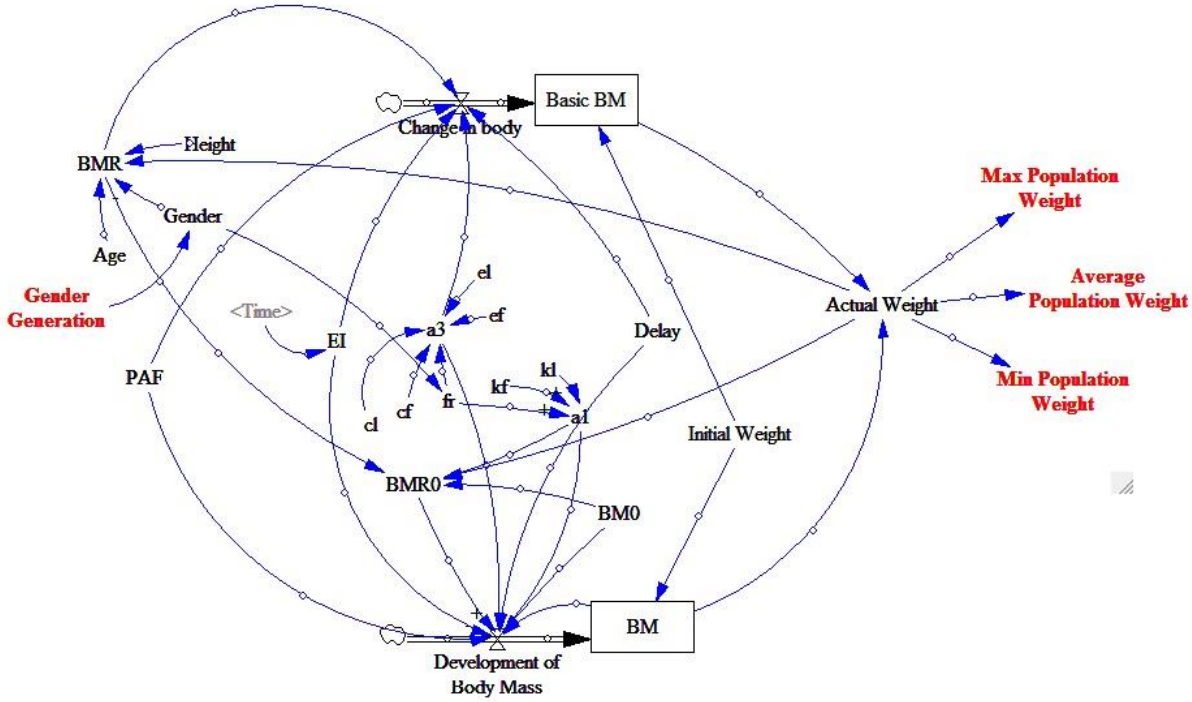

Figure 4-1 - Obesity System Model for a Population

Multiple replications of the individual level model can be simulated together to generate population-level response (Rahmandad & Sabounchi, 2011). By defining subscripts in the individual model we can run the model for population to see how the weight of population will change under different scenarios. Population weight changes can be observed by adjustments of Physical Activity Factor, Energy Intake or Age.

To run the model on a population level, some modifications need to be done to the model. First step is to define distribution for height, age and weight for both male and female. These variables follow specific distributions. Distributions used in the model are defined as follow.

Age: RANDOM NORMAL (20, 69, 35, 12.3)

It is well known that adult male heights follow a normal (Gaussian) distribution. The same is true of adult female heights. First, assume adult male heights are normally distributed with mean 177.8 cm and standard deviation 7.62 cm. Assume also that adult female heights are normally distributed with mean 162.55 cm and standard deviation 7.62 cm (Cook, 2008).

Height for men: NORMAL (160, 190, 177.8, 7.62)

Height for women: NORMAL (155, 175, 162.55, 7.62)

The normal distribution is a remarkably good model of heights for some purposes. It may be more interesting to look at where the model breaks down.

Initial Weight for men: NORMAL (60, 150, 76.7, 12.1)

Initial weight for women: NORMAL (45, 150, 61.5, 11.1)

4.2. The formula used in the model for population

$$Gender\ Generation = \text{RANDOM 0 1 ()} \tag{4-1}$$

This function is used to generate numbers 0 and 1 randomly

$$Gender = IF\ THEN\ ELSE(Gender\ Generation < 0.5,\ 0\ ,\ 1\ ) \tag{4-2}$$

The structure of RANDOM NORMAL in the model is as follow:

$$RANDOM\ NORMAL(Min, Max, Mean, Stabdard\ Deviation, Seed)$$

$$Age\ =\ RANDOM\ NORMAL(20, 69\ , 35\ , 12.3\ , 5) \tag{4-3}$$

$$Height =$$
$$IF\ THEN\ ELSE(Gender = 1, RANDOM\ NORMAL(160, 190\ , 177.8\ , 7.62\ , 3\ ),$$
$$RANDOM\ NORMAL(\ 155\ , 175\ , 162.55\ , 7.62\ , 4\ )\ ) \tag{4-4}$$

$$Initial\ Weight =$$
$$IF\ THEN\ ELSE(Gender\ =\ 1, RANDOM\ NORMAL(60, 150\ , 76.7\ , 12.1\ , 1)\ ,$$
$$RANDOM\ NORMAL(45, 150\ , 61.5\ , 11.1\ , 2\ )\ ) \tag{4-5}$$

$$Average\ Population\ Weight\ =\ SUM(Actual\ Weight[Person!])/\ population\ number \quad (4\text{-}6)$$

There is a little change in the model as well. To run the model for the population, gender is an input for both initial weight and height. Another one is Average population Weight which shows how population weight trend will change.

CHAPTER 5

MODEL VALIDATION AND SCENARIOS FOR BOTH INDIVIDUAL AND POPULATION LEVEL

5.1. Model Validation

It is vital to establish the validity of the structure of SD model which is the validity of the set of equations used in the model. Structure validity is followed by checking the accuracy of model behavior. However, it should be noted that a point-by-point match between the real behavior and the model behavior is not as important as it is in classical forecasting modeling (Barlas, 2000; Forrester & Senge, 1980). There are many tests of model structure and behavior for SD models which are not possible with other types of models. Contrarily, some commonly used tests for other models are inappropriate for SD models (Forrester & Senge, 1980).

(Forrester and Senge, 1980) suggested a number of direct structure tests, for example, structure and parameter verification tests, and dimensional consistency test. In this study, the structure and parameter verification tests are conducted by comparing the model equations with the existing knowledge on the obesity system modeling in the literature. Finally, dimensional consistency test for all model equations is conducted.

Tests of model behavior evaluate the adequacy of model structure through analysis of the behavior generated by the structure (Forrester & Senge, 1980). Some of these tests which are conducted in this study include behavior sensitivity. The behavior anomaly test is extensively used in both phases of model development and model validation. After discovering some model behavior which significantly conflicts with behavior of the real system, the abnormal behavior was traced to find the obvious flaws in the model or assumptions. Furthermore, the behavior sensitivity test is conducted by Monte Carlo simulation.

5.2. Comparative Analysis

To understand usefulness of the model, as well as, test and validate the results, a set of techniques have been used. Actual data was provided by a Medical Weight Loss Clinic in Windsor. There are also some actual data taken from online website (The Dynamics of Weight Control).

The model has been run for different individuals and the results are as follows:

Example 1:

A 20 year-old woman who is moderately active, 162 cm tall and 56.70 kg in weight needs 1700 kcal per day to maintain her weight. At age 30, this same woman would need 1650 kcal/day to maintain her weight of 56.70 kg.

Many people find a way to gradually adjust their weight with age, so they eat less or increase their physical activity. If the woman in this example failed to modify her calorie intake, after ten years by the time she reached age 30, she could gain 4.5 kg. The model was ran for her for 10 years and she reached 62.77 kg.

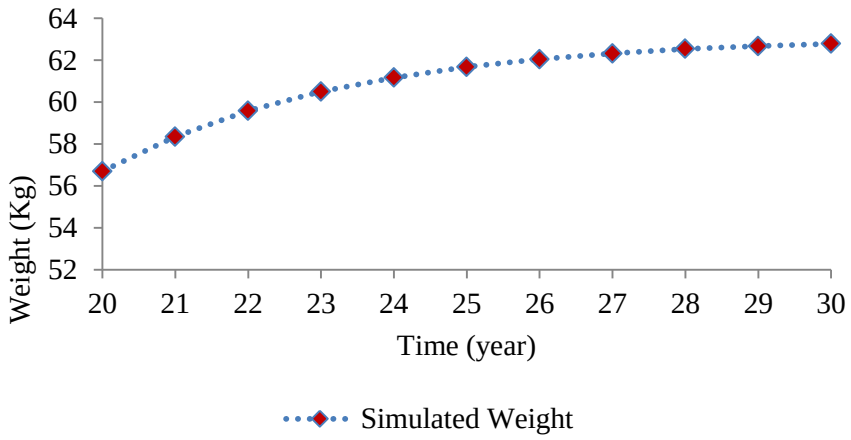

Figure 5-1 – Ten-year simulation for a 20 years old female

Example 2:

For this case the data is provided by the Medical Weight Loss Clinic in Windsor. In this actual field data, patients went through a particular type of diet, at much lower caloric amount than they would normally consume. That is complemented by a particular choice

of foods, administered under the supervision of the trained personnel. The patients keep a daily of calories consumed as other details of their daily diet. The program varies in length and the patients measure their weight weekly. There were several other input parameters provided by the clinic. They include: Age (of the individual), Gender, Calories and a qualitative assessment of the PAF. A weekly recording of the actual Weight of the individual was also provided. A portion of the data structured is shown in Appendix A.

Figure 5-2 – is showing the results of running the model for a 39 year-old male who is 160 cm tall, 141.5 Kg in weight with the physical activity factor of 1.45.

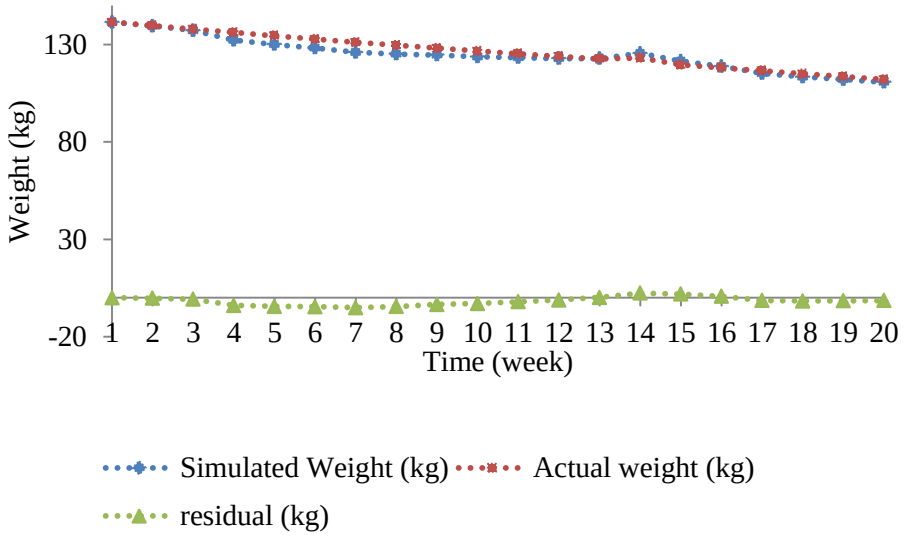

Figure 5-2- Weight simulation for a 39 year-old male

5.3. Overeating Scenario for the Population Level Model

We carried out a Task to understand the dynamic of weight gain/loss. The target of this experiment is to determine how body weight varies in response to changes in energy intake during the holiday season based on an overeating scenario. In this task the individual's energy intake increases (from 2000 kcal/day) linearly and progressively peaking at 2800 kcal/day for four days and then decrease back to 2000 kcal/day. This dietary pattern repeats again during the next holiday season such as Christmas. To simplify the experiment, physical activity factor stays constant.

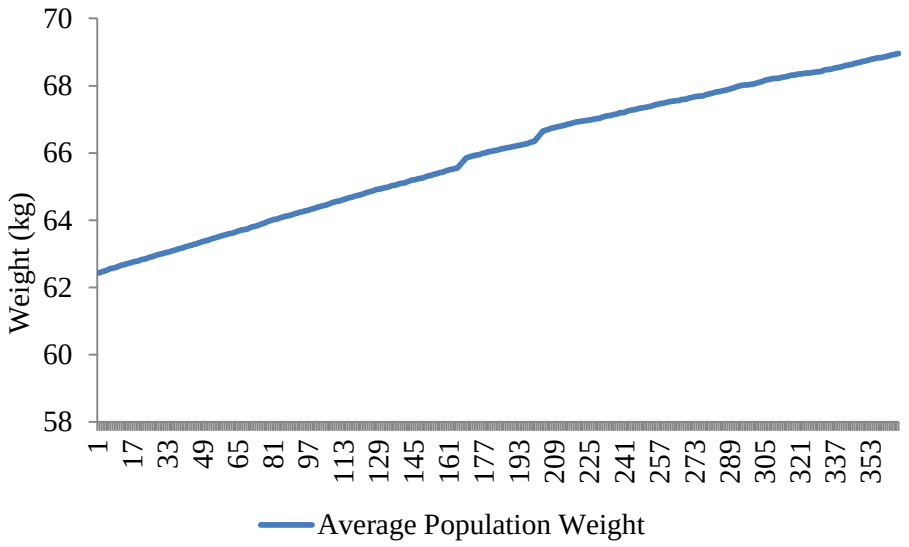

Figure 5-3 – Average population weight due to overeating

Upper and lower bounds of population weight can be seen in Figure 5-3.

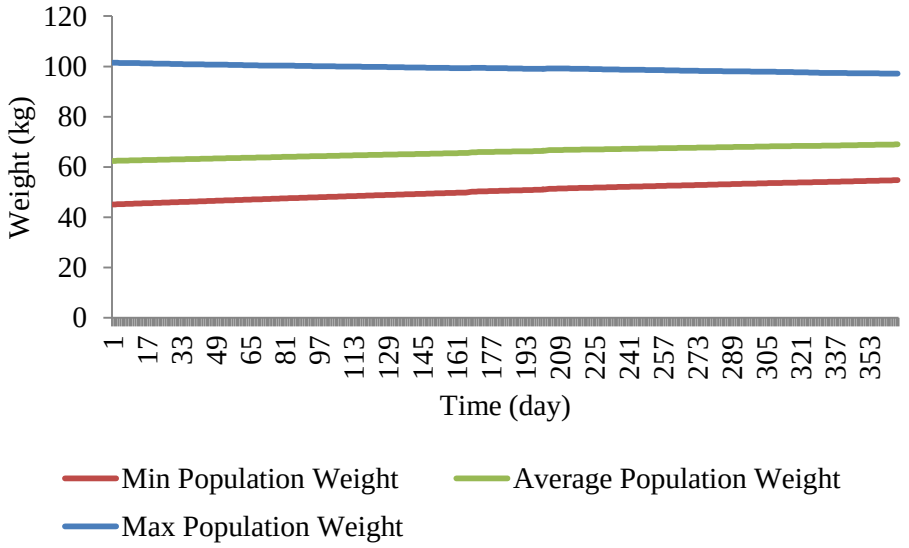

Figure 5-4 – Minimum, Maximum and average population weight due to overeating

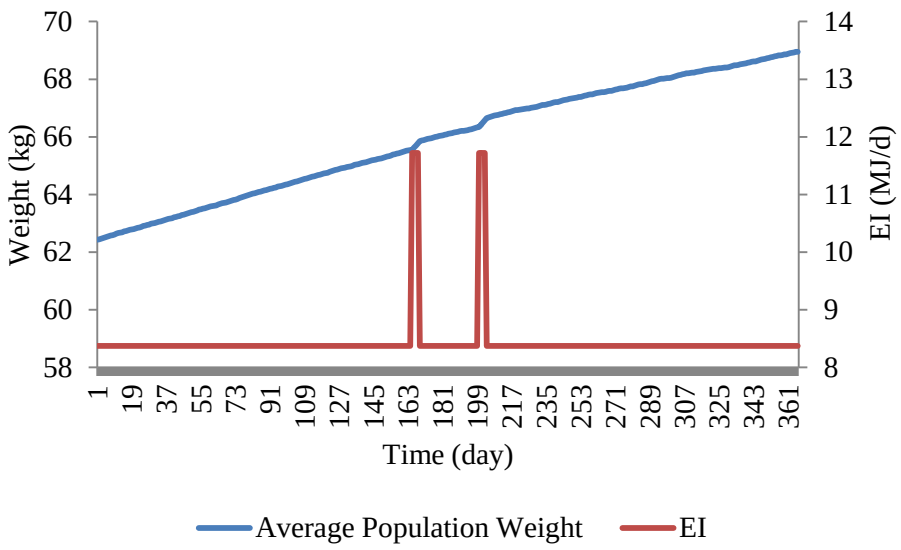

Figure 5-5 – Comparison of average population weight with energy intake

The primary vertical axis is showing the weight and the secondary one is showing the EI. It can be seen that overeating creates a jump in weight in a short amount time. The body tends to move back due to equilibrium but it may not go back to the same point where the individual was before overeating unless caloric shortage recompenses the excess intake.

5.4. Two-day a Week Fasting Scenario

We want to determine how body weight responds to fasting (500 kcal/day) two days of a week.

In this scenario the individuals (1000 individuals) consume 2000 kcal/d for three days and then their energy intake is kept at 500 kcal/day for two days and then goes up again to 2000 kcal/day. This dietary pattern is repeated over the period of six months.

To run the model for this scenario, some modifications were required. The equations which are needed are as follows:

Random EI = PULSE TRAIN(3, 2 , 3 , 180) (5-1)

Pulse Train function is used to generate numbers 0 and 1. This function generates 1 for three days and then 0 for two days. This pattern repeats for 180 days.

EI = IF THEN ELSE(Random EI = 1 , 2.092 , 8.37) (5-2)

This equation means if Random EI equals to 1 then take 2.092 MJ/day (500 kcal/day) as energy intake otherwise choose 8.37 MJ/day (2000 kcal/day).

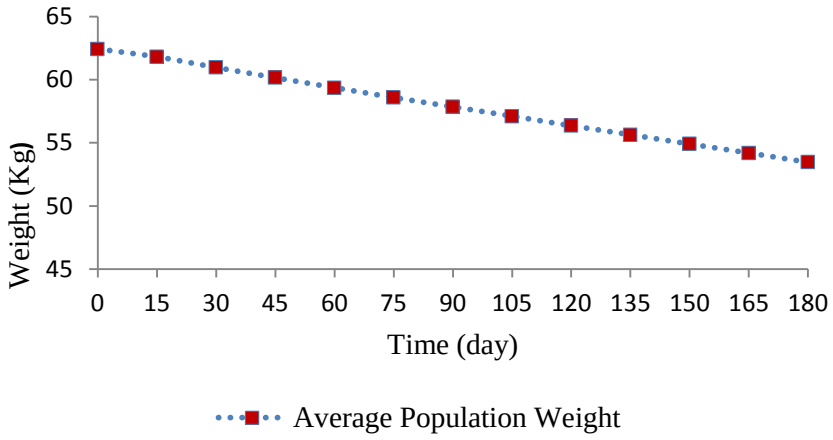

Figure 5-6 – Average population weight due to two days a week fasting

At the beginning, before following this particular dietary regimen, the average population weight was 62.46 kg, after six month the population weight declined to 54. 61 kg.

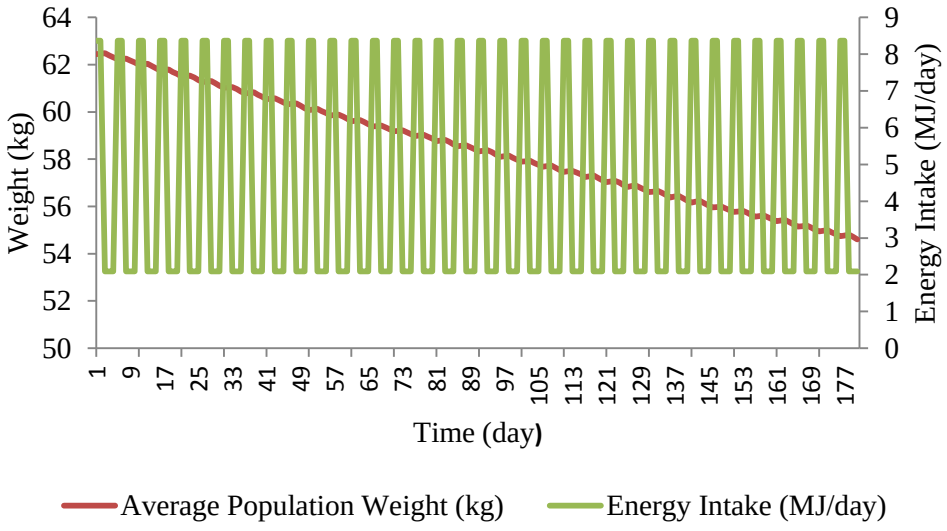

Figure 5-7 – Comparison of average population weight with energy intake based on two days a week fasting

Based on this scenario, by this amount of EI, when people fast two days after every three days, they lose weight. The average weight loss was 7.85kg.

In this scenario, we increase energy intake from 8.37 MJ/day (2000 kcal/day) to 14.65 MJ/d (3500 kcal/day) to see how the population weight changes. Figure 5-8 – shows how the body responds to this change in EI.

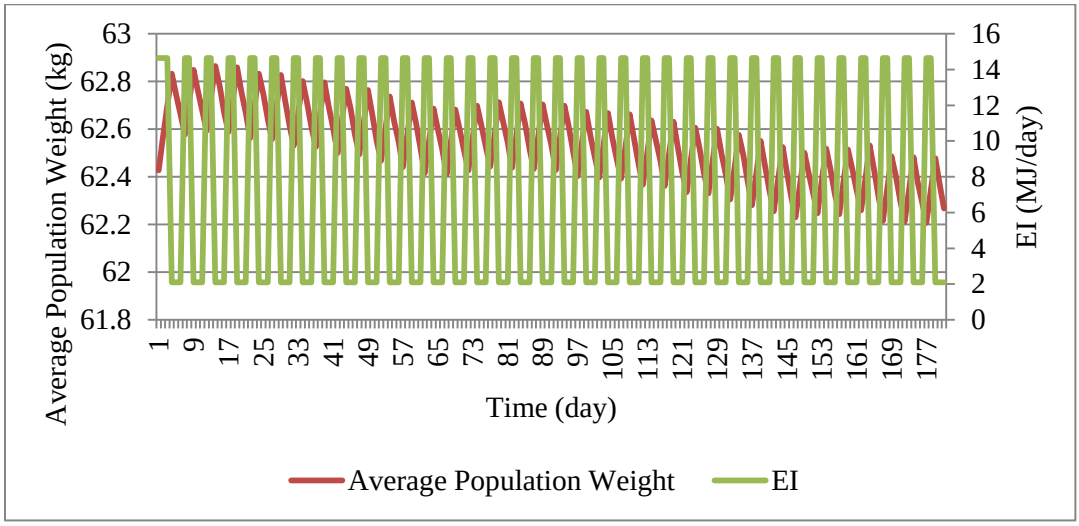

Figure 5-8 – Average population weight when EI has been increased in two-day fasting scenario

In this scenario, the average population weight was 62.42 kg at the beginning and the after one year it reached 62.26 kg. It can be seen that even though there are some fluctuations in the population weight, the overall behavior shows that the population is capable of maintaining the weight (it changes between 62.2 kg and 62.8 kg).

CHAPTER 6

REGRESSION AND SENSITIVITY ANALYSIS

Sensitivity analysis is the procedure of changing the assumptions about the value of Constants set in the model and assessing the resulting output. Manual sensitivity analysis includes changing the value of a Constant (or several Constant at once) and simulating, then changing the value of the constant again and simulating again and redoing this action many times to get a spread of output values.

6.1. Regression Analysis

To perform regression analysis we ran the model for standard male and female (an individual with average characteristics of age, weight, height) over a period of 30 days. In this analysis one parameter (EI or PAF) is kept constant and another factor (PAF or EI) varies then the output (weight) is recorded at the end of the 30^{th} day.

We ran the model 42 times for each standard male and female to find a relation between Weight, EI and PAF. In other words, we wanted to know whether we can predict the weight if we have EI and PAF?

Table 6-1 – Characteristics of standard male and female

	Male	Female
Age	35	35
Height (cm)	177.8	162.55
Initial Weight (kg)	76.7	61.5

Table 6-2 - Weight for standard male at the 30th day for specific amount of EI and PAF

PAF / EI(MJ/d)	1	1.2	1.4	1.6	1.8	2
4	74.76	73.93	73.10	72.28	71.46	70.65
6	75.89	75.06	74.23	73.40	72.58	71.77
8	77.03	76.19	75.36	74.53	73.70	72.89
10	78.16	77.32	76.48	75.65	74.82	74.00
12	79.30	78.45	77.61	76.77	75.94	75.12
14	80.43	79.58	78.74	77.90	77.06	76.24
16	81.56	80.71	79.86	79.02	78.18	77.35

Excel produces the following results:

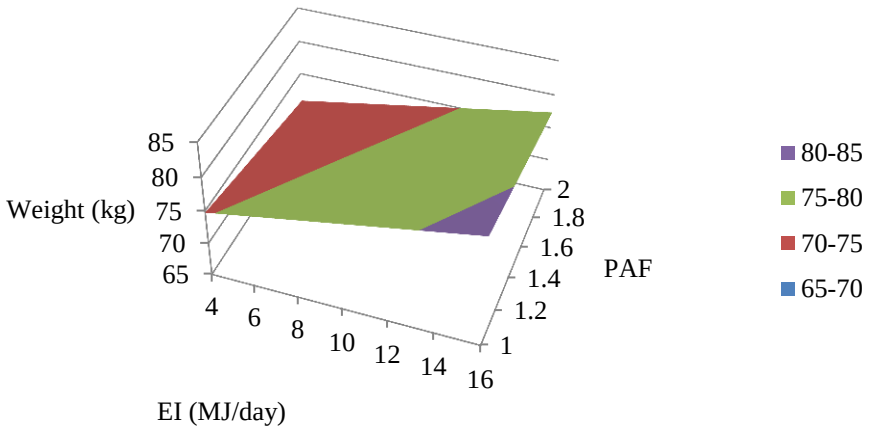

Figure 6-1 – Relation between EI, PAF and Weight for a standard male

Figure 6-1 shows that weight values for different level of EI and PAF follow surface. It means that there is a linear relationship between weight values that can be predicted.

Excel produces the following results:

Table 6-3 – Regression Statics for a standard male

SUMMARY OUTPUT	
Regression Statistics	
Multiple R	0.999987
R Square	0.999974
Adjusted R Square	0.999972
Standard Error	0.014168
Observations	42

Table 6-4 – ANOVA for a standard male

	df	*SS*	*MS*	*F*	*Significance F*
Regression	2.0E+00	3.0E+02	1.5E+02	7.4E+05	4.9E-90
Residual	3.9E+01	7.8E-03	2.0E-04		

	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*
Intercept	76.688	0.011	6809.735	0.0000	76.665	76.711
EI	0.563	0.001	1029.363	0.0000	0.562	0.564
PAF	-4.161	0.006	-650.083	0.0000	-4.174	-4.148

Residual and probability output are shown in Appendix.

Table 6-5 shows weight values for a standard female at the day 30.

Table 6-5 – Weight for standard female at the 30th day for specific amount of EI and PAF

PAF / EI(MJ/d)	1	1.2	1.4	1.6	1.8	2
4	60.60	60.00	59.41	58.83	58.24	57.66
6	61.64	61.04	60.45	59.86	59.27	58.69
8	62.68	62.08	61.49	60.89	60.30	59.72
10	63.72	63.13	62.52	61.93	61.34	60.75
12	64.76	64.16	63.56	62.96	62.37	61.78
14	65.80	65.15	64.59	63.99	63.40	62.81
16	66.84	66.23	65.63	65.03	64.43	63.83

The following results are produced by excel for standard female.

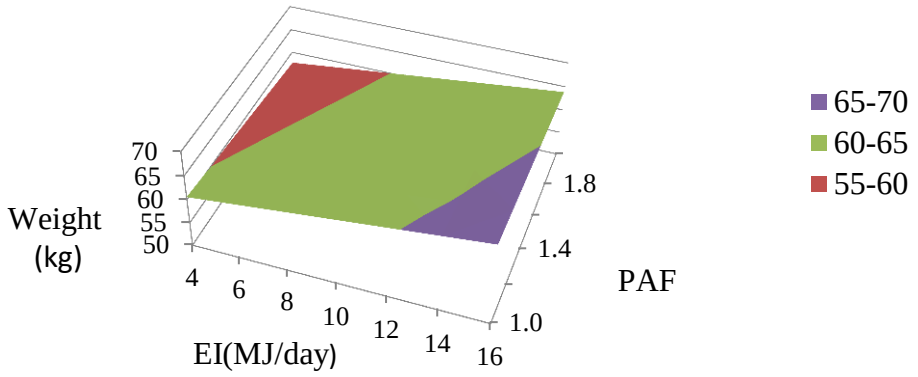

Figure 6-2 – Relation between EI, PAF and Weight for standard female

Figure 6-2 shows that the values of weight for the standard female at the day 30 follow surface.

Table 6-6 - Regression Statics for standard female

SUMMARY OUTPUT	
Regression Statistics	
Multiple R	0.999988
R Square	0.999978
Adjusted R Square	0.999977
Standard Error	0.0112786
Observations	42

Table 6-7 – ANOVA for standard female

	df	*SS*	*MS*	*F*	*Significance F*
Regression	2	222.910	111.455	876168.400	0.000
Residual	39	0.005	0.000		
Total	41	222.915			

	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*
Intercept	61.510	0.009	6861.142	0.000	61.492	61.528
EI	0.517	0.000	1188.609	0.000	0.516	0.518
PAF	-2.969	0.005	-582.705	0.000	-2.979	-2.959

The regression surface for male and female are as follows:

$$W\ male = 0.5626\ \times EI - 4.1082\ \times PAF + 76.6882 \quad (6\text{-}1)$$

$$W\ female = 0.5171\ \times EI - 2.9689 \times PAF + 61.5097 \quad (6\text{-}2)$$

To understand which factor (EI or PAF) has a greater impact on weight, contribution of each parameter should be calculated. To evaluate impact of EI and PAF, proportion of 0.5626EI and 4.1082PAF (0.5171EI, 2.9689PAF) and then the ratio of 0.5626EI/4.1082PAF (0.5171EI/2.9689PAF) for standard male (female) should be calculated. Results are shown in tables 6-8 and 6-9.

Table 6-8 – Ratio of 0.5626EI/4.1082PAF for standard male

PAF / EI(MJ/d)	**1.0**	**1.2**	**1.4**	**1.6**	**1.8**	**2.0**
4	0.548	0.456	0.391	0.342	0.304	0.274
6	0.822	0.685	0.587	0.514	0.456	0.411
8	1.096	0.913	0.738	0.685	0.609	0.548
10	1.369	1.141	0.978	0.856	0.761	0.685
12	1.643	1.369	1.174	1.027	0.913	0.822
14	1.917	1.598	1.369	1.198	1.065	0.959
16	2.191	1.826	1.565	1.369	1.217	1.096

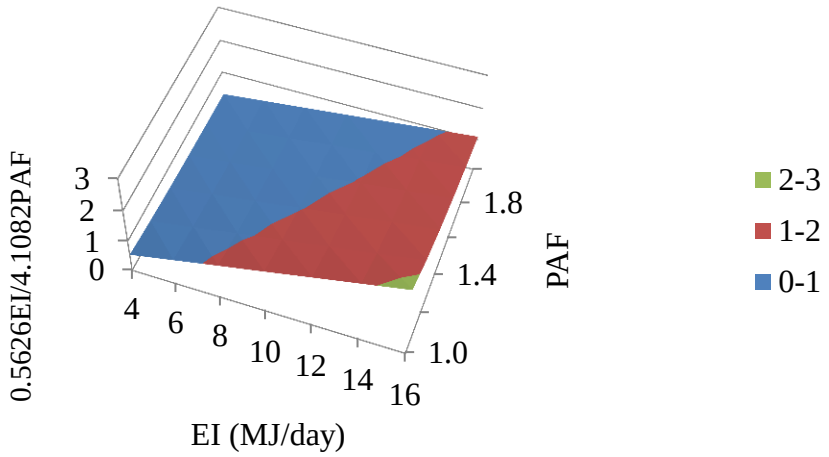

Figure 6-3 – Relation between EI, PAF and ratio of 0.5626EI/4.1082PAF for a standard male

Results for standard female are as follows:

Table 6-9 – Ratio of 0.5171EI/2.9689PAF for a standard female

PAF / EI(MJ/d)	**1.0**	**1.2**	**1.4**	**1.6**	**1.8**	**2.0**
4	0.697	0.581	0.498	0.435	0.387	0.348
6	1.045	0.871	0.746	0.653	0.581	0.523
8	1.393	1.161	0.995	0.871	0.774	0.697
10	1.742	1.451	1.244	1.089	0.968	0.871
12	2.090	1.742	1.493	1.306	1.161	1.045
14	2.438	2.032	1.742	1.524	1.355	1,219
16	2.787	2.322	1.991	1.742	1.548	1.393

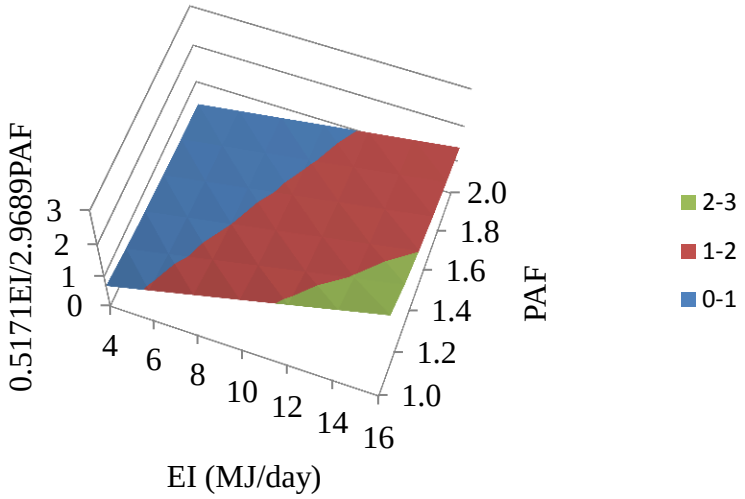

Figure 6-4 – Relation between EI, PAF and Ratio of 0.5171EI/2.9689PAF for standard female

According to tables 6-8 and 6-9, there are different ratios for different levels of EI and PAF (from 0.274 to 2.191 for male and from 0.348345 to 2.786756 for female). It means at different levels of EI and PAF, body respondse in a different way. For example at the highest level of EI (16 MJ/day) energy intake has more significant impact on body mass than physical activity even though PAF has the highest level.

R Square

R Square is 0.999974 (0.999988 for female), which is a very good fit. It means 99% of the variation in Weight is explained by the independent variables EI and PAF. The close to 1, the better regression line.

Significance F and P-values

Significance F is used to check reliability of the results. If this value is less than 0.05, results are reliable. Variables with a high P-value (greater than 0.05) should be deleted and reran the regression until Significance F drops below 0.05.

Coefficients

According to these two formulae (e.g., equation 6-1 and 6-2) we can understand that for each unit increase in EI, weight increases with 0.5626 and 0.5171 for male and female respectively. For each unit increase in PAF, weight decreases with 4.1082 and 2.9689 for male and female respectively.

Residuals

The residuals show how far away the actual data are from the predicted data.

6.2. Monte Carlo Simulation

Method of automating this process is Monte Carlo simulation known as multivariate sensitivity simulation (MVSS). Hundreds or even thousands of simulations can be carried out, with Constants sampled over a range of values, and output for subsequent analysis.

Sensitivity testing is based on Monte Carlo simulation. The method choses the values randomly from a specific distribution type in order to evaluate the behavior of the output. Sensitivity analysis is carried out for the two main inputs (PAF and EI) against the main output (Weight). For sensitivity simulations, probability distribution values for each parameter should be defined. Random Uniform Distribution which is suitable for most sensitivity testing is selected by default in the Vensim. In this distribution, any number between the minimum and the maximum has the equal probability to occur. Random Normal Distributing is chosen in this work because values near the mean are more likely to occur than values far from the mean.

To quantify the influence of each factor on the variability of the outcome (Weight), both the set point (mean) of the energy intake and physical activity factor were kept constant at 10 MJ/day and 1.5, respectively.

In the Population Level Model, EI and PAF are assumed to be 8.37 MJ/d (2000 kcal/day) and 1.375, respectively. It can be seen in Figure 6-1 that by this amount of energy intake and physical activity factor, the average population weight increases over time.

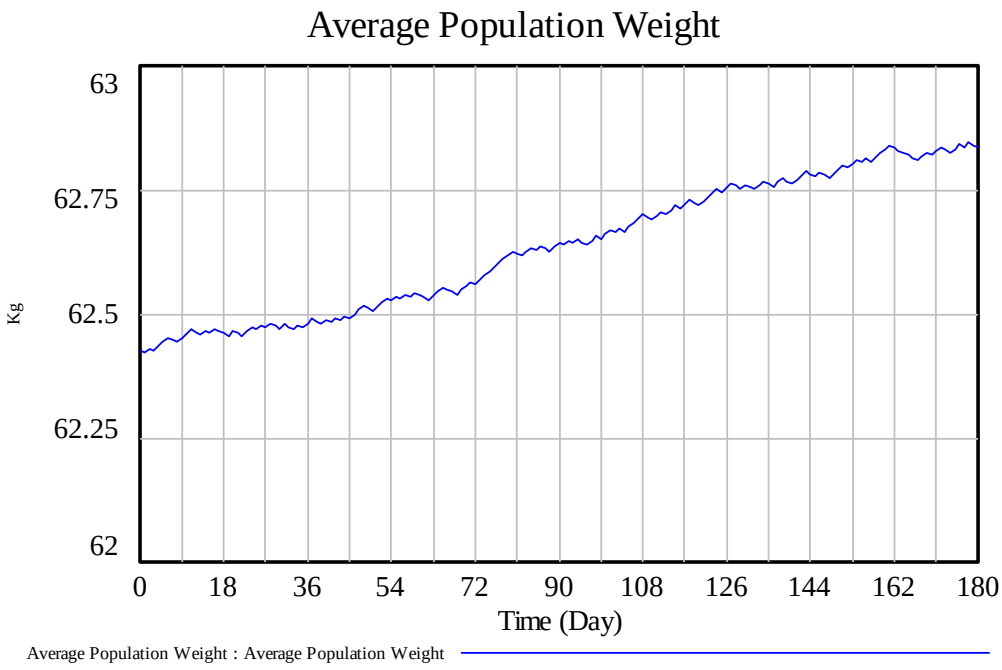

Figure 6-5 – Average Population Weight at EI = 8.37 MJ/d & PAF = 1.375

To observe the effects of EI and PAF, all model parameters in the model should be constant during the sensitivity analysis. The number of simulations is set at 200. To perform on multivariate test, the distribution for each parameter specified sampled, and the resulting values used in a simulation. This procedure will be repeated 200 times. Sensitivity scenarios are defined in Table 6-2.

Table 6-10 – Sensitivity Scenarios

Fig.	Energy Intake					Physical Activity Factor				
	Distr.	Min. (MJ/d)	Max (MJ/d)	Std. Dev.	Mean (MJ/d)	Distr.	Min.	Max	Std. Dev.	Mean
6-6	RN	4	16	4	10	Constant				
6-7	RN	7	13	4	10					
6-8	RN	4	16	2	10					
6-9	Constant					RN	1	2	0.2	1.5
6-10						RN	1.3	1.7	0.2	1.5
6-11						RN	1	2	0.1	1.5
6-12	RN	4	16	4	10	RN	1	2	0.2	1.5
6-13	RN	7	13	2	10	RN	1.3	1.7	0.1	1.5
6-14	RN	4	16	2	10	RN	1	2	0.1	1.5
6-15	RN	7	13	4	10	RN	1.3	1.7	0.2	1.5

In Figure 6-6, Monte Carlo simulation results are shown with energy intake at its maximum Range and Variation. It can be seen that in spite of an average weight gain, the overall behavior allows for both weight gain and loss.

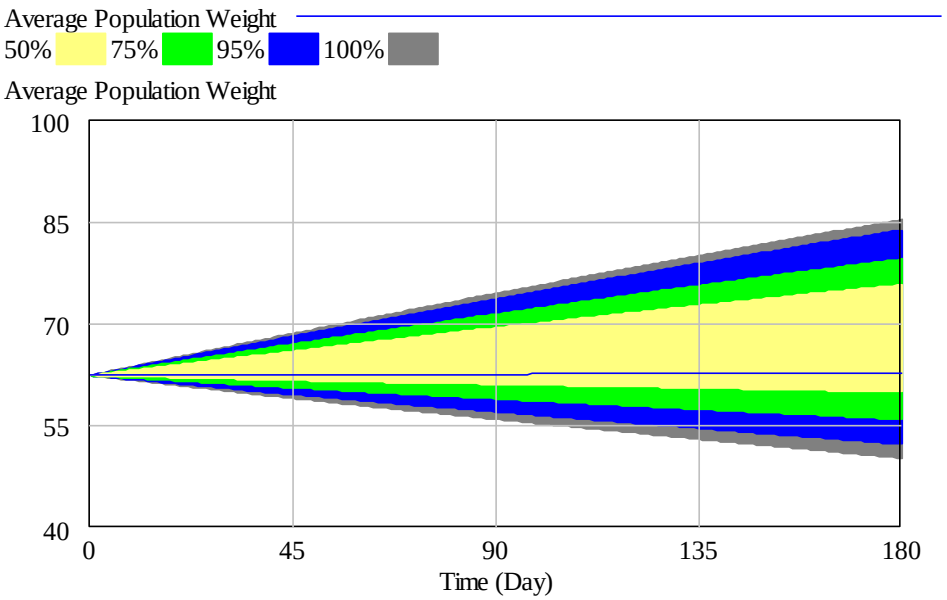

Figure 6-6 – Sensitivity EI with Maximum Range and Variation

For all sensitivity analysis graphs presented in this study, the four ranges presented around the mean are at 50%, 70%, 90% and 100% starting from the closest to the mean and moving outwards respectively. Graphs generated in this analysis, shows confidence bounds for all the output values of Average Weight Population that were generated when EI & PAF were randomly varied about their distributions. The outer bounds of uncertainty (100 %) show maximum values of approximately 85 kg and minimum values of approximately 45 kg at the end of the simulation. Note that there is the possibility of a decline in average population weight. The first simulation run (with the values of the Constants contained in the model) is plotted as a line indicated by the run name Average Population Weight.

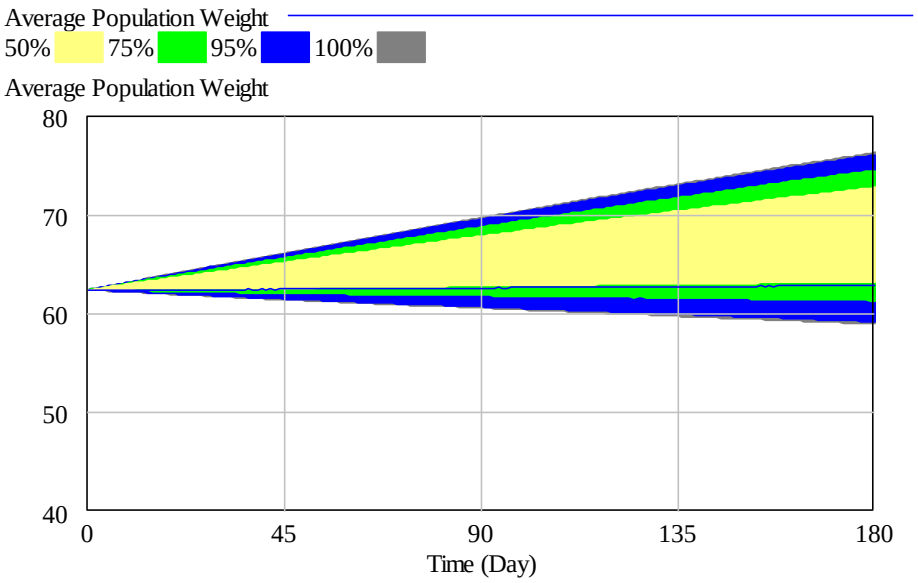

Figure 6-7 – EI Small Range/Maximum Variation

In Figure 6-7, the standard deviation and all other parameters are kept constant; the difference is just the range (i.e., minimum and maximum values) that is reduced down to 7 MJ/day and 13 MJ/day, respectively. There is a remarkable decline in the variation of the results and the range.

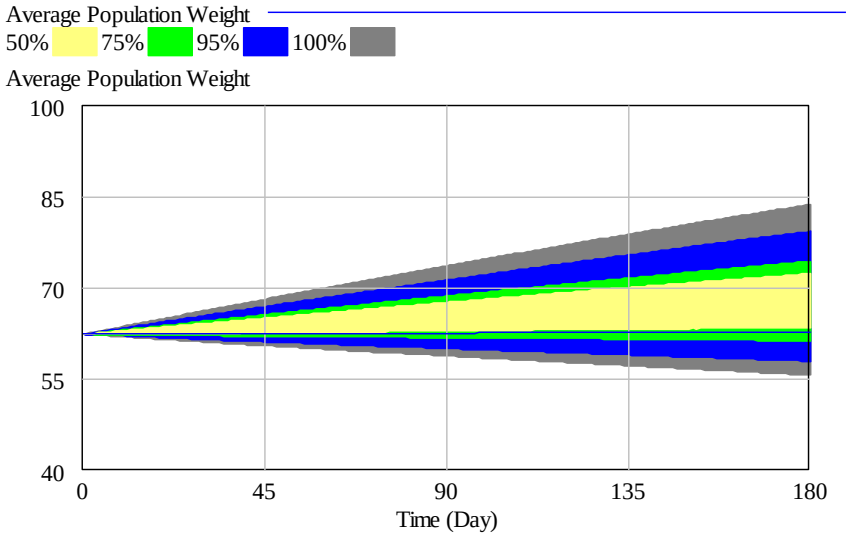

Figure 6-8 – EI Maximum Range/Small Variation

In Figure 6-8, the average remains constant at the initial level and the range is reduced a little bit, while the variation is reduced from 4 to 2 MJ/day.

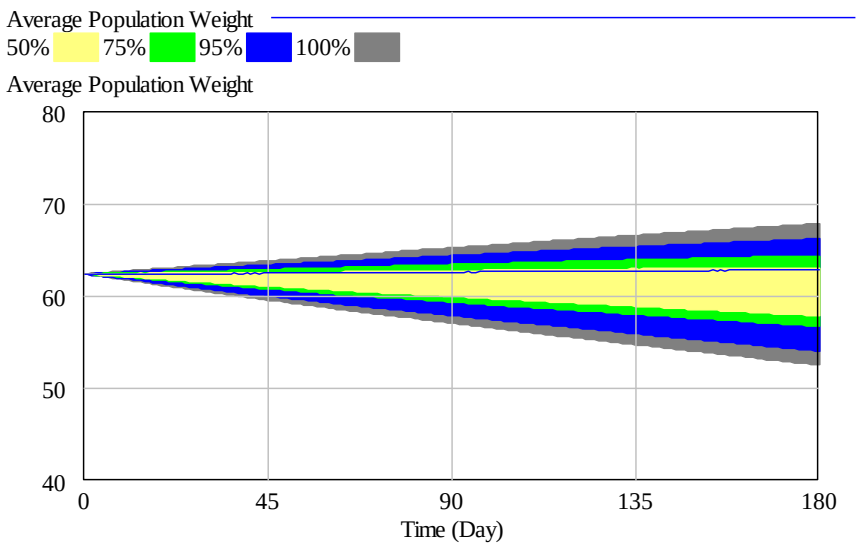

Figure 6-9 – PAF with Maximum Range and Variation

In Figure 6-9, a similar analysis using Physical Activity Factor is provided. All other parameters including EI are kept constant so the effect of PAF alone can be identified. According to the Figure 6-9, it can be concluded that the rate of losing weight is faster.

A smaller range of PAF (1.3 to 1.7) was used in Figure 6-10, while the variation is remained constant. Alternately, in the simulation presented in Figure 6-11 a smaller variation of PAF from 0.1 versus 0.2 was used.

The behavior of the weight shows similar trends as the EI analysis. Based on these scenarios, it is better to sustain a balanced program without large extremes while focusing on program consistency. It means that keeping a stable average is the preferred policy while maintaining a smaller range.

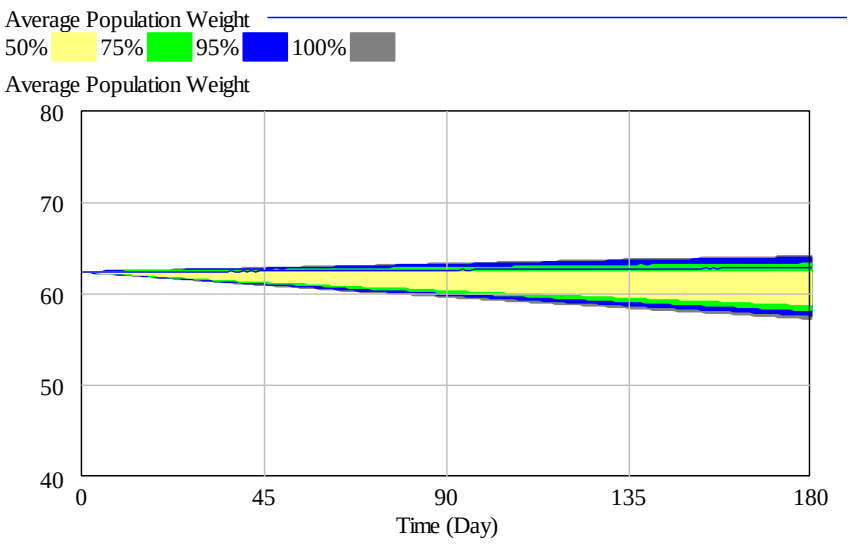

Figure 6-10 – PAF Small Range/High Variation

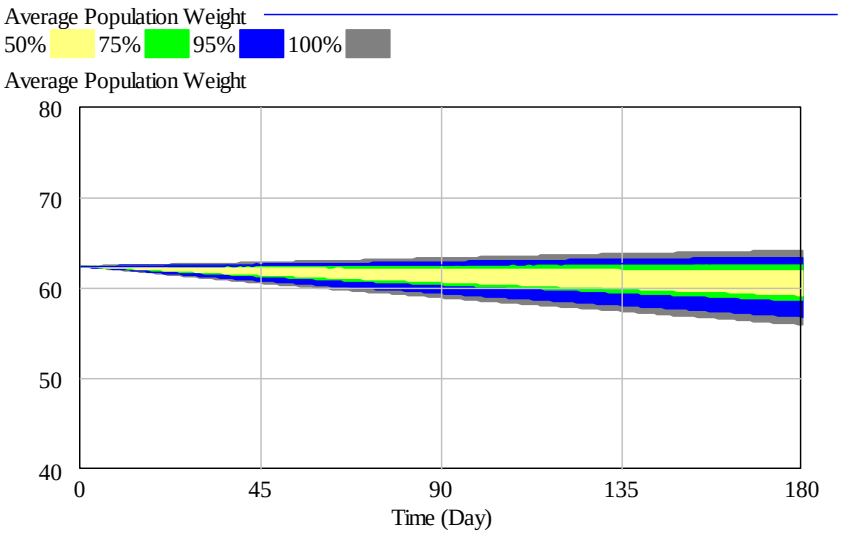

Figure 6-11 – PAF Large Range/Small Variation

The last four figures (6-12 – 6-15) presented, the two main factors (EI & PAF) are simulated at the same time so that that their integrated effect can be observed. The sensitivity analysis can be carried out with different setting. For instance, the two main parameters, EI and PAF, can be set in a way that they cancel each other on an average and examine the effect of different setting from the perspective of the range, variation and even the mean. For example, if an individual suddenly eats and drinks more, there is a compensating value of the PAL that cancels out the impact of the excess caloric intake (e.g. by exercising more).

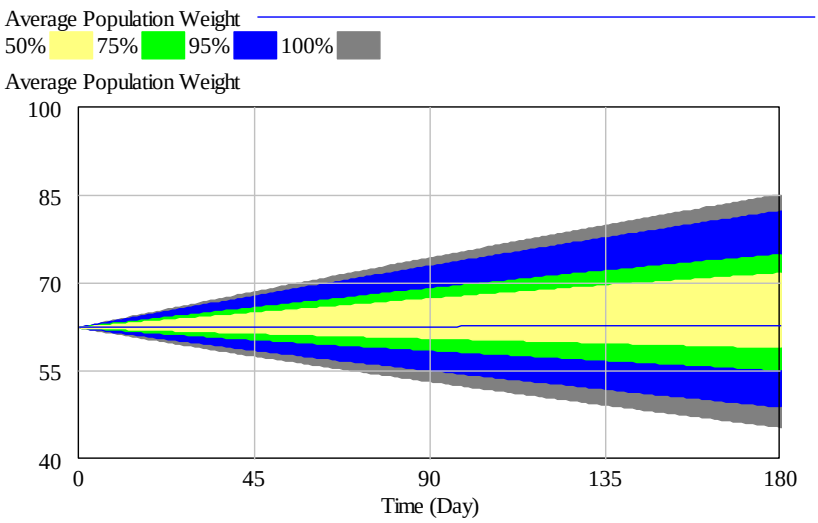

Figure 6-12 – EI & PAF Both Maximum

In Figure 6-12, both parameters are simulated between the largest range and variation, while keeping a constant mean. It can be seen that when additional variation is induced, it leads to more values of weight gain and weight loss by the individuals in a faster way.

The last three figures (6-13, 6-14 and 6-15) have been created by simultaneously simulating both the EI and PAF in different ranges or variation.

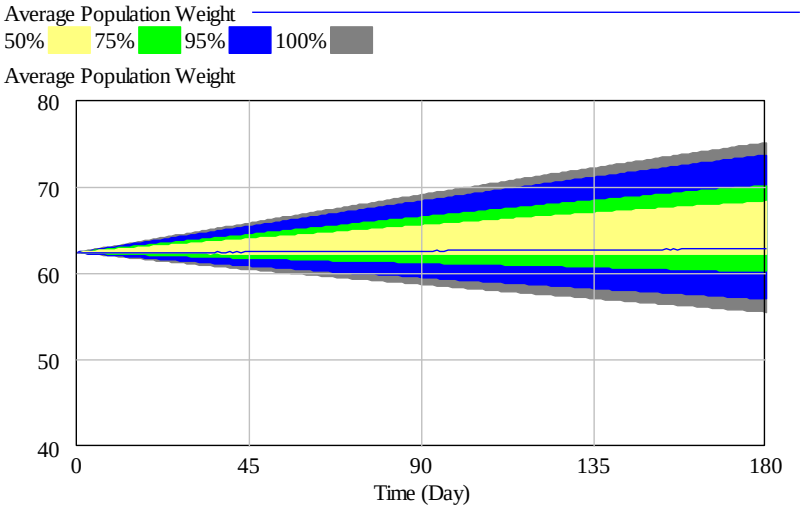

Figure 6-13 – EI & PAF Both Minimum

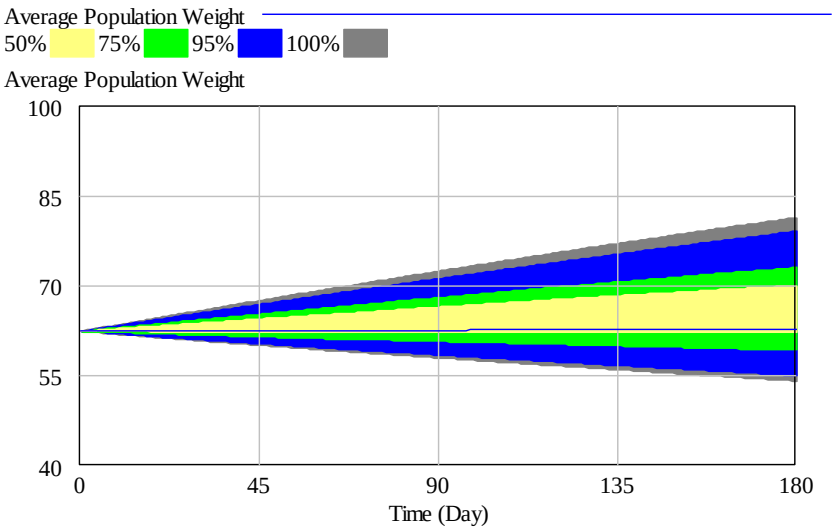

Figure 6-14 – EI & PAF Minimum Variation

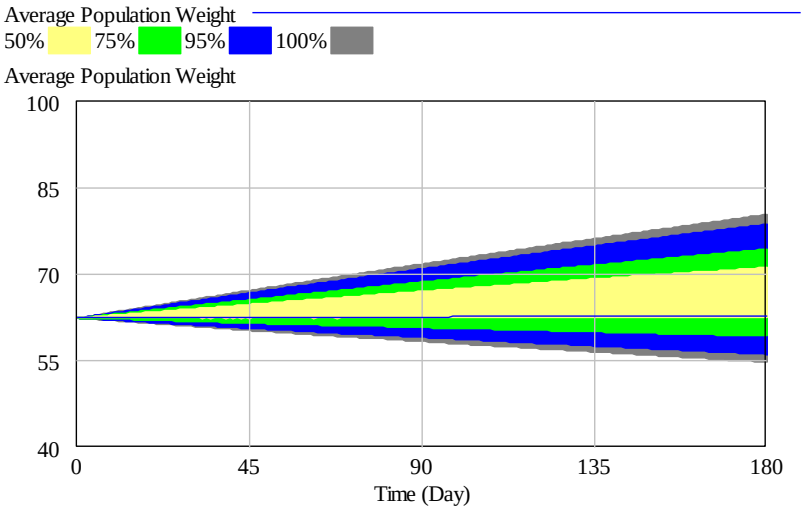

Figure 6-15 – EI & PAF Minimum Range

CHAPTER 7

LIMITATIONS AND FUTURE WORK

- In the presented model, many significant factors were not considered in the final solution. Particularly, the Non-physical factors such as socio-economics status, education, individual's living habitat, stress, social network, etc. are not included. While some of these elements play a significant role in the process of weight gain/loss. Including this set of factors can be a future research effort.
- To maximize the impact of this methodological innovation in future research, computational challenges in the estimation of process should be considered (Rahmandad & Sabounchi, 2011).
- The amount of data was available from the Medical Weight Loss Clinic was limited. Identifying other public sources of data, designing experiments and developing a research specific database is the goal of a future work.
- Obesity is a complex, multifactorial disease that not only has a significant impact on physical health but also on psychosocial well-being and therefore on quality of life. Obese people experience considerable impairments in quality of life as a consequence of their weight, and these can impact significantly their mental health, which in turn can further affect on their physical health. It is not yet clear how quality of life differs among different subsets of weight and genders, ethnicities etc., but the literature is growing and should be incorporated into future versions of the obesity model (McPherson, Marsh, & Brown, 2013)

Appendix A

Table A-1 - Weekly recording of the actual and simulated weight for a 39 year-old male who is 160 cm tall

Week	EI (MJ/d)	Actual Weight (kg)	Simulated Weight (kg)
0	2.092	141.5	141.5
1	2.092	139.72	139.72
2	2.092	137.97	137.97
3	2.092	136.23	136.23
4	2.092	134.51	134.51
5	2.092	132.81	132.81
6	2.092	131.13	131.13
7	3.3472	129.62	129.62
8	3.3472	128.14	128.14
9	3.3472	126.68	126.68
10	4.184	125.33	125.33
11	4.184	124.01	124.01
12	4.184	122.7	122.7
13	2.092	123.17	123.17
14	2.092	119.62	119.62
15	2.092	118.08	118.08
16	2.092	116.56	116.56
17	2.092	115.06	115.06
18	2.092	113.57	113.57
18	2.092	112.1	112.1

Table A-2 – Residual output for a standard male

Observation	*Predicted W*	*Residuals*	*Observation*	*Predicted W*	*Residuals*
1	74.778	-0.018	22	72.281	-0.001
2	75.903	-0.013	23	73.406	-0.006
3	77.028	0.002	24	74.532	-0.002
4	78.153	0.007	25	75.657	-0.007
5	79.278	0.022	26	76.782	-0.012
6	80.404	0.026	27	77.907	-0.007
7	81.529	0.031	28	79.032	-0.012
8	73.946	-0.016	29	71.449	0.011
9	75.071	-0.011	30	72.574	0.006
10	76.196	-0.006	31	73.699	0.001
11	77.321	-0.001	32	74.825	-0.005
12	78.446	0.004	33	75.950	-0.010
13	79.571	0.009	34	77.075	-0.015
14	80.697	0.013	35	78.200	-0.020
15	73.113	-0.013	36	70.617	0.033
16	74.239	-0.009	37	71.742	0.028
17	75.364	-0.004	38	72.867	0.023
18	76.489	-0.009	39	73.992	0.008
19	77.614	-0.004	40	75.118	0.002
20	78.739	0.001	41	76.243	-0.003
21	79.864	-0.004	42	77.368	-0.018

Table A-3 – Probability output for a standard male

Percentile	Weight
1.190	70.650
3.571	71.460
5.952	71.770
8.333	72.280
10.714	72.580
13.095	72.890
15.476	73.100
17.857	73.400
20.238	73.700
22.619	73.930
25.000	74.000
27.381	74.230
29.762	74.530
32.143	74.760
34.524	74.820
36.905	75.060
39.286	75.120
41.667	75.360
44.048	75.650
46.429	75.890
48.810	75.940
51.190	76.190
53.571	76.240
55.952	76.480
58.333	76.770
60.714	77.030
63.095	77.060
65.476	77.320
67.857	77.350
70.238	77.610
72.619	77.900
75.000	78.160
77.381	78.180
79.762	78.450
82.143	78.740
84.524	79.020
86.905	79.300
89.286	79.580
91.667	79.860
94.048	80.430
96.429	80.710
98.810	81.560

Table A-4 - Residual output for a standard female

Observation	*Predicted W*	*Residuals*	*Observation*	*Predicted W*	*Residuals*
1	60.609	-0.009	22	58.828	0.002
2	61.644	-0.004	23	59.862	-0.002
3	62.678	0.002	24	60.896	-0.006
4	63.712	0.008	25	61.931	-0.001
5	64.746	0.014	26	62.965	-0.005
6	65.781	0.019	27	63.999	-0.009
7	66.815	0.025	28	65.034	-0.004
8	60.015	-0.015	29	58.234	0.006
9	61.050	-0.010	30	59.268	0.002
10	62.084	-0.004	31	60.303	-0.003
11	63.118	0.012	32	61.337	0.003
12	64.153	0.007	33	62.371	-0.001
13	65.187	-0.037	34	63.405	-0.005
14	66.221	0.009	35	64.440	-0.010
15	59.422	-0.012	36	57.640	0.020
16	60.456	-0.006	37	58.675	0.015
17	61.490	0.000	38	59.709	0.011
18	62.525	-0.005	39	60.743	0.007
19	63.559	0.001	40	61.777	0.003
20	64.593	-0.003	41	62.812	-0.002
21	65.627	0.003	42	63.846	-0.016

Table A-5 - Probability output for a standard female

Percentile	Weight
1.19048	57.66
3.57143	58.24
5.95238	58.69
8.33333	58.83
10.7143	59.27
13.0952	59.41
15.4762	59.72
17.8571	59.86
20.2381	60
22.619	60.3
25	60.45
27.381	60.6
29.7619	60.75
32.1429	60.89
34.5238	61.04
36.9048	61.34
39.2857	61.49
41.6667	61.64
44.0476	61.78
46.4286	61.93
48.8095	62.08
51.1905	62.37
53.5714	62.52
55.9524	62.68
58.3333	62.81
60.7143	62.96
63.0952	63.13
65.4762	63.4
67.8571	63.56
70.2381	63.72
72.619	63.83
75	63.99
77.381	64.16
79.7619	64.43
82.1429	64.59
84.5238	64.76
86.9048	65.03
89.2857	65.15
91.6667	65.63
94.0476	65.8

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