

THE PENNSYLVANIA STATE UNIVERSITY  
SCHREYER HONORS COLLEGE

DEPARTMENT OF MECHANICAL ENGINEERING

DYNAMIC MODELING OF FOOD TO FUEL CONVERSION AND ITS EFFECT ON  
INDIVIDUAL WEIGHT GAIN

BRADFORD PECHIN  
SUMMER 2017

A thesis  
submitted in partial fulfillment  
of the requirements  
for a baccalaureate degree  
in Mechanical Engineering  
with honors in Mechanical Engineering

Reviewed and approved\* by the following:

Kathleen Keller  
Professor of Nutrition  
Thesis Supervisor

Sean Brennan  
Professor of Mechanical Engineering  
Honors Adviser

Jacqueline O'Connor  
Assistant Professor of Mechanical Engineering  
Faculty Reader

\* Signatures are on file in the Schreyer Honors College.

## ABSTRACT

Obesity is an impactful issue across the globe, especially in America, and a simple explanation for the increase in obesity rates is that people are eating more. However, this thesis hypothesizes that caloric intake alone does not fully explain differences in weight gain due to food type. Americans' diets have changed in recent decades, and historical changes in the types of food consumed appear to relate to trends in obesity. In particular, the increase in consumption of carbohydrates is notable. This thesis examines very simple models of human digestion to explore the possible connection between consumed carbohydrates and weight gain due to rapid digestion of them by the body.

To explore this potential relation, the body's uptake of nutrients from consumed food is modeled as a first order low-pass filter, with varying time constants for the uptake dependent on the food type. Food mass is modeled as eating events where the digestive process preserves input/output mass flow, modeling input and output of mass as a time-delayed effect. It is assumed as well that the individual strives to maintain homeostasis by associating hunger with instantaneous caloric deficit at the time of food ingestion, eating a quantity of food proportional to perceived hunger. The model includes the known effect that metabolic detection of caloric content during eating is delayed by a very small amount of time. It also includes a first-order dynamic model of the body's conversion of excess food energy into fat, as well as a similar model for the conversion of body fat into energy for cellular use.

Via this simulation, different food inputs are tested with different digestive rates to compare their effects on potential weight gain and loss. The developed model exhibits strong variation in bodily response to different food types, based particularly on the different

magnitudes of instantaneous available energy. These examples lend strength to the thesis's hypothesis by showing how differently the body reacts to different types of foods, with the goal to bring awareness to the potential weight-gain consequences of sugar and similarly rapidly-digested foods.

## TABLE OF CONTENTS

|   |     |
|---|-----|
| LIST OF FIGURES .....                                   | v   |
| LIST OF TABLES .....                                    | vii |
| Chapter 1 Introduction and Overview of the Thesis ..... | 1   |
| Hypothesis.....   | 1   |
| Societal Relevance .....                                | 5   |
| Outline of Remaining Chapters.....                      | 6   |
| Chapter 2 Literature Review .....                       | 8   |
| Obesity and its Consequences.....                       | 8   |
| Causes of Obesity.....                                  | 13  |
| Macronutrients .....                                    | 15  |
| Diet Trends.....  | 17  |
| Chapter 3 Governing Equations and Relations .....       | 21  |
| Model Construction.....                                 | 21  |
| Subsystem Dynamics .....                                | 24  |
| Metabolism.....   | 24  |
| Mass Dynamics. ....                                     | 29  |
| B.M.R. ....   | 31  |
| Voluntary Choice .....                                  | 33  |
| Implementation .....                                    | 38  |
| Chapter 4 Discussion and Model Validation .....         | 40  |
| Starvation Test .....                                   | 40  |
| Fiber Test .....  | 43  |
| Equilibrium Test .....                                  | 46  |
| Hunger Test.....  | 48  |
| Chapter 5 Results and Conclusions.....                  | 52  |
| Test Results .....                                      | 52  |
| Model Faults .....                                      | 59  |
| Conclusions.....  | 62  |
| Appendix: Simulation Code.....                          | 64  |
| BIBLIOGRAPHY .....                                      | 67  |

## LIST OF FIGURES

|  |    |
|--|----|
| Figure 1: Leading causes of death among Americans (Obesity Information, 2016) .....  | 9  |
| Figure 2: Obesity growth over time based on different BMI ranks (Sturm & Andreyeva, 2004) .....  | 12 |
| Figure 3: Macronutrient trends in American diets over time (Ogden, 2014).....  | 19 |
| Figure 4: Macronutrient Distribution of the caloric increase in American diets (Ogden, 2014) .....   | 20 |
| Figure 5: Constructed Model via Simulink.....  | 22 |
| Figure 6: Inside the Metabolism block .....  | 25 |
| Figure 7: Mass digestion test .....  | 28 |
| Figure 8: Inside Mass Dynamics block.....  | 29 |
| Figure 9: Inside BMR block .....   | 31 |
| Figure 10: Inside Voluntary Choice block.....  | 33 |
| Figure 11: hunger multiplier for fat .....   | 35 |
| Figure 12: Mass vs. time for starvation test.....  | 42 |
| Figure 13: fiber test – mass flow rate of poop (kg per hour) vs. time; demonstrates pooping events repeat and work effectively .....                           | 44 |
| Figure 14: fiber test –body mass (kg) vs time; verification that excretion events do not influence mass change in the long run .....                           | 45 |
| Figure 15: Equilibrium test – mass (kg) vs time; proves effectiveness of metabolism simulation by maintaining mass in a time of no net calories .....          | 47 |
| Figure 16: hunger test for quantity approach; plots mass vs time to demonstrate effect on mass of hunger multiplier that measures quantity of energy debt..... | 49 |
| Figure 17: hunger test for rate approach; plots mass vs time to demonstrate effect on mass of hunger multiplier that measures rate of energy debt.....         | 50 |
| Figure 18: mass vs time for equilibrium carbohydrate input – solely carbohydrate diet on diet equal to calories of BMR .....                                   | 53 |
| Figure 19: mass vs time for equilibrium fat input – solely fat diet on diet equal to calories of BMR.....  | 54 |
| Figure 20: mass vs time for equilibrium protein input – solely protein diet on diet equal to calories of BMR .....   | 55 |

|   |    |
|---|----|
| Figure 21: mass vs time for equilibrium, half fat/half carbs; diet with BMR calories comprised of half fat and half carbs .....         | 56 |
| Figure 22: mass vs time for equilibrium, half fat/half protein diet with BMR calories comprised of half fat and half protein .....      | 57 |
| Figure 23: mass vs time for equilibrium, half protein/half carbs; diet with BMR calories comprised of half protein and half carbs ..... | 58 |

**LIST OF TABLES**

|  |    |
|--|----|
| Table 1: potential effects caused by obesity (Kopelman, 2007) .....                | 10 |
| Table 2: BMI Classifications and corresponding rank (Garrow & Webster, 1985).....  | 11 |
| Table 3: Macronutrients and their respective energy contents (Buchholz, 2004)..... | 15 |

## **Chapter 1**

### **Introduction and Overview of the Thesis**

Obesity is an impactful issue across the globe, especially in America, and a simple explanation for the increase in obesity rates is that people are eating more. However, this thesis hypothesizes that caloric intake alone does not fully explain differences in weight gain due to food type. American's diets have changed in recent decades, and historical changes in the types of food consumed appear to relate to trends in obesity. In particular, the increase in consumption of carbohydrates, especially refined sugar, is notable (Bentley & Kantor, 2016). This thesis examines very simple models of human digestion to explore the possible connection between consumed sugar and weight gain due to the rapid digestion of sugars by the body.

### **Hypothesis**

According to a study published by the American Journal of Public Health, "...there is a need for more integrated and quantitative modeling that can account for dynamic feedback" within the human body, and that there have been very few modeling efforts that use this type of analysis (Hall, Hammond & Rahmandad, 2014). There is a particular lack of dynamic models, i.e. simulations that include the differential equations describing the time evolution of nutrient uptake and usage within the body. This need for new models, coupled with increasing weight problems in our society, has inspired the goal of this thesis: to model the body dynamically in an effort to demonstrate the effects of food choices on weight gain.



The thesis that follows provides details of this modeling effort, but a summary is as follows: to explore this potential relation, the body's uptake of nutrients from consumed food is modeled as a first order low-pass filter with varying time constants for the uptake dependent on the food type. Food mass is modeled as eating events where the digestive process preserves input/output mass flow, modeling input and output of mass as a time-delayed effect. It is assumed as well that the individual strives to maintain homeostasis by associating hunger with instantaneous caloric deficit at the time of food ingestion, eating a quantity of food proportional to perceived hunger. The model includes the known effect that metabolic detection of caloric content during eating is delayed by a very small amount of time. It also includes a first-order dynamic model of the body's conversion of excess food energy into fat, as well as a similar model for the conversion of body fat into energy for cellular use.

A low-pass filter is a specific form of a differential equation that allows passage of low frequency signals and alters those of high frequency, while preserving the accumulated energy content of the signal. The name "low pass" arises for this very common equation because low frequency signals that are introduced as inputs to the equation are allowed to pass through unchanged, while any signal introduced with a frequency that is deemed too high, or past the set cutoff point, will be attenuated before it is allowed to pass. Low-pass filters are pervasive in human-build devices such as electrical circuits and mechanical systems, but they are also pervasive in natural systems as well.

The human body, in the simplest model of the time-dynamics of nutrient uptake, also acts as a low-pass filter in an effort to maintain homeostasis. Specifically, the body cannot instantly uptake ingested calories and thus must require time after ingestion to convert a fast signal (eating) into available energy. It is assumed in this thesis that the digestive process is the primary

mechanism for this low-pass filtering effect, that regardless of energy deficit, humans cannot significantly increase or decrease the nutrient uptake rate of food. The need for rapid energy intake in times of starvation, in spite of fixed digestive processes, is likely what dispossesses humans to the flavor of simple sugars

Homeostasis in humans refers to the body's ability to regulate its physiological environment to ensure its stability in response to fluctuations in the outside environment (Rosenbaum and Liebel. 2016). Homeostasis is important for our survival, as it helps maintain the proper environmental balance within the body to ensure available energy for organ function and bodily activity. “For instance, heart failure may occur when negative feedback mechanisms become overwhelmed and destructive positive feedback mechanisms take over,” (Marieb, Nicpon & Hoehn, 2007). In the release of energy into the bloodstream from fat and other stored energy sources, the body again exhibits behavior like a low pass filter. It aims to prevent any introduced signal or influence, a sudden exercise event for example, from altering the body to a point causing organ or activity failure. If a system within the body reaches a point past this cutoff, homeostasis mechanisms seek to return the body to its normal state. For example, in the presence of caloric debt, the body may signal an unwillingness to continue a strenuous activity, at least not at the intensity that would be normal without caloric debt. This process is analogous to how a low-pass filter attenuates a signal that is faster than a low-pass filter's cutoff frequency.

Most systems within the human body use feedback to self-regulate body activity. Examples include the digestive system and those linked to it that involve the methods of dealing with and processing any ingested food. After the consumption of food, the digestive process extracts energy which then circulates throughout the body in the form of blood sugar, or glucose in the blood. “The liver acts as the body's glucose (or fuel) reservoir, and helps to keep your

circulating blood sugar levels and other body fuels steady and constant,” (UCSF Medical Center, 2007). The liver in this case is acting as a low pass filter that aims to keep any introduced fluctuating signal at a lower, constant level. Or to restate this effect: the liver tries to keep blood sugar levels “steady and constant,” aiming to prevent them from deviating too far from the desired level. It is for these reasons that this thesis uses a low-pass filter model to represent the body, as it is a simple yet descriptive model for the body’s functions and efforts to maintain homeostasis. Further, numerical simulations allow straightforward implementation of such filters.

The simulations in this thesis are developed to explain how different types of foods cause different weight gain effects on individuals. According to the National Institute of Diabetes and Digestive Kidney Diseases, the rate at which the food/calories are digested does influence how the food is digested (Wallace, 2013). The food can either be used as energy, or stored for later use via conversion to fat. It is a hypothesis of this thesis that this buildup of fat could be a potential cause for obesity.

The rate of digestion contributes to weight change in that it has influence on how soon an individual will feel hungry after they have eaten a meal. According to the International Journal of Obesity, “It is likely that short-term satiety signals, activated following the intake of a meal and emanating largely from the gut, improve digestive efficiency, nutrient utilization and prevent large fluctuations in circulating nutrients by preventing consumption of large meals.” (Hussain and Bloom, 2012). Essentially, the feeling of fullness after eating a meal has digestive and health benefits, as well as preventing deviations via from homeostasis via nutrient circulation, like blood sugar, and eating bigger meals. Because increased caloric intake is likely the main factor in weight gain, it would follow that a lasting feeling of fullness that reduces caloric intake

has favorable weight effects. Based on this hypothesis, foods that provide slower release of calories may then prevent weight gain.

Via simulations, this thesis tests the idea that caloric release rates might affect weight gain. Different food inputs are simulated with different digestive rates to compare their effects on hunger response, which in turn affects weight gain and loss. The developed model exhibits strong variation in bodily response to different food types, based particularly on the different magnitudes of instantaneous available energy. These examples lend strength to the thesis's hypothesis by showing how differently the body reacts to different types of foods, with the goal to bring awareness to the potential weight-gain consequences of sugar and similarly rapidly-digested foods.

### **Societal Relevance**

In an investigation conducted by the American Medical Association, it was found that more than one-third of adults and 17% of youth are obese, and that that number has been rising ever since (Ogden, 2012). The study also deemed that “obesity prevalence remains high and thus it is important to continue surveillance,” as it is a serious and growing issue in today's world

Obesity can be a negative health condition considering its vast list of accompanying ailments (Ogden, Carroll, Kit & Flegal, 2012), such as heart disease, diabetes, high cholesterol, and high blood pressure. Furthermore, the prevalence of severe obesity in children and adolescents seems to be increasing, (Flegal, Graubard, Williamson & Gail, 2005) indicating not only that obesity will continue to be problem in the future as children become adults, but that the

magnitude of obesity is also growing, meaning more health problems in general and with greater intensity.

### **Outline of Remaining Chapters**

The remainder of this thesis is organized into four more chapters, each building upon the hypothesis and relating back to its claim. The chapters are as follows: Literature Review, Governing Equations and Relations, Discussion and Model Validation, and Results and Conclusions. In Chapter 2, the Literature Review, the thesis reviews research done on the topics of this thesis, such as obesity and the rate of digestion of different foods. While these topics have already been briefly mentioned, they will be discussed in more detail as to paint a better picture of the national obesity situation, as well as what causes may be behind the undesirable condition and the science behind them. This discussion shows awareness in the literature of the core ideas of this thesis, and provides additional support to the hypothesis and the reasoning behind the simulation developed in the chapters that follow.

The next chapter, Governing Equations and Relations, discusses each equation used in the model and the reasoning behind it, as well as their origin. Furthermore, it provides detail about the model itself and how it was constructed, explaining the model's organization and the chosen software used to build it and run the simulation. By doing this, readers may better understand the relationship of the model to bodily functions, making the parallels between the two more clear and giving more credibility to the technological representation of living human biology.

Chapter 4, called Discussion and Model Validation, explains in detail the model's ability to predict known outcomes. Using the model, validation tests demonstrate the model's ability to replicate factual results under situations commonly used for metabolic testing. These results seek to establish the simulation's fidelity prior to using it to predict outcomes for the experiment at hand.

Lastly, the fifth chapter, Results and Conclusions, discusses the primary results of the experiment. Discussions compare the simulation results to the hypothesis. A summary of the primary results concludes the thesis, including areas that need more time, effort, and evaluation.

## **Chapter 2**

### **Literature Review**

Obesity is a problem that afflicts a large percentage of people, and can be caused by a variety of factors (Kopelman 2007). Not only is there societal pressure to avoid this condition, but there are many adverse health effects associated with it as well (Kopelman, 2007). Though there are multiple avenues that lead to obesity (Hall, Hammond & Rahmandad, 2014), this thesis focuses on the connection between the digestion rates of one's food choices and the weight gain that results from it. Faster-digesting foods have become more popular in recent history (Bentley & Kantor, 2016), and during nearly the same exact timeframe, obesity spiked as well (Sturm & Andreyeva, 2004). It is very possible these two phenomena are connected, and the details are discussed in the following sections.

### **Obesity and its Consequences**

Obesity is defined as excess adipose tissue, or a vast surplus of fat (Olsen & Board, 2014, identified from a certain BMI classification, which are explained in paragraphs to come. Excess fat is dangerous to the body for many reasons, such as the inhibition of organ function and increases stress on the heart, increasing one's risk for heart disease (Kopelman, 2007). Aside from cancer, heart disease kills nearly three times more Americans than anything else, and is the leading killer of Americans overall (Obesity Information, 2016). With heart disease as one of its consequences, obesity could be considered a dangerous condition.

## Leading Causes of Death

Percentage by Subgroup

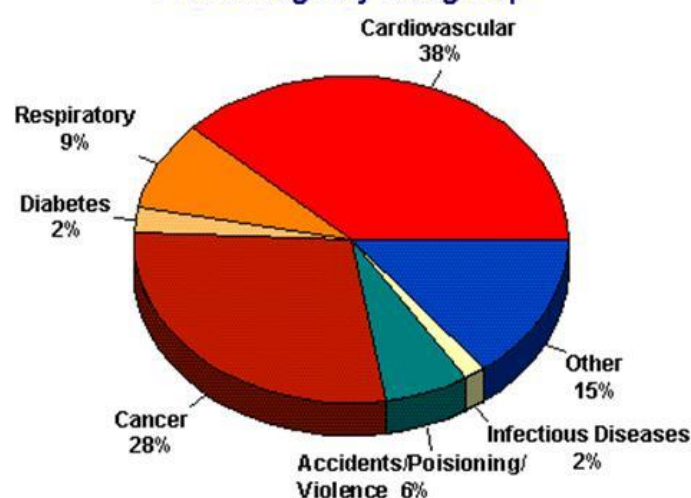


Figure 1: Leading causes of death among Americans (Obesity Information, 2016)

Obesity puts an individual at additional risk for a variety of ailments, including high blood pressure, high blood cholesterol, and diabetes (Kopelman, 2007). More specifically, bad cholesterol is raised and good cholesterol is lowered (Kopelman, 2007). This effect, along with the increase in blood pressure, both increase someone's likelihood to experience a stroke or develop heart disease (Kopelman, 2007). Therefore, obesity severely endangers the heart, straining it to work harder to pump blood through clogged inflamed arteries (Kopelman, 2007). With such dire consequences and a recent upward trend, obesity has become a great concern for the United States. A table of various obesity symptoms (Kopelman 2007) can be observed in Table 1.



**Table 1: potential effects caused by obesity (Kopelman, 2007)**

|  |   |
|--|---|
| Metabolic syndrome                       | 30% of middle-aged people in developed countries have features of metabolic syndrome  |
| Type 2 diabetes                          | 90% of type 2 diabetics have a body mass index (BMI) of $>23 \text{ kg m}^{-2}$   |
| Hypertension                             | 5× risk in obesity<br><br>66% of hypertension is linked to excess weight<br><br>85% of hypertension is associated with a BMI $>25 \text{ kg m}^{-2}$  |
| Coronary artery disease (CAD) and stroke | 3.6× risk of CAD for each unit change in BMI<br><br>Dyslipidaemia progressively develops as BMI increases from $21 \text{ kg m}^{-2}$ with rise in small particle low-density lipoprotein<br><br>70% of obese women with hypertension have left ventricular hypertrophy<br><br>Obesity is a contributing factor to cardiac failure in $>10\%$ of patients<br><br>Overweight/obesity plus hypertension is associated with increased risk of ischaemic stroke |
| Respiratory effects                      | Neck circumference of $>43 \text{ cm}$ in men and $>40.5 \text{ cm}$ in women is associated with obstructive sleep apnoea, daytime somnolence and development of pulmonary hypertension   |
| Cancers                                  | 10% of all cancer deaths among non-smokers are related to obesity (30% of endometrial cancers)  |
| Reproductive function                    | 6% of primary infertility in women is attributable to obesity<br><br>Impotency and infertility are frequently associated with obesity in men  |
| Osteoarthritis (OA)                      | Frequent association in the elderly with increasing body weight – risk of disability attributable to OA equal to heart disease and greater to any other medical disorder of the elderly   |
| Liver and gall bladder disease           | Overweight and obesity associated with non-alcoholic fatty liver disease and non-alcoholic steatohepatitis (NASH). 40% of NASH patients are obese; 20% have dyslipidaemia<br><br>3× risk of gall bladder disease in women with a BMI of $>32 \text{ kg m}^{-2}$ ; 7× risk if BMI of $>45 \text{ kg m}^{-2}$   |

Obesity can be determined by a variety of methods, but the most commonly referred to and relied upon method is the use of BMI, or body mass index, measurements (Ogden, Carroll, Kit & Flegal, 2014). BMI is defined as an individual's weight in kilograms divided by the square of the individual's height in meters (Ogden, Carroll, Kit & Flegal, 2014).

$$BMI = \frac{kg}{m^2} \quad (1)$$

Essentially, a higher BMI means that someone has more weight for each inch tall he or she is. Depending on an individual's body mass index, they are given a certain weight classification, with higher BMIs being classified as more and more overweight. The weight rankings and corresponding BMI ranges are shown in Table 2 (Garrow & Webster, 1985).

**Table 2: BMI Classifications and corresponding rank (Garrow & Webster, 1985)**

| Classification | BMI (kg/m <sup>2</sup> ) |
|----------------|--------------------------|
| Healthy weight | 18.5-24.9                |
| Overweight     | 25-29.9                  |
| Obesity I      | 30-34.9                  |
| Obesity II     | 35-39.9                  |
| Obesity III    | 40+                      |

Currently, obesity affects 35.7% of Americans – more than one third of the entire population (Wallace, 2014). Furthermore, “nearly 70% of American adults are either overweight or obese,” (Obesity Information, 2016) which shows that the condition is even more apparent as we age, and therefore will affect more people than may currently seem. The trend in obesity in America is also very concerning, as, in the recent history, it has been increasing year over year. The increase has so much magnitude, that now, over one third of Americans are obese. What is even *more* worrisome is the fact that the number of “clinically obese” individuals has followed the same growth pattern, but at an even greater rate of increase. The phrase “clinically obese” refers to the severely obese, usually those with a BMI greater than 40 or 50.

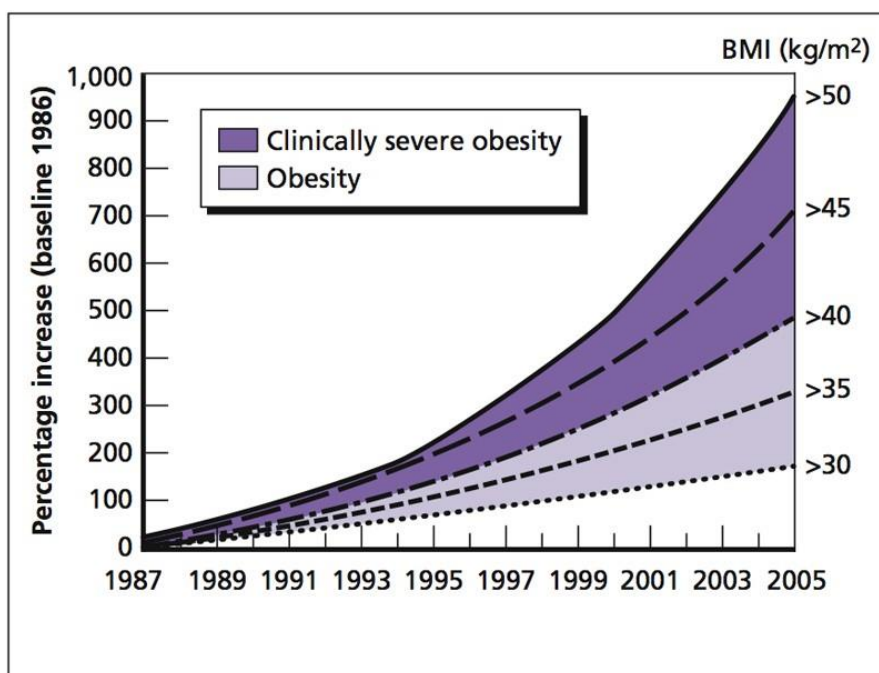


Figure 2: Obesity growth over time based on different BMI ranks (Sturm & Andreyeva, 2004)

As can be seen in Figure 3, obesity overall has more than doubled since 1987, which is very significant in terms of the population (Sturm & Andreyeva, 2004). Furthermore, it can be observed that the higher the body mass index classification, the faster the growth. The most shocking number is that of the BMI >50, where a 900% growth was seen. “Many physicians believe that clinically severe obesity is a rare pathological condition affecting only a fixed percentage of the population,” but the rapid growth disproves this theory, as it no longer affects a “few” individuals, and shows that severe obesity is an integral part of the United States’ population’s weight distribution (Sturm & Andreyeva, 2004).

A body mass index as high as the clinically obese classification is extremely dangerous (Kopelman, 2007) – 40 is the highest ranking for obesity – let alone the risk all the lesser classifications still entail by themselves alone. Severe obesity such as this is of far more concern than regular obesity, being responsible for a much longer list of ailments, most of which are

more serious progressions of conditions caused by lesser obesity. Along with worsening the conditions and individual's body is already subject to by being obese to begin with, morbid obesity causes things like osteoarthritis, reproductive problems, metabolic issues, some types of cancer, and even sleep apnea (Kopelman, 2007). The body struggles to operate with all of the excess fat people carry with morbid obesity, and it greatly accelerates the pace at which the body wears down (Finkelstein, Trogdon & Dietz, 2009).

Obesity and severe obesity are serious, growing problems in the United States, and they place a solid portion of the population in danger of health risks (Kopelman, 2007). With this knowledge, obesity (especially severe obesity) should be of major concern in terms of the health and longevity of the current population and generations of the future.

### **Causes of Obesity**

Obesity can be caused by a variety of factors, such as consuming excess calories on a consistent basis, lack of exercise, or simply poor genetics that make an individual predetermined to have a husky body type (also makes it harder for them to lose weight). However, the *real* determining factor is the presence of an energy imbalance. "Energy imbalances...are known to cause overweight or obesity," (Gibbons, 2017), and those who eat more often are more prone to such energy imbalances.

For the sake of this study, the focus will be on energy imbalances related to food consumption, rather than imbalances created through other situations, such as medicines and hormonal influences that cause the body to behave differently than it normally would. Since this thesis's focus considers the average American, a study done on diet would be more applicable,

because it better relates to the average population, rather than those with abnormally bodies. The goal of such assumptions is to make the results more encompassing and meaningful.

An energy imbalance, as in the world of thermodynamics, simply means that “...energy in does not equal energy out,” (Gibbons, 2017). When talking about food, the energy that this statement refers to is the energy content of the food itself, described as calories – another type of energy. To put a calorie in perspective, a Joule is equal to 0.239 calories (Rosen & Spiegelman, 2014). This type of calorie is abbreviated by “cal” and is, contrary to popular belief, not the actual unit used when describing a given food’s accessible or usable energy content. In fact, a “kilocalorie” is what is actually used on nutrition facts labels and is what is commonly referred to when the word “calorie” is mentioned, when it really represents one thousand calories. This term is subsequently abbreviated as “kcal” or just “Cal,” using a capital “C” to distinguish it from the smaller caloric unit, which uses a lower case letter. To help avoid confusion from a reader’s standpoint, from here on out and for the remaining duration of this study, kilocalorie will simply be referred to as “calorie”, not to be confused with its smaller counterpart.

“Calories are essential to human health; the key is taking in the right amount. Everyone requires different amounts of energy per day depending on age, size and activity levels,” (Vandevijvere & Swinburn, 2015). Clearly, consuming more calories than one uses within a given timeframe would result in an energy imbalance, with a certain net caloric gain. It is this excess number of calories that is responsible for weight gain involved in energy imbalances. However, as previously mentioned, it is more than just calories that can be responsible for unfavorable weight gain – rate of digestion of the food and its respective calories also has a hand in such effects, due to the lasting feeling of fullness that the said food provides. For instance, if one consumes a meal with a lot of fat in it (fat digests very slowly, as is discussed in the next

section), it will take him or her longer to digest the food, and therefore will feel fuller for a longer duration of time (Vandevijvere & Swinburn, 2015). A quicker-digesting food is absorbed speedily, causing the individual to regain the feeling of hunger sooner after eating is finished (UCSF Medical, 2007). More hunger means more consumption, which leads to more calories and a greater chance of creating an energy imbalance. Different foods are handled differently by the body, and therefore digest at different rates (Marieb, Nicpon & Hoehn, 2007). This variation mostly lies in the classification of the food, or what macronutrients the food is composed of, explained in the following section.

## Macronutrients

If one is to study food's effects on the body based on digestion rate, it must be made known the food's constituents' effects on said rate. There are four general types of food, called macronutrients: fats, proteins, carbohydrates, and alcohols – each of which has a different energy content, which are outlined in Table 3 (Buchholz, 2004).

**Table 3: Macronutrients and their respective energy contents (Buchholz, 2004)**

| Macronutrient | Calorie Content (calories/gram) |
|---------------|---------------------------------|
| Fat           | 9                               |
| Carbohydrate  | 4                               |
| Protein       | 4                               |
| Alcohol       | 7                               |

While protein and carbohydrate contain four calories per gram each, fat has nine calories for every gram (Buchholz, 2004), making consumption of fat more apt to pushing people above their caloric equilibrium and into an energy imbalance, causing them to gain weight if the imbalance is maintained. Yet, the macronutrients' difference in caloric content is not the only significant detail that influences weight – the other is each macro's rate of digestion, or how fast the body can convert the food into usable energy for bodily functions (Buchholz, 2004).

As is commonly known, whenever a person eats or drinks something, it is broken down via the process of digestion. This process begins in the mouth, making its way into the stomach, through the intestines, and ends in the toilet. Digestion is "...breaking down food into nutrients which the body uses for energy, growth, and cell repair," (Wallace, 2013). To facilitate nutrient absorption, the food is broken down or changed into smaller molecules (Dietary Guidelines, 2015). "The body breaks down nutrients from food and drink into carbohydrates, protein, fats, and vitamins," (Dietary Guidelines, 2015). Since vitamins have no caloric value (Buchholz, 2004) and do not contribute to food-related energy imbalance, they will not be considered here.

When individual consume food, his or her digestive tract works to break it into smaller pieces both physically, through mechanical motions, and chemically, by using digestive enzymes (Marieb, Nicpon & Hoehn, 2007). The mechanical breakdown of different food occurs at relatively consistent rates, but different foods chemically digest at very different rates (Hur, Lim, Decker & McLements, 2010). This difference in chemical breakdown speed among macronutrient may play a key role in weight gain due to the feeling of fullness it provides, which is related to the pace of energy absorption of the food. The resultant energy balance can be different depending on how much energy is introduced at once from the food. This entails that the pace at which energy is extracted from what we consume via digestion may be important in

determining whether or not an unfavorable energy imbalance, and whether or not that individual gains weight or not, due to its effect on signaling hunger on a timely basis (Shin & Berthoud, 2009). This rate-dependent phenomenon is commonly overlooked because it is not printed on standard nutrition labels. Only the caloric content of the food is expressed, giving the impression that all types of calories are treated the same by our bodies.

This discrepancy lies in the different enzymes that are used to digest the different macronutrients. Amylase is the enzyme that breaks down carbohydrates, protease digests proteins, and lipase is the enzyme for fats. These enzymes are released at different locations, and therefore have different acting times on the foods they break down (Marieb, Nicpon & Hoehn, 2007). Amylase is contained in saliva, causing the digestion of carbohydrates to begin essentially as soon as an individual begins chewing. With chewing being the first step in the digestion process, this enzyme has the most time to engage its respective macronutrient, hence carbohydrates being digested most rapidly. Protease is released in the stomach, triggering protein's digestion once the food reaches that location. In general, protein digests the second fastest because fat is not digested until it reaches the intestines. Due to its inability to mix with water, and the fact that our stomach acid is dissolved in water, fat cannot be digested in the stomach – it simply floats on top of the acidic liquid until it passes into the small intestines, where lipase enters the picture and begins breaking it down (Marieb, Nicpon & Hoehn, 2007).

### **Diet Trends**

This study aims to model and demonstrate the effects of weight gain from different ratios of macronutrients in a diet due to the variation in their respective digestion rates. Interest was



lent to this topic upon finding evidence that obesity may have been correlated to this phenomenon (Jonnalagadda, 2011), in that recent diet trends among Americans reflected more fast-digesting foods. Even more curiously, these trends occurred near the same timeframe as the increase in national obesity (Bentley & Kantor, 2016), making it seem even more like the trends may have played a part in making our nation an overall heavier one.

As discussed earlier in this thesis, obesity began to surface in the American population during the 1980s, and continued to increase until present day, doing so at a significant rate (Sturm & Andreyeva, 2004). Coincidentally enough, during roughly the same timeframe certain diet changes among Americans also took place that reflect a tendency for extra weight gain. Starting around the same time, there has been an increase in carbohydrate intake among Americans (Bentley & Kantor, 2016). As carbohydrates digest the fastest of all the macronutrients, when considering digestion rate, they have the greatest potential in leading to excess weight gain among their consumers (Jonnalagadda, 2011).

As previously mentioned, foods that digest more quickly leave the consumer feeling full for a shorter amount of time, causing them to feel hungry more quickly after eating (Hur, Jin, Decker & McLements, 2010). This effect would often lead to extra eating, as the general population eats when they are hungry. So, if an increase in this sort of foods took place, then it would be plausible, if not probable, that an increase in weight gain would also take place. As the time periods seem to match up, even more strength is lent to this claim, giving potential to a relationship between the occurrences of these two phenomena.

Referring to Figure 3, obesity can be seen to begin rising among American citizens in the year 1980, increasing up until now (Sturm & Andreyeva, 2004). Furthermore, referring to

Figure 1, the same trend can be observed. In Figure 5, it can be observed that a significant change in macronutrient intake took place around roughly the same time.

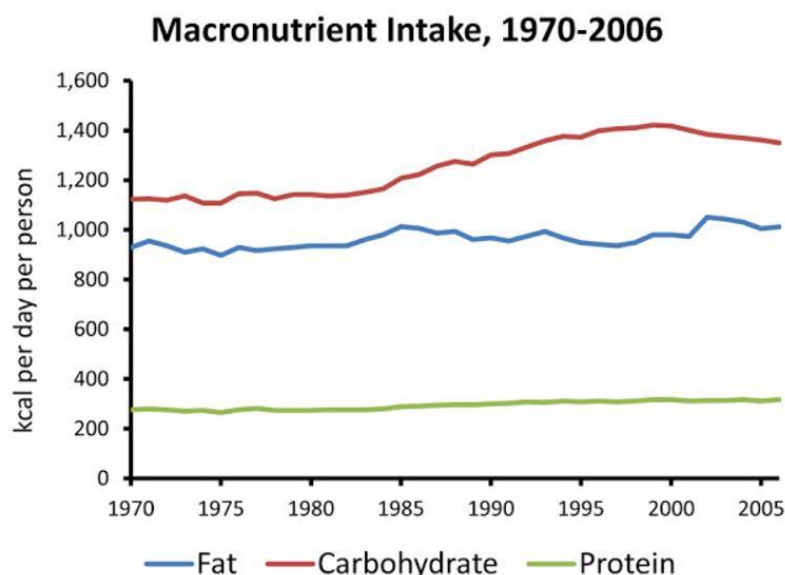


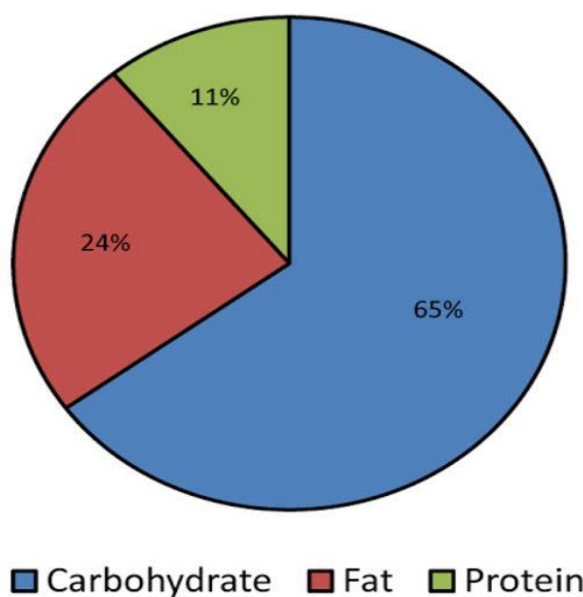
Figure 3: Macronutrient trends in American diets over time (Ogden, 2014)

In roughly the year 1980, Americans began consuming more calories overall, as the lines for fat, protein, and carbohydrate all rose up the chart (Ogden, 2014). However, it is not only the uptake in calories that is significant here – the sharp increase in carbohydrates is also important to note. The increase in this macronutrient is of much more magnitude than the increase in fat (protein stayed relatively constant, if not a very slight increase). As this is the macronutrient that leads to more frequent pangs of hunger, it would follow that an increased consumption of it would lead to more overall eating, perpetuating the trend of eating more calories, as well as increasing the amount of obesity seen among Americans (Jonnalagadda, 2011).

In regards to the overall caloric increase in American diets, Figure 6 details the exact breakdown of said caloric increase in terms of the macronutrients that composed it (Ogden,

2014). As can be seen in Figure 6, nearly two-thirds of the increase in calories was comprised of carbohydrates.

**Contribution of Macronutrients to Calorie Increase Since 1970 (USDA)**



**Figure 4: Macronutrient Distribution of the caloric increase in American diets (Ogden, 2014)**

With such a large portion of the additional food introduced in American diets being faster-digesting foods, it would make sense that obesity numbers would increase. Though there is no proof of the existence of such a relationship, it logically complies that weight gain would arise as people would be eating foods that cause them to be hungry more often. This situation would also create somewhat of a vicious cycle, as Americans would consume more calories, mostly from carbohydrates, become hungry quickly and eat again, only to consume more carbohydrate-dense calories, and this could lead to increased obesity.

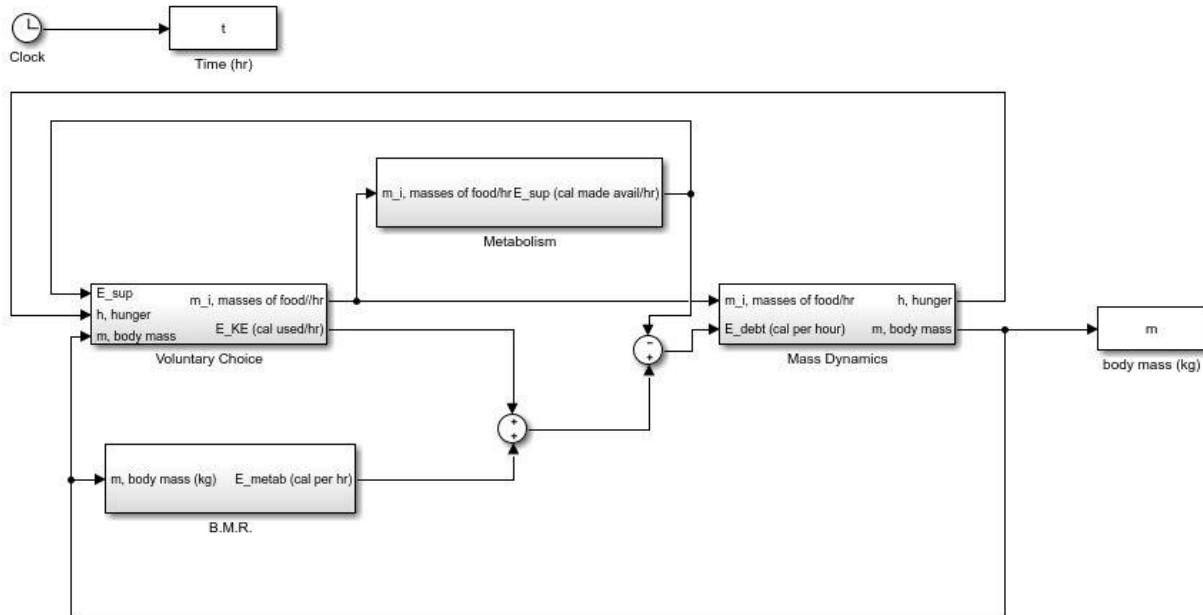
## **Chapter 3**

### **Governing Equations and Relations**

This thesis takes an engineering approach in dissecting a nutritional phenomenon and describing digestion. More specifically, it attempts to demonstrate the effects of food on weight gain by modeling the body and the digestive process via a feedback loop using a low-pass filter, as previously discussed. To do this, various different equations and modeling techniques are deployed, differing from block to block depending on what each system is modeled. When put together in a feedback loop, the mathematical operations all interact in time together in a process that seeks to resemble how the human body deals with ingested food, and how that ingested food causes mass changes in an individual.

### **Model Construction**

As previously mentioned, the model uses a feedback loop in order to represent and replicate the body and the process of food digestion. The simulation is implemented with the MATLAB software suite, using the Simulink subroutines. Simulink is a graphical programming environment that allows various representations of interacting differential equations. The program uses graphical blocks to build diagrams representing each equation and their interactions. Each block can be further customized, depending on desired inputs and outputs, along with various mathematical functions being available. The highest-level representation of the model constructed for this thesis can be seen in Figure 8.



**Figure 5: Constructed Model via Simulink**

As is displayed in Figure 8, the model is built with four separate blocks, representing four separate equations, detailed in the following sections. The model varies with time, as can be interpreted from the clock in the top left corner of the picture, and the main dependent variable is mass. Other outputs were also determined; however, they are not shown in this model as they were only used for graphical and data-processing purposes – these outputs are the individual outputs of each block and are not the focus of this study. The varying input for the model is described within the Voluntary Choice block, which is the individual's choice in the rate of food intake. In reality, the individual is the ultimate determining factor in how much food is being consumed, regardless of whatever influence he or she experiences. Simply put, the model assumes that the individual's choice of the quantity to eat is a function of many factors, but is primarily influenced by the perception of hunger.

The feedback loop of the model represents the body using summations of different bodily aspects or functions. To start, the Voluntary Choice block determines the food input, as explained above. This block also determines the kinetic energy,  $E_{KE}$ , expenditure of the individual, which is the rate of energy used from physical activity. This quantity is part of Voluntary Choice because, like eating, the model assumes an individual controls the choice of quantity and level of exercise. This energy expenditure is then added together with  $E_{metab}$ , which is the rate of energy used at rest (the determination of this quantity will be further discussed in a later section) to determine the total energy demand by the body ( $E_{dem}$ ), given by:

$$E_{dem}(t) = E_{KE}(t) + E_{metab}(t) \quad (2)$$

From there, the energy demand is subtracted from the body's available energy supply ( $E_{sup}$ ). This energy supply value is the supply of energy in the body created from consumed food or conversion of energy reserves, both taking place in the Metabolism block described later. This summation block (shown below) subtracts energy supply from energy demand to yield energy debt ( $E_{debt}$ ), which represents the deficit or surplus of energy that the body is experiencing at any given time.

$$E_{debt}(t) = E_{dem}(t) - E_{sup}(t) \quad (3)$$

This determined energy debt, which can be either positive or negative, as stated above, is the factor that determines if an individual gains or loses mass in the simulation. A caloric surplus causes an individual to gain weight in whatever timeframe said surplus takes place, while a caloric deficit will do the opposite, causing him or her to lose weight (Vandevijvere & Swinburn, 2015). Hence, the energy debt value is calculated to account for mass gain or loss, explained in the section detailing the Mass Dynamics block. This energy debt is also used in determining hunger, which, along with mass, are used as feedback signals in determining how

the model will react each time through feedback loop. Energy supply is also used in this regard.

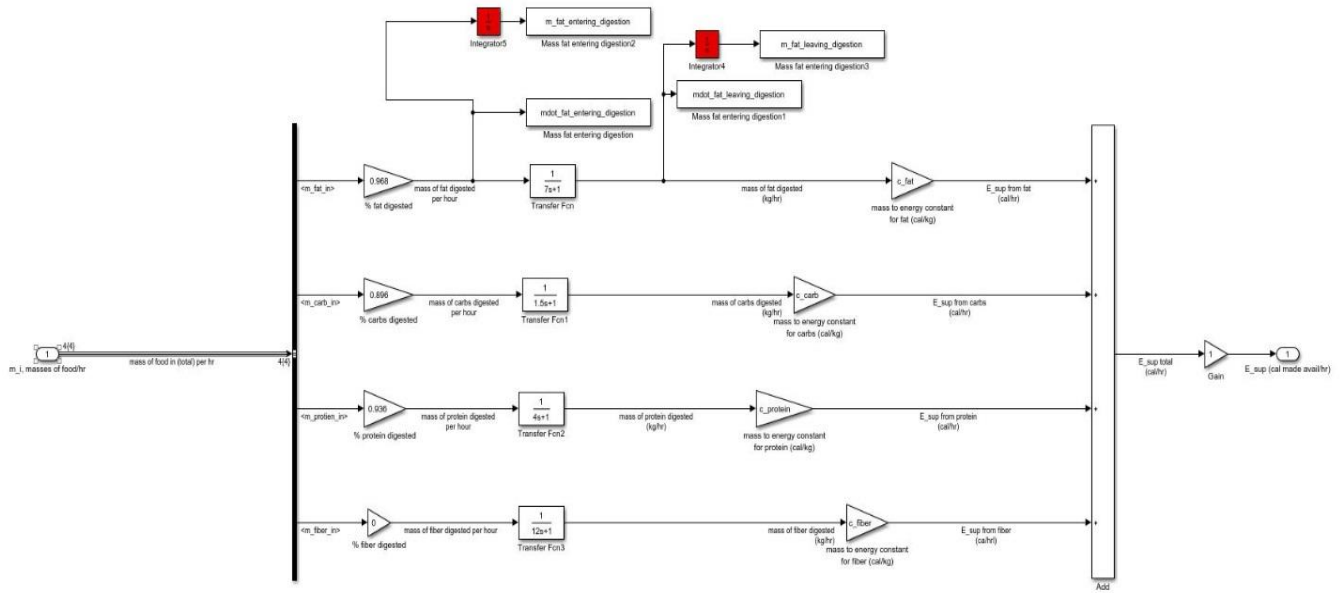
How these feedback signals work and influence the model's mechanisms is discussed in the following sections.

### **Subsystem Dynamics**

Each subsystem of the model includes further blocks of more detail to represent the equations used to represent core metabolic and digestive mechanisms. Each block represents a different equation that in turn, represents a different step of the digestive process within the body. These equations enable the simulation of food digestion once it is consumed, stored as body mass, utilized as metabolic energy, and excreted. Each block and its respective equation are explained in the subsections below.

#### **Metabolism**

The metabolism block is one that represents the actual digestion of food and its conversion into usable energy for the body. The input of this block, which comes from Voluntary Choice, is the rate of food consumed per hour, broken up into four different macronutrients: carbohydrates, fat, protein, and fiber. These food categories were chosen because they are the four main constituents of food, and have significant impact on digestion (Dietary Guidelines, 2015). The fourth macronutrient is alcohol, but is not included in this thesis as it is not generally contained in average foods, nor is it usually consumed to be a primary source of nutrients. Therefore, it is assumed that large caloric intake of alcohol is not reflective of the general population's average caloric intake.



**Figure 6: Inside the Metabolism block**

The block's output, as is shown in Figure 9, is the energy supply to the body. This quantity is obtained from the equation below, converting the rate of food ingested into a rate of energy available, deemed energy supply, or  $E_{sup}$  (as shown in Figure 9). The equations for converting each macronutrient into energy are as follows:

$$\frac{dm_{fat,digest}}{dt} = f_{digest,fat} \cdot \frac{1}{\tau_{fat}} m_{fat,in}(t), \quad \dot{E}_{fat}(t) = c_{fat} \cdot \dot{m}_{fat,digest}(t) \quad (4)$$

$$\frac{dm_{carb,digest}}{dt} = f_{digest,carb} \cdot \frac{1}{\tau_{carb}} m_{carb,in}(t), \quad \dot{E}_{carb}(t) = c_{carb} \cdot \dot{m}_{carb,digest}(t) \quad (5)$$

$$\frac{dm_{protein,digest}}{dt} = f_{digest,protein} \cdot \frac{1}{\tau_{protein}} m_{protein,in}(t), \quad \dot{E}_{protein}(t) = c_{protein} \cdot \dot{m}_{protein,digest}(t) \quad (6)$$

$$\frac{dm_{fiber,digest}}{dt} = f_{digest,fiber} \cdot \frac{1}{\tau_{fiber}} m_{fiber,in}(t), \quad \dot{E}_{fiber}(t) = c_{fiber} \cdot \dot{m}_{fiber,digest}(t) \quad (7)$$

The equation for each macronutrient is slightly different, as the percent of each macronutrient that is digested varies between macronutrients, as does the caloric content of each macronutrient, or the energy extracted from the digestion of each. The caloric content of each macronutrient is



represented by the  $c$  constant in the equations, and was developed per data from table 3. The percentages of each macronutrient digested, referred to by  $f_{name}$  in the equations, were found from a study published in the United States National Library of Medicine, which analyzed human feces for macronutrient composition (Kurai, 2012).

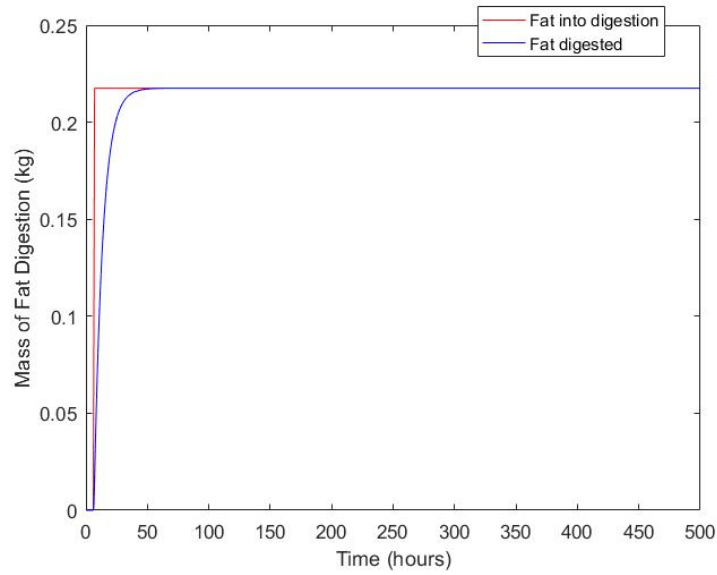
**Table 4: Macronutrient absorption percentages determined from fecal composition; left column for normal feces, right for diarrhea (Kurai, 2012)**

|   |                   |                   |                   |
|---|-------------------|-------------------|-------------------|
| Fecal composition                       |                   |                   |                   |
| <b>F<sub>WW</sub> (g/day)</b>           | $157 \pm 79$      | $796 \pm 942$     | <b>&lt; 0.001</b> |
| <b>Percentage of dry weight (%)</b>     | $17.6 \pm 5.8$    | $13.5 \pm 4.8$    | <b>0.047</b>      |
| <b>F<sub>Energy</sub> (kcal/day)</b>    | $146.4 \pm 86.7$  | $445.5 \pm 201.3$ | <b>&lt; 0.001</b> |
| <b>Energy wet feces (kcal/g)</b>        | $0.97 \pm 0.36$   | $0.78 \pm 0.37$   | 0.135             |
| <b>Energy dry feces (kcal/g)</b>        | $5.49 \pm 0.69$   | $5.59 \pm 1.09$   | 0.720             |
| <b>F<sub>Fat</sub> (g/day)</b>          | $2.4 \pm 2.6$     | $11.4 \pm 14.3$   | <b>&lt; 0.001</b> |
| <b>F<sub>Nitrogen</sub> (g/day)</b>     | $0.9 \pm 0.5^a$   | $2.6 \pm 1.1^b$   | <b>&lt; 0.001</b> |
| <b>F<sub>Protein</sub> (g/day)</b>      | $5.6 \pm 3.1^a$   | $16.2 \pm 7.1^b$  | <b>&lt; 0.001</b> |
| <b>F<sub>Carbohydrate</sub> (g/day)</b> | $24.1 \pm 17.7^a$ | $52.1 \pm 18.9^b$ | <b>0.003</b>      |
| Absorption capacity                     |                   |                   |                   |
| <b>Energy absorption (%)</b>            | $93.1 \pm 4.1$    | $76.5 \pm 10.6$   | <b>&lt; 0.001</b> |
| <b>Fat absorption (%)</b>               | $96.8 \pm 3.3$    | $84.7 \pm 17.2$   | <b>&lt; 0.001</b> |
| <b>Protein absorption (%)</b>           | $93.6 \pm 3.8^a$  | $82.5 \pm 7.2^b$  | <b>&lt; 0.001</b> |
| <b>Carbohydrate absorption (%)</b>      | $89.6 \pm 6.7^a$  | $74.5 \pm 12.9^b$ | <b>&lt; 0.001</b> |

The equations from Figure 9 begin with the input rate of mass ingested, and by multiplying by the percentage of each ingested macronutrient, one can obtain the rate of food ingested by type. Then, the low-pass model of nutrient absorption is applied by multiplying the inverse of each macronutrient's respective time constant, which determines the rate of digestion of the macronutrient at hand. This process accounts for the fact that the energy from the food is not all extracted instantaneously, but rather as a gradual process.

The time constant of nutrient uptake was determined via averaging recorded digested times of different types of each macronutrient, as there is a lot of variation within the literature. Specifically, the digestion times that were used to create the averages were obtained from a study detailing the digestion of foods (Hur, Lim, Decker & McLements, 2010). This way, the most encompassing results can be obtained, rather than simply picking one of the types and using that digestion time.

To ensure the mass quantities are equal before and after the filter was applied, the rates from before and after the filter were integrated (summed in time) and plotted against one another. This way, it is proven that the filter only impacts rate of digestion, rather than quantity digested. The results from are displayed in Figure 11. This test was just done for fat arbitrarily, as it was only necessary to prove the filter did not alter the amount of mass, and the same filter structure is used for all nutrients. One meal of solely fat was used as the input, and the simulation ran for two days in order to give digestion ample time to take place. As can be seen in Figure 11, the mass into digestion instantly rises to its value, as is expected, and the mass digested lags and rises more slowly to the input value, but after enough time (once all the food goes through digestion), the post-digestion mass levels out at the same value as the pre-digestion mass. This test proves the fact that the filter only slows the rise time, while leaving the cumulative signal unaffected.



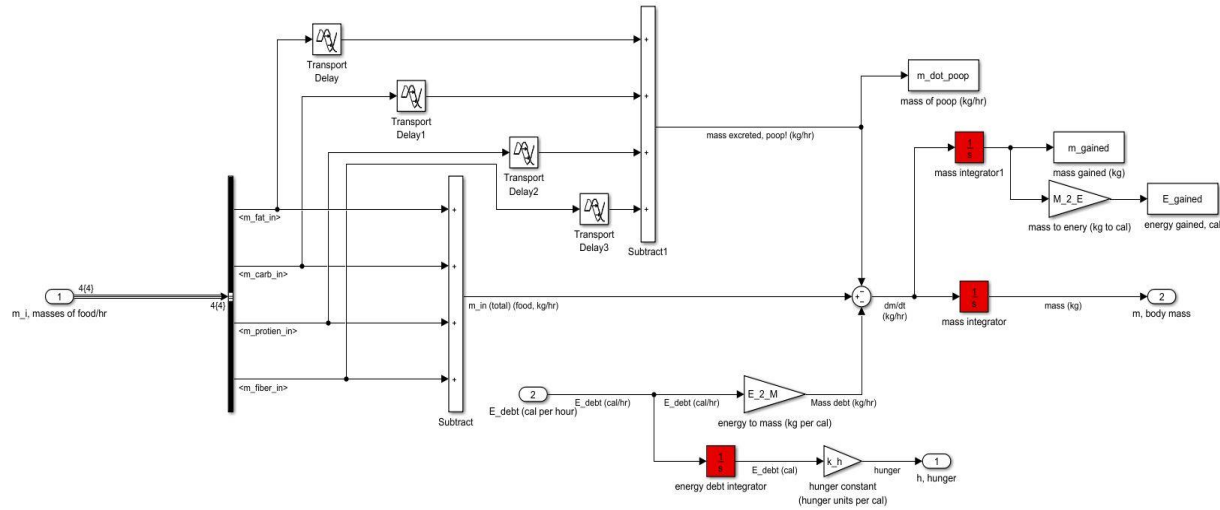
**Figure 7: Mass digestion test**

The rate of mass digested is then converted into energy by multiplying the digested mass of each macronutrient by its respective energy content ( $c_{fat}/carb/protein/fiber$ ). These energy conversion equations are also displayed in Figure 9, following the equations for calculating rate of food digested. From there, the energies from each macronutrient are summed together in order to yield a total energy supply as the final output for the block, as described by the equation:

$$E_{sup} = E_{carb} + E_{protein} + E_{fat} + E_{fiber} \quad (8)$$

This total energy supply rate is then fed into a summation to calculate energy debt as well as fed back into the Voluntary Choice block. This is because energy supply may be a primary factor that influences one's choice to exercise, as an individual is less inclined to exercise if they feel drained, or have no supply of energy, and more inclined to do so if they feel the opposite.

## Mass Dynamics.



**Figure 8: Inside Mass Dynamics block**

The energy debt that is calculated from the summation of energy demand and energy supply, then feeds into the Mass Dynamics block as one of the inputs, which is displayed above. The energy debt is the determining factor in whether or not an individual gains or loses weight (Vandevijvere & Swinburn, 2015). The energy debt is then further manipulated in two ways: to determine mass gain or loss, and to determine how hungry the individual feels. The equations describing these relations are:

$$\frac{dm}{dt} = -E_{debt} + \dot{m}_{in} - \dot{m}_{poop} \quad (9)$$

$$h(t) = k_{hunger} \cdot E_{debt}(t) \quad (10)$$

First, the energy debt is converted to mass, using information published in the US National Library of Medicine that indicates 3500 calories equates to one pound of mass change, whether it is a surplus or a deficit of said calories (Hall, 2008). This constant is then inverted and converted to kg rather than pounds, so as the units stay consistent. In the diagram, this is represented by the E\_2\_M variable. This way, the influence that energy debt had on mass

change was accounted for, in that the simulation would account for mass increase from caloric surplus, and loss from deficit.

Next, the energy debt was integrated to remove the time aspect, and multiplied by a hunger constant, which determines how hungry an individual feels for each calorie they are lacking. This product, being hunger, can be positive or negative, indicating if the individual feels hungry or full, and the distance from zero indicates the magnitude of said hunger. However, in regards to hunger, while the hunger quantity was determined in theory, no conclusive information was found on how to directly convert between energy debt and hunger. Hence, more research is needed on this subject in order to determine a hunger constant in this instance and for it to be utilized in the model to add further complexity and accuracy to the simulation. As of now, it was left as an estimated quantity, the derivation of which is explained in later sections, and fed into the Voluntary Choice block as such.

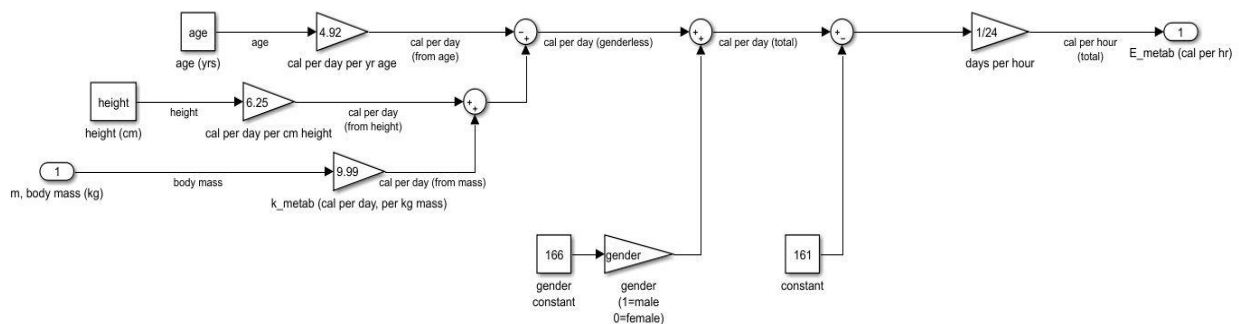
The energy debt rate was fed into a summation along with  $m_{in}$  (total), representing the total rate of mass ingested, as well as the total rate of mass excreted, represented by  $m_{dot\_poop}$  in the diagram. This equation, as displayed above, represents an individual's mass change, as mass is gained or lost from energy debt, gained from food ingested, and lost from that which is excreted. The ingested mass rate was obtained from Voluntary Choice, while the excreted mass rate was calculated via a transport delay. A transport delay was used in conjunction with each mass input rate, to delay the excretion in order to represent the time it takes for food to move through the body. The excreted masses were then summed back together to represent a total, and used in the summation as described above.

The mass change rate obtained from this summation was then integrated to obtain a total change in quantity of mass, as this is the main focus of the study and the change that is being

observed. This quantity was used in number of data plots, as well as an output for the block to be used in the overall feedback loop, since body mass is a signal that influences changes in metabolic rate, as well as the choices an individual makes about their diet and activity level. The mass quantity was also used as a tool for debugging, as can be seen in its top branch in the diagram, in case there were inconsistencies in the results.

### B.M.R.

The BMR block is used to represent the amount of energy that the body uses at rest. A human being, even when not doing any physical activity whatsoever, still uses calories simply to perform necessary bodily functions in order for them to survive (Mifflin, Scott & Daugherty, 1990). These processes account for the fact that even if an individual refrains from any sort of movement for an entire day, they will still become hungry at some point and need to eat, due to their need for energy to perform bodily functions. This block gets its name from the value that represents this amount of calories needed each day to survive, called the “basal metabolic rate,” or BMR for short. The diagram for the block is shown in Figure 13 for reference.



**Figure 9: Inside BMR block**

There are many ways to calculate the BMR of an individual, but the one chosen for this thesis was the Mifflin St. Jeor equation, as described in the American Journal of Clinical Nutrition. The equation, with  $E_{metab}$  represents the calories needed per day (Mifflin, Scott & Daugherty, 1990) and is given by:

$$E_{metab} = 9.99 \cdot weight + 6.25 \cdot height - 4.92 \cdot age + 166 \cdot sex - 161 \quad (11)$$

The input for this block was the mass, which is the weight in this equation, and the main input. Height (in centimeters) and age (in years) are variable inputs set by the user, but are held constant in this study to generate results that can be compared to one another without involving other variables that may cause different results. The sex or gender is also an input similar to those just described, but 1 is used for males, and 0 is used for females.

This equation, out of all the other available ways to calculate the BMR of an individual, was chosen because based on research, it proved to be the most accurate. According to a study published in the US National Library of Medicine that tested all the methods to calculate BMR against one another, the Mifflin St. Jeor equation was deemed the best. “The Mifflin-St Jeor equation is more likely than the other equations tested to estimate RMR to within 10% of that measured,” (Frankenfield, Roth-Yousey & Compher, 2005) clearly indicating that it is the best choice for calculating the BMR. RMR is just another name for BMR, meaning “resting metabolic rate (Frankenfield, Roth-Yousey & Compher, 2005). Therefore, this equation was the best choice for this model in terms of generating accurate results that realistically represent the human body.

The last step of the equation, which was added for the sake of this model, was to divide by a factor of 24. As the Mifflin St. Jeor equation is used to calculate calories per day needed by an individual, it was necessary to divide this quantity by 24 before it was sent as an output to the

rest of the model, as the rest of the model is based in hours, and the units must be consistent for the simulation to run accurately.

## Voluntary Choice

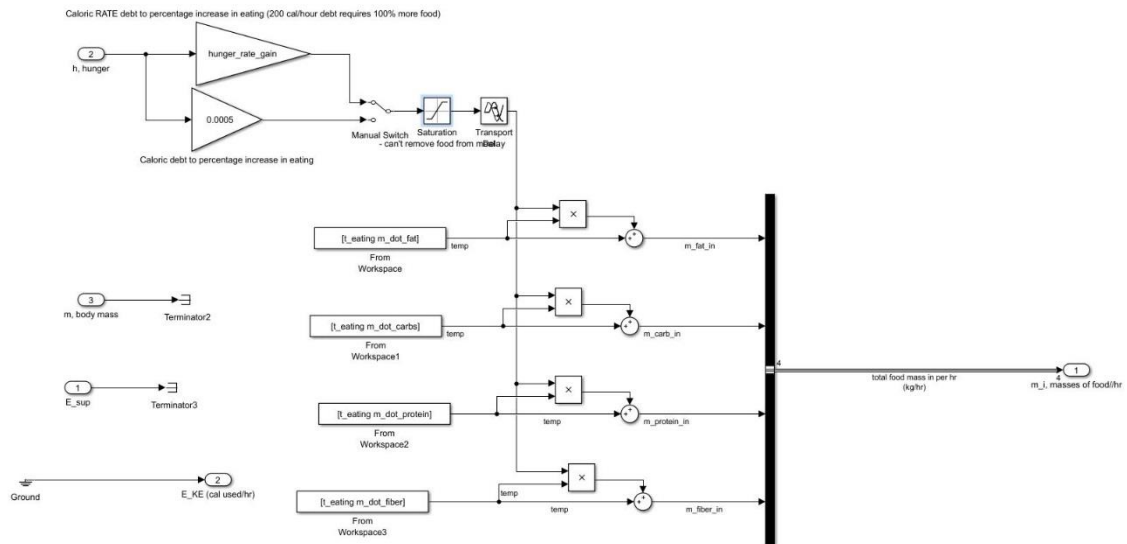


Figure 10: Inside Voluntary Choice block

The Voluntary Choice block is shown in Figure 13. It is mostly constructed via assumptions of human behavior, due to inability to find sufficient research on the mechanisms it is supposed to represent. It is likely that eating and exercise choices draw from much more than just the digestive process, making them very difficult to model without immense levels of complexity and many different variables. The outputs of this block are the rate of food intake, as well as the rate of energy used for exercise, which are both choices made by an individual. The inputs, as shown, are body mass, hunger, and energy supply. These were chosen as inputs, because they all, in some way, influence an individual's decision to eat or exercise. For instance, if an individual is hungry, it is possible that he or she will likely have more desire to eat, and less



desire to exercise. If the individual feels full, the opposite will likely be true: he or she will have less desire to eat, and more capacity for exercise, as his or her body would not be yearning for fuel.

However, it is important to note that non-caloric influences and messaging pathways may affect eating and exercise decisions. If an individual deems his or her body mass to be too high, they might restrict their eating and try to exercise more, in an effort to reduce mass to a level he or she is more happy with. If the individual wants to gain weight, he or she may alter their type of exercise, or not exercise at all, and would also be inclined to eat more food. Though these inputs would lead an individual to make certain choices regarding their food intake and energy expenditure, it is exceptionally difficult to represent these relations into a concise equation. And such equations would likely vary greatly between individuals, simply due to personal preference. The structure of the Voluntary Choice block allows investigation of various inputs in the future.

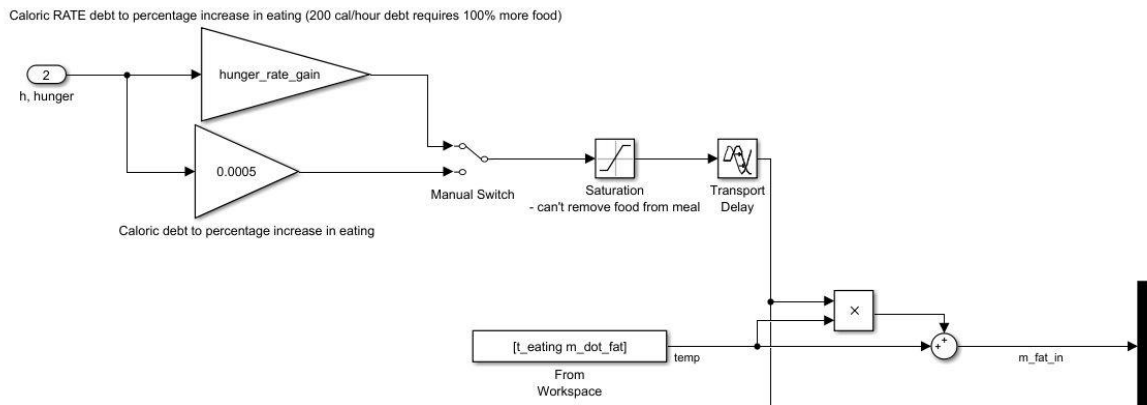
For early analysis of model behavior, instead of having the eating decisions depend on hunger, the eating behaviors were initially entered in an open-loop fashion. As the relationship between varying food intake and mass is the focus of this thesis, this technique was beneficial also because of the ease of altering the amount of food consumed, rather than having it be affected by previous signals. To do this, time arrays were created in the MATLAB workspace, with each index representing an hour of the day. On specific indices relating to chosen mealtimes, the mass input rate variables were signaled to activate and input a value at that given time. This way, multiple meals could be specified easily. The meals were designed to repeat themselves every 24 hours in order to represent a realistic diet with recurring meals each day, rather than building a signal for every single desired meal. The chosen mass rates were then summed together in order to make signal transmission easier (rather than sent out four signals),

and then sent out of the block as an output to be used in the rest of the model. The total mass input is then given by:

$$\dot{m}_{in} = \dot{m}_{fat,in} + \dot{m}_{protein,in} + \dot{m}_{carb,in} + \dot{m}_{fiber,in} \quad (12)$$

The same could be done for energy expenditure (E\_KE), without the summing, but given time constraints, this value was simply held constant in order to prevent extra variable interference and to focus on the main aspect of this study, being the mass-food relationship.

Later, to account for hunger's impact on the simulation, a mechanism to manipulate hunger was added to the intake of food, as is depicted for the fat's input in Figure 15, just to make viewing and explaining the details more simple.



**Figure 11: hunger multiplier for fat**

After the amount of food is input from the user (fat, in the example above), it is fed into a product block in order for to hunger to manipulate the amount. A hungrier individual is assumed to eat more food. Though there was little available data on how to numerically manipulate energy debt into hunger, estimates were used and methods were compared against one another via iteration to determine what seemed most realistic. The multiplier using hunger was attempted with two different approaches: using energy debt itself, and the rate of energy debt. A manual switch inserted between the two methods as means of changing the desired method

throughout the thesis, to allow comparison of hunger responses based on different algorithms for eating response to hunger signals.

The former method calculated a hunger response based on an amount of energy debt. Essentially, this approach presumed that the body detects the accumulated calories of energy debt and triggers hunger in accordance to the magnitude of said debt. A baseline estimate was used in that for a 100 calorie energy deficit, an individual would eat 5% more food than they normally would to account for the extra need for energy. The larger the energy deficit, the more hungry an individual would become, and the more food they would consume at a given meal. So, the amount of food is multiplied by the percentage derived from hunger, then added back to itself to achieve a percent increase effect.

The other method used instantaneous rate of energy debt to gauge hunger, rather than the accumulated caloric debt. The ideology is similar to the other method, but simply a different means of measuring the debt. Here, the body is presumed to register the current rate at which debt is accumulating and respond in hunger accordingly. The baseline estimate used here was that if an individual neglected to eat for an entire day, they would be missing a day's worth of calories, which is 2000-3000 for most individuals. A response of 2400 was chosen, simply to make the conversion advantageous, as there are 24 hours in a day, meaning a debt of 200 calories per hour. Hence, for a debt of roughly 200 calories an hour, an individual was estimated to double their consumption, as, at that rate, a day's worth of calories would be lost, which doubling intake would compensate for.

Regardless of the method used, its result was then fed through a saturation block, as well as a transport delay before it was used as multiplier with in the food input. The saturation block was used to account for the fact that there is no such thing as "negative eating". If the energy

debt happened to be negative, meaning there was a caloric surplus, then hunger would then become negative. This change of sign then would cause a negative multiplier to be used in conjunction with the food input, causing less food to be fed into the diagram than what was input by the user. The implications of this are unrealistic, which say that if an individual feels too full, he or she would then be able to “negatively eat”, or rid themselves of food. The saturation block helps avoid this effect by adjusting all negative values to zero, so they do not cause the simulated individual to eat “negative” food. Furthermore, the transport delay was utilized to account for the fact that the feeling of hunger is not instantly registered by the body as soon as food is introduced – it takes time for people to feel hungry after they have eaten. The transport delay simply allows for time to pass before the hunger signal can start affecting food intake, as would be realistic.

For each iteration through the feedback loop, the individual’s food intake would change slightly based off of how hungry he or she was, allowing for a more realistic representation of eating patterns, rather than having the individual consistently eat the same number of calories at every meal. Furthermore, this use of a feedback loop accounts for weight gain effects. Namely, if one is eating foods that digest faster, then they can accrue energy debts at a faster rate once digestion is complete. Not only does this make the model more realistic, but allows for better results in regards to the focus of this thesis, in that the rate of digestion’s impact on hunger at least has *some* effect rather than none. The methods were later tested against one another to determine the better choice, which is further explained in chapter 4.

## Implementation

In running the simulations in order to study this mass-food relationship, certain parameters were established and settings chosen to ensure realistic results, as well as prevent unwanted errors while running the simulations. These included altering the way the program runs, changing the model's configuration, as well as carefully choosing the information to be utilized and manipulated by the model. The following paragraphs detail said techniques used to ensure the model's functionality.

First, the issue of time was necessary to determine, in regards to what unit to use, as well as how long to run these simulations. In order to better compare the results of this study to published literature, the simulated time for each test run was aimed to match that of studies that examine mass changes based on diet in individuals. This number, in order to observe statistically significant body mass changes, was deemed to be two weeks to a month. Therefore, all simulated tests with this model were ran for at least 2 weeks duration. Furthermore, as is the case with all models, units must be consistent throughout in order for things to run smoothly. Hence, the issue with picking a unit of time. In the end, hours was chosen, due to the average digestion time of foods being used ended up being in hours, rather than days or minutes (Hur, Lim, Decker & McLements, 2010). Instead of converting these units to something else, the model was built around the unit of hours in order to stay consistent and avoid confusion.

After choosing the unit of hours based on the average digestion rate of different macronutrients, a sampling rate must be chosen for the simulation in order to send information to MATLAB in order to be graphed. To avoid any sampling errors, the Nyquist sampling theorem was applied to the smallest digestion rate of a macronutrient, being about 1.5 hours for carbohydrates, as can be seen in Figure 9 in the time constant for carbs. This theorem

recommends 2 times the digestion rate to be used for sampling rate, but to avoid error and be cautious, a number *much* greater than that was chosen, at 400 times per hour. This way, it could be said with much confidence that any sampling error could be avoided. Moreover, various outputs were sent to MATLAB aside from just mass, such as energy debt and food intake, in order to be used for graphical comparison purposes in literature comparison or as debugging tools. A visual interpretation makes it easier to spot erroneous trends, as well as to get a quick, generalized idea of the information without pouring through the data.

Lastly, the method of entering food was chosen so that meal time could also be accounted for, rather than have all the food be instantaneously entered. Not only would this not reflect reality and take away from the accuracy of the model, but it would affect the digestion rate and skew the results. To avoid this, signal builder blocks were used, as briefly mentioned before. These blocks enabled the user to input a rate of food over a certain amount of time, depending on however long the meal is desired to be (an hour was used in this thesis, as it was realistic and conveniently cancelled units without adding a multiplier), which then yielded a quantity of food due to unit cancellation. A bus signal was then used to transmit the food signal throughout the model, in order to avoid breaking and combining the signals repeatedly as they entered and left different model blocks. As it was necessary to maintain individual food signals throughout the model to keep track of individual macronutrient quantities, the bus signal was chosen to allow for the individual signals to be maintained, while also allowing manipulation of all four signals as if they were one. Not only was this beneficial and convenient in regards to the model, but it also reflected reality in that food goes through the body as one and isn't broken up into different macronutrients and dealt with separately, yet each macronutrient within the food has its own unique impact on the body and digestion.

## **Chapter 4**

### **Discussion and Model Validation**

The goal of the simulation model is to replicate the processes in which food is digested and contributes to mass change. But even if the governing equations make sense in theory, the simulation may not produce expected results. In an effort to avoid this unfavorable effect, certain preliminary tests were performed in order to ensure the model was indeed functioning properly. Once the tests confirmed the model's effectiveness, the validated model was used to perform simulated eating experiments in an effort to model and test the mass changes in an individual when different meal combinations, in terms of macronutrient ratio and content, were introduced.

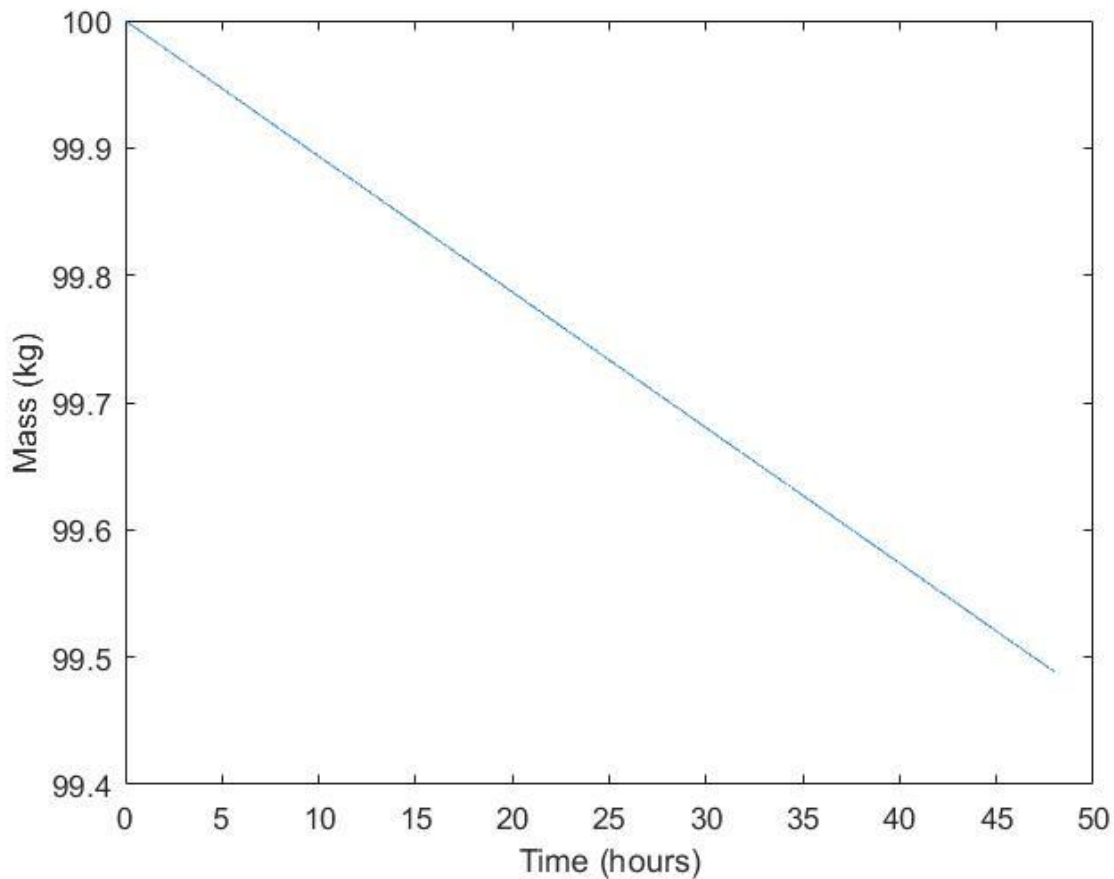
#### **Starvation Test**

The first test that was performed in order to verify the model's accuracy and proper function was a starvation test. This "no food" test was done in order to, before any amount of food was introduced, validate that the utilization of energy at rest was working correctly in the BMR subroutine. Because there was no food to digest, there was no conversion of food into energy that may have had an impact on mass in the opposite fashion. As is common practice in engineering, isolating a variable is the most effective method of determining its effects, and in this case, determining if these effects matched with what is supposed to happen in reality.

The starvation test was performed by running the simulation for two days without introducing any food into the model and monitoring mass over the course of those two days in an effort to see how much was lost. Hence, the test's name – the model, or individual, was starved for two days to see how much mass would be lost. Mass was expected to be lost as there was no food supply to be converted into energy supply for the body, and energy demand remained, due to the BMR, or energy utilized at rest. And, as mentioned earlier, according to the US National Library of Medicine, a net change of 3500 calories over a certain time span is needed to cause a mass change of one pound of whatever sign the net change was (i.e. a deficit of 3500 calories would result in a one pound loss, while a surplus of the same amount would result in a one pound gain) (Hall, 2008). As mass in this model is in kilograms, the units were converted, with one pound being equal to roughly 0.45 kilograms.

The individual tested here, and that was used for the remainder of this study, was a 20 year old male, who was 172.72 centimeters tall, and weighed in at 100 kilograms. These specifications, when plugged into the Mifflin St. Jeor equation, indicated that the individual had a BMR of 1985.1 calories per day. This number shows that over the course of two days, there would be 3970.2 calories burned, equating to slightly more than one pound of mass loss, or roughly 0.52 kilograms. When the simulated test was run, the individual showed a steady decrease in mass as expected, as there were no calories introduced for the tested timeframe, and the total mass loss was, as can be seen in Figure 14 on the following page, just slightly more than half a kilogram, which is exactly what was expected. The test displayed was run only for two days, as to prevent enough mass change to significantly alter metabolic rate.



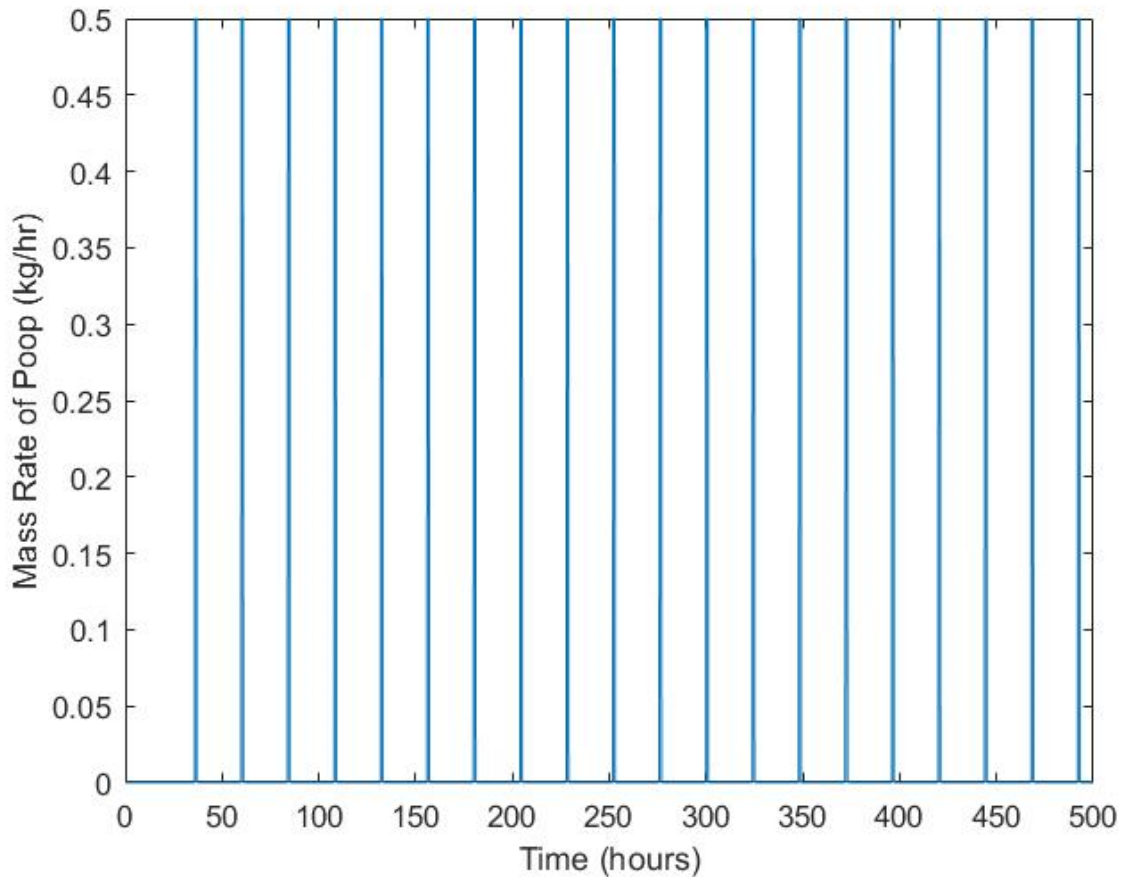


**Figure 12: Mass vs. time for starvation test**

As the starvation test proved that the simulation predicted the same amount of mass loss was expected, it was deemed a success. Now, it could be said with confidence that the energy burning half of the model was working properly, as there was a proper amount of mass loss over a certain period of time, given the lack of food introduction. Since this test was a success, it was then acceptable to test the other half of the model in the energy conversion from food. As there would be two variables involved in a test involving energy conversion that could potentially cause incorrect or inconsistent results, it was necessary to first verify that one of them (the energy utilization) was working properly in order to isolate the second (the energy conversion) and therefore prove its own validity.

## **Fiber Test**

Before the energy conversion part of the diagram was tested, it was first necessary to test the mass excretion, without affecting the energy supply. In order to do this, a fiber test was performed. In said test, the objective was to determine if the excretion part of the diagram was effective, so it was needed to use some sort of food. Since fiber has no caloric content and simply aids in digestion, it was the chosen food input for this experiment. Naturally, if the food excretion was being tested, it would be necessary to input some sort of food in order to trigger digestion and hence, excretion. However, any other food besides fiber that would be inputted would affect energy supply, as they all carry some sort of caloric value. This would trigger the energy conversion part of the diagram, which is supposed to be avoided in this test, as it adds an extra variable and makes the results inconclusive. Hence, fiber was chosen for just one meal each day of about half a kilogram. The amount and frequency here was arbitrary, as it was only desired to inspect if the pooping part of the diagram was working and that mass, though fluctuating due to entry and exit of food mass, would maintain the same downward trend like the starvation test, because no real calories were being introduced.

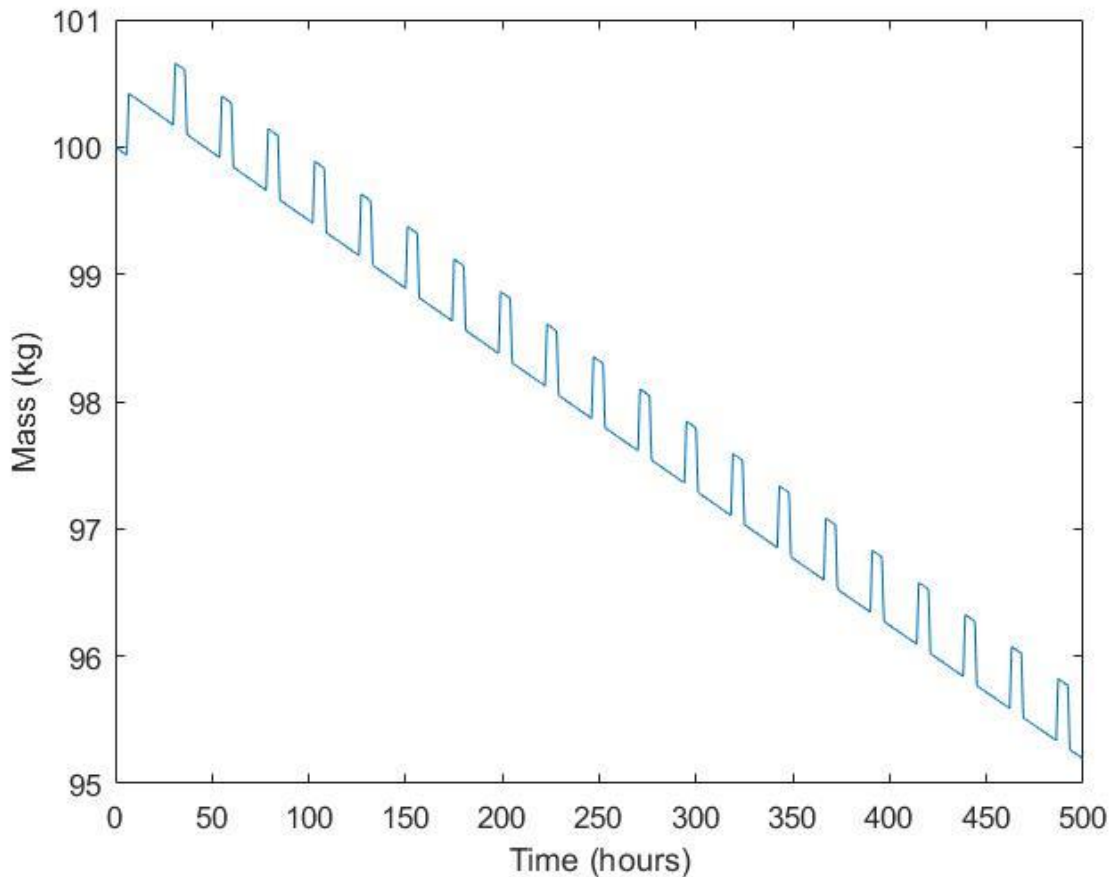


**Figure 13: fiber test – mass flow rate of poop (kg per hour) vs. time; demonstrates pooping events repeat and work effectively**

In Figure 16, the mass rate of excreta (“poop”) was plotted against time, to ensure that there were in fact instances of excretion occurring. As can be seen from the figure, this test was a success, as periodically, after inputting the mass of fiber, it was excreted, shown with the spikes of mass flow rate. The periods of inactivity were simply the times after the food was excreted, but before more was introduced. How long it took for the spikes to occur after eating were represented by the time used in the transport delay, which was 30 hours. This represents reality in that it takes about 30 hours in general for food to pass completely through the body and

out of the colon, based off an average of different types of food and how they affect this rate (Rose, Jefferson & Cartmell, 2015).

In order to then determine if mass was consistently dropping, though fluctuating as explained above, body mass was plotted against time, as is displayed in Figure 18.



**Figure 14: fiber test –body mass (kg) vs time; verification that excretion events do not influence mass change in the long run**

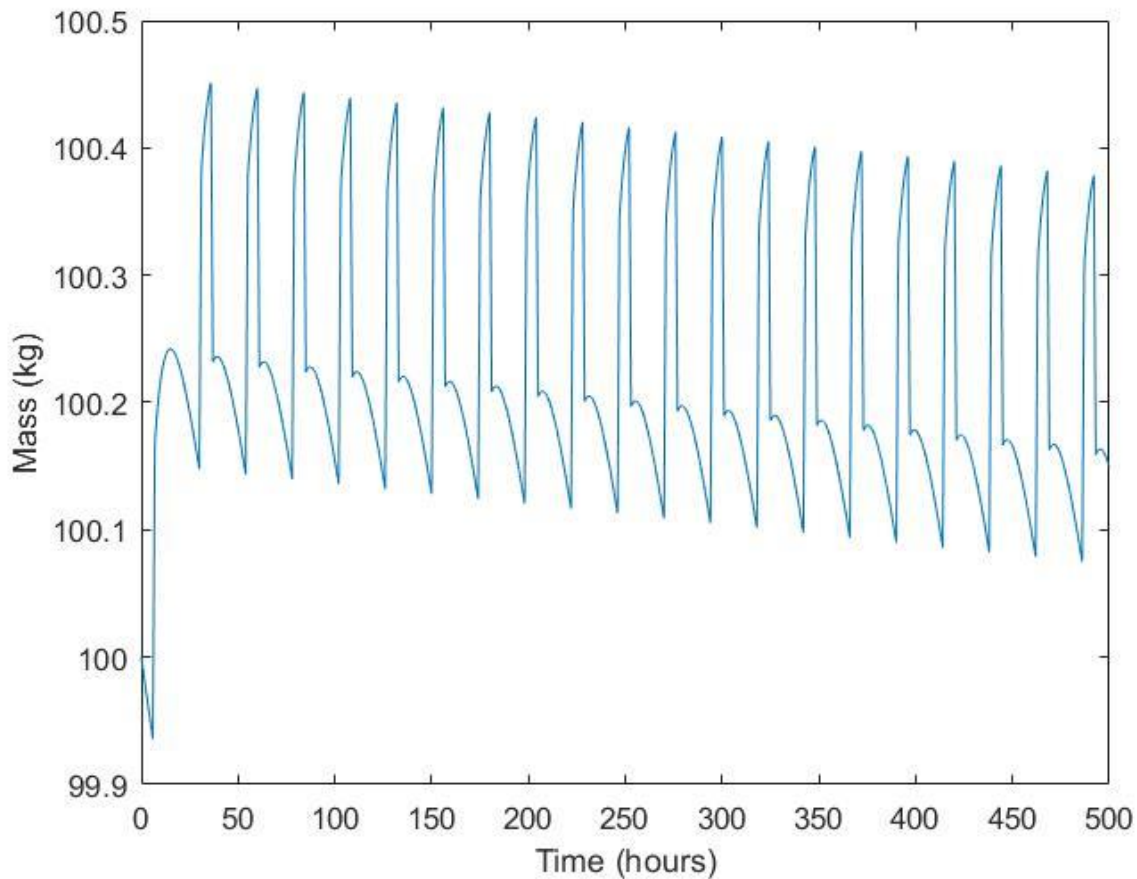
As is shown in Figure 18, the predicted downward trend in mass was verified, proving this test also to be a success, and therefore verifying the accuracy of the excretion mechanism of the diagram. Mass spiked up due to intake of fiber mass, but dropped back down again when it was excreted to the level it would have been had no food been introduced. Mass would then continue

the downward trend, burning calories while none are introduced due to fiber's lack of caloric content, until another mass of fiber was input, triggering a rise and decline for excretion once again. Now that the fiber test was proven accurate, verifying the excretion was working effectively, it was safe to move on to testing the energy conversion's effectiveness by inputting food that *does* have calories.

### **Equilibrium Test**

The equilibrium test, as briefly explained above, was one that was used to test the model's ability to deal with a food input and properly convert it to energy. As the starvation test confirmed that the baseline energy burning facet of the model was working properly, this test's results would only demonstrate the effectiveness of the energy conversion facet.

In order to perform this test, the same basal metabolic rate established in the starvation test (that will be used henceforth in this thesis, as previously noted) was used, and food ingestion rate was inputted that would yield the same amount of calories supplied to the body. This way, there would be no net energy debt, and the simulated individual should retain relatively the same amount of body mass from the start time to end time. As the type of food is arbitrary here, one meal per day of fat was inputted to achieve equilibrium, just to simplify the analysis. Though the graph of mass versus time may show some fluctuation due to different food digestion rates, food excretion, and meal inputs, the end mass should be the same (or close to it) as the start mass, as the net calories over the measured timespan would be roughly zero.



**Figure 15: Equilibrium test – mass (kg) vs time; proves effectiveness of metabolism simulation by maintaining mass in a time of no net calories**

Figure 19 shows that over a span of 500 hours, equating to a little more than 20 days, the individual maintained mass within one tenth of a kilogram, proving the equilibrium test a success. When inputting nearly the exact same amount of calories it would take to match the BMR, and therefore create zero energy debt, there was little to no mass fluctuation, as is expected. The small initial drop can be attributed to the fact that there was no food until the first meal, which led to a slight drop in mass due to an increasing energy debt. Then, once food/fuel was introduced, there was a spike in mass due to energy surplus as well as the physical mass of the ingested food. The subsequent spikes simply represent subsequent meals each day. The

drops in mass can be accounted for by excreting mass, as well as the BMR running through fuel for the remainder of the day after the equilibrium meal was consumed. Now that this test and the others were successes, all the parts were confirmed to work, and the model was considered accurate and effective, enabling experimental tests to be run in order to get to the main point of this thesis: to observe differences in mass based on digestion rate of foods consumed.

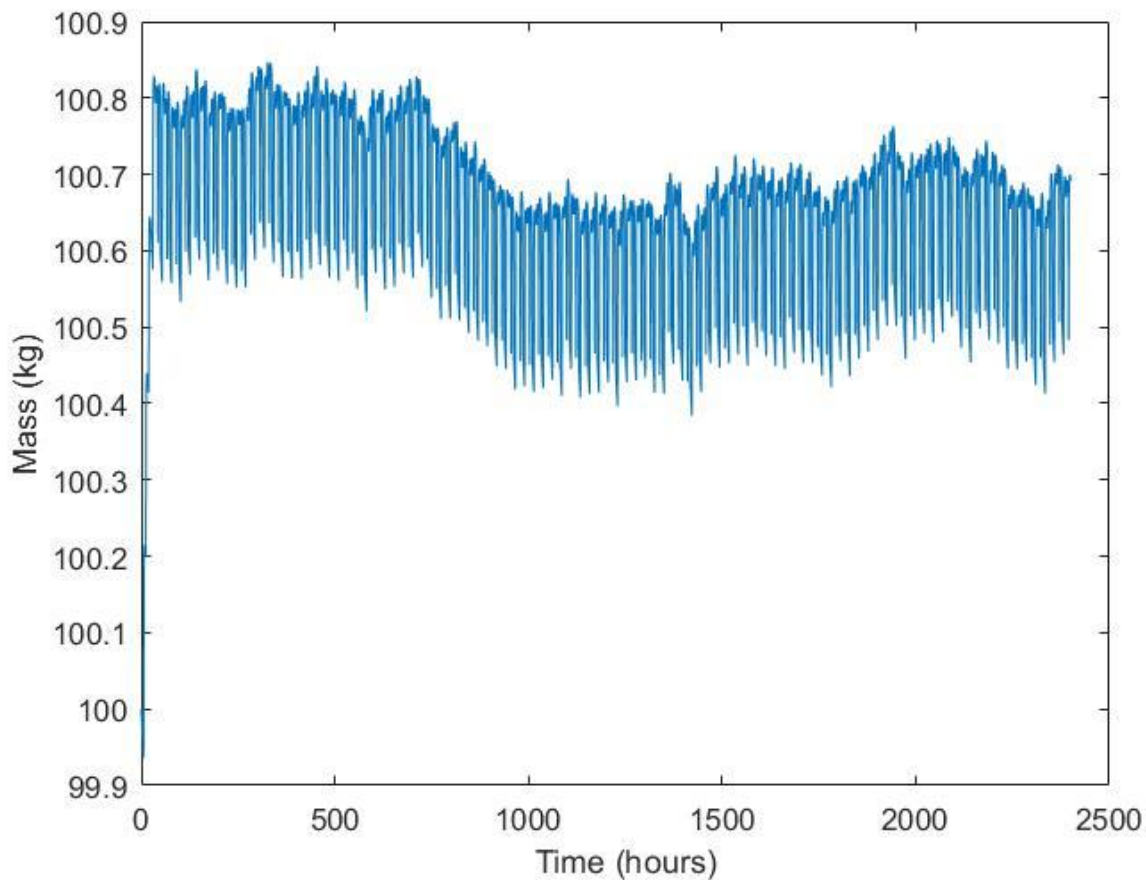
### **Hunger Test**

Once the rest of the model was established to be in working order, it was then time to establish which method of including hunger would be most effective, as well as to test if the inclusion of hunger in the model would affect it in the way that it should. As the aim of this thesis is to produce evidence that digestion rate has an impact on weight gain, this facet of the model is among the most important because it enables the digestion rate to have an effect. Without the hunger mechanism in the model, individuals would consistently eat the same amounts of food, regardless of their energy debt or how hungry they were.

In order to verify the test's effectiveness, first, the method in detecting hunger had to be chosen, as two potential options were used in the model. In order to choose between the two methods, a test was run using them both, in order to see which produced more accurate results. To do this, a diet of only carbohydrates was used in order to create a hunger effect, as carbs digest the quickest, and that diet was made to be calorically equal the BMR used in the simulation. This way, any weight gain effects that were observed would be attributed to the increased eating due to the greater hunger from eating food that digests quickly, rather than a net energy surplus within the body. If there were an energy surplus or deficit present, there would

then be an effect on mass over time, making it hard to determine what changes arose from the energy imbalance rather than the higher digestion rate of the food.

The first approach tested was using the quantity of energy debt in terms of calories to measure hunger. The simulation was run for 100 days, giving the diet ample time to have its effect on mass change. The results from the simulation are shown in Figure 20.



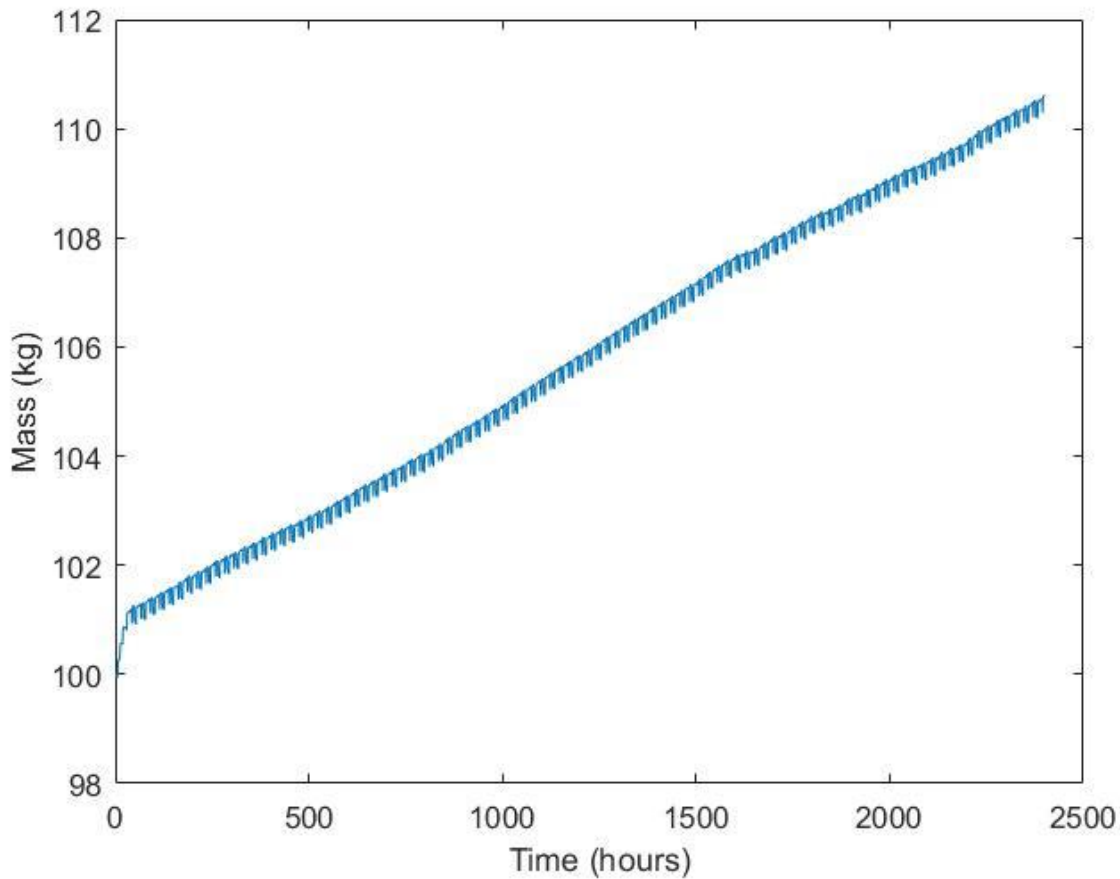
**Figure 16: hunger test for quantity approach; plots mass vs time to demonstrate effect on mass of hunger multiplier that measures quantity of energy debt**

The results show an increase in mass at first, due to ingestion of food into an initially empty stomach, but proceed to decline from there. As one would tend to eat more from eating nothing but carbohydrates due to the shorter feeling of satiety, a decrease in mass was not expected.



Therefore, the second method had to be tested, as this one produced peculiar results, losing mass with eating programmed to increase.

Then, the method of using energy debt rate as a basis for hunger, rather than the quantity of energy debt, was tested in the same way. The results are displayed in Figure 21.



**Figure 17: hunger test for rate approach; plots mass vs time to demonstrate effect on mass of hunger multiplier that measures rate of energy debt**

Here, it can be seen that the mass of the individual in the simulation steadily increased over time, yielding the expected result when consuming a solely-carbohydrate diet. Hence, this method was chosen as more accurate in portraying hunger and was used in the tests going forward. This test was suspected to be more effective due to the use of rate, as the use of quantity had some

problems with what it implied. When using quantity of energy debt to determine hunger, the individual would respond by increasing the eating to where there was no longer a numerical deficit, quickly eliminating the hunger signal, and therefore mitigating hunger's impact on eating increase. The rate of energy debt, however, was immune to this error in that it couldn't be shut off by a sharp increase of food – it took a bit more time to register hunger this way, so it had more lasting effects.

## **Chapter 5**

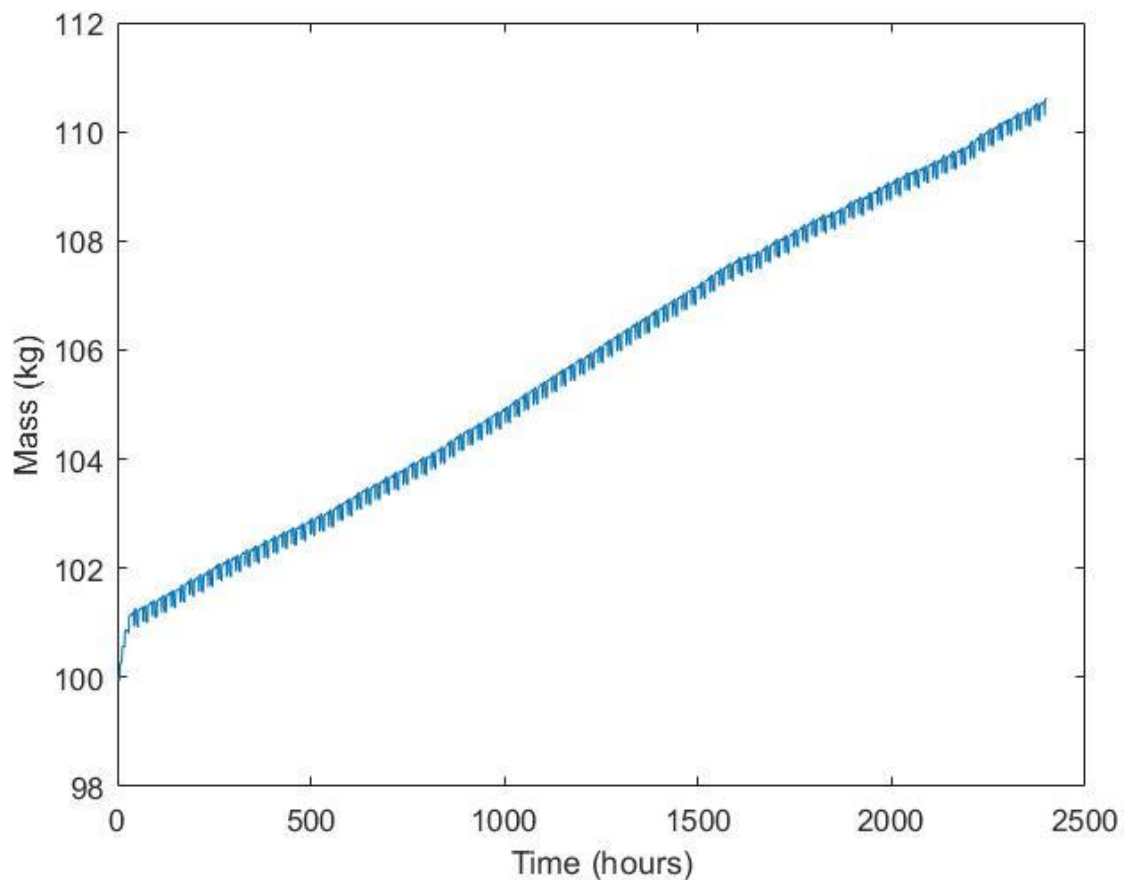
### **Results and Conclusions**

Once the model was deemed effective via testing its various facets, experimentation took place. The simulation was used to test diets of different macronutrients against one another in an effort to determine the accuracy of the hypothesis and validate the societal relevance of this thesis. In order to demonstrate that rate of digestion of foods that people consume could be a factor in weight gain, rates of digestion were simulated and compared to one another in the form of different macronutrients. The three macronutrients all have different average digestion times, as previously mentioned, and therefore were used in separate simulation tests to demonstrate different digestive rates' effects on body mass.

### **Test Results**

As noted above, simulations were ran using different diet variations based on macronutrient composition. These tests were done in order to verify the hypothesis which points to digestion rate of food as a cause of weight gain, and help lend strength to the claim that the increase in carbohydrates in Americans' diets may have helped lead to the rise in obesity levels that occurred at roughly the same time. The diets were chosen to reflect different digestion rates, varying the ratio of carbs to protein to fat to change the digestion rate of the overall consumption. Yet, the baseline ingestion of calories was maintained at equilibrium regardless of the macronutrient combination in an effort to mitigate the effects of mass gain simply from input

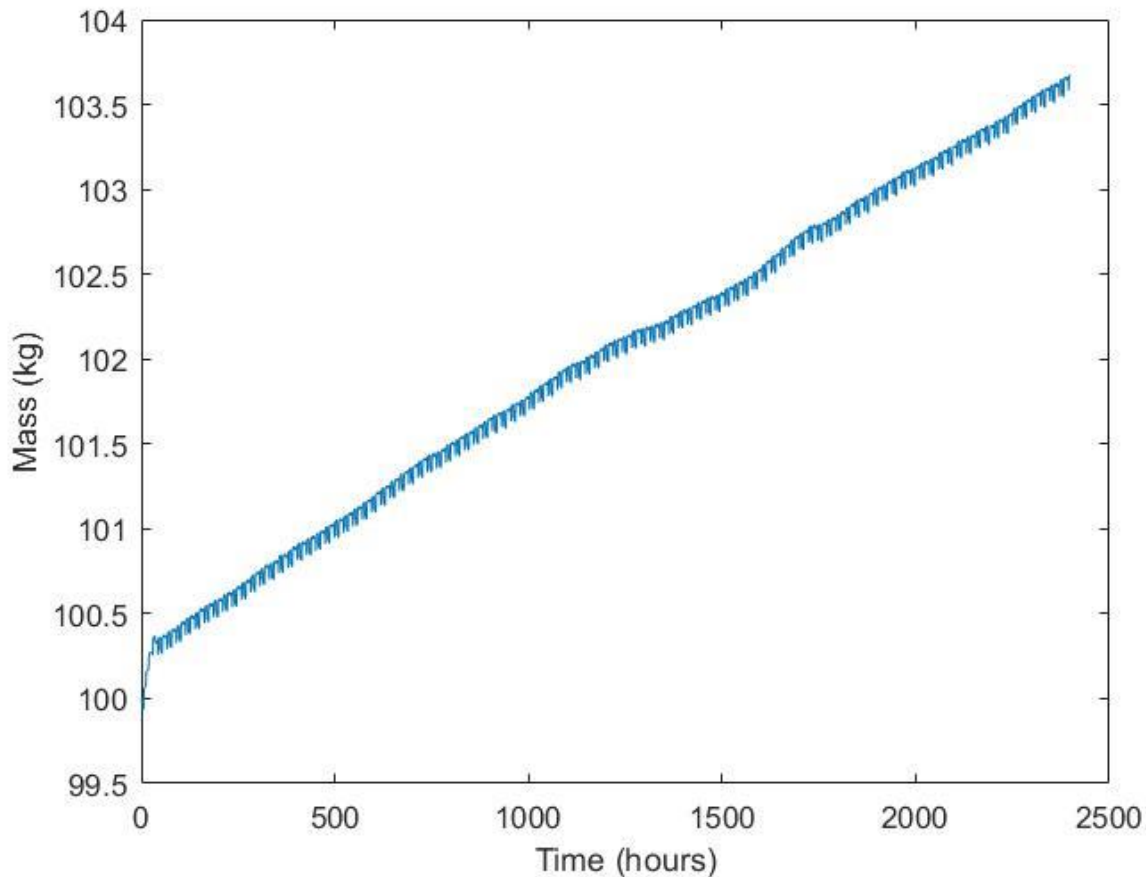
ingested surplus. This way, the effects of the surplus developed from hunger change due to different digestion rates can be more noticeable and other variable influence can be eliminated. Furthermore, the tests were run for 100 days, in order to give ample time for any mass change to occur. The results of these tests are displayed in Figures 22-24.



**Figure 18: mass vs time for equilibrium carbohydrate input – solely carbohydrate diet on diet equal to calories of BMR**

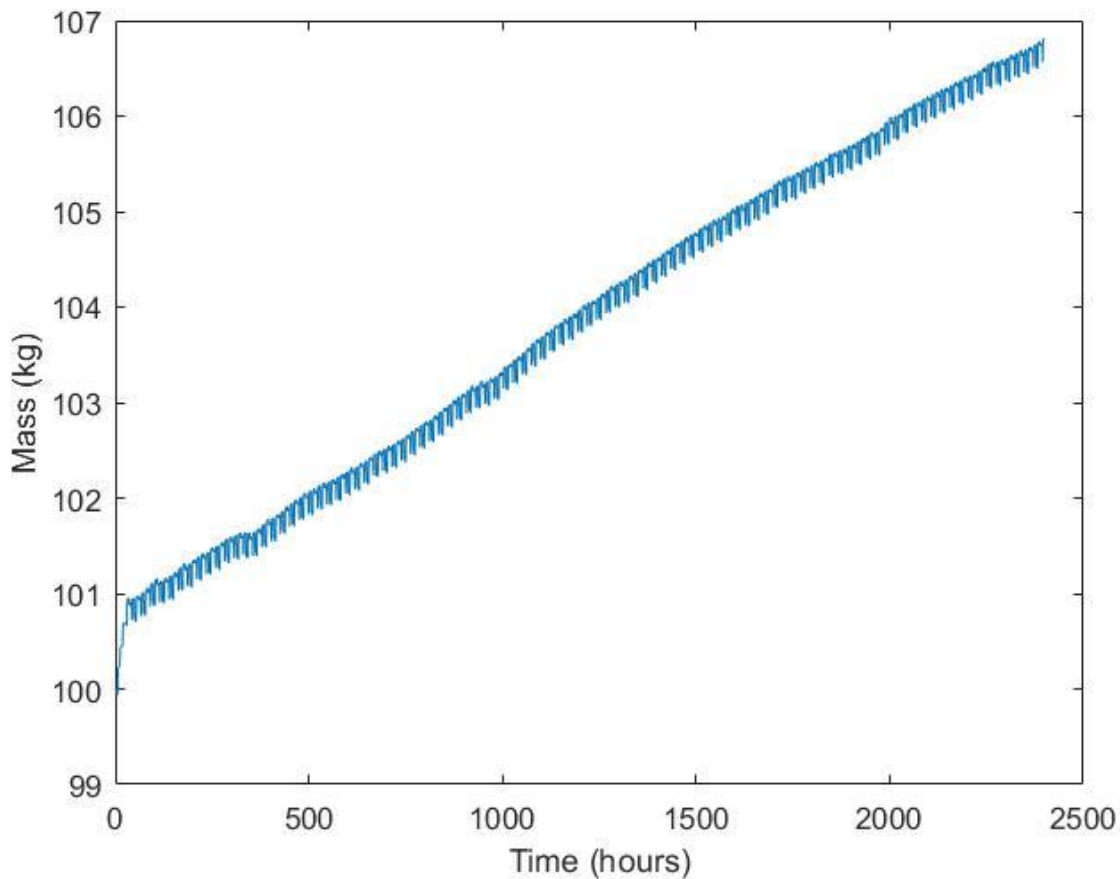
Figure 22 depicts the results from the test in which the individual consumed equilibrium calories of solely carbohydrates. This test, though extreme, is reflective of many American diets that are so carbohydrate-dominated, as shown in Figures 5-7. The mass gain was significant, as was

expected from a diet of only fast-digesting food. The end value for body mass reached about 111 kg, starting from 100 kg.



**Figure 19: mass vs time for equilibrium fat input – solely fat diet on diet equal to calories of BMR**

Figure 23 displays the results for a diet of the same kind, matching the BMR in calories to create equilibrium, but doing so with a diet comprised completely of fat. Though the individual still did gain mass from this (an effect accounted for in the next section), the amount was noticeably less, totaling at about 103.5 kg, resulting in only an increase of 3.5 kg. This went as hypothesized, as its average digestion time being the slowest producing the smallest amount of mass gain.

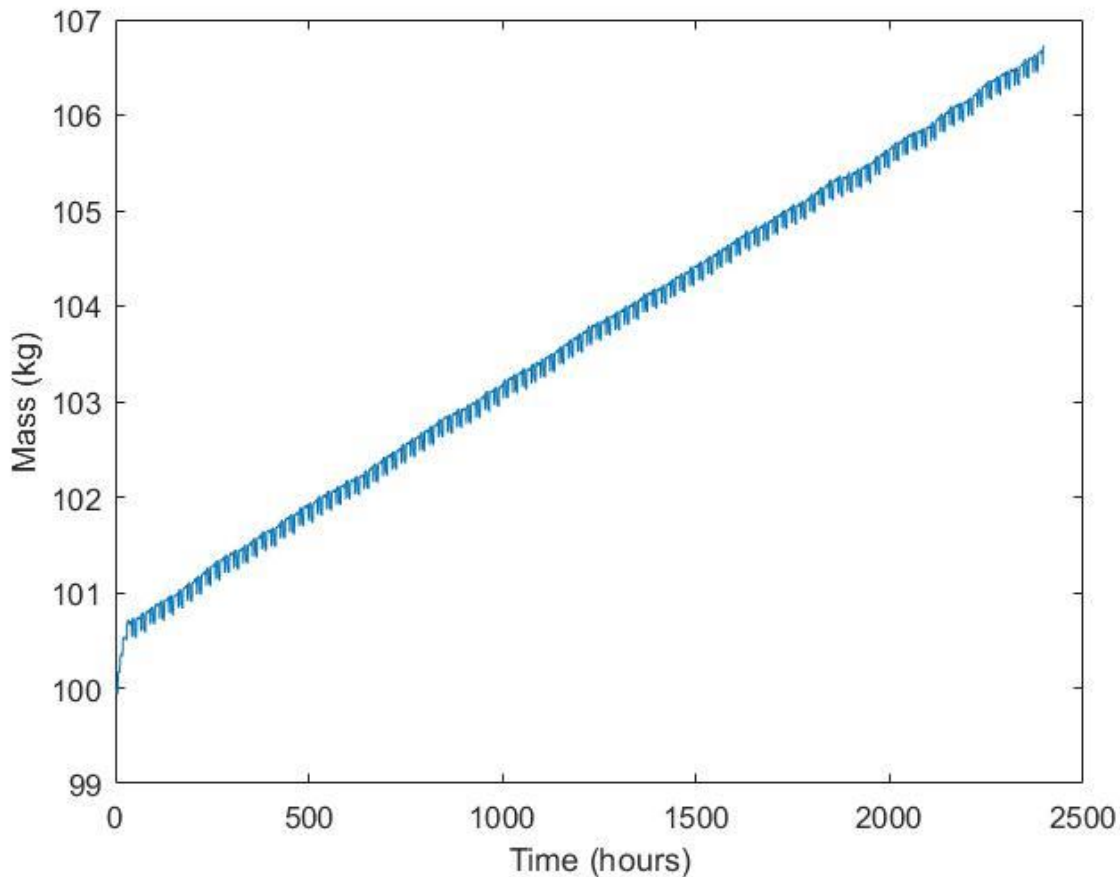


**Figure 20: mass vs time for equilibrium protein input – solely protein diet on diet equal to calories of BMR**

Figure 24 shows the results for protein, which also turned out as expected. The same mass gain was present, but to a degree between that of the previous two tests. Protein digestion time was determined to, on average, be longer than that of carbohydrates, but less than that of fat. In light of this, the results were in line with the hypothesis, as mass gain from protein rose to a total of roughly 7 kg, greater than that of fat, but less than that of carbohydrates.

To further experiment with different macronutrient ratios and to observe the effects of these ratios, macronutrients were then used in pairs with one another, splitting 50/50 in terms of fraction ingested. This way, the hypothesis that was supported by previous experiments can be

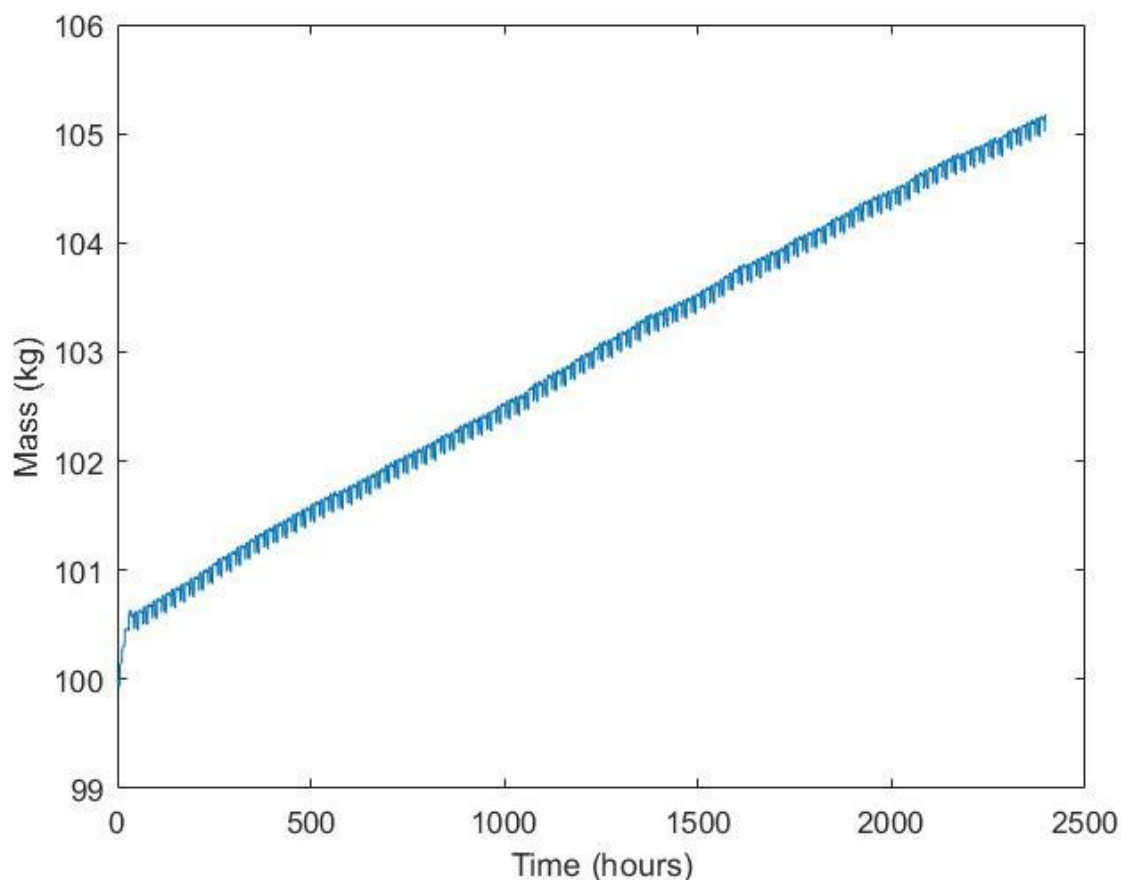
further tested, if predicted results continue to occur in accordance with the digestion rates of the nutrients involved. The results of all these three tests are shown in Figures 25-27.



**Figure 21: mass vs time for equilibrium, half fat/half carbs; diet with BMR calories comprised of half fat and half carbs**

Figure 25 displays the results of the test run splitting the input total between fat and carbohydrates. The end result was a net gain of about 6.5 kg in body mass, making it another test that fell in line with the hypothesis, causing the middle amount of mass gain among the latter three mass tests. With an input combination split between the fastest digesting macronutrient and the slowest digesting one, it would be predicted from the hypothesis that it would produce less mass gain than the protein/carb combination, but more than that of fat/protein, and that is

indeed what happened. The results for these two combinations are displayed in Figures 26 and 27.

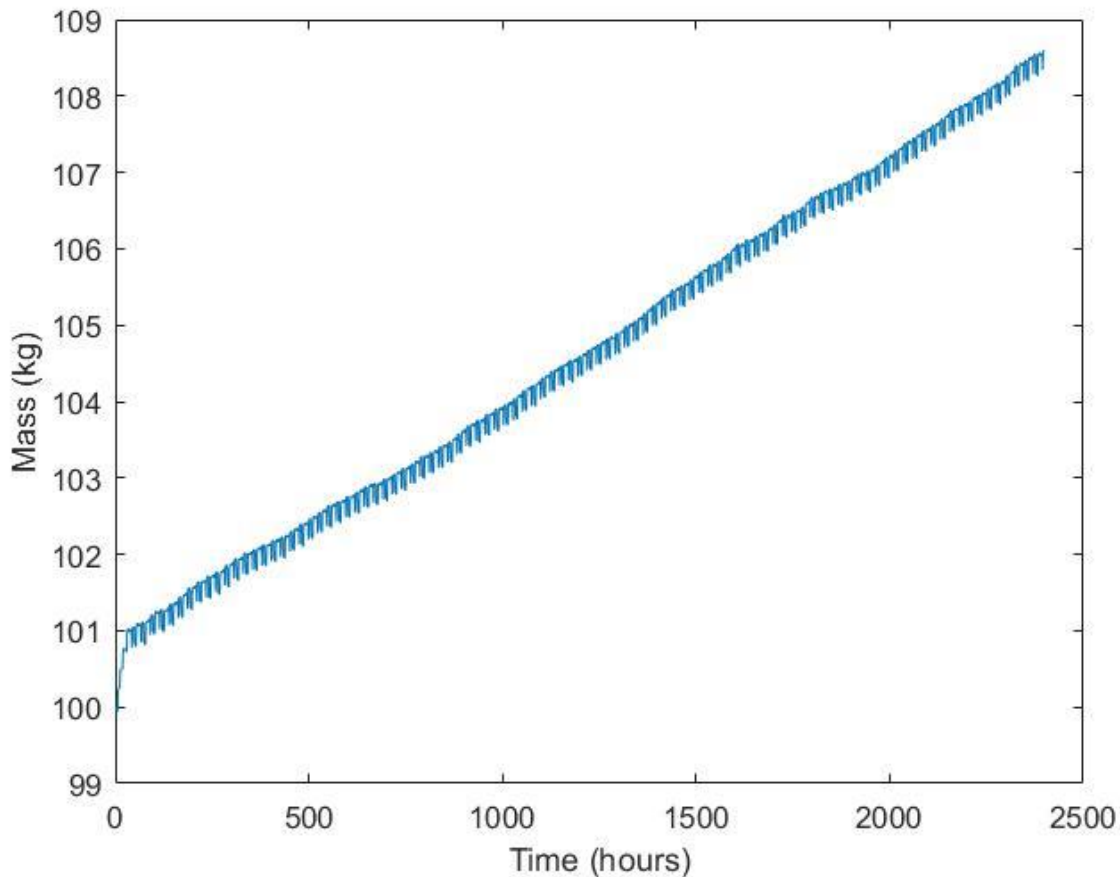


**Figure 22: mass vs time for equilibrium, half fat/half protein diet with BMR calories comprised of half fat and half protein**

Figure 26 displays the results from the protein and fat combination, which caused an end mass of about 105 kg, resulting in a net gain of 5 kg over time. As mentioned on the previous page, this result was expected in accordance with the hypothesis, producing the least mass gain of the three combination tests while having the slowest average digestion time. It should also be noted that an all fat diet produced a gain of roughly 3.5 kg, and one with all protein yielded a gain of about 7 kg. The total gain for half fat/half protein was between these two totals, which lent more



strength to the hypothesis, as it shows that the mass gains in the first three tests (all one macronutrient) were not anomalous, and that they follow the trend that the hypothesis predicts.



**Figure 23: mass vs time for equilibrium, half protein/half carbs; diet with BMR calories comprised of half protein and half carbs**

Figure 27 shows the results of the last of the three combination tests, using an input comprised of half protein and half carbs. The figure demonstrates that this test showed the most mass gain of the three combo tests, as the net mass gain was about 8.5 kg – 3.5 kg more than the fat/protein combination, and 2 kg more than that of fat/carbs. Furthermore, similar to the half protein and half fat combination, this test's end mass lied between that of its constituents' end masses for their respective tests. Protein showed a mass gain of roughly 7 kg, and carbs showed one of

about 11 kg, putting the protein/carbs test's mass gain right in the middle at 8.5 kg. This test was, like the rest, in line with the hypothesis. The weight gain remained proportional to speed of digestion of the simulated diet's components, was in line with expectations, and was consistent with the trend shown by the rest of the diagrams.

### **Model Faults**

In any good experiment, it is always useful and important to reflect on areas where one could have improved or done better, as well as potential areas for error within the conducted tests. Though this model was created in an effort to be as realistic as possible, there were still certain areas of uncertainty that developed throughout the experimentation and they should be noted here. While they did not greatly affect the end results of this study, these uncertainties could have been improved upon given more time and could have made small improvements to the model, making it an ever better representation of the human body.

The first point of potential error lies in the hunger constants, or the numbers used to convert energy debt into a certain amount of eating increase. Though the use of these developed constants yielded expected results, there was no way of knowing they were truly accurate. The constants were developed with logical approaches as described in previous sections, yet, they were estimates nonetheless and should be considered as such when reflecting upon the results of this thesis. Hence, individuals may respond in reality more or less intensely to hunger than depicted in these results, which would lead to different amounts of mass gain over time, potentially causing the perpetual increase of mass at equilibrium calories.

Next, it should be pointed out that, based on the model and produced results, the equation for Basal Metabolic Rate, or BMR, needed to be modified slightly. Though the equation used was effective in the short term before significant mass gain, it proved ineffective in the long run. This effect was a result of the BMR not properly adjusting for mass gain, as the individuals in the tests proceeded to gain weight throughout months while consuming a consistent number of calories. The BMR, which uses body mass as an input, should have accounted for this, eventually causing the mass increase to plateau as body mass reached a sufficient number to account for any excess calories. This is hypothesized to be because the equation was developed using populations to generate data, rather than one individual at various body weights. For instance, a 100 kg man who loses 20 kg may have a different BMR than that of an originally 80 kg man. This, however, is only a possible explanation, and further research would be required to determine the reason behind this phenomenon.

Third, another constant, the energy-to-mass conversion constant, may have been off by a small magnitude from its true value. This number was found to be roughly 3500 surplus or deficit calories per pound of body weight change, which was then converted into cal/kg. The constant at hand may have been another possible cause of the perpetual mass gain found in the simulations, as if it were too low, individuals would be gaining more weight than they should be based off of their net caloric surplus, throwing a system that is built on the concept of equilibrium out of rhythm. Furthermore, when running food input amounts through the equations, the energy on both sides of the equations tended to be slightly unequal, though only by a small magnitude. This effect could have been caused by an erroneous conversion constant, as it would throw the equation from its true balance and cause an energy inequality. Further research would be necessary in order to determine the accuracy of the constant used here.

Lastly, it should be noted that the simulations run here were for one type of individual: a 20 year-old, 100 kg, 172.72 cm tall man. For more accurate and encompassing results, further simulations should be run across different types of individuals, varying in all categories just described. Also, more combinations of food inputs could be utilized in order to further verify the hypothesis and lend more data in support of its claim. Various different combinations of food are possible, and different amounts of targeted energy imbalance could be tested as well (designing for a mass gain or loss). This way, the trends seen in the results could be verified across different individuals, diets, and situations, rather than one individual. This would rule out the possibility of an anomaly and lend strength to the claims made here, as a larger sample size is always more beneficial to increasing result accuracy.

As the human body is so immensely complex, there are a great deal of factors that play into each bodily mechanism, and therefore a lot of room for error. Not only are there more opportunities for something to go wrong, but anyone wishing to model the body in any certain way is going to have to choose only a few of the numerous bodily processes to focus on, and even fewer variables to use in said simulation. A variety of factors and levels of complexity could have been added to this model, like specific types of each macronutrient or hormonal effects based on dietary influence, but given time constraints, they were unable to be explored, and would have made the model too complicated to focus on just the digestion rates of foods and their effects. Therefore, even with all the modifications made along the way, this model still is not a perfect representation of the body, and results may vary further still, simply because of the differences between a given individual's body and the next.

## Conclusions

Though there were imperfections with the model and the methods in which it was run, the simulation results, via tests described in chapter 4, showed consistency with expectations on controlled tests and gave expected results when considering the rate of digestion for various foods. Tests were run to verify that all involved simulated bodily functions were working correctly, and that proper responses were generated under known conditions with verified results. When the expected results were indeed verified, the model's accuracy was proven, and therefore made it a viable means to simulate the body and diet patterns and their respective effects on mass change in an individual.

Tests were then run with said model, and the results from these tests were consistent with the results of the hypothesis. Diets with foods containing macronutrients that digest more quickly produced more mass gain in every experiment done. With this effect being observed every time, more strength was lent to the result. Even more convincing was the trend that was verified by the latter set of experiments, using the combinations of macronutrients. These diets resulted in mass gains that lied perfectly within the range between the macronutrients involved, strengthening the claim to the relationship between mass change and rate of digestion of ingested foods.

With the model's strength being verified and in light of the results detailed above, the hypothesis was confirmed, at least for the individual simulated. The model, that appeared to be accurate from comparison to known data, showed from several simulations that there appears to be a relationship between rate of digestion of foods and mass gain of an individual. The slower the rate of digestion of the foods chosen for consumption, the more less mass gain the individual will see over time, and the opposite appears to be true for foods with a fast rate of digestion.

However, it should be noted that these conclusions were based on results from tests using only caloric equilibrium, and that the individual is assumed to consume strictly the stated inputs, with the only dietary variation resulting from a hunger response.

The simulation model showed that carbohydrates produce the most weight gain in individuals if consumed over time, versus other food types, based on a hunger model where the food ingestion rate is proportional to the rate of caloric deficit. It can be considered plausible that an increase in carbohydrates of such proportions among Americans could have resulted in weight gain among the population. The results of this study may provide a dietary alternative to people who wish to prevent excessive mass gain: this includes changing eating patterns to include more slow-digesting foods. With this knowledge, individuals would be more empowered to make educated decisions in their diet and better suited to achieve and maintain healthy weight levels.

These conclusions are only from tests for in individual and for one specific situation used for the simulation. To generally determine relationships between food types and mass gain, even for the assumptions of the simulation, much more testing is needed to verify the results are consistent among all ages, genders, weights, and heights. Otherwise, the results may not apply to the general population, as they should not be assumed for any other type of individual.

## Appendix: Simulation Code

```

%% set up workspace
clear all; %clear workspace
close all; %closes all figures
clc; %clears command window
%% fill in the simulation parameters
k_h=2; %set gain for hunger (not used yet)
height=172.72; %set height input in cm
age=20; %set age input in yrs
gender=1; %set gender (1 for males, 0 for females)
E_2_M=1/(3500*2.205); %convert 3500 calories/lb to kg per calories
M_2_E=1/(E_2_M); %convert kg to calories
c_fat=9000; %conversion factor for mass to energy of fat (calories per kg)
c_fiber=0; %conversion factor for mass to energy of fiber (calories per kg)
c_protein=4000; %conversion factor for mass to energy of protein (calories per kg)
c_carb=4000; %conversion factor for mass to energy of carbs (calories per kg)

flag_include_mass = 1; % shut on (1) or off (0) the mass dynamics of eating and pooping

hunger_rate_gain = 1/100;

%% Fill the time signals for eating
num_days = 100;
t_end = 24*num_days; % Change this number if you want to run the sim longer! (hrs)
t_eating = (0:0.01:t_end)'; % Units are hours
t_24_hr = mod(t_eating,24); % Convert to 24 hour cycle (temp variable)
BMR = 1985.1;

% Initialize all the food values to zeros
m_dot_fat = 0*t_eating;
m_dot_protein = 0*t_eating;
m_dot_carbs = 0*t_eating;
m_dot_fiber = 0*t_eating;

% Define fractions of types of food to eat (should add to 1)
f_fat = 0; % Fraction of food mass from fat
f_protein = 0.5; % Fraction of food mass from protein
f_carbs = 0.5; % Fraction of food mass from carbs

% Define fraction of food eaten for breakfast, lunch, and dinner (should
% add to 1)
f_breakfast = 1/3;
f_lunch = 1/3;
f_dinner = 1 - f_breakfast - f_lunch;

% Breakfast is from 6 to 7
indices_breakfast = find(t_24_hr>6 & t_24_hr<7);
m_dot_fat(indices_breakfast) = f_breakfast * f_fat * BMR / c_fat * (1/0.968); % kg/hour
m_dot_protein(indices_breakfast) = f_breakfast * f_protein * BMR / c_protein * (1/0.936); % kg/hour
m_dot_carbs(indices_breakfast) = f_breakfast * f_carbs * BMR / c_carb * (1/0.896); % kg/hour
m_dot_fiber(indices_breakfast) = 0; % kg/hour

% Lunch is from 12 to 1
indices_lunch = find(t_24_hr>12 & t_24_hr<13);
m_dot_fat(indices_lunch) = f_lunch * f_fat * BMR / c_fat * (1/0.968); % kg/hour
m_dot_protein(indices_lunch) = f_lunch * f_protein * BMR / c_protein * (1/0.936); % kg/hour
m_dot_carbs(indices_lunch) = f_lunch * f_carbs * BMR / c_carb * (1/0.896); % kg/hour
m_dot_fiber(indices_lunch) = 0; % kg/hour

% Dinner is from 7 to 8
indices_dinner = find(t_24_hr>19 & t_24_hr<20);
m_dot_fat(indices_dinner) = f_dinner * f_fat * BMR / c_fat * (1/0.968); % kg/hour
m_dot_protein(indices_dinner) = f_dinner * f_protein * BMR / c_protein * (1/0.936); % kg/hour
m_dot_carbs(indices_dinner) = f_dinner * f_carbs * BMR / c_carb * (1/0.896); % kg/hour
m_dot_fiber(indices_dinner) = 0; % kg/hour

% Check the inputs via plots of everything
b_fig=figure(123456);
set(b_fig,'Name','Mass inputs vs time');
plot(...)
    t_eating,m_dot_fat,'c',...
    t_eating,m_dot_protein,'r',...
    t_eating,m_dot_carbs,'b',...
    t_eating,m_dot_fiber,'g')
xlabel('Time (hours)');
ylabel('Mass input rates (cal per hr)');
legend('fat','protein','carbs','fiber');

```

```

%% run the simulation
junk=sim('mass_model__6th_order_20170720_BP'); %simulate the model
%% plot the results
a_fig=figure(1);
set(a_fig,'Name','Mass vs Time');
plot(t,m);
xlabel('Time (hours)');
ylabel('Mass (kg)');

if l==0
    a_fig=figure(111111);
    set(a_fig,'Name','Mass_flow_rate of Poop vs Time');
    plot(t,m_dot_poop);
    xlabel('Time (hours)');
    ylabel('Mass Rate of Poop (kg/hr)');

end

if l==0

    a_fig=figure(22222);
    set(a_fig,'Name','Mass_flow_rate of Fat Digestion');
    plot(t,mdot_fat_entering_digestion,'r',...
        t,mdot_fat_leaving_digestion,'b');
    xlabel('Time (hours)');
    ylabel('Mass Rate of Fat Digestion (kg/hr)');
    legend('Fat into digestion','Fat digested');

    a_fig=figure(33333);
    set(a_fig,'Name','Mass of Fat Digestion');
    plot(t,m_fat_entering_digestion,'r',...
        t,m_fat_leaving_digestion,'b');
    xlabel('Time (hours)');
    ylabel('Mass of Fat Digestion (kg)');
    legend('Fat into digestion','Fat digested');

end

if l==1
    b_fig=figure(2);
    set(b_fig,'Name','Energy Debt Rate vs Time');
    plot(t,E_debt);
    xlabel('Time (hours)');
    ylabel('Energy Debt Rate (cal per hr)');

end

if l==0
    b_fig=figure(21);
    set(b_fig,'Name','Energy Debt vs Time');
    plot(t,E_debt_integrated);
    xlabel('Time (hours)');
    ylabel('Energy Debt (cal)')

    b2_fig=figure(6);
    set(b2_fig,'Name','Mass vs Energy Debt');
    plot(E_debt,m);
    xlabel('Energy Debt (cal per hr)');
    ylabel('Mass (kg)');

    c_fig=figure(3);
    set(c_fig,'Name','Metabolised Energy vs Time');
    plot(t,E_metab*24);
    xlabel('Time (hours)');
    ylabel('Metabolised Energy (cal per hr)');

    c2_fig=figure(7);
    set(c2_fig,'Name','Mass vs Metabolised Energy');
    plot(E_metab,m);
    xlabel('Metabolised Energy (cal per hr)');
    ylabel('Mass (kg)');

    d_fig=figure(4);
    set(d_fig,'Name','Energy Supply vs Time');
    plot(t,E_sup);
    xlabel('Time (hours)');
    ylabel('Energy Supply (cal per hr)');

    d2_fig=figure(8);
    set(d2_fig,'Name','Mass vs Energy Supply');
    plot(E_sup,m);
    xlabel('Energy Supply (cal per hr)');

```



```
ylabel('Mass (kg)');

e_fig=figure(5);
set(e_fig,'Name','Mass of Food In vs Time');
plot(t,m_in);
xlabel('Time (hours)');
ylabel('Mass of Food In (kg per hr)');

e2_fig=figure(9);
set(e2_fig,'Name','Mass vs Mass In');
plot(m_in,m);
xlabel('Mass of Food In (kg per hr)');
ylabel('Mass (kg)');
end
```

## BIBLIOGRAPHY

- And, Andrea C Buchholz. "Andrea C Buchholz." *The American Journal of Clinical Nutrition*.  
Department of Nutritional Sciences, University of Wisconsin-Madison, 01 May 2004. 24  
July 2017.
- Bentley, Jeanine, and Linda Kantor. "Food Availability (Per Capita) Data System." *Food  
Availability (Per Capita) Data System*. United States Department of Agriculture -  
Economic Research Service, 23 Nov. 2016. 24 July 2017.
- Casazza, K., Fontaine, K. R., Astrup, A., Birch, L. L., Brown, A. W., Bohan Brown, M. M., ...  
Allison, D. B. (2013). Myths, presumptions, and facts about obesity. *New England  
Journal of Medicine*, 368(5), 446–54.
- Chaumont, Caroline. "How Do You Measure Hunger? | WFP | United Nations World Food  
Programme - Fighting Hunger Worldwide." *UN World Food Programme*. N.p., 03 Jan.  
2009. 24 July 2017.
- Flegal, K. M., B. I. Graubard, D. F. Williamson, and M. H. Gail. "Excess Deaths Associated with  
Underweight, Overweight, and Obesity." *JAMA*. U.S. National Library of Medicine, 20  
Apr. 2005. 24 July 2017.
- Frankenfield, D., L. Roth-Yousey, and C. Compher. "Comparison of Predictive Equations for  
Resting Metabolic Rate in Healthy Nonobese and Obese Adults: A Systematic Review."  
*Journal of the American Dietetic Association*. U.S. National Library of Medicine, May  
2005. 24 July 2017.

Finkelstein, E. A., J. G. Trogon, J. W. Cohen, and W. Dietz. "Annual Medical Spending

Attributable to Obesity: Payer-and Service-specific Estimates." *Health Affairs (Project Hope)*. U.S. National Library of Medicine, 28 Oct. 2009. 24 July 2017.

Garrow, J. S., and J. Webster. "Quetelet's Index (W/H<sup>2</sup>) as a Measure of Fatness." *International Journal of Obesity*. U.S. National Library of Medicine, 1985. 24 July 2017.

"GI Enzymes and Their Importance in Digestion." *Everything You Need to Know about Your GI System*. Penn State University, 10 Mar. 2014. 24 July 2017.

Gibbons, Gary H. "Causes." *National Heart Lung and Blood Institute*. U.S. Department of Health and Human Services, 23 Feb. 2017. 24 July 2017.

Hall, Kevin D., Ross A. Hammond, and Hazhir Rahmandad. "Dynamic Interplay Among Homeostatic, Hedonic, and Cognitive Feedback Circuits Regulating Body Weight." *American Journal of Public Health*. American Public Health Association, July 2014. 24 July 2017.

Hall, Kevin D. "What Is the Required Energy Deficit per Unit Weight Loss?" *International Journal of Obesity*. U.S. National Library of Medicine, Mar. 2008. 24 July 2017.

Hur, Sun Jin, Beong Ou Lim, Eric A. Decker, and Julian McLements. "In Vitro Human Digestion Models for Food Applications." *In Vitro Human Digestion Models for Food Applications - ScienceDirect*. ScienceDirect, 16 Aug. 2010. 24 July 2017.

Hussain, S. S., & Bloom, S. R. (2012). The regulation of food intake by the gut-brain axis: implications for obesity. *International Journal of Obesity*, 37(5), 625–633.

Jonnalagadda, Satya S., Lisa Harnack, Rui Hai Liu, Nicola McKeown, Chris Seal, Simin Liu, and George C. Fahey. "Putting the Whole Grain Puzzle Together: Health Benefits Associated with Whole Grains—Summary of American Society for Nutrition 2010

- Satellite Symposium." *The Journal of Nutrition*. American Society for Nutrition, May 2011. 24 July 2017.
- Kopelman, P. "Health Risks Associated with Overweight and Obesity." *Obesity Reviews*. Blackwell Publishing Ltd, 19 Feb. 2007. 24 July 2017.
- Kurai, H. "[Differential Diagnosis of Acute Diarrhea]." *Nihon Rinsho. Japanese Journal of Clinical Medicine*. U.S. National Library of Medicine, Aug. 2012. 24 July 2017.
- "The Liver & Blood Sugar." *UCSF Medical Center*. University of California, San Francisco, 2007. 24 July 2017.
- Marieb, Elaine Nicpon, and Katja Hoehn. *Human Anatomy & Physiology*. San Francisco, CA 94111: Pearson Benjamin Cummings, 2007. Print.
- Mifflin, M. D., S. T St Jeor, L. A. Hill, B. J. Scott, and And S A Daugherty. "M D Mifflin." *The American Journal of Clinical Nutrition*. American Society for Clinical Nutrition, 01 Feb. 1990. 24 July 2017.
- MRP, Cynthia L. Ogden PhD. "Prevalence of Obesity and Trends in Body Mass Index Among US Children and Adolescents, 1999-2010." *JAMA*. American Medical Association, 01 Feb. 2012. 24 July 2017.
- "Obesity Information." *HEART*. American Heart Association, 18 Oct. 2016. 24 July 2017.
- Ogden, C. L. L., Carroll, M. D. D., Kit, B. K. K., & Flegal, K. M. M. (2014). Prevalence of childhood and adult obesity in the United States, 2011-2012. *Jama*, 311(8), 806–814.
- Olson, S., & Board, N. (2014). *The Current State of Obesity Solutions in the United States : Workshop Summary Steve Olson , Rapporteur ; Roundtable on Obesity Solutions ; Food and*.

- Rose, C., A. Parker, B. Jefferson, and E. Cartmell. "The Characterization of Feces and Urine: A Review of the Literature to Inform Advanced Treatment Technology." *Critical Reviews in Environmental Science and Technology*. Taylor & Francis, 02 Sept. 2015. 24 July 2017.
- Rosen, Evan D., and Bruce M. Spiegelman. "Adipocytes as Regulators of Energy Balance and Glucose Homeostasis." *Nature*. U.S. National Library of Medicine, 14 Dec. 2006. 24 July 2017.
- Rosen, E. D., & Spiegelman, B. M. (2014). What we talk about when we talk about fat. *Cell*, 156(1–2), 20–44.
- Rosenbaum, M., & Leibel, R. L. (2010). Adaptive thermogenesis in humans. *International Journal of Obesity* (2005), 34 Suppl 1(S1), S47-55.
- Rosenbaum, M., & Leibel, R. L. (2016). Models of energy homeostasis in response to maintenance of reduced body weight. *Obesity*, 24(8), 1620–1629.
- Shin, A. C., Zheng, H., & Berthoud, H. R. (2009). An expanded view of energy homeostasis: Neural integration of metabolic, cognitive, and emotional drives to eat. *Physiology and Behavior*, 97(5), 572–580.
- Speakman, J. R., Levitsky, D. a, Allison, D. B., Bray, M. S., de Castro, J. M., Clegg, D. J., ... Westerterp-Plantenga, M. S. (2011). Set points, settling points and some alternative models: theoretical options to understand how genes and environments combine to regulate body adiposity. *Disease Models & Mechanisms*, 4(6), 733–45.
- Sturm, R., J. S. Ringel, and T. Andreyeva. "Increasing Obesity Rates and Disability Trends." *Health Affairs (Project Hope)*. U.S. National Library of Medicine, 23 Apr. 2004. 24 July 2017.

Tchernof, A., & Després, J.-P. (2013). Pathophysiology of human visceral obesity: an update.

*Physiological Reviews*, 93(1), 359–404.

U.S. Department of Health and Human Services and U.S. Department of Agriculture. 2015–2020

Dietary Guidelines for Americans. 8th Edition. December 2015.

Vandevijvere, S., Chow, C. C., Hall, K. D., & Swinburn, B. A. (2015). Increased food energy

supply as a major driver of the obesity epidemic : a global analysis. *Bull World Health Organ*, 93(November 2014), 446–456.

Wallace, Michael. "The Digestive System & How It Works." *National Institute of Diabetes and*

*Digestive and Kidney Diseases*. U.S. Department of Health and Human Services, 01 Sept. 2013. 24 July 2017.

Wierdsma, Nicolette J., Job HC Peters, Peter JM Weijs, Martjin B. Keur, Armand RJ Girbes, Ad

A Van Bodegraven, and Albertus Beishuizen. "Malabsorption and Nutritional Balance in the ICU: Fecal Weight as a Biomarker: A Prospective Observational Pilot Study."

*Critical Care*. BioMed Central, 2011. 24 July 2017.

Woods, S. C. (2013). Metabolic signals and food intake. Forty years of progress. *Appetite*, 71,

440–444.

---

**Academic Vita of Bradford A. Pechin**  
Bap5346@psu.edu

---

Education

Major(s) and Minor(s): Mechanical Engineering

Honors: Mechanical Engineering

Thesis Title: Modeling of Food to Fuel Conversion and its Effect on Societal Weight Problems

Thesis Supervisor: Dr. Sean Brennan

Work Experience

Date: June-August 2015, June-August 2016

Title: Intern

Description: Engineering Support intern, Power Sales intern

Institution/Company (including location): Exelon Corporation, Kennett Square, PA

Supervisor's Name: Joe Schival, Kathy Willard

Grants Received *[whether from Schreyer Honors College (e.g., Schreyer Ambassador Awards) or from other sources; please specify]*: Provost Award

Awards: Magna Cum Laude

Professional Memberships: Phi Eta Sigma Honor Society

Publications: N/A

Presentations: N/A

Community Service Involvement: Alpha Firehouse, Rugby involvement

International Education (including service-learning abroad): N/A

Language Proficiency: Spanish