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Diet and Health Capital: An American Case Study

Abstract

This paper begins to model diet's contribution to national stocks of Health Capital as defined in Kenneth Arrow et al.'s (2012) "Sustainability and the Measurement of Wealth" by developing a set of equations that link the diet of a nation's average citizen to that nation's per capita welfare over time. The model is applied to the American diet from 2005 to 2009. This research finds that even a small change in diet can have a large impact in whether or not a nation is sustainable over time.

Keywords

Health Capital, BMI, diet

Cover Page Footnote

I would like to acknowledge Sabina Shaikh, Tara Massad and Trevor Gallen for their help in writing this paper.

Introduction

This paper seeks to advance Kenneth Arrow *et al.*'s (2012) concept of health capital by modeling the role of diet in health. Last year, Arrow and his colleagues sought to improve the way in which economists measure a nation's capacity to be sustainable. In their paper, Arrow *et al.* conceptually develop health capital as a framework to discuss how the health of a nation's average citizen impacts per capita welfare levels. They find that "health capital makes a huge difference to our estimates of changes in per capita wealth. The value of this capital is more than twice as large as all other forms of capital¹ combined." And, as they determine whether a country is sustainable over time by evaluating changes in holistic wealth, health capital has a huge impact on how sustainability is measured. They gracefully end their paper with the caveat that, while they have done a phenomenal job developing health capital as a tool to advance their argument, "the analysis of health capital is an innovation that will require much further study to understand."

This paper endeavors to connect the diet of the average citizen of a nation to that nation's capacity to be sustainable. To this end, it will develop a series of links: that of diet to energy imbalance, energy imbalance (over time) to weight gain, weight gain to various detrimental health effects² and mortality rates, and mortality rates to some number of quality adjusted life years lost for the population. Then, the model developed in this paper borrows two equations from Arrow *et al.*'s to find diet's effect on a nation's health capital and, in turn, on its capacity to be sustainable. This paper will then apply this model to the average American diet from 2005 to 2009. The findings of this exercise frame a discussion of potential improvements to the model and to the institutions surrounding diet in America. The key assumptions made in this paper are laid out as they become relevant. As such, it is important to note here that this paper assumes that malnutrition can be ignored. For a discussion of the American diet, this assumption is not excessively crippling. However, in some countries, malnutrition is a much more important consideration than is obesity. Additionally, this paper will ignore changes in morbidity that result from changes in a nation's average BMI. This model must be expanded before it can relate to countries dissimilar to America.³ Lastly, a wider question remains unanswered: how much of health capital is explained by diet? This paper tentatively hypothesizes that diet only plays an important role in health capital in the absence of a few factors, including exposure to warfare and disease and access to health care. In America, the values attached to these other factors may be close to zero so that the largest detriments to health capital are the costs accrued by the diet-driven increases in mortality and morbidity.

Literature Review

In the existing literature, the links between an average citizen's diet and a nation's capacity to be sustainable range from being well discussed to being relatively unremarked upon. In the most extreme cases, no consensus exists on topics discussed in this paper. For example, it is unclear

¹ human capital, natural capital, reproducible capital, etc.

² obesity, diabetes, coronary heart disease, stroke, and some cancers.

³ It is my hope that this short coming, and others, will be rectified in later papers.

exactly how much of energy imbalance is due to energy intake, energy expenditure, or biological and genetic factors. The following is a review of the existing works that this paper will draw upon, including a brief summary of Arrow *et al.*'s seminal paper.

The American Government has published data on dietary trends for the past four decades that vary in completeness depending on food type (United States Department of Agriculture [USDA], 2002). This data provides an excellent picture of the average American diet in 2000, characterized by high consumption of added fats and sugars and low consumption of vegetables as compared to recommended consumption patterns. It also shows that Americans are eating more on average than they were in any past decade, (USDA, 2002). In 2000, American's were consuming just under 2,700 calories per person per day, 530 calories above the 1970 level of consumption, (USDA, 2002). This case study will clearly display this data.

A substantial amount of literature exists on obesity and malnutrition rates worldwide. The Behavioral Risk Factor Surveillance System (BRFSS), and the National Health and Nutritional Examination Survey, as well as others, publish data on obesity prevalence and trends. The CIA World Factbook (Central Intelligence Agency [CIA], 2012) reports that 33.90% of American adults are obese. That is, they have a body mass index (BMI) equal to or greater than 30, where BMI is measured as $\frac{\text{weight}}{(\text{height})^2}$.⁴ This positions the United States as the sixth most obese nation in the world, (CIA, 2012). Olshansky *et al.* (2005) note that "the prevalence of obesity among adults in the United States increased by approximately 50 percent per decade throughout the 1980s and 1990s... and that "two thirds of adults in the United States today are obese or overweight." They (Olshansky *et al.*, 2005) further note that "28 percent of men, 34 percent of women, and nearly 50 percent of non-Hispanic black women are currently obese." Additionally, they (Olshansky *et al.*, 2005) write, "the distribution of body-mass index has shifted in a skewed fashion such that the proportion of people with extreme obesity has increased at an especially rapid rate."⁵ The World Health Organization publishes a rank of the world's countries by mortality rates due to malnutrition, (WHO 2010). While obesity is an epidemic in America and has widespread health effects, malnutrition only kills one American in one hundred thousand per year.

Next, there is a wealth of information concerning the mechanics of weight gain and the observed increase in obesity rates over the past several decades. Scientists agree that weight gain (loss) is the result of a positive (negative) imbalance in energy intake over time. However, as noted above, no consensus exists as to exactly how much energy intake, energy expenditure and biological considerations affect changes in weight. This paper will follow Jeffery and Linde (2005) who argue that changes in energy intake, more so than either of the other factors, influence changes in weight. They also posit that changes in energy expenditure are the next most influential for weight gain or loss, followed by biological and genetic factors. They write: "The cause of the obesity epidemic that has affected the world for the last 30 years remains unknown. Although changes in body weight and fatness are surely the result of changes in energy intake and energy expenditure that are mediated by changes in food and activity choices, clear data identifying the specific contribution of energy intake vs. energy expenditure ... are not

⁴ This measurement will be used throughout the paper. Note that BMI is an imperfect measurement of weight and health. Specifically, it may misreport the health of athletes and those with large amounts of muscle mass.

⁵ This will increase the average American BMI.

available. ...consideration of both biological and behavioral aspects of energy intake and utilization strongly favor change in **energy intake as the most likely cause,**” (emphasis added, Jeffery & Linde, 2005). Their full argument is worth reading but a short summary will suffice for the purposes of this paper: they argue that the obesity epidemic sprung up too quickly for the changes to be attributable to any genetic or biological change and that changes in energy expenditure have been too minimal to explain very much of the increase in average American BMI. And, there has been an unhealthy change in American diet (and an increase in the sheer amount of calories consumed per day). So, they conclude, it is probable that changes in weight are most attributable to diet, at least in the American case.

As for how to rigorously quantify diet, this paper will follow Linda Kantor (1998), who published the first analysis of how much average diet deviates from recommended diet. In her 1998 study, she coalesced 27 years of American food pyramid dietary recommendations. This allowed her to achieve a food pyramid that she assumed to be more accurate than any of the individual constituent food pyramids. This paper will make the same assumption. Then, she contrasted actual dietary patterns to the recommendations by adapting two sets of data: a set detailing amounts and types of food available to Americans and actual consumption data. She is then able to show how much average diet differs from recommended diet. Later, with Judy Putnam and Jane Allshouse (2002), she updated these measures to fit consumption data from 2000. This paper uses Kantor’s method to quantify a nation’s diet. And, this paper will make reference to recent findings (Basu, Yoffe, Hills, & Lustig 2013) that suggest that calories from different food groups impact weight gain by differing degrees.

There is no disagreement as to the fact that obesity and certain diet related health conditions can reduce life span. There is disagreement, however, as to how much they do so. Olshansky *et al.* (2005) find that obesity can potentially shorten expected lifespan (for the entire population) by somewhere between 1/3 of a year to a full year. Peto *et al.* (2010) find that “an increase of 2 in the BMI in overweight populations” may result in a reduction in life expectancy of up to one year. Elizabeth Frazão (1999) looks at four diseases that changes in BMI impacts (Coronary Heart Disease, Stroke, Diabetes and Cancer) and how they affect lifespan by measuring life years lost due to premature death – defined as death after 55 but before 75. She finds that these four diseases contribute to a loss of quality adjusted life years roughly equal to 1,000,940 life years per year (the number of deaths multiplied by how premature the death was). So, there is a significant amount of disagreement about the exact value of the covariance between BMI and life expectancy. This paper will make an estimate as to the value of that covariance by drawing heavily upon a meta-analysis of 57 studies published in *The Lancet* (United States National Center for Biotechnology Information [USNCBI], 2009). This study estimates that obesity and diet related diseases may shorten the life spans of the obese by anywhere between one and ten years, depending on the severity of the case.

The last major work that this paper will draw upon is Arrow *et al.*’s (2012) “Sustainability and the Measurement of Welfare.” The economists set up a holistic measurement of sustainability in terms of per capita welfare, and measure welfare as a function of a number of variables including human capital, natural capital, and reproducible capital. They also consider “the meaning of wealth in a non-optimal course of development, the role of ecosystem resources and global public goods, the implications of resource exhaustion in a world of international trade, and the measurement of health as a form of capital.” To develop the concept of health capital, they employ a measurement of the statistical value of human life to find the value of an

additional quality adjusted life year⁶ for an individual. This allows them to quantify changes in average lifespans. They (Arrow *et al.* 2012) write: “What matters for sustainability are the changes in the values of the various forms of capital.” So, changes in life span allow them to consider health capital’s role in sustainability. Finally, a nation is said to meet their sustainability standard if the change over time of all combined welfare considerations sum to a non-negative number, (Arrow *et al.* 2012).

The Basic Model

This model takes advantage of the first law of thermodynamics: all energy not consumed or expelled by the body is stored. The creation of fat is the easiest way for the body to achieve this and, thus, when more energy is consumed than used, BMI increases for an individual. Conversely, when the body uses more energy than is available, stored energy must be used and BMI decreases. For those with moderate or high BMIs, this results in a loss of fat. For those with low BMIs, this can result in the destruction of muscle tissue – a very negative reality for those suffering from malnutrition.

Diet and Energy Imbalance

The first link to be established is that between diet and energy imbalance – a link that will be applicable to both weight gain and weight loss and which the rest of the paper will build on. It has three components: diet (energy intake), energy expenditure, and some genetic or biological factor that will be referred to as the “biologic factor” from here on out. The biological factor describes the difference in metabolic rates across a population. As will become apparent in a moment, the aggregate biological factor will be equal to one and so will drop out of the equation. Equation (1) gives the most basic description of the relationship between energy imbalance, energy intake, energy expenditure, and the biological factor.

(1) ***Energy Imbalance =***

$$\left[\left(\mathbf{1} + \left| \frac{\delta_{recommended} - \delta_{actual}}{\delta_{recommended}} \right| \right) (\lambda)(energy\ intake) * (\mathbf{1} - \lambda - \gamma)(biological\ factor) \right. \\ \left. + (\gamma)(energy\ expenditure) * (\mathbf{1} - \lambda - \gamma)(biological\ factor) \right]$$

Here, γ is the coefficient for *energy expenditure* and describes how much it contributes to energy imbalances. λ is the weight for *energy intake*: it describes how much diet affects the total energy imbalance. This notation has the convenient result that no modifier is needed to discuss the importance of the biological factor: its coefficient is one minus the sum of γ and λ . Finally, δ is the total number of calories consumed so that $\left(\mathbf{1} + \left| \frac{\delta_{recommended} - \delta_{actual}}{\delta_{recommended}} \right| \right)$ is a weight that describes how many more (or less) calories are consumed as a part of the average

⁶ Adjusted for age and nationality

diet than is recommended. As has been noted, there exists no consensus on what values should be assigned to γ or to λ . This paper will estimate values for them by considering Jeffery and Linde's (2005) conclusions and the role of epigenetics in weight gain.⁷ Specifically, in the case study, this paper will weight γ at 0.367 and λ at 0.483, so that $(1 - \gamma - \lambda)$ is equal to 0.15.⁸ Importantly, the biological factor will be treated as a constant as it will not change significantly over the course of a life span, perhaps even with epigenetic changes considered. And, the biological factor will not matter when considering only one nation over a period of time of less than 35 years. As may be determined given future research, it may not even differ very much from nation to nation, or over a much longer period of time.⁹ Dropping the biological factor and adding in these weights yields equation (2).

(2)

Energy Imbalance =

$$\left[\left(1 + \left| \frac{\delta_{recommended} - \delta_{actual}}{\delta_{recommended}} \right| \right) (0.483)(energy\ intake) \right] \\ + (0.367)(energy\ expenditure)$$

Here, *energy intake* is a function of diet to be discussed shortly.¹⁰ It will be a positive number measured in kilocalories per day. *Energy expenditure* is also a function, however this paper does not attempt to comprehensively model it. It will be a negative number also measured in kilocalories per day, and $\left| \frac{\delta_{recommended} - \delta_{actual}}{\delta_{recommended}} \right|$ is, theoretically, a number between 0 and ∞ with no units.¹¹ This bound occurs because $\delta_{recommended}$ and δ_{actual} are measured in kilocalories per day where δ_{actual} is the number of kilocalories consumed by someone following a nation's average diet and $\delta_{recommended}$ is the recommended consumption of kilocalories per day. Importantly, the sign associated with energy imbalance has to be maintained over time in order for average BMI to change appreciably. This will be incorporated into the model after *energy intake* is defined. This measure is the absolute value of recommended expenditure of kilocalories per day minus actual energy expenditure in kilocalories per day. The recommended expenditure of kilocalories per day is equal to the recommended number of kilocalories per day consumed, $\delta_{recommended}$, multiplied by the relative importance of *energy expenditure* in *Energy Imbalance*. So, *Energy Imbalance* is measured in kilocalories per day.

⁷ This is a contested topic. It may be the case, however, that the covariance between the prevalence of obesity in a nation and the value of γ may increase on a generational basis. This might greatly impact this model.

⁸ The reasons for valuing the weights at 0.483, 0.367 and 0.15 are given in Appendix I.

⁹ It may however, vary from relatively wealthy countries (where obesity is a leading concern) to relatively poor countries (where malnutrition is a leading concern).

¹⁰ See equation (3)

¹¹ Realistically, the value of the upper limit of $\left(1 + \left| \frac{\delta_{recommended} - \delta_{actual}}{\delta_{recommended}} \right| \right)$ will probably not exceed 2 for someone observing the nation's average diet.

Quantifying Energy Intake

This link will be established by following Kantor's (1998) model of how the average American diet should be modified to meet US recommendations. Here, quantified energy intake (quantified diet) is a function of how much actual consumption differs from recommended consumption. It is described by equation (3) where (*food group**) is the absolute value of the recommended consumption of that food group (in kilocalories per day) minus actual consumption of that food group (in kilocalories per day). And, *a - g* are unitless values that sum to 1. This results in an *energy intake* value measured in kilocalories per day.

$$(3) \quad \text{energy intake} = \\ a(\text{grains} *) + b(\text{vegetables} *) + c(\text{fruits} *) + d(\text{dairy} *) + e(\text{meats} *) \\ + f(\text{added fats} *) + g(\text{added sugars} *)$$

Here, *a - g* are coefficients that act as weighting factors. They describe how insidious a deviation from the recommended level of consumption is for a certain food group. This is to say, each coefficient represents how much a calorie from each food group differs in its impact on *Energy Imbalance*. These coefficients are necessary because, as one journalist puts it, "not all calories are created equal," (Bittman, 2013). For example, if it turns out that eating a little more added sugar than is recommended (in calories) leads to a relatively large change in *Energy Imbalance*, the value of *g* will be relatively high. A recent study (Basu, Yoffe, Hills, & Lustig 2013) suggests that is the case. Importantly, this model can account for shifts in the value of these coefficients over time by using averages for *a - g*.¹² Again, there is no consensus on what values to assign to these coefficients.¹³

Energy Imbalance, Weight Gain and Obesity

The first step in describing weight gain is identifying some average value for energy imbalance over a specified period of time. This may be obtained by finding $\frac{\text{Energy Imbalance}_{(\text{period } 2)} - \text{Energy Imbalance}_{(\text{period } 1)}}{2}$. Then this value, denoted by *Energy Imbalance_{avg}*, can be plugged into equation (4). This model assumes that changes in weight are not impacted by deviations from the average caloric intake within the timeframe considered. Letting μ be a conversion rate measured in weight per calorie per day, we get equation (4) – the mathematical relationship between a change in BMI and *Energy Imbalance* over time.

¹² For example, beef may have become less healthy on average as a result of the increased prevalence of CAFO's in the American meat production system. This would be reflected in this model by an increase in the value of the weight assigned to *meats* *.

¹³ See Appendix I for an attempt to derive these values by examining the American diet from 1970 – 2000.

$$(4) \quad \Delta BMI_{avg} = \frac{weight_{period 1} + \mu * (Energy Imbalance_{avg})}{(height_{period 2})^2} - \frac{weight_{period 1}}{(height_{period 1})^2}$$

Equation (4) is inspired by the National Heart, Lung and Blood Institute's "Balancing Food and Activity" web page, (USDA 2013). They say that more calories consumed (burned) than burned (consumed) yields a weight gain (loss) and give some estimation as to a weight for μ by positing that "eating just 150 calories more a day than you burn can lead to an extra 5 pounds over 6 months. That's a gain of 10 pounds a year," (USDA 2013). Extrapolating, we get that one calorie extra per day results in a gain of .000182 pounds (.000082 kilograms).¹⁴ This is the value of μ employed in the case study.¹⁵ This value does not account for the fact that a calorie from one type of food may not equal a calorie from another type of food – Equation (3) already attempts to do that. Note that this value is an average for men and women and that, when strictly considering the theoretical implications of this paper, this specific value may be ignored.

Equation (5) rearranges and simplifies equation (4). It holds height in a population constant over time – an assumption that will not hold true for some populations in some time periods. A population recovering from a devastating famine, for instance, may witness an increase in height over time. Immigration and emigration may also result in changes in average height for specific populations.

$$(5) \quad Energy Imbalance_{avg} = \frac{1}{\mu} * \Delta BMI * (height)^2$$

So, to this point, there are links established that describe how adherence to a certain diet over time affects the BMI of the average individual in a population over that time period.

Obesity and Changes in Mortality Rates

Now equation (6), the relationship between changes in BMI and changes in mortality rates can be developed.¹⁶ This has been well studied and there exists a meta-analysis of 57 papers on this topic, (USNCBI, 2009). This work will draw heavily on the findings of this meta-analysis: that, for BMIs above 25, a positive correlation exists between BMI and mortality rate and, for BMIs below 25, a negative correlation exists between the two.¹⁷ The result is a U-shaped curve as presented in Graph 3 in Appendix I. This also reflected in Chart 1 in Appendix I. The authors of this paper summarize their findings by writing: "BMI is in itself a strong predictor of

¹⁴ This is in line with Jeffery and Linde who write: "For a man of average height, 30 percent excess body weight represents about 150,000 kcal of energy storage as fat ... A daily error in energy balance of 100kcal⁴ would be capable of producing a net increase in energy storage of this magnitude in less than five years. To produce obesity in a person over a 20-year time span, a very common course, requires a daily error of only 25kcal." They go on to say that a 300 calorie imbalance per day could account for the American obesity epidemic.

- Jeffery and Linde, "Evolving Environmental Factors," page 57.

¹⁵ See page 16 for an example.

¹⁶ Obesity is rarely the cause of death in itself. The real cause of death is a group of diseases and disorders collectively referred to as Metabolic Syndrome.

¹⁷ This will be important in the formulation of a model to describe malnutrition.

overall mortality both above and below the apparent optimum of about 22.5–25 kg/m². The progressive excess mortality above this range is due mainly to vascular disease and is probably largely causal. At 30–35 kg/m², median survival is reduced by 2–4 years; at 40–45 kg/m², it is reduced by 8–10 years.” While this is important for this paper on a whole, they (USNCBI, 2009) present another finding that is more directly important for the formation of equation (6): “each 5 kg/m² higher BMI [than this optimal range is] on average associated with about 30% higher overall mortality.” Extrapolating, every increase in BMI of 1 kg/m² roughly results in a 6% increase in mortality rate. So, from this meta-analysis, equation (6) is derived. It describes the relationship between $\Delta avgBMI_{period\ 2-period\ 1}$ and change in mortality rate, σ .

$$(6) \quad \sigma = \varphi * \Delta BMI$$

Here, φ is the above mentioned conversion rate, a 6% increase in mortality rate per 1 kg/m². Plugging this value in results in the following: $\sigma = (.06) * (\Delta BMI)$. This is to say, an additional six people per thousand in a population die (per year) for every unit increase in average BMI.

Mortality Rate and Life Years Lost

It is now necessary to determine how premature these deaths are on average. This value may be multiplied by σ to determine the average number of life years lost in a population due to an adherence to some diet for a period of time. Following Frazão (1999), individuals who succumb to diet related diseases (not including malnutrition) rarely do so before the age of 55. And, a death cannot be labeled premature if the individual dies after she has surpassed the expected life span. So the number of life years lost due to premature death is described by equation (7). This paper again follows Frazão in that equation (7) splits *Number of Life Years Lost* in two: premature deaths between ages 55¹⁸ and 64, and premature deaths between the ages 65 and the life expectancy. This allows the model more accuracy as the number of premature deaths is generally skewed so that extremely premature deaths are rarer than moderately premature deaths.

$$(7) \quad \text{Number of Life Years Lost} =$$

$$\left(\frac{64 + 55}{2}\right) (\text{number of premature deaths in individuals between 55 and 64 years of age})$$

$$+ \left(\frac{\text{expected life span} + 64}{2}\right) \left(\text{number of premature deaths in individuals younger than the expected life span but older than 55}\right)$$

Here, the values $\left(\frac{64+55}{2}\right)$ and $\left(\frac{\text{expected life span}+64}{2}\right)$ detail the average prematurity of deaths for each group. Frazão (1999) finds that the number of these deaths that occur between the ages of

¹⁸ 55 is retirement age. This may play a role in the impact of increased mortality from BMI. However, in America at least, most of the individuals harmed by an increase in the average BMI are poor who may retire later 55. This will be brought up again in the discussion section of this paper.

55 and 64 account for approximately 1/3 of premature diet related deaths. So equation (7) can be rewritten.

$$(8) \quad \text{Number of Life Years Lost} = \left[\text{expected life span} - \left(\left(\frac{64+55}{6} \right) + \left(\frac{\text{expected life span}+64}{3} \right) \right) \right] (\text{number of premature deaths})$$

So, for example, when the average expected life span (for females and males) is 80, this model calculates that the average prematurity of death from an increase in BMI is just over 12 years. The model assumes that 3/4 of the deaths that occur as a result of increases in the average BMI will be premature (with the other 1/4 being allotted to individuals older than the expected life span or younger than 55). While it is safe to assume that not all of the deaths incurred by an increase in average BMI come between the ages of 55 and 80, this 3/4 value is largely arbitrary and may underestimate the number of life years lost.

$$(9) \quad \omega = \text{Number of Life Years Lost} = \left[\text{expected life span} - \left(\left(\frac{64 + 55}{6} \right) + \left(\frac{\text{expected life span} + 64}{3} \right) \right) \right] * \left[\left(\frac{3}{4} \sigma \right) \left(\frac{\varepsilon_{\text{period 1}} + \varepsilon_{\text{period 2}}}{2} \right) \right]$$

Here, ε is the number of individuals younger than the life expectancy but older than 55 in a population. The value of ε is averaged over time so as to account for the eventuality of individuals leaving and entering this age group at uneven rates. ε is drawn from data while σ is calibrated using the developed model. The result is that ω is equal to the number of premature deaths times how premature they are.

Life Years Lost and the Impact on a Nation's per capita Health Capital

The link between life years lost to a population and that nation's capacity to be sustainable is developed by Arrow *et al.* (2012). They write: "The value of the health capital for an individual is the expected discounted years of life remaining multiplied by the value of an additional year of life (which is assumed to be independent of age)." So, the impact on the health capital of a nation's average individual from a change in the average BMI in a nation is a function of how much that change reduces expected discounted years of life remaining. This is represented by equation (12). Before developing this link, however, it is necessary to define the value of an additional life year. Arrow *et al.* (2012) do this – the following two equations are taken from their paper. Here, VSL is the value of a statistical life for a nation.¹⁹ Additionally, h

¹⁹ Arrow *et al.* (2012) write: "Viscusi and Aldy (2003) performed a cross-country meta-analysis and concluded that the VSL in other countries is approximately proportional to the 0.6 power of per capita GDP." Viscusi also argues that there is a societal benefit in avoided health care costs for sick individuals dying earlier. This

is the value of an additional life year, $\pi(a)$ is the proportion of a population at age a , people discount future additional life years at a rate 0.05, and $f(T|T \geq a)$ is the conditional mortality rate, defined in equation (10), where $f(T)$ is the probability density that someone born will die at age T and $F(T)$ is the corresponding cumulative distribution.

$$(10) \quad f(T|T \geq a) = \begin{cases} 0, & T < a \\ \frac{f(T)}{[1-F(a)]}, & T \geq a \end{cases}$$

$$(11) \quad h = VSL / \left(\sum_{a=0}^{100} [\pi(a) (\sum_{T=0}^{100} (f(T|T \geq a) (\sum_{t=0}^{T-a} ((1 - .05)^t)))] \right)$$

However, an increase in mortality rate results in some decrease in life expectancy and, increasing morbidity will adversely affect this calculation. So, as the amount of time that a certain diet is adhered to increases, the more the values of $f(T)$ and $F(T)$ in period two will differ from their values in period 1. So, as *BMI* increases, the value of an additional year of life decreases. But, for simplicity, in this paper the value of h will be held constant over the timeframe considered. This will underestimate the impact of diet on a nation's stock of health capital. This impact is a dollar amount given by the following:

$$(12) \quad \text{Negative Impact on a Nation's a Health Capital} = (h * \omega)$$

Equation (13) describes how an adherence to a certain diet for a specified period of time impacts a nation's health capital. As the average *BMI* in a nation increases, health capital decreases (for nations with a starting average *BMI* greater than 25).

$$(13) \quad f(HC) = (\theta - \omega)h$$

Here, $f(HC)$ is a function that demonstrates how health capital in a nation decreases with an increase in *BMI*. θ is the total expected remaining life years in a population (which is equal to the number of people in a population in period 1 times their average expected remaining number of life years).

Life Years Lost and the Impact on a Nation's Capacity to be Sustainable

Changes in health capital, reproducible capital, natural capital and human capital, oil net capital changes, and carbon damages are all summed to measure a nation's wealth over time, (Arrow *et al.* 2012). If the growth of a nation's welfare is non-negative, then that nation meets Arrow *et al.*'s sustainability constraint – this nation is sustainable in the time frame considered. It is clear that increases in obesity directly affect a nation's capacity to be sustainable by reducing a

may reduce the amount to which increased mortality rates (from increased *BMI*) affect a nation's capacity to be sustainable.

nation's health capital. Holding all other forms of capital constant from period 1 to period 2, the impact on a nation's capacity to be sustainable is equivalent to the impact on a nation's health capital.

$$(14) \quad \text{Impact on a Nation's Capacity to be Sustainable} = (h * \omega)$$

This impact is negative for countries like the United States that suffer from an obesity epidemic. Increases in average BMI will be positive for countries in which the average BMI is below 22.5. As a final thought, it is important to reiterate the fact that Arrow *et al.* (2012) find this form of capital to be twice as important as all other forms of capital combined when measuring changes in per capita wealth. So, even small values for ω may have a large impact a nation's capacity to be sustainable.

A Case Study: America from 2005 - 2009

In order to find the impact of average American diet on America's capacity to be sustainable from 2005 to 2009, it is necessary to first quantify America's diet by using equation (3) to solve for *energy intake*. This value can be coupled with data on energy expenditure in equation (2) to find *Energy Imbalance*. Here, this paper will follow Jeffery and Harnack (2007) to estimate values for γ , λ and $a - g$. Then, the change in the average individual's BMI can be calculated (and compared to the real average BMI in 2000), followed by σ , ω and the *Impact on a Nation's Capacity to be Sustainable*. Arrow *et al.* (2012) find that the value of an additional year of life in the United States in 2009 was roughly \$367,343, (or \$392,109 in 2012). This value will be used to find the cost of the diet held between 2005 and 2009.

Estimating Values for γ , λ , and $a - g$

These coefficients will be estimated by carrying out a tautological application of this model to America from 1970 to 2000. Here, change in BMI and height, held constant, are plugged into equation (5) to find *Energy Imbalance_{avg}*. This value and data on energy expenditure can then be plugged into equation (2) to arrange a situation in which there is one equation but three unknowns γ , λ and *energy intake*. This problem can be slightly alleviated by reexamining equation (3), in which there are seven unknowns, $a - g$. Then, drawing (again) on the number of calories in a gram of fat, starch and protein, and on recent research that reveals that added sugars are especially important in energy imbalance, $a - g$ can be guessed at. This process is repeated for 1970 and 2000 data to establish an average over that time period. By considering the results given by Jeffery and Linde, the role of epigenetics in weight gain, and the determined value for *energy intake*, *energy expenditure*, and *Energy Imbalance*, can be estimated. Then, all of the values, if guessed well, correctly predict the change in BMI from 1970 – 2000. This is entirely unsurprising as these coefficients were tautologically determined using the actual change. This process is carried out in Appendix 1 and the results are as follows: λ is 0.483, γ is 0.367, $a - c$ are 0.0976, d and e are 0.1341 and f and g are 0.2195.

Energy Imbalance in America in 2005 and in 2009

This case study will retrospectively predict “future” changes in BMI starting in 2005. The *Energy Imbalance* in 2005 will be set as the *Energy Imbalance_{avg}* for this five year period. This will overestimate the change in BMI as this time period actually saw a decrease in the consumption of meats, added fats, and added sugars (USDA, 2012) and an increase in the consumption of fruits, (Cook, 2012). And, while this period also witnessed a decrease in the per capita consumption of vegetables, the overall trend of food consumption from 2005 to 2009 was one of a shift towards a healthier diet, (Cook, 2012). However, this model still predicts an increase in average BMI because the health gains were not enough to overcome how poor the average diet was in 2005.

The recommended change in consumption in 2005 for each food group in pounds per year was as follows (the numbers in parentheses are in kilocalories per day): grains -15.52 (-63.85), vegetables +.31 (+.07), fruits +366.98 (+242.63), dairy + 148.17 (+129.39), meat +4.007 (+3.53), added fats -89.36 (-426.61), add sugars -45.375 (-519.29).²⁰

$$(15) \quad \text{energy intake}_{2005} = a(63.85) + b(0.07) + c(242.63) + d(129.39) + e(3.53) + f(426.61) + g(519.29)$$

Adding in the value of the coefficients gives that $\text{energy intake}_{2005} = 252.00$ kilocalories per day. This is substantially higher than the values for quantified diet in both 2000 (roughly 144) and 1970 (roughly 175). For reference, by following the same procedure, in 2009 *energy intake* was 224.53. This paper will also assume constant total caloric consumption between 2000 and 2009 so that $\left(1 + \left|\frac{\delta_{\text{recommended}} - \delta_{\text{actual}}}{\delta_{\text{recommended}}}\right|\right)$ is 1.34. In finding a number for $\text{energy expenditure}_{2005}$, this paper will summarize findings published by the United States Center for Disease Control and Prevention (2011) and Jeffery and Harnack’s (2007) findings that one hour of moderate exercise is sufficient to burn roughly 150 kcals. This recommended amount of physical activity per day augments natural caloric expenditure. In 2005, the CDC reported that 14.2 percent of Americans engaged in 10 minutes or fewer of moderate or strenuous physical activity with roughly one fourth of these individuals doing none at all. 37.7 percent of Americans, engaged in more than 10 minutes per week of moderate or vigorous activity but less than the recommended 30 minutes of moderate activity at least five days a week. This paper will assume that the average energy intake per person in this group was halfway between that of the two bookend groups. And, these studies find that 48.1 percent of Americans at least meet the recommendation. If the average for this group was 30 minutes of moderate physical activity per day, then the $\text{energy expenditure}_{2005} = 104.989$ kcals per day.²¹

$$(16) \quad \text{Energy Imbalance}_{2005} = (1.34(.483)(252.00) + (.367)(-104.99)) \left(\frac{\text{Kcal}}{\text{day}}\right)$$

²⁰ Notice that the amount of fruits and vegetables was held constant from 2000 to 2005. This was based off of the findings of Blanck *et al.* (2008) who write, “The frequency of fruit and vegetable consumption changed little from 1994 through 2005.”

²¹ $.142*(.75*60\text{kcal}/(7/5)\text{days} + .25*0) + .377*(75/7/5\text{kcal}/\text{day}/2) + .481*(150\text{kcal}/\text{day}) = 104.989$ kcals per day.

This value, also equal to $Energy\ Imbalance_{avg}$ for this time period, is 124.57 Kcal per day.

Predicted and Actual Change in Average BMI from 2005 to 2009

Equation (4) gives the relationship between change in BMI and $Energy\ Imbalance_{avg}$. Plugging in our value of $Energy\ Imbalance_{avg}$, μ , average weight in 2005 (81.5 kg, or 179.7 lbs.), (McDowell, Fryar, Ogden & Flegal, 2008) and average height in 2005 (1.6925 meters, or 66.63 inches) gives the following:

$$(17) \quad \Delta BMI_{avg} = \left[\left(\frac{179.7\ lbs. + \left(.000182 \frac{\text{lbs.}}{\text{day}} \right) \left(124.57 \frac{\text{kilocalorie}}{\text{day}} \right)}{(66.63\ in)^2} \right) - \left(\frac{179.7\ lbs.}{(66.63\ in)^2} \right) \right] * 703$$

This represents a change in BMI of 0.15 (from 28.45 to 28.6), a modest change. However, as can be seen in figures 10 and 11 in Appendix II, obesity rates increased dramatically from 2005 to 2009.

Perhaps this occurred because some individuals were becoming healthier while many others were just crossing the BMI = 30 thresh-hold. Indeed, this is likely the case as the observed average BMI (in men) in 2010 was 28.7, (Hellmich 2012).

American Life Years Lost from Diet from 2005 to 2009

The increase in mortality rate in this time frame as a result in the increase in BMI is $0.06 * 0.15$, or nine tenths of a percentage point. This number, while seemingly small, is non-trivial. This can be seen in equation (18). Here, the average prematurity of death was 10.813 years and σ is $\frac{9}{100}$.

$$(18) \quad \omega = \text{Number of Life Years Lost} = 10.813 \left[\left(\frac{3}{4} * \frac{9}{10,000} \right) \left(\frac{\epsilon_{period\ 1} + \epsilon_{period\ 2}}{2} \right) \right]$$

Here, the average number of individuals between the ages of 55 and 77.97 in 2005 and 2009 (roughly 67.5 million) is multiplied by 0.0073 to get that $\omega = 492,667$.

The Impact on America's Capacity to be Sustainable

At a value of \$367,343 per life year, the total impact on American health capital was \$45,244,443,445/year (in 2009 dollars). That's only \$150.8 per capita per year. However, this value is, by itself, roughly 1/3 the amount that America spends on obesity prevention per year,

(Reinberg 2013).²² To provide even more context, the American stock of oil in 2000 was valued at \$49.69 billion (in 2009 dollars). And, this number does not include the costs of morbidity; it is a (perhaps large) underestimate of the total impact on America's health capital in this time period. As has been noted, even as average BMI did not increase substantially in this time period, prevalence of obesity did, and so the costs associated with increases in morbidity may have increased dramatically. From Equation (14), the impact on America's capacity to be sustainable from 2005 – 2009 was, in 2009 dollars, \$180,977,773,780.²³

Discussion

The American obesity epidemic overwhelmingly impacts poor minority groups, (Olshansky *et al.* 2005). Assuming that energy expenditure rates (and metabolic rates) are similar across socio-economic boundaries²⁴, the explanation is that America's poor eat relatively unhealthy diets as compared to their more affluent counterparts. This suggests that an unhealthy diet is an inferior good while a healthy diet is a normal good.²⁵ Furthermore, the nation currently subsidizes unhealthy food, to an extent, via corn subsidies, (Scientific America, 2012). In some ways, therefore, the United States' government may subsidize the mortality and morbidity costs incurred by increases in BMI. The impact on a nation's capacity to be sustainable ought to be accounted for in determining subsidies that impact food consumption. This is to say, healthier foods (which are a normal good) ought to be subsidized instead of unhealthy foods – for reasons already argued in existing literature, and also because of the implications for a nation's capacity to be sustainable. Of course, this paints a very simplistic portrait of the institutions that influence the value of *energy intake** in America. In reality, the ways in which various agricultural subsidies influence how Americans eat are extremely complex.

This paper also has theoretical implications for policy written to reduce the severity and prevalence of food deserts. In theory, given that grocery stores that provide healthier food face an increased chance to fail (from high insurance costs, a possible reduced demand, and other factors) but stand to reduce the value of *energy intake** in populations living in food deserts, a market failure currently exists in that the contemporary market structure does not account for the opportunity cost of the forgone value of health capital. The government ought to insure against failure for grocery stores (whose presence may reduce aggregate values of *energy intake**) at a

²² Note that this set of equations (once morbidity is accounted for) could be used to determine optimal annual spending on obesity prevention. Once the model is expanded to account for malnutrition, it may also be able to determine optimal annual spending on malnutrition prevention. This has interesting applications for development theory.

²³ Had America witnessed an increase in BMI of 1 in that time frame, the impact on its capacity to be sustainable would have been near 1.3 trillion dollars.

²⁴ Again (partially to avoid entering into an extremely controversial debate that is beyond the scope of this paper) this assumes away adverse effects of epigenetics.

²⁵ This may not be true when considering developing nations; Abhijit V. Banerjee and Esther Duflo (2006) write the following: "Finally, to the extent that we can tell, the trend seems to be to spend even less money on food [with increases in income]. In India, for example it went from 70 percent in 1983 to 62 percent in 1999-2000, and the share of millet in the food budget dropped to virtually zero (Deaton, 2005). Not surprisingly, the poor are also consuming fewer calories over time (Meenakshi and Vishwanathan, 2003), though it is possible that this change reflects the fact their work involves less physical effort (Jha, 2004)." This model may eventually be able to contribute to the discussion of development and nutrition by describing the quality of calories being consumed and the returns to health.

value equal to the total expected impact on health capital from eliminating food deserts.²⁶ If this occurs in the form of annually-recalculated lump sum subsidies to these grocery stores at a value equal to the total benefit divided by the number of grocery stores receiving the subsidy, the market would internalize this currently unaccounted for cost. Here, the market would correct the number of grocery stores in food deserts. However, this theoretical framework is also overly simplistic; further research is required on multiple frontiers if this model is to be a useful policy tool in attempts to reduce the magnitude of the negative effects associated with food deserts.

Conclusion

The results of the case study indicate that the value of health capital per capita could be higher if Americans ate healthier on average. So, the costs incurred by an unhealthy diet are opportunity costs of health capital. This is to say, the costs incurred by an unhealthy diet are an opportunity cost of aggregate welfare levels. It is uncertain whether this analysis easily extends to the level of the individual. However, the idea that, for an individual, the costs incurred by an unhealthy diet for are opportunity costs is not outlandish. And, diet is an omnipresent component of health capital. In many developed countries, it may be the most prevalent component of health capital as access to health care is relatively high and exposure to violence and disease is relatively low. This paper has briefly discussed another important factor in health capital: physical activity. It is also uncertain how GDP and energy expenditure are linked. As wage increases, the income effect might reduce leisure time physical activity. Or, as wage increases, the substitution effect might increase total leisure time and so physical activity might increase. Smoking is the other major preventable American killer. It too should be considered in health capital eventually and may also present an opportunity cost of welfare. Future research is needed here as well. With morbidity and mortality accounted for, it may turn out that diet is the most important factor in health capital in countries suffering from obesity epidemics. As health capital is twice as important as all other forms of capital, reducing the prevalence of obesity might have extremely large ramifications for a nation. To conclude, this paper is the next of many stepping stones in the path to understanding health capital. Future stepping stones may more fully explore physical activity, malnutrition and morbidity, exposure to warfare, pollution and disease, access to health care and other factors. They may also more full explore the relationships between this and other types of capital.

²⁶ Given that the demographic affected by food deserts is also the demographic experiencing the obesity epidemic, this expected impact may be close to the national total.

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Appendix I: Estimating Values for γ , λ , and $a - g$

Before values for γ and λ can be estimated, values for $a - g$ must be guessed. This will be done by working backwards from equation (5). Holding height and the biological factor constant, known values for average height, average weight (Centers for Disease Control and Prevention, 2012) average BMI,²⁷ energy expenditure and energy intake measurements (from Jeffery & Harnack, 2007) can be plugged into equation (5) to find average energy imbalance from 1970 to 2000.

$$(19) \quad \frac{1}{\mu} * \Delta BMI * (height)^2 = \frac{1}{.000082} * 1.95 * 1.69^2 = 67.9195 \frac{\text{Kcal}}{\text{day}}$$

So, this model predicts that the change in average BMI of 1.95 in America from 1970 to 2000 would require a 67.9195 Kcal/day *Energy Imbalance*. As a point of reference, Jeffery and Linde (2005) write: “to produce obesity in a person over a 20-year time span, which is a very common course, requires a daily error of only 25 Kcal.” It is possible that this model will overstate the number of obese people in a nation if the average BMI of an obese person is much higher than 30. Olshansky *et al.* (2005) find that the shift in BMI’s in America fits this trend. So, the question becomes how much of this imbalance is due to diet, how much to a lack of physical activity and how much to the average American’s biological factor.

$$(20) \quad 67.9195 \frac{\text{Kcal}}{\text{day}} = \left[\left(1 + \left| \frac{\delta_{recommended} - \delta_{actual}}{\delta_{recommended}} \right| \right) (\lambda) (energy\ intake) \right] + (1 - \gamma - \lambda) (energy\ expenditure)$$

The observed value of $\left| \frac{\delta_{recommended} - \delta_{actual}}{\delta_{recommended}} \right|$ in 2000 was roughly 680/2000, or about 0.34 (the United

States government estimates that the average daily caloric intake need for all age groups and both genders is roughly 2,000, (USDA, 2002). In 1970, this value was 0.15 so that the average value from 1970 to 2000 of $\left(1 + \left| \frac{\delta_{recommended} - \delta_{actual}}{\delta_{recommended}} \right| \right)$ is 1.245. In the early 2000s, the Center for Disease Control [CDC] recommended that adults exercise “a minimum of 30 minutes of moderate-intensity activity on most days of the week. In 2001, a total of 54.6% of persons were not active enough to meet these recommendations,” (CDC, 2003). This paper assumes that the same was true in 2000. Jeffery and Harnack (2007) find that “a 300 kcal [change] in energy expenditure would require ~60 min of walking” so that this 30 minutes of walking per day would result in a roughly 150 kcal change in calories. Jeffery and Linde (2005) write that “by most estimates, the proportion of energy expenditure in humans that is occupied by non-modifiable

²⁷ See Figure 8 in Appendix II

activities is 60-80 per cent. The remaining 20-40 percent goes to intentional physical activity.” So, this 150 kcal recommendation for energy expenditure from physical activity is likely the value that would establish an *Energy Imbalance* of 0 given that the rest of the calories from energy intake are used to maintain biological homeostasis. And, as the 150 kcal recommendation was only met by 54% of the population in 2000 (and as there was an increase in physical activity between 1970 and 2000 of 5.15 minutes per day according to Harnack and Schmitz (2006) the recommended value of *energy expenditure* between 1970 and 2000 in America minus the actual value is 81.875 kcal per day. Lastly, Harnack and Schmitz (2006) also find that, unless there were significant changes in household activities such as meal preparation or household chores, energy expenditure remained roughly the same from the 1970’s to the 2000’s (the slight increase in physical activity was offset by increases in sedentary leisure activities such as watching television).

$$(21) \quad 67.9195 \frac{\text{Kcal}}{\text{day}} = 1.245(\lambda)(\text{energy intake}) + (\gamma)(-81.875) \frac{\text{Kcal}}{\text{day}}$$

In order to reduce the number of unknowns in this equation again, the value of *energy intake* can be found by inserting appropriate values for $a - g$, the degree to which an increase in the deviation from the recommended consumption of any food group impacts *energy intake*. These weighting factors can be arrived at by considering the conclusions reached by Basu *et al.* (2013) and the number of calories in grams of fat, protein and starch. There are 9 calories per gram of added fat. Accordingly, this gives added fat the highest weight at 9. Added sugars will also be given a weight of 9 (their negative impact with respect to weight gain may be even higher than that of added fat, however), grains a weight of 4, vegetables and fruits a weight of 4 (their original weight, 2, was doubled to account for the problems associated with under consuming in these groups), and dairy and meats a weight of 5.5 (mostly protein with some fat). Summing and dividing by the total value gives the following:

$$(22) \quad \text{energy intake} = \\ .0976(\text{grains} *) + .0976(\text{vegetables} *) + .0976(\text{fruits} *) + .1341(\text{dairy} *) \\ + .1341(\text{meats} *) + .2195(\text{added fats} *) + .2195(\text{added sugars} *)$$

These values signify that deviating from recommended consumption levels of added fats and added sugars is most impactful, deviating from dairy and meats the second most impactful, and deviating from fruits, vegetables and grains least impactful. Now, values for 1970 and 2000 for *food group** can be plugged into the equation, summed and divided by two to get the average energy intake in this period.

*Quantifying the American Diet in 1970 and 2000*²⁸

An important clarification must be made: the Kantor data that details the recommended consumption levels of various food groups is not specifically from 1995. However, these

²⁸ Figures 5 - 7 in Appendix II document the quantification of American diet.

recommendations will be assumed to be accurate for Americans from 1970 to 2010. This may not be an issue because the Kantor recommendations are a compilation of 27 years (from the 1970s to the 1990s) of food group consumption guidelines.

Actual consumption patterns in 1995, in pounds per person per year were as follows (USDA, 2012): Grains 190.6, Vegetables 408.2, Fruits 280.1, Dairy 576.2, Meat 190.5, Added Sugars 144.1, Added Fats and Oils 64.2. Kantor recommends the following shifts in 1995 consumption patterns in pounds per person per year: Grains -14.82, Vegetables +20.41, Fruits +366.28, Dairy +169.47, Meat +13.607, Added Sugars -91.26, Added Fats and Oils -24.08. The actual shift that occurred between 1995 and 2000, however, did not meet these radical recommended shifts. Actual consumption patterns in 2000, again in pounds per person per year, were as follows: Grains 199.9, Vegetables 428.3, Fruits 279.4, Dairy 591.1, Meat 196.8, Added Sugars 148.9, Added Fats and Oils 81.7. This represented only slight changes for each food group: Grains +9.3, Vegetables +20.1, Fruits -0.7, Dairy +14.9, Meat +6.3, Added Sugars +4.8, Added Fats and Oils +17.5. So, recommended changes in 2000, based on actual changes in consumption patterns and the recommended changes are, in pounds per person per year, as follows (the values in parentheses are recommended changes in 2000 in calories per person per day): Grains -24.12 (-98.68), Vegetables +0.31 (+0.07), Fruits +366.98 (+241.30), Dairy +154.57 (+134.23), Meat +7.307 (+6.41), Added Sugars -96.06 (-456.09), Added Fats and Oils -41.575 (-464.99). These calorie per person per day measurements can be plugged into equation (3) to get *energy intake* in terms of calories per day.

$$(23) \quad \text{energy intake}_{\text{America},2000} = \\ a(98.68) + b(0.07) + c(241.30) + d(134.23) + e(6.41) + f(464.99) + g(456.09)$$

The values of *a-g* can be plugged in here to get $\text{energy intake}_{\text{America},2000} = 144.1429$. A similar process can be applied to find the *food group** values in 1970. Between 1970 and 2000, consumption of grains increased by 45%, fruits and vegetables by 20%, dairy by 8%, meats by 10%, added fats by 39.5%, and added sugars by 23%, (USDA, 2002).²⁹ Which is to say, the recommended change in consumption in pounds per person per year in 1970, based off of Kantor's 1996 estimate, was as follows: grains: +37.48 (+153.75), vegetables: +89.81 (+20.18), fruits: +397.68 (+261.49), dairy: +197.67 (+171.67), meat: +26.907 (23.59), added sugars: -70.86 (-336.44), and added fats: -13.275 (-148.47). Here, the numbers in parentheses are measured in calories per day. These values can be plugged into equation (3) to quantify diet in 1970.

$$(24) \quad \text{energy intake}_{\text{America},1970} = \\ a(153.75) + b(20.18) + c(261.49) + d(171.67) + e(23.59) + f(336.44) + g(148.47)$$

²⁹ As a side note, this government document reports slightly different consumption totals in 2000 than the document used before.

Adding in the values for $a - g$ gives that $energy\ intake_{America,1970} = 175.12$. The average energy intake for this period is 159.63. Plugging this value into equation (24) yields the following:

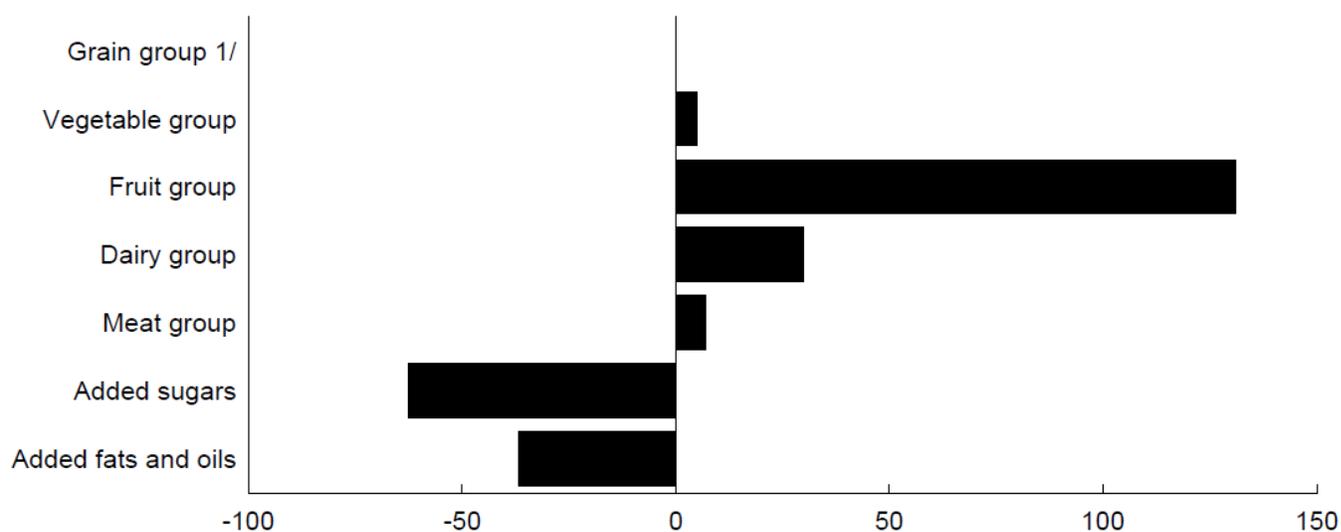
$$(25) \quad 67.9195 \frac{\text{Kcal}}{\text{day}} = 1.245(\lambda)(159.63) + (\gamma)(-81.875) \frac{\text{Kcal}}{\text{day}} = 198.73 * \lambda - \gamma * 81.875$$

So a guess must be made as to the values of λ and γ . Many values satisfy equation (25) as well as Jeffery and Linde's argument for a larger value of λ than γ . However, this paper will estimate that the value of these coefficients are $\lambda = 0.483$ and $\gamma = 0.367$.

Appendix II: Graphs and Charts

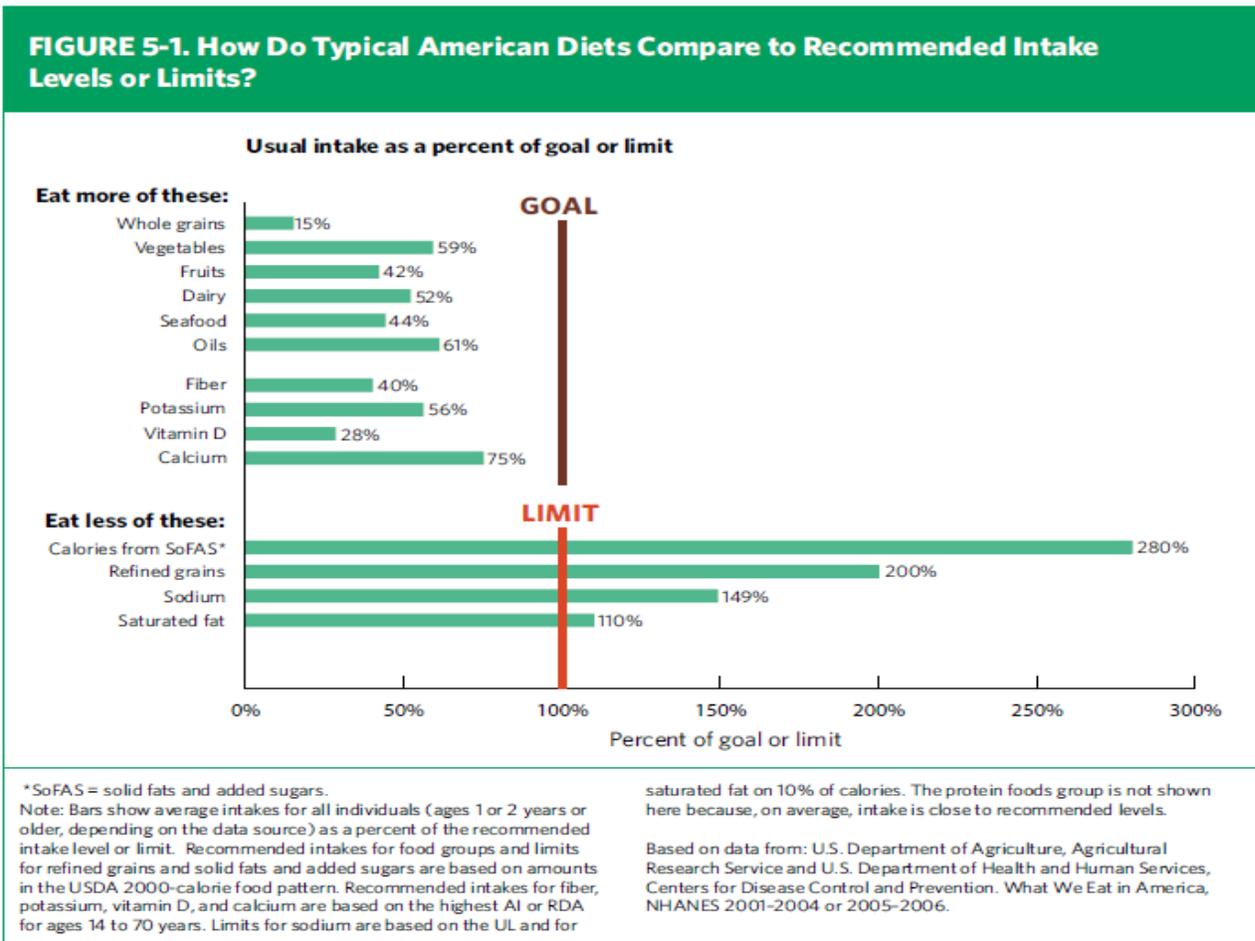
Figure 1)³⁰

Change in food supply servings needed to meet Food Guide Pyramid serving recommendations for a 2,200-calorie diet, 1996



³⁰ Kantor, "A Dietary Assessment," 1998

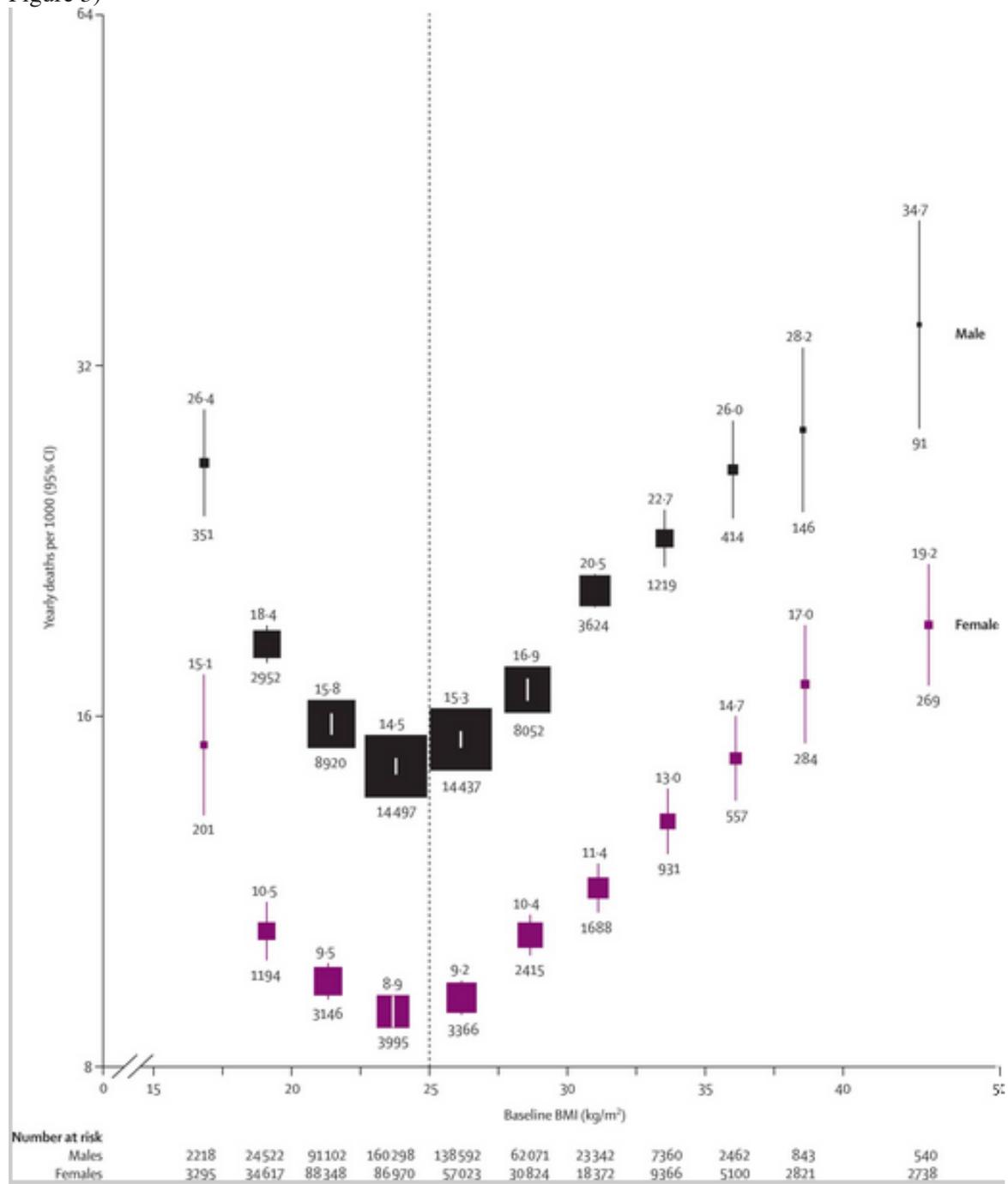
Figure 2)³¹



³¹ Figure 2 is included to legitimize the value of *added sugar**, which largely drives the value of *energy intake* in the case study.

U.S. Department of Agriculture and U.S. Department of Health and Human Services. *Dietary Guidelines for Americans*, 2010. 7th Edition, Washington, DC: U.S. Government Printing Office, December 2010.

Figure 3)³²



³² United States National Center for Biotechnology Information, "Body-mass," 2009

Figure 4)³³All-cause mortality versus baseline BMI in the ranges 15–25 kg/m² and 25–50 kg/m²

	All participants				Never smokers only			
	15–25 kg/m ²		25–50 kg/m ²		15–25 kg/m ²		25–50 kg/m ²	
	Deaths	HR (95% CI)						
Overall	35 256	0.79 (0.77–0.82)	37 493	1.29 (1.27–1.32)	7054	0.87 (0.81–0.94)	9849	1.32 (1.28–1.36)
Male	26 720	0.79 (0.76–0.82)	27 983	1.32 (1.29–1.36)	3694	0.87 (0.78–0.97)	4811	1.44 (1.36–1.53)
Female	8536	0.80 (0.75–0.85)	9510	1.26 (1.23–1.30)	3360	0.87 (0.78–0.97)	5038	1.27 (1.22–1.32)
Age at risk (years)								
35–59	9333	0.76 (0.71–0.81)	8386	1.37 (1.31–1.42)	1665	0.88 (0.74–1.04)	1667	1.43 (1.32–1.55)
60–69	11 514	0.77 (0.73–0.82)	13 007	1.32 (1.27–1.36)	1782	0.88 (0.75–1.03)	2841	1.36 (1.28–1.45)
70–79	10 078	0.82 (0.77–0.87)	11 358	1.27 (1.23–1.32)	2116	0.93 (0.80–1.06)	3364	1.33 (1.25–1.40)
80–89	4331	0.89 (0.80–0.97)	4742	1.16 (1.10–1.23)	1491	0.86 (0.74–1.01)	1977	1.15 (1.07–1.25)

Figure 5)³⁴

Type of Food	recommended servings	Ounces per serving	recommended ounces	Recommended Grams	average kilocalories per serving	recommended caloric intake per day (kcal)
Grains	9	1.5	13.5	382.71825	140	1260
Vegetables	4	8	32	907.184	41	164
Fruits	3	8	24	680.388	120	360
Dairy	2.2	5.83	12.833	363.8091335	115.5	254.1
Meat	1	6	6	170.097	120	120
Added Sugars	1	1.3404	1.3404	37.9996698	341.997*	<341.997*
Added Fats and Oils	1	2	2	56.699	510.291*	<510.291*

*Based on the number of kilocalories per gram of fat (9) and assumed kilocalories per gram of sugar (9)

³³ United States National Center for Biotechnology Information, "Body-mass," 2009³⁴ Data taken from USDA, ERS, "Food Consumption" Page 1

Figure 6)³⁵

Types of Food	Recommended Change in 1996*	1995 data*	2000 data*	Actual Change*	Recommended change in 2000*
Grains	-14.82	190.6	199.9	9.3	-24.12
Vegetables	20.41	408.2	428.3	20.1	0.31
Fruits	366.28	280.1	279.4	-0.7	366.98
Dairy	169.47	576.2	591.1	14.9	154.57
Meat	13.607	190.5	196.8	6.3	7.307
Added Sugars	-91.26	144.1	148.9	4.8	-96.06
Added Fats and Oils	-24.08	64.2	81.7	17.5	-41.575

*measured in pounds/year

Figure 7)³⁶

Type of Food	Recommended change in 2000*	Recommended change in pounds per day	Kilocalories per pound	Recommended change in kilocalories per day
Grains	-24.12	-0.066082192	1493.333333	-98.68273973
Vegetables	0.31	0.000849315	82	0.069643836
Fruits	366.98	1.005424658	240	241.3019178
Dairy	154.57	0.423479452	316.9811321	134.2349961
Meats	7.307	0.020019178	320	6.406136986
Added Sugars	-96.06	-0.263178082	1733	-456.0876164
Added Fats and Oils	-41.575	-0.11390411	4082.328	-464.9939359

*measured in pounds/year

$$\frac{(\text{Number of Servings} * \text{Calories per Serving})}{(\text{Number of Servings} * \text{Ounces Per Serving} * \text{Ounces per Pound})} = \text{Calories per Pound}$$

³⁵ Data taken from USDA, ERS, "Food Consumption" Page 1

³⁶ Ibid.

Figure 8) Average BMI in America by age and gender over time³⁷

Sex and age	NHES I, 1960-62			NHANES I, 1971-74			NHANES II, 1976-80			NHANES III, 1988-94			NHANES 1999-2002		
	Sample size	Mean	Standard error of the mean	Sample size	Mean	Standard error of the mean	Sample size	Mean	Standard error of the mean	Sample size	Mean	Standard error of the mean	Sample size	Mean	Standard error of the mean
Male¹															
20 years and over	---	---	---	---	---	---	---	---	---	7,755	26.7	0.1	4,262	27.8	0.1
20-74 years	2,895	25.1	0.1	4,992	25.7	0.1	5,604	25.6	0.1	6,860	26.8	0.1	3,775	27.9	0.1
20-29 years	585	24.3	0.2	986	24.5	0.1	1,261	24.3	0.1	1,638	25.2	0.2	712	26.6	0.2
30-39 years	714	25.2	0.2	654	26.1	0.2	871	25.6	0.1	1,468	26.5	0.2	704	27.5	0.3
40-49 years	649	25.6	0.2	715	26.2	0.2	695	26.4	0.2	1,220	27.3	0.2	774	28.4	0.3
50-59 years	487	25.6	0.2	717	26.0	0.2	691	26.2	0.2	851	27.8	0.2	594	28.7	0.3
60-74 years	460	24.9	0.2	1,920	25.4	0.1	2,086	25.7	0.1	1,683	27.2	0.2	991	28.6	0.2
75 years and over	---	---	---	---	---	---	---	---	---	895	25.9	0.2	487	26.8	0.2
Female¹															
20 years and over	---	---	---	---	---	---	---	---	---	8,480.0	26.5	0.1	4,243	28.1	0.2
20-74 years	3,231	24.9	0.1	7,919	25.1	0.1	6,161	25.3	0.1	7,459.0	26.6	0.2	3,719	28.2	0.2
20-29 years	672	22.2	0.2	2,122	23.0	0.1	1,290	23.1	0.2	1,663.0	24.3	0.2	654	26.8	0.3
30-39 years	749	24.1	0.2	1,654	24.7	0.2	964	24.9	0.2	1,773.0	26.3	0.3	698	27.9	0.3
40-49 years	759	25.2	0.2	1,232	25.7	0.2	765	25.7	0.2	1,354.0	27.1	0.3	783	28.6	0.4
50-59 years	554	26.4	0.2	780	26.2	0.2	793	26.5	0.2	996.0	28.4	0.3	591	29.2	0.4
60-74 years	497	27.2	0.2	2,131	26.5	0.2	2,349	26.5	0.1	1,673.0	27.4	0.2	993	29.2	0.2
75 years and over	---	---	---	---	---	---	---	---	---	1,021.0	25.9	0.2	524	26.8	0.4

--- Data not available.

¹Statistically significant trend or difference p<0.05 for all years available.

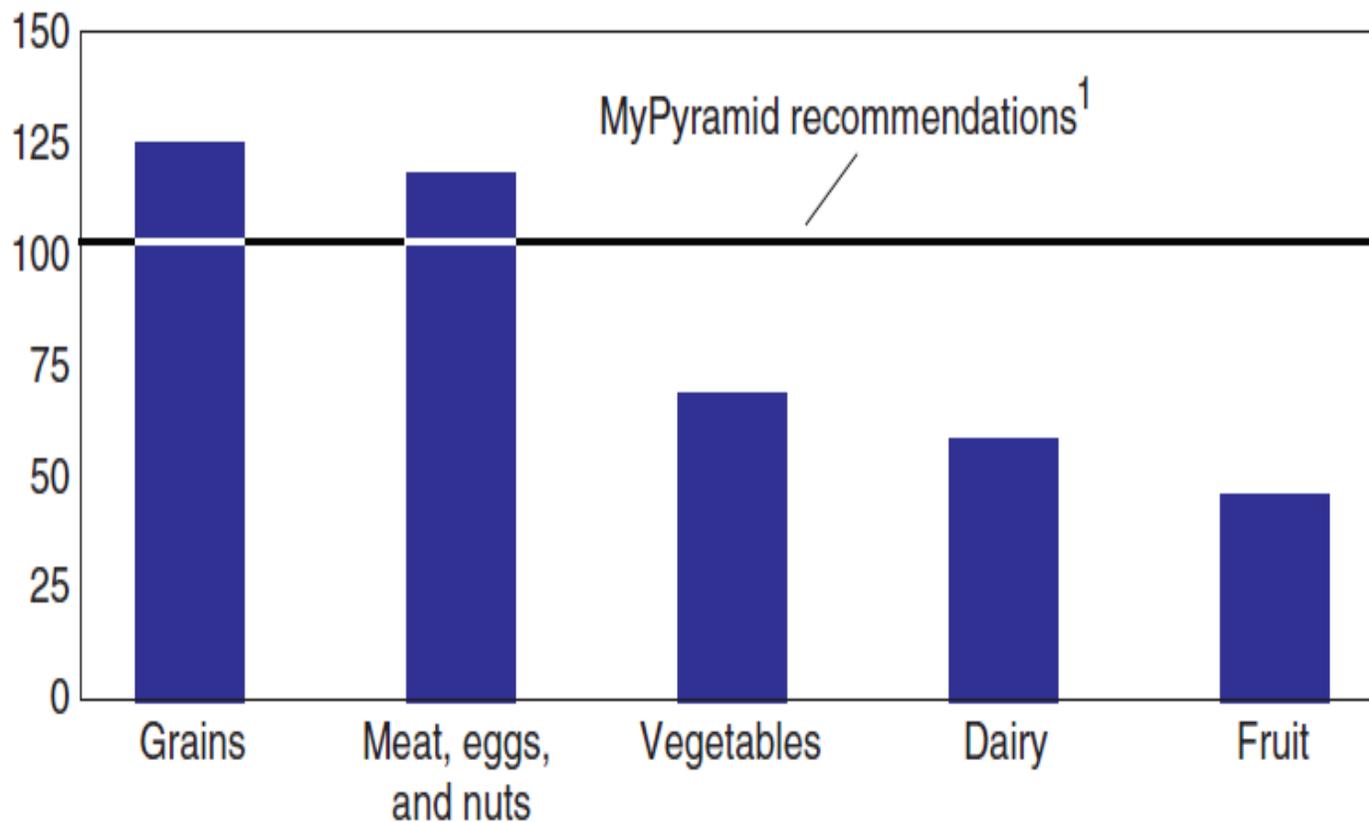
NOTES: BMI is calculated as weight in kilograms divided by square of height in meters. NHES I: National Health Examination Survey, Cycle I, ages 20-74 years; and NHANES: National Health and Nutrition Examination Survey.

³⁷ Ogden *et al.* "National Health" 2008

Figure 9)³⁸

Loss-adjusted per capita food availability out of balance with dietary recommendations

Percent of recommendation

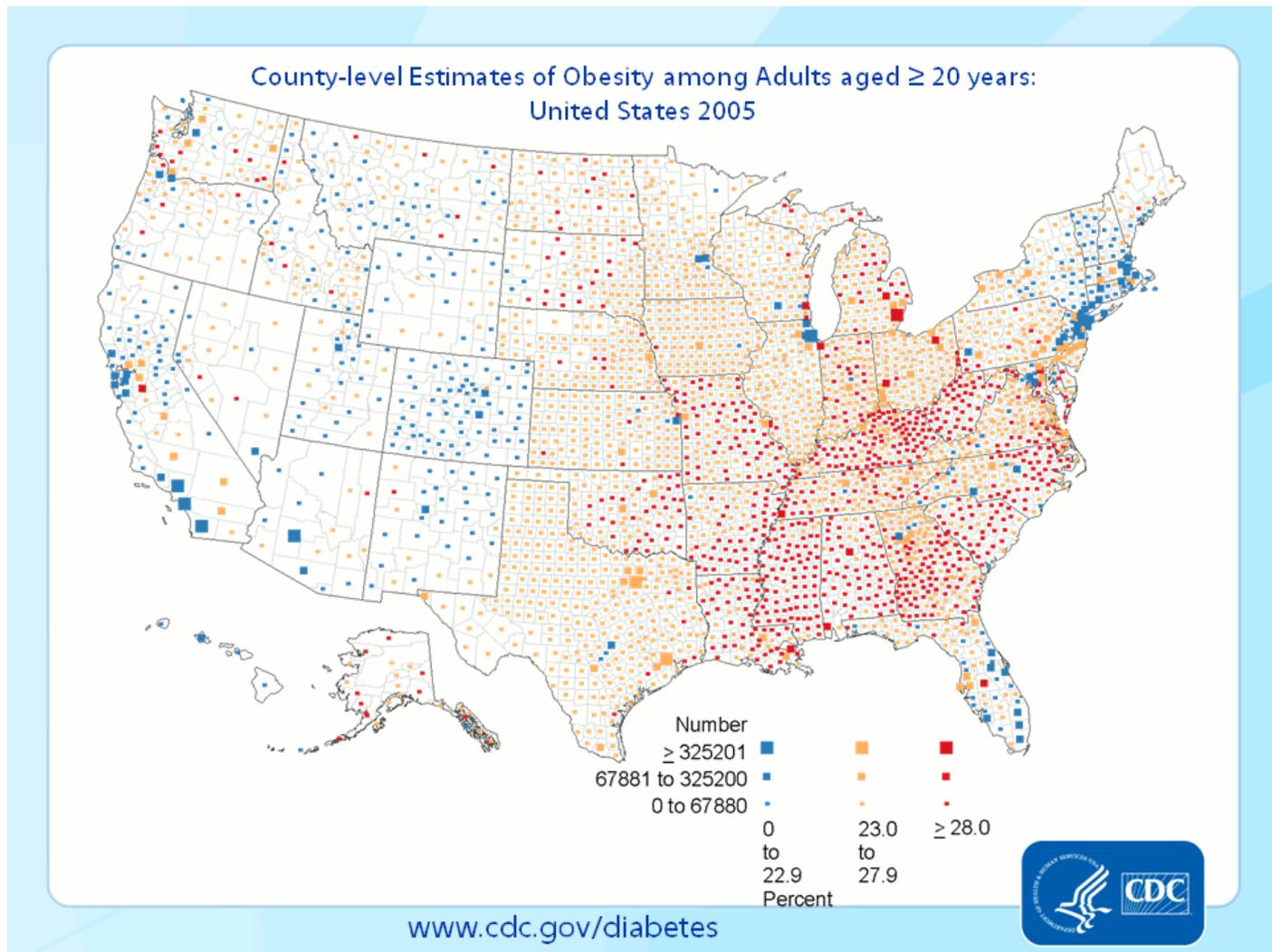


¹2005 data based on a 2,000-calorie diet.

Source: USDA, ERS Food Availability (Per Capita) Data System.

³⁸ http://www.ers.usda.gov/media/210681/eib33_1_.pdf

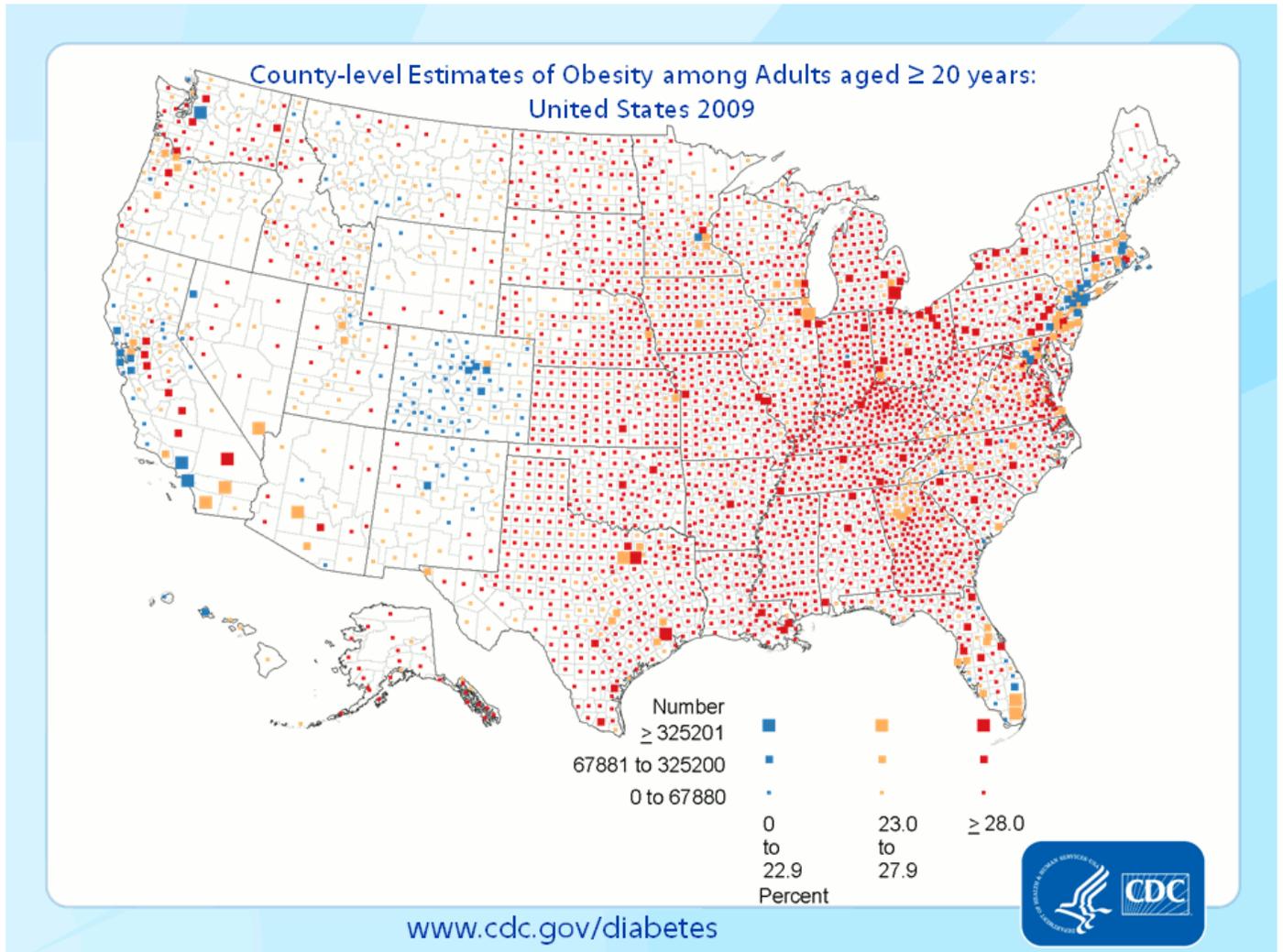
Figure 10)³⁹



³⁹ United States Center for Disease Control and Prevention "County Level Estimates of Obesity, U.S. Maps." *Diabetes*

Data and Trends County Level Estimates.: National Diabetes Surveillance System.,
<http://apps.nccd.cdc.gov/DDT_STRS2/NationalDiabetesPrevalenceEstimates.aspx?mode=OBS>.

Figure 11)⁴⁰



⁴⁰ United States Center for Disease Control and Prevention "County Level Estimates"