Low-Fat Diet on Global Health Outcomes—A Two-Stage Difference-in-Difference

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Abstract

I examine the effects of the low-fat diet promoted in a number of countries. I will examine the following public health outcomes: (1) mean body mass index (age-adjusted), (2) prevalence of adult, children, and adolescent obesity, (3) prevalence of overweight adults, children, and adolescent, (4) incidence of systolic high blood pressure, and (5) incidence of type 2 diabetes. In the US, the low-fat diet was intended to decrease the sharp increase in heart attacks during the 1960's and to promote weight loss. Many countries that implement similar food-based dietary guidelines address the same or similar health outcomes. Using the two-stage difference-in-difference method, I compare countries that implemented similar food-based dietary guidelines—in terms of food groups and quantities recommended—as the United States compared to untreated countries where no such food-based dietary guidelines have been promoted.

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Important preface: I am still thinking about the economic angle for this paper. Ray March has recommended reading Hamowy (2007), Leeson et al. (2020), and Merton (1938), in order to make a broader appeal to examining government influence in health/ health science/ healthcare.

1 Introduction

Worldwide increases in weight-gain and cardiovascular disease (CVD) risk has led many experts and governments searching for dietary recommendations to curb and reverse this trend. National health and dietary guidelines were implemented and promoted. This includes pictorial tools used to promote food-based dietary guidelines (FBDGs), such as: Food Pyramid (United States), Healthy Eating Pyramid (Australia), Balance of Good Health (Great Britain), Food Guide Pagoda (China), and Food Guide (Canada) used to promote certain food groups and their daily quantities consumed. Per the Food and Agriculture Organization (FAO) of the United Nations, these FBDGs "are intended to establish a basis for public food and nutrition, health and agricultural policies and nutrition education programmes to foster healthy eating habits and lifestyles. They provide advice on foods, food groups and dietary patterns to provide the required nutrients to the general public to promote overall health and prevent chronic diseases."[1]

Most countries (89%) recommend reducing fat intake and fewer than half of all countries (44%) have a message on the quality of fats (Herforth et al., 2019). This is based off of the *diet-heart hypothesis*, which stipulates dietary consumption of cholesterol can lead to changes in blood serum cholesterol levels that, in turn, increases the risk of atherosclerosis[2] and other coronary heart disease events and potentially premature death. On 1968, the Senate created the Select Committee on Nutrition and Human Needs in order to tackle the heart epidemic. On 1977, the Select Committee published a report *Dietary Goals for the United States* (1977) recommending Americans to (1) increase carbohydrate consumption to 55-60 percent of total caloric intake, (2) reduce fat consumption to 40-30 percent of total caloric intake, (3) reduce saturated fat consumption to 10 percent of total caloric intake, and (4) reduce cholesterol consumption to 350-250mg a day. In other words, to maintain a similar of daily caloric intake, Americans were recommended to eat considerably less meat and dairy.

Although the United States has been making dietary recommendations since 1916, when Caroline Hunt authored the first United States Department of Agriculture (USDA) food guide, *Food for Young Children* (Hunt, 1916), it was not until 1990 that the recommendations quantified less than 30 percent of fat for daily caloric intake and not until the Food Pyramid in 1992 that greater attention was put on meat and dairy products. For the first time, the hierarchical imagery of the Food Pyramid conveyed

[1] "Food-Based Dietary Guidelines", 2021.

[2] Atherosclerosis is the buildup of plaque along the arterial walls, which is a disease of the arteries that supply blood to the heart. Accumulation over decades results in calcification, turning arteries stiff. This can result in blood clots that completely block the blood supply to whatever organ that particular artery was supplying

proportionality with the bread, cereal, rice, and pasta group being the base; fruits and vegetables on the next level; fewer portions of meat and dairy; and the least amount of fats, oils, and sweets. In 2005, with the release of MyPyramid, the recommended servings of meat and beans shrunk while fats, oils, and sweets disappeared altogether. With the release MyPlate in 2011, this time meats, beans, fish, nuts, eggs, and poultry disappeared from the graphic and was incorporated into the general protein foods group.[3] Similar trends occur for Sweden and Germany. While they implemented dietary guidelines around the middle of the 20^{th} century, it was in the 1980s and 1990s that they recommended decreased fats and meats intake.

Countries use pictorial tools, similar to the US Food Pyramid, to convey the correct proportionality of food groups. In China and South Korea, the Food Pagoda promotes grains as a staple at the bottom and fats and oils at the top. The Philippines and Puerto Rico adopted a pyramid as well. Great Britain, Germany, Mexico, Portugal, and Sweden adopted a plate or circular shape divided into similar proportions of the food groups as the US Food Pyramid. All of the pictorial tools mentioned earlier recommended the most servings in grains, followed by fruits and vegetables, then by meats, dairy, beans, and nuts, and the least of all, fats, oils, and sugar. Other countries, with and without pictorial tools, starting from the 1980s until today have pushed for similar recommendations daily serving sizes for each food group.

I will attempt to study the global effects of countries that implement national FBDGs. The health outcomes I will examine are: 1) mean body mass index (BMI) (age-adjusted), (2) prevalence of adult, children, and adolescent obesity, (3) prevalence of overweight adults, children, and adolescent, (4) incidence of systolic high blood pressure (SHBP), and (5) incidence of type 2 diabetes (T2D). These outcomes are either the health outcomes or related health outcomes that countries who implement FBDGs explicitly address. The aim of this paper is to add to body of literature examining whether low-fat diets have had a significant impact on worldwide health outcomes.

The structure of the paper is as follows: I will provide the historic background on the low-fat diet and a literature review on the effects of diet related to weight loss, cholesterol, SHBP, and T2D in Section 2. The method of the paper, two-stage difference-in-difference, will be discussed in Section 3. Why I chose these health outcomes and how I obtained the data will be discussed in Section 4. The results are in Section 5. Problems to solve and potential weaknesses to my paper are looked at in Section 7. The discussion and conclusion are not yet included in this rough draft, since the results may change as countries are added and dropped.

[3]See: https://www.myplate.gov/eat-healthy/protein-foods "All foods made from seafood; meat, poultry, and eggs; beans, peas, and lentils; and nuts, seeds, and soy products are part of the Protein Foods Group."

2 Historic Background

The low-fat diet is based off of research by Ancel Keys, a professor of physiology at the University of Minnesota, who studied the diets of six countries: Australia, Canada, England and Wales, Italy, Japan, and the United States. He found that as the percentage of fat in the diet rose, the death rates from heart disease rose proportionally (A. Keys, 1953). Keys later, in 1956, began work on an enormous project examining the relationship between diet and heart-disease risk. This would be the famous Seven Countries study that followed around thirteen thousand middle-aged men in sixteen mostly rural populations in Italy, Yugoslavia, Greece, Finland, the Netherlands, Japan, and the US (A. Keys, 1970). Studying over 12,000 men ages 40-59, Keys found that cigarette smoking, body fatness, and relative bodyweight did not seem to explain differences in coronary heart disease. However, there were tendencies related to serum cholesterol values and saturated fatty acids in the diet with coronary disease. Later, Keys continued to argue for the relationship between saturated fatty acids, dietary cholesterol, and blood serum cholesterol level, hypothesizing that

> If saturated fatty acids provide 18% of the calories in the diet, reducing to half that amount, with isocaloric replacement by simple carbohydrate, can be expected to produce an average reduction of some 23 mg/dl in the serum cholesterol. Add the effect of cutting in half the dietary cholesterol and the total effect should be, on the average, more than 30 mg/dl decrease in serum cholesterol. [...] It is enough to agree that a reduction by some 30 mg/dl in serum cholesterol is certainly not negligible. (A. Keys, 1984, p. 358)

It should be noted here that there is a difference between dietary cholesterol and blood serum cholesterol levels. Dietary cholesterol is the main steroid from animal tissues.[4] Meat, eggs, grain products, and milk were four of the highest sources of dietary cholesterol, contributing a total of 96 percent of the total consumption in the 2013-2014 survey cycle (Xu et al., 2018). Blood serum cholesterol level, on the other hand, consists of "good," or high-density lipoprotein (HDL), and "bad," or low-density lipoprotein (LDL), cholesterol. HDL has two important functions: it promotes reverse cholesterol transport and it modulates inflammation. LDL transports cholesterol to peripheral tissues. According to the diet-heart hypothesis, if LDL blood cholesterol levels are elevated, lipids can be deposited in the arterial lumen leading to plaque formation, which is an prerequisite development of atherosclerosis. Papers focus on either the absolute amounts of HDL and LDL, the sum of HDL and LDL, or the ratio of HDL and LDL cholesterol (DiNicolantonio & O'Keefe, 2017).[5] Healthy lipids include monounsaturated and polyunsaturated fats. Unhealthy lipids include trans fat. Saturated fats are considered unhealthy or neutral.

[4] Foods with the most dietary cholesterol include: egg yolk, shrimp, beef, pork, poultry, cheese, and butter.

[5] There are additional lipoprotein cholesterols, including very-low-density lipoprotein (VLDL) and intermediate-density lipoprotein (IDL)

Population studies have consistently shown that HDL cholesterol levels are inversely proportional to CVD. The literature will be further discussed in Section 2.2.

Early on, there were some studies questioning the relationship between diet and CVD risk. Mann et al. (1964) conducted a field survey of 400 Masai men, women, and children. Despite a lifetime diet of exclusively meat and dairy, there was no evidence for arteriosclerotic heart disease (Mann et al., 1964, p. 311). Even Keys, in 15 year follow-up to the Seven Countries study, writes

> in spite of greatly increased awareness of the coronary problem in the past two decades and many surveys showing a substantial rise in the average serum cholesterol associated with a trend toward "westernization" of the diet in Japan, vital statistics mortality from coronary heart disease (CHD) has increased little and is still very low by American and European standards. The 15-year experience of CHD in Japan remains puzzling (A. Keys et al., 1984, p. 153).

In the book *How to Eat Well and Stay Well the Mediterranean Way* (1975) Keys promotes the low-fat diet that he initially called the "Mediterranean diet." The diet, however, did not gain widespread popularity until the 1990s. The Mediterranean diet consists of abundant plant foods (fruits, vegetables, breads, beans, nuts, and seeds), olive oil as a source of fat, dairy products (mainly cheese and yogurt), and fish and poultry consumed in low to moderate amounts, zero to four eggs consumed weekly, and meat and wine consumed in low amounts (Willett et al., 1995). Saturated fats make up around 7-8 percent of total daily energy intake with total fat ranging from 25-35 percent.

2.1 Weight Loss

Weight gain occurs when daily energy (caloric) intake is greater than daily energy expenditure (Buchholz & Schoeller, 2004). Excess caloric intake is stored in the body as lipids.[6] Weight is maintained when daily energy intake is equal to daily energy expenditure. When daily energy intake is lower than daily expenditure or daily expenditure is higher than daily intake, weight loss occurs. There is, however, an ongoing debate on whether or not the macronutrient proportions in diet can help or hinder weight loss.

Of the few studies that studied various diets for one year and longer, one study showed that a low-fat diet was superior to a moderate-fat diet (Toubro & Astrup, 1997); two showed that a moderate-fat, Mediterranean diet was superior to a low-fat diet (McManus et al., 2001; Shai et al., 2008); and one showed a low-carbohydrate diet was superior to a low-fat diet (Shai et al., 2008). While the low-fat

[6]Over time, as the accumulation increases, the amount of lipids on the body increases, resulting in being overweight. Overweight is classified as having a BMI of 25 but less than 30. Obesity is classified of having a BMI between 30 and 40. BMI over 40 is considered morbidly obese. BMI is calculated as a ratio of weight to height. Alternative indices of body fat include relative fat mass (RFM), body adiposity index (BAI), waist circumference (WC), and waist-to-hip ratio (WHR). BMI remains the most popular and most accessible measure of body fat (Adab et al., 2018).

diet has not been conclusively shown to alleviate cardiovascular disease risk and weight gain in these long-term randomized control studies, the US government continues to promote this low-fat, high-carbohydrate diet.[7]

Americans nonetheless complied with the recommendations, shifting their diet characterized by low fiber and high saturated fats and sugars to one with reduced consumption of saturated fats and sugar and increasing intake of grains and dietary fiber (Zhou et al., 2010). However, this was followed by a sharp nationwide increase in the prevalence of obesity starting from the mid-1980's and a second rapid increase in the prevalence of diabetes in the late 1990's (Zhou et al., 2010). Worse, despite Americans shifting their diet, data from 1980 to 2005 suggest persons age 20 years and over moved upward from 23 percent obesity rates to 34 percent (*Prevalence of Overweight, Obesity and Extreme Obesity among Adults: United States, Trends 1976-80 through 2005-2006*, 2008). It could be argued that weight gain trends would have been worse if not for the guidelines recommended by the US government. Indeed, there could be endogeneity among countries that adopt FDBGs: governments with countries experiencing worsening CVD risk and increased weight gain would feel pressured to address these outcomes.

In popular writings, Atkins (2002), Fumento (1998), Oliver (2005), Campos (2004), Michaels et al. (2019), and Taubes (2001, 2008, 2011) challenges the efficacy of the low-fat diet to combat cardiovascular disease and weight loss. In the literature, scholars make less exaggerated claims. A growing number of literature has found no significant difference on weight loss in varying the ratio of macronutrients (Astrup et al., 2004; Bradley et al., 2009; Bravata et al., 2003; Brinkworth et al., 2009; Dansinger et al., 2005; Das et al., 2008; de Souza et al., 2012; Foster et al., 2010; Golay et al., 1996; Sacks et al., 2009; Shai et al., 2008).

This is, of course, by no means a comprehensive review of the literature. The conclusion these scholars reached is that restriction in daily caloric intake, no matter emphasis on the macronutrient content, will result in weight loss. That is not to say, however, that in other aspects, such as cholesterol, adherence to the diet, individual preferences for types of foods, and other aspects, are also equal among different diets. For example, McManus et al. (2001) observed greater participation retention among moderate-fat Mediterranean style diet compared to low-fat Mediterranean style diet. Johnston et al. (2006) found ketogenic low-carbohydrate diets had no significant weight loss differences over nonketogenic low-carbohydrate diets, however, they observed several adverse metabolic and emotional effects associated with the ketogenic diet.

[7]The US government links dietary cholesterol and blood serum cholesterol: "Oils are fats that are liquid at room temperature, like vegetable oils used in cooking. They come from many different plants and from fish. Oils are not a food group, but they provide you with important nutrients such as unsaturated fats and vitamin E. Choosing unsaturated fat in place of saturated fat can reduce your risk of heart disease and improve "good" (HDL) cholesterol levels"("More Key Topics", 2021).

2.2 Cholesterol and Lipids

At the same time, while one aspect of the low-fat diet was aimed at promoting weight loss, researchers also looked into the relationship between diet and cholesterol levels. Globally, all countries include starchy staples in their FBDGs, though few are quantitative. Americans, on average, followed the low-fat diet. From 1950 to 1998, US CVD mortality did, in fact, decrease by 53 percent. Nonetheless, the incidence of CVD remained the same (La Berge, 2007). Unlike the research on the relationship between diet and weight loss, most epidemiologic studies and several intervention studies found support that the Mediterranean helps reduce CVD risk (Furtado et al., 2008; Sofi et al., 2010). Though there are scholars and medical doctors that question the relationship between fat consumption and blood serum cholesterol levels.

The French paradox is frequently brought up when discussing the relationship between saturated fats and CVD (Ferrieres, 2004). One explanation that was suggested concludes ethanol, specifically wine ethanol, is inversely related to CVD but decreases the longevity of the French population (Criqui & Ringel, 1994; Renaud & de Lorgeril, 1992). Keys in an interview agreed with the noncausal relationship between dietary cholesterol and blood-cholesterol levels, but continued to believe in the relationship between saturated fat and blood-cholesterol levels saying,

> There's no connection whatsoever between cholesterol in food and cholesterol in blood. None. And we've known that all along. Cholesterol in the diet doesn't matter at all unless you happen to be a chicken or a rabbit. Now we know that both rabbits and chickens are highly sensitive to dietary cholesterol. Their blood-cholesterol levels soar. But all carnivores—not just human beings, but rats and dogs—are not sensitive. It doesn't make a bit of difference how much cholesterol is in the diet. What matters is saturated fat, which we've proved raise serum cholesterol (Jaret, 1997).

In other words, the diet-heart hypothesis now turned into the *saturated fat diet-heart hypothesis*. However, one meta-analysis covering twenty-one studies during a 5-23 year follow-up of over 300,000 subjects found no significant relationship between dietary saturated fats and an increased risk of CVD (Siri-Tarino et al., 2010). Seshadri et al. (2004) found similar beneficial effects on LDL and HDL levels between the low-carbohydrate diet and the conventional diet (a diet consistent with the national recommendation of $\leq$30% of calories from fat). Kendrick (2008) and Ravnoskov (2002; 2018; 2016) have written a number of books and studies criticizing the lipid/diet-heart hypothesis. Examining the literature from the 1960s until present, Ravnskov et al. (2018) concludes

> The idea that high cholesterol levels in the blood are the main cause of CVD is impossible because people with low levels become just as atherosclerotic as people with high levels and their risk of suffering from CVD is the same or higher (Ravnskov et al., 2018, p. 966)

In an earlier review of 263 studies, Ravnskov et al. (2016) attempts to find a relationship between LDL and mortality, but none were found.

2.3 Systolic High Blood Pressure (SHBP)

SHBP, or hypertension, is characterized when blood pressure within artery walls is consistently too high. Two measurements are used: systolic for when the heart contracts (during beats) and diastolic when the heart relaxes (in-between beats). Hypertension is one of the most important risk factors for CVD. There are two types of hypertension: (1) primary (essential) hypertension has no identifiable cause but tends to develop gradually over years and (2) secondary hypertension, which is caused by an underlying condition. [8] One reason to look at SHBP was mentioned earlier. Elevated LDL levels in the blood can cause constriction in the blood vessels, which could be an early symptom of CVD. However, given the variety of factors that may cause SHBP, the results obtained from this outcome may not be robust.

2.4 Type 2 Diabetes (T2D)

The International Diabetes Federation (IDF) estimates worldwide prevalence of diabetes at 463 million people in 2019, 90 percent of which have T2D ("IDF Diabetes Atlas, 10th Edn." 2021). T2D is an impairment in the way the body regulates sugar (glucose) as fuel. Typically, two interrelated symptoms are associated with T2D: (1) production of insulin, a hormone that regulates blood sugar levels, from the pancreas is inhibited and (2) cells respond poorly to insulin and intake less sugar. High blood sugar levels can lead to circulatory, nervous, and immune system disorders. The underlying cause for T2D is not yet known, though the rise of prevalence is hypothesized as a result of environmental and behavioral changes.There is no cure, though symptoms can be managed with insulin, diet, weight loss, and exercise. FBDGs often do not directly address diabetes. However, since diabetes is related to the other health outcomes, including weight, CVD, and SHBP, it seems pertinent to include diabetes as part of the analysis. The low-fat Mediterranean diet may provide some protective mechanisms against diabetes (Georgoulis et al., 2014; Sleiman et al., 2015). There is a growing amount of attention and research dedicated to the prevalence of diabetes (Aziz et al., 2015; Saeedi et al., 2019).

[8] Various conditions can lead to secondary hypertension, including: obstructive sleep apnea, kidney disease, adrenal gland tumors, thyroid problems, congenial birth defects, medication, and illicit drugs.(List of conditions obtained from the Mayo Clinic website ("High Blood Pressure (Hypertension) - Symptoms and Causes", 2021)).

3 Data

3.1 Outcome Variables and Covariates

I would prefer to analyze cardiovascular disease as an outcome, because many FBDGs directly address this outcome. However, the effects of diet on cardiovascular diseases will manifest after a long period, perhaps a few decades, and any changes—if there are changes—in cardiovascular outcomes within the duration of my study will unlikely be due to the federal dietary recommendations. Therefore, I will instead look at the outcomes of shorter manifesting diseases, such as SHBP and T2D, along with obesity and overweightness.

My data on prevalence of obesity and overweight for adults, children, and adolescents, as well as the data on mean BMI are taken from the World Health Organization (WHO) indicators.[9] Data on SHBP and T2D were retrieved from the Global Health Data Exchange (GHDx).[10] BMI, obesity, and overweight are studied over years 1976-2016 and SHBP and T2D are studied over years 1990-2016. The dataset consists of 141 total countries, 73 of which adopted FBDG over this time period.

For the covariates, I have included income taken from the Penn World Tables 10.0[11] and females as a percentage of the population from the World Bank.[12]

3.2 Identification of the Treated Countries

In order to find the countries that have implemented FBDGs and the respective food groupings and intake recommendations, I used the following sources: (1) Painter et al. (2002), who compared pictorial representations of international food guides, (2) Blake et al. (2018) conducted an international examination of FBDGs, (3) Herforth et al. (2019) wrote a comprehensive global review of FGDBs, (4) the WHO report for the European region (*Comparative Analysis of Food and Nutrition Policies in WHO European Member States*, 2003; *Food-Based Dietary Guidelines in the WHO European Region*, 2003), and lastly, (5) the FAO encourages countries to submit their FBDGs to their online database.[13] While the FAO website contains the most up-to-date information on FBDGs, past dietary guidelines are not as well documented in terms of food group and quantity recommendation.

My current strict treated group includes: The United States (*USDA's Food Guide Pyramid*, 1992), Australia (Syrett, 2015), Great Britain (Gatenby et al., 1995), China ("Dietary Guidelines and the Food Guide Pagoda", 2000; Ge et al., 2007), Canada (Bush & Kirkpatrick, 2003; "History of Canada's Food Guides from 1942 to 2007", 2019), and South Korea (Lee & Kim, 1998). More work needs to be done

[9] "Indicators", 2021.
[10] "GBD Results Tool — GHDx", 2021.
[11] Feenstra et al., 2015.
[12] "Indicators", 2021.
[13] "Food-Based Dietary Guidelines", 2021.

sifting through the literature in order to determine the countries with strict recommended dietary fat.

3.3 Strict and Loose Treated Countries

Many countries will group foods and macro-nutrients into the same groupings as the US Food Pyramid but will often differ in quantitative recommendations. Therefore, I will create *strict* and *loose* categories of my treated group. The *strict* treated group will contain countries with FBDGs that quantify 30 percent or less of daily energy intake come from total fat. The *loose* treated group will include all countries that have implemented a FBDG, since as Herforth et al. (2019) found, most FBDGs advise, generally, less fat intake.

4 Method

The focus of this paper is to determine whether or not government dietary recommendations, along with the mass media and schooling, in promoting the low-fat diet, managed to curb the health outcomes I named earlier. Since the treatment in my treated group is staggered, two-way fixed effects (TWFEs) should not be used. Using early treated countries as controls for the late treated countries would result in biased estimands (Baker et al., 2021; Bertrand et al., 2004; Callaway & Sant'Anna, 2021; Goodman-Bacon, 2021). Specifying TWFEs as:

$$Y_{gpit} = \lambda_g + \gamma_p + \beta D_{gp} + \epsilon_{gpit}$$

assumes a conditional expectation function that is linear in group, time period, and treatment status. Here, Y_{gpit} are the health outcomes variables (obesity, overweight, SHBP, T2D), λ_g is the group(country) fixed effect, γ_p is the time (year) fixed effect, and D_{gp} is the treatment (adopting a FBDG). However, if the model is misspecified—and we do not know beforehand the functional form of these relationships—it will attribute some heterogeneity impacts of the treatment of the treatment to group and period fixed effects.

The longer the treatment (that is, D_{gp} is longer), the more the group's treatment effect will be absorbed by the group fixed effects. In other words, TFWE will not recover $E(\beta_{gp}|D_{gp} = 1)$. Nonetheless, I will include estimates from TWFEs. Countries that implement FBDGs should have treatment effects build up over time as more of the population learns of the guidelines, create dietary habits, and shift their lifestyles.

Included in each difference-in-difference is the Goodman-Bacon decomposition in order to determine if the comparisons between the early treated countries and the later treated countries are not weighted biasedly. For the Goodman-Bacon decomposition, later vs earlier treated are the biased com-

parisons and the earlier vs later treated are the correct comparisons (Goodman-Bacon, 2021). As a result, I will use the two-stage differences-in-differences (2SDID) method formulated by Gardner (2021).

4.1 The Two-Stage Approach

In studies with staggered treatment, later treated units should not be compared with earlier treated units. However, as long as there are untreated and treated observations for each group and period, we can control for group and time fixed effects. Mis-specified difference-in-difference models will attribute heterogeneous treatment effects onto group and time fixed effects rather than the true treatment effect (Gardner, 2021). The 2SDID procedure outlined by Gardner (2021) is as follows:

1. Estimate the model

$$Y_{gpit} = \lambda_g + \gamma_p + X'_{gpit}\delta_{pt} + \epsilon_{gpit}$$

   on the sample of observations for which $D_{gp} = 0$, retaining the estimated group and time effects $\hat{\lambda}_g$ and $\hat{\gamma}_p$ and including interactions between covariates (time-varying or not) $X'_{gpit}\delta_{pt}$.

2. Regress adjusted outcomes $Y_{gpit} - \hat{\lambda}_g - \hat{\gamma}_p$ on $D_{gp} = 0$. Since parallel trends implies that

$$E(Y_{gpit}|g, p, D_{gp}) - \lambda_g - \gamma_p = \beta_{gp}D_{gp} = E(\beta_{gp}|D_{gp} = 1)D_{gp} + [\beta_{gp} - E(\beta_{gp}|D_{gp} = 1)]D_{gp},$$

   where $E\{[\beta_{gp} - E(\beta_{gp}|D_{gp} = 1)]D_{gp}|D_{gp}\} = 0$. This procedure identifies $E(\beta_{gp}|D_{gp} = 1)$, even when the adoption and average effects of the treatment are heterogeneous for groups and periods.[14]

However, the standard errors are wrong on the second stage because the dependent variable uses estimates obtained in the first stage. The asymptotic distribution of the second stage can be obtained by interpreting the two-stage procedure as a joint generalized methods of moments (GMM). However, I alternatively obtain the correct standard errors with bootstrapping. With this approach, once the country adopts an FBDG, it will be permanently removed from the untreated group. Therefore, early treated countries will not be used as a comparison for later treated countries, which could potentially cause biased estimands and even the wrong coefficient sign in the average treatment effect on the treated (Goodman-Bacon, 2021). In my paper, as soon as a country implements a FBDG, the country will be taken out of the untreated group and will not be replaced.

[14] I can adjust outcomes in the second stage according to the last pre-treatment realization $X^*_{gpit}\delta_{pt}$ or by controlling for X^*_{gpit} via conventional propensity-score reweighting methods (or a doubly combination of methods), though this remains more parametric than their inverse-probability-weighting approach (Gardner, 2021).

5 Results

Some of the data is not yet complete, such as data on what I defined as strict and loose FBDG countries. Additionally, variable names will be changed later to be clearer. See Table 2 for summary statistics.

In my study, for prevalence of obesity and overweight, we see an increase in prevalence for countries who adopted a FBDG that is significant (obesity children: 1.441, obesity adolescents: 1.047, obesity adults: 1.217; overweight children: 1.664, overweight adolescents: 1.333, overweight adults: 0.6792). Prevalence of T2D also saw a significant increase (447.5). For mean BMI, the results were not as significant, though mean BMI did slightly increase for children (0.0301) and adolescents (0.0289), while mean BMI decreased for adults both in crude measures (-0.0850) and age-adjusted (-0.2199). The rate of SHBP also saw a somewhat significant decrease (-0.3864). See Table 3 for the results. Countries that adopt FBDGs, from these results, experience more weight gain, greater prevalence of obesity and overweight, and T2D.

Tables 4-29 are the complete breakdown of Goodman-Bacon decompositions, TWFEs, and 2SDID estimates for each respective health outcome.

5.1 Other Potential Covariates

Rather than controlling for dietary and life choices that impact health, I should control for economic factors associated with dietary choices. As such, I should examine following covariates for health outcomes: median age, percent male, percent black, percent Hispanic, and possibly a measure of unemployment. However, the data on median age would force me to drop too many years or too many countries. Additionally, I was unable to racial and ethnic data over the duration of my study. To capture a measure of healthcare capacity, I could use health insurance coverage, physicians per capita, and surgical measures (to measure sophistication). Health spending as a percent share of GDP may be fine, but many countries price cap certain aspects of their healthcare sector. Again, however, I was unable to find data commensurate with the period of my study.

6 Problems to Solve and Possible Limitations

6.1 Stable Unit Treatment Value Assumption (SUTVA)

The US and Great Britain exhibit more cultural, economic, and scientific influence, spillover effects can occur and populations where their government may not explicitly recommend dietary guidelines, may nonetheless follow a diet similar to the one suggested by the United States. This is not spillover.

However, once the US adopts dietary guidelines, other individuals in other countries may follow these the US guidelines, even when their own respective countries have not yet adopted these guidelines. International committees and institutes consisting of joint research, such as the FAO and WHO, will influence similar macronutrient recommendations. Much research is led by US researchers and done in English. Another potential occurrence of spillovers are countries and regions with close relations, such as the Nordic countries adopting the Nordic Nutritional Recommendations in 1980 (before the adoption of FBDGs), Canada and the US, Belgium and Luxembourg,[15] and there are other joint efforts between Latin American countries and between African countries. It is also unreasonable to assume that countries that adopted FBDGs late did not change their diets beforehand, such as Afghanistan (2016), Kenya (2017), United Arab Emirates (2019), Peru (2019), Ecuador (2018), Cambodia (2017), and Sierra Leone (2016). As a result, my analysis is potentially more likely to underestimate the true effect of FBDGs, since non-treated individuals are potentially more likely to change their diet in adherence with the US and other more influential country's FBDGs.

6.2 Anticipation Effect

Before FBDGs are officially adopted by countries, most countries create new committees and research institutes in order to fund studies on diet and health. The results of these studies are often known years before the FBDG is officially published. In addition, related to the SUTVA assumption, countries that have conducted researched earlier will also affect the diets other countries.

6.3 Outcomes Affecting Treatment

Mentioned earlier in the paper, countries that experience worse health outcomes, such as CVD risk and mortality, diabetes, and obesity, could feel pressured to enact FBDGs. This would violate one of the assumptions for difference-in-difference. One way to test if this is the case, which is planned for future improvements of this paper, is to see if adoption of FBDG is spurred by increasing cases of the health outcomes I examine.

6.4 Differences in Implementation

Diet fads are notorious for becoming quickly and widely adopted by a new celebrity promotion or popular magazine. These fads vary between country to country, which will affect if I may underestimate or overestimate the effect of the FBDGs. There are also potential spillover effects for both diet fads and FBDGs. Culture and traditional diets may change how many individuals follow the dietary recommendations.

[15] Belgium adopted their FBDG in 2005, but Luxembourg never officially adopted one.

Differences in advertisements and advertising laws may distort the population's desire to follow the recommended diet. For example, Keller and Schulz (2011) found evidence that advertisements on candy, fast food, and other products high in fats and sugars distorted nutritional diet in children in Switzerland. Different countries have different laws on advertisements targeting children. On the other hand, countries that adopt a FBDG may promote or implement in varying methods and degrees. And even if countries do promote FBDGs strongly, different citizenry may have different priorities or worries other than their weight or CVD risk. Varying proportions of children in each country may consume school lunches provided by public schools. Public schools in each country would also differ in how well these lunches adhere to their national FBDGs. Lastly, some countries create FBDGs targeting subgroups, such as infants under age two, pregnant women, children, and adults. As a result, the entire population may not uniformly adopt these national dietary recommendations.

Diet and fitness are also correlated with each other and affect the health outcomes of my study. Guthold et al. (2018) found that globally more than a quarter of adults were insufficiently physically active. Guthold et al. (2018) also found that differences in income levels results in differences in the prevalence of insufficient physical activity. Therefore, countries with populations that rapidly change physical activity levels or experience rapid changes in economic growth or decline may affect exercise and the outcomes I am measuring.

Meat and dairy producers may also attempt to lobby and influence federal dietary recommendations (Nestle, 1993). They may fund research or run advertisements that influence the population's dietary choices. This could directly or indirectly influence FBDGs and the diets of the population. On the other side, there are individuals who ignore federal dietary recommendations and would be more likely to ignore other health advice, such as on smoking and exercising. Lastly, mentioned earlier, adopting FBDGs may be affected by the outcomes measured in this study. Countries with higher mean BMI and CVD risk may seek to adopt FBDGs.

7 Conclusion

With the limitations noted above, it may be the case that, ultimately, the parallel trends and SUTVA assumptions do not hold. Additional tests and econometrical techniques are required in order to extract a causal relationship between FBDGs and the health outcomes examined in this paper. Despite the lack of consensus among the proportion of macronutrient to facilitate weight loss and to prevent CVD risk, nearly every country encourages a lower fat diet and recommends starchy carbohydrates as a dietary staple. This paper has shown, with significant results, that the lower fat diet most FBDGs recommend has not promoted overall health and prevented chronic diseases. Although I found these significant results, I have not believe I have included enough covariates, due to country and period limitations.

Additionally, there are other health outcomes and chronic that may be more relevant to overall health and that the low-fat diet most FBDGs helps prevent.

Despite the variety of diets that exists today and existed in the past, additional research is necessary to determine if the low-fat diet does indeed promote overall health—and given the range individual sensitivity to certain diets and food preferences, it is questionable to recommend one type of diet across the population.

Appendices

A Tables

Table 1: FBDG Countries

Country	FBDG Name	Year Start
Albania	Recommendations on healthy nutrition in Albania	2008
Australia	Healthy Eating Pyramid	1980
Austria	The Austrian food pyramid - 7 steps to health	2005
Bangladesh	Dietary Guidelines for Bangladesh	2000
Belgium	Practical guidelines for healthy eating	2005
Benin	Benin's Dietary Guidelines	2015
Bulgaria	Food based dietary guidelines for adults in Bulgaria	2006
Canada	Canada's food guide	1992
China	Dietary guidelines for Chinese residents	1997
Cyprus	National nutrition and exercise guidelines	2007
Denmark	The Official Dietary Guidelines - good for health and climate	1995
Fiji	Food and health guidelines for Fiji.	1987
Finland	Food Circle	1987
France	The French National Nutrition and Health Program's dietary guidelines	2002
Germany	Ten guidelines of the German Nutrition Society for a wholesome diet	2005
Great Britain	Eatwell Guide	1994
Greece	National Nutrition Guide for Greek Adults	1999
Hungary	Dietary guidelines for the adult population in Hungary	1987
Iceland	Food-based dietary guidelines for adults and children from two years of age	2006
India	Dietary Guidelines for Indians	1998
Indonesia	Balanced Nutrition Guidelines	1995
Iran	Food-based dietary guidelines for Iran	1990
Ireland	Healthy Food for Life – the Healthy Eating Guidelines	2012
Israel	The Israeli food pyramid	1998
Italy	Dietary Guidelines for Healthy Eating– Revision 2018	1986
Japan	Dietary guidelines for Japanese	2000
Lebanon	The Food-Based Dietary Guideline Manual for Promoting Healthy Eating in the Lebanese Adult Population	2013
Malaysia	Malaysian Dietary Guidelines	1999
Malta	The healthy plate: Dietary guidelines for Maltese adults.	1986
Mongolia	Dietary guidelines for Mongolians	1990
Namibia	Food and nutrition guidelines for Namibia	2000
Nepal	Food-based dietary guidelines - Nepal	2004
Netherlands	Food-based dietary guidelines for the Netherlands	1981
Nigeria	Food-based dietary guidelines for Nigeria - a guide to healthy eating	2001
Norway	Norwegian guidelines on diet, nutrition and physical activity	2014
Oman	The Omani guide to healthy eating	2009
Philippines	Nutritional guidelines for Filipinos	1990
Poland	Principles of healthy eating	2009
Portugal	Food Wheel Guide	1977

Table 1 continued from previous page

Country	FBDG Name	Year Start
Romania	Guidelines for a healthy diet	2006
Saudi Arabia	Dietary guidelines for Saudis: the healthy food palm	2013
South Africa	Food-Based Dietary Guidelines for South Africans	2003
South Korea	General Dietary Guidelines for Koreans	1991
Spain	Eat healthy and move: 12 healthy decisions	2005
Sri Lanka	Food-based dietary guidelines for Sri Lankans	2002
Sweden	Find your way to eat greener, not too much and be active!	1989
Switzerland	Swiss Food Pyramid	1998
Thailand	Food-based dietary guidelines for Thai	1986
Turkey	Dietary guidelines for Turkey	2004
United States	Food Pyramid	1992
Vietnam	10 tips on proper nutrition for period 2013-2020	1995
Uruguay	Dietary guidelines for the Uruguayan population: for a healthy, shared and enjoyable diet	2005
Argentina	Dietary guidelines for the Argentinian population	2000
Bahamas	The new dietary guidelines for the Bahamas	2002
Barbados	Food-based dietary guidelines for Barbados	2009
Belize	Food-based dietary guidelines for Belize	2012
Bolivia	Food-based dietary guidelines for the Bolivian population	2013
Brazil	Dietary Guidelines for the Brazilian Population 2014	2006
Chile	Dietary guidelines for the Chilean population	1997
Costa Rica	Dietary guidelines for Costa Rica	1997
Cuba	Dietary guidelines for the Cuban population over two years of age	2000
Dominican Republic	The mortar of food and nutrition	2009
El Salvador	Dietary guidelines for Salvadorian families	2001
Grenada	Food-based dietary guidelines for Grenada	2006
Guatemala	Dietary guidelines for Guatemala. Recommendations for healthy eating	1996
Guyana	Food-based dietary guidelines for Guyana	2004
Honduras	Dietary guidelines for Honduras. Tips for healthy eating	2011
Jamaica	Food-based dietary guidelines for Jamaica: Healthy eating - Active living	2015
Mexico	Dietary and physical activity guidelines in the context of overweight and obesity in the Mexican population	2015
Panama	Dietary guidelines for Panama	1997
Paraguay	Dietary guidelines of Paraguay	2003
Saint Lucia	Dietary guidelines for Saint Lucia	2007
Venezuela	Dietary guidelines for Venezuela	1991

Table 2: Summary Statistics (1976-2016)

treatment		**No**			**Yes**		
Variable	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**	**Test**
year	2542	1996	11.834	3239	1996	11.834	F=0
high.blood.pressure.sum	1674	24.614	6.099	2133	26.614	8.456	F=66.521***
diabetes.type2.prev	1674	2896.2	2192.227	2133	4476.383	2302.407	F=460.712***
diabetes.type2.inc	1674	188.73	140.39	2133	245.786	112.007	F=194.529***
mean.bmi	2542	22.872	2.368	3239	24.538	2.086	F=806.235***
obesity.adult	2542	7.958	7.446	3239	13.008	7.497	F=650***
overweight.adult	2542	26.945	15.338	3239	41.115	15.367	F=1213.035***
obesity.children	2542	3.349	4.485	3239	6.503	4.832	F=646.1***
overweight.children	2542	9.836	9.1	3239	18.454	10.459	F=1082.655***
obesity.adol	2542	2.182	3.403	3239	4.18	3.457	F=482.401***
overweight.adol	2542	8.767	8.066	3239	16.142	9.136	F=1027.614***
median.age	496	20.083	4.907	632	27.943	7.718	F=390.535***
female	2542	49.662	3.078	3239	50.034	2.586	F=24.961***
physician	936	0.617	0.728	1847	1.955	1.209	F=967.689***
unemployment	1560	7.369	6.554	2028	7.683	5.319	F=2.506
gdp	2368	4491.92	11684.43	3108	11274.18	14960.36	F=332.257***
income	2542	9561	19395.71	3239	17536.36	19436.21	F=240.247***
year.start	0	NaN	NA	3239	2001.316	9.694	
… No	2501	98.40%		2009	62%		
… Yes	41	1.60%		1230	38%		
code	2542	69.903	39.854	3239	71.861	41.347	F=3.295*
treat	2542			3239			X2=1241.723***
… No	2542	100%		1995	61.60%		
… Yes	0	0%		1244	38.40%		

Statistical significance markers: * p<0.1; ** p<0.05; *** p<0.01

Table 3: Results

Outcomes	**Years**	**Observations**	**2SDID Estimates**
Mean BMI			
Children	1976-2016	5,781	0.0301* (0.0117)
Adolescents	1976-2016	5,781	0.0289* (0.0119)
Adults	1976-2016	5,781	-0.0850*** (0.0183)
Adults (age-adjusted)	1976-2016	5,781	-0.2199*** (0.0193)
Obesity			
Children	1976-2016	5,781	1.441*** (0.0862)
Adolescents	1976-2016	5,781	1.047*** (0.0632)
Adults	1976-2016	5,781	1.217*** (0.0910)
Overweight			
Children	1976-2016	5,781	1.664*** (0.1477)
Adolescents	1976-2016	5,781	1.333*** (0.1219)
Adults	1976-2016	5,781	0.6792*** (0.0905)
Type 2 diabetes	1990-2016	3,456	447.5*** (33.36)
High blood pressure	1990-2016	3,456	-0.3864* (0.1913)

Note: *p<0.1; **p<0.05; ***p<0.01

Table 4: Goodman-Bacon Decomposition (BMI age adjusted)

type	weight	avg_est
Earlier vs Later	0.19176	-0.15877
Later vs Earlier	0.12010	0.23310
Treated vs Untreated	0.68813	-0.19226

Table 5: TWFE (BMI age adjusted)

	Dependent variable:
	mean.bmi.age.adjusted
treat	-0.135^{***}
	(0.017)
Observations	5,781
R^2	0.011
Adjusted R^2	-0.021
F Statistic	61.734^{***} (df = 1; 5599)
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 6: Static Treatment (BMI age adjusted)

Dependent Variable:	mean.bmi.age.adjusted
treat = TRUE	-0.2199*** (0.0204)
S.E. type	Bootstrap (n = 500)
Observations	5,781
R^2	0.05381
Adj. R^2	0.05381
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 7: Goodman-Bacon Decomposition (obese children)

type	weight	avg_est
Earlier vs Later Treated	0.19176	-0.34571
Later vs Earlier Treated	0.12010	0.12446
Treated vs Untreated	0.68813	1.80501

Table 8: TWFE (obese children)

	Dependent variable:
	obesity.children
post	1.191***
	(0.077)
Observations	5,781
R^2	0.041
Adjusted R^2	0.010
F Statistic	238.929*** (df = 1; 5599)
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 9: Static Treatment (obese children)

Dependent Variable:	obese.children
treat = TRUE	1.441*** (0.0889)
S.E. type	Bootstrap (n = 500)
Observations	5,781
R^2	0.11228
Adj. R^2	0.11228
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 10: Goodman-Bacon Decomposition (obese adolescents)

type	weight	avg_est
Earlier vs Later Treated	0.19176	-0.25957
Later vs Earlier Treated	0.12010	0.14440
Treated vs Untreated	0.68813	1.33747

Table 11: TWFE (obese adolescents)

	Dependent variable:
	obesity.adol
post	0.888***
	(0.060)
Observations	5,781
R^2	0.038
Adjusted R^2	0.007
F Statistic	221.941*** (df = 1; 5599)
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 12: Static Treatment (obese adolescents)

Dependent Variable:	obesity.adol
treat = TRUE	1.047*** (0.0596)
S.E. type	Bootstrap (n = 500)
Observations	5,781
R^2	0.10006
Adj. R^2	0.10006
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 13: Goodman-Bacon Decomposition (obese adults)

type	weight	avg_est
Earlier vs Later Treated	0.19176	-0.70310
Later vs Earlier Treated	0.12010	-0.07906
Treated vs Untreated	0.68813	1.72410

Table 14: TWFE (obese adults)

	Dependent variable:
	obesity.adult
post	1.042***
	(0.084)
Observations	5,781
R^2	0.027
Adjusted R^2	−0.005
F Statistic	153.537*** (df = 1; 5599)
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 15: Static Treatment (obese adults)

Dependent Variable:	obesity.adult
treat = TRUE	1.217*** (0.0888)
S.E. type	Bootstrap (n = 500)
Observations	5,781
R^2	0.10006
Adj. R^2	0.10006
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 16: Goodman-Bacon Decomposition (overweight children)

type	weight	avg_est
Earlier vs Later Treated	0.19176	-0.77254
Later vs Earlier Treated	0.12010	0.31456
Treated vs Untreated	0.68813	2.18306

Table 17: TWFE (overweight children)

	Dependent variable:
	overweight.children
treat	1.392***
	(0.116)
Observations	5,781
R^2	0.025
Adjusted R^2	−0.006
F Statistic	144.875*** (df = 1; 5599)
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 18: Static Treatment (overweight children)

Dependent Variable:	overweight.children
treat = TRUE	1.664*** (0.1462)
S.E. type	Bootstrap (n = 500)
Observations	5,781
R^2	0.06636
Adj. R^2	0.06636
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 19: Goodman-Bacon Decomposition (overweight adolescents)

type	weight	avg_est
Earlier vs Later	0.19176	-0.67914
Later vs Earlier	0.12010	0.34623
Treated vs Untreated	0.68813	1.79986

Table 20: TWFE (overweight adolescents)

	Dependent variable:
	overweight.adol
treat	1.150***
	(0.098)
Observations	5,781
R^2	0.024
Adjusted R^2	−0.007
F Statistic	138.601*** (df = 1; 5599)
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 21: Static Treatment (overweight adolescents)

Dependent Variable:	overweight.adol
treat = TRUE	1.333*** (0.1205)
S.E. type	Bootstrap (n = 500)
Observations	5,781
R^2	0.06029
Adj. R^2	0.06029
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 22: Goodman-Bacon Decomposition (overweight adults)

type	weight	avg_est
Earlier vs Later	0.19176	-0.66512
Later vs Earlier	0.12010	0.06404
Treated vs Untreated	0.68813	0.95571

Table 23: TWFE (overweight adults)

	Dependent variable:
	overweight.adult
treat	0.538***
	(0.086)
Observations	5,781
R^2	0.007
Adjusted R^2	−0.025
F Statistic	39.226*** (df = 1; 5599)
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 24: Static Treatment (overweight adults)

Dependent Variable:	overweight.adult
treat = TRUE	0.6792*** (0.0950)
S.E. type	Bootstrap (n = 500)
Observations	5,781
R^2	0.02119
Adj. R^2	0.02119
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 25: Goodman-Bacon Decomposition (diabetes)

type	weight	avg_est
Earlier vs Later	0.12507	164.5181
Later vs Always Treated	0.12385	-404.3213
Later vs Earlier	0.12228	-166.8900
Treated vs Untreated	0.62879	569.0526

Table 26: TWFE (diabetes)

	Dependent variable:
	diabetes.type2.prev
treat	307.910***
	(41.358)
Observations	3,807
R^2	0.015
Adjusted R^2	−0.030
F Statistic	55.428*** (df = 1; 3639)
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 27: Static Treatment (diabetes)

Dependent Variable:	diabetes
treat = TRUE	447.5*** (34.43)
S.E. type	Bootstrap (n = 500)
Observations	3,456
R^2	0.08475
Adj. R^2	0.08475
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 28: Goodman-Bacon Decomposition (high blood pressure)

type	weight	avg_est
Earlier vs Later	0.12507	0.06858
Later vs Always Treated	0.12385	2.51916
Later vs Earlier	0.12228	0.79910
Treated vs Untreated	0.62879	0.51414

Table 29: TWFE (high blood pressure)

	Dependent variable:
	high.blood.pressure.sum
treat	0.742***
	(0.128)
Observations	3,807
R^2	0.009
Adjusted R^2	−0.036
F Statistic	33.809*** (df = 1; 3639)
Note:	*p<0.1; **p<0.05; ***p<0.01

Table 30: Static Treatment (high blood pressure)

Dependent Variable:	high.blood.pressure.sum
treat = TRUE	-0.3864* (0.1743)
S.E. type	Bootstrap (n = 500)
Observations	3,456
R^2	0.00402
Adj. R^2	0.00402
Note:	*p<0.1; **p<0.05; ***p<0.01

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