Of Junk Food and Junk Science

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Abstract: Of Junk Food and Junk Science

The popular press has triumphantly announced that the cause of the obesity epidemic is "junk food." After a moment's reflection, however, it seems likely that the true causal structure of the obesity epidemic can be neither single-equation nor univariate. Therefore, while the hypothesis that "junk food" is *the* cause of obesity has little *a priori* plausibility, these articles in the popular press present a testable hypothesis that, in spite of some measurement impossibilities, is tested here. While one can always argue about p values etc., it is safe to say that the results show no evidence to indicate support for a causal link. The second section of the paper explains this result and suggests a rudimentary structural model of obesity which begins to address the issues of specification error, simultaneity, etc., that plague much of the obesity research. This model shows that because of the dynamic nature of weight status, there is no necessary reason to expect to find a statistical relation between a person's observed weight and the amount he or she is currently eating or exercising. Therefore, studies which regress weight, obesity, or the probability of obesity on eating and exercise patterns have serious specification error. Further development of structural econometric models of obesity may lead to consistent estimates of the partial effects of exogenous variables on obesity levels. We conclude with a discussion of the implications for policy development and industry.

Of Junk Food and Junk Science

Initially, the idea of a Granger causality study of junk food and obesity arose as an attempt to inject a little humor into a professional meeting. There is some precedent for such econometric humor. Thurman and Fisher published a Granger causality study of eggs and chickens in the AJAE about 20 years ago. Since it is 100% certain that all chickens are caused entirely by eggs and all (chicken) eggs are entirely caused by chickens, this was more of a humorous test of the methodology than a potential resolution of the age-old philosophical question. Fortunately, the results showed a reasonable likelihood of a connection between eggs and chickens, so Granger's Nobel prize is probably safe. However, after some thought, we realized that a Granger causality study of junk food and obesity was not in the same league as one for chickens and eggs, and had the potential to make a contribution to the obesity literature.

In recent years, the popular press has announced that the American obesity epidemic is being caused by the food industry, specifically companies selling "junk food." This claim may be found in articles such as, "Junk Food, TV Driving Kids to Obesity (Gordon in the Washington Post), "Junk Food Is Fooling People into Overeating" (Henderson in the Times London), "TV Ads Entice Kids to Overeat, Study Finds" (Mayer in the Washington Post), "Junk Food Giants Spend Billions Brainwashing Consumers & Buying Politicians" (Organic Consumers), and many others. Other articles warn of the potential danger of junk food, with titles such as "Don't Even *Think* of Touching that Cupcake" (Kershaw in the New York Times), and "Fighting Obesity, but Fronting for

Junk Food" (Melz in the Boston Globe). Even reading past the headlines, the articles typically present a simple picture of cause and effect, i.e. increases in junk food consumption have led to increases in obesity rates. Most articles contained little or no discussion of the complexity of factors that may be related to the rise in obesity. While these articles generally present little or no evidence to support their strident claims, they may not be totally devoid of legitimate content. They may contribute to scholarly inquiry by presenting a clear, testable hypothesis. If the popular press is correct that junk food is *the* cause of obesity, then one would think that a statistical relationship between per capita consumption of junk food and aggregate rates of obesity in the United States could be observed.

It is not clear that the actual line of causality between junk food and obesity is as obvious as these articles suggest. It is clear that companies specializing in donuts, candy, fried chicken, etc. sell foods that are high in calories and/or sugar or fat. This is not disputed. It is also clear that there is a fairly predictable relationship between calories consumed and the common measure of obesity, the body mass index (BMI), but the exact nature of this connection is not so obvious. While it is certain that consumption of large quantities of these junk foods can certainly cause obesity, it is not at all certain that this is the underlying causative factor. Indeed, the line of causality could run the other way. The obesity epidemic may be arising as a result of other causative factors that create an increased demand for junk foods. As people become obese, their maintenance calorie level increases substantially. Therefore, obese people become hungry unless they eat

large quantities of calories. These junk foods are a readily-available source of cheap, tasty calories which can provide the function of preventing hunger for the obese. Therefore, if junk food consumption and obesity were both increasing, it is a logical possibility that the increase in obesity rates is the cause of the increasing the demand for high calorie foods rather than the result of it.

However, since obesity is one of the factors determining caloric consumption, and caloric consumption is one of the factors determining obesity, it is obvious that obesity and food consumption are both endogenous variables in a system of structural equations. While a significant amount of responsible analysis of the obesity problem is underway, it is fair to say that the true casual structure of obesity is not well-understood. At this point, it seems likely that in addition to the underlying basic thermodynamics, other factors including, physiological, psychological, social, cultural, and economic components are involved in what may be a very complex set of relationships.

Therefore, even though it is highly unlikely that the causal structure of obesity can be adequately represented by any univariate, single equation structure, an econometric analysis of such a model is justified as a test of the hypothesis currently being trumpeted in the popular press that junk foods are the cause of the obesity epidemic. It is equally reasonable to examine the possibility that the obesity epidemic is causing a rise in junk food consumption. If the linkages were really this simple, it should be possible to present empirical evidence to support one or the other of them with a Granger causality test.

Methodology

Every sophomore is aware of the *post hoc, ergo proper hoc* fallacy. It is clearly too simple to suggest that correlated events have a causal relationship. However, Granger's suggestion is more sophisticated than that. He suggested a clever test statistic to see if patterns in preceding values of a variable add a statistically significant amount to the ability to forecast subsequent values. While this is a form of "after this, therefore because of this," it is a more sophisticated form. While there are many possible ways of calculating a Granger causality test, the most straightforward simply compares the residual sum of squares from a vector autoregression that contains lagged values of the proposed "casual" variable with residual sum of squares from one that does not. For example, to test if Y Granger-causes X, first estimate two vector autoregressions:

$$x_t = \alpha_1 + \sum_{i=1}^{\rho} \beta_i x_{t-i} + \gamma_i y_{t-i} + \varepsilon_t$$

.

$$x_t = \alpha_2 + \sum_{i=1}^{\rho} \phi_i x_{t-i} + e_t$$

Then, denoting the residual sums of squares from these two regressions as SS_1 and SS_2, the test statistic for the null hypothesis that y does not Granger-cause x is:

$$\frac{(SS_2 - SS_1)/\rho}{SS_1/(T - 2\rho - 1)},$$

which is distributed F with ρ and T - 2ρ -1 degrees of freedom as long as the residuals are normally distributed, or the sample sizes are large enough to rely on the central limit theorem. This amounts to a test of whether adding lagged values of Y add a statistically significant amount of reduction in the error sum of squares of the regression.

Data

As with many empirical studies in social science, acquiring the data needed to test these hypotheses is not a trivial task. Even defining the concepts of obesity and junk food is not straightforward. Therefore we generated some proxies. While it would be fairly easy to find disagreement on the definition of obesity, some objective measures are available. Defining and measuring "junk food" is considerably more difficult. The Body Mass Index (BMI) is a commonly-used measure of obesity. It is calculated as the weight (in kilograms) divided by the square of height (in meters).[1] The World Health Organization considers adults with a BMI between 25.0 and 29.9 to be overweight, and those with a BMI of 30.0 and above as obese. (World Health Organization; Centers for Disease Control and Prevention). Therefore, we measure the rate of obesity in the U.S. by the proportion of the population with a BMI of 30 or above.

To attempt to measure the rate of national per capita "junk food" consumption, we developed an *ad hoc* "junk food index" (JFI). First we collected the annual sales data for

[1] To calculate BMI using pounds and inches, the formula is the product of weight (in pounds) and 703, divided by height (in inches) squared.

a group of companies who sell mostly foods with high sugar or fat content and/or are generally believed to be mostly devoid of other healthful nutrients. Sales data were collected for the period 1990 through 2006 for the following companies (with some of their signature products and brands in parentheses): Interstate Bakeries (Twinkies, HoHos), McDonalds (Big Mac), Hershey Foods (Hershey's Chocolate Bar, Reese's, Almond Joy), Coca Cola (Coca Cola, Sprite), Pepsico (Pepsi, Mountain Dew) and YUM Brands (Pizza Hut, Taco Bell, KFC), and Kelloggs (Frosted Flakes, Pop-Tarts). Sales data were obtained from the Compustat North America database. Because of the spin-off of Yum Brands from Pepsico, we combined the sales for these two companies in the years after the spin-off. Second, the aggregate annual sales of this group of companies was deflated to real dollars by the food component of the consumer price index and divided by population. This gives an *ad hoc* measure of real, per capita "junk food" consumption in the United States. These data are shown in table 1 and plotted in figure 1. A brief glance at the data casts doubt on the hypotheses that obesity rates are caused by "junk food" consumption. Real per capital consumption of "junk food" from these companies is essentially the same in 2005 as it was in 1996, but the incidence of obesity has grown from 16.8% of the U.S. population in 1996 to 24.4% in 2005, a 45% increase.

Data Validity

While it might at first appear that our *ad hoc* "junk food" index is a somewhat arbitrary measurement device, this criticism raises several other issues. The problem is not just a measurement problem; it is more serious than that. The concept of "junk food" itself has

little scientific validity and is not really subject to measurement. There are several reasons for this. First, all foods fall on a multivariate continuum of nutritional merit. Assuming one could actually measure "aggregate nutritional merit" of a food item in some reasonable way, as one goes toward zero on that continuum, what threshold level should cause a food to be categorized as junk? Clearly it is not reasonable to pick an arbitrary cut-off level and simply classify all foods as "junk" or "non junk". Nutrition is much more complex than that. Second, the nutritional merit of a food has many dimensions; one for each important nutrient that potentially promotes health and one for each potentially harmful ingredient. Moreover, some compounds may be beneficial at low levels and harmful at high levels. Even if one could find agreement on which compounds should be included in which category, there is the problem of determining how increasing amounts of favorable ingredients could compensate for lack of other favorable ingredients or the inclusion of harmful ingredients.[2] This mind-boggling task makes it clear that a threshold concept of "junk food " is not a concept that has much potential scientific validity and is certainly not a concept that lends itself to precise measurement.

Despite the problems described in the preceding paragraph, the term "junk food" is one that is widely used in both the popular press and the vernacular. While it may defy easy definition, to paraphrase Justice Potter Stewart's comment on obscenity, most people

[2] A good example is the debate about whether the beneficial fats in fish more than compensate for possible damage from toxic metallic compounds.

"know it when they see it." Therefore, while our "junk food" index is admittedly imprecise, it does measure something related to the claims found in the media. It seems clear that the aggregate sales of these firms we include measures something about the what the popular press, which commonly targets foods such as Big Macs and Twinkies, considers to be "junk food" consumed in the United States. The lack of precision of the measurement is certainly not as much of a problem as the lack of validity of the concept being measured. Furthermore, any possible potential lack of validity of the "junk food" index will be rendered irrelevant by the arguments made in the second part of this paper. There we show that regressing obesity levels on eating and exercise patterns is a severely flawed econometric structure. Because of the dynamics of obesity, not only is there no reason to expect to find any statistical relationship between aggregate junk food consumption and overall obesity levels, but there is no reason to expect to find a statistical relationship between aggregate total food consumption and overall obesity levels. These issues are explored after the econometric results are examined.

Econometric Results

Given the nature of the problem, it seems likely that only short lags are relevant. This assertion is verified by the econometric results. Lags longer that two periods were highly insignificant. The Granger causality hypotheses and results are shown below.

H_0: "Junk food" consumption does not Granger-cause obesity.

The two necessary regression equations are:

$$\%Obese_t = \alpha_1 + \beta_1 \%Obese_{t-1} + \beta_2 \%Obese_{t-2} + \gamma_1 JF_{t-1} + \gamma_2 JF_{t-2} + \varepsilon_t$$

$$\%Obese_t = \alpha_2 + \phi_1 \%Obese_{t-1} + \phi_2 \%Obese_{t-2} + e_t$$

The first regression had an $R^2 = 0.9869$ and the second had an $R^2 = 0.9850$. This gives a hint about how the results will come out. The parameter estimates, t and p values for regression one and two are shown in table 2.

The calculated value of the $F_{2,10}$ test statistic is 0.719. The critical value of the F statistic for alpha = 0.05 is 4.1028. Therefore, do not reject the null hypothesis that junk food consumption does not Granger-cause obesity. While failure to reject the null hypothesis does not constitute evidence for accepting the null, one has to note the nearly total absence of evidence for rejecting the null in this case. Even if one were willing to accept an alpha level of 0.2, the critical value of the F statistic would be 1.899, and the calculated F value of 0.719 still is not even close.

The alternative logical possibility is that obesity is causing the demand for junk food. The formal hypothesis statement is:

H_0: Obesity does not Granger-cause junk food consumption.

The two necessary regression equations are:

$$JF_t = \alpha_1 + \beta_1 JF_{t-1} + \beta_2 JF_{t-2} + \gamma_1 \%Obese_{t-1} + \gamma_2 \%Obese_{t-2} + \varepsilon_t$$

$$JF_t = \alpha_2 + \phi_1 JF_{t-1} + \phi_2 JF_{t-2} + e_t \quad .$$

The third regression had an $R^2 = 0.767$ and the fourth had an $R^2 = 0.746$. The parameter estimates, t and p values are shown in Table 3. The calculated value of the $F_{2,10}$ test statistic is 0.45 which is again a very long ways from the critical value for alpha = 0.05 of 4.1028 or the critical level for alpha level of 0.2 of 1.899. Therefore, do not reject the null hypothesis obesity does not Granger-cause junk food consumption. Again, the lack of support for rejection of the null hypothesis is nearly total.

Sample Size, Power and Distribution of Residuals

Given the relatively small number of observations compared to the number of parameters being estimated, one can legitimately wonder if the test statistics have adequate power. When one fails to reject a null hypothesis for alpha = 0.05 or even 0.2, it is certainly a logical possibility that a larger sample would cause rejection of the null hypothesis. However, in this case, the calculated values of the F statistic are so tiny that no amount of data would cause rejection of the null. For example, consider the largest F statistic which is for the main hypothesis that "junk food" consumption does not Granger-cause obesity. The calculated $F_{2,10}$ value is 0.719. In order to reject the null hypothesis at the 5% level, the calculated $F_{2,10}$ value would have to exceed 4.1. Clearly, more data would affect the threshold rejection level. If there were 65 data points, the test statistic would have 2 and 60 degrees of freedom and the critical value would fall to 3.15. With infinite data, the test statistic is $F_{2,\infty}$ and the critical value would be 3.0, still far above the calculated value

of 0.719. Therefore, even though the length of the obesity epidemic does not provide us with many annual data points, the extremely small values of the test statistics suggest that failure to reject the null hypotheses is not simply due to lack of data.

Additionally, the relatively small sample size raises legitimate questions about the distribution of the test statistic. In order for the test statistic to have an F distribution with a small sample, the residuals must be normally distributed. The normal probability plots for the residuals for the four regressions all appear reasonably normal, but if non-normality were suspected, it would be a suggestion of slightly "fat tails" i.e. "chubby tails." This would imply that the test statistic might also have "chubby tails" meaning that the test might have a tendency to reject the null too often. Therefore, if the null had been rejected, one could argue that it was a spurious result due to a violation of the assumptions. However, this departure from normality can not be blamed for a failure to reject, and one is forced to conclude that the evidence is adequate to reject the single equation univariate model structure that "junk food" is *the* cause of obesity.

A formal test of the assertion commonly found in the popular press shows that the lack of evidence in support of Granger causality is nearly total. Moreover, given the complex multivariate continuum of foods characteristics that exista, the concept bifurcating food into "junk food" and "non-junk food" has very little validity.

Toward a Structural Model of Obesity

The true causal structure of obesity is likely much more complex than the simplistic relationships proposed in the popular literature and tested above. Almost certainly, the

actual causal structure of obesity is multi-equation, multivariate, and dynamic. It is not necessary to point out to most scholars the dangers of specification error, the difference between a reduced form and a structural model, or the folly of estimating a dynamic structure with a static form. But in the interest of promoting discussion of an adequate econometric structure for the obesity problem, a simple structural form is suggested below.

Without making any claims of physiological expertise, basic thermodynamics requires that people gain weight[3] when the calories they consume exceed the calories they burn. Since it is important to separate the effects of biology from individual choices, calories burned may be divided into the maintenance requirement (M) and the amount consumed by exercise and factors other than maintenance. Net caloric intake (NC) may be defined as calories eaten minus calories burned by work, exercise, etc. Then, we can conceptualize excess calories per unit of time as the difference between net caloric intake and the maintenance requirement (M). This conceptualization separates the issues of biology and individual choice. The maintenance requirement is primarily determined by who we are and net caloric intake is primarily determined by the eating and exercise that we choose. The maintenance requirement is a function of many predetermined variables that would have to be accounted for, but ignoring those temporarily, it is also a function of the endogenous variable weight (W). The simplest possible form for a maintenance

[3] We switch from BMI to weight because height will be one of the predetermined variables in a more complete model.

caloric requirement function would be a linear function of weight, $M = \alpha_0 + \alpha_1 W$. While the coefficients would be affected by the characteristics of the individual, example coefficients would be $\alpha_0 = 800$ and $\alpha_1 = 8$ giving a maintenance level of 2000 calories per day for a 150 pound person. While the Forbes equation (Forbes) suggests that the relationship between excess calories and weight gain may not be quite linear, for the sake of simplicity we will assume that each 3500 excess calories causes one pound of weight gain. Given this simplifying assumption, weight gain is given by a simple first-order, first-degree differential equation:

$$\frac{dW}{dt} = \left[NC - \alpha_0 - \alpha_1 W \right] \phi ,$$

where phi is 1/3500.

Therefore, basic thermodynamics says that overeating and lack of exercise do not cause obesity, they cause weight gain. While everyone knows this, it is a crucial distinction that has not been adequately incorporated in much obesity research. If a person is obese, it is not because of their current eating and exercise habits. Indeed, one might expect the obese to have a motivation to exercise and diet more. While the existence of obesity reflects a history of excessive caloric consumption relative to exercise, it says nothing in particular about their current eating and exercise habits. As a result, the common practice of regressing obesity levels (or the probability of obesity) on a vector of eating and exercise habits is severely mis-specified. While there is no necessary connection between a person's obesity status and their current eating habits, one might argue that if

one has constant eating and exercise habits, then it would be valid to look for relationships between eating, exercise and obesity. However this assertion is also flawed because of the dynamics of obesity.

The dynamics of obesity cause there to be no necessary relationship between current eating and exercise habits and weight even if eating and exercise habits are constant over time. This may be explained by the simple differential equation for weight gain shown above. For a net caloric intake which is greater than maintenance, the change in weight is positive. If the net caloric intake is constant at this level, weight increases over time. The increase in weight causes the maintenance requirement to rise and the rate of weight gain to fall until an equilibrium weight is reached where the net caloric intake equals the maintenance requirement. Therefore, while a constant set of eating and exercise habits totally determine a person's *equilibrium* weight, they have no relationship to this person's weight at a point in time. For example, consider a person weighing 150 pounds who consumes a net caloric intake of 2400 calories every day. Since the maintenance level for a 150 pound person is 2000 calories, initially there will be an excess intake of 400 calories per day. But with a constant intake of 2400 calories there will be a weight gain each day, and the weight gain increases the maintenance requirement thereby reducing the excess intake until eventually the person's weight reaches 200 pounds. At this point, the maintenance requirement will have risen from the 2000 calories/day required for a weight of 150 pounds to 2400 calories, and weight will equilibrate at 200 pounds. Therefore, while a 150 pound person who has a net caloric intake of 2400 calories has an

equilibrium weight of 200 pounds, since his current weight is not in equilibrium, it will be misleading to associate their eating and exercise habits with a weight of 150 pounds. Conversely, a 300 pound person who consistently eats 2400 calories per day will also eventually arrive at a weight of 200 pounds because at 300 pounds her maintenance requirement exceeds 2400 calories. In fact with this model, anyone who has a constant net caloric intake of 2400 calories will eventually weigh 200 pounds, regardless of his or her current weight.

This means that in a cross-sectional sample of individual observations[4], there is no reason to expect to find a connection between weight and net caloric consumption when a significant part of the sample is not at their equilibrium weight. This is especially significant in a country like the U.S. where a large portion of the population has been gaining weight over the last several decades. Unless the sample is restricted to individuals whose weight is stable, the common econometric structure regressing obesity on eating, exercise, TV watching, etc. is severely flawed. By integrating the differential equation for weight gain, solving for W(t) and using La Hopital's rule to take the limit of W(t) as t gets large, we see that the equilibrium weight is $[NC- \alpha_0]/\alpha_1$. Therefore, given the parameters of the model, a person's *equilibrium weight* is totally determined by net rate of caloric intake. However, for those whose weight is not in equilibrium (possibly a large percentage of the population), there is no necessary relationship of any kind between a

[4] Of course, there is no hope for aggregate measures.

person's weight at a point in time and the net rate at which they are consuming calories.[5] For example, one could find 100, 200, 300 and 400 pound people all who have a net caloric intake of 2400 calories. The weight of the first person would be increasing, while the third and fourth persons' weights would be decreasing. Only the second person would have a stable weight at 200 pounds. A sample of these four people would have a beta of zero if weight were regressed on net caloric consumption.

In general, if some people in the sample are not at their equilibrium weight, the partial effects of exogenous variables on weight can be estimated accurately only when the rate of change in weight is also accounted for. This may be seen by re-arranging the differential equation for weight by putting the rate of change of weight on the right hand side:

$$W(t) = -\frac{\alpha_0}{\alpha_1} + \frac{1}{\alpha_1} NC(t) - \frac{1}{\alpha_1 \phi} \frac{dW}{dt}.$$

Therefore, when obesity (W) is regressed on net caloric consumption (NC), we are really estimating a reduced form with an omitted variable, if the rate of change of weight is omitted. Since we know that the rate of change of weight is not orthogonal to net caloric consumption, the estimates of the reduced form coefficients will be biased. This is especially important in the context of a country, like the U.S. where a substantial proportion of the population is gaining weight.

[5] There also is no necessary relationship between the probability a person is obese and their choices if their weight is not in equilibrium.

Suggestions for Model Specification

Obesity is an important social problem which unfortunately has been the victim of a fair amount of junk science in the popular press. Creation of a multivariate multi-equation dynamic structural econometric model of individual obesity with correct functional forms suggested by nutritional scientists (rather than linear approximations) could make a significant contribution to public policy decisions.

The basic overview might be as follows. Let **X** be a vector of predetermined variables measuring individual physiologic characteristics such as age, height, thyroid function, etc. Let **Y** be a vector of choice variables such as diet and exercise choices. Since maintenance calories might be affected by choices as well as individual characteristics and body weight (W), let M = m(**X,Y**,W). Total caloric intake may also be of the form C = c(**X,Y**,W). Since the amount of calories consumed by a 300 pound person running a mile might be more than what a 100 pound person would use, the function describing calories burned by activity choices may also be of the form B = b(**X,Y**,W). Hopefully, the physiology literature can give some guidance for the proper functional forms for these functions, which probably will involve some non-linearities. Then, the basic differential equation would probably be:

$$\frac{dW}{dt} = f\left(C - B - M\right) \ ,$$

where f, again, has the proper functional form. Even using linear approximations for these equations may be a significant improvement over single-equation models with obesity or the probability of obesity as the dependent variable. The dynamic problems may be avoided by using the rate of weight gain (loss) as a dependent variable rather than weight or obesity.

Discussion and Implications

An accurate and full understanding of the interaction of food choices (including what is commonly referred to as "junk food" and other quantity and quality factors), lifestyle choices (including work and leisure choices), and weight status is critical to the development of sensible policy choices. The news is full of blanket condemnations of the current evil, be it "fast food," "junk food," "too much TV," "too little exercise," or "inadequate physical education in the schools." A better understanding of the causes of obesity will help us get the policies right so that they have the intended effects. Individuals will then be able to make choices based on sound science.

One of the principal implications of our findings is that they argue against a simplistic explanation of the obesity problem. Similarly, our findings argue against a simple solution, i.e. eliminating the simple cause. It is tempting to fall prey to the fallacy that coincident events must somehow be related and to search for a cause and effect relationship. Over the last few decades obesity rates have increased along with the consumption of junk food, sales of video games, and a decline in physical education in

the schools. It is tempting to blame some or all of these factors for the rise in obesity and it is easy to construct a convincing rationale for the case. Of course, the last few decades have also seen the introduction of cell phones, widespread adoption of the Internet, and rapid social change. Which of *these* factors, if any, is responsible for the rise in obesity? The search for a single cause and a single solution grossly oversimplifies a problem that is almost certainly extremely complex.

The attention surrounding the publication of findings implicating a single cause of a health problem is often substantial. We are frequently told that a single factor is associated with an increased or decreased risk of a disease. The natural reaction is to avoid the poison and seek the cure. However, after numerous food scares and warnings, what are we to believe? First caffeine is bad for you, then it's good for you. Alcohol is bad for you ..... well maybe alcohol in moderation is actually beneficial. What are Joe and Jane consumer to believe? The outcome is a confused public that has tried many of the quick fixes that have failed to solve its problems and eventually becomes inured to health and nutrition news of any kind. Furthermore, the sound-bite nature of most news reporting makes it difficult to sort out "good" science from "junk" science and encourages the proclamation of simple prescriptions that do not accurately reflect the complexity of the problems.

Despite the widespread nature and increasing severity of the problem, little progress has been made in addressing the obesity epidemic that is sweeping the U.S. and much of the developed world. The problem is severe. It manifests itself in poor health, time away

from work, higher public and private health costs, and increased mortality rates. We don't have a good understanding of the causes and potential solutions and we certainly don't know what public policies might be most effective. What is needed is research that incorporates the best medical and social science to develop an understanding of the complex causes of obesity both at both the individual and societal levels. This knowledge could serve as a basis for sound public policy that could help consumers make informed choices and encourage the pursuit of healthy lifestyles.

There are a host of public policy issues associated with the obesity epidemic that could be informed by a better understanding of how the various factors interact to influence weight gain and weight status. For example, we expect that much public policy would be targeted at schools where the audience is impressionable and lifelong diet and exercise patterns are in the formative stages. Notable programs include school lunch programs, policies on what types of food and beverages may be served on campus, physical education and after school sports programs, and curriculum addressing nutrition and exercise. Restaurants have also been the subject of significant debate regarding the impact of "fast food" on weight status. Recently, Los Angeles banned new fast food restaurants in the low income area of south Los Angeles. Policies encouraging or discouraging certain types of restaurants and the availability and placement of nutritional information could benefit from a better understanding of the causes of obesity. Finally, given the importance that diet and exercise play in the nation's health, government has an interest in educating citizens to lead healthful lives. From an economic perspective,

policies that encourage healthy eating and exercise may translate into more productive workers, less time off due to sickness, and lower health care costs for companies and governments.

The food industry also has a large stake in the public policy debate over the causes of obesity. Policies and regulations concerning where and when their products may be sold, how they are taxed, and what information must be made available to consumers will be influenced by our collective understanding of how food consumption affects weight status. There are many opportunities for food companies to exploit the obesity epidemic. Over the years, many companies have tried to market the diet food of the day to Americans hungry for the latest slimming fad-food. More recently, Kraft has marketed "100 Calorie Packs" of items including Oreos, Chips Ahoy, and Wheat Thins. Disney has recently partnered with Imagination Farms to sell Disney-branded fruits and vegetables to children. A more complete understanding of the causes of obesity should enable companies to profitably design, package, and market products that will enable consumers to make wise, healthful choices.

Concluding Comments

Even though it is preposterous to suppose that the cause of the obesity epidemic is either single-equation or univariate, a formal test of this type of causal relationship between per capita "junk food" consumption and the rate of obesity shows that the lack of evidence in support of Granger causality in either direction is nearly total. We also showed that there is no relationship between a person's weight and how much he or she eats and

exercises. Rather, these factors determine whether a person is gaining or losing weight (not actual weight). The implication of this result is that studies in which current weight (or the proportion of the population that is overweight) is regressed on factors related to current eating and exercises habits are seriously flawed. We should be very cautious in interpreting the results of these studies, which have serious specification error. Of course, our research neither suggests that food choices are not an important causative factor in the obesity epidemic, nor that obesity is not an important factor in food choices. Almost certainly, the actual causal structure of obesity is multi-equation, multi-variate, and dynamic. A more promising approach for econometric models is to use the rate of weight change as a dependent variable rather than weight when trying to determine the partial effects of things like "junk food" consumption, TV watching, etc. In general, more attention needs to be paid to the underlying thermodynamics in econometric research of obesity, and over-simplified explanations must be avoided. The popular press is rife with this articles that gloss over the complexities and then do not pass the test when confronted with the evidence. Perhaps the media should stick to their time-honored "man bites dog" stories rather than pretending to contribute to the discourse on public policy.

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Table 1. U.S. Real Per Capita "Junk Food" Consumption and Obesity Rates

Year	% Obese*	"Junk Food" Index
1990	11.6	$135.24
1991	12.6	$141.33
1992	12.6	$152.17
1993	13.7	$159.44
1994	14.4	$172.39
1995	15.8	$186.36
1996	16.8	$188.13
1997	16.6	$182.88
1998	18.3	$180.42
1999	19.7	$172.83
2000	20.1	$171.79
2001	21.0	$180.73
2002	22.1	$171.82
2003	22.8	$178.31
2004	23.2	$182.98
2005	24.4	$188.62
2006	25.1	$191.63

*Sources: 1990-2002: Centers for Disease Control and Prevention, Behavioral Risk Factor Surveillance System, Trends Data, http://apps.nccd.cdc.gov/brfss/Trends/TrendData.asp; 2003-2006: Centers for Disease Control and Prevention, Behavioral Risk Factor Surveillance System, Prevalence Data, http://apps.nccd.cdc.gov/brfss/index.asp.

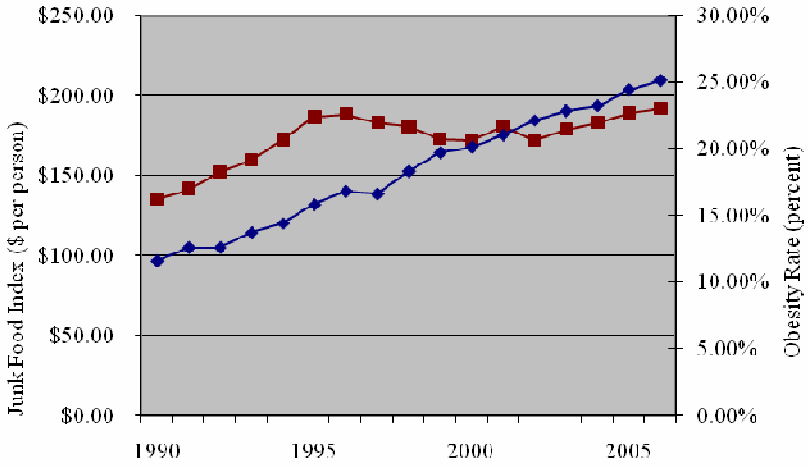

Figure 1. Real, Per Capita "Junk Food" Index and Obestiy Rates. 1990 to 2006

Table 2. Parameter estimates for H_0: Junk food consumption does not Granger-cause obesity

Parameter	Estimate	t value	p value
α_1	-1.18	-0.60	0.56
β_1	0.58	2.04	0.07
β_2	0.39	1.41	0.19
γ_1	0.02	0.75	0.47
γ_2	-0.002	-0.09	0.93
α_2	1.02	1.50	0.16
φ_1	0.64	2.38	0.03
φ_2	0.36	1.35	0.20

Table 3. Parameter estimates for H_0: Obesity does not Granger-cause junk food consumption

Parameter	Estimate	t value	p value
α_1	59.37	2.63	0.025
β_1	0.92	3.32	0.008
β_2	-0.29	-1.15	0.28
γ_1	-1.54	-0.48	0.64
γ_2	1.97	0.61	0.55
α_2	54.96	2.66	0.02
φ_1	0.93	3.50	0.004
φ_2	-0.23	-1.03	0.32