WHY SHOPPING FREQUENCY IS A KEY DETERMINANT OF DIET-BASED DISEASES

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There are many economic and medical studies that attempt to explain why poor community health is correlated with limited access to stores that carry a full assortment of affordable foods, most notably, healthful fresh fruits and vegetables. Medical studies tend to treat the built environment as exogenous, implicitly suggesting that poor community diets and resulting health outcomes are caused by a supply-side market failure. Economic studies prudently recognize that the built grocery environment is an equilibrium outcome that depends on consumer demand. Yet both economic studies that control for the endogeneity of the build environment and medical studies find only weak—or even nonexistent—associations between store densities and food choice (see Colby (2016) for a review).

While the basic idea—that consumers will purchase less healthful foods if healthful foods are more difficult to obtain—is intuitively appealing, formal theory that provides an economic foundation has not been well developed. Such economic theory is needed because the argument stemming from popular intuition has a major hole. If shopping at a store that offers wanted healthful foods is inconvenient, what's to stop households from circumventing the difficulty by simply stocking up on healthful foods as they shop less frequently? Given the underwhelming empirical supporting evidence for the popular intuition, a theory is needed that can answer that question. I provide an economic model that answers that question when combined with food facts from food chemistry and medical sciences. The quick answer is that healthful food tends to decay quickly, so that carrying such perishable foods over longer periods of times is at best costly, and at worse, infeasible.

This hypothesis is empirically explored by seeing if households that shop less frequently shift their diets away from nutrients that decay rapidly. Focus is on specific food characteristics and nutrients that fulfill the following criteria.

1. Clearly related to the stability of food products.

2. Scientific consensus says consuming foods containing the characteristic or nutrient on health outcomes affects health outcomes.

3. The magnitude and/or variance of current consumption levels of the food characteristic or nutrient imply that public policies aimed at changing the consumption of those foods or nutrients have the potential to prevent a large number of deaths.

All measured associations between shopping frequency and food and nutrient consumption levels support the hypothesis. While the endogeneity of shopping frequency is not controlled for, I argue that the persistence of the correlations provide strong evidence that shopping frequency is an important causal driver of dietary patterns that are known to cause diet-based disease. Given shopping frequency's association with grocery store densities, I therefore conjecture that shopping frequency may be the "missing link" between the built environment and community health outcomes. Since shopping frequency—*not store densities*—is likely to be the true causal mechanism determining food and nutrient choices, focusing on built environments while ignoring

shopping frequencies may explain the weak relationships between built environments, food choice, and health outcomes.

Section I presents a theory of perishable vs. nonperishable food choice, shopping frequency, and inventory management that generates testable hypothesis. Section II discusses the relationships between nutrient/food perishability and health outcomes, with a discussion about the role of food processing. Specific nutrients that fulfill the three criteria above are identified for empirical consumption analysis. Section III empirically tests the hypotheses that people living in households that grocery shop frequently consume foods with nutrient compositions that cause the foods to decay more rapidly. The results indicate that people living in high grocery shopping frequency households eat more nutritiously. A final section concludes.

I. Theory of Perishable Consumption

A household's food inventory management problem lies at the heart of the household's decisions of how often to grocery shop and food choices. Food inventory management is complicated by the wide range of product options that, to varying degrees, can be substitutes, with some being behavioral complements. Some foods can be stored practically forever, while others decay rapidly over a few days. Later I will discuss nutritional differences between nonperishable foods and nonperishable foods. At this point, nutrition is ignored, as I differentiate foods only according to their decay rates and treat them as weakly separable in a utility function to explore the behavioral responses to key factors.

I.A. Model Setup

The household maximizes utility function

$$u(x, y) \tag{1}$$

where x the amount of a perishable good consumed, and y is the amount of a nonperishable food consumed. The household's per unit time budget constraint is

$$m = k(s;\theta) + qI_x^0 + I_y^0 \tag{2}$$

where I_x^0 and I_y^0 are the inventory levels of goods x and y, respectively, immediately after purchases are made. I_y^0 is the numeraire and q is the unit market price of perishable inventory I_x^0. $k(s;\theta)$ is the fixed cost of a shopping trip and is a function of the time between shopping trips, s, and a parameter θ.

Since I_y^0 does not perish, all of I_y^0 is consumed so that $y = I_y^0$. This is not the case for the perishable food. The amount of I_x^0 that is consumed is a function of s and a parameter δ. Specifically, $x = g(s;\delta)I_x^0$ where $g \in [0,1]$. Therefore (2) can be written as

$$m = k(s;\theta) + p(s;\delta,q)x + y \tag{3}$$

where $p(s;\delta) = q / g(s;\delta)$ is the cost per unit of perishable *consumption*. For example, if ¼ of the perishable food goes to waste, g = ¾ so that the consumption price is 1/3 higher than the market price.

The fixed cost function, $k(s;\theta)$, represents the burden of making a shopping trip to the store. Suppose that each time the household makes a shopping trip, they face psychological, monetary, and opportunity costs equal to θ. Over a time period T, they will face an aggregate fixed cost of θ times the number of shopping trips they make: $k(s;\theta) = \theta T / s$. Without loss of generality, measure time so that T = 1, and we have

$$k(s;\theta) = \frac{\theta}{s} \tag{4}$$

Note that $k_s = -\theta s^{-2} < 0$, $k_{ss} = 2\theta s^{-3} > 0$, and $k_{s\theta} = -s^{-2} < 0$.

The signs of the derivatives of $p(s;\delta,q)$ are motivated by a functional form that is generated from a simplified intertemporal consumption model in Colby (2015). Suppose the household obtains $V(s) = \max_{c_x(t),c_y(t)} v(c_x(t),c_y(t))$ with $v(c_x(t),c_y(t)) = \int_0^s [a\ln(c_x(t)) + \ln(c_y(t))]dt$ subject to the dynamic inventory constraints $\dot{I}_x(t) = -\delta I_x(t) - c_x(t)$ and $\dot{I}_y(t) = -c_y(t)$, with initial conditions $I_x(0) = I_x^0$ and $I_y(0) = I_y^0$. The optimal paths are $c_x(t) = I_x^0 e^{-\delta s} / s$ and $c_y(t) = I_y^0 / s$. Integrating $c_x(t)$ over $t \in (0,s]$ implies

$$p(s;\delta,q) = q\frac{\delta s}{1-e^{-\delta s}}. \tag{5}$$

Lemma 1: $p_s > 0$, $p_{ss} > 0$, $p_{s\delta} > 0$, and $p_\delta > 0$ for all $q,\delta,s > 0$.

All proofs are in the Appendix.

I.B. First Order Conditions

The household's problem is simplified by solving for y in (3) and substituting in (1) to form the unconstrained maximization problem

$$\max_{x,s} u(x, m - k(s;\theta) - p(s;\delta,q)x) \tag{6}$$

The first order conditions are

$$\frac{\partial u}{\partial x} = u_1 - pu_2 \equiv 0 \tag{7}$$

and

$$\frac{\partial u}{\partial s} = -u_2(k_s + p_s x) \equiv 0 \tag{8}$$

where u_i is the derivative of $u(\cdot)$ with respect to its ith argument. Equation (7) is the usual rule that the marginal rate of substitution equals the price ratios, with the caveat that the price of perishable food consumption incorporates the cost of food waste. Equation (8) implies that s is chosen so that the combined expenses of long-run fixed costs of shopping and food waste are minimized. Put another way, for any consumption levels, (x, y), the household chooses their shopping frequency to minimize nuisance costs.

I.C. Comparative Statics

I.C.1. Changes in Income

I have found that income is positively correlated with shopping frequency (Colby, 2016). Obsequiously, this is a paradox in light of travel cost models that assume that the (fixed) cost of travel(ling to grocery stores) is measured by wage rates. Incorporating food decay and inventory management, however, resolves this paradox.

Proposition 1. *Consumption of the perishable food increases and shopping frequency increases in income if and only if the perishable food is a normal good (in a fixed shopping frequency world).*

Intuitively, as income rises, the household consumes more perishable food, which pressures them to shop more frequently to avoid costly food waste. The comparative static results that follow depend crucially on perishable food being a normal good, an assumption supported by demand analyses that include fresh produce (e.g. Cox & Wohlgenant, 1986).

I.C.2. Changes in Fixed Cost Parameters

An economic interpretation of a household having "limited access" to healthful food retailers is the household faces high fixed costs per associated with a trip to a grocery store. For example, travel costs, including the shopper's value of time, can be high enough to discourage shopping trips (as anyone knows who has delayed going to the store because of the burden). Surprisingly, higher fixed costs do not necessarily imply that the household will shop less frequently. This is because if the perishable food is *not* a normal good, then the decreased income from higher fixed costs shifts consumption towards *more* perishable food. With more perishable food in inventory, the household is impelled to avoid costly food waste by shopping *more* frequently. If that force outweighs the benefits of avoiding less fixed costs from shopping less, the household will find itself in the perverse position—akin to Giffen goods—of making more shopping trips when the cost of making a shopping trip has gone up. However, if the perishable food is a normal good, then households facing high fixed costs will shop more frequently. Given that the most important highly perishable foods, fresh fruits and vegetables, are normal goods (Cox & Wohlgenant, 1986), this is almost certainly the case.

Proposition 2. *If the perishable food is a normal good, then an increase in the per-trip fixed cost of shopping, θ, will decrease the amount of the perishable food consumed and decrease the household's shopping frequency.*

Proposition 2 does not necessarily imply that an increase in the cost of making a grocery trip will decrease the amount of perishable *inventory* purchased. Rather, it is a statement about *ingested* quantities. It is possible for the household to increase the amount of the perishable food that they purchase, but consume a lesser amount due to a disproportionate amount going to waste. Indeed…

Corollary 3. *If the perishable food is a normal good, then an increase in the fixed costs of a shopping trip will increase the proportion of perishable food that is wasted.*

Note that Corollary 3 does not necessarily imply that the total amount of food wasted increases when the fixed cost increases. This is because the household adjusts by purchasing less perishable foods.

Some households may find high levels of food waste unacceptable, and cap the proportion of food that they discard. When this is the case, the countervailing force discussed above—wherein the perishable food is not a normal good, and the household avoids increasingly costly waste as their perishable food inventories rise in response to a decrease in income from higher fixed costs—no longer applies, and we can dispense with assumption that perishable food is a normal good.

Corollary 4. *If the proportion of the perishable inventory that is wasted is independent of shopping frequency, then an increase in the fixed cost of a shopping trip will decrease shopping frequency.*

Corollary 4 implies that if the decay rate of the perishable food, δ, is close to zero, then an increase in the fixed cost of a shopping trip will always decrease shopping frequency whether or not the perishable food is a normal good.

II. Food Decay, Health & Nutrition, and Processed Food

II.A. What is "Processed Food"?

Popular use of the term "processed food" is pejorative, and connotes nutritional inferiority. Yet food processing adds value to raw agricultural commodities by improving taste and texture, reducing post-purchase preparation time, creating more convenient forms, and extending shelf-lives. None of those value-adding processes deliberately make foods less nutritious. Some processing—such as fortification, artificial sweetening, fat removal—are deliberate attempts to *improve* nutrition (Smith, 1999). Thus if "food processing" makes food less nutritious, it is an unfortunate accident. After all, consumers prefer nutritious foods, *ceteris paribus*.

I now discuss how many of the chemical reactions that make foods decay can be slowed or eliminated by food processes that incidentally make foods less nutritious. Because of this, consumers that shop less frequently will consume less nutritious food as they opt for (i) within-category options that have been altered for longer shelf-lives, or (ii) a different food category with longer shelf-lives.

Generally, as households opt for more chemically stable foods to sustain them over long shopping cycles, their diets will have more sodium, more saturated fat, less non-saturated fat, less ω-3 fatty acids, and higher energy density. Diets altered in this way are obesogenic, and lead to greater risk of heart disease, cancer, hypertension, diabetes, and other diseases.

II.B. Food Decay, Nutrients, and Health

II.B.1. Energy Density and Water Activity

Energy density is related to perishability through the food chemistry property 'water activity'[1]. Reducing water activity is a basic and ubiquitous way that food manufacturers extend the shelf-lives

[1] The water activity of a food is the ratio of the vapor pressure of the water in the food divided by the vapor pressure of water only. Essentially water activity can be thought of as a measure of the rate of water being evaporated in a food. The more water and the more freely the water is able to migrate to the surface, the higher the food's water activity.

of foods. Reduced water activity limits microorganism growth and chemical reaction rates (deMan, 1999), such as the breakdown of lipids which creates off-putting rancid flavors (Troller, 1978). In this way, *processes* that reduce water activity add value to food commodities, allowing consumers to shop less, pay less, and store a greater variety of foods.

The greater caloric energy density resulting from the removal of water from edible substances is likely one reason "processed food" has attracted a negative connotation by consumers and food consumer advocates. For example, Michael Pollan recommends avoiding shopping in the interior of grocery stores where dry, shelf-stable packaged foods are found that do not require refrigeration (Pollan, 2008). The negative health consequences of energy dense foods are most severe with low income households as a result of a negative correlation between energy density and energy costs (Drewnowski & Specter, 2004). At least part of that association is driven by the lower per calorie shipping and inventory costs of energy dense, shelf-stable foods. *Consumption* prices of these shelf-stable foods is further reduced beyond differences in market prices, as less goes from pantry to trash can.

Consumption of high energy density foods does not necessarily imply that people will consume more calories. *A priori* it is reasonable to hypothesize that the human body maintains a relatively constant long-term food energy equilibrium. Under this hypothesis, the body measures the amount of bioavailable calories from ingested food, compares that amount to metabolic requirements and energy reserves, and modulates the amount of food consumed through signals of satiation or hunger. However, this hypothesis has been refuted by experiments that adjust food characteristics (such as energy density), and environmental cues (such as plate sizes).

There is strong evidence that the volume of consumed food—not food energy—is the quantity that the body uses to determine satiation in all but toddlers and infants (Ello-Martin et al., 2005). For example, Rolls et al. (1998) adjusted the volume of milk drank between breakfast and lunch by experimental subjects while keeping nutrient quantities (e.g. calories) fixed, and found that those that drank the less energy density milk consumed less volume and energy in subsequent lunches and dinners. Wansink, Painter & North (2005) rigged soup bowls so that they would stay at a constant level to see if that would induce greater consumption. Those with rigged bowls ate 73% more soup (and calories), despite not believing they had consumed more, nor feeling more satiated, than the control group who ate out of normal bowls. This suggests satiation is dependent on visual volumetric cues.

These experiments and many other studies show that satiation is based on perceived volume in the *short-term* (Poppitt & Prentice, 1996). But this does not rule out *long-term* energy consumption adjustments. Do consumers ingest fewer calories later that day or in subsequent days following elevated caloric consumption? Perhaps it is the case that those "tricked" into consuming excess calories—such as bottomless soup bowl diners—experience delayed hunger cues, impelling them to consume fewer calories later: "I had a big lunch." This turns out not to be the case. An experiment by Rolls, Roe, and Meengs (2007) resoundingly refuted that hypothesis by controlling all the food options of experimental participants over two 11 day periods. The treatment group were

given 50% larger portions during a baseline period. All subjects were free to choose how much food to eat. The large 423 kcal increase in consumption resulting from larger portion sizes was constant over the 11 days. The portion size effect was *positively* correlated with energy *density*, indicating that energy dense foods are more readily overconsumed. That is to say, with humans tending to keep food consumption *volume* constant, consumption of higher density foods results in greater energy consumption. Taken together, these findings suggest that foods with low water activity (e.g. from low food water concentrations) will have longer shelf-lives, be energy dense, and result in elevated energy consumption.

Consumption of a high energy dense diet is associated with elevated fasting insulin[2], obesity, and the metabolic syndrome[3] (Mendoza et al., 2004). The metabolic syndrome is associated with alarmingly elevated risks of CVD (*Relative Risk* (*RR*) = 2.35), CVD mortality (*RR*= 2.40), myocardial infarction (*RR* = 1.99), stroke (*RR* = 2.27), and all-cause mortality (*RR* =1.58) (Mottillo et al., 2010). These studies are careful to control for possible confounding nutrients that are associated with energy density, such as fat.

Table 1 Nutrients, Shelf-Lives and Diet-Based Diseases

Nutrient	**Nutrient Change to Extend Shelf-Life During Processing**	**Disease/Condition**	**Attributable U.S. Deaths (per year)[b]**
Energy Density/Water[a]	-	Obesity, Metabolic Syndrome, Insulin Resistance, CVD, Myocardial Infarction, Stroke,	
Omega-3 Fats	-	Liver Disease, Inflammation, CVD, Diabetes, Hypertension, Cancer, Mental Disorders	84,000
Saturated (Polyunsaturated) Fats	+ (-)	Cancer, CVD, CHD	15,000
Sodium	+	CVD, Stroke, Hypertension, Diabetes	70,000-120,000

Notes: [a]Water is removed from food to reduce the chemical property "water activity." Doing so makes foods more energy dense, which results in increase energy consumption. Entries for "Shelf-Lives" and "Diseases/Conditions" are supported by food chemistry and medical literatures, respectively. [b]Figures taken from Danaei et al. (2009) and Coxson et al. (2013).

[2] A key predictor for diabetes. An additional way that water activity is commonly reduced is by adding solutes such as salts to meats and sugars to fruits (Rahman & Labuza, 2007). Sugars reduce water activity in fruit spreads by binding the water.

[3] Defined as a state in which a person has at least three of the following: (i) abdominal obesity, (ii) elevated triglyceride levels, (iii) low HDL levels, (iv) high blood pressure, and (v) high blood sugar (NCEP, 2001).

II.B.2. Omega-3 Fatty Acids and Rancidity

Food manufacturers find the presence of rapidly decaying omega-3 (ω-3) fatty acids problematic beacuse they produce off-putting rancid flavors and odors (Kolanowski & Berger, 1999; Allport, 2006). According to lipid expert Gary List, "Any product with significant amounts of ALA [an ω-3] has a stability problem, and are likely to develop rancid and off flavors...making it a food manufacturer's nightmare" (Allport, 2006). For example, ω-3 oxidation is what gives fish its fishy smell. Beef with higher concentrations of ω-3—e.g. from grass fed cattle—develop rancid flavors and changes color quicker (Vatansever et al., 2000). Fortifying foods with ω-3s is unrealistic except for perhaps foods that already have very short shelf-lives (Kolanowsk & Laufenberg, 2006). Food manufacturers commonly remove ω-3s from foods to extend their shelf-lives.

Humans evolved on a diet consisting of an ω-6 to ω-3 ratio of 1:1. U.S. diets have a ratio of 16:1 (Simopoulos, 2006). Greater ω-3 fatty acid consumption—especially eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA)—is recommended by the American Heart Association, FAO/WHO, USDA, the U.S. National Academy of Sciences, and many other health organizations throughout the world (Mozaffarian & Wu, 2011). EPA and DHA are primarily consumed in seafood.

Recent meta analyses conclude that the consumption of ω-3 polyunsaturated fatty acids (PUFAs) (1) reduce the risk non-alcoholic fatty liver disease (a disease associated with obesity, type-2 diabetes, dyslipidemia, and hypertension) (Parker et al., 2012); (2) reduce joint inflammation (Goldberg & Katz, 2007); (3) reduce symptoms of childhood attention-deficit/hyperactivity disorder (ADHD) through known physiological mechanisms (Bloch & Qawasmi, 2011); (4) reduce depressive symptoms of those with bipolar and major depressive disorder (MDD)[4] (Sarris et al., 2012; Grosso et al., 2014); (5) a modestly reduce the risk of diabetes mellitus (Wu et al., 2012); (6) reduced blood pressure (Miller et al., 2014); (7) improve cognitive abilities for those with some forms of cognitive impairment[5] (Mazereeuw et al., 2012); (8) reduce the risk of cardiovascular (CV) events in people with CV disease (Casula et al., 2012), and reduce the risk of heart failure (Djoussé et al., 2012) and CV disease (Delgado-Lista et al., 2012) in healthy populations when from marine sources[6]; (9) reduced risk of breast cancer (Zheng et al., 2013); (10) reduced prostate cancer (Chua et al., 2013); and (11) reduced post-surgical infection rates (Pradelli et al., 2012). The important role that ω-3s play in (12) neurological development and (13) visual acuity (Boucher et al., 2011) combined with the bodies inability to endogenously produce them has led to be added to baby formula.[7] It is estimated that low relative levels of dietary ω -3 consumption is responsibility for 84,000 deaths/yr. in the U.S. (Danaei et al., 2009).

[4] But not depression in general populations (Bloch & Hannestad, 2012; Grosso et al., 2014).
[5] With no cognitive benefit for healthy adults or those with dementia (Mazereeuw et al., 2012).
[6] Recent meta-analyses do not find a statistically significant reduction with ω-3 supplements (Casula et al., 2012; Rizos et al., 2012).
[7] There are reports of other benefits of ω-3 consumption in medical journals, but we restrict our list to only benefits that have been replicated and have meta

II.B.3. Saturated Fatty Acids Are Most Stable

The saturation of a fatty acid refers to the proportion of hydrogen bonds along the fatty acid's carbon-chain compared to the maximum possible number. Saturated fats have hydrogens in all potential locations. A fat is unsaturated when double bonds use up possible hydrogen bonding sites along the carbon-chain. Hydrogen bonds are highly stable; the double bonds of unsaturated fats are not. Off-flavors, odors, and free radicals are created when lipid oxidation occurs around the site of a double bond. Generally speaking, the less saturated the fatty acid, the more opportunities for lipid oxidation, and the faster the food decays, becoming less palatable. For example, the relative rates of spoilage by oxidation of 18-carbon fatty acids with 1, 2, and 3 double bonds compared to the completely saturated 18-carbon fatty acids is 100:1,1200:1, 2500:1 (DeMan, 1999). Saturating the double bonds of PUFAS extends shelf-life, which is why food manufacturers prefer saturate fats. Unfortunately, replacing PUFAS with saturated fat eliminates health benefits (Kolanowski & Laufenberg, 2006).

Randomized controlled trials find that replacing dietary PUFA consumption with more chemically stable SFAs—such as monosaturated and ω-3 fatty acids—increases the risk of CHD (Mozaffarian et al., 2010).[8] Similar finding apply to cancer. It is estimated that the high dietary levels of SFAs in place of substitutable but perishable PUFAs causes 15,000 deaths/yr. in the U.S. (Danaei et al., 2009).

II.B.4. Sodium, the Number One Preservative

Nearly all dietary sodium in food is from processing (Mattes & Donnelly, 1991). The extent to which foods are processed is positively correlated with sodium concentrations (Poti et al., 2015). Sodium is added for its salty flavor[9], to enhance other flavors, and to increase shelf lives by lowering water activity (Troller, 1978 pp 72-3). Sodium decreases the chemical property of water activity even in high moisture foods such as with pickled products (Troller, 1978 pp 112). Salt is an inexpensive ancient preservative that probably will be widely used forever.

There is currently great effort being expended by medical and policy institutions to create and launch policies for the reduction of dietary sodium intake. The Institute of Medicine has recommended strong regulatory action, going so far as to suggest that the Food and Drug Administration (FDA) consider revoking salt's generally recognized as safe (GRAS) status, thereby making salt an additive that can be closely regulated and limited by the FDA (Boon et al., 2010). The Food and Drug Administration (2016) recently published a draft outlining ambitious voluntary industry guidelines to be used by food manufactures to reduce "mean and upper bound concentrations for sodium in commercially processed, packaged, and prepared foods."

[8] Contrary to popular belief, however, the consumption of SFAs is not directly associated with CHD or CVD (Siri-Tarino et al., 2010).
[9] Saltiness is one of the five primary tastes, meaning we have a type of gustatory receptor specific to our sense of salt.

Eighty-nine percent of Americans consume more than the 2,300 mg/d sodium upper limit recommended by the *2015–2020 Dietary Guidelines for Americans*, with an average of 3,400 mg/d (Jackson et al., 2016). Consumers that already have high sodium intakes experience increased risks of mortality when they consume more sodium[10] (Graudal et al., 2014). Higher sodium consumption is associated with higher blood pressure[11] (Aburto et al., 2013) which is a key mediating factor for stroke (relative risks (*RR*) = 1.23) and cardiovascular disease (*RR* = 1.14) (Strazzullo et al., 2009). Simulations predict that reducing U.S. sodium consumption down to recommended levels would prevent 700,000 to 1,200,000 deaths over ten years: 3% to 5% of all deaths over the *first* decade (Coxson et al., 2013).

II.B.5. Nutrients Related to Decay, Not Considered

A number of other food chemicals are related to both perishability and health outcomes. For example, processed meat consumption is associated with CHD and diabetes incidence (Micha et al., 2010) and colorectal cancer (Chan et al., 2011). In the interest of investigating only the consumption of nutrients that satisfy the three criteria delineated in the introduction, these and other associations are ignored in the empirical analysis.

Many important nutrients decay in storage. This is the case even for canned, packaged, and to a lesser extent, frozen food products. For example, the iron content of wheat flour decreases over storage time (Marathe et al., 2002); antioxidant concentration decreases over time as they neutralize free radicals produced by deterioration; the bioavailability of B vitamins, vitamin C, vitamin E, carotenoids, calcium, and potassium all decay rapidly in foods (especially fresh produce) (Rickman et al., 2007a & 2007b). Therefore, households that store food over longer periods will experience an inferior diet *even if they consume the same food.* These nutrients are not included in the analysis, however, because while they decay in the chemical sense, their decay is not observable in the data and not associated with the deterioration of the gustatory quality of the food. Thus they are unlikely to be associated with shopping frequency.

III. Data and Analysis

III.A. Data

Data comes from Continuing Survey of Food Intakes by Individuals (CSFII) 24-hour diet recall surveys, years 1994, 1995, 1996 and a supplemental children's survey in 1998. CSFII surveys were designed and conducted by the USDA. In 2002, the CSFII and National Health and Nutrition

[10] The opposite is true for those with low levels of sodium, a concern neither here nor there.

[11] African Americans are particularly prone to hypertension from salt intake. This is not the case for non-American Africans. A leading theory explaining the discrepancy is that those genetically predisposed to retain high levels of sodium were more likely to survive dehydration from ailments on slave ships. However, the evidence supporting this hypothesis has been questioned.

Examination Survey (NHANES) combined. Respondents are surveyed a second time at a later date for a second 24-hour recall.

CSFII is uniquely suited for the research question because (i) respondents report all the food they consume (not just food at home), (ii) nutrient quantities are available, (iii) the panel is sufficiently large to obtain reliable estimates, (iv) the sample is representative of the U.S., (v) extensive demographic and (vi) geographic control variables are provided, and (vii) shopping frequency is reported.

Survey participants report the foods that they eat. Nutrient quantities are derived using the Survey Nutrient Database, which contains the nutrient quantities for recipes. Salt is not included in recipes where it is considered optional, but is added in if in a follow up question the participant indicates that they added it either in meal preparation or at the table. Given the timeframe of the survey, the gradual recognition of the importance of omega fatty acids, and the present prevalence of missing values in the current USDA National Nutrient Database for Standard Reference (SR-28), reported EPA and DHA are likely significantly understated.

The crucial variable of shopping frequency is categorical. Interviewers asked:

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Let's begin by talking about the general food shopping practice of
this household. On the average, how often does someone do a major
shopping for this household? Would you say...
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- More than once a week
- Once a week
- Once every two weeks
- Once a month or less
- Never
- Don't know
- Not ascertained

III.B. Nutrient Consumption Models

I model the consumption of a given nutrient as dependent on shopping frequency conditional on demographic and income data. Assuming perishable foods are normal goods, Proposition 2 hypothesizes that nutrients that cause foods to decay faster will be consumed in higher quantities by people who live in households that shop more frequently, *ceteris paribus*. Thus EPA and DHA will be consumed more by people who live in households that shop more frequently. Conversely, nutrients that are associated with longer shelf-lives are hypothesized to be consumed in higher quantities by people who live in households that shop less frequently. Therefore sodium and saturated fat will be consumed in higher quantities by people who live in households that shop infrequently. Insofar as water activity is associated with food water concentrations in turn lower

energy density—which is associated with higher energy consumption—I hypothesize that people in households that shop more frequently will consume fewer calories and more food water.

A considerable number of observations contain zero values. This is especially the case for fatty acids. Tobit models are used to avoid bias from failure to account for censoring at zero units. Let

$$y_i = \begin{cases} y_i^* = \beta X + u_i & \textbf{if } y_i^* = \beta X + u_i > 0 \\ 0 & \textbf{if } y_i^* = \beta X + u_i \le 0 \end{cases} \tag{9}$$

with $u_i \sim N(0, \sigma_i^2)$, for nutrient *i*, where *y* is the quantity consumed and *X* are explanatory variables including shopping frequency. With nutrients being consumed through foods with fixed nutrient profiles, nutrient quantities are surely correlated. Efficiency is gained by accounting for cross-equation error correlation. Unfortunately—but not surprisingly given the 64 censoring possibilities—estimation of the 6 equation multivariate likelihood function with cross-equation error correlation fails to converge (see the Appendix for a fuller discussion). Instead, I collect equation-by-equation errors and run a standard second stage seemingly unrelated regression estimation.

III.C. Estimated Relationships between Shopping Frequency and Nutrient Consumption

Estimates provide strong evidence in favor of all hypotheses (Table 2). People in households that shop frequently consume less salt, saturated fat, and fewer calories; and more EPA, DHA, and food water. For example, compared to a person in a household that shops once every 2 weeks, a person living in a household that shops at least once per week will consume 71 fewer calories, 144 mg less sodium, 1.14 g less saturated fat, 0.0057 g less EPA[12], 0.0056 g less DHA, and 5 ml less food water per day (Figure 1). All of these suggest that shopping more frequently is associated with a more nutritious diet, and reduced risk of the diseases and conditions listed in Table 1. The lower calorie and greater food water consumption of higher shopping frequency households suggest shopping frequency is associated with the consumption of lower energy dense foods, and therefore lower rates of obesity.

While any one of the monotonic relationships between shopping frequency and nutrient consumption exhibited in Figure 1 would provide only suggestive—and perhaps spurious—evidence for the hypothesis that shopping frequency is correlated with greater consumption of nutrients that rapidly decay, taking all six together provides very strong evidence. It is worthwhile to assure the reader that all nutrients that adequately satisfy the three criteria in the introduction were included in the analysis. That is to say, nutrients were not cherry picked to support the hypothesis, and the reported estimates do not suffer from post estimation bias.

[12] EPA and DHA estimates are likely understated due to lack of data of concentrations in foods at the time the surveys were conducted.

Table 2: Tobit Models of Nutrients Consumed

	Dependent Variable					
	Calories	Sodium	Saturated Fat	EPA	DHA	Food Water[a]
Shop > 1/wk	-72	-117	-1.35	0.011	0.012	9.23
	(12.3)	(23.9)	(0.21)	(0.0020)	(0.0027)	(5.25)
Shop 1/wk	-26.9	-30.6	-0.53	0.0049	0.0063	9.29
	(10.3)	(20)	(0.17)	(0.0017)	(0.0022)	(4.4)
Shop 1-per-2 wks	-0.88	26.7	-0.21	0.0053	0.0064	4.26
	(10.8)	(21)	(0.18)	(0.0018)	(0.0023)	(4.62)
Food Stamps	-21.3	78.2	-0.76	0.0052	0.0056	5.58
	(11.4)	(22.1)	(0.19)	(0.0019)	(0.0025)	(4.88)
1994	351	699	4.25	0.0072	0.017	36.1
	(10)	(19.4)	(0.17)	(0.0017)	(0.0022)	(4.3)
1995	317	619	3.71	0.010	0.021	38.1
	(10.4)	(20.2)	(0.17)	(0.0018)	(0.0023)	(4.46)
1996	399	741	4.57	0.0092	0.021	40.7
	(10.2)	(19.9)	(0.17)	(0.0018)	(0.0022)	(4.39)
Age	2.38	9.61	-0.010	0.00092	0.0012	3.32
	(0.15)	(0.3)	(0.0026)	(0.000026)	(0.000033)	(0.065)
Male	441	737	6.15	0.0084	0.014	80.5
	(6.5)	(12.6)	(0.11)	(0.0011)	(0.0014)	(2.78)
Income as % of Poverty Line	0.4	0.46	0.00047	0.000015	0.000031	0.19
	(0.042)	(0.081)	(0.0007)	(0.0000071)	(0.000009)	(0.018)
Northeast	-19.6	-10.4	0.095	0.0028	0.0069	-4.61
	(10.5)	(20.4)	(0.18)	(0.0018)	(0.0023)	(4.49)
Midwest	98.4	251	1.61	-0.009	-0.0048	2.17
	(9.89)	(19.2)	(0.17)	(0.0017)	(0.0021)	(4.23)
South	-57.2	5.9	-0.45	-0.00045	-0.00062	-25
	(9.13)	(17.8)	(0.15)	(0.0016)	(0.002)	(3.92)
MSA, central city	11.3	-22.3	-1.15	0.013	0.012	23.7
	(9.43)	(18.3)	(0.16)	(0.0016)	(0.002)	(4.03)
MSA, outside central city	-0.99	-17.4	-0.87	0.0082	0.0063	13.1
	(8.53)	(16.6)	(0.14)	(0.0015)	(0.0018)	(3.65)
White	-23	-35.2	0.16	-0.016	-0.019	-42.8
	(13)	(25.3)	(0.22)	(0.0022)	(0.0028)	(5.59)
Black	-42.5	-28.4	0.16	0.0082	0.012	-35.6
	(15.4)	(30)	(0.26)	(0.0026)	(0.0033)	(6.63)
Asian	-171	-114	-4.3	0.047	0.056	65
	(23)	(44.7)	(0.39)	(0.0038)	(0.0049)	(9.95)
Native American	2.24	109	-0.82	-0.00047	-0.0092	14
	(37.4)	(72.7)	(0.63)	(0.0064)	(0.0082)	(15.9)
Intercept	1182	1532	19.1	-0.075	-0.056	272
	(23.3)	(35.3)	(0.39)	(0.004)	(0.0051)	(7.8)
Sigma	804	1563	13.6	0.12	0.17	279
	(2.3)	(4.48)	(0.039)	(0.00048)	(0.00056)	0.98
Censored Observations	400	404	420	35518	16095	15
Total Observations	61546	61546	61546	61546	61546	40371

Note: Standard errors in parentheses. Fats are measure in grams, sodium in milligrams, and food water in milliliters. Omitted Variables: shopping frequency once per month or less, not on food stamps, 1998, female, west, Non-MSA"other." [a]Does not include beverages.

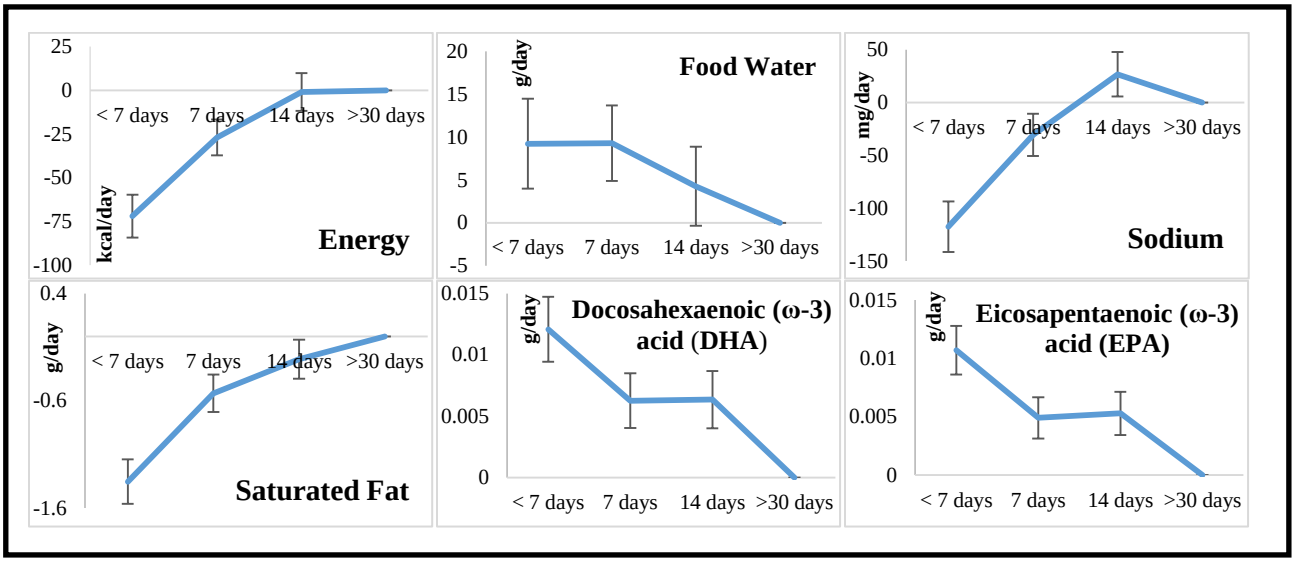

Figure 1. Level Effects of Shopping Frequency on Nutrient Intake

Note: Bars represent standard error.

IV. Concluding Remarks

Diet-based disease is one of society's most pressing public policy concerns. For the first time in U.S. history, the life expectancy of newly born Americans has decreased (Xu et al., 2016). It is widely believed that a large proportion of U.S. deaths are preventable through dietary or behavioral interventions (Danaei et al., 2009). That has inspired policymakers and researchers to look for a smoking gun signaling a cause of diet-based mobidity that can be remedied through policy prescriptions. "Food deserts" were initially believed to be just that smoking gun (Bitler & Haider, 2011). However, that is now in doubt with recent studies concluding that market interventions in the build environment—such as the introduction of supermarkets in locations where store densities are low and health is poor—are ineffective at impelling people to eat healthier (Cummins et al., 2014).

This article provides a more rigorous theoretical foundation to the intuition of "food access" and food choice. In so doing, focus is shifted away from the built environment, squarely onto the relevant household behavior for which the built food retail environment has served as proxy: shopping frequency. The conclusion is that, under typical conditions, low "access" will cause a household to buy less perishable food only if it also gets them to shop more frequently. The reason is because more food goes to costly waste if the household shops infrequently. The nutrition of perishability is considered, and it is concluded that key nutrients are closely related to both health and perishability, with highly perishable nutrient profiles being more healthful. The consumption of these healthful but perishable nutrient profiles are shown to be systematically related to shopping frequency, as the theory suggests.

The framework and evidence in this article recasts the question of food access in a new way, that may be fruitful for the creation of new policy prescriptions that prevent diet-based disease.

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Appendix

Proof of Lemma 1

The derivative of $p(s;\delta,q) = q\dfrac{\delta s}{1-e^{-\delta s}}$ with respect to *s* is

$$p_s = q\delta\frac{1-(1+\delta s)e^{-\delta s}}{(1-e^{-\delta s})^2} > 0 \tag{10}$$

Since $e^{\delta s} = \sum_{n=0}^{\infty}(\delta s)^n / n! > 1+\delta s$, the numerator is positive $\forall(\delta, s) > (0,0)$, and the sign in (10) follows. Noting that the parameter δ and *s* enter (5) symmetrically, we have

$$p_\delta = qs\frac{1-(1+\delta s)e^{-\delta s}}{(1-e^{-\delta s})^2} > 0 \tag{11}$$

by the same argument.

Taking a second derivative with respect to *s* and gives

$$\begin{aligned} p_{ss} &= q\delta\frac{(1-e^{-\delta s})\delta^2 s e^{-\delta s} - 2\delta e^{-\delta s}(1-e^{-\delta s})(1-e^{-\delta s}-\delta s e^{-\delta s})}{(1-e^{-\delta s})^4} \\ &= \frac{q\delta^2 e^{-\delta s}}{(1-e^{-\delta s})^3}\left[\delta s + \delta s e^{-\delta s} + 2e^{-\delta s} - 2\right], \end{aligned} \tag{12}$$

which implies $p_{ss} > 0$ if and only if the quantity in the square bracket is greater than zero. To show that this is indeed the case, multiply the square bracket by $e^{\delta s}$ and let $w = \delta s$. It will be shown that the result, $we^w + w + 2 - 2e^w > 0$, $\forall w > 0$. Expanding the first and last term gives

$$\begin{aligned} we^w + w + 2 - 2e^w &= \left(w + w^2 + \frac{w^3}{2!} + \frac{w^4}{3!} + \cdots\right) + w + 2 - \left(2 + 2w + \frac{2w^2}{2!} + \frac{2w^3}{3!} + \frac{2w^4}{4!} + \cdots\right) \\ &= \left(\frac{w^3}{2!} + \frac{w^4}{3!} + \cdots\right) - \left(\frac{2w^3}{3!} + \frac{2w^4}{4!} + \cdots\right) \\ &= \left(\frac{3w^3}{3!} + \frac{4w^4}{4!} + \cdots\right) - \left(\frac{2w^3}{3!} + \frac{2w^4}{4!} + \cdots\right) \\ &= \left(\frac{(3-2)w^3}{3!} + \frac{(4-2)w^4}{4!} + \frac{(5-2)w^5}{5!} + \cdots\right) > 0, \end{aligned} \tag{13}$$

since all the terms in the last line of (13) are positive. Thus $p_{ss} > 0 \ \ \forall w > 0$.

The sign of the cross derivative $p_{s\delta}$ will now be derived in a similar manner. Taking the derivative of (10) with respect to δ gives

$$p_{s\delta} = \frac{q}{(1-e^{-\delta s})^3}\left[1-2e^{-\delta s}+e^{-2\delta s}+\delta^2 s^2 e^{-\delta s}+\delta^2 s^2 e^{-2\delta s}-2\delta s e^{-\delta s}+2\delta s e^{-2\delta s}\right] \tag{14}$$

The sign of $p_{s\delta}$ inherits the sign of the quantity in the square brackets. Once again let $w=\delta s$, and distribute e^{2w} through the square bracket in (14) so that the square bracket is transformed to

$$e^{2w}-2e^{w}+1+w^2e^{w}+w^2-2we^{w}+2w \tag{15}$$

Now expand (15) to get

$$\begin{aligned}&\left(1+2w+\frac{2^2w^2}{2!}+\frac{2^3w^3}{3!}+\cdots\right)-2\left(1+w+\frac{w^2}{2!}+\cdots\right)+1+\left(w^2+w^3+\frac{w^4}{2!}+\frac{w^5}{3!}+\cdots\right)\\&+w^2-\left(2w+2w^2+\frac{2w^3}{2!}+\frac{2w^4}{3!}+\cdots\right)+2w\end{aligned} \tag{16}$$

Making cancelations and multiplying fractions by unity so that terms with a given polynomial degree have the same denominators gives

$$\begin{aligned}&\left(\frac{2^2w^2}{2!}+\frac{2^3w^3}{3!}+\cdots\right)-\left(\frac{2w^2}{2!}+\frac{2w^3}{3!}+\cdots\right)+\left(\frac{3\cdot 2w^3}{3!}+\frac{4\cdot 3w^4}{4!}+\cdots\right)-\left(\frac{3\cdot 2w^3}{3!}+\frac{4\cdot 2w^4}{4!}\right)\\&=w^2+\sum_{n=3}^{\infty}\frac{2^n+n(n-1)-2n-2}{n!}w^n\end{aligned} \tag{17}$$

The first term in the sum (n = 3) has coefficient equal to $1>0$. 2^n is an increasing function and $n(n-1)-2n-2$ is a u-shaped parabola with minimum at 1.5 and is increasing thereafter. It follows that for all n > 3, the coefficients in the sum in (17) are positive so that all terms are positive. Therefore (17) is positive $\forall w>0$ and $p_{s\delta}>0$. ■

Standard Model with Fixed *s*

The only first order condition is $u_1-pu_2=0$, and the assumed second order condition is $u_{11}-2pu_{12}+p^2u_{22}<0$. Perishable demand changes with income according to

$$x_m=-\frac{u_{12}-pu_{22}}{(u_{11}-2pu_{12}+p^2u_{22})}, \tag{18}$$

Thus the perishable food is consumed more after an increase in income when shopping frequency is fixed if and only if $u_{12} - pu_{22} > 0$. When prices change,

$$\begin{aligned} &u_{11}x_p - u_{12}(x + px_p) - u_2 - pu_{12}x_p + pu_{22}(x + px_p) = 0 \\ &(u_{11} - 2pu_{12} + p^2u_{22})x_p - u_2 - x(u_{12} - pu_{22}) = 0 \\ &x_p = \frac{u_2 + x(u_{12} - pu_{22})}{(u_{11} - 2pu_{12} + p^2u_{22})} = \frac{u_2}{(u_{11} - 2pu_{12} + p^2u_{22})} - xx_m < 0 \end{aligned} \tag{19}$$

if $x_m > 0$, as expected since the second term is the income effect and the first term, the substitution effect is always negative.

Proof of Proposition 1

Applying standard comparative static methods gives

$$\frac{\partial x}{\partial m} = \frac{(u_{12} - u_{22}p)u_2(k_{ss} + p_{ss}x)}{|H|} \tag{20}$$

and

$$\frac{\partial s}{\partial m} = -\frac{u_{12} - u_{22}p}{|H|} \tag{21}$$

where $|H|$, the determinant of the Hessian is assumed positive to ensure that the first order conditions characterize a local utility maximum. The numerator $u_{12} - u_{22}p > 0$ is necessary and sufficient for *x* to be a normal good in the analogous model that does not include *s* a choice variable (see Appendix). From (20) and (21) we see that if *x* is a normal good in the standard consumption model, then *x* is normal here as well. ■

Proof of Proposition 2

Taking the full derivatives of the first order conditions (7) and (8) gives

$$\frac{\partial^2 u}{\partial x \partial \theta} = u_{11}x_\theta - u_{12}(k_\theta + k_s s_\theta + p_s x s_\theta + p x_\theta) - u_2 p_s s_\theta - u_{21} p x_\theta - u_{22} p(k_\theta + k_s s_\theta + p_s s_\theta x + p x_\theta) = 0 \tag{22}$$

and

$$\frac{\partial^2 u}{\partial s \partial \theta} = -u_2(k_{s\theta} + k_{ss}s_\theta + p_{ss}xs_\theta + p_s x_\theta) - u_{21}(k_s + p_s x)x_\theta + u_{22}(k_s + p_s x)(k_\theta + k_s s_\theta + p_s x s_\theta + p x_\theta) = 0 \tag{23}$$

Using the identity $(k_s + p_s x) = 0$ implied by (8), equations (22) and (23) can be written in matrix form as

$$\begin{bmatrix} u_{11} - 2u_{12}p + u_{22}p^2 & -u_2 p_s \\ -u_2 p_s & -u_2(k_{ss} + p_{ss}x) \end{bmatrix} \begin{bmatrix} \frac{\partial x}{\partial \theta} \\ \frac{\partial s}{\partial \theta} \end{bmatrix} = \begin{bmatrix} (u_{12} - u_{22}p)k_\theta \\ u_2 k_{s\theta} - u_{22}(k_s + p_s x) \end{bmatrix} \tag{24}$$

Applying Cramer's rule gives

$$\begin{aligned} \frac{\partial x}{\partial \theta} &= \frac{-(u_{12} - u_{22}p)k_\theta u_2(k_{ss} + p_{ss}x) + u_2[p_s k_{s\theta} u_2 - u_{22}(k_s + p_s x)]}{|H|} \\ &= \left(\frac{\partial x}{\partial m}\right)(-k_\theta) + \frac{u_2 p_s[k_{s\theta}u_2 - u_{22}(k_s + p_s x)]}{|H|} \\ &= -\left(\frac{\partial x}{\partial m}\right)\Big/ s + \frac{u_2 p_s[k_{s\theta}u_2 - u_{22}(k_s + p_s x)]}{|H|} \\ &= -\left(\frac{\partial x}{\partial m}\right) \times (\textbf{\# of trips}) + \frac{u_2 p_s[k_{s\theta}u_2 - u_{22}(k_s + p_s x)]}{|H|} \end{aligned} \tag{25}$$

and

$$\frac{\partial s}{\partial \theta} = \frac{(u_{11} - 2u_{12}p + u_{22}p^2)[p_s k_{s\theta} u_2 - u_{22}(k_s + p_s x)] + (u_{12} - u_{22}p)k_\theta p_s u_2}{|H|} \tag{26}$$

which is are both positive if $u_{12} - u_{22}p > 0$, the condition for *x* to be normal. ■

Proof of Corollary 3

The proportion of inventory consumed is $q / p(s)$ so that the proportion of inventory that is wasted is $L = 1 - q / p(s)$. Using (5) and taking the derivative of *L* with respect to *s* gives

$$\frac{\partial L}{\partial s} = \frac{1 - e^{-\delta s}(1 + \delta s)}{\delta s^2} > 0, \quad \forall \delta > 0, \forall s > 0. \tag{27}$$

Thus if *s* increases then the proportion of perishable food wasted, *L*, increases. ■

Proof of Corollary 4

Set $p_s = 0$ in (26). ■

Effects of Changes in Price and Decay Parameters

Applying standard comparative static techniques gives

$$\begin{bmatrix} u_{11}-2u_{12}p+u_{22}p^2 & -u_2 p_s \\ -u_2 p_s & -u_2(k_{ss}+p_{ss}x) \end{bmatrix} \begin{bmatrix} \dfrac{\partial x}{\partial \delta} \\ \dfrac{\partial s}{\partial \delta} \end{bmatrix} = \begin{bmatrix} (u_{12}-u_{22}p)p_\delta x + p_\delta u_2 \\ u_2 p_{s\delta} x \end{bmatrix} \tag{28}$$

Solving for the behavioral derivatives gives

$$\begin{aligned} \frac{\partial x}{\partial \delta} &= \frac{-[(u_{12}-u_{22}p)p_\delta x + p_\delta u_2][u_2(k_{ss}+p_{ss}x)] + u_2^2 p_s p_{s\delta} x}{|H|} \\ &= -\frac{\partial x}{\partial m} p_\delta x + \frac{-p_\delta u_2[u_2(k_{ss}+p_{ss}x)] + u_2^2 p_s p_{s\delta} x}{|H|} \end{aligned} \tag{29}$$

and

$$\frac{\partial s}{\partial \delta} = \frac{\left[u_{11}-2u_{12}p+u_{22}p^2\right]\left[u_2 p_{s\delta} x\right] + \left[(u_{12}-u_{22}p)+p_\delta x\right]u_2 p_s}{|H|} \tag{30}$$

Comparative static results for changes in the decay rate of food are ambiguous even when the perishable food is a normal good.

Multivariate Tobit Models: Computational Complexity

Assume $\mathbf{u} = [u_1 \cdots u_N]^T \sim N(\mathbf{0}, \Sigma)$ where Σ need not be diagonal. For *N* censored equations, there are 2^N possible censoring outcomes, where each power of 2 represents the two possibilities a y_i being censored or not. Let $a_i = 1$ if $y_i = 1$ and $a_i = 0$ otherwise. Let $I^{a_1 a_2 \cdots a_N} = I^a = 1$ if the observation is of type $a_1 \cdots a_N = a$, and zero otherwise. The superscript is a representation of the observation type in the binary number system.[13] Note that if none of the dependent variables are censored, then $a_1 \cdots a_N = 0 \cdots 0$ so that $I^0 = 1 - \sum_{a=1}^{1\cdots 1} I^a$. The likelihood function of a single observation is

$$L = \left[\phi_{\tilde{a}}(\mathbf{y} \mid \mathbf{x}) \Phi_a(\mathbf{y} \mid \mathbf{x})\right]^{I^a}$$

where $\tilde{a} = 1 \cdots 1 - a$, $\phi_{\tilde{a}}$ is the marginal pdf of the dependent variables that are not censored which is has a variance covariance found by eliminating the rows and columns corresponding to covariances

[13] Following convention, leading zeroes are dropped.

of uncensored random variables, and $\Phi_a(\mathbf{y} \mid \mathbf{x})$ is the marginal CDF of the censored random variables. Written another way,

$$L = \phi_{\bar{a}}(\mathbf{y} \mid \mathbf{x}) \int_{-\infty}^{0} \cdots \int_{-\infty}^{0} \phi_a(\mathbf{y} \mid \mathbf{x}) d\mathbf{y} .$$

This form illustrates why cross-equation error correlation introduces a great deal of computational complexity. The integrals in the equation must be numerically calculated repeatedly to find the maximum likelihood estimates.