Journal of Bioeconomics (2021) 23:125–149
https://doi.org/10.1007/s10818-021-09316-w

EDITORIAL

Ordinaries

Big Macs & Economics: Why we love foods that kill us

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Published online: 10 July 2021

Abstract
Neoclassical and behavioral economics disagree over the human consumption of dietary fat, a pervasive behavior that increases morbidity and mortality. Neoclassical economics assumes that people are choosing optimal diets, trading off utility and money today in return for disease and early death. In contrast, behavioral economics argues people are making poor dietary decisions. Evolutionary biology suggests that, for our human ancestors, dietary choices were optimal, in a constrained manner consistent with the neoclassical economic model. In the modern environment, which has more and different foods, biology provides no support for the neoclassical view.

Ordinary: "With no special or distinctive features; normal. Not interesting or exceptional; commonplace."

-Oxford English dictionary.

1 Why do Big Macs taste so good?

"Would the Ache enjoy a Big Mac?"

The Ache are an indigenous people in South America who have been studied extensively by anthropologists (Hill and Hurtado, 2017). When these anthropological studies commenced, the Ache lived in a manner thought to have some important similarities to the way that all humans lived before the invention of agriculture. Before agriculture, all humans can be characterized as Pleistocene foragers—people who lived by hunting animals and gathering plants.

So would the Ache enjoy a Big Mac? We asked this question of one of the anthropologists who lived with, and published extensively, on the Ache. The answer, "Yes, the Ache would love a Big Mac, and they would kill you for one."

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A Big Mac contains 8 g of saturated fat. The US government divides dietary fat into four types: monounsaturated, and polyunsaturated fats are labeled as good for humans, and saturated and *trans* fats as bad (U.S. Department of Agriculture, 2020).

The US government recommends limiting saturated fat intake to no more than 10% of total calories. One Big Mac represents almost half of the daily recommendation for saturated fat intake. While most people believe eating saturated fats is unhealthful, 77% of Americans nonetheless consume more than the recommended amount (U.S. Department of Agriculture, 2020).

Eating dietary fat, particularly "bad" fats, is an official US government source of death. Furthermore, this behavior is entirely avoidable. At a societal level, we could avoid the production of foods that kill us. At an individual level, we could navigate a world filled with death-causing food and eat only those substances that are good for us.

Given that dietary fat reportedly kills, why do we eat so much? At one level, the answer is obvious, Big Macs taste good. At another level, however, there is a deeper puzzle. Violet, a high school student, recently asked, "Why are ***all*** (her emphasis) the foods that taste good, bad for us? Shouldn't evolution have made us enjoy foods that are good for us?" Good question, Violet.

In fact, eating foods that taste good is killing people. Heart disease is the leading cause of death in the world (Mente et al., 2009). The consumption of dietary fat is reported to be one of the most significant contributors to heart disease (Milanlouei et al., 2020). Published research studies attribute 9% of all heart attacks and 16% of heart events to consumption of excessive dietary fat (Hooper et al., 2001).

In this article, we examine the consumption of dietary fat, but not with the goal of improving people's health. Rather, our primary intention is to improve economics. This is because although the facts about food and health are generally so well-known as to be trite to most people—"eat your vegetables"—the topic is important for economics.

This article overlaps with and extends our previous article on genetic mismatch (Burnham & Phelan, 2020a). In that article, we argue that the anomalies of behavioral economics, and many modern human problems including heart disease, stem from ancestral genes that are out of sync with industrialized life. This article focuses on the specific aspects of dietary fat, including a more detailed explanation of their chemical structures and the physiology of their breakdown and usage by humans, that are responsible for causing significant health problems.

This discussion has broad relevance because it exemplifies one of the two types of behavior that are challenging to economics. First, behaviors that are bad for us, but feel good. Second, behaviors that are good for us, but feel bad (see Table 1).

By focusing on dietary fat consumption in some detail, we present here an in-depth examination of a fundamental human behavior that feels good, but has negative effects, within the mission of this series (Burnham & Phelan, 2019):

> The Ordinaries column will interpret economic behavior from the perspective of evolutionary biology. From this view of life, the anomalies of behavioral economics will disappear into a coherent biological framework that incorporates elements of neoclassical maximization.

Table 1 Being bad can feel good, and being good can feel bad (from Burnham, 2016; Burnham & Phelan, 2020a)

		Feels	
		Good	Bad
Outcome in evolutionary terms of survival and reproduction	Good		Colonoscopy, dental visit, flossing, saving money, college, vaccines, helmets, prudence
	Bad	Crack cocaine, Big Mac, TV, motorcycle, pizza, **some types of dietary fat**, cigarettes	

2 Economic views of the consumption of dietary fat, without natural science insights

Why do people kill themselves by consuming unhealthful foods? The neoclassical explanation for suicide by Big Mac is the same as the explanation for other self-destructive behaviors.

People make themselves poor and kill themselves by smoking cigarettes, gambling, injecting heroin, and, most directly, by committing suicide. The neoclassical explanation is that the individuals exhibiting these behaviors, although killing and impoverishing themselves, are also maximizing utility. Nobel laureate Gary Becker argues that heroin produces so much dopamine today that becoming an addict with its concomitant costs is a good decision for some individuals (Becker & Murphy, 1988).

Similarly, the neoclassical economist argues that the people consuming dietary fat are making an optimal choice. A shorter life is a rational and informed outcome of a desire to eat good-tasting food today, which might also be cheaper. Lamenting the consequence of an early death caused by dietary choices is simply an *ex post* failure by the Big Mac lover to hold up their end of the utility bargain.

In sharp contrast, behavioral economists condemn the consumption of dietary fat as a mistake. In the behavioral view, humans—the biologically most successful species on the planet—suffer from crippling heuristics and biases.

People are so flawed, according to behavioral economists, that we are incompetent at almost everything. The list of behavioral mistakes ("anomalies") numbers into the hundreds. We suffer from loss aversion, hyperbolic discounting, overconfidence, projection bias, framing effects and dozens more defects, all the while navigating the world with tiny, boundedly rational brains.

Given the behavioral economic list of mistakes, it is amazing that humans have grown to a global population close to eight billion, harnessed nuclear power, flown to the moon, and developed vaccines in rapid response to novel pathogens.

In summary, there is a sharp divide within economics regarding individuals' decisions to consume dietary fat and, consequently, die sooner than they would otherwise. To the neoclassical economist, premature death is the rational outcome of trade-offs made with full knowledge of the consequences. In contrast, behavioral

Table 2 Economics and the consumption of dietary fat, circa 2021

Phenomenon	People kill themselves by consuming dietary fat. Dietary fat is a major contributor to heart disease, the leading cause of death globally. These deaths are avoidable if people were to make different dietary choices.
Neoclassical economics	People make optimal dietary decisions. There are trade-offs between health, lifespan, enjoyment of food, and money. Each person's level of dietary fat consumption is perfect for that individual.
Behavioral economics	Consuming dietary fat is a mistake. People are flawed vessels who make all sorts of mistakes and eating the wrong foods is one of these mistakes.

economists consider death by Big Mac another error in the long litany of mistakes made by flawed human beings (see Table 2).

3 The chemistry of dietary fat

We begin by categorizing the structural features of dietary fats. All types of fats have carbon backbones: a chain of carbon atoms—often a dozen or more—linked together with one or two hydrogen atoms attached to each carbon—with a terminal carboxyl group. The variation in appearance, taste, and health impact are caused by subtle differences in the nature of the carbon–carbon bonds.

Health agencies around the world categorize dietary fats into four categories, based on these carbon–carbon bond differences. One of essential chemical features used in categorizing fats is the level of saturation. A fat's level of saturation reflects the extent to which each carbon in the carbon–carbon chain has the maximum number of hydrogen atoms bound (two), rather than just one (Table 3).

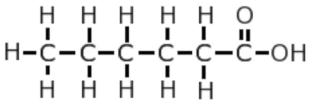
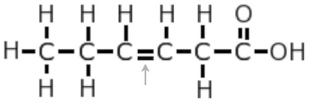
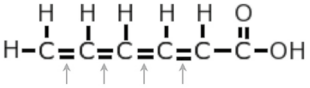
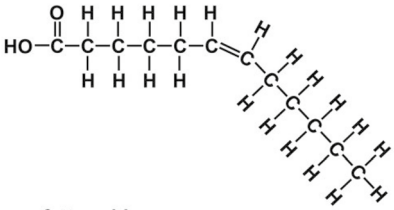
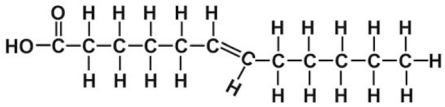
In a chain of linked carbon atoms, because each carbon is attached to a carbon on either side, two potential bonds remain. In some cases, these two bonds are each with a hydrogen atom. In other cases, just one hydrogen is bound to the carbon and there is a double bond between two of the carbon atoms, indicated as C=C. (A great deal of energy is stored within these carbon–carbon and carbon-hydrogen bonds. Much of life on earth is possible only because organisms are able to capture and use that energy.)

An unsaturated fat is one in which one or more pairs of adjacent carbon atoms are linked with double bonds, because a hydrogen atom can be added to each carbon that has a double bond. Since more hydrogen can be added to any fat with one or more double bonds, such molecules are not saturated. Unsaturated fats are liquid at room temperature.

Saturated fats have no C=C double bonds. There is not room to add any additional hydrogen to these fats, hence they are saturated. Typically solid at room temperature, saturated fats are found in meat, dairy products including cheese, and some oils such as coconut oil and palm oil.

Monounsaturated fats contain a single C=C double bond. There is room to add two hydrogens—one to each carbon on either side of the double bond. Olive oil, canola oil, avocados, and many nuts contain monounsaturated fats.

Table 3 The structure of four types of dietary fat

	Chemical structure
Saturated fats	
Monounsaturated fats	
Polyunsaturated fats	
Trans fats	***cis*-fatty acid**  ***trans*-fatty acid** 

Polyunsaturated fats have more than one C=C double bond. Polyunsaturated fats are found in some fish, including salmon and herring, as well as some seeds, such as flax seed.

***Trans* fats** start out as unsaturated plant fats. Then through an industrial process of heating the fat and passing hydrogen bubbles through the liquid, the number of carbon–carbon double bonds is reduced as hydrogen atoms bind to carbon atoms. Creating partially-hydrogenated plant oils with this process makes it possible for food scientists to alter the texture and stability of foods. That is, they are able to create foods with a more desirable texture (think: "melts in your mouth, not in your hands") and a long shelf-life.

The saturation of vegetable fats, however, creates something called *trans* fats. In contrast with *cis* fats (the naturally-occurring fats found in plants and animals), *trans* fats have hydrogen atoms bound in a slightly different orientation on either side of the double bond. In *trans* fat, the carbon atoms on either side of double bonds are on opposite sides. In *cis* fats, the carbon atoms on either side of a double bonds are on the same side (see Table 3 and the figures within). This subtle difference, as we will see, has significant consequences.

4 Biology: What is food?

Can you imagine trying to eat wood? Or, worse yet, rocks? For humans, such a diet would quickly lead to starvation. To stay alive, animals need to eat food, and rocks and tree trunks are not food. Or are they?

"Food" doesn't exist as an objective category of substance. Substances simply exist as molecules—a collection of atoms of one or more different types—bound together by chemical bonds. In contrast with "food," atoms and molecules *do* exist in an absolute sense. Whether named and described by humans or not, they have a chemical make-up, defined solely by their physical properties.

It is the *interaction* of a substance with a living organism that determines whether or not the substance is food for the organism. The molecules comprising the substance and the specific ways they are bonded to one another influence the amount of chemical energy contained within it. But that energy is accessible to a living organism only if it has the molecular machinery to disassemble the substance, reassemble those molecules into other molecules and tissues, and capture the energy released when the new bonds store less energy than those in the original molecules.

The human digestive system-as well as the digestive systems of other organisms–is like an assembly line running backward. Imagine starting with an assembled car and dismantling it into its many parts: the tires, doors, windshield, steering wheel.

Food entering the digestive system is like the intact car. It enters the assembly line and then passes through several phases, during which the food is chewed up and broken down, the nutrients absorbed by the body, and the non-usable portion of the raw materials discarded as waste products. But it's not an all-purpose dis-assembly line. Not every substance can be broken down by any given organism.

4.1 Cellulose is food, but also "not-food."

Locked in a library, humans would die of starvation. The lowly silverfish, on the other hand, would thrive–growing and reproducing. This is because the paper and glue in books is a tasty meal–that is, "food"–for silverfish. Yet those very same substances are not "food" for humans.

Here's why that silverfish (*Ctenolepisma lineata*) can eat books. The chief molecule in paper is cellulose, a linear chain of hundreds to thousands of ß-1–4-linked glucose units. Stored within the bonds of those molecules is a huge amount of energy, which silverfish can harvest.

Living on a diet of cellulose alone, the silverfish gains weight and shows a respiratory quotient (the ratio of carbon dioxide production to oxygen consumption) of close to 1.0 (Lasker & Giese, 1956)—indicating complete utilization of carbohydrates—in this case, cellulose. Moreover, when bacteria-free silverfish are fed cellulose containing radioactively labeled carbon, they respire $^{14}CO_2$, indicating that the cellulose has been digested and metabolized by the silverfish.

Silverfish digest cellulose by producing the enzyme C1-cellulase. This enzyme is also produced by other species which are able to break down cellulose (Martin,

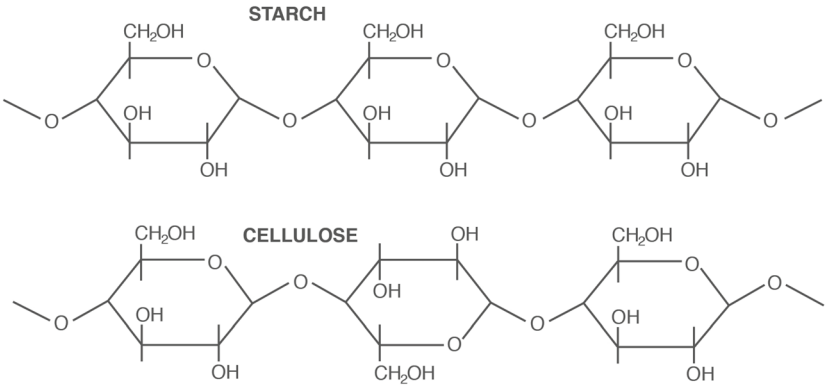


Fig. 1 Starch and cellulose are both glucose polymers, with different types of bonds

1983, 1991), including termite species (*Trinervitermes trinervoides*; Potts & Hewitt, 1973, 1974; *Mastotermes darwiniensis*; Veivers et al., 1982) and cockroaches (Scrivener et al., 1989).

More recent evidence establishes the existence of cellulose-digesting enzymes in a wide range of invertebrate animals (Watanabe & Tokuda, 2001) and also documents that the genes for these enzymes are derived from a common ancestor (Davison & Blaxter, 2005). The modes of action of these enzymes, such as endo-ß-1,4-glucanases and cellobiohydrolases, have been described in detail (Watanabe & Tokuda, 2010).

Just as cellulose is a chain of glucose molecules, the starch contained in myriad human foods, such as potatoes and pasta, also consists of a chain of glucose molecules. There is one subtle, but crucial chemical difference between starch and cellulose, however, in the conformation of the chemical bonds between adjacent glucose molecules. In starch, the glucose molecules are connected by **alpha** 1–4 glycosidic linkages, whereas in cellulose the connections are via **beta** 1–4 glycosidic linkages.

The difference between alpha and beta linkages is in the orientation of hydroxyl (OH) groups on adjacent glucose molecules. In starch, all the hydroxyl groups are on one side, while in cellulose the hydroxyl groups alternate in orientation (see Fig. 1).

Humans (and all other vertebrates), unfortunately, have not evolved the useful cellulose-digesting genes. As a consequence, cellulose does not function as food for us. We do consume many foods that contain cellulose—but we only digest, metabolize, and make effective use of other, non-cellulose, molecules within those foods. Fecal analyses confirm that humans excrete nearly all of the consumed cellulose molecules, undigested and unused, with a very small, nutritionally insignificant, amount broken down by bacteria in the colon (Kelleher, 1984). Nearly all of that potential energy in the chemical bonds within cellulose goes unused by us.

4.2 How do we extract value from food?

Many substances besides cellulose aren't food for humans. Rocks, as we noted above, aren't on the menu either. Yet the molecules within them do store energy in their bonds. So it shouldn't come as much of a surprise that many microbe species have the metabolic machinery to extract energy and nutrients from rocks. And in the process of "eating," they produce a variety of minerals as by-products (see, e.g., Hazen et al., 2008; Amenabar & Boyd, 2019).

But humans clearly are not going hungry. In fact, the range of substances humans can consume for energy and raw materials is huge. In every case, these substances contain one or more of the three groups of macromolecules: proteins, carbohydrates, and fats. Proteins provide raw materials for growth and for the production of enzymes. Carbohydrates are the primary fuel on which our bodies run. And dietary fats function primarily as a dense source of energy that can be efficiently stored in the body.

As we saw in the case of cellulose, for digestion of any macromolecule to be possible, an organism must produce a very specific chemical enzyme that can catalyze the metabolic degradation and uptake reactions. Typically, many different digestive enzymes work in sequences of reactions to break down food into molecules that can be absorbed into the bloodstream and used by the body's cells for energy and raw materials.

Without all of the proper enzymes—including numerous proteases, trypsins, and pepsins for protein breakdown, lipases for lipid breakdown, as well as amylases, maltases, and many, many more enzymes for carbohydrate breakdown—a consumed item is useless to us and eventually excreted.

4.3 Why is there so much variation in the diets of different species?

It might seem strange and inefficient that there is so much variation among all the different species on earth when it comes to the substances that they can and cannot break down for energy and raw materials. Some species can digest cellulose, others can't. Some can digest lactose, others can't. Why isn't there one universal set of digestive enzymes?

The answer is evolution. Natural selection can be remarkably efficient when it comes to eliminating versions of genes that carry the instructions for structures—or digestive enzymes—that provide no value for organisms. As a result, when species become adapted to a habitat, they tend to have digestive tracts and enzymes highly specific for their diet in that habitat. Researchers have documented dramatic adaptive evolution for the genes encoding digestive enzymes.

In one detailed investigation of 48 bird species, researchers documented strong selection on 16 different digestive enzyme genes. Specifically, they noted improved enzymatic efficiency and catalytic capacity driven by the specific diets of each species (Chen & Zhao, 2019).

For example, in species consuming larger amounts of seeds and meat than other very closely related species, scientists observed that there had been intense selection

for versions of the genes coding for improved enzymatic breakdown of those substances. Conversely, they noted reduced selection on those genes in species consuming diets with less of those food types.

Across numerous other taxa and diverse feeding habits, researchers have observed similarly dramatic genetic changes in response to dietary diversification. This includes the dietary switch from carnivorous to herbivorous in pandas (Zhao, 2010), from carnivorous to omnivorous in dogs (Axelsson et al., 2013), from omnivorous to herbivorous in colobine monkeys (Zhang et al., 2002), and from herbivorous to carnivorous in Cetaceans (including whales, dolphins, and porpoises) adapted to aquatic habitats following the transition from land to water (Wang et al., 2016).

Put simply, a consistent theme in nature is the adaptation of enhanced ability to digest the macromolecules associated with new feeding habits (and the loss of digestive functions associated with feeding habits no longer employed) when such changes increase the evolutionary fitness of those individuals manifesting them.

5 Biology: Natural selection and the evolution of food preferences

Economics assumes that people choose behaviors based on preferences. But where do preferences originate? The answer to this question, too, is evolution by natural selection. Over evolutionary time, preferences evolve so that people maximize the biological measure of inclusive fitness by choosing the behaviors that provide the most pleasure.

So, physiologically and biochemically we understand what food is and how we extract value from it. And evolutionarily, we recognize dramatic digestive enzyme adaptations in response to the specific habitats of populations. But the behavioral question remains: How do organisms—including humans—"know" which substances to seek out and consume? And how do we know which substances to pass over?

That's where taste comes in, and is where food preferences generate outcomes that can improve organisms' evolutionary fitness. Within populations, the individuals that enjoy and consume more nutritious foods and energetically-dense items are more successful when it comes to relative survival and reproduction rates. Over evolutionary time, natural selection can create a finely-tuned system for locating and consuming optimal diets. We experience this as food preferences, as do organisms of other species.

The old-field mouse (*Peromyscus polionotus*) is a rodent that feeds primarily on seeds and fruit. In experiments in captivity, as well as in outdoor enclosures within their habitat, they eat about a gram of food per day, regardless of whether they are given millet, sunflower seeds, or peanuts.

When old-field mice are given a choice—in the form of multiple dishes, each filled with a different seed type—however, they have clear preferences. If one of the dishes contains peanuts, they'll almost completely restrict their intake to that dish. In the absence of peanuts, they almost completely restrict their intake to sunflower seeds. They'll eat millet, but only when sunflower seeds or peanuts are not available (Phelan & Baker, 1992).

Why do these rodents have such strong and consistent taste preferences? Their food preferences closely reflect the net energetic payoff of each seed type to the mouse. These reflect what is termed the "E/h" value for each type, where E is the caloric density of the food and h is the handling time for each type. Preference for maximizing E/h conforms to the most fundamental prediction of optimal foraging theory (Emlen, 1966; Charnov, 1976; Stephens & Krebs, 1986).

Humans, too, have food preferences. And ethnographic tests of optimal foraging theory hypotheses have given us useful insights into these preferences. Across numerous studies, researchers have documented that food choices are largely consistent with diet-breadth models predicting: 1) maximization of net energy returns per unit handling time, 2) preferentially foraging within locales with higher yields, and 3) altering foraging sites in response to shifts in relative yields (Smith et al., 1983).

These include studies of the Ache of eastern Paraguay (Hawkes et al., 1982), the Siona-Secoya of Ecuador and the Ye'kwana and Yanomamo of Venezuela (Hames & Vickers, 1982), and Samoan horticulturists (Gage, 1980).

Consistent with these findings, energy density predicts preferences among fruits and vegetables in 4-year-old children (Gibson & Wardle, 2003). Working with adults and extending food choices to high-fat foods with much greater energy density–from cucumber (0.1 kcal/g) up to chocolate (5.3 kcal/g)–Brunstrom et al. (2018) found a similar significant correlation between food preference and energy density. Put simply, love of high calorie, energy-dense foods—specifically dietary fat—is an adaptation.

Our hearty appetites are nothing more than an incentive system to get us to eat. Furthermore, to be healthy, all people need to consume dietary fat. Failure to consume enough dietary fat, and the right types, leads to the condition "Essential Fatty Acid Deficiency." In modern, well-fed human populations, Essential Fatty Acid Deficiency is seen most commonly in people who are fed intravenously (Yamanaka et al., 1980; Davila & Konrad, 2017).

The health complications from eating too little fat highlight the fact that humans do need to consume some dietary fat. Far from being a food item to be shunned, fat is essential for normal growth, aids in the creation and maintenance of membranes, mediates the expression of genes, and provides myriad health benefits that may include cancer prevention (Fuentes et al., 2018).

As hunter-gatherers, a reward system for finding and consuming calories, with an emphasis on fats, was an effective mechanism for enhancing evolutionary success. For our hunter-gatherer ancestors, when it came to food, our preferences led us to optimum, appropriate behaviors. Fats tasted good to ancestral humans. And they were good for those people.

Biology thus explains the origin and nature of preferences. Human preferences evolved as an incentive scheme to induce people to undertake the behaviors that led to the most biologic success. We enjoy dietary fat because its consumption led to more and healthier babies for our ancestors (as well as more nieces, nephews and other genetic relatives). For our ancestors, eating dietary fat tasted good and was good for them (Table 4).

Table 4 Eating fat tasted good and was good for our ancestors

		Feels	
		Good	Bad
Outcome in evolutionary terms of survival and reproduction	Good	**Dietary Fat**	
	Bad		

6 Biology: Dopamine from consuming dietary fat

From the evidence described above, it is clear that taste preferences for energetically dense foods exist in animals as well as in humans across a wide range of diverse cultures. Moreover, these preferences reflect the significant evolutionary fitness advantages that accrue to individuals able to obtain relatively greater amounts of energy with greater efficiency. But are humans or other animal species motivated to seek out such foods as a result of conscious effort to win some contest for maximizing reproductive success? Of course not.

Humans and other animal species have a nervous system, enabling us to detect sensations such as light, sound, touch, taste, and smell, and to respond to that sensory information. The neurological systems have evolved to reward fitness-maximizing behavior—those behaviors that increase an individual's survival and reproduction relative to others in the population. And numerous lines of evidence indicate that within our brain reward systems, dopamine is the primary currency.

For example, in vivo recordings of individual neurons demonstrate the link between dopamine release and reward perception (Cohen et al., 2012; Schultz, 2013). We are built to seek out and repeat those behaviors that make us feel good (Burnham & Phelan, 2020b).

Our brain's pleasure centers—chiefly, our dopamine systems—mediate food-based rewards and motivate our diet choices (Smith, 1995; Cooper & Al-Naser, 2006). This regulation involves numerous brain circuits and includes the modulation of food intake in response to factors including satiation, energy, taste, and smell (Pfaffmann & Norgren, 1977; Norgren et al., 1989; Meguid et al., 2000).

Consistent with observations that organism diet choices reflect maximization of energy/handling-time, reward intensity corresponds to the energy density of foods consumed (Li et al., 2009). Numerous studies document relatively greater dopamine release in response to fat-rich diets relative to low-fat diets (Kannel & Wilson, 1995; Martel & Fantino, 1996; Liang et al., 2006). This enhanced reward system stimulation, in turn, leads to an increase in the pleasure associated with these diets (Kelley, 2004; Costa, 2007).

Interestingly, dopamine rewards are produced in response to gastrointestinal tract signaling, reflecting the energetic content of ingested food (de Araujo et al., 2012). In fact, brain dopamine levels directly reflect caloric density even when mice receive intra-gastric infusions of fat emulsions (Ferreira et al., 2012; DiFeliceantonio, 2018). It's not even necessary for there to be flavor cues present for the brain to detect *and reward* the obtaining of fat calories!

Our calorie-sensing-and-rewarding system has additional features that further enhance its effectiveness in motivating diet choices. For example, when we are hungry, we experience greater dopamine release and reward-center activation, and when we're satiated we experience less (Roseberry, 2015).

In addition to the direct observations of dopamine reward system activation corresponding to the caloric value of ingested food, numerous indirect experimental approaches further bolster our understanding of these processes. Researchers have documented that lesions that damage the reward centers produce aphagia (Teitelbaum & Epstein, 1962). While the animals don't lose their ability to taste or to distinguish between sweet and bitter substances, they simply ignore food (Berridge et al., 1989).

Similarly, drugs that enhance the sensitivity of the reward centers stimulate significant increases in consumption (Wise & Dawson, 1974; Cooper, 1980; Cooper & Moores, 1985). Drugs which suppress the dopamine reward centers, on the other hand, produce aphagia (Wise & Colle, 1984; Wise & Raptis, 1986; Schneider et al., 1990).

Preferences evolved by natural selection to produce fitness maximizing behavior. This is the ultimate cause of human preference for dietary fat (Tinbergen, 1963). The proximate or mechanistic cause of our love for dietary fat is a sophisticated neurologic system, functioning as a reward system to induce behaviors that produce dopamine.

We have specific systems to detect nutritious food and send dopamine to the reward centers. These systems exist in all humans. Thus, even without prior exposure to a Big Mac, nor any advertisements, the anthropologist could confidently predict that the Ache would love calorically-dense packet in the form of a fast food meal.

7 Biology: Mismatch is the reason that eating fat kills modern humans

"Strangers in a strange land: mismatch and economics," is the second article in this Ordinaries series (Burnham & Phelan, 2020a). It contains a fuller description of genetic mismatch and describes the most important themes relating the 'out of sync' idea to economics. Here we summarize the general mismatch view and apply it to the topic of dietary fat.

People in industrialized economies suffer from a mismatch between the evolved, genetic human nature and the modern environment. This mismatch is the source of behavioral economic anomalies and a host of actually-deleterious human behaviors.

Mismatch occurs because evolution by natural selection can be a slow, multi-generation process, whereas technology changes more rapidly. Our genes cannot always keep up with the pace of change.

This is why our tastes ('preferences') reflect ancestral payoffs. Natural selection favors the evolution of preferences that induce biological fitness maximization. In equilibrium, living for today by maximizing dopamine release results in maximization of inclusive fitness (biological success).

The human struggle to eat a healthy diet is an example of mismatch. The most compelling evidence is that modern hunter-gatherers have healthy hearts.

Apparently, these healthy hearts come without any effort to restrict calories or retrain dietary choices. The short version is that modern hunter-gatherers eat what and when they want, and have no issues with either dietary fat consumption or too many calories.

Even though we have written more extensively about modern hunter-gatherers (Burnham & Phelan, 2020a) and there are extensive popular press accounts (e.g., Reynolds, 2017), the findings regarding heart health are amazing and worth repeating.

For example, the hearts of the Tsiname of Bolivia have been analyzed by CAT scan (CT). Popularly cited in newspapers and magazines as having the "healthiest hearts in the world," the original academic article states that the Tsiname have "the lowest reported levels of coronary artery disease of any population recorded to date." (Kaplan et al., 2017).

Our modern struggles with diet are caused by mismatch. Let us examine mismatch specifically with regard to the FDAs two 'bad' fats—*trans* fats and saturated fats.

7.1 Genetic mismatch and *trans* fats.

Recall that in *trans* fats hydrogen atoms bound to adjacent carbon atoms are on the opposite side (trans) from each other. Most fats in nature are *cis*, with small amounts of *trans* fats in some animal products (Dhake et al., 2011 In the twentieth century, humans learned how to create *trans* fats by the partial hydrogenation of vegetable oils (Phelan, 2018). We have written extensively on *trans* fats in textbook format (Phelan, 2018). Here is a brief version of that textbook account.

When a method for hydrogenating vegetable oils was discovered, it was an exciting advancement in the world of food technology. Adding hydrogen bonds to unsaturated plant oils made it possible to create fats with desirable physical properties. Margarines, for example, are soft enough to be spread on bread right out of the refrigerator—something not possible with butter. What's more, foods made with hydrogenated vegetable oils had a longer shelf-life and were less expensive.

Over time, however, data accumulated indicating that consumption of *trans* fats increased the risk of heart disease. For example, in a study of some 120,000 nurses, those who consumed just 2% more *trans* fats than their cohorts experienced nearly double the risk of heart disease. Greater consumption of saturated fats was associated with a much smaller increase in heart disease.

As noted, the nurses study was large—and spanned decades—but it was observational and did not include any controlled experimental manipulations. The question "Do *trans* fats increase your risk of heart disease?" still loomed ominous and unresolved.

A later study tried to more clearly identify causation. In it, researchers recruited approximately 60 participants. Their average age was 25, and about 60% were women. The volunteers were restricted to identical diets except that 10% of the total calories came from (1) *cis* (i.e., not trans) unsaturated fat, or (2) *trans* fat, or (3)

saturated fat. Each diet was consumed by each person for three weeks, in random order, for a total of nine weeks.

After each three-week period, the researchers analyzed blood samples taken from participants. They measured a lipoprotein (a mix of lipid and protein) called LDL that damages arteries and is associated with increased risk of heart disease. They also measured another lipoprotein, called HDL, which reduces damage to arteries and is associated with a reduced risk of heart disease.

The results? Blood levels of LDL—sometimes referred to as "bad cholesterol"—were significantly higher after three weeks on either the *trans* fat diet or the saturated fat diet, compared with the unsaturated fat diet. Perhaps more importantly, after three weeks on the *trans* fat diet, levels of HDL—"good cholesterol"—were significantly lower. This HDL decrease occurred only on the *trans* fat diet.

The ratio of LDL to HDL is one of the most useful measures of heart disease risk. By this measure, when research participants were consuming the *trans* fat diet, they had significantly greater risk of heart disease than when consuming either the unsaturated or saturated fat diets. The researchers concluded that *trans* fats in the diet do cause harm.

Trans fat in abundance are evolutionarily novel. Industrialized *trans* fats were created in a laboratory by a novel process. Suddenly, from an evolutionary perspective, the world was filling up with products loaded up with *trans* fats. This substance was great for producers, whose cookies and other products could sit in warehouses, trucks, and on shelves for months. Furthermore, by some measures *trans* fats seem to taste even better than 'natural' fats (something we will see again with the artificial sweetener, sucralose).

Upon the invention and widespread adoption of *trans* fats, people were immediately put in a situation of genetic mismatch (Table 5). Industrially-created *trans* fats became ubiquitous, tasted great, and killed people (Remig, 2010).

7.2 Genetic mismatch and saturated fats

Does the consumption of saturated fat cause heart disease? There is ongoing disagreement on this question (see Astrup et al., 2020 and O'Neill & Raggi, 2020 for two current articles). We are not going to review this extensive literature in detail, nor take a position on either side of the debate. Rather, we are going to discuss saturated fat in the context of economics and our framework for the issues that challenge economics.

From our prospective, there are two different versions of the saturated fats and genetic mismatch idea. The first version of this is similar to that of *trans* fats. Ancestral humans consumed saturated fat in animal meat, yet suffered no negative effects. The mismatch arose with the invention of animal husbandry. Livestock enables people to consume more meat, and the meat from livestock has higher fat levels than wild meat.

Other dietary changes came along with agriculture and animal husbandry. Humans domesticated animals to produce eggs, milk, and other dairy products.

Table 5 *Trans* fats are novel, taste good and are unhealthful

		Feels	
		Good	Bad
Outcome in evolutionary terms of survival and reproduction	Good		
	Bad	Industrially produced, partially hydrogenated, *Trans* fats	

Because of these changes, the amount of saturated fat in the human diet increased significantly.

Some early studies argue that saturated fats in the diet directly cause heart disease. For example, the 'seven countries study' argues for a causal relationship where eating saturated fats leads to higher blood levels of cholesterol, which causes heart diseases (Keys, 1970; Andrade et al., 2009). The seven country study states, "there was a tendency for [coronary heart disease] incidence to be related to the prevalence of hypertension, serum cholesterol values and saturated fatty acids in the diet." (Keys, 1970, p 186.)

From this perspective, eating more saturated fat causes heart disease. The mismatch is caused because industrialized humans retain their ancestral taste for dietary fat, and now much greater amounts of that fat can be obtained (Table 6).

However, the idea that saturated fat consumption always causes heart disease is no longer widely accepted. Other dietary factors, including carbohydrates, are implicated along with dietary fat in coronary heart disease. Overall, the relationship between saturated fat and coronary heart disease is multifactorial (Siri-Tarino et al., 2015). A relatively recent review article summarizes the data as follows, "the effect of individual foods on coronary heart disease cannot be predicted solely on the basis of their content of saturated fats" (Bier, 2016, p. 1945).

This second view is often summarized as a Paleolithic or 'Paleo' diet prescription. Humans co-evolved with food sources for millions of years. All of this changed with the invention of agriculture about 10,000 years ago. Our genes have not caught up with the impact of agriculture. To eat a healthy diet, humans should return to a pre-agriculture diet and lifestyle.

In the Paleo view, what is new is not the consumption of saturated fat from animal meat, but rather the consumption of large amounts of carbohydrates, and, importantly, a decrease in physical activity (Table 7).

For the purposes of this article, and from an evolutionary perspective, the competing views are both examples of mismatch. Eating saturated fats did not cause heart disease for our ancestors. Retaining our ancestral genes in a different world leads to heart disease. Genetic mismatch is the cause of dietary induced heart disease.

Table 6 Saturated fats and heath. Mismatch version 1

		Feels	
		Good	Bad
Outcome in evolutionary terms of survival and reproduction	Good	Saturated fat, obtained in moderate amounts by hunting and gathering	
	Bad	Saturated fat, obtained in massive amounts by industrial food production	

Table 7 Saturated fats and heath. Mismatch version 2

		Feels	
		Good	Bad
Outcome in evolutionary terms of survival and reproduction	Good	Saturated fat, consumed without agricultural carbohydrates, and as part of physically active lifestyle	
	Bad	Saturated fat, consumed with agricultural carbohydrates, and as part of sedentary lifestyle	

8 Applying the Ordinaries' taxonomy of improvement to dietary fat

"Surviving desire: the causes and cures of self-control issues" is an Ordinaries paper that describes a taxonomy of approaches for obtaining better outcomes (Burnham & Phelan, 2020c). There are four strategies in the ordinaries taxonomy: (1) Will Power, (2) Innovation, (3) Mast Strapping, and (4) Dopamine Modulation. Let us investigate these strategies in the specific context of consuming dietary fat.

8.1 Strategy 1: Will power

Will power is the ability to eat a healthful diet even though junk food tastes better. As noted in our previous article on self-control, there are deep philosophical issues regarding will power. Consider a person who eats grilled, wild-caught salmon (rich in Omega-3 fatty acids), but would get more pleasure from a Big Mac. An economist might say there is no such thing as will power; the behavior of eating the salmon reveals that it is preferred to a Big Mac.

We appreciate the philosophical concept but continue to believe that will power is a useful idea in the context of diet. Strategy 1 in the Ordinaries taxonomy is to decide on a diet and try to eat the foods that you want to eat, even if they taste worse to you than foods on your preferred menu.

Most people have gotten as far as they can using will power. To obtain more improvement requires use of the three additional strategies.

8.2 Strategy 2: Innovation

Innovation is the creation of novel products that taste good and are not bad for us. At the simplest level, this could be a well-seasoned vegetable dish using some combination of spices and cooking techniques that make the healthful food also super tasty.

A different approach is to engineer a new type of molecule. There have been a significant number of these produced by corporations. Our view is that none of them are total successes, but we will describe two to make the concept clear.

Gatorade-zero is a sweet drink that contains no sugar and has zero calories. The sweetener in Gatorade-zero is sucralose, which is very similar in structure to sucrose, commonly referred to as table sugar.

Sucralose is an engineered molecule where three of the hydroxyl groups (OH) of sucrose have been replaced with chlorine (see Fig. 2).

People feel good when eating sugar. We perceive a sweet item in our mouth, and this perception system generates a cascade leading to dopamine release in our brain's pleasure centers (Wintjens et al., 2011). As noted, this system evolved as an adaptation to motivate ancestral humans to obtain sugar, with its valuable energy.

The goal of artificial sweeteners is to stimulate the human perception system for sweetness, thereby releasing dopamine, by using a product that mimics key features of natural sugars. The goal of the artificial sweeteners is to stimulate all of the dopamine release, while yielding none of the calories. Interestingly, sucralose is reported to be much sweeter than sucrose—up to 1000 times as sweet (de Souza, et al., 2013).

Sucralose is effective in creating the taste of sweetness without any calories. There are conflicting studies on the effectiveness of sucralose in weight loss and concerns about negative side effects (Thomson, et al., 2019). Even if sucralose turns out to have more costs than benefits, it illustrates the idea of innovation via new molecules.

Directly on the topic of dietary fat consumption, Olestra is a fat substitute designed to create the pleasure associated with the consumption of fat, but without the negative effects. The idea is the same as with sucralose. Create a molecule that mimics the key features of dietary fats, but cannot be metabolized by humans. A normal fat has a head region and three long fatty acid chains attached. Olestra uses sucrose at the center, with 6 long fatty acids chains attached.

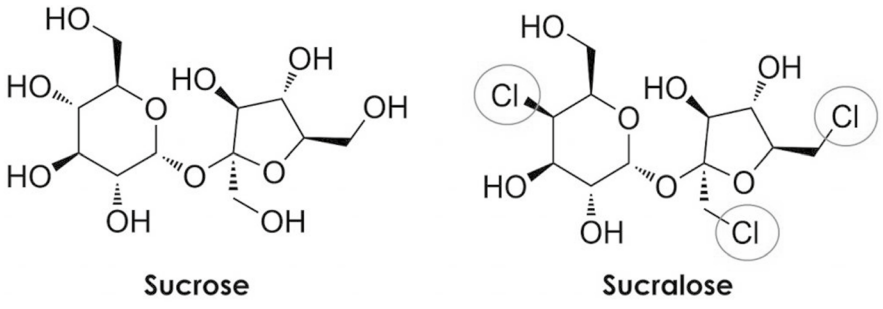


Fig. 2 Sucralose has been designed to mimic sugar

Olestra is successful in creating pleasure from the fat-sensing mechanisms in humans, and it passes undigested through the human body. Unfortunately for the manufacturers, it can pass too quickly through the body, and in some people it causes abdominal pain (Barlam & McCloud, 2003). Because of these gastrointestinal effects, the use of Olestra has been largely discontinued (Gélinas, 2013).

8.3 Strategy 3: Mast strapping

Mast-strapping is named after Odysseus's solution to the Sirens on his way home from the Trojan War. The paradoxical idea being that having fewer options can improve the outcome. By being tied to the mast, Odysseus was able to obtain his goal. More often, we think that having more options and flexibility increase our likelihood of finding a good outcome. Mast strapping is a form of "less is more."

As of 2019, 40 countries have passed laws restricting the use of industrially produced trans-fatty acids (iTFAs). The US and Canada have banned iTFAs completely. These restrictions have dramatically decreased the consumption of iTFAs with a significant positive impact on health (Li, 2019).

Restrictions of consumption of artificial *trans* fats is an effective example of mast-strapping. Companies are not forced to compete to lower food prices by using *trans* fats. All companies are on a level playing field in areas where *trans* fats are banned.

At an extreme, mast-strapping means completely removing an option. More subtle applications involve making the better dietary fat option relatively more attractive. In our prior article, we discuss eating before going to the supermarket as folk wisdom that works because it changes the perception of options.

Along these lines, one can use a variety of such low-tech solutions to increase the likelihood of consuming a better composition of dietary fat. Stocking the house with foods you want to eat, and doing this stocking when satiated, may tip the balance toward health. If the paleo advocates are correct that carbohydrates—and not saturated fats—are the problem, then stocking tasty paleo options, may also work.

8.4 Strategy 4: Dopamine modulation

Antabuse is designed to make a person feel bad if they consume alcohol. Nicotine vaccines reduce the pleasure of smoking. These are examples of dopamine modulation—taking steps to reduce the pleasure received from engaging in a behavior we wish to avoid.

We are not aware of any high-tech methods akin to antabuse or nicotine vaccines to alter the consumption of dietary fat. Low-tech solutions such as the shopping-only-when-satiated idea mentioned above may help. Similarly, pre-ordering foods or meals in advance may tip the balance a bit in the favor of better foods.

8.5 Summary of behavioral strategies.

There are four methods to alter the consumption of dietary fat. Will power, create something new that is more healthful, mast strap to restrict access to something unhealthful, or change the impact of food on your happiness in a strategic manner.

None of the ideas that we mention in this article are going to change the situation immediately. We do not have a magic secret to make people alter their diet and live longer. Nonetheless, we are optimistic for two reasons.

First, behavioral changes for hard problems, such as eating too much of the wrong food, take years and many small steps. Having a productive framework for those smaller adjustments can dramatically increase the pace and likelihood of success.

Second, we are in the infancy of technological change. Our ability to innovate has never been greater. So even in the absence of magic bullets today, there is the promise of future innovation.

9 Biological economic views on the consumption of dietary fat.

We recap what we have learned from the natural sciences, then return to the economics of unhealthful foods.

First, food is not an absolute concept. Organisms derive energy and raw materials from food. Each species of organism has its own set of food. The heat generated by burning wood, demonstrates the vast stored energy in cellulose. Humans, however, derive no calories from cellulose because we lack the enzymes to break the relevant bonds in that carbohydrate.

Wood is not food for humans. However, wood is food for termites, silverfish, and other organisms that have the appropriate enzymes to break the glucose-glucose bonds in cellulose.

Second, our taste for dietary fat arose as a genetic adaptation. Whereas modern humans suffer from eating too much, ancestral humans confronted the exact opposite problem—they were often hungry. Dietary fat is calorically-dense, providing a large amount of energy per unit of mass and handling time. Furthermore, essential fats are required for important metabolic pathways and cannot be synthesized.

We have specific physiologic machinery that detects dietary fat and generates a dopamine reward within the brain's pleasure centers. We are built to enjoy the consumption of dietary fat. When we attempt to alter our diet, our opponent is our own taste and desires. This is not an easy opponent to overcome or sidestep.

All humans must eat fat to remain healthy. And the value of extra fat was positive for ancestral humans. We are built to love the consumption of dietary fat.

Third, genetic mismatch is the reason that eating fat can harm our health. Industrialized humans live in an alien environment, different in systematic ways from the ancestral environment. Heart disease caused by dietary choices is caused by the mismatch between ancestral genes and modern settings.

Let us return to economics. Neoclassical economics assumes that people's choices about the consumption of dietary fat are optimal. When people consume

foods that shorten lifespan and create disease, the neoclassical economist sees a trade-off.

How does a neoclassical economist view *trans* fats? The benefits of *trans* fats may include lower cost and better taste. Because *trans* fats help foods stay fresh for longer, the price might be lower. An Oreo cookie made with *trans* fat might, for example, be priced 5% lower than a similar Oreo made without *trans* fat. Additionally, some people prefer the taste of *trans* fats.

The neoclassical economist argues that the consumer of *trans* fats is making an optimal decision. Save a few pennies today and get better taste in return for years of heart disease and an earlier death. "Who are we," the neoclassical economists believe, "to get between a person and their inexpensive cookies."

The biological view, however, is that *trans* fats are evolutionarily novel. While there are very small quantities of *trans* fats in the wild, the recent creation of industrialized, partially-hydrogenated *trans* fats can supply them in large amounts. Thus, there has been no time for evolution to evolve preferences to reflect the current negative dietary value of *trans* fats. Furthermore, such preferences might never occur.

What are we to do with *trans* fats? Behavioral economists argue that *trans* fats are bad, and people need help to improve their lives. Without nudges, people will make bad food choices. Consistent with this view, the United States and some other countries have banned manufactured *trans* fats. *Trans* fats kill, humans have the wrong preferences when it comes to *trans* fats, and the world is a better place with a ban on human-made *trans* fats.

Now let us turn our attention to the consumption of saturated fats. Neoclassical and behavioral economics retain their standard views. Neoclassical economics argues that eating saturated fat is optimal, even if it creates sickness and death. Behavioral economics argues that people need help avoiding the consumption of saturated fats.

Biological insights illuminate a different possible take on saturated fats. Modern hunter-gatherers eat saturated fats yet have tremendously healthy hearts. The source of heart disease from diet is mismatch.

One view is that mismatch is in the *quantity* of saturated fats that are available. Focusing just on meat, for example, wild animals are leaner (and harder to catch) than domesticated livestock. Thus, modern humans might be harmed by saturated fat consumption because we eat more meat and that meat has more fat per ounce.

An intriguing alternative, however, is that carbohydrates are the most significant cause of genetic mismatch involving diet. From this view, ancestral humans consumed both saturated fat and carbohydrates. The change to a modern diet involves change in both – more-readily-available fatty meats, along with an agriculture-enabled increase in carbohydrate consumption.

Proponents of paleolithic diets argue that restricting novel foods, including many grains, is central to a good diet. As stated, we are not taking a stand on the restriction of saturated fats vs. paleolithic approaches; to us, the evidence is not completely persuasive for either side. In either case, however, the source of the negative health effects is genetic mismatch.

Table 8 Economics and consumption of dietary fat with the natural sciences

Phenomenon	People kill themselves by consuming dietary fat. Dietary fat is a major contributor to heart disease, the leading cause of death globally. These deaths are avoidable if people were to make different dietary decisions.
Neoclassical economics	People make optimal dietary decisions. There are trade-offs between health, lifespan, enjoyment of food, and money. Each person's level of dietary fat consumption is perfect for that individual.
Behavioral economics	Consuming dietary fat is a mistake. People are flawed vessels who make all sorts of mistakes and eating the wrong foods is one of these mistakes.
Biological economics	The pleasure that humans derive from consuming dietary fat is an adaptation. Our human ancestors were more successful biologically if they were able to obtain extra calories from fat. Genetic mismatch is the reason that dietary fat consumption is bad for humans today. We have more food, more carbohydrates, more fats, different fats, and less physical activity than our ancestors. Human-engineered *trans* fats are novel and bad for our health. Our preferences are out of sync with respect to *trans* fats. Humans, and our non-human ancestors, have enjoyed saturated fats dating back millions of years. Genetic mismatch is the cause of saturated fats causing health problems. There are two different versions of the mismatch and saturated fat hypothesis. First, more saturated fat is bad by itself. Second, carbohydrate consumption causes the consumption of saturated fat to be unhealthful.

In conclusion, the natural sciences provide no support for the neoclassical economic view. In our novel modern environment, humans prefer some foods that kill us. Left to our own devices, dietary choices will not be optimal.

Behavioral economics thus finds support in the natural science view. Because of genetic mismatch, people will make bad dietary choices. Do the natural sciences help behavioral economics, or could we simply not have written this article?

We find value in the natural science insights, with the theoretical foundations that illuminate the specific causes and consequences of mismatch and its framework that points toward practical solutions. Evolution by natural selection has built us to love eating fat. We need to be strategic and disciplined to navigate our way to a better diet (Table 8).

Acknowledgments Paul Greenberg provided timely and valuable comments.

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