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**SOCIAL AND SCIENTIFIC DISORDER AS EPISTEMIC
PHENOMENA, OR THE CONSEQUENCES OF GOVERNMENT
DIETARY GUIDELINES**

BY

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Social and Scientific Disorder as Epistemic Phenomena, or The Consequences of Government Dietary Guidelines

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Abstract

We begin with an economic model of science according to which signals concerning *scientific reputation* both serve to coordinate the plans of individuals in the scientific domain and ensure that the knowledge that emerges from interactions between scientists and the environment is reliable (McQuade and Butos 2003). Under normal circumstances, *scientific order* emerges from the *publication-citation-reputation* (PCR) processes of science (Butos and McQuade 2012). We adopt and extend F.A. Hayek's "non-standard" epistemology according to which *knowledge affords successful plan-based action* (Scheall 2016). We develop an *epistemic theory of social order* (Scheall 2015). We discuss the *distorting* effects on scientific knowledge and, thus, on scientific and social order more broadly, of government interference with the PCR processes of science. Finally, we use the history of the United States federal government's development of standardized dietary guidelines for American consumers and its concomitant interference in the PCR processes of nutritional science as an illustration of our synthesized theory of social order. We conclude that this interference contributed to social *disorder* in dietary science and beyond.

Keywords: social order; scientific order; F.A. Hayek; pretence of knowledge; discovery procedure; epistemic theory; dietary guidelines; fat hypothesis; carbohydrate hypothesis; Gary Taubes

JEL Codes: B31, D02, D72, H11, H51, I18

The Publication-Citation-Reputation (PCR) System as a Discovery Procedure

Our argument presupposes an economic model of the normal operation of science in the absence of government involvement or, more generally, in the absence of exogenous intervention.

According to this model (McQuade and Butos 2003, 2005; Butos and McQuade 2006, 2012; McQuade 2007), science is an institutionalized form of interaction between generally self-

interested individuals each of whom publishes their own work and, in turn, uses and cites the works of others. Under normal circumstances, the various incentives that individuals encounter when engaged in this mode of interaction, including the incentive provided by the norm that emphasizes the significance of empirical correspondence, promote the emergence of *scientific knowledge about the world*.

We adopt F.A. Hayek's "non-standard" epistemology according to which *knowledge affords successful plan-based action* (Scheall 2016). More exactly, Hayek's conception of knowledge implies that a subject *S* knows proposition *P* insofar as both *P* is presupposed (perhaps only tacitly) by a possible plan *L* of *S*'s to achieve goal *G* and, were *S* to act on *L*, *S* would achieve *G* as a result of *L*. *P* is an element of a "possible plan" when, were *S* to both have goal *G* and make plan *L* to achieve *G*, *L* would presuppose *P*.

Under normal circumstances, the scientific process manifests bidirectional feedback loops that constrain scientific activity to promote the emergence of scientific knowledge. A *publication* is evaluated by other scientists who might either adopt or criticize it in the form of a (positive or negative) *citation*, which feeds back upon the *reputation* of the original author, thereby affecting the reception of their subsequent publications, and their prospects for professional advancement and additional funding. This "PCR" system of institutionalized incentives ensures that what is ultimately accepted as scientific knowledge has been exposed to the daylight of rational scrutiny. The scientific knowledge that emerges from the PCR process has been subjected to, criticized from the perspective of, and deemed in acceptable conformance with, the assessment standards of the relevant scientific community. In short, it is through the PCR process that scientific error (or, worse yet, deliberate fraud) is identified and corrected.

However, where this process is either absent, superseded, or perverted, so too are its error-identification and -correction functions, and, other things equal, the knowledge that emerges from such science is less actionable than the knowledge that emerges from a normally-functioning PCR process. Any plan of action that presupposes a proposition the result of a negated or perverted PCR process is more prone to failure than any otherwise identical plan that presupposes no such proposition.

The only exception to this general rule would seem to be the case where the consequences of exogenous interference in the PCR process happen to mirror what would have occurred counterfactually in the absence of this interference. However, practically speaking, this qualification is of little relevance. There is no reason to think that, in the absence of an unfettered PCR process, anyone who would endeavor to exogenously mandate the contents of scientific knowledge would be able to imaginatively conjure the results of such a process. This would amount to predicting the contents of future science. Like market competition, science is what Hayek (2014a) called a *discovery procedure*: a process for the discovery of knowledge that does not exist in the absence of that process. Just as no one can know what the market price of a particular quantity of tin would be in the absence of market competition (see pp. 6-7 below), no one can know what would emerge from the PCR process without the PCR process.

Why Worry about the State?

A thorough investigation of the various ways in which the PCR process might be overridden or distorted by exogenous forces arising from either the public or private realms is surely in order. However, such an inquiry would take us too far afield in the present context. As anyone familiar with the infamous Lysenko affair in mid-century Soviet genetic science can attest, the

institutions of the state can be used to manipulate and, in the extreme, negate the normal operation of the PCR process. This is not to deny that there are circumstances in which *private* non-scientific actors might exert a similarly deleterious influence on the emergence of actionable scientific knowledge, but the most obvious cases should surely be handled first. Even ignoring Lysenko-like cases, government's role as primary funding source, principal external regulator, and prominent beneficiary of both scientific and technological research make it an obvious place to begin an investigation of the effects on science of the impingement of exogenous forces.

An Epistemic Theory of Social Order

The model of the emergence of scientific knowledge described above is a particular instance of a general *epistemic theory of social order* that, like our assumed epistemology, has its roots in the work of F.A. Hayek. In "Hayek's Epistemic Theory of Industrial Fluctuations," Scheall (2015) argues that an epistemic theory of economic order can be synthesized from various parts of Hayek's methodological writings (see Hayek 2014b, 2014c, and, especially, 2014d).¹ Our goal in the present section is to extend this theory of economic order into a more general epistemic theory of social order.

According to this epistemic theory, *social order* emerges endogenously in some domain – be it the economy, science, politics, or society itself – to the extent that the presuppositions of the plans (i.e., "beliefs") of the individuals active in this domain are internally and externally coordinated, i.e., mutually compatible and adequate to environmental circumstances. To say that the beliefs of different individuals are mutually compatible and adequate to environmental

¹ Hayek (1948a) offered an epistemic conception of economic *equilibrium* according to which "equilibrium exists to the extent that economically-relevant beliefs of individual market participants are mutually consistent and accurate with respect to the external facts" (Scheall 2015, p. 115). Hayek (1978b) subsequently preferred to describe this condition as one of economic *order*, which is the locution we adopt here.

circumstances is to say that, barring changes in either the relevant beliefs of other individuals or in environmental conditions, the individuals active in this domain will all be able to effectively implement their respective plans—it is to say, in other words, that these people *know* in our sense of knowledge. Social order obtains in some domain to the extent that the plans of the relevant individuals are actionable. Maximal order in some social domain is a condition in which the individuals active in that domain possess perfectly actionable knowledge of everything relevant to acting in that domain. It is a condition in which everyone can act without resistance from the environment, including other actors.

It follows from this conception that social *disorder* in some domain is also epistemic, i.e., a matter of the beliefs of the individuals active in the respective domain being uncoordinated either with each other or with other aspects of the environment. Social disorder in some domain constitutes a lack of knowledge in our sense: it means that the plans of some individuals in the relevant domain are bound to fail. Indeed, according to the epistemic theory, the marks of social disorder are disappointed expectations and failed plans, and the measure of disorder is the extent of disappointment and failure.

It should be obvious that the realization of perfect social order is unlikely if not practically impossible. Simply put, the data *do* change, and typically too rapidly to afford perfect coordination by anyone other than the omnipotent and all-knowing. However, our epistemic conception of social order does not assume or imply either that any individual ever knows anything entirely – actionability is a continuum, not a dichotomy – or that perfect social order in some domain is possible. That some expectations are disappointed is part and parcel of social life.

Given this epistemic theory of social order, the central problem of the social sciences is the discovery and understanding of the various mechanisms operative in different domains (and ultimately, across society itself) that either promote or impede the coordination of individual knowledge.

Hayek (2014c) argued that, with respect to the economy, a system of freely-adjusting prices is a necessary element in minimizing the disappointment of expectations. Hayek's (2014c, p. 99-100) famous "tin example," which illustrates the role of the price system in the coordination of knowledge in the economic realm and, thus, its central part in the maintenance of economic order, is always worth quoting at length:

"Fundamentally, in a system in which the knowledge of the relevant facts is dispersed among many people, prices can act to coordinate the separate actions of different people...It is worth contemplating for a moment a very simple and commonplace instance of the action of the price system to see what precisely it accomplishes. Assume that somewhere in the world a new opportunity for the use of some raw material, say, tin, has arisen, or that one of the sources of supply of tin has been eliminated. It does not matter for our purpose—and it is very significant that it does not matter—which of these two causes has made tin more scarce. All that the users of tin need to know is that some of the tin they used to consume is now more profitably employed elsewhere and that, in consequence, they must economize tin. There is no need for the great majority of them even to know where the more urgent need has arisen, or in favor of what other needs they ought to husband the supply. If only some of them know directly of the new demand, and switch resources over to it, and if the people who are aware of the new gap thus created in turn fill it from still other sources, the effect will rapidly spread throughout the whole

economic system and influence not only all the uses of tin but also those of its substitutes and the substitutes of these substitutes, the supply of all the things made of tin, and their substitutes, and so on; and all this without the great majority of those instrumental in bringing about these substitutions knowing anything at all about the original cause of these changes. The whole acts as one market, not because any of its members survey the whole field, but because their limited individual fields of vision sufficiently overlap so that through many intermediaries the relevant information is communicated to all. The mere fact that there is one price for any commodity—or rather that local prices are connected in a manner determined by the cost of transport, etc.—brings about the solution which (it is just conceptually possible) might have been arrived at by one single mind possessing all the information which is in fact dispersed among all the people involved in the process”² (emphasis added).

As we have argued above, the PCR system plays an analogous (but by no means identical) role in coordinating the beliefs of individuals active in the realm of science. An unfettered PCR system is the mechanism whereby the presuppositions of individual plans of action in the scientific domain come to be coordinated both with each other and with other facets of the external environment.

We can put a finer point on the operation of the PCR process in light of Hayek’s tin example. Imagine that a novel theoretical explanation of some phenomenon has been published. Just as price signals may or may not be acknowledged, and put to use, by market participants, “publication is a signal inviting response, which may or may not be recognized and acted on by

² The knowledge imparted by a freely-adapting system of prices is necessary but, of course, not by itself sufficient for individuals to adjust their economic plans to changing data.

other scientists” (McQuade and Butos 2003, p. 140). The interaction between the publishing scientist and the broader scientific community will serve to coordinate plans only if some among the latter find the explanation “useful in furthering one’s own work” (McQuade and Butos 2003, p. 140). Scientific custom typically requires that the use of someone else’s work be acknowledged in the form of an explicit citation to the author of the original publication. However, it is important to recognize that “[u]ses will vary, and so citations will vary in reputation-building effect. There will not only be use-citations, but a variety of forms, *including negative ones*. There will be impediments to the recognition of publication quality and potential usefulness, and there will be at least some basic criteria for a work even to be considered for possible use. In an environment in which most authors are acting as both contributors and users, there will be competition for access to better publication vehicles and also more direct confrontation involving both positive and negative citation” (McQuade and Butos 2003, p. 140; emphasis added).

Provided the PCR system is functioning without interference – that is, provided that scientists privy to a publication are not hindered from registering their impressions via the PCR system – then many members of the scientific community require no immediate first-person knowledge of the contents of the respective publication. If only some scientists know the contents of a publication and respond by citing it (positively or negatively), and if the scientists who receive this signal respond accordingly, by either accepting the new explanation, remaining neutral, or rejecting it in favor of some other, then, as Hayek noted, “the effect will rapidly spread throughout the whole...system and influence not only all the uses” and criticisms of the new explanation, but also those of its relatives and rivals. “The whole acts as one” scientific community, “not because any of its members survey the whole field, but because their limited

individual fields of vision sufficiently overlap so that through many intermediaries the relevant information is communicated to all.” Thereby, a consensus concerning scientific knowledge is either created, maintained, or destroyed: an unfettered PCR system “brings about the solution which (it is just conceptually possible) might have been arrived at by one single mind possessing all the information which is in fact dispersed among all the people involved in the process.”³

Our general theory of social order is *epistemic* in several different senses. It starts from the conception of economic order as well-coordinated knowledge, but it also builds upon Hayek’s epistemic explanation of the advent of economic disorder, i.e., as a consequence “of humans (typically, for Hayek, policymakers) acting *on the basis of knowledge that they don’t in fact possess* in such a way that interferes with the functioning of a freely-adjusting price system, and which thus hampers the operation of the tendency” for price changes to facilitate knowledge coordination (Scheall 2015, p. 104-105).

Economic policymakers falsely believe that they possess the knowledge necessary for effective macroeconomic management, i.e., their respective plans falsely presuppose that they know how to make explicit and deliberate policy so as to either maintain economic order or ameliorate economic disorder when it appears. In other words, economic disorder emerges not only because policymakers hold false beliefs about the economy. Such false beliefs would be relatively harmless were they never made the basis of policy. Rather, economic disorder emerges because policymakers both hold false beliefs about the economy and hold false beliefs about their false beliefs about the economy: “economic policymakers *don’t know that they don’t know* how to effectively administer economic [order]. Indeed, quite to the contrary, the policymaker

³ The standard caveats apply: the signals indicated by the PCR system are necessary but not sufficient for the emergence of scientific knowledge and, at best, serve this function imperfectly. Expectations are often disappointed in science, as elsewhere.

typically believes she can possess the knowledge both necessary and sufficient for effective political management of economic [order] – but she is wrong, or so Hayek argues” (Scheall 2015, p. 105).

Hayek (2014d) attributed this false belief in the adequacy of their own presuppositions about the economy – which he described as the economic policymaker’s “pretence of knowledge” – to the prevailing acceptance among policymakers of a “scientific” economic methodology that glorifies quantification and measurability, a (Keynesian) economic theory according to which the causally relevant variables are those that just happen to be measurable, and statistical techniques for the measurement of the phenomena represented by these variables. But, according to Hayek (2014d), quantification and measurability are less relevant than defenders of scientism assume, Keynesian theory is inadequate for policymaking purposes, and the available statistics fail to express the relevant causal phenomena. The consequences of the economic policymaker’s ignorance of their own ignorance are clear: “when policymakers pretend to possess the relevant economic knowledge and make policy on the basis of this pretence, their decisions typically impede, either directly or indirectly, the price system’s knowledge-coordinating function” (Scheall 2015, p. 109). According to the epistemic theory of industrial fluctuations, economic disorder is a condition in which the plans of individual economic actors are uncoordinated, most often, in virtue of policies made on the basis of a pretence of knowledge.⁴

Our explanation of social disorder as it emerges from the scientific domain will ultimately be similarly epistemic: acting on the basis of the false presupposition that they possess

⁴ This is the most common, but not necessarily the only possible, cause of economic fluctuations according to the epistemic theory. Anything that interferes with the knowledge-coordinating function of the price system will bear the same consequence.

the counterfactual knowledge of what would emerge from an unfettered PCR process without the possibility of recourse to such a process, politicians make policy that impedes (directly or indirectly) the PCR system's epistemic functions, namely, the coordination of knowledge and the identification, and correction, of scientific errors. The scientific knowledge, such as it is, that emerges from a politically-perverted PCR process is more likely to encounter resistance from the world, either from the beliefs of others or from further elements of the environment. For this reason, a plan that presupposes a proposition the result of a negated or perverted PCR process is more prone to failure than an otherwise identical plan that presupposes no such proposition. Social disorder – marked by the failure of plans that presuppose elements of this bastardized scientific knowledge – appears (or, more exactly, since something less than perfect order is the normal state of things, is aggravated) as a consequence.

Simply put, social disorder is a consequence of policy made on the basis of the policymaker's false belief in the adequacy of their knowledge for the purposes of maintaining social order. The knowledge that emerges from a politically-distorted PCR process is, for anyone who would presuppose it as part of their individual action plan, less actionable (less reliable) than the scientific knowledge that emerges from an unfettered PCR process. Just as economic disorder is a consequence of economically ignorant policies that override the epistemic function of the price system, such social disorder as emerges from the scientific domain flows from scientifically ignorant policies that override the epistemic function of the PCR system.

At the highest level of generality, our theory of social order predicts that, wherever there is exogenous interference with the epistemic mechanism that serves to coordinate knowledge in some social domain, failed plans tend to follow—disorder results. Other things equal, a plan of

action is more prone to fail if based on a presupposition which is the consequence of a superseded or distorted price system, or a superseded or distorted PCR system.

Confessing Our Own Ignorance

Hayek's explanation of how economic policymakers come to falsely believe that they possess the knowledge required of effective macroeconomic management – i.e., acceptance of the *mélange* of scientific methodology, Keynesian theory, and the adequacy of economic statistics – cannot be transferred to contexts in which politics impact the PCR process. This explanation works (as far as it does) only in the economic realm, and only as far as scientific and Keynesian presuppositions enter the plans of policymakers.⁵ Social disorder may typically be, to some degree or other, a consequence of policy made on the basis of a false belief in the adequacy of the policymaker's knowledge of the relevant phenomena, but a general explanation of how policymakers come to hold false beliefs, in particular, both about science and about their beliefs about science is not to be expected. It is comparatively easy to explain – which is not to say justify – the decisions of central bankers and other economic policymakers. We have a fair understanding of their methodology, preferred theories, and the data upon which they rely. It is not so simple to explain how any particular policy that impacts the PCR process emerges from political interactions, much less provide a *general* explanation of these phenomena.

This being said, Hayek's explanation of the economic policymaker's pretence of knowledge does provide some nebulous hints, which we are unable to better specify, of what such a general explanation might look like. The economic policymaker's mistaken beliefs about the economy follow from accepting an economic theory inadequate for their professed policy

⁵ We refer to Keynesianism here because that was Hayek's explicit target in "The Pretence of Knowledge" (2014d), but other, similarly scientific, forms of interventionism are subject to the same critique.

objectives, namely, the maintenance of economic order. Here we might note merely that, while there are competing theories as to the operation of the economic system and making policy on the basis of an inadequate theory can only fail for the purposes of economic order, the policymaker who would intervene upon science is altogether without theoretical guidance. There is no second-order science of science the results of which are obviously relevant to the maker of science policy in their ostensible goal of promoting social order through policies that impact science's PCR processes. Similarly, while there is a scientific methodology that tells the economic policymaker that applying, for example, Keynesian theory to economic statistics is adequate for the purposes of economic order, there is no explicit methodology that might assist the maker of science policy in the discovery of the theoretical and empirical knowledge required of the effective promotion of social order through science policy. In other words, the policymaker whose decisions impact the PCR process is typically even more epistemically enfeebled than the economic policymaker.

Yet, policies that impact the PCR process nevertheless get made. The question that we cannot answer in the current context is how, regardless of their patent ignorance of the results of an unfettered PCR process, policymakers not only fail to recognize their ignorance, but become convinced of their wisdom. It is one thing to say that such policymakers do not know what they are doing, it is quite another to explain why they act *despite this manifest ignorance*. Hayek's pretence of knowledge explanation of the economic policymaker's muddle, while it doesn't justify the relevant policy blunders, at least makes some sense of the policymaker's mistaken belief that their policies are justified. The science policymaker cannot be exculpated on similar grounds.

In order to keep the discussion as tractable as possible, we are deliberately ignoring a high-cardinality set of considerations almost certainly relevant to explaining this phenomenon. We are purposely setting aside all considerations specifically concerning public choice and any questions related to the motivations of, and incentives confronted, by policymakers. If science policy is made on the basis of considerations of what is best for the policymaker rather than in the interests of the public, then – to the extent policy-relevant presuppositions enter the plans of members of the public – this self-interested political behavior is the best explanation of the failure of these plans.⁶ However, accounting for the epistemic complications that contribute to the failure of plans based on presuppositions related to science policy while keeping an eye on the motivations of political actors would make this already complex analysis more convoluted still. For this reason, we have adopted the admittedly inadequate assumption (yet another unrepayable loan from Hayek) that policies which impact the PCR process, regardless of their consequences, are made on the basis of what policymakers honestly believe to be in the interests of their constituents.⁷

Moreover, there do seem to be examples (the case study below may be one such instance) in which, whatever the truth may be, policymakers at least *seem* to honestly believe they make science policy not in their own but in the public's interest. While this does not make issues of the

⁶ More generally, if policymakers act on the basis of motivations that are, in whole or in part, self-interested rather than constituent-minded, the epistemic problem of their ignorance of the requirements of making effective policies for their constituents is diminished. To the extent policymakers worry about their own interests, this latter, often intractable, quandary is replaced with a different and typically far simpler epistemic problem: the knowledge requirements of conducting policy in pursuit of personal, rather than public, goals. In many, perhaps most, political contexts, engaging in self-interested political acts is epistemically easier than realizing the policy goals of one's constituents. If this is right, then, other things equal, the relative epistemic complications of satisfying the wishes of the public should incentivize more self-interested policymaking. We should expect to find more self-interested political behavior where (*ceteris paribus*) the epistemic burden of making effective constituent-minded policy is comparatively heavy.

⁷ Political-economic analyses in the Austrian tradition with which Hayek is associated typically ignore considerations of the ethics of lawmakers. Hayek's (2014d) "Pretence of Knowledge" argument against the effectiveness of countercyclical policymaking assumes that policymakers aim always and only at effective macroeconomic management.

motivations of political actors irrelevant in such cases, it does provide some cover for our decision to ignore these complications in the present context.

We should probably also indicate what we take to be the positive value of the theory: it provides some clues where to find instances of both scientific disorder and social disorder caused by science-policymaking, namely, in cases of disappointed expectations among the users of scientific knowledge; it also provides a first approximation of an explanation of such cases that might be extended by integrating some of the public choice considerations we have opted to avoid herein.

However, we must confess yet one further limitation of our theory, namely, its limited testability: it is extremely difficult, if not plainly impossible, to observe either the presuppositions of an individual action plan or the plan itself, and, thus, its success or failure. However, there is an important sense in which its limited testability is not as damaging as might seem at first glance. Indeed, given its basis in the work of F.A. Hayek – who did more than anyone to remind its practitioners, critics, and false prophets of the very limited predictive possibilities (and, thus, policy relevance) of the social sciences – the model would, in effect, be self-undermining were it capable of predicting particular events with any precision. We are constrained to the prediction of patterns – generally, that any action plan that presupposes a proposition the result of overriding or perverting the operation of a relevant epistemic mechanism is more prone to fail than any otherwise identical plan that presupposes no such proposition – which is as it should be in the social sciences.

A Brief History of Federal Government Interference in Nutrition Science

To the extent that there was a medical-scientific consensus at the start of the 1970s concerning the dietary causes of coronary heart disease, obesity, and diabetes, it stressed their connection with the consumption of carbohydrates. The prevailing medical wisdom held that a diet including too many carbs, especially refined sugars and flours, contributed to tooth decay, as well as to weight gain and the various maladies associated with it. However, within a few short years, Americans were being advised to consume less of their calories in the form of *dietary fat*, especially, the *saturated fat* primarily found in red meat and animal-based foods.⁸ Since carbohydrates and dietary fat are nutritional rivals – for any given caloric load, consuming less of one must mean consuming more of the other – and since this novel advice encompassed a recommendation to at least maintain, if not decrease, their caloric intakes, the implication was that Americans should avoid red meat and indulge in bread, pasta, rice, fruits, and vegetables. This change was a consequence of the U.S federal government’s interference in the PCR processes typically operative in nutritional science.

The government placed the political cart before the scientific horse. In an ostensive effort to provide Americans with healthful dietary advice, the government short-circuited the PCR process from which a consensus on nutrition science would have otherwise been likely to eventually emerge.

There are several indications at the time of this writing that scientific as well as public opinion is circling back in favor of some variation of the view that carbohydrates are to be convicted (i.e., the “carbohydrate hypothesis”). This reversal is due in no small part to the work

⁸ Chemically speaking, dietary fat is dissimilar to the adipose tissue – also called “fat” – that composes not only oft-lamented beer bellies, paunches, and spare tires, but, more auspiciously, breasts and derrières. One should avoid any inference that there is something more than an unfortunate homonymic relationship between the two distinct concepts, especially, the inference that, since they share the same name, consumption of the first must be the cause of the second.

of Gary Taubes, an award-winning science journalist, whose remarkable 2007 book *Good Calories, Bad Calories: Fats, Carbs, and the Controversial Science of Diet and Health* documents in considerable technical detail the history of the last century (or so) of dietary science and the deleterious consequences of the government's pronouncement of a nutritional consensus without recourse to a daylighting PCR process.

Government officials are gradually acknowledging this reversal in the fortunes of the carbohydrate hypothesis. The eighth and most recent (2015) iteration of the U.S. Department of Agriculture's (USDA) *Dietary Guidelines for Americans* takes a few, albeit slight, steps back from the more trenchant injunctions on dietary fat proclaimed in past editions. If Taubes is right that there is essentially no evidentiary basis for the "fat hypothesis" that the dietary fat and, in particular, the saturated fat we consume contributes to ill health, there is reason to think this trend will continue.

We take no stance concerning the adequacy of any particular dietary hypothesis. Indeed, if our model of science is at all representative, then such a judgment requires the sort of scientific knowledge that can only emerge from an unfettered PCR process. But, as Taubes has shown and we will examine below, this is a case in which the PCR process was essentially countermanded. It thus elucidates how the social order and its scientific sub-order are impacted when the PCR process is superseded by exogenous forces.

Our model of social order bears interesting implications for the fact that the USDA treats *Dietary Guidelines* as a living document. Each subsequent modification implies that the knowledge upon which each of the previous iterations was based was inadequate to the professed goal of the *Guidelines*. To respond that the *Guidelines* require frequent updating, because what counts as scientific knowledge is constantly changing, is to beg the question. It is to assume,

rather than establish via argument, that the knowledge that enters into each restatement of the *Guidelines* is the product of processes that ensure as far as possible under prevailing circumstances its actionability. As a matter of historical fact, the knowledge that has figured in the various versions of the *Guidelines* has always been less a product of an open PCR process than of the unreliable caprices of political whim.

The crucial evidentiary coordinates in the advent of the hypothesis that consuming dietary fat, saturated fat, in particular, is the prime mover of chronic disease concerned two apparent phenomena both of which, if Taubes' analysis is sound, turned out later to be *merely* apparent.

First, there was the perception, widely accepted at mid-century, that Americans were in the midst of a heart disease "epidemic." Not coincidentally, Congress passed the clumsily-titled National Heart and Lung Institute National Heart Act in 1948, which created the National Heart Institute and the National Heart Council, precursors to today's National Heart, Lung, and Blood Institute (NHLBI) (one of the National Institutes of Health [NIH], itself an agency of a cabinet-level department, the U.S. Department of Health and Human Services). The U.S. federal government is the biggest player in heart disease research, and the NHLBI the main vehicle through which it funds research on heart disease and related disorders.⁹ In its first year, the Institute provided \$9 million in research funding; this number increased to \$54 million in 1960 and to \$2.5 billion in 2008.

Second, there was the belief that the increasing industrialization and urbanization of America in the latter half of the nineteenth and first part of the twentieth centuries coincided with

⁹ See Koppl (2002) on "big player" theory

a radical “nutritional transition” away from the widespread consumption of cereals, fruits, and vegetables (i.e., carbohydrates), toward increased consumption of animal foods (i.e., dietary fat).

It was but a short step from this purported correlation between the “nutritional transition” and the “epidemic” to the hypothesis that the first was the cause of the second, i.e., that the increased intake of dietary fat at the expense of carbohydrate in the first few decades of the twentieth century led to a rampant increase in the incidence of heart disease a few decades later.

However, according to Taubes (2007, pp. 5-13), the epidemic and the nutritional transition are both more myth than fact. The perception at mid-century of a heart disease epidemic was largely an artifact of new medical-screening techniques developed in the first few decades of the 20th century that led to a marked increase in the diagnosis, but not necessarily the *incidence*, of coronary heart disease (Taubes 2007, p. 6). Further complicating matters was the fact that, by the middle of the 20th century, early death from infectious diseases and inadequate nutrition had effectively been eradicated in the United States. Heart disease is common only among those over 50. The marked decrease in the number of premature deaths “left Americans living long enough to die of chronic diseases—in particular, cancer and heart disease” (Taubes 2007, p. 7). Another factor propagating the perception of a heart disease epidemic was the proliferation of new medical categories of heart disease, which led to more deaths *being classified as* due to heart disease (Taubes 2007, pp. 7-8).

Taubes (2007, pp. 9-13) argues that the evidence is similarly specious for the “changing-American-diet story” according to which Americans switched their eating habits *en masse* in the latter part of the nineteenth and first half of the twentieth centuries from allegedly healthy carbohydrates to dangerous animal foods. The main cheerleader for both the changing-American-diet story and the hypothesis that fat is responsible for fattening, diabetes, and heart

disease was cardiologist Dr. Ancel Keys of the Laboratory of Physiological Medicine at the University of Minnesota: “Keys formulated the argument initially based on [U.S. Department of Agriculture (USDA)] statistics suggesting that Americans at the turn of the century were eating 25 percent more starches and cereals, 25 percent less fats, and 20 percent less meat than they would be in the 1950s and later...[these] statistics, however, were based on guesses, not reliable evidence” (Taubes 2007, p. 10). The USDA began collecting the relevant data in the early 1920s, but, through 1940, only periodically and only on a selection of food groups. “Only with World War II looming did USDA researchers estimate what Americans had been eating back to 1909, on the basis of the limited data available.” (Taubes 2007, p. 11). Data on homegrown foods were especially scarce. A number of eighteenth- and nineteenth-century observers of American habits noted a pronounced fondness for the consumption of red meat: “One French account from 1793...estimated that Americans ate eight times as much meat as bread. By one USDA estimate, the typical American was eating 178 pounds of meat annually in the 1830s, forty to sixty pounds more than was reportedly being eaten a century later” (Taubes 2007, p. 11). Moreover, many carbohydrate foods – pasta and rice, in particular – were considered strange and exotic imports through the first several decades of the 20th century. There are reasons to believe that meat consumption did indeed fall for a time in the decade of the 1900s due, first, to a temporary lag in livestock production relative to the growth of the American population, and, second, to the shocking claims of Upton Sinclair’s fictional treatment of meatpacking in *The Jungle*, published in 1906, which “caused meat sales in the United States to drop by half...All of this suggests that the grain-dominated American diet of 1909 [the base year for the changing-American-diet story], if real, may have been a temporary deviation from the norm” (Taubes 2007, p. 12). Finally, the evidence after 1940, when the USDA began collecting and publishing these statistics quarterly,

indicate that “the increase in total fat consumption, to which Ancel Keys and others attributed the ‘epidemic’ of heart disease, paralleled not only *increased* consumption of vegetables and citrus fruit, but of vegetable fats, which were considered heart-healthy, and a *decreased* consumption of animal fats” (Taubes 2007, p. 13).

Keys played the central role, not only in publishing research that supported the fat hypothesis, but in convincing the American Heart Association (AHA) to adopt the hypothesis as its central operating assumption, and – perhaps most importantly – in acting as an uncompromising advocate for the hypothesis both among the relevant communities of researchers and in the public eye.

Keys’ hypothesis consisted of two sub-theses. First, there was the thesis that the consumption of fat – originally total fat, but later, especially saturated fat – increases the levels of total blood (or serum) cholesterol. Second, there was the thesis that, since cholesterol is the main component of the atherosclerotic plaques associated with coronary heart disease (CHD), it is total cholesterol that induces the formation of these plaques. Thus, Keys invoked a purported mechanism from eating saturated fat to increased total cholesterol in the bloodstream, to the formation of atherosclerotic plaques and, thus, to heart disease, elevated blood pressure and hypertension, myocardial infarction (heart attack), stroke, etc.

Keys based his hypothesis on data gathered from a number of countries that, at the time, were just emerging from the catastrophe of World War II. Given that postwar conditions included both famine and food rationing, Keys’ data may not have constituted the best evidence for or against any hypothesis concerning the health effects of different diet regimens under normal conditions. What might have appeared to be a correlation between fat consumption and heart disease could have been caused by any number of other hardships related to the war.

Moreover, whether deliberately or coincidentally, the evidence upon which Keys based his hypothesis was highly selective: “Keys had chosen only six countries for his comparison though data were available for twenty-two countries. When all twenty-two were included in the analysis, the apparent link between fat and heart disease vanished” (Taubes 2007, p. 18).

In 1957, the AHA insisted that the available evidence failed to support Keys’ hypothesis of a causal relation between dietary fat and heart disease. However, though no new evidence had appeared in the meantime, three years later an *ad hoc* AHA committee issued a report, co-authored by Keys, that reversed the Association’s earlier skepticism. From this point forward, the AHA advocated increasingly stringent recommendations to reduce the consumption of dietary fat and none of its members was a more indefatigable crusader than the Minnesota cardiologist.

Keys’ bum rush of the media, American public, and his scientific peers, not to mention the AHA’s increasingly restrictive recommendations concerning dietary fat, contributed to the growing acceptance of the fat-cholesterol hypothesis over the third quarter of the 20th century. However, the evidence for the hypothesis remained stubbornly recalcitrant. Had it not been for George McGovern’s Senate Select Committee on Nutrition and Human Needs, which both reified the alleged connection between fat, cholesterol, and chronic disease as nutritional-medical wisdom and enshrined the suggestion to reduce fat consumption in favor of carbohydrates as the main “dietary goal” of all Americans, then either controversy would have continued in perpetuity or a normal PCR process would have eventually generated something approaching an unalloyed consensus concerning dietary-scientific knowledge. Either way, without the McGovern committee’s stamp of official government approval, Keys’ hypothesis would likely have remained popular only among a smaller part of the population rather than the nutritional lodestar it became for the majority of Americans.

The report that the McGovern committee issued on January 14, 1977, the first *Dietary Goals for the United States*, was written “almost single-handedly” by a scientific novice, committee staffer Nick Mottern, a “former labor reporter [...and...] researcher for a consumer-products newsletter” (Taubes 2007, p. 46). Mottern managed to avoid confronting the refractory evidence concerning Keys’ hypothesis by ignoring it in favor of a near-exclusive emphasis on the research of a single investigator, Harvard’s Mark Hegsted, an unreserved believer in the connection between fat consumption, cholesterol, and heart disease.¹⁰ *Dietary Goals* marked “the first time that any government institution (as opposed to private groups like the AHA) had told Americans they could improve their health by eating less fat. In so doing, *Dietary Goals* sparked a chain reaction of dietary advice from government agencies and the press that reverberates still, and the document itself became gospel. It is hard to overstate its impact. *Dietary Goals* took a grab bag of ambiguous studies and speculation, acknowledged that the claims were scientifically contentious, and then *officially bestowed on one interpretation the aura of established fact*” (Taubes 2007, pp. 44-45; emphasis added).

The resulting controversy over *Dietary Goals* led McGovern to hold eight subsequent hearings. However, the skeptical “experts were sandwiched between representatives from the dairy, egg, and cattle industries, who also vigorously opposed the guidelines, for obvious reasons. This juxtaposition served to taint the legitimacy of the scientific criticisms” (Taubes 2007, p. 47). A revised version of *Dietary Goals* was soon published that “included a ten-page preface that attempted to justify the committee’s dietary recommendations in light of the uproar

¹⁰ It would later be discovered that at least one of Hegsted’s research projects the results of which unhesitatingly endorsed the fat-cholesterol hypothesis, a 1965 literature review published in the *New England Journal of Medicine*, had been funded (without disclosure of said funding) by the Sugar Research Foundation (SRF), and that the “SRF set the review’s objective, contributed articles for inclusion, and received drafts” (Kearns, Schmidt, and Glantz 2016, E1).

that had followed” (Taubes 2007, p. 48). The revised report acknowledged the presence of disagreement among nutrition scientists and the belief among some that its recommendations could lead to harm, but the committee deemed such destructive consequences unlikely. “McGovern’s *Dietary Goals* had turned the dietary-fat controversy into a political issue rather than a scientific one, and Keys and his hypothesis were the beneficiaries” (Taubes 2007, p. 48). The consensus that has marked the last forty years of dietary science was gerrymandered in large part by the McGovern committee and the subsequent piling-on of other government agencies.

The USDA entered the fray in the spring of 1977. Assistant Secretary of Agriculture Carol Foreman, a former consumer advocate, “believed it was incumbent on the USDA to turn McGovern’s recommendations into official government policy” (Taubes 2007, 48). Once again, like Mottern before her, Foreman would have no truck with scientific controversy.

After drafting an agreement with the Food and Nutrition Board of the National Academy of Sciences (NAS) to appraise the McGovern committee’s dietary recommendations, Foreman backed out when it became clear that members of the Academy’s brass were skeptical of *Dietary Goals*.¹¹ Indeed, the Board soon released its own guidelines in the form of *Toward Healthful Diets*, which advised Americans simply to “watch their weight...everything else, dietary fat included, would take care of itself” (Taubes 2007, p. 50). Foreman encountered similar skepticism similar at the National Institutes of Health and the Food and Drug Administration, where the McGovern committee’s report was (at the time) deemed less scientific than political.

Foreman eventually found the Surgeon General’s Office sufficiently pliant and representatives of the two agencies set about writing an official set of government-certified dietary guidelines. The USDA was represented by the first head of the Department’s Human

¹¹ The Food and Nutrition Board is responsible for determining Recommended Dietary Allowances.

Nutrition Center, Mark Hegsted. Suffice it to say that the first USDA *Dietary Guidelines for Americans*, written by Hegsted and the Surgeon General's J. Michael McGinnis, and released in 1980, made Keys' fat-cholesterol hypothesis the quasi-official scientific position and the recommendation to reduce fat intake in favor of carbohydrates the official dietary guideline of the U.S. federal government. This was the effective end of the policy debate.

It was little surprise when the American public soon accepted the government's low-fat dogma, which was quite harmonious with environmental concerns popular in the 1970s, especially the budding vegetarian and anti-meat movements. The American food industry soon flooded the market with reduced-fat alternatives, such as low-saturated fat margarines and vegetable oils, and products engineered to conform to the requirements of America's new dietary paradigm.

Meanwhile, the science concerning Keys' hypothesis of a connection between fat, cholesterol, and heart disease was left to catch up. Four smaller trials attempted to establish such a connection within different populations. None succeeded (Taubes 2007, p. 53). Further studies aimed less to test any particular hypothesis than to reconcile the evidence with the government's official dietary guidelines. However, much of this research was waylaid by the unexpected discovery of what appeared to be a link between *low* cholesterol and cancer (Taubes 2007, p. 54). This link was eventually erased from scientific concern when two NHLBI workshops in the early 1980s concluded it merely a mark of the uniquely cancer-prone; for everyone else, the Institute insisted, the connection between high cholesterol and coronary heart disease held. The results of two large trials conducted by the NHLBI in the 1970s were similarly ambiguous for any connection between fat, cholesterol, and heart disease, yet the Institute soon committed to a

massive public-marketing campaign designed to convince Americans to lower cholesterol through reduced fat consumption (Taubes 2007, p. 58).

Despite the stubborn evidence, the acquiescence of the medical and scientific communities was soon obtained, largely by ignoring anyone who raised a fuss against Keys' low-fat hypothesis. The NIH held a "consensus conference" in December 1985 that ended the scientific debate. Three skeptics of Keys' hypothesis presented testimony (out of twenty speakers), but their message was effectively silenced in the conference report. As Taubes (2007, p. 59) puts the point: "The NIH Consensus Conference officially gave the appearance of unanimity where no unanimity existed."

This brief history illustrates a case in which a theretofore undecided scientific controversy was determined by political means rather than through an unfettered PCR process. Between the McGovern committee's original pronouncement, the USDA's subsequent declaration of the scientific and nutritional wisdom of low-fat diets, and the NIH's gerrymandered consensus conference, the government effectively delegated the contents of dietary-scientific knowledge, such as it was for several decades to follow.

The Consequences of the Federal Government's Interference in Nutrition Science

According to our epistemic theory of social order, the government's pronouncement of a nutritional consensus without recourse to the error-identification and -correction functions of a daylighting PCR process made the resulting scientific knowledge, such as it was, less actionable than would have otherwise been the case. Thus, if our epistemic theory of social order is at all correct, any plan of action that presupposed a proposition the result of this politically-perverted

process was more prone to fail than an otherwise identical plan that presupposed no such proposition.

We can imagine this knowledge-by-fiat figuring among the presuppositions of many different kinds of plans, but, given our concern to test the epistemic theory of social order against the evidence as far as it will permit, two kinds of plan would seem most pertinent, namely, plans of *scientific* action and plans of (for lack of a better word) *dietary* action.

According to our theory, a plan that presupposed that Keys' hypothesis would pass a particular experimental test without encountering falsifying evidence was more likely to fail than a plan that did not presuppose the scientific adequacy of the fat hypothesis. Simply put, if you expected the government's quasi-official scientific stance to pass your experimental test, you were likely to be disappointed.

Similarly, a plan that presupposed the nutritional adequacy of low-fat diets, e.g., a plan which presupposed that a low-fat diet would contribute to weight loss, and minimize the risks of heart disease and diabetes, was more likely to fail than a plan that did not presuppose the nutritional adequacy of low-fat diets. If you expected the government's official dietary guidelines to make you svelte and lower your risk of heart disease, you were likely to be disappointed.

Evidence concerning plans of scientific action and social disorder in dietary science

Those in control of research monies are able to influence scientific inquiry in pre-approved ways. Naturally, the more prominent the funding source within the broader world of science funding, the greater these effects. Related to this, the more important the source of research monies, the greater the effects on the reputations of those awarded (or denied) such funds (Butos and

McQuade 2012, 22). However, privately-funded research is often seen as dubious work done merely to support the funder's interests and the reputations of scientists who accept private research monies is commensurately discounted.¹²

Given the government's position as biggest player in the funding of medical-scientific research, the PCR model predicts that its influence on the direction of this research and the reputations of researchers is uniquely powerful. Given the incentive provided by government funders to clinical investigators and medical researchers to engage in research consistent with the government's priorities, in the wake of federal acceptance of the cholesterol hypothesis, we would expect to have observed relatively more scientists adopting plans that presupposed the adequacy of Keys' fat-cholesterol hypothesis and relatively fewer adopting plans that did not. According to Taubes (2007, pp. 51-52), this is precisely what occurred:

“Scientists were believed to be free of conflicts if their only source of funding was a federal agency, but all nutritionists knew if their research failed to support the government position on a particular subject, the funding would go instead to someone whose research did...The NIH expert panels that decide funding represent the orthodoxy and will tend to perceive research interpreted in a contrarian manner as unworthy of funding. David Kritchevsky, a member of the Food and Nutrition Board when it released *Toward Healthful Diets*, put it this way: ‘The U.S. government is as big of a pusher as industry. If you say what the government says, then it's okay. If you say something that

¹² Taubes (2007, Ch. 3) discusses the apparent double-standard of treating privately-funded research as ethically dubious and public funding as the epitome of scientific probity, as if government funders were utterly without bias or incapable of misdirecting the scientific enterprise.

isn't what the government says, or that may be parallel to what industry says, that makes you suspect'."

Not only should more researchers have developed plans based on the government's presupposition of the scientific adequacy of Keys' fat-cholesterol hypothesis, but we would expect to have observed the relative failure of experimental tests that presupposed Keys' fat-cholesterol hypothesis. The available evidence, though too extensive to be comprehensively reviewed here (the interested reader is directed, in particular, to Chapter Two and the whole of Part Two of Taubes' *Good Calories, Bad Calories*), appears at least consistent with this pattern prediction as well.

Recall the two sub-theses of Keys' fat-cholesterol hypothesis. There is the first sub-thesis that eating fat, especially, saturated fat, increases levels of total cholesterol in the bloodstream; and there is the second sub-thesis according to which total cholesterol induces the formation of the atherosclerotic plaques so intimately associated with coronary heart disease. The evidence from repeated tests of the second sub-thesis have definitively undermined the notion that there is any connection between *total* cholesterol and CHD. Indeed, this is now accepted even among those who cling to a modified version of Keys' fat hypothesis.

It had already been recognized by the 1950s that cholesterol is one of several different fat-like substances that circulate in the bloodstream, collectively referred to as *lipids*, including free fatty acids and *triglycerides* (the molecular form of fat in the bloodstream; three fatty acids bound by a glycerol molecule). *Lipoproteins* act as a vehicle carrying cholesterol and triglycerides through the bloodstream. Though it was recognized that these other kinds of lipids and their conveyance vehicles might figure in heart disease, either in isolation or in conjunction

with cholesterol, Keys' hypothesis held the advantage that total cholesterol levels were measurable on the basis of existing technology, while the other varieties of lipids and lipoproteins were not. However, as medical science continued to progress and measurements of the relevant disaggregated magnitudes were made possible (*possible* but, importantly, not *common* or *standard*: the only device capable of such measurements at the time was an ultracentrifuge housed at the University of California, Berkeley) the true complexity of the relationships between different elements in the bloodstream and heart disease became apparent.

Lipoproteins are classified in terms of their density. Even in the early days of cholesterol science, it was recognized that *low-density lipoproteins* (LDL) are more numerous in atherosclerosis sufferers and especially prominent in diabetics, and that LDL and total cholesterol do not vary together (Taubes 2007, pp. 154-155). The one researcher with access to the Berkeley ultracentrifuge at the time, medical physicist John Gofman, who performed the majority of the relevant research, "described the measurement of total cholesterol as a 'false and highly dangerous guide' to the effect of diet on heart disease. Total-cholesterol measurements tell us nothing about the status of VLDL [*very low-density lipoproteins*, which also figure in the CHD process] and LDL" (Taubes 2007, p. 156). Perhaps unfortunately, Gofman's research on the components of the human bloodstream and their relation to atherosclerosis and heart disease was left fallow when he departed the field in the early 1960s.¹³

Other researchers picked up the triglyceride thread around the same time. Margaret Albrink, then a young physician at Yale University, reported in 1961 – soon after the American

¹³ Incidentally, a further implication of Gofman's research was that Keys' first sub-thesis – the alleged connection between the consumption of fat, especially, saturated fat, and serum cholesterol – was, at best, tenuous. VLDL was also recognized as critical to the heart disease process and "[t]hrough Gofman's studies had demonstrated that the amount of LDL in blood can indeed be elevated by the consumption of saturated fat, it was *carbohydrates*, he reported, that elevated VLDL...and only by restricting carbohydrates could VLDL be lowered" (Taubes 2007, p. 156).

Heart Association pronounced Keys' hypothesis its official doctrine – that triglycerides raise the risk of heart disease and that low-fat diets raise triglyceride levels (Taubes 2007, p. 159).

Albrink's research was largely ignored by the media and she was personally attacked by Keys' supporters. By the early 1970s, Albrink's interpretation of the evidence had been confirmed independently" by researchers in Pennsylvania, Stockholm, and Seattle (Taubes 2007, p. 159).

Despite these falsifications of Keys' fat-cholesterol hypothesis, the government, via the National Institutes of Health ("effectively the only source of funding for this research in the United States" [Taubes 2007, p. 160]), had by this time committed too many resources to testing the effects of total cholesterol – which, as compared to lipoproteins, triglycerides, etc., remained easy to measure – to encourage research that might further undermine Keys' fat-cholesterol hypothesis (Taubes 2007, p. 160). Unfortunately for the NIH, these large-scale tests of the connection between fat consumption, total cholesterol levels, and heart disease all failed to support Keys (Taubes 2007, p. 161ff). It was not until the late 1980s that the importance of the intimate connection between low levels of *high-density lipoproteins* (HDL, which carry what we now call "good" cholesterol), high triglycerides, obesity, and diabetes would be recognized, "but by then the heart disease researchers [were] committed to the recommendations of a national low-fat, high-carbohydrate diet" (Taubes 2007, p. 163).

So, the evidence seems consistent with the implications of the PCR model and the epistemic theory of social order, i.e., both a relative increase in research dedicated to investigating the scientific position approved by the federal government and a tendency for these plans to fail, other things equal.

The epistemic theory of social order would seem to bear a further consequence of the government's quasi-scientific position: so long as this position remained effectively unaltered

and the government retained its role as biggest player in scientific funding, we would expect to observe failed plans of scientific action being modified so as to retain the presupposition of the adequacy of Keys' hypothesis. That is, we would expect to observe scientists making *ad hoc* modifications to their existing presuppositions in the wake of the failure of plans based on these presuppositions, e.g., by either altering other of their presuppositions except those relevant to Keys' hypothesis or changing these latter presuppositions in the least destructive manner possible.

Again, the evidence of the effects of the government's adoption of a quasi-official scientific stance seems to support this implication of the epistemic theory of social order. Very few researchers responded to the experimental failure of the second sub-thesis of Keys' hypothesis by removing it from the presuppositions of their plans.

“Those who believed that dietary fat caused heart disease had always preferentially interpreted their data in the light of that hypothesis. Now they no longer felt obliged to test any hypothesis, let alone Keys'. Rather, they seemed to consider their obligations to be that of 'reconciling [their] study findings with current programs of prevention', which meant the now official government recommendations. Moreover, [government-financed studies of Keys' hypothesis] were expensive, and one way to justify the expense was to generate evidence that supported the official advice to avoid fat. If the evidence didn't support the recommendations, then the task was to interpret it so that it did” (Taubes 2007, pp. 53-54).

The relevant presuppositions were modified so that the basic substance of the hypothesis – if not the details of its original formulation in terms of *total* cholesterol – could be retained in light of the falsifying evidence. The new bogeyman became the connection between consumption of saturated fat and elevated levels of LDL, and the cholesterol they carry through the bloodstream (Taubes 2007, pp. 163-5): “the American Heart Association and the proponents of Keys’ hypothesis now shifted the focus of scientific discussions from the benefits of lowering total cholesterol to the benefits of lowering LDL cholesterol...Making LDL the ‘bad cholesterol’ oversimplified the science considerably, but it managed to salvage two decades’ worth of research” (Taubes 2007, p. 166).

Evidence concerning plans of dietary action and social disorder in cardiac health

As we have seen, with the advent of McGovern’s *Dietary Guidelines* in 1977, the federal government took on the role of official nutritionist and dietician to the American public. For the most part, over the next several decades, Americans faithfully adopted the federally-sanctioned advice to reduce consumption of dietary fat, saturated fat in particular. “[M]ost reliable evidence suggests that Americans *have* indeed made a conscious effort to eat less fat, and particularly less saturated fat, since the 1960s. According to the USDA, we have been eating less red meat, fewer eggs, and more poultry and fish; our average fat intake has dropped from 45 percent of total calories to less than 35 percent” (Taubes 2007, p. *xvii*). In the terms of the present essay, it would seem that many Americans adopted plans of dietary action predicated on a presupposition of the adequacy of the government’s nutritional advice.

Alas, though Americans dutifully followed the purported wisdom of the original McGovern committee report and its successors, social disorder in the domain of diet and

nutrition followed: the incidence rates of obesity, cardiac heart disease, and diabetes – the very conditions the politicians ostensibly meant to ameliorate – either rose or failed to fall over this time: “there is little evidence that the incidence of heart disease has declined, as would be expected if eating less fat made a difference. This was the conclusion, for instance, of a ten-year study of heart disease mortality published in *The New England Journal of Medicine* in 1998” (Taubes 2007, p. xviii). “Indeed, if the last few decades were considered a test of the fat-cholesterol hypotheses of heart disease, the observation that the incidence of heart disease has not noticeably decreased could serve in any functioning scientific environment as compelling evidence that the hypothesis is wrong” (Taubes 2007, p. xviii).

According to the NIH’s National Institute of Diabetes and Digestive and Kidney Diseases, since the early 1960s, the incidence of obesity among adults over the age of 20 has more than doubled, increasing from 13.4 to 35.7 percent. The figures for younger Americans are little better: the incidence of obesity among children and adolescents rose during the 1980s and 1990s, stabilizing in the last decade at 17 percent.¹⁴ These changes corresponded with a decrease in the percentage of fat (and saturated fat) in the American diet, and an uptick in the consumption of carbohydrates (Taubes 2007, pp. 232-233).

“Diabetes rates have increased apace” (Taubes 2007, xviii). According to one study of a population of middle-aged adults in a single Massachusetts community, the incidence of type-2 (i.e., adult-onset) diabetes doubled in just under three decades following the McGovern committee report (Fox et al. 2006). Another study showed that, though the incidence of undiagnosed diabetes remained stable between 1988 and 2002, the prevalence of diagnosed

¹⁴ <https://www.niddk.nih.gov/health-information/health-statistics/Pages/overweight-obesity-statistics.aspx>

diabetes “rose significantly” – over 27 percent – from 5.1 percent to 6.5 percent of the population (Cowie et al 2006).

According to the epistemic theory of social order, these dispiriting facts are all, at least in the first instance, consequences of political interference with the epistemic mechanism that ensures the actionability of the accepted results of nutritional medicine. The theory implies the pattern prediction that the knowledge that emerged from this politically-manipulated PCR process was less actionable than would have been the case otherwise. Americans who adopted the adequacy of the advice associated with Keys’ dietary-fat hypothesis as a presupposition of their plans to avoid obesity, heart disease, and diabetes, were mostly disappointed. The government’s perversion of the processes of dietary science that, under normal circumstances, serve to ensure the actionability of its results, contributed to social disorder.

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