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RESEARCH ARTICLE

Social and scientific disorder as epistemic phenomena, or the consequences of government dietary guidelines

Scott Scheall*, William N. Butos and Thomas McQuade

College of Integrative Sciences and Arts, Arizona State University Polytechnic Campus, Mesa, AZ, USA, Department of Economics, Trinity College, Hartford, CT, USA and Independent Scholar, Honolulu, HI, USA
*Corresponding author. Email: scott.scheall@asu.edu

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Abstract

We begin with a process-oriented model of science according to which signals concerning *scientific reputation* serve both to coordinate the plans of individuals in the scientific domain and to ensure that the knowledge that emerges from interactions between scientists and the environment is reliable. Under normal circumstances, *scientific order* emerges from the *publication–citation–reputation* (PCR) process of science. We adopt and extend F. A. Hayek's epistemology according to which *knowledge affords successful plan-based action* and we employ this in the development of an *epistemic theory of social order*. We propose that external interferences with the PCR process have *distorting* effects on scientific knowledge and, thus, on scientific and social order more broadly. We support this claim by describing the history of the US federal government's development of standardized dietary guidelines for American consumers and its concomitant interference in the PCR process of nutritional science. We conclude that this interference contributed to social *dis*order 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

1. The publication–citation–reputation (PCR) system as a discovery procedure

Our argument presupposes a process-oriented 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, 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 empirical correspondence, promote the emergence of *scientific knowledge about the world*.[1]

Normally, 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 publications and prospects for professional advancement. This 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

[1]For a full exposition of this model in terms of "adaptive systems theory," including citations to the extensive literature on systems theory in general and science as a social system in particular, see McQuade and Butos (2003, 2005), Butos and McQuade (2006, 2012), and McQuade (2007). For connections to the literature on the philosophy of science, see McQuade (2010).

the relevant scientific community. It is through the PCR process that scientific error (or, worse yet, deliberate fraud) is identified and corrected.[2]

However, where this process is either absent, superseded, or perverted, so too are its error identification and correction functions, and, other things being 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 resulting from a negated or perverted PCR process is more prone to failure than any otherwise identical plan that presupposes no such proposition.

As antecedents for this model of the system of science, we particularly cite R. K. Merton, D. L. Hull, M. Polanyi, and F. A. Hayek. Merton (1996: 333), summarizing a lifetime of work in the sociology of science, observed:

> the institutionalized practice of citation and references in the sphere of learning is not a trivial matter … [for] these are in truth central to the incentive system and an underlying sense of distributive justice that do much to energize the development of knowledge.

Hull (1988: 309), in a detailed study of two competing groups of research biologists, underlined with first-hand observation the seriousness with which the researchers regard the reputational effects of the citation of publications: "Individual scientists want credit for their publications, as much credit as possible. … [They] readily acknowledge that they crave for recognition[.]" Polanyi (1962; 1967: 54–55) conceived of science as a dynamic order of freely coordinating individuals who are "in fact cooperating as members of a closely knit organization" by virtue of "the adjustment of the efforts of each to the hitherto achieved results of the others," and argued passionately (against much opposition) for science to be free from external direction, pointing out the inherent contradiction between such direction and the process of discovery. Hayek (1960: 388–389) who, contemporaneously with Polanyi, saw science as a spontaneous order, also argued that the advance of knowledge requires that scientists have the freedom to pursue their work autonomously within the evolved conventions of science and unencumbered by "political interference."[3] However, the most important antecedent for our model was Hayek's (2014b) exposition of the epistemic characteristics of the market's price system, which inspired the identification of its analog, the PCR process, in science.

2. An epistemic theory of social order

This model of the emergence of scientific knowledge 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. Scheall (2015) argues that an epistemic theory of economic order can be synthesized from various parts of Hayek's methodological writings (see Hayek, 2014a, 2014b, and, especially, 2014c).[4] Our goal 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. This is to say that, barring changes in either the relevant beliefs of other individuals or in environmental conditions, the

[2]There is no guarantee that the identification and correction of scientific errors occurs quickly. The rate at which specific scientific errors are found and fixed is likely to vary inversely with the complexity of, and difficulty of testing, the phenomena under investigation.

[3]For a detailed analysis and critique of both Hayek's and Polanyi's treatments of spontaneous order, including their characterizations of science, see Butos and McQuade (2017).

[4]Hayek (2014a) 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: 115). Hayek (2014c) subsequently preferred to describe this condition as one of economic *order*, which is the locution we adopt here.

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individuals active in this domain will all be able to effectively implement their respective plans – in other words, that these people *know*, in our sense of knowledge (Scheall, 2016). Social order obtains in some domain to the extent that the plans of the relevant individuals are actionable.

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. 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 practically impossible. Simply put, the data *do* change, and typically too rapidly to afford perfect coordination by anyone. 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 of mechanisms operative in society that either promote or impede the coordination of individual knowledge.

Hayek (2014b) 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 (2014b: 99–100) famous "tin example" 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. Hayek describes how changes in supply and demand, although known first hand by only a few, spread through the economic system, affecting the prices of substitutes and complements, without downstream market participants needing to know more than the prices relevant to their local plans.[5]

The PCR system plays an analogous (but by no means identical) role in coordinating the beliefs of scientists. 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 describe the operation of the PCR process in terms mirroring 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 other scientists" (McQuade and Butos, 2003: 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 their own work. 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: 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

[5]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.

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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 (2014b: 99–100) 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.[6]

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: 104–105). 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 being equal, a plan of action is more prone to fail if based on a presupposition that is the consequence of a superseded or distorted price system, or a superseded or distorted PCR system.

A thorough investigation of the various ways in which the PCR process of science 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, the 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.

3. Policy-induced social disorder

Economic policymakers falsely believe that they possess the knowledge necessary to make explicit and deliberate policy so as to either maintain economic order or ameliorate economic disorder when it appears. By themselves, such false beliefs would be relatively harmless were they never made the basis of policy. But economic disorder emerges because policymakers hold false beliefs both about the economy and 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 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: 105)

The consequences of economic policymakers' ignorance of their own ignorance are clear: "when policymakers pretend to possess the relevant economic knowledge and make policy on the basis of this pretense, their decisions typically impede, either directly or indirectly, the price system's knowledge-

[6]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.

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coordinating function" (Scheall, 2015: 109). 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 pretense of knowledge.[7]

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 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 that resulted from a negated or perverted PCR process is more prone to failure than an otherwise identical plan that presupposes no such proposition.

Social disorder, then, is a consequence of policy made on the basis of policymakers' 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.

4. Acknowledging our own ignorance

To keep the discussion as tractable as possible, we are deliberately ignoring many considerations that are almost certainly relevant to explaining why policies that interfere with the PCR process get enacted despite policymakers' manifest ignorance of the knowledge required to realize the goals of the relevant policies. 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 that 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.[8] 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 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 public interest.[9] Moreover, there are examples (like the case study below) in which this does *seem* to be the case. While this does not make issues of the motivations of political actors irrelevant in such cases, it does provide some cover for our decision to ignore these complications in the present context.

[7]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.

[8]To the extent that policymakers worry about their own interests, we have 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 altruistic policy goals. Other things being equal, the relative epistemic complications of satisfying the wishes of the public should incent more self-interested policymaking. We should expect to find more self-interested political behavior where (*ceteris paribus*) the epistemic burden of making effective public-minded policy is comparatively heavy.

[9]Political-economic analyses in the Austrian tradition with which Hayek is associated typically ignore considerations of the ethics of lawmakers. Hayek's (2014c) "Pretence of Knowledge" argument against the effectiveness of countercyclical policymaking assumes that policymakers aim always and only at effective macroeconomic management. Also see Koppl (2018).

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The positive value of the theory is that 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.

We acknowledge the limited testability of the theory: 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 Hayek – who did more than anyone to point out the very limited predictive possibilities (and, thus, limited policy relevance) of the social sciences – the model would be self-undermining were it capable of predicting particular events with any precision. We are constrained to the prediction of patterns, which is as it should be in the social sciences.

The specific pattern prediction that we are making here is that external interference in the operation of the PCR process of science compromises the reliability of the knowledge produced by that system, thereby increasing disorder, both in the science itself and in the greater society. We are not making a claim of optimality for the unhampered process, but we are noting that, like the price mechanism in its domain, it harnesses individual incentives and individual knowledge to produce knowledge of the system's environment that is useful, but inevitably limited and contingent, and that there are no credible alternative institutional arrangements for producing that type of knowledge. In describing the involvement of government in nutrition science, we are presenting a detailed specific illustration supporting our pattern prediction.[10]

5. An overview of trends 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 (CHD), obesity, and diabetes, it stressed their connection with the consumption of carbohydrates (Butos and McQuade, 2012). 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 fewer calories in the form of *dietary fat*, especially the *saturated fat* primarily found in red meat and animal-based foods. 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, 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 US federal government's interference in the PCR process of 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 that scientific as well as public opinion is circling back in favor of the view that carbohydrates are to be convicted (i.e. the "carbohydrate hypothesis"). Government officials are gradually acknowledging this reversal in the fortunes of the carbohydrate hypothesis. The eighth and most recent (2015) iteration of the US 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 our argument is sound, the deleterious consequences of the government's pronouncement of a nutritional consensus without recourse to a daylighting PCR process have made this turnabout prudent. Recent scientific research suggests that there is no decisive evidentiary basis

[10]Butos and McQuade (2012) complement the case study described here with an analysis showing how the PCR process was distorted and bypassed by government actors in the science of immunology.

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for the "fat hypothesis" that the dietary fat (in particular, the saturated fat) we consume contributes to ill health.

We take no stance concerning the adequacy of any particular dietary hypothesis. Indeed, we maintain that such a judgment requires the sort of scientific knowledge that can only emerge from an unfettered PCR process. But we do claim that the ascendance of the fat hypothesis is a case in which the PCR process was essentially countermanded by government mandate.

Our model of social order bears interesting implications for the fact that the USDA treats *Guidelines* as a living document. Each subsequent modification implies that the knowledge upon which each of the previous iterations was based was inadequate. To respond that this is because what counts as scientific knowledge is constantly changing is to beg the question, for it assumes, rather than establishes via argument, that the knowledge that enters into each restatement of *Guidelines* is the product of a process that ensures as far as possible its actionability. As a matter of historical fact, the knowledge that has figured in the various versions of *Guidelines* has always been less a product of an open PCR process than of the unreliable caprices of political whim.

6. The "fat hypothesis" and government interference in nutrition science

The fat hypothesis emerged mid-century in the context of two widely accepted social beliefs. First, there was the perception that Americans were in the midst of a heart disease "epidemic." Not coincidentally, Congress in 1948 created the National Heart Institute and the National Heart Council, precursors to today's National Heart, Lung, and Blood Institute (NHLBI). The US federal government is the biggest player in heart disease research, and the NHLBI is the main vehicle through which it funds research on this and related disorders.[11] Second, there was the belief that the increasing industrialization and urbanization of America in the latter half of the 19th and first part of the 20th centuries coincided with 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 carbohydrates in the first few decades of the 20th century led to a rampant increase in the incidence of CHD a few decades later.

However, the alleged epidemic and the nutritional transition are both more myth than fact. Taubes (2007: 5–13) argues that the perception at mid-century of a heart disease epidemic was largely an artifact of new medical-screening techniques that led to a marked increase in the diagnosis, but not necessarily the *incidence*, of CHD. 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. CHD 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: 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: 7–8). The evidence is similarly suspect for the "changing-American-diet story," according to which Americans switched their eating habits *en masse* in the latter part of the 19th and first half of the 20th 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 CHD was cardiologist Ancel Keys of the Laboratory of

[11]See Koppl (2002) on "big player" theory. The NHLBI provided $9 million in research funding in its first year; this increased to $54 million in 1960 and to $2.5 billion in 2008. The NHLBI budget authority for 2016–2018 has been $3.1, $3.2, and $2.5 billion, respectively. See https://www.nih.gov/about-nih/what-we-do/nih-almanac/national-heart-lung-blood-institute-nhlbi (accessed August 29, 2018).

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Physiological Medicine at the University of Minnesota.[12] Keys initially based his argument on USDA data that Americans at the turn of the century had consumed 25% more starches and cereals, 25% less fat, and 20% less meat than in the 1950s and afterward. These claims were not as definitive as Keys imagined. The first systematic reckoning by the USDA of food consumption in the US was not published until 1949.[13] Basic statistics of food production in that report were generated by the Census of Agriculture taken every five years, while intermediate years were estimated from sample data. The estimates of meat consumption only referred to interstate commerce of meat production. Given the absence of data for "intermediate years" and non-federally inspected meat, the consumption data were precarious. Interestingly, the data for foodstuffs not subject to interstate commerce, and especially for meat consumption, beginning in 1909 through 1943, were not only used by Keys but have been regularly reported in more recent data analyses. Taubes (2007: 11) notes:

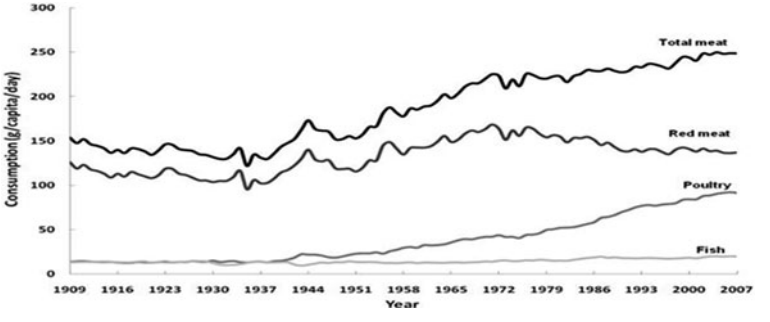
> Only with World War II looming did USDA researchers estimate what Americans had been eating back to 1909, on the basis of the limited data available … By one USDA estimate, the typical American was eating 178 pounds of meat annually in the 1830s, forty to sixty pounds more than was reportedly being eaten a century later.

The 1942 Report noted the "basic data for computation of estimates of retail slaughter up to 1943 were even more incomplete" than the "year-to-year changes shown by sample data" (Taubes, 2007: 19).[14]

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 a persistent advocate for the hypothesis

[12]Research on the association of diet with cardiovascular diseases took hold in the late 1940s. During World War II Keys conducted the so-called "starvation study" on 36 conscientious objector volunteers to examine the effects of dietary factors on cardiovascular and other body functions that were formerly believed to be relatively fixed (see Kalm and Semba, 2005). Also, the ongoing long-term "Framingham Study" commenced in 1948 with over 5,000 adult subjects to study the epidemiology of cardiovascular diseases (see Mahmood *et al.*, 2013; Oppenheimer, 2010). The "Seven Countries" study by Keys and associates originated as a prospectus by Keys in 1948 and began in 1956 with a yearly grant of $200,000 from the US Public Health Service.

[13]This chart from Daniel *et al.* (2011) replicates the data for the years 1909–1942 from the USDA Report of 1942. Also see O. C. Wells (1942), a major contributor to the 1942 USDA Report, who notes that the figure for 1909–1916 was an estimate of an eight-year average. He adds: "I cannot argue for absolute accuracy of these estimates" (463).



[14]There are reasons to believe that meat consumption did indeed fall for a time in the first decades 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 Taubes (2007) notes "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." 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: 12–13).

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both among the relevant communities of researchers and in the public eye. Keys' (1953) hypothesis consisted of two sub-theses. First, that the consumption of fat – originally total fat, but later, especially saturated fat – increases the levels of total blood (or serum) cholesterol. Second, that, since cholesterol is the main component of the atherosclerotic plaques associated with 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, heart attack, stroke, etc.

Keys (1970) based his hypothesis on data for 1958 through 1964 from seven countries which, at the time, were still emerging from the catastrophe of World War II.[15] 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, the evidence upon which Keys based his hypothesis appeared selective and led to severe criticism of his methodology. Before the field work even began, Yerushalmey and Hilleboe (1957) pointed out that the strength of the association observed by Keys depended on the countries selected for the study. If countries other than those included by Keys had been selected, the observed association would have been weak.

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 Keys.

Keys' persuasiveness with the media, the American public, and his scientific peers, together with the AHA's increasingly restrictive recommendations concerning dietary fat, fed 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 elevated the alleged connection between fat, cholesterol, and chronic disease to the level of 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. Without the McGovern committee's stamp of official government approval, Keys' hypothesis would likely have remained popular only among a small part of the population rather than becoming a nutritional lodestar for the majority of Americans.

This is the critical point. Although private actors were involved, the distortion of the PCR process in this case could not have occurred without the intervention of government actors. Such a combination of private and public opportunism in science is reminiscent of "crony capitalism" and could be labeled "crony science."[16]

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 novitiate, committee staffer Nick Mottern, a "former labor reporter [… and …] researcher for a consumer-products newsletter"

[15]See https://www.sevencountriesstudy.com/about-the-study/investigators/ancel-keys/ (accessed August 29, 2018).

[16]It is undeniable that corporations and other private interests often fund research in the hope of getting the results they prefer. Both proponents of the fat hypothesis and of the competing sugar hypothesis (see Yudkin, 1988) were promoted and opposed by relevant corporate interests. But so long as the interests at play are more or less in competition – so long as neither has a monopoly on funding nor is especially "big" relative to the other – there is no reason to think that one side can out-fund the other and permanently (or even for very long) capture the PCR process. Only a government, and in this case the US government in particular, is big enough to declare an "official" consensus and enforce it through the funding process, regulatory prerogatives, and its bully pulpit, thereby short-circuiting the PCR process. The US government led the way in preempting the science, but others, including the UK, France, and others, soon followed.

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(Taubes, 2007: 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.[17] *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: 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: 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 that had followed" (Taubes, 2007: 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. In effect, *Dietary Goals* transformed the dietary fat controversy from a scientific into a political issue. The consensus that has marked the last 40 years of dietary science was gerrymandered in large part by the McGovern committee and the subsequent piling-on of other government agencies.

Although the Department of Health, Education, and Welfare and the National Institutes of Health (NIH) considered dietary research to fall under their respective purviews, Congress in 1977 designated the USDA lead agency. In entering the fray, 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, perhaps because of the massive amount of research money at stake. As William Broad (1979: 6) noted, "the fighting began in 1977, and the story of its origin elucidates the politics of an evolving area of research."

After drafting an agreement with the Food and Nutrition Board of the National Academy of Sciences 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 the McGovern committee's report. Indeed, the Board soon released a report highly critical of *Dietary Goals* titled *Toward Healthful Diets*:

> The Food and Nutrition Board is concerned about the flood of dietary recommendations currently being made to the American public … These recommendations, which have come from various agencies in government, voluntary health groups, consumer advocates, and health-food interests, often lack a sound scientific foundation, and some are contradictory to one another. In an effort to reduce the confusion in the mind of the public that has resulted from these many conflicting recommendations, the Board has prepared the following statement … The Board considers it scientifically unsound to make single, all-inclusive recommendations to the public regarding intakes of energy, protein, fat, cholesterol, carbohydrate, fiber, and sodium. Needs for energy and essential nutrients vary with age, sex, physiological state, hereditary factors, physical activity, and the state of health. (NRC 1980: 2–3)

[17]It would later be discovered that at least one of Hegsted's research projects, a 1965 literature review published in the *New England Journal of Medicine*, the results of which unhesitatingly endorsed the fat-cholesterol hypothesis, 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 *et al.*, 2016: E1).

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Foreman encountered similar skepticism at the NIH and the Food and Drug Administration, where the McGovern committee's report was (at the time) deemed less scientific than political (Taubes, 2007: 50). But she 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 Nutrition Center, Mark Hegsted. 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 US federal government. This was the effective end of the policy debate.

It was not surprising that 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 movement. 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 CHD was left to catch up. Four smaller trials attempted to establish such a connection within different populations. None succeeded (Taubes, 2007: 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: 53–54). 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 CHD held. The results of two large trials conducted by the NHLBI in the 1970s were similarly ambiguous for any connection, yet the Institute soon committed to a massive public-marketing campaign designed to convince Americans to lower cholesterol through reduced fat consumption (Taubes, 2007: 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' hypothesis. The NIH held a "consensus conference" in December 1985 that ended the scientific debate. Three skeptics of Keys' hypothesis presented testimony (out of 20 speakers), but their message was effectively silenced in the conference report. As Taubes (2007: 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 an 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 regulated the contents of dietary-scientific knowledge, such as it was, for several decades to follow.[18]

7. The consequences of the 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 less actionable than would have otherwise been the case. Thus, according to our epistemic theory of social order, any plan of action that presupposed a proposition following from this politically perverted process was more prone to fail than an otherwise identical plan that presupposed no such proposition.

[18]Nissen (2016: 558–560) of the Cleveland Clinic argues that the history of US dietary guidelines is an "evidence-free zone." He contends that the absence of randomized controlled clinical trials has "left dietary advice to cult-like advocates" and that "decades of dogma" regarding dietary cholesterol has led to erroneous dietary advice. "We reduced dietary fat but binged on carbohydrates and became increasingly obese."

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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 that 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 preapproved 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.[19]

Given the government's position as biggest player in the funding of medical-scientific research, the PCR model predicts that its influence on both 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: 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 US 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 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, seems consistent with this pattern prediction as well.[20]

[19]Taubes (2007: Chapter 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.

[20]See Chapter 2 and Part Two in Taubes (2007).

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Recall the two sub-theses of Keys' fat-cholesterol hypothesis: first, eating fat, especially, saturated fat, increases levels of total cholesterol in the bloodstream; and second, total cholesterol induces the formation of the atherosclerotic plaques associated with CHD. 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' 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). *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 CHD, 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 technology progressed and measurements of the relevant disaggregated magnitudes became possible,[21] 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: 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, found that:

> attention to the blood cholesterol alone provides only the most naïve approach to the problem of clinical management of myocardial infarction. Indeed, if preventive and therapeutic measures are considered only in the light of the blood cholesterol level serious errors of management will eventuate and many patients will be denied effective therapy … Since it is the level of certain lipoprotein classes, and not the serum cholesterol level, that is of primary importance in arteriosclerosis and in myocardial infarction, the measurement of the serum cholesterol level alone can serve as a false and highly dangerous guide. (Gofman, 1958: 272, 281)

Perhaps unfortunately, Gofman's research on the components of the human bloodstream and their relation to heart disease was left fallow when he departed the field in the early 1960s.[22]

Other researchers picked up the triglyceride thread around the same time. Soon after the AHA pronounced Keys' hypothesis its official doctrine, Margaret Albrink, a young physician at Yale University, reported in 1961 that triglycerides raise the risk of heart disease and that low-fat diets raise triglyceride levels.[23] Albrink's research was largely ignored by the media and she was personally attacked by Keys' supporters. But by the early 1970s, Albrink's interpretation of the evidence had been confirmed independently by several other researchers (Taubes, 2007: 159).[24]

Despite these falsifications of Keys' fat-cholesterol hypothesis, the government, via the NIH ("effectively the only source of funding for this research in the United States" [Taubes, 2007: 160]), had by this time committed too many resources to testing the effects of total cholesterol – which

[21] *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.

[22] 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]hough 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: 156).

[23] See Albrink *et al.* (1961), Albrink (1962).

[24] See Kuo (1967) and the *JAMA* editorials (1967) independently supporting Kuo's findings. Among other corroborating findings, see Carlson and Bottiger (1972).

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remained easy to measure as compared to lipoproteins, triglycerides, etc. – to encourage research that might further undermine Keys' fat-cholesterol hypothesis (Taubes, 2007: 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: 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: 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 being 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 the 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 their presuppositions other than 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. Few researchers responded to the experimental failure of the second sub-thesis of Keys' hypothesis by removing it from the presuppositions of their plans (Taubes, 2007: 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: 163–165): the AHA 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: 166).

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

As we have seen, with the advent of *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% of total calories to less than 35%. (Taubes, 2007: xvii)

In the terms of the present paper, it would seem that many Americans adopted plans of dietary action predicated on a presupposition of the adequacy of the government's nutritional advice.

But the plans went astray and social disorder in the domain of diet and nutrition followed: the incidence rates of obesity, CHD, 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 … Indeed, if the last

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> 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: 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%. 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%.[25] These changes coincided 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: 232–233). 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 diabetes "rose significantly" – over 27% – from 5.1% to 6.5% of the population (Cowie *et al.* 2006).

Conclusion

According to the epistemic theory of social order, these trends are 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. We conclude that the available evidence concerning the consequences of this government interference for both science and the nutritional health of Americans is consistent with the pattern predictions implied by the epistemic theory of social order.[26]

[25]See https://www.niddk.nih.gov/health-information/health-statistics/Pages/overweight-obesity-statistics.aspx (accessed August 31, 2018).

[26]Our analysis of the effects of government's involvement in nutrition science and the social disorder that has resulted therefrom has concentrated exclusively on the American case. Yet government-generated nutrition guidelines are the rule around the world. The United Nations' FAO (2016) reports that over 100 countries (predominantly in Europe, Asia and the Pacific, North America, the Caribbean, and South America) have produced dietary guidelines. However, only seven African countries have nutrition guidelines, and several North African countries (including Libya, Egypt), Iraq, Saudi Arabia, Russia, North Korea do not have any. The guidelines among the more developed countries are roughly consistent, but with some variations (e.g. France has several different food guidelines), while those of lesser developed countries typically emphasize caloric intake and disease prevention. The Nordic countries were probably the first promoters of government dietary guidelines (see Mozaffarian, 2018). In 1935 Sweden created the Swedish Nutrition Council, an advisory scientific body, under the auspices of the Health Department to address issues of public nutrition. Similar institutions were created by Denmark in 1935, Finland in 1936, and Norway in 1938. In 1938 the Swedish Population Commission, led by Gunnar Myrdal, published a report on the "nutrition issue" as part of the emerging welfare state and social and educational programs (Kjaernes, 2003). Despite Nordic nutrition science advances, which highlighted balanced nutrition, exercise, reduced fat intake, after World War II nutrition policies among the Nordic countries started to reveal fractures due to welfare policy differences and conflicts between the private sector and nutrition policies, especially in the dairy industry. By the late 1980s, however, most of the Nordic countries settled on political decisions that made nutritional goals integral to health policy. In contrast to the Nordic countries and the US, the UK lagged in providing government nutrition guidelines, but substantial interest in the links between diet and health preceded those guidelines. Various official reports (numbering nine) on diet, nutrition, and health from 1979 through 1994 were issued by the Department of Health (see Hunt *et al.*, 1995). In 1994 the UK's food guide was launched as "The Balance of Good Health," a product of the Department of Health, Ministry of Agriculture, and the Health Education Authority, that was subsequently published in 1994 as the National Food Guide (NFG). In its final form the NFG recommendations argued for a low-fat and low-sugar diet; the Guide advised that "breads, other cereals and potatoes" and "fruit and vegetables" should comprise each about 30% in the diet, equal percentages of "meats, fish, and alternatives" and "milk and dairy" at about 15% each and the remainder of 10% "fatty sugary foods"

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(see Hunt *et al.*, 1995). In the Guide's 2015 iteration ("The Eatwell Guide") starchy carbohydrates comprise 38% of the diet, fruits and vegetables 40%, dairy 8%, meats and fish plus eggs and legumes 12% and oils and spreads 1% (Buttriss, 2016).

It would indeed be a useful project to document, for all of these countries, the influences on local science and possible social disorder resulting from the promulgation of nutritional guidelines that short-circuit the scientific process, but we do not have in hand sufficient data or access to relevant sources for a comprehensive study. However, while we might expect the results of such a study to differ in detail country by country, we see no reason why they would show experiences very different from that in the US, where the data are clear and compelling.

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