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Dietary Policy, Controversy and Proof: Doing Something versus Waiting for the Definitive Evidence

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Dietary Policy, Controversy and Proof: Doing Something versus Waiting for the Definitive Evidence

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Dietary Policy, Controversy and Proof: "Doing Something" versus Waiting for the "Definitive Evidence"

Controversies over diet and its relationship to health are never out of the headlines for long. At the beginning of the 21st century, a heated debate is in progress about the causes of the obesity epidemic sweeping the United States and some other nations (1, 2). The percentage of the population classified as obese in the US jumped from 14.5 % in 1971 to 30.9% in 2000 (3). Many commentators attribute the increase to simple overeating. In their view, Americans, spurred on by advertising and ever-increasing serving sizes, are ignoring dietary advice and consuming more calories than they are expending (1, 2, 4). Others maintain that the problem is more complex. A few researchers claim that recent dietary advice (5, 6), which advocates restricting fats and increasing carbohydrate intake, is *contributing* to weight gain. According to them, dieters following stricter versions of this regime are upsetting the insulin-based physiological system that regulates blood sugar, appetite and fat metabolism, with the result that they gain weight and often develop diabetes (1, 2). Alongside this recent skepticism about the wisdom of fat avoidance is a renewed interest on the part of mainstream medicine in the controversial Atkins diet – a high fat, low carbohydrate regime that was for decades relegated to the realm of quackery. Baffled and frustrated by obese but starving patients who fail to lose weight, some orthodox physicians have begun testing Atkins' claims (7, 8) much to the chagrin of those who find any questioning of the anti-fat message a danger to health (9).

As these recent controversies illustrate, discussions about diet, disease and health take place in a highly politicized arena. In an ideal world, scientific research would settle controversies, as the opposing parties would be forced to agree on hard and indisputable facts. However, within the vast body of knowledge related to diet and disease, there are many contradictory claims and experimental results that are open to conflicting interpretations. Nonetheless, because of intense public, commercial and

political interest, health policy makers are under considerable pressure to come up with answers and advice. Although policy decisions draw on and lend legitimacy to some interpretations of the "evidence", other interpretations are often possible, and controversies erupt again. This chapter traces the development of one view of the healthy diet – a diet that is low in fats, and high in carbohydrates. Although it became mainstream and widely accepted during the second half of the 20th century, this diet was the subject of considerable controversy. It was primarily developed and promoted as a response to a disease that was rife in the decades after World War II – coronary heart disease (CHD). The rise of the low fat diet is therefore closely bound up with attempts to "do something" about CHD at a population-wide level.

The chapter covers the period from the 1940s, when medical and lay awareness of the increasing incidence of CHD began to grow, to 1985, the year the National Cholesterol Education Program (NCEP) began its widespread and concerted effort to sell the anti-fat, anti-cholesterol message to the nation. This campaign marked a victory for advocates of fat reduction over skeptics who, for decades, continued to question the efficacy of low fat diets as a means of preventing disease. To make sense of the scientific knowledge, its policy ramifications, and the controversy as a whole, it is useful to divide the knowledge linking fats, cholesterol and heart disease into three separate but related hypotheses. These are (1) that higher serum cholesterol levels are associated in some way with an increased risk of CHD, (2) that serum cholesterol levels can be reduced by modifying the fat and cholesterol content of the diet, and (3) that a cholesterol-lowering diet will reduce the risk of developing cardiovascular disease. By the mid-1960s, scientists had established the validity of hypotheses 1 and 2. However, hypothesis 3 remained problematic. While scientists struggled to test its validity, public, commercial and political interest in the link between diet and disease intensified, stimulating the creation of policy before the issue was resolved to everyone's satisfaction. The history of dietary policy in post-war

America provides a fascinating insight into the way science, culture, economics and politics intertwine with policy-making in the field of public health.

Putting Cardiovascular Diseases on the Public Health & Medical Research Agenda

Although rates of CHD in the US increased markedly during the first half of the 20th century (10, see also the accompanying chapter), there was initially little public awareness of the disease. Lay people and medical organizations were still primarily interested in treating and preventing infectious diseases. In 1945, for example, a report on fund-raising by voluntary health agencies stated that the funds raised that year represented $94 for each case of infantile paralysis, $22 for each case of tuberculosis, $8 for cancer, and 3 cents for each case of heart disease (11). Over the next 10 years, a group of cardiologists and medical research lobbyists worked diligently to raise the profile of cardiovascular diseases (CVD), or diseases of the circulation. Their efforts were closely bound with another highly successful campaign, led by wealthy philanthropists Mary and Albert Lasker, to increase funding for research into chronic diseases in general. The Laskers and their allies fervently believed that well funded research would soon produce cures for CVD and cancer. In 1948, the Federal government rewarded their efforts by establishing the National Heart Institute (NHI) (12-14). Funds for research rose steadily. Between 1950 and 1967, annual funding escalated from $16 million to $164 million (14).

The American Heart Association (AHA) was also involved in these efforts. In 1946 the Association, until then a private, professional body, voted in favor of becoming a national voluntary health agency, which allowed it to expand its public education and fund-raising activities (15). It established a National Heart Week, and used newspapers, radio, magazines, Hollywood stars and community organizations to publicize heart disease and the need for more money for research. It distributed heart-shaped collection boxes to drugstores emblazoned with the slogan “Open Your Heart...Give to Fight

the Heart Diseases, America's Number One Killer" (16, 17). The 1949 campaign raised $2,850,000, and the 1950 campaign raised a further $4 million (18, 19). The Association estimated that during its first 12 years as a voluntary health agency, it channeled nearly $50 million into research (20).

Before 1950, scientific knowledge about the causes of CVD was indeed scanty. Early experiments with rabbits suggested some kind of link between diet and atherosclerosis. However, many scientists believed that these findings could not be extrapolated to humans (21, 22). During the 1930s and 1940s, scientists found higher levels of serum cholesterol in humans with various diseases of the kidneys and circulation (23, 24). Hints also began to emerge that serum cholesterol levels could be decreased by manipulating the diet. In the late 1940s patients with high blood pressure were often treated with a very strict rice-fruit diet, which contained no cholesterol and virtually no fat. Researchers observed that patients on this diet experienced substantial decreases in serum cholesterol levels (25, 26). They conducted experiments to investigate the comparative effects of vegetable and animal fats on serum cholesterol. However, results were confusing and contradictory (27, 28). They would remain so until the second half of the 1950s, when scientists redefined the problem in terms of fat saturation.

Early publicity reflected the uncertainty about the causes of cardiovascular diseases. In 1947, *Better Homes and Gardens* published an article titled "The 100% American Way to Die", which informed readers of the growing incidence of several forms of cardiovascular disease, including coronary thrombosis, stroke, hypertension and rheumatic heart disease (29). It canvassed several theories of causation, including "high-cholesterol foods". The section on cholesterol ended with a caution: "It's only a theory yet, nothing that should justify tampering with a good diet *unless your doctor himself tells you to*" [emphasis in original]. Instead, the advice given was vague and general, and based on an assumption that cardiovascular diseases were caused by some mixture of stress, overeating and lack of

exercise: - “calm down and get out into the sun” readers were advised, “instead of rushing and worrying and getting flabby and stuffing your paunch with the $3 dinner” (29).

Early Heart Disease Epidemiology – Ancel Keys & The Framingham Heart Study

Of the many projects funded during the medical research boom of the 1950s, only a few stand out as having played pivotal roles in the development of knowledge about CHD and its causes. In 1951 Ancel Keys met a fellow physiologist from Naples who told him that the disease was not a problem in his home city. This stimulated Keys to set up a comparative study of the heart disease rates and diets of various socio-economic groups in Naples, Madrid and Minnesota. He found very little heart disease among the poorer populations of Naples and Madrid and attributed this to their diets, which contained little meat and few dairy products (27). Keys presented these findings at two international congresses in 1952. In a memoir published in 1990, he wrote that the findings “were politely received but few were convinced that diet had anything to do with coronary heart disease” (30). At the time, dietary policies aimed to prevent deficiencies, not to deter excess. As milk, meat and eggs supplied protein, iron and valuable vitamins, they were considered healthy – not potentially pathogenic (31).

The large gap between Keys’ ideas and those of the majority of his colleagues is evident in a symposium on atherosclerosis published in the AHA journal *Circulation* in 1952 (32). In the symposium, Louis Katz, an eminent atherosclerosis researcher, said that he would only prescribe a low-fat, low-cholesterol diet for obese patients or for those who had already had *two or more* heart attacks because: “prohibitions should not be carelessly advocated until such time as it is clearly revealed that the prohibition has a great chance of being beneficial to the patient” (32). Keys, on the other hand, boldly asserted that “if mankind stopped eating eggs, dairy products, meats and all visible fats”,

atherosclerosis would become “very rare”. His diet for atheroschlerosis patients included skim milk, lean meat or fish and “a boiled or poached whole egg for Sunday” (32). It would be at least another decade or two before other doctors would consider such a strict diet to be appropriate for heart disease patients.

In contrast to his later seven countries study (33), Keys’ early investigations were retrospective – that is, they compared variables at a single point in time, seeking to correlate possible causes with disease *after* the disease had manifested itself. Critics found it easy to question these studies because there was no guarantee that the putatively causal variables (for example, high fat diet, high cholesterol levels) chronologically preceded the putative outcome (CHD). To overcome these problems, epidemiologists devised prospective studies. In these, they chose persons, randomly or otherwise, and characterized them according to various criteria. A research team then periodically investigated the incidence of new disease among subjects, and determined causes of death in those who died between examinations. Epidemiologists argue that these studies are more rigorous because the characteristics of the persons under observation are measured *before* the disease appears.

In post-war America, epidemiologists set up many prospective studies (34-39). The most famous and influential was – and is - the Framingham Heart Study. Begun in 1947, it was taken over by the newly formed NHI in 1949. The original aim was to study the incidence of heart disease over time in a defined community - the town of Framingham, about 20 miles from Boston. However, the new NHI directors decided that it could also be used to search for “constitutional and conditioning factors” (later known as “risk factors”) associated with the development of the disease. Accordingly, they expanded the range of personal and medical information to be collected (40). The original cohort consisted of 2,282 men and 2,846 women aged 30-59 years; all were subjected to a thorough medical examination and questioning, then recalled every two years for re-examination. Causes of death were sought for

those who died between examinations (41). In 2004, the study was still collecting data on the remaining members of the original cohort, as well as their spouses and offspring.

Data from the first four years of the Framingham study did not appear until 1957 (42), and the risk factors that are now so well known did not become clear until the early 1960s. In the meantime, there was a great deal more publicity about CHD and its causes, much of it fuelled by the work of Ancel Keys.

More Publicity and the First Battles over Policy

In the mid-1950s some sectors of the popular press became much less circumspect in their reports about the links between diet and heart disease. A 1954 *Newsweek* article reporting on a recent conference was titled “Fat's the Villain”. Much of the article was taken up with descriptions of the retrospective country comparisons made by Keys and his colleagues. The author summarized the work with the claim: “A world survey of recently discovered facts shows that cardiovascular troubles are most common in countries where there is the most fat in the diet” (43). In December 1955 the *Reader's Digest* also published a flattering article describing Keys' research, and cautiously advocated dietary change (44). The news-worthiness of CHD received a further boost in September 1955 when President Eisenhower suffered a heart attack (45).

While publicity about heart disease and its possible links to fat consumption increased, the question of the effects of different types of fats on serum cholesterol levels remained unresolved. This issue was of great interest to sectors of the food industry, some of which were linked to scientific research through the Nutrition Foundation, an industry-funded research body (46). In exchange for a membership fee, companies gained access to the latest knowledge about nutrition. Although the direction of research

was decided by a Scientific Advisory Committee, it was open to influence by industry representatives. During the 1950s, member companies offered to donate an extra million dollars over and above their usual payments if the Foundation would fund an intensive research effort into the effects of different types of fats on serum cholesterol (46). The Committee agreed. It was during this time that scientists managed to sort out the confusion by reframing the problem as one of fat saturation. The findings were consistent and non-controversial. Despite some variation among persons, the lowest cholesterol levels were obtained by ingesting oils with high concentrations of polyunsaturated fatty acids (47). Soon after these studies were published, the first polyunsaturated margarine appeared, and vegetable oil manufacturers began to claim that their products could prevent heart disease (48, 49).

The new findings quickly found their way into the popular press and stimulated another spate of articles on diet, cholesterol and heart disease. By this time, Keys was no longer alone in advocating dietary change. In 1956 *Time* published this bold claim by New York nutritionist Norman Jolliffe: "No prudent person who has had, or wishes to avoid, coronary heart disease should eat a high-fat diet of the type consumed by most Americans.... Stress and strain, physical indolence, obesity, luxury living or tobacco play but a minor role" (50). Jolliffe and colleagues persuaded more than 1,000 men in New York to join an "Anti-Coronary Club" in which they would abide by his dietary rules (51). He hoped that within five years he would have enough data to prove the salutary effects of diets low in saturated fats (50).

The public advocacy of low-fat diets troubled some of the more conservative members of the scientific profession. In May 1957, two researchers went public with a message of caution published in a *Newsweek* cover story titled "The Diet Mania - Do Fats Really Kill?". The article began: "Currently the dieters are swarming to a new fad - the anti-cholesterol or low-fat diet - though scientists have not yet reached any considerable measure of agreement as to whether such a diet does anything but harm"

(52). The author asserted that cholesterol was “the mysterious compound around which one of medicine's most heated controversies is now raging” and contrasted the views of Keys and Jolliffe with those of Frederick Stare (a Harvard University nutritionist), and Irvine Page (a former president of the AHA). Page was particularly critical of Keys' country comparison studies. Foreign CHD statistics were unreliable, he said, because of “poor methods of reporting, understaffed health departments, and dubious autopsy proceedings”. He claimed that there was not yet enough evidence to justify “wholesale tinkering with the American diet” (52).

Page and Stare were also the leading authors of the first AHA policy statement on diet and heart disease, published in *Circulation* in August 1957. Although some later accounts cite this report as the first policy statement advocating reductions in dietary saturated fat (e.g. 5), it was actually quite cautious and circumspect. The five authors expressed concern about a “flood of diet fads and quackery” (53). They noted that, “great pressure is being put on physicians to do something about the reported increased death rate from heart attacks in relatively young people”. However, they warned, “some scientists have taken uncompromising stands based on evidence that does not stand up under critical examination” (53). They expressed skepticism about Keys' studies, and warned against the extrapolation of results from formula dietary experiments to the general population. They argued that there was not enough evidence to “permit a rigid stand” on the link between diet and heart disease and concluded: “We are certain of one thing: the evidence now in existence justifies the most thorough investigation” (53).

Several leading scientific and medical organizations supported the conservative stance of the AHA, including the Research Council of the National Academy of Sciences (54), the AMA Council on Foods and Nutrition (55) and the Nutrition Foundation (49). In December 1959, the FDA issued a statement announcing that “the role of cholesterol in heart and artery diseases has not been established. A causal

relationship between blood cholesterol levels and these diseases has not been proved". Therefore, advertising claims linking consumption of vegetable oils and margarine to a decreased risk of heart disease were "false and misleading" (56).

Changes in Policy

The late 1950s and early 1960s were pivotal years in the history of dietary policy. While the AHA and other organizations initially advised caution, Mary Lasker, the wealthy philanthropist and influential advocate of medical research, tried to persuade doctors to "do something" about cardiovascular disease, even in the face of imperfect knowledge (12) . A decade had passed since her lobbying efforts had helped establish the NHI, and she and her allies now turned their efforts to public education. In 1959, she persuaded eight physicians, including five former AHA presidents (erstwhile conservatives Page and Stare among them), to issue a statement under the auspices of the National Health Education Committee - a Committee which she chaired. The statement outlined five "factors predisposing to arteriosclerosis" – "heredity, overweight, elevated blood cholesterol level, elevated blood pressure and excessive cigarette smoking". People were advised to see their doctors if any of these factors were present (57).

By now, generous funding for research was yielding results, and some aspects of the links among diet, cholesterol levels and CHD were becoming clearer. In 1960, *Time* magazine reported that at the AHA's annual meeting, the "great cholesterol controversy" was beginning to subside, and that "even onetime skeptics were prepared to concede that abnormal quantities of fatty material in the blood should be regarded as one of the major factors in producing heart-artery disease" (58). Shortly after this, the AHA released another policy statement (59). This time, it cautiously endorsed dietary change. The statement was formulated by an elite group of six scientists, including Page and Stare. However, the

group now had two new members - Keys and another staunch supporter of dietary change, Jeremiah Stamler. The document began by announcing: “Current available knowledge is sufficient to warrant a general statement regarding the relation of diet to the possible prevention of atherosclerosis” (59). As supporting evidence it cited Keys’ work, about which no doubts were raised, and animal and human dietary experiments. The AHA worded its recommendations carefully: “These and other research studies have given clues as to the prevention of atherosclerosis by dietary means. A reduction in blood cholesterol by dietary means, which also emphasizes weight control, may lessen the development or extension of atherosclerosis and hence the risk of heart attack or strokes. It must be emphasized that there is as yet no final proof that heart disease or strokes will be prevented by such measures” (59). The policy statement recommended dietary changes for overweight people, those who had already had a heart attack or stroke, men with a family history of heart disease, high cholesterol, high blood pressure and for those “who lead sedentary lives of relentless frustration” (59).

The AHA statement listed 23 references in support of its recommendations. Most described experiments in which cholesterol levels were reduced by modifying the diet. Others were epidemiologic studies linking high cholesterol levels or other variables with an increased risk of heart disease. In other words, the studies were concerned with the first two of the three hypotheses outlined above. Only two of the references discussed experiments designed to test the crucial final link in the chain - whether dietary change (through a reduction of cholesterol levels) could also reduce the incidence of cardiovascular disease. These studies were Jolliffe's Anti-Coronary Club (60) and an English study with people who already had CHD (61). Both papers described how the studies were set up, but were too recent to yield any data.

The Search for the Definitive Evidence

Before, during and after the AHA's first cautious endorsement of dietary change, scientists continued to work towards the "definitive proof". Ideally, this would be an experiment demonstrating a reduction in the incidence of heart disease in people consuming a cholesterol-lowering diet. Some early studies were suggestive. In 1950 and 1951, John Gofman and colleagues published reports claiming that diet could reduce the incidence of further attacks in heart disease patients (62, 63). Lester Morrison, a doctor from Los Angeles, made similar claims on the basis of a twelve year study of 100 patients (64). However, neither study held up well under later standards of methodology and reporting, and were not cited in the AHA's 1961 policy statement.

The 1950s and 1960s were decades of rapid change in the design of clinical trails (65). What passed for a reasonable experiment in the 1950s was deemed to be full of errors by the end of the 1960s. For example, Jolliffe's Anti-Coronary Club recruited subjects with a previous history of heart disease, obesity, hypertension or diabetes alongside those who were healthy. No thought was given to the establishment of a control group. The investigators tried to improve their study later by adding a retrospective control group, and excluding men with a previous history of heart disease from the analysis. Results published in 1966 claimed that men who lowered their fat intake suffered less heart disease (51). However, because of its flaws, the study did not qualify as the "definitive proof". The same was true of other early studies (66).

During the 1960s, scientists did strive to set up a large, well-designed study that would finally settle the issue. In 1960 the NHI began funding a National Diet-Heart Study that involved many prominent researchers. Their aim was to investigate the effects of dietary change on normal, healthy men. However, they calculated that, for such an experiment to yield a statistically significant result, they would need to enroll 100,000 subjects for five years (67) – a formidable task, but one that the scientists were determined to carry out. Because of the expense and sheer logistical difficulties, they spent

several years conducting feasibility studies. Their report, published in 1968, claimed that the experiment was feasible, and recommended that the NHI carry it out as soon as possible (68).

However, a few years later, an NHI task force recommended against a purely dietary study. Instead, it called for a "multiple risk factor intervention trial" (MRFIT), designed to test whether a combined attack on smoking, high blood pressure and cholesterol levels would prevent deaths from CHD (69). Investigators began planning this experiment in 1971, and the results were published in 1982 (70). Scientists thus spent more than two decades designing and conducting a large trial of the cholesterol hypothesis. In the meantime, those concerned with promoting health found it difficult to wait. Instead, public, political and commercial interest in the links between diet and disease mounted. More policies were formulated. By the time MRFIT produced a result, the urgency had passed.

The 1960s: More Controversy and More Policies

There is a fine line between scientifically respectable treatment and so-called faddism – the use of unproven remedies and preventative measures. Such practices are beyond doctors' control (71), and orthodox medical organizations often warn against the use of "fad" diets – that is, diets outside the realm of official recommendations – for weight loss and/or health reasons (72, 73). Without definitive evidence of their efficacy, cholesterol-lowering diets in the early 1960s hovered on the edge of "faddism". However, mounting suggestions of a positive correlation between high cholesterol levels and disease, and the possibility of lowering levels through diet, gradually made cholesterol-lowering diets a scientifically respectable, if still experimental, treatment for patients at high risk of heart disease.

The AHA's cautious endorsement in 1960 of cholesterol-lowering diets for high risk patients may have been an attempt to regain control over knowledge linking diet to cardiovascular diseases (74, 75). Ironically, however, its equivocal tone encouraged the opposite effect. Its cautious endorsements and careful provisos were exploitable by both sides of the controversy. Vegetable oil companies tested the limits of the FDA ban on health claims by highlighting the AHA statement in advertisements. The National Dairy Council, on the other hand, seized on the disclaimer about "no final proof" to support its counter-claim that "The idea that replacing some 'saturated' fats with 'unsaturated' fats will help prevent heart disease is clearly unproved" (76).

In 1962, the AMA Council on Foods and Nutrition published a statement on "The Regulation of Dietary Fat" that, like the AHA report, cautiously endorsed dietary change for those at increased risk of heart disease (77). It received wide coverage in the popular press (74,75). The Council was so disturbed by the enthusiastic media and lay response that it issued a press release titled "Latest Food Fad is Wasted Effort", that tried, once again, to regain control. It pointed out that laboratory tests were necessary to determine cholesterol levels, and stressed that doctors should be in charge of any dietary change (78, 79). Again, vegetable oil companies and the dairy industry exploited the situation. Another round of claims and counter-claims created even more confusion (80-82).

During the 1960s, the lobbyists whose efforts had initially helped to stimulate the massive research effort into the causes of cardiovascular disease became increasingly impatient with the scientists' equivocation. Mary Lasker and her allies used their influence in Congress and the NIH to push for comprehensive policies aimed at "conquering" heart disease. When these failed to materialise, they asked President Kennedy to establish a "President's Conference on Heart Disease and Cancer" (83). Kennedy was assassinated before these efforts bore fruit. However, President Johnson, a sufferer of CHD and personal friend of Lasker's, was a staunch ally of the health lobbyists. His Commission on

Heart Disease, Cancer and Stroke was the second such commission established during his administration, after the Commission on the Assassination of President Kennedy (84). In 1964, it produced a report recommending the establishment of new regional centers for research and treatment of cancer and heart disease, extra training for physicians and education for the public (85). The AMA opposed the effort, known as the Regional Medical Program, because it smacked of state intervention in medical practice (86). The recommendations were watered down. Nevertheless, one outcome of the Program did affect policy. An Inter-Society Commission on Heart Disease Resources brought together representatives from 29 medical organizations to formulate policies on prevention, diagnosis, treatment and rehabilitation (87).

All these activities – the articles in the popular press, the conflicting advertisements, the lobbying, and the Commission - were indications of mounting interest in the relationship between diet and heart disease. The demands that something be done created problems for scientists, as developments in clinical trial methodology made it increasingly difficult for them to produce quick answers. Nevertheless, while the definitive proof of the efficacy of cholesterol-lowering remained elusive, investigations of other aspects of the links between cholesterol and CHD continued. During the 1960s, more epidemiologic studies were reported, including several from Framingham (88-90). The studies affirmed a consistent positive correlation between serum cholesterol levels and risk of cardiovascular diseases.

In the mid-1960s, the AHA and the AMA decided to broaden their dietary recommendations to include people who did not already have heart disease (91, 92). Both statements cited new epidemiologic knowledge to justify the move to primary prevention. The AMA Council stated: “The observations regarding risk which support this position are derived chiefly from the Public Health Service Study in Framingham, Mass.” However, it also noted: “it must be recalled that definitive proof that lowering

serum cholesterol, or preventing a rise in serum cholesterol, will lower the morbidity and mortality associated with coronary heart disease, is still lacking" (91). Over the next decade, researchers continued to refine and expand their understanding of many aspects of CHD. Several more policy statements advising dietary change were published (66, 93). For a time, it seemed as though the cholesterol controversy had died down. It re-ignited, however, when the Federal Government re-entered the dietary policy arena in the 1970s.

Government Involvement in Nutrition Policy

Since 1917, when it issued its first set of dietary guidelines, the Department of Agriculture (USDA) has been the federal agency responsible for formulating and disseminating nutrition policies in the United States. For most of the century, the policies were aimed at preventing deficiencies, not deterring excess. Consumers were advised to choose foods from "protective" groups - dairy products, meat and eggs, fruits and vegetables and so on. The first four editions of the USDA dietary guidelines, published between 1917 and 1946, recommended daily consumption of up to eight food groups, including fat. Such advice benefited the agriculture and food industries (31). However, the new scientific knowledge linking fats and cholesterol to disease upset the comfortable relationship between food producers and nutrition policy-makers. The USDA avoided provoking the wrath of industries selling foods containing saturated fats and cholesterol through two decades of controversy. The 1958 edition of the dietary guidelines did not mention fat at all, and the Department refrained from publishing any more guidelines until 1980. The first United States government guidelines recommending reductions in fat and cholesterol intakes were highly controversial. They did not emerge from the USDA, but from a temporary body - the Senate Select Committee on Nutrition and Human Needs (SCN) (31).

The SCN was initially established in 1968 to tackle malnutrition due to poverty. In 1976, it turned its attention to what it called "Diet Related to Killer Diseases" and held hearings to "consider the role of diet in preventive health care and the degree to which diet contributes to the development of major diseases including heart disease, cancer and diabetes" (96-98). A few months later, the SCN released the first edition of *Dietary Goals for the United States*. Among the recommendations were some very provocative suggestions, including, "Decrease consumption of meat…Decrease consumption of butterfat, eggs and other high cholesterol sources" (99).

The meat, diary and egg industries had made some sporadic, but unsuccessful, attempts during the 1970s to challenge claims that their products caused disease (100, 101, 97). Now, the alarm bells really rang. If *Dietary Goals* were adopted as official government policy, there could be changes to nutrition education, labeling and advertising laws, and the content of diets fed to millions of people, including school children, hospital patients, prisoners and armed services personnel. Income price support mechanisms might also be affected. Food industry lobbyists exerted pressure in Washington, and SCN hearings on the *Dietary Goals* were re-opened. Sessions were set aside to hear testimony from the meat and egg industries (102, 103)**.**

Food industry lobbyists were able to accumulate numerous scientific statements expressing skepticism about the efficacy of dietary change (104)**.** The SCN revised the *Dietary Goals* and released a second edition in December 1977. Although some proponents of dietary change claimed that industry had exerted strong and illegitimate control over policy (95, 101), the changes were minor. The recommended daily allowances of fats and cholesterol were unchanged. Suggestions for food selection were reworded as follows: "Decrease consumption of animal fat, and choose meats, poultry and fish which will reduce saturated fat intake… Decrease consumption of butterfat, eggs and other high cholesterol sources. Some consideration should be given to easing the cholesterol goal for pre-

menopausal women, young children and the elderly in order to obtain the nutritional benefits of eggs in the diet “ (105).

Select Committees can act only in an investigative and advisory capacity and are not empowered to present legislation to congress (98). In order for *Dietary Goals* to affect government activities, it had to be taken up and used. Between 1977 and 1980 the situation was quite confused, as it was unclear which sector(s) of the federal health or agricultural bureaucracy, if any, would take responsibility for implementing policies aimed at reducing fat and cholesterol intake. For a time, there was a “turf war” between the USDA and the Department of Health, Education and Welfare (DHEW) over nutrition research and policy. Some food activists and their political allies expected the DHEW to take a more proactive role in the dietary prevention of disease. However, scientists at the National Heart Lung and Blood Institute (NHLBI), as the NHI was now called, were in a bind. They were still engaged in two long–term and expensive experiments designed to provide the long awaited definitive proof of the efficacy of lowering cholesterol levels. The results of MRFIT and a drug trial – the Lipid Research Centers Coronary Primary Prevention Trial (LRC-CPPT) – would not be available until the early 1980s. Speaking out in favor of dietary change before then would be tantamount to anticipating the results of the trials (97, 106-108).

Although the NHLBI scientists were reluctant to speak out, three sets of dietary recommendations did emerge from various other corners of the federal bureaucracy during 1979 and 1980 – a Surgeon General's Report titled *Healthy People* (109), a joint USDA-DHEW document, *Dietary Guidelines for Americans* (110), and a report from the Food and Nutrition Board (FNB)of the National Academy of Sciences called *Towards Healthful Diets* (111). The first two of these advocated reductions in fat and cholesterol intakes, though the former used more emphatic language. The controversy that erupted around the documents followed a pattern that was, by now, quite familiar. Food activists praised

Healthy People and blamed the meat industry for the more cautious wording of the *Dietary Guidelines* (108). Meat industry representatives, on the other hand, repeated their claims that the link between diet and disease was still unproven (112).

Of the three statements, the FNB's *Towards Healthful Diets* caused the most uproar. The Board had long taken a conservative position on the cholesterol issue (54, 113). Now, members expressed concern about what they saw as an excessive and unrealistic hope that nutrition could prevent diseases such as cancer and heart disease, which they believed were not primarily nutritional in nature. The Board took a skeptical approach to the evidence linking saturated fats and cholesterol to heart disease, stating that "intervention trials in which diet modification was employed to alter the incidence of coronary artery disease and mortality in middle-aged men have generally been negative". It also claimed that "epidemiology establishes coincidence, but not cause and effect" (111).

By 1980, at least 18 organizations in the US and elsewhere had formulated policies recommending dietary change (114). The FNB was a very prestigious body, and its deviation from the prevailing viewpoint provoked much media interest (74, 75). The debate illustrated the degree to which diet had become a political issue (114–116). Consuming less fat and cholesterol had become a "progressive", pro-health, pro-consumer cause, a means through which people could protest against corporate greed. On the other hand, political conservatives praised the FNB for taking a stance against the "Naderites" who "mope around Washington DC proclaiming that everything we eat is unsafe" (117). However, although the conservatives were gathering momentum (the Reagan administration was about to begin), they were unable to gain the high moral ground on the cholesterol issue. Food activists pointed out that some of the FNB scientists had links to the egg, dairy, meat and processed food industries (114, 118). The FNB's consumer liaison panel resigned in protest, and members of the Board were required to justify their views in hearings before a House Agriculture Subcommittee (116, 119). After the FNB

report, it was difficult for scientists to question the status of evidence in favor of cholesterol-lowering without being labeled dupes of those sectors of the food industry that profited from the sale of saturated fats and cholesterol..

The Large Trials and an End to Controversy

In 1982, the results of the Multiple Risk Factor Intervention Trial (MRFIT) were published (70). Researchers had designed this trial to test the combined effects of diet, blood pressure medication, and smoking cessation in middle-aged men exhibiting multiple risk factors for heart disease. After screening, 12,866 men were enrolled for an average of seven years. Despite careful planning, the results were not what the investigators had expected (70). There was no statistically significant difference between the control and intervention groups in the primary end-point – death from CHD. In designing the trial, scientists had used Framingham data to predict a death rate of 29 per 1,000 men in the untreated group and 21.3 per 1,000 in the intervention group. However, the death rates were 19.3 and 17.9 respectively. The investigators gave several explanations. There were more deaths than expected among men taking blood pressure medication. Death rates from CHD across the nation had fallen, for reasons that were not clear. Finally, men in the intervention group had not changed their risk factors as much as expected, while many in the "untreated" group had made changes (70). A report in the *New York Times*, based on interviews with men assigned to the control group, gives an insight into factors that affected the results. One of the men stated: "I said to myself I'm not going to be a part of the control group and kill myself for the sake of their statistics… Once I realized that I had two risk factors, I made some modification "(120). Despite all the careful preparation, definitive proof of the effects of cholesterol-lowering on the incidence of coronary disease remained elusive.

The results of the other large NHLBI test of the cholesterol hypothesis were published in 1984 (121) . The Lipid Research Clinics Coronary Primary Prevention Trial (LRC-CPPT) was a drug trial, which enrolled 3,806 middle-aged men in the top 5% of the risk profile for heart disease. After an average of 7.4 years of medication or placebo there was slightly less heart disease in the treated group. This result was statistically significant, provided a one-sided test for significance was used (121). This type of test assumes that the result of the experiment can only go one way – that is, that the treatment can only be beneficial. It is customary to use a two-sided test in clinical trials, thus allowing for the possibility that treatment may be harmful (122).

Although the LRC-CPPT produced a marginal result, despite enrolling only middle-aged men at very high risk, the NHLBI claimed that it provided the long-awaited definitive proof of the efficacy of cholesterol-lowering drugs *and* diets in preventing CHD in the general population (123). There was a lively debate over the interpretation of the results in the medical journals (122, 124-126). In addition to the unorthodox use of the one-tailed test, scientists questioned the validity of extrapolating the results to dietary recommendations for the whole population. The deaths of men in the treatment group also provoked concern. More men taking the drug had died from accidents, suicide and violence than in the control group. According to some commentators, this warranted further investigation (122, 124-126).

The controversy in medical circles did not spill into the public arena. In the popular press, any lingering uncertainty had been clarified. *Time* magazine, for example, featured a cover story titled “Sorry, It's True. Cholesterol really is a Killer”. It quoted leading NHLBI investigator Basil Rifkind: “It is now indisputable that lowering cholesterol with diet and drugs can actually cut the risk of developing heart disease and having a heart attack” (123). Not long after the LRC-CPPT results were published, scientists at the NIH organized a Consensus Conference on Lowering Blood Cholesterol to Prevent Heart Disease. Despite a few dissenting voices, the conference attendees produced a strong statement in

favor of dietary change for everyone in the US over the age of two years (127, for objections see 128, 129). It also recommended the establishment of a National Cholesterol Education Program, to convince everyone in the US of the importance of monitoring their cholesterol levels, and if deemed appropriate, reducing them through diet or drug treatment (130).

Conclusion

By the end of the 20th century, the anti-fat, anti-cholesterol message was widespread and well-entrenched (5,6). From time to time, dissenters have argued that the evidence in favor of dietary change is less substantial than is commonly supposed (129, 131, 132). However, so far these arguments have had little or no effect on policy. The institutional mechanisms in favor of fat and cholesterol restriction have become large and well-developed. Only time will tell if, and when, and how, policy will change. As CHD rates decline and the incidence of obesity increases, we may see a fundamental reassessment of what constitutes healthy eating, similar to that which occurred when medical attention shifted in the mid 20th century away from infectious diseases and vitamin deficiencies towards cancer and CHD. On the other hand, the rise in obesity may merely provoke policymakers into intensifying their anti-fat message.

The story of the controversies over fat, cholesterol and CHD does not hold any easy lessons for the policymakers of the future. It provides, at best, a cautionary tale that illustrates the degree to which policies in the field of public health are inextricably bound up with cultural, political and economic concerns. The recent trend towards evidence-based medicine seeks to deflect pressures and influence from these quarters by strengthening the link between policy and reliable scientific evidence (133). However, it is unlikely that such efforts will be able to neutralize these pressures altogether. "Evidence" is often a slippery concept, and hard to come by, and lay people,

politicians and others will continue to demand that scientists and policy makers "do something" about health problems in the community. Given the pressures, and the difficulties of obtaining definitive evidence, complex political imbroglios are bound to develop. There is no way to insulate medical science from the rest of society. The conduct and interpretation of experiments occurs within a social context, and policy-making is inevitably a political process.

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