Diet-Related Chronic Diseases: Moving from Cause to Prevention

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There is a need for better communication between people involved in public-health issues, of which I am one, and representatives of the food industry. My objective here is to initiate the kind of dialog that I feel is necessary on public-health nutrition issues and how they relate to agricultural biotechnology and food production.

By way of background, I will discuss the current disease burden in the United States and globally, and public-health objectives and approaches: as public-health workers, what are we trying to achieve with health and how does that relate to food? Then I will discuss how food technology makes our lives as public-health people better on one hand and makes it worse on the other. Some implications for the food supply are noteworthy, based on a presentation at a World Health Organization/Food and Agriculture Organization (WHO/FAO) Consultation on Diet, Nutrition, and Chronic Diseases, Geneva, Switzerland, in January 2002, in which I participated. And I will conclude with policy implications and research needs, from my perspective.

Current Disease Burden

An article by Michael McGinnis, formerly the Deputy Assistant Secretary for Health, and Bill Foege, who was at the Centers for Disease Control (CDC), Atlanta, and also was President of the American Public Health Association, is often cited to establish that most deaths are now related to modifiable "lifestyle" factors: tobacco, alcohol, diet, and other personal behaviors (McGinnis and Foege, 1993). This picture of chronic disease is increasingly applicable globally, as shown by the *Global Burden of Disease* report (Murray and Lopez, 1996), which was a topic of much discussion at the January 2002 WHO/FAO meeting. Heart disease is now the most prevalent ailment globally, *i.e.* for developing as well as for developed countries. Although communicable diseases remain important in developing countries, they kill fewer people than non-communi-

cable or chronic diseases. Even in regions where there are food shortages, and people continue to strive for survival, some are dying from heart disease and diabetes. It is projected that, by 2025, most of the people affected by chronic diseases will be outside the United States, *i.e.* in India, China, the Russian Federation, Japan, Pakistan, and Indonesia.

Attempts have been made to calculate how much of the United States healthcare budget is devoted to conditions such as obesity. In 1995 dollars, type-2 diabetes cost about $50 billion, for example. Some 5 to 8% of the national health care budget is spent on obesity-related diseases—a huge fraction for one condition, and it is probably an underestimate. In Australia it is over 2%, and 4% in the Netherlands and France. In developing countries in which healthcare budgets are small, and where diabetes is emerging, as much as 25% of the healthcare budget is being spent treating complications like end-stage renal disease. In such countries, there is potential for overwhelming effects of chronic diseases. Therefore, there is need to deal with these conditions even where there is need to get food to the hungry.

The latest data, for 1999, from the CDC website, show continuing increasing trends in obesity, including children in the 6 to 11 and 12 to 18 age groups. It is to be expected that 5% would be above the ninety-fifth percentile standard; instead 14 or 15% are above it, and this does not include the children who are considered to be at risk of being overweight, *i.e.* up to 30% of some populations. Not only does childhood obesity often lead to adult obesity, it is associated with problems during childhood.

The burden is not evenly distributed by ethnicity and income status; 6-to 11-year-old black girls were no more obese than white girls in the 1950s, whereas they were more likely to be above the ninety-five percentile in 1994 data, and a similar trend is seen in Hispanic girls. There is more obesity and more chronic disease within some minority populations than in the general population. For death rates, the excess shows up primarily in African Americans. Deaths per 100,000 population for heart disease and cancer are higher for blacks, whereas other minority groups have lower rates than for the white population. This indicates ethnicity-linked protective factors. One of our goals, from a public-health perspective, is to elucidate and preserve the factors that contribute to lower death rates—to understand why these groups have not acquired all of the risk factors in these diseases, at least to the point of mortality.

For heart disease and cancer, similar patterns by ethnicity are seen for men and women. For diabetes, Asian Americans are the only group with rates lower than for whites. More years of potential life are lost from diabetes among American Indians, Hispanics, and blacks compared to whites.

Finally, on disease burden, there is considerable low birth weight in this country among black and Puerto Rican Americans, and the chronic-disease-risk profile now includes this factor. The concept is that something happens *in utero*

to compromise an adequate pattern of growth. Children are born small, and, although they may gain weight rapidly, epidemiologically they remain at higher risk for chronic diseases in adulthood. This was seen in the Dutch-famine and in other studies leading to the "Barker hypothesis" or the "fetal-origins-of-disease" hypothesis. We are still trying to elucidate the reasons why low birth-weight due to undernutrition during pregnancy results in a predisposition in certain communities to chronic diseases later in life.

Public Health Perspective on Diet

The task of the public-health worker is to prevent and control disease. Anything in the system that appears to cause or aggravate disease is a possible focus for intervention. We are also responsible for positioning these issues in the larger discussion: in my work, I am interested in engaging those whose work has effects on what people eat or how active they are, or on other aspects of their health, *e.g.* urban planners, bus route planners, bankers. The role of the public-health scientist is to show that everything in the system is connected: apparently unrelated activities can have unexpected repercussions in terms of adverse effects on the health of the population. We are trying harder to quantify relative impact in order to allot priority in terms of numbers of people affected as well as severity. We are advocating for an objective policy process, *i.e.* to prevent vested interests from unduly influencing the policy process. I was at the infamous release of dietary guidelines when Secretary Glickman had a pie thrown in his face; part of the concern thus expressed was that vested interests had been influential. The only vested interest entering the policy process should be that of protecting vulnerable populations. We need to be one step removed when we are setting policy.

How do we set dietary guidelines? We try to keep track of everything—what people eat and how it affects their health—*i.e.* the nutrition-monitoring system that is now endangered because of lack of funds. We try to track food and where it is eaten, we set nutrient requirements, measure health outcomes, monitor consumer behavior, *etc*. With the appropriate data, we can monitor downstream effects of various choices, both in terms of industry activity and in terms of consumer behavior. It is complicated, especially when teaching epidemiology students, because it involves not just what is eaten but also host-specific factors. Whether supplements are being taken and in what forms and whether a nutrient needs activation all influence what doses people are getting and the ultimate health effects. It becomes a complicated process also from the conceptual and causal-model points of view; our data are seldom precise, and we are never quite sure whether an apparent effect is real or not.

We are now using an outcomes-based approach for devising dietary guidelines. Formerly, the approach was to look at whether intake seemed to be low or high based on our best data, and then examine evidence linking the nutrient to a particular disease or other adverse effects associated with intake at

that level. If there were possible adverse effects then we would track them, and if we could confirm them then we would change the level in the food supply, increase it or decrease it to alleviate the risk. However, there are too many components in the food supply. Going nutrient by nutrient, even with just fifty items to track, rapidly becomes too ponderous. Therefore, as we obtain better data—in the United States and in other countries, and the WHO is now using this approach—we can start with disease outcomes. Heart disease, diabetes, obesity, cancer, osteoporosis, and bone and dental diseases were most recently addressed by the aforementioned WHO/FAO diet and disease consultation. We then look at how convincing is the evidence that appears to associate a disease with food intake. If it is convincing, if it is a definite public-health/nutrition issue, we make a recommendation to increase or decrease intake. If it is probable, a potential issue, and we are not quite sure—food changes are considered to be harmless compared to, say, drugs with which physiological effects are unknown—any recommendation to increase or decrease intake would not be made aggressively, but offered as advice. And then, if there is no evidence of a pattern, *e.g.* with coffee—although past studies have proven negative—there may still be a potential research issue. As long as consumers or scientists *think* that there is potential for harm, we keep studying it. And when something new comes on the market, even in the absence of apparent adverse effects it will be a potential research issue, but, without evidence of disease, no effort will be expended to affect intake.

As an aside: intake is likely to change. If one item is modified because of convincing evidence, then it will shift at least part, if not all, of the dietary pattern. Also, intake may change unexpectedly because of alterations in the food supply. Again, it is difficult to draw sound conclusions from such complexity.

The type of evidence we obtain contrasts with data that are generated in controlled experiments. We seek ecological validity for human populations rather than for small numbers of individuals in a laboratory setting. We glean ideas by comparing countries or by following people over time and by seeking patterns in retrospect in those afflicted with disease, having examined what they were eating. In clinical trials we investigate whether changing that component of the diet has the desired effect. Then larger-scale trials are conducted before we consider new policy; simultaneous studies in the lab examine mechanisms to elucidate what should be tested in the trials.

To decide whether an association is causal, however—because it is not laboratory based—we use logic, graded logic, to determine if we have consistent, unbiased, strong, coherent, repeated, predictive, and plausible evidence. Although such firm evidence is seldom obtained, areas of relative certainty exist. We assign importance to relative risk; we are most concerned about factors that have large effects on people with diseases, but small effects on a lot of people are also of great concern.

The World Cancer Research Fund Panel published a comprehensive coverage of studies that attempted to relate cancer to diet up to 1997, and the evidence rules that they used with slight modification were also those used in the WHO/FAO report referred to above. In the strongest category there is consistency, with strong laboratory support and evidence of a dose-response, *e.g.* the more fruit and vegetables you eat the better off you are compared to people who eat less or none. In the "probable" category, there is less consistency, possibly resulting from fewer studies, but there is strong mechanistic or laboratory support. Then there is the "possible" category, which is generally supportive but no firm conclusion is possible. Lastly, in the "insufficient" category are issues that have public appeal, but no supporting evidence; they may garner media coverage but there is no persuasive reason for formal study.

The following are common themes, and areas in which we feel secure: calorie intake should be controlled, correct energy balance maintains weight, fat and cholesterol should be limited, a variety of plant foods have positive effects, and moderation in intake of sugar and salt is important, as are adequate physical activity, alcohol in moderation and avoidance of smoking. A conference was convened in 1997 (*Preventive Nutrition: Pediatrics to Geriatrics* in Salt Lake City, Utah by the Nutrition Committee of the American Heart Association, with invitees from the American Cancer Society, Diabetes Association, Dietetic Association, National Institutes of Health, American Academy of Pediatrics, and the USDA Dietary Guidelines) to discuss targets for change in the American diet (Deckelbaum *et al.*, 1999). At first, some of the commonality that emerged was thought to be coincidental, but, as more data are obtained, a convergence is emerging in mechanisms causing various diseases once considered to be very different. For example, at the time the conference was held, insulin resistance was not known to be related to cancer, but much data now indicate a link. Inflammation was thought to be strictly of infectious or other origin, but now it is understood to be linked to the atherosclerotic pathway. And apoptosis related to cancer is now understood to be related also to diabetes and atherosclerosis. So, it could be that one set of physiological processes, variously expressed in individuals with different predispositions or different exposures, leads to different diseases.

What is wrong with this approach? First of all, it is essentially outside the agricultural sector. So new recommendations on dietary guidelines are made with no knowledge of implications for the food supply, which has global implications. This type of public-health approach should be taken in conjunction with people in the production sector. Also, this perspective on food is very different from that of the consumer. We look at food as a carrier of risk, whereas few people sit down at the table thinking they that they are partaking of risk factors, or in the supermarket feel a need to minimize risk factors as they shop for food. Our reductionist chemical perspective is difficult to communi-

cate to consumers. In large part, our approach still emphasizes individual diseases and is simplistic with respect to dietary interactions. Epidemiology is very bad at handling interactions: we need broad control in order to investigate one part, even though in nature it is impossible to control one component that is influenced by others. It is also non-experimental because that is the only means of investigating natural scenarios in human populations, therefore, we can never be sure about cause. And a long lag-time is necessary for definitive answers; suspicion over a new addition to the food supply is likely to require thirty or forty years before it is proven to be justified, or not.

Food Technology

Food technologists are addressing many of these issues, but outside of the public-health sector. There are unlimited possibilities, which is problematical from a study-design point of view. When I assess diet I have no idea what I am looking for in the population, because the many new variables that are entering the food supply are changing our bases for risk assessment.

Furthermore—a pet peeve of mine—the health-based marketing of single foods is not really consonant with dietary guidance, because we are moving more towards patterns. The emphasis on health effects from single foods is making life more difficult, because consumers have the tendency to look for magic bullets, as some claims seem to promise.

Food Supply

From a food-supply perspective, let's consider the United Kingdom, where most people are not meeting dietary guidelines—*e.g.* very few, especially women, meet the guidelines for fat or fiber—and also consider that huge changes are taking place in countries like China because of the globalization of the food supply. A model developed by the International Obesity Task Force, the policy and advocacy arm of the International Society for the Study of Obesity (London) gives ideas on how links between global markets and development factors and advertising reach across national boundaries effect changes. It becomes very difficult to devise generally applicable methods to elicit change.

Prakash Shetti at the Food and Agriculture Organization, addressing the WHO/FAO consultation, posed the question of whether, in recommending 400 g of fruits and vegetables per day, anybody had multiplied the global population by 400 to see if enough fruits and vegetables are actually available? Can dietary guidelines be met without talking to producers? Also, he pointed out that 80% of fish imports are to Japan, the United States and the European Community; a third of the catches from developing countries enter international trade, supplying 50% of total exported fish. We may be creating a situation with dietary guidelines in which we take food from developing countries so that we can have the right amounts of fish and fruits and vegetables in affluent countries. These issues need careful consideration.

It is difficult to predict trends in consumer preferences. Even with intense advertising campaigns, it is hard to tell if consumers will accept something different in the diet. An analysis of national-survey data between 1965 and 1991 by Barry Popkin documented changes in whites and blacks for fiber products, pasta, *etc*. For example, blacks increased their high-fiber cereal consumption by 1,500%, whereas whites increased theirs by a smaller amount. In some cases, trends for blacks and whites were in opposite directions. When the food supply changes, we still cannot predict long-term effects.

Policy and Research Issues

Top-down strategies are being discussed, making some people uneasy because they conflict with the free-market ethic. Finland, for example, has successfully instituted such a strategy, although it requires community involvement, including cooperation from people who resist regulation and who object to the government telling them what to do. Certainly, integration and harmonization of public-health strategies with the food industry are essential. Having drawn up dietary guidelines, integration of agricultural and public-health policies becomes necessary. However, when agricultural policy is being set, improvement in public health is not a chief objective; clearly, coordinated policy development is needed.

With the top-down approach, there is less reliance on consumer education than hitherto. In the Finnish example, high rates of cardiovascular disease were reversed by environmental changes, with taxation and other strategies that did not rely only on direct appeals to individuals to change their behavior. Technology played a key role in developing a locally adapted rapeseed oil. The concept of a cholesterol-lowering oil produced domestically gained popular acceptance. The reductions in chronic heart-disease rates over a 25-year period are well documented—not merely through improved treatment (as here in the US), but by significantly reducing new cases. Dr. Pekka Puska, who led this successful program in Finland, is now with the WHO leading a global effort to reduce rates of heart disease (Puskka, 2000).

What are the implications of agricultural biotechnology for dietary guidance? If the food supply changes as a result of genetic engineering, we need to blend those changes with dietary guidance. This morning, my orange-juice carton indicated a calcium content equal to that in milk. Nutrients that were here are now also there. This may be good, but how does it affect the food pyramid? We need to forecast trends and consumer reactions with which to match the guidelines. Clearly, we need to study not only what people are eating and what is in the food, but, especially, shifts in food intake and particularly in vulnerable populations.

And finally, regarding environmental issues: in fact, there is very little understanding of how to change the environment in a way that would be beneficial for both producers and consumers with respect to diet and health.

References

Deckelbaum RJ *et al.* (1999) Summary of a scientific conference on preventive nutrition: pediatrics to geriatrics. Circulation 100 450–456.

McGinnis JM Foege WH(1993) Actual causes of death in the United States. Journal of the American Medical Association 270 2207–2212.

Murray CJL Lopez AD eds. (1996) The Global Burden of Disease. A Comprehensive Assessment of Mortality and Disability from Diseases, Injuries, and Risk Factors in 1990 And Projected To 2020. Cambridge: Harvard University Press.

Popkin BM *et al.* (1996) A comparison of dietary trends among racial and socioeconomic groups in the United States. New England Journal of Medicine 335 716–720.

Puskka P (2000) Nutrition and mortality: the Finnish experience (Tutorial). Acta Cardiology 55 213–220.