Ending the Cardiovascular Disease Epidemic

An Interview With Lewis Kuller

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SG: Is cardiovascular disease eradicable?

LK: It’s feasible, although it wouldn’t be easy. We’re dealing with 4 interrelated “common-source” epidemics. One is dietary intake of saturated fats and polyunsaturated fats. The second is excess salt intake. The third is excess caloric intake relative to energy expenditure, leading to obesity. The fourth is tobacco use.

Unfortunately, the emphasis has been only on changing individual behaviors. It is difficult to control a common-source epidemic strictly on an individual basis. The way to control a common-source epidemic is to modify the sources. This means changing the types of foods that are available, reducing salt in the food chain, improving opportunities for physical activity, and discouraging smoking. Unfortunately, political and economic conditions make these difficult to accomplish. Small successes have been the legislation and political action to reduce trans fatty acids in restaurants and in the food chain and the recent support by the American Medical Association to reduce salt intake in the food chain.

SG: And recent smoking bans in restaurants in many US cities.

LK: Yes—smoking is a good example of controlling the epidemic at its source. The reduction in smoking in the United States is not primarily due to smoking cessation programs. The reduction of smoking is from the increasing cost of cigarettes, the reduction in number of places where people can smoke, and the fact that smoking has become socially unacceptable. Tobacco smoking in the United States is pretty much restricted to lower socioeconomic populations, unfortunately—that, plus the relatively small number of people at all levels who are addicted to nicotine and require intensive efforts to stop smoking.

SG: It’s interesting that legislation to stop smoking in restaurants and bars has been associated with a decline in rates of myocardial infarction in those cities.

LK: We don’t know whether this is an effect of reduced smoking in the community, or reduced environmental tobacco smoke, or whether this is just a chance phenomenon. Without any question, smoking is the most important precipitant of an acute coronary event, and the most potent risk factor for coronary heart disease, especially in women. It is likely that changes in smoking prevalence have had the biggest impact, along with the diet, on the declining CHD [coronary heart disease] rate in the United States.

Coronary death rates in the United States have dropped by 50% and are continuing to decline at close to 4% a year. This is most likely related to the decline of smoking, the dramatic 30–40 mg/dL decline of blood cholesterol levels, and decreases in blood pressure levels. Control of hypertension has played a big role, as well as improvement in the treatments of cardiovascular disease. So we’ve had a big winner and this has increased life expectancy.

SG: Is this evidence that public health really can affect cardiovascular disease rates?

LK: Differences in populations can give us clues about what public health interventions might work. The incidence rates of cardiovascular disease in Japan are one-tenth those in the United States. The Japanese are heavier cigarette smokers than in the United States, so there’s something else in the Japanese lifestyle that accounts for their low coronary disease rates. The Japanese have a diet very high in omega 3 fatty acids. They eat 100 g of fish a day, they have about 7% saturates, and they have lower body weight.

In the United States there are dramatic differences by areas. Connecticut, for example, has a very low death rate in middle-aged men. In Pennsylvania, middle-aged whites have one-third the rate of coronary disease in Montgomery County as in Allegheny County. Differences that big are not due to medical care—they’re due to lifestyle. This strongly suggests that modifying these lifestyles is feasible and could have a dramatic effect on CHD rates.

SG: What role does education play?
LK: The Nurses’ Health Study showed a huge gradient in coronary disease rates by educational level. Education determines 2 things. One, education is related to the levels of risk factors, things like smoking, obesity, dietary intake of saturated fat and cholesterol, exercise. And 2, education affects adherence to lifestyle modification and long-term therapy.

SG: Earlier we talked about the change in the social view of tobacco from accepted behavior to pariah. Can we look at the overweight epidemic in much the same way? Are we in a very early stage of changing people’s ideas about what a healthy weight ought to be?

LK: I think you’re right. Epidemics related to behavior change usually begin in the better educated, more prosperous parts of the population. Those behaviors gradually migrate down. Then the middle and upper social classes develop approaches to prevent or modify their behavior. If you look at the smoking epidemic, it began in middle and upper class men and women. In the United States, the AIDS epidemic began in middle and upper class men, and then became an epidemic of low income and poor and minority men and women. I think the same thing may be happening with the obesity epidemic.

The critical factor in these epidemics is to understand how they’re transmitted in the population—then you can prevent them. One of the failures of our approach to the obesity epidemic is that we haven’t paid enough attention to how the epidemic evolved, and we haven’t successfully identified the points of intervention. We just have a long litany of approaches to prevention and treatment, none of which has really worked.

The obesity epidemic is related to 3 primary factors. The first is a change in family structure in the United States. Most family members now work and are out of the home. The second is the development of techniques for making cheap fast foods. This is like the smoking epidemic. Techniques for making cheap cigarettes were developed in the 1890s. Without cheap cigarettes you wouldn’t have had a smoking epidemic. Without cheap fast foods you wouldn’t have as big an obesity epidemic. The third problem is the reduction in time available for leisure activity. It’s clear that the obesity epidemic can be affected by modifying any of those 3 factors. These are the common sources for the epidemic. If you understand the interrelationships of the social and built environment, and the agents of the transmission, you can basically control the epidemic.

SG: Can we do more with educating the public?

LK: We’ve done a poor job of informing the public about the importance of maintaining low risk factors through their life. We’ve also done a poor job of educating both the public and the health care community that this disease begins early in life. Obesity is a lifetime problem, not a five- or ten-year event.

SG: Maybe we should apply the concept of readiness-to-change to society, like we do to individuals.

LK: We need to convince the public and the political powers that they should commit resources to this particular approach. Our efforts in prevention are being subsumed by a lot of buzzwords—genomics, proteomics, metabolomics—and this leads to a presumption that you can’t prevent heart disease.

Given our current environment, I suspect that we might have trouble developing a polio vaccine today unless we could specify the biologic mechanism, and identify those people at risk. We seem to have lost track of the logic of how to prevent disease. Epidemiologists are equally guilty because of our fascination with complex statistical analysis, the best logistic regression models, and how many P values we can find, rather than relating biology to epidemiologic observations and prevention.

SG: Say more about the polio epidemic. What does it teach us?

LK: It’s important to understand polio. I was a budding physician in the days of the polio controversy, and there are similarities to atherosclerosis. The viral disease was extraordinarily common. Practically all kids were infected, but only a small number of the kids actually developed clinical poliomyelitis. We weren’t really sure why some kids got paralytic polio and why some kids got bulbar polio and died. We still don’t know why.

If you look at atherosclerosis the same way, you can say atherosclerosis is extremely common, but only a relatively small number of individuals at any one time have a heart attack. Still, if you follow people with atherosclerosis over their lifetime, the majority will sooner or later have a cardiovascular event. We could approach prevention by identifying the very few people who are at high risk and developing a high risk therapeutic approach—but this would have very little effect on the population. It would be the same argument as developing a polio vaccine limited to those kids who were going to get paralytic polio. The probability that that would have worked is extremely remote. As a rule, efforts that focus an intervention on the very high-risk group, say through genetic testing, will be a dismal failure for a common-source epidemic.

The longer we delay our treatment of atherosclerosis, especially if we delay it to the time when people already have a heart attack, the benefit for the population is small. If you compare the rates of disease in people who have been put on statins after a heart attack (a very effective therapy) against the rates of disease in people with minimal atherosclerosis, the rates of the people with little atherosclerosis are far, far lower.

SG: So to “inoculate” the population against cardiovascular disease, we need to start young.
LK: Absolutely. You can prevent atherosclerosis in children and young adults, and that should be the highest priority, without question. We should aim to drive LDL cholesterol down to around 70 to 100 mg/dL, which has been suggested as the level where you won’t see much atherosclerosis. That’s going to be the big winner in the long run.

A key question is how high should the polyunsaturated fats be in the diet, and what type of polyunsaturates should be there? As you get older, the ability to modify atherosclerosis gets harder. We tell people age 65 and 70 that they should change their diet, and reduce the saturated fat, and lower their LDL cholesterol by 5 or 6 mg/dL. It’s a waste of time. As you get older, the approach has to be different—you need a much more aggressive approach. In older men and women, over age 50 or 55, you need very aggressive intervention to modify the disease process. Probably most high-risk older individuals should get aggressive drug therapy.

Dietary efforts should be for the primary prevention of atherosclerosis. We should focus more of our research on the early stages of atherosclerosis, using our new technologies for measuring atherosclerosis to determine how effective we are in preventing this early development.

SG: And then older people who don’t have atherosclerotic disease don’t need aggressive intervention.

LK: Probably a quarter to a third of women and a few men are at very low risk their entire life. Interestingly enough, you can identify these women before they are even menopausal by their healthy lifestyles, their low levels of LDL cholesterol and blood pressure, their nonsmoking status. Your risk factors in middle age and even younger determine your risk of heart disease at 75 or 80—not your risk factors when you’re 75 or 80.

SG: There are very few men who have low risk factors during their first 30 or 40 years.

LK: You’re right—it’s an absolute disaster for men in the United States. We focus so much of our efforts on the short-term, worrying about which is the best vegetable to eat or the best drug of the day, all of which have relatively small effects. We keep forgetting that the entire population of US men, especially over age 50 or 55, have a terrible atherosclerotic disease that could have been totally preventable by healthy lifestyles early in life.

SG: What can the epidemiology community do to “inoculate” society against cardiovascular disease?

LK: The first question is who should take the lead. Those who led the charge 30 or 40 years ago are retired or gone—Jerry Stamler, Henry Blackburn, Nemat Borhani, Paul Dudley White. The National Heart, Lung, and Blood Institute at NIH gives lip service to prevention. They are heavily focused on end-stage disease and on genomics. Genes may be important, but they are unlikely to be the solution to the problem. The Centers for Disease Control has some excellent people, but the CDC is caught up in mindless process review with little funds for effective implementation. The state health departments are, unfortunately, undermanned, underfunded, weak, and ineffective. So the first thing that epidemiologists and prevention people have to do is find the leadership to develop a really first-rate prevention program.

The second is to figure out where the resources should come from to implement an effective program. Even voluntary organizations like the American Heart Association have, to some degree, lost their focus on prevention. They have become heavily committed to drug treatment and new devices (ie, to clinical cardiology). It may be correct for them to do this, but there’s no complementary organization and leadership within the federal government or other agencies in the area of preventive cardiology.

SG: Should we also be designing studies to eliminate the common sources of the epidemic?

LK: Yes. The AIDS epidemic in many ways got out of hand because we didn’t use good epidemiologic thinking to understand how the disease would spread. If you understood AIDS as a venereal disease, and knew how venereal diseases spread in the population, then you would have anticipated how the AIDS epidemic spread.

In the obesity epidemic, the answer is to focus on the 3 biggies. We need to test the hypothesis that we can increase activity by offering better approaches to leisure time activity. We have to find effective ways to modify people’s eating behaviors. Fast food and eating out are here to stay. We have to deal with that. We’ve got to get industry on our side, and change the way food processing is done. That would be the winning strategy, not blaming industry. The food industry has made a big investment in alternatives to trans fats in the United States. Let’s partner again. Most important, we need to get out the message that cardiovascular and cerebrovascular disease are mostly preventable.