Much has been written on the relationship of diet to heart disease and almost anything can be "proven" if one selects the appropriate references. The basic problem which has precipitated the debate on dietary fat and other related controversies is the extremely high mortality in the United States from cardiovascular disease. There are about 600,000 deaths annually from complications of atherosclerosis and about 1,000,000 from all types of cardiovascular trouble. It has been estimated that if we could eliminate cancer as a cause of death in the U.S. the average length of life would be extended about 2 1/2 years, but if we could eliminate cardiovascular disease, the average length of life would be increased 8 to 10 years. These startling statistics have led to a great proliferation of research to determine the cause of the inordinately high incidence of cardiovascular disease in the U.S. as compared to other populations and to develop means whereby it can be reduced. The following are some of the factors which are often mentioned as possible contributors to our problem:

1. Heredity--most certainly a contributing factor.

2. Stress--Many believe this is a very important factor but early settlers in the U.S. must have been stressed. Germans and Norwegians certainly were stressed during World War II; yet coronary heart disease was markedly reduced.

3. Smoking (especially cigarettes)--shown to be involved, but people in some countries with much less coronary heart disease (CHD) smoke about as much as we do.

4. Lack of exercise--relationship not clearly established but data are strongly suggestive.

5. Diet--some components which have been suggested as factors are: too many calories, too much sucrose, too little of certain vitamins such as vitamin E and C, too little of certain minerals such as magnesium and chromium, too much salt (NaCl), too much fat (or saturated fat). Data are available to support or contradict (as you prefer) each of these suggested relationships and many others.

6. Hypertension--probably a contributing factor but certainly not the only one.

7. Hypercholesteremia--associated with atherosclerosis but is this a cause and effect relationship?

8. Obesity--some relationship but not the principal factor.

The last three might be considered secondary (i.e., they themselves are abnormalities which have their own set of causes).

I believe we in the animal industry should be interested in this subject because of:

1. The tragic number of deaths due to CHD and other cardiovascular troubles, and

2. The need, in my opinion, for the animal industry to be thoroughly knowledgeable so that it can:

   a. Refute unwarranted criticism of its products.

   b. Make appropriate alterations of some of its products if evidence ever shows definitively that such changes are needed.

Our research group has, for many years, been engaged in research related to cardiovascular disease, with particular emphasis on the relationship of diet and exercise to blood plasma cholesterol and the development of atherosclerosis. Ruminants were chosen as our principal models because of the similarity of arterial changes in some of these species to arterial changes in humans, the adaptability of some of these species to surgical alterations necessary for many of our studies, and several other advantages. Now let us turn to specifics, some of which I will illustrate with slides.

One sometimes hears the statement that the reason we have so many heart attacks is that we live longer and must die of something. The facts are, however, that people in many other countries, with a much lower incidence of cardiovascular disease, live longer than we.

Cardiovascular disease is a broad term and I want to restrict my comments largely to atherosclerosis (lipid deposition and other degenerative changes in the arterial wall). In many species, as the animal grows older the walls of the arteries thicken, sometimes to the point of seriously restricting blood flow and of precipitating formation of thrombi.

It has been amply demonstrated, with experimental animals, that degenerative changes in the arteries can be hastened by certain dietary manipulations, particularly when these changes are extreme. Feeding high levels of cholesterol, accompanied by fat, causes higher blood plasma cholesterol and increases lipid deposition in the walls of the arteries. The maximum effect from a given amount of dietary cholesterol usually has been seen when the level of fat is about 25 to 30 times the level of cholesterol. But, many such experiments have involved feeding cholesterol at levels much higher than those usually encountered in natural diets (e.g., the equivalent, in some
cases, of the cholesterol intake of a man eating several dozen eggs daily). Certain dietary manipulations can lower plasma cholesterol also. Usually, but certainly not always, the "saturated" fats cause somewhat higher plasma cholesterol than do the more unsaturated fats. Why this happens is not clear. Some believe it involves only a redistribution of cholesterol in the various tissues of the body.

We must be careful in manipulating diets so that we do not create problems as great or greater than those we are trying to correct. In one study, death rate from complications of atherosclerosis was lowered by increasing unsaturated and decreasing saturated fats in the diet of men but incidence of cancer was increased and the overall death rate was not altered. Also, there is some evidence that unsaturated fats may decrease longevity by hastening the aging process in the tissues. Furthermore, changing one dietary component may affect requirements for others. For example, an increase in dietary unsaturated fatty acids greatly increases the requirement for vitamin E. The proportion of unsaturated fatty acids in milk, meat and eggs can be greatly increased, if desirable, by manipulation of the diet of the animal. But some questions and problems are involved, not the least of which is this: What is the effect of feeding a diet high in unsaturated fats over a long period of time? Some animal experiments suggest there may be some problems. Certainly, the ultimate effects of various major dietary manipulations on cardiovascular disease and other parameters is far from clear.

Let us now look at a few other facets of this situation. According to best estimates, the per capita consumption of saturated fats and cholesterol has changed very little in the U.S. during the past 60 years. Yet the incidence of deaths from coronary heart disease has increased dramatically. During the same period, consumption of unsaturated fats has increased.

Milk and meat have been criticized because of their comparatively high content of saturated fatty acids but many countries which consume far more milk and milk products per capita than we do have a lower incidence of coronary heart disease. And countries with both high and low meat consumption have less of this problem than we.

Atherosclerosis is by no means restricted to humans. It has been observed in many species (e.g., swine, cattle, deer, caribou, rabbits, pigeons, dogs, ducks, pheasants, sharks). Whether it is a common cause of death in any of these species, except under experimental conditions, is not known.

An extensive comparison of Irish in Ireland with Irish in Boston was conducted cooperatively by personnel of the Harvard School of Public Health and the Trinity College School of Medicine in Dublin. A total of 1,994 middle aged men were involved, including about 500 pairs of brothers, one of whom lived in Ireland and the other in Boston. Irish in Ireland had much less atherosclerotic heart disease. Brothers living in Ireland consumed more calories, more complex carbohydrates, more butter (7.3% of calories vs. 2.9% in Boston) more magnesium, more fluorine (in tea), more milk, more
stout and more beer and had more physical activity but less coffee, less whiskey, and less gin, and less unsaturated fat (4.5% of calories vs. 14.0% for Boston). There was no significant difference in serum cholesterol, blood pressure, cigarette smoking or proportion of calories from fat. Body weight and the number of abnormal electrocardiograms were higher in the Boston Irish. Autopsies showed much earlier atheromatous involvement in the coronaries and aortas of the Boston Irish. One of the key differences, if not the principal one, seemed to be the much greater physical activity of the men in Ireland.

Much must yet be learned before the shocking death rate from cardiovascular disease in the U.S. can be reduced to a more acceptable level. Any attempt to attribute the problem to only one factor is unrealistic and misleading; many factors undoubtedly are involved. It would, however, seem prudent to exercise great caution in promoting major changes in the American diet (except to practice moderation).

In my opinion, two of the major contributors to our problem in the U.S. are (1) overindulgence in food and drink, and (2) underindulgence in physical exercise. We need to get these back in balance. Furthermore, many more liberties can be taken in the diet if it is counterbalanced by appropriate physical activity.

A. M. MULLINS: Ladies and Gentlemen your conference chairman was kind enough to let me introduce some people this morning before the conference program gets under way. First, I would like to introduce the new directors to you. The names we put on the board yesterday. The new directors for the American Meat Science Association for next year are James A. Christian. Jim, are you in the audience? Jim’s a hard worker and a late sleeper. Fred Parrish, Fred is up in the visual aid stand in the back and Dr. Warren Tauber, Union Carbide, Warren where are you? In the back and up in the right hand corner. Congratulations to all of you gentlemen. We’ve introduced the incumbent directors previously.

There’s one other man in the audience that I would like to introduce and I know most of you know this man by name but I’m sure that there are a lot of people in the audience that haven’t had the privilege of meeting him personally and that is Dr. J. L. Lush of the Iowa State University, a renowned animal breeder and geneticist. We’re delighted to have you with us this morning, Dr. Lush. I saw you in the audience Monday and didn’t have the opportunity to introduce you at that time.

C. E. ALLEN: The next reporting this morning is the Meat Cookery Committee, and the chairman of this committee is Miss Reba Staggs, from the National Live Stock and Meat Board, and I think rather than do better than Dave Stroud did yesterday in introducing Reba, I’ll only say that she is widely known throughout the United States, and of course we all know her through her activities at the National Live Stock and Meat Board, so, Reba, we’re looking forward to hearing from your committee. And I see they’re all intact up here in front, so you can have the program.
REBA STAGGS: Thank you, Dr. Allen. The Meat Cookery Committee members are listed on page 12 in your program. I'm not going to call their names but I do want to make this public announcement—that each one on the committee has made a very important contribution. In deciding the subjects to be discussed this morning, it was not so difficult to find a great number of subjects in this area, but was more challenging to decide just what subjects we would discuss. We hope that after the program this morning you will feel an inroad has been made in your thinking and that the two areas are not only important to meat processing but also to meat as prepared in the home. Since we don't eat meat raw, it would seem that both presentations should be of interest to all of us. We will have the two presentations and, as indicated in your program, following them there will be a question and answer period.

Our first speaker this morning is a member of this organization. He holds membership in many very prominent organizations, but I'm not going to take your time to name all of them because that would take his time for speaking. He took his B.S. degree at Ohio State in Food Technology, his further academic training at that university in biochemistry, he has been associated with the Hy-Grade Packing Company as a food technologist in the research department, he has been a professor, and associate professor of biochemistry at Purdue University, and he is currently employed by Peter Eckrich & Sons, Inc. He went there in 1965 as a chemist in the Research Department, is currently the Manager of the Basic Research of that organization. I present to you, Dr. Ned Draudt. Dr. Draudt will speak on "Changes in Meat During Cooking."