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Dear doctors,

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Once again I would like to thank the hospital for inviting me to speak to the medical staff and community about nutrition. Thanks for taking time out of your busy day to attend. As requested I am providing some of the scientific evidence supporting low carb diets. I also wish to make some additional comments.

Point 1 – Dr. A, stated that there are years and years of outcomes data looking at thousands and thousands of patients and that these studies show that a diet high in saturated fat increases the chances of developing outcomes such as heart attack and death related to heart attack.

This is not actually the case. What Dr. A was apparently referring to was the observational literature, in which large cohorts are followed for decades and attempts are made to associate diet as measured typically by food frequency questionnaires with future disease status. Despite the ubiquity of these studies, they are incapable of establishing a cause and effect (with the possible exception of smoking and lung cancer, in which the effect of cigarettes is so large that it is virtually impossible to come up with a reasonable alternative explanation for the association seen). While these studies make attempts to assess saturated fat consumption, they tended to ignore such critically important variables as the refinement of the carbohydrates and grains consumed and the sugar (sucrose and high fructose corn syrup) consumed. Food diaries and food frequency questionnaires are known to be subjective and flawed measurement instruments, and it's well accepted that the quality of data collected over 15 to 20 year spans of time is suspect at best. Even if one accepts that these studies can measure with reasonable accuracy the variables that the investigators claim to measure, the best that can ever be said about these data is that they are hypothesis-generating, not that they are meaningful evidence of causality.

I have attached a list of some of these major observational studies looking at saturated fat intake and heart disease. Worth noting is that the two most recent meta-analyses of these studies -- in 2009 by Siri-Torino et al and in 2010 by WHOTK et al, -- looked at 347,000 subjects in 21 separate cohorts followed between 5-23 years. Both of the Meta analysis studies found **NO** association between dietary saturated fat consumption and heart disease.

I have also included 24 additional separate observational outcomes studies. 16 of those studies have found **NO** association between dietary saturated fat and heart disease. While eight of these studies did find a statistically significant association, that association vanished in four of them when adjusted for multiple confounding variables.

Prior to this past decade, public health policy on the saturated fat content of the diet was based primarily on prospective clinical feeding trials that adjusted the ratio of polyunsaturated to saturated fats as a means of adjusting serum cholesterol and LDL cholesterol levels. The rationale was that the only way to lower cholesterol levels in the blood without raising triglyceride levels and so balancing out any predicted effect on heart disease was to replace saturated fat with polyunsaturated fat rather than carbohydrates. The hypothesis to be tested was whether this intervention would reduce heart disease risk.

Many such studies were done along these lines, and I have included them. Only two were reasonably well-randomized controlled trials: the Minnesota Coronary Survey and the Los Angeles Veterans Trial, and they failed to show that lowering cholesterol by increasing the ratio of polyunsaturated fat to saturated fat reduced heart disease. Three meta-analysis published recently confirmed the absence of evidence to implicate saturated fat in the diet as a cause of heart disease. A fourth meta-analysis – by the Cochrane Collaboration, an international organization dedicated to doing unbiased reviews of medical interventions – is expected to be published shortly. In 2006, the Women’s Health Initiative published its low-fat diet trial, in which 20,000 women were randomized to a low-fat diet, high in fruits, vegetables and whole grains, and compared to 29,000 randomized to a control diet. The trial failed to show that a low-fat diet had any effect on heart disease, diabetes, breast cancer or obesity.

My conclusion, as I pointed out in my lecture, is that while we have long believed that saturated fat is a cause of heart disease, the actual evidence continues to refute that hypothesis.

I have also included a list of now several dozen randomized-controlled trials comparing diets restricted in carbohydrates to relatively low-fat diets that are also restricted in carbohydrates. Although, these trials do not provide us with outcomes data – with hard endpoints -- they universally demonstrate that low carb diets improve heart disease risk factors at least over a year or two – the length of the trials -- compared to low-fat, calorie-restricted diets. Low-carb, high-fat diets raise HDL-C, lower triglycerides and may sometimes elevate the LDL-C, considered to be old technology. LDL particle sub-fractionation and LDL-particle number, now considered the single best indicators of heart disease risk by the lipidology community, is invariably more favorable on the low-carbohydrate, high-fat diets. It troubles me that Dr. A does not see much value in this new technology. It’s arguably the case that any decisions on treatment options for obese and overweight patients should be based on the results of these clinical trials alone. In my lecture, I discussed, for instance, the results of the Stanford University A-to-Z trial, which were published in 2007. The principal investigator actually admitted to his bias against low-carbohydrate, high-fat diets and his surprise at finding that this diet was superior in terms of both weight loss and heart disease risk factors compared to the other diets studied.

Getting back to outcomes vs. prospective randomized trials. Do we ignore these well-designed trials because they do not have outcomes data, despite showing an improvement in cardio-metabolic markers? With luck (and significant funding) we will see head-to-head outcomes studies comparing diets, but this will take 10 to 20 years or longer. Until that time we need to forge forward with the highest quality data we have and those data argue clearly for the prescription of low carbohydrate, high fat diets for obese, overweight and diabetic patients.

Point 2 - Dr. A, in our meeting afterwards, expressed concern that his patients would want to stop taking cholesterol medication after hearing my lecture. I was not telling anyone to stop taking Statins or that there was never a need to take this type of medication. I was simply discussing weight loss and the cardio-metabolic effects. I do however acknowledge his concern.

Low carb high fat diets, lower insulin and address inflammation. Metabolic markers improve as a result, including cardiac CRP, c-peptide, Hga1C, adiponectin, and atherogenic lipoprotein variables.

Low fat diets may also improve metabolic markers, if a patient can maintain it, but not always in a favorable way. Although there is no scientific basis for a low fat high carb diet, metabolic markers do improve. A likely reason why is because any diets that restrict calories significantly, even if they're defined as "low-fat diets", will also reduce carbohydrate consumption significantly and improve the quality of the carbohydrates that are consumed.

If patients lose weight the need for Statin therapy diminishes. However, for those who fail both diets, patients tend to eat carbs and fats together in great quantity, what I refer to as the typical American diet. These patients need all the help of modern medicine. We have no choice but to reply on poly-pharmacy and expensive procedures to prolong life but not necessarily to improve the quality of life.

Point 3 - Dr B, asked me to comment on the work of Dr Weston Price and life expectancy. His legacy lives on with his foundation. To this day the foundation supports the original ideas that whole and unprocessed foods, containing macro as well as micronutrients better support health. Dr Price pointed out that modern civilization and the refining and processing of food has lead to deterioration in our health as a society. I too am frustrated that back in the 1930's nutrition was not center stage. Other pressing issues like World War II was a distraction. After the war it appears that popular opinion, the government, money and industry were the predominant forces shaping nutrition.

During this time the food industry focused on the notion of a "balanced diet" being absolutely necessary for health, or at least the best way to hedge against vitamin or mineral deficiencies. (It also guaranteed each segment of the food industry would have a market for its goods, which was indeed a stated goal of the Harvard nutritionists who were most responsible in the 1940s for pushing the concept of a balanced diet in the U.S.) The lipid hypothesis, low fat and low calorie became popular and supported the continued production of inexpensive grains, soy legumes and sugar, mostly refined and processed. Science supporting diets high in saturated fats and low-in-carbohydrate was unpopular in large part because it conflicted with the hypothesis – that saturated fat causes heart diseases. When the U.S. government began giving dietary advice in the late 1970s, many researchers – including the head of the National Academy of Sciences – objected, calling it nothing more than a nutritional experiment with the U.S. public as the subjects. Those scientists whom objected were not supported. The experiment however was backed and supported by the USDA, HHS, CMS, FDA, NIH, NHLBI and the American Heart Association. Those outside of the government -- like Dr Price, forty years earlier – had little influence.

I also pointed out that prior to 1900 nutrition was different. Food was less processed, refined and contained less carbohydrate. Whole foods were consumed such as animals, vegetables and fruits when available. The primary cooking oils were Butterfat, Lard and Beef Tallow. Life expectancy was low at this time not because of nutritional factors but because child mortality and deaths from infectious diseases were far higher. You pointed out that people live longer today. I did not get a chance to answer. Much of the advances in life expectancy since then can be attributed to modern medicine, improvements in emergency medical services, pharmaceuticals and other new technologies, not to mention smoking cessation, etc. Any role of nutrition in this increase in life expectancy is difficult to tease out and opinions among the experts differ greatly.

What I'm proposing is that as a nation and as individuals, we're eating too much sugar and too much refined and processed carbohydrates and it is making us fat and ill. And the evidence in support of this is from well-controlled randomized trials – put individuals on diets devoid of refined grains and sugars and allow them to eat as much as they want of fat and saturated fat, they get leaner and they get healthier. Changing out diet may help to prevent disease, reduce morbidity, improve quality of life and or extend life even further. We might even save some healthcare dollars along the way.

We should all be open to advances in science and nutrition, for the benefit of community, our patients and ourselves. I welcome any other comments you might have.

Sincerely,

A handwritten signature in black ink, appearing to read "J Gerber MD". The signature is written in a cursive, flowing style.

Jeffrey N. Gerber, M.D., FAAFP
Board certified family physician