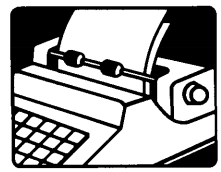
Editorials



Lipids, Diet, Diabetes, and Atherogenesis

The pathogenesis of atherosclerosis is still uncertain but most theories invoke a central role for abnormalities in lipid and/or lipoprotein metabolism.[1] Current dogma has it that dietary consumption of cholesterol, saturated fat, and total fat contributes to the development of atherosclerosis. The logical extension of this argument is that decreasing intake of these substances will result in both a lowering of circulating lipid levels and a decreased risk of atherosclerosis. This is the "lipid hypothesis," as Ahrens has dubbed it.[2] Although not yet substantiated, there probably is sufficient circumstantial evidence for us to adopt the lipid hypothesis, at least tentatively.

Diabetes is associated with accelerated atherosclerosis. The complex interrelationships between diabetes, diet, obesity, lipid metabolism, and vascular disease is the subject of a recent volume of *Advances in Modern Nutrition*.[3] The reader of that volume will find no certain answers, but will find many new questions to contemplate. In this issue of DIABETES CARE, Hsia and his colleagues give us another factor to contemplate—serum cholesterol binding reserve (or SCBR).[4] This is the capacity of serum to solubilize cholesterol and presumably correlates with capacity of the serum to function in the normal clearance of cholesterol from tissue. Hsia has shown that his SCBR can be accounted for by high density lipoprotein (HDL) and very low density lipoprotein (VLDL). Others have focused most attention on HDL.[5,6] The current hypothesis is that the greater the serum capacity for clearing cholesterol (as measured by either HDL or SCBR), the less the risk of atherosclerosis. Hsia has preliminary evidence (unpublished), in a prospective study, that these may be much more powerful predictive risk factors of myocardial infarction than serum lipid levels. Diabetic patients are deficient in their capacity to clear cholesterol, and this may contribute to their enhanced atherogenesis. The real question is whether HDL or SCBR levels can be influenced by diabetic control, diet, or anything else we do. Additionally, if these factors do indeed prove important in cholesterol clearance, we may have to modify the lipid hypothesis to account for the interaction between lipids and those lipoproteins that clear tissue of lipids.

If the lipid hypothesis is correct, we should be especially concerned about the level of dietary fat consumption by our diabetic patients. Indeed, it has been suggested by some that the accelerated atherosclerosis seen in diabetes is a consequence of the dietary prescription we have advocated for diabetic patients over the last half-century or so. This latter thesis is based on the fact that at constant caloric intake, if we disproportionately limit dietary carbohydrate consumption, we must as a consequence liberalize dietary fat consumption. Thus, the practice of limiting carbohydrate intake, coupled with the encouragement of the use of milk and eggs as protein sources, may have inadvertently contributed to the accelerated atherosclerosis seen in diabetes. This, then, was one of the reasons for the 1976 revisions of the *Exchange Lists for Meal Planning*,[7] and the 1977 *Guide for Professionals*[8] using those lists. Unfortunately, these were at least five years in the genesis, from the time of the first acknowledgment of the problem by the ADA Committee on Food and Nutrition in 1971.[9]

It might be supposed that we and our patients can rest more comfortably now, if we adopt the precepts of decreasing our cholesterol and saturated fat intake. After all, that is a position advocated by all of the relevant reputable health agencies—the American Diabetes, Dietetic, and Heart Associations; the National Heart, Lung and Blood Institute (NHLBI); the Senate Select Committee on Nutrition; etc. Indeed, NHLBI has spent many millions of dollars supporting the Lipid Research Clinics Program. Should we be bothered, then, by the fact that the lipid hypothesis is still hypothesis? Should we be bothered by the realization that we do not yet have answers to the question of what is the best nutritional program for our health? Should we be bothered by the sobering thought that the dietary wisdom

of yesterday's experts may have resulted in the enhanced risk of atherogenesis run by our diabetic patients?

This issue of DIABETES CARE includes two articles that are germaine to these questions. Anderson and Ward outline their experience with a high-fiber diet in the treatment of diabetes.[10] They and others[11–15] have been finding that such diets may be beneficial for diabetic patients, both in terms of blood glucose control, and control of blood lipids. Yet, if the lipid hypothesis is unproven, the fiber hypothesis is even less well established. And Monnier et al.,[16] also in this issue of DIABETES CARE, provide evidence that not all fibers have the same effects, at least in terms of glucose metabolism.

The conclusion I must draw from this is that we have much yet to learn about the complex interrelationships between lipids, diet, fiber, diabetes, and atherogenesis. Personally, I happen to believe both that the lipid hypothesis is correct and that high fiber intake is beneficial. Yet, before we adopt either or both as dogma, I think we should recall again that standard practice for many years called for disproportionate carbohydrate limitation (and hence high fat intake), and, although an attractive approach at the time, it is now viewed as having been a wrong, and even harmful, approach. And at the time, the authorities supported that position. I should also point out that those who advocated liberalizing carbohydrate intake were often severely criticized at that time. Several years ago, those advocating high fiber intake were also often criticized. And, today, we hear criticism of those who advocate megavitamin programs, vegetarian or natural foods, and other unconventional nutritional positions. Let us not cast stones.

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Why Don't We Teach and Treat Diabetic Patients Better?

I would like to start with outlining an *ideal* situation concerning the relationship between the patient, the primary care physician, and the diabetes specialist. First, all three are highly motivated, very intelligent, and well aware of the needs and the extent of the knowledge and of the ignorance of each other. All three are cooperative.

The patient comes to the primary care physician with his problem: recently discovered insulin-dependent diabetes. The primary care physician immediately refers the patient to the diabetes specialist. The latter is immediately available and remains so day and night for a couple of weeks and thereby starts on a professional but also friendly relationship with a confident and highly motivated patient. The patient was sent to him by a highly motivated primary care physician who is fully aware of what is at stake and fully confident in the reliability of the diabetes specialist. After three weeks, the patient is sent back to his family doctor with full knowledge of diet, insulin, urine-testing, and test-recording; self-adjustment of insulin dosage; awareness of insulin