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This work was supported in part by the U.S. Department of Energy under Contract No. DE-AC03-76SF00098, by the National Institutes of Health Program Project Grant HL 18574 from the National Heart, Lung, and Blood Institute, and by a grant from the National Dairy Promotion and Research Board, administered in cooperation with the National Dairy Council.
FASEB Symposium Papers

Overview

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This work was supported by the National Institutes of Health Program Project Grant HL 18574 from the National Heart, Lung, and Blood Institute, a grant from the National Dairy Promotion and Research Board and administered in cooperation with the National Dairy Council, and was conducted at the Lawrence Berkeley Laboratory through the U.S. Department of Energy under Contract No. DE-AC03-76SF00098.
The topics addressed in this symposium relate to a fundamental issue of emerging importance in nutrition namely, the role of one's genetic milieu in influencing dietary response to nutrients, and hence in determining optimal dietary intakes of these nutrients. In the case of dietary fat and cholesterol, we are dealing with key components that influence metabolic pathways involved in the development of atherosclerosis and cardiovascular disease. These in turn are now coming to be understood in terms of the genes involved in their regulation. At this time there are dozens of well-characterized genes that have been found to determine the structure and metabolic processing of plasma lipoproteins, and yet, as the papers in this section illustrate, we are only getting a hint of the much larger number of genes that are likely to be found to have a role in regulating plasma lipoprotein levels. In view of the intimate connection between diet and plasma lipoprotein metabolism, it is clear that many of the involved genes will be responsive to change in fat and cholesterol intake, and others will influence the nature and extent of the lipoprotein response to these nutrients. Indeed, it is reasonable to suggest that some genes affecting plasma lipoproteins evolved differently in different population and ethnic groups in conjunction with differing nutritional environments and metabolic needs.

The information presented in the papers from this symposium are based on studies in animal models, and in subgroups of human populations defined by genetic and metabolic parameters that have been found to influence the lipoprotein response to dietary fat and cholesterol.
Animal models are of considerable importance in this area since they afford us the opportunity to study regulatory mechanisms at the molecular and cellular level and to relate dietary response to atherosclerosis directly. Using molecular and biochemical tools, Dr. Lawrence Rudel and his group have investigated the basis for hypo- and hyperresponsiveness to dietary fat and cholesterol in nonhuman primates. Dr. Beverly Paigen has pioneered the mouse as a model for genetic effects on atherosclerosis susceptibility and dietary responsiveness. The mouse has proved to be particularly useful for investigating genes modulating the protective effects of HDL on diet-induced atherosclerosis, and Dr. Paigen reviews her work in this area, as well as recent studies identifying genes responsible for formation of gallstones on high-fat diets.

The remaining papers focus on genetically-influenced factors affecting dietary fat and cholesterol response in humans. Dr. Margo Denke reviews evidence for interindividual variability in lipid and lipoprotein response to dietary fat and cholesterol, and describes recent studies linking dietary fat responsiveness to carefully defined metabolic markers that distinguish pathophysiologic mechanisms underlying lipoprotein disorders. Dr. Darlene Dreon and I present recent work from our laboratory in which the definition and measurement of individual subclasses of human plasma LDL has proven useful for characterizing genetic influences on lipoprotein response to dietary fat. Finally, Dr. Barbara Howard reviews emerging data dealing with the importance of gender, race, and other intrinsic modulators of dietary responsiveness in human populations.

The work presented here is but an early indication of the wealth of information that is likely to arise from the application of new genetic and molecular tools to the understanding of factors influencing responsiveness to dietary fat and cholesterol. In advancing our understanding of these phenomena,
we are also recognizing that optimal dietary practices will differ among
individuals. This has important implications not just for defining specific
metabolic mechanisms and gene-diet interactions that may be of importance in
the pathogenesis of coronary artery disease (as well as a number of other
conditions affected by dietary fat intake), but also for helping the medical,
nutritional, and general community to appreciate the biological basis for the
wide interindividual differences in dietary responsiveness that they encounter in
attempting to promote and follow standard dietary recommendations for heart
disease prevention. Ultimately, we might anticipate a sufficient fund of
knowledge regarding gene-diet interactions in cardiovascular disease so as to be
able to tailor optimal dietary recommendations to individuals or groups with
differing needs.

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