Preface

In this book and atlas entitled High Density Lipoproteins, Dyslipidemia, and Coronary Heart Disease, I am indebted to the many chapter authors who have contributed their writing as well as their insights. The work grew out of a meeting on high density lipoproteins (HDLs), chaired by me and Dr. Vassilis Zannis of Boston University, and held on June 19 and 20, 2009 in Newport, Rhode Island, as a satellite meeting following the 15th International Symposium on Atherosclerosis, held in Boston. In the introduction our understanding of overall lipoprotein metabolism is reviewed, as well as the common genetic lipoprotein disorders associated with premature coronary heart disease (CHD). These familial disorders include lipoprotein(a) excess, dyslipidemia (high triglycerides and low HDL), combined hyperlipidemia (high cholesterol and high triglycerides often with low HDL), hypoalphalipoproteinemia (low HDL), and hypercholesterolemia. We discuss the management of these disorders. We also review other disorders including cerebrotendinous xanthomatosis, phytosterolemia, deficiency of apolipoprotein B, and severe hypertriglyceridemia. The latter disorder can be associated with pancreatitis.

Thereafter, the focus is on HDL, beginning with chapters on the regulation of apolipoprotein (apo) A-I gene expression, the composition, remodeling, and the metabolism of HDL particles, as well as HDL structure, function, and its anti-inflammatory properties. Then the focus shifts to chapters on disease states characterized by apoA-I deficiency, apoA-I variants, Tangier disease due to lack of ABCA1 function, the ABCG1 transporter, the interaction of HDL particles with transporters and receptors, human lecithin:cholesterol acyl transferase (LCAT) deficiency, human cholesteryl ester transfer protein (CETP) deficiency, and scavenger receptor-B1 (SR-B1) and HDL metabolism. Then the focus shifts again to topics of greater interest to the practicing physician including chapters on the genetics of HDL in the general population, the role of nutrition, alcohol, and exercise in modulating HDL metabolism, and the effects of estrogens, niacin, statins, fibrates, CETP inhibitors, and HDL infusion therapy on HDL metabolism and CHD risk reduction. We end with a concluding chapter focusing on the current and future status of the management of lipoprotein disorders, with a special focus on HDL. In our view, HDL raising is the next frontier in CHD risk reduction.

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