Diabetes mellitus (DM) is becoming increasingly epidemic globally. The World Health Organization (WHO) estimates that the prevalence of DM varies between 8 and 10% in all regions of the globe. Millions of new cases are diagnosed every year, and a substantial percentage of people with DM are undiagnosed either because they are not screened for the condition or because of inadequate access to healthcare. The epidemics of obesity, increased mechanization and reduced physical activity, cigarette smoking, and the fact that people are living longer have all contributed to the rise in Type 2 DM incidence. The incidence and prevalence of Type 1 DM is also increasing, perhaps also related to changes in the environment. Obesity, sedentary lifestyle, and cigarette smoking potentiate insulin resistance, which also promote atherosclerosis and the vascular complications of DM, as well as of Type 2 DM itself. Longer lifespan is associated with increased weight, lower levels of physical activity, and progressive loss of pancreatic islet cell mass. The US Centers for Disease Control estimates that 26.5% of Americans 65 years of age or older have DM. According to the American Heart Association, in 2008, 18 million Americans had diagnosed DM, with another 7.1 million having undiagnosed DM; it is estimated that the prevalence of pre-diabetes in the US is 81.5 million. These staggering numbers are not unique to the United States. The worldwide rate of rise in DM is just as alarming. It is estimated that by the year 2030, 340 million people around the world will have DM and the figure is likely to be higher.

The risk for DM is strongly influenced by genetic and environmental factors. Risk for new onset DM is strongly influenced by race and ethnicity. Insulin resistance (IR) is the hallmark of pre-diabetes and Type 2 DM and is characterized by impaired transduction of insulin signaling pathways. Insulin resistance, which also occurs in Type 1 DM, results in hyperglycemia and is also associated with visceral organ steatosis, endothelial dysfunction, hypertension, increased systemic inflammatory and oxidative tone, a prothrombotic state, intracellular accumulation of toxic lipid intermediates (diacylglycerol, ceramide), as well as atherogenic dyslipidemia, among other changes. These metabolic disturbances greatly augment risk for the development of microvascular and macrovascular disease. The epidemic of DM is expected to result in one of the steepest rises in human morbidity and mortality ever observed outside of wartime. DM is the leading cause of proliferative retinopathy and adult onset blindness in working age adults, peripheral vascular disease and lower extremity amputation, end-stage renal disease and
need for dialysis and renal transplantation, peripheral and autonomic neuropathy, and it magnifies the risk of myocardial infarction, stroke, and sudden death at least two- to four-fold. In addition to the human cost of this disease, there is an enormous economic burden associated with the clinical management and treatment of complications associated with DM.

*Lipoprotein in Diabetes Mellitus* is meant to be an authoritative and comprehensive reference on the many changes wrought by IR and DM on lipid and lipoprotein metabolism. Reducing the burden of atherogenic lipoproteins in serum is unequivocally associated with reductions in risk for cardiovascular events and may also ameliorate microvascular damage. The book begins by summarizing the various techniques to measure lipoproteins and their subclasses. In addition to delineating the molecular basis for how IR and DM alter lipid and lipoprotein handling in the gut, adipose tissue, liver, blood, and blood vessel wall, this volume explores how IR induces dyslipidemia, the glycation and oxidation of lipoproteins, and how alterations in immunity and cell surface receptor expression can impact lipoprotein metabolism. The mechanistic basis for why IR and DM increase risk for atherosclerosis as well as diabetic retinopathy and nephropathy are explored in detail. The design of clinical trials and the impact of lifestyle modification and of specific approved and investigational drug classes on diabetic dyslipidemia and risk for diabetes-related complications comprise the latter third of this volume. We thank our international panel of contributors for their clinical and basic scientific expertise and many insights. It is our sincerest hope that the clinicians who care for patients with IR and DM and the basic science researchers who explore mechanisms of vascular damage and protection will find this treatment of the issues covered herein timely and relevant and that it will significantly impact patient care in a positive and lasting way.

Oklahoma City, OK, USA

Peoria, IL, USA

Oklahoma City, OK, USA

Alicia J. Jenkins, MD, FRACP

Peter P. Toth, MD, PhD

Timothy J. Lyons, MD, FRCP