Preface

Lipids, Lipoproteins, Atherosclerosis and Cardiovascular Disease

John R. Burnett
Department of Core Clinical Pathology & Biochemistry, Royal Perth Hospital, Perth, WA 6487, Australia.
For correspondence: Dr John R. Burnett email: john.burnett@health.wa.gov.au

Cardiovascular disease (CVD) due to atherosclerosis is the leading cause of morbidity and mortality in Westernised countries. Atherosclerosis is a complex disease, involving many cell types and circulating mediators and resulting in an inflammatory state. Atherosclerotic lesions form *de novo* from focal accumulation of lipoproteins, monocyte-derived macrophages, and lymphocytes within the arterial wall. These lesions can develop as early as the second decade of life and progress into clinical disease over time. However, despite recent advances in cardiology, atherosclerosis remains an important medical problem.

There has been renewed interest in the protective role of high-density lipoprotein (HDL) against atherosclerosis and CVD. Patients with central obesity, insulin resistance, hypertension, and type 2 diabetes mellitus, conditions associated with the metabolic syndrome, have a unique dyslipoproteinaemia characterised by hypertriglyceridaemia, elevated levels of apolipoprotein B, small dense low-density lipoprotein (LDL) cholesterol, and low levels of HDL-cholesterol. Moreover, these lipoproteins represent major cardiovascular risk factors in these conditions.

Although the focus in treating lipid disorders is on reducing LDL-cholesterol concentrations, additional lipid-related independent risk factors, such as triglyceride, HDL-cholesterol, and lipoprotein(a) levels should be used clinically to assess cardiovascular risk. Among other factors, lipoprotein lipase, hepatic lipase, lecithin: cholesterol acyltransferase, and cholesteryl ester transfer protein play an important role in abnormal HDL metabolism in insulin resistance and type 2 diabetes mellitus. Insights gained from study of the postprandial state on atherosclerosis and CVD may also aid in risk assessment.

Familial hypercholesterolaemia (FH) was the first genetic disease of lipid metabolism to be clinically and molecularly characterised and is the most common and severe monogenic form of hypercholesterolaemia. Historically, the study of FH has provided insights into lipoprotein metabolism and the subsequent development of therapeutic agents for lowering LDL-cholesterol, such as the statins. The recent identification of new genes causing FH underscores the complexity of lipoprotein metabolism.

The complexity and chronicity of atherosclerosis in humans hinders the study of the key mechanisms involved. Thus, over the past decade, the mouse has become the model of choice for atherosclerosis research. All of the current mouse models are based on perturbations of lipoprotein metabolism through dietary and/or genetic manipulations. These models have shown that many non-lipid factors can also influence the severity and characteristics of the atherosclerotic lesions.

In this issue of *The Clinical Biochemist Reviews*, internationally recognised authors with experience in the above areas share their insights into the current state of lipids, lipoproteins, atherosclerosis and cardiovascular disease. I wish to thank each of the contributors for their time and effort in this endeavour.