Preface

Over the course of the last 30 years, cardiovascular disease (CVD) morbidity and mortality rates in the Western world have progressively declined on the basis of more intensive prevention approaches aimed at smoking cessation and lowering levels of cholesterol and blood pressure. However, there remains a considerable residual risk of clinical events with CVD continuing to be the most common cause of death in most countries. Accordingly, there is an ongoing need to identify additional targets for therapeutic intervention in order to achieve more effective prevention of one of the world’s major public health problems.

During the same period of time, seminal insights from pathology and clinical studies have characterized a pivotal role for inflammation in the pathogenesis of CVD. Cellular and humoral mediators of inflammation have been implicated in the formation, progression and clinical expression of a broad range of CVD disorders including the vasculature, myocardium, and pericardium. As a result, there has been increasing interest in the development of inflammation-related biomarkers for diagnosis and risk prediction, and targeted anti-inflammatory therapies for both disease prevention and treatment.

As the role of inflammation in CVD has been increasingly elucidated, there has been considerable interest focused on the CVD manifestations of a range of systemic inflammatory diseases. In particular, an abundant body of literature has now demonstrated clear associations between rheumatoid arthritis (RA) and CVD, both commonly encountered conditions worldwide. Recent studies have demonstrated that the presence of RA portends a high CVD risk, comparable to that observed in the setting of diabetes mellitus. Subsequent efforts have attempted to define the most effective approaches to reducing CVD risk in RA patients, whether that be via use of RA-targeted therapies or by intensification of conventional CVD prevention strategies. Small clinical studies have provided important insights, although larger trials are urgently needed.
Semb and colleagues have presented an elegant overview of the role of rheumatoid arthritis in a broad range of CVD disorders. They comprehensively review the evidence spanning from studies of biological samples and populations through to the clinical bedside. It provides the reader with a contemporary analysis of what is known and what remains to be further clarified. It represents an important compilation of the literature by a star-studded group of clinical investigators in the field. It will further stimulate us to identify better ways to prevent the alarmingly high CVD risk observed in RA. To many, inflammation remains the next frontier as a target for modification in CVD. RA represents the prototype from which our foray continues beyond this frontier. As a result, this book provides a great road map for that journey.

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