Preface

The World Health Organization (WHO) estimates that worldwide, diabetes occurs in more than 180 million people. Because the incidence of Type 1 diabetes mellitus (T1D) and Type 2 diabetes mellitus (T2D) is increasing globally, it is estimated that the number of people with diabetes will more than double by 2030. In parallel, it is anticipated that comorbid states associated with diabetes will also rise; thus, understanding and treating complications of diabetes will be a very high priority going forward in order to decrease morbidity and mortality, as well as to better control health care expenditures. Historically, most attention has been focused on four major complications known to afflict many individuals with T1DM and T2DM: retinopathy, neuropathy, nephropathy, and cardiovascular disease. However, epidemiological data now show that other tissues and organs may be significantly impacted by the diabetic state—and the skeletal system is now emerging as a primary target of diabetes-mediated damage (i.e., diabetic bone disease).

Studies have demonstrated that osteopenia and osteoporosis may be frequent complications of T1D, both in children and adults, and that T1D is associated with decreased bone density and increased fracture risk. In contrast to T1D, T2D has typically not been associated with osteopenia or osteoporosis and, in fact, has been more often associated with increased BMD. However, newer data show that bone quality and bone microarchitecture may be compromised in both conditions, suggesting that underlying mechanisms related to increased risk to fracture may be contributory to both forms of diabetes.

In this volume, we provide the reader with up-to-date information about what is currently known about diabetic bone disease and what are the challenges still facing the research and clinical care communities. In the first two chapters, the clinical and epidemiological data about diabetic bone disease is evaluated and reviewed for T1D and T2D, respectively. Chapter 3 discusses how the propensity to fracture in diabetic bone disease can impact fracture risk assessments and how it can be adjusted for using current clinically relevant fracture risk models. Chapter 4 provides a comprehensive overview of orthopedic complications observed in diabetes, and Chapter 5 focuses on the consequences of diabetes on periodontal disease. The utility
of skeletal biomarkers in assessing diabetic bone disease is reviewed in Chapter 6. Chapter 7 shows how drugs used to treat diabetes may also have skeletal consequences. Diabetes may fundamentally impact early progenitor cells of various bone lineages, and through this mechanism globally impact bone; Chapter 8 reviews the literature related to this possibility. How diabetes ultimately may impact the architecture, integrity, and quality of bone is discussed in Chapters 9–11.

As editors, we are truly indebted to the authors who have allowed us to catalogue their unique insights and expertise in diabetic bone disease into one comprehensive text. We hope the reader will find this volume, the first ever to be devoted specifically to diabetic bone disease, to be a useful and thought-provoking resource.

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