Cancer Microenvironment (2011) 4:219
DOI 10.1007/s12307-011-0093-4

EDITORIAL

Introduction to the Special Issue on The Microenvironment of Bone Metastasis

Rebecca Silbermann

Published online: 2 December 2011

Introduction

Bone metastases occur frequently in patients with advanced cancer. It is estimated that up to 70% of patients with advanced prostate or breast cancer and 15 to 30% of patients with lung, colon, stomach, bladder, uterus, rectum, thyroid, or kidney cancer have bone metastases [1]. While the exact incidence of bone metastasis is not known, it is estimated that 350,000 cancer patients die with bone metastases in the United States annually [2]. The consequences of bone metastases are severe and include pain, hypercalcemia, pathologic fractures, and nerve-compression syndromes. This special issue of Cancer Microenvironment focuses on recent developments in our understanding of the microenvironment of bone metastases and includes articles on the homing of tumor cells to the bone, the roles of hematopoietic stem cell and stromal cell populations in the development of metastatic lesions and a review of the interactions between prostate cancer cells and the tumor microenvironment in the development of bone metastases from prostate cancer. In addition, this issue includes submissions discussing the role of TGF-β in the bone microenvironment of breast cancer and the effect of rigidity on the regulation of genes that stimulate tumor-induced bone disease. Finally, three articles are dedicated to the unique pathophysiology of the bone microenvironment in myeloma bone disease. Myeloma bone disease is distinct from bone disease caused by other tumors that are metastatic to bone as myeloma bone leisons are characterized by severely decreased or absent osteoblast activity in addition to increased osteoclastic bone resorption [3]. This issue includes articles discussing recent progress in the understanding of the mechanisms regulating myeloma bone disease, the angiogenic properties of the myeloma marrow microenvironment, and a summary of our current understanding of osteoblast inhibition in myeloma and the potential therapeutic utility of bone anabolic agents in managing this devastating disease.

References

1. Roodman GD (2004) Mechanisms of bone metastasis. N Engl J Med 350:1655–1664
2. Mundy GR (2002) Metastasis to bone: causes, consequences and therapeutic opportunities. Nat Rev Cancer 2:584–593
3. Bataille R, Chappard D, Marcelli C, Dessauw P, Sany J, Baldet P, Alexandre C (1989) Mechanisms of bone destruction in multiple myeloma: the importance of an unbalanced process in determining the severity of lytic bone disease. J Clin Oncol 7:1909–1914

R. Silbermann (✉)
Department of Medicine, Division of Hematology and Oncology,
University of Pittsburgh,
Pittsburgh, PA, USA
e-mail: silbermannr@upmc.edu