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Nota di contenuto	Section 1: Historical Background -- Chapter 1: Mast cell/platelet heparanase/Heparan sulfate biosynthesis and turnover -- Chapter 2: gene cloning/overview -- Chapter 3: gene cloning/melanoma metastasis -- Chapter 4: gene cloning/cancer/immune system -- Chapter 5: heparin/HS modifying enzymes -- Section 2: Crystal Structure/substrate specificity/gene regulation -- Chapter 6: crystal structure -- Chapter 7: molecular dynamics, KKDC peptide -- Chapter 8: Biochemistry/active site -- Chapter 9: substrate specificity -- Chapter 10: gene regulation, promoter/Egr1/methylation -- Chapter 11: SNPs -- polymorphism -- Chapter 12: Splice variants -- Section 3: Cell & tumor biology (general functions & mode of action) -- Chapter 13: Exosomes/heparan sulfate/heparanase -- Chapter 14: Exosomes/drug resistance -- Chapter 15: Nuclear

heparanase/transcriptional activity -- Chapter 16: Non-Enzymatic functions/Signal transduction/cellular trafficking/autophagy -- Chapter 17: Heparan sulfate/stem cells/inflammation -- Chapter 18: Danger signals/HS/platelet heparanase -- Chapter 19: Heparanase/Integrins/Melanoma -- Section 3: Immune Cells/Immuno-Modulation -- Chapter 20: Heparain. Heparanase and Selectins in Cancer Metastasis and Inflammation --- Chapter 21: Trans-Endothelial Migration, Lymphocytes, Neutrophils/T-cells --Chapter 22: Macrophages, dendritic cells, autoimmunity -- Chapter 23: Macrophages, Heparanase and the tumor microenvironment, neutralizing antibodies -- Chapter 24: NK Cells -- Section 4: Cancer (heparanase in specific types of cancer) -- Chapter 25: Myeloma, inhibition, drug resistance -- Chapter 26: Breast Cancer/Pancreatic Cancer/Cancer and Inflammation -- Chapter 27: Brain Metastasis/MIR-1258 -- Chapter 28: Gastric cancer/immunization -- Chapter 29: Head and Neck Cancer -- Chapter 30: Glioma -- Chapter 31: Sarcoma -- Section 5: Inhibitors/clinical trials/cancer -- Chapter 32: Chemistry/synthesis of heparanase inhibitors PI-88, PG -- Chapter 33: PG series/biology/Tumor models and clinical trial -- Chapter 34: Chemically modified heparins/Heparin mimetics -- Chapter 35: Medicinal Chemistry (Ronesparstat/small molecules/clinical trials) - Section 6: Other indications/diseases -- Chapter 36: IBD/inflammation and cancer/diabetes/obesity -- Chapter 37: Immune Diabetes -- Chapter 38 Inflammation, Sepsis/Amyloidosis -- Chapter 39: Kidney dysfunction -- Chapter 40: Fibrosis -- Chapter 41: Viral infection -- Chapter 42: Cariomyocytes/Endothelial cell-cardiomyocyte crosstalk in diabetic cardiomyopathy -- Chapter 43: Eye research -- Chapter 44: atherosclerosis, nuclear localization -- Chapter 45: Yona Nadir (coagulation/tissue factor) -- Section 7: Heparanase-2 (Hpa2) -- Chapter 46: Hpa2 gene cloning -- Chapter 47: UFS -- urofacial syndrome/peripheral neuropathy -- Chapter 48: Hpa2: tumor suppressor.

Sommario/riassunto

Proteases and their involvement in cancer progression have been well addressed and documented; however, the emerging premise presented within this book is that Heparanase is a master regulator of aggressive cancer phenotypes and crosstalk with the tumor microenvironment. This endoglycosidase contributes to tumor-mediated remodeling of the extracellular matrix and cell surfaces, augmenting the bioavailability of pro-tumorigenic and pro-inflammatory growth factors and cytokines that are bound to Heparan sulfate. Compelling evidence ties Heparanase with all steps of tumor progression including tumor initiation, growth, angiogenesis, metastasis, and chemoresistance, supporting the notion that Heparanase is an important contributor to the poor outcome of cancer patients and a validated target for therapy. Unlike Heparanase, heparanase-2, a close homolog of Heparanase, lacks enzymatic activity, inhibits Heparanase, and regulates selected genes that promote normal differentiation and tumor suppression. Written by internationally recognized leaders in Heparanase biology, this volume presents a comprehensive understanding of Heparanase's multifaceted activities in cancer, inflammation, diabetes and other diseases, as well as its related clinical applications to scientists, clinicians and advanced students in cell biology, tumor biology and oncology.
