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Altri autori (Persone)	HayatM. A. <1940->
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Nota di contenuto	Preface.-I. Tumor dormancy -- Is tumor dormancy clinically relevant?- Microenvironmental influence on breast cancer Dormancy and metastasis -- Determination of breast cancer dormancy: analysis of Circulating free dna using snp 6.0 arrays -- Clonogenicity of cultured prostate cancer cells is controlled by dormancy : significance and comparison with cell culture models of breast cancer cell dormancy -- Dormancy and metastasis of melanoma cells to lymph Nodes, lung, and liver -- Late recurrence is a sign of melanoma dormancy: need of Life-long follow-up of elanoma patients. II. Quiescence -- Hematopoietic stem cell quiescence and long term Maintenance: role of scl/tal1 -- Regulation of muscle stem cell quiescent and Undifferentiated state: roles of hesr1 and hesr3 genes -- The kinase mirk/dyrk1b mediates a reversible quiescent State in a subset of ovarian, pancreatic, and colon cancers. III. Cellular senescence -- Stress -induced senescence: molecular pathways.-11 Accumulation of reactive oxygen species and induction Of premature senescence: role of ddb2 -- P21 mediates

senescence by a mechanism involving accumulation of reactive oxygen species -- Role of micrornase and zeb1 downmodulation in oxidative Stress-induced apoptosis and senescence.- Hypoxic response in senescent brain is impaired: possible Contribution to neurodegeneration.-Enhancing reprogramming to pluripotency by controlling senescence -- Histone deacetylase inhibitor induces replicative Senescence of mesenchymal stem cells -- Senescence arrest of endopolyploid cells renders Senescence into one mechanism for positive tumorigenesis -- The two faces of senescence-associated epigenetic Alterations: tumor suppressors and oncogenic drivers. - Chemotherapy- and radiation-induced accelerated senescence: implications for treatment response, tumor progression, and cancer survivorship -- Suppression of cellular senescence in glioblastoma: role Of src homology domain-containing phosphatase 2 -- Chemotherapy ofmalignant pleural mesothelioma induces both senescence and apoptosis -- Microrna as a modulator of cell proliferation and Senescence: role in lung cancer cells -- Role of senescence induction in cancer therapy -- Cellular senescence limits the extent of fibrosis Following liver damage -- Formation of secretory senescent cells in prostate tumors: the role of androgen receptor activity and cell cycle regulation.-Index .

Sommario/riassunto

With a particular emphasis on tumor dormancy in breast, lung, prostate, and liver cancers, as well as in melanoma, this first volume of a new Springer series focuses on the interrelationship between biological processes of aging and tumors—both dormant and quiescent. With detail supplied by numerous international researchers at the forefront of cancer research, the book examines a host of differing aspects of the topic. Featured contributions analyze the role of the quiescent state in regulating hematopoietic and muscle stem cells. They also explore the mediation, by the kinase, in the reversible quiescent state of a subset of ovarian, pancreatic, and colon cancers. The book includes key research on the molecular mechanisms underlying stress-induced cellular senescence, in addition to those governing the accumulation of reactive oxygen species, and the induction of premature senescence. It also provides information on suppressing cellular senescence in the most common, and most aggressive malignant primary brain tumor in humans, glioblastoma multiforme. With comprehensive and cutting-edge information on therapeutic interventions and on the correct diagnosis of relevant neoplasms, and with numerous color illustrations, this is the most up-to-date assessment of current medical knowledge in this crucial area of medical research.
