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Nota di contenuto	Contents; Foreword; Preface; Chapter 1 Oncogenic Viruses, Cellular Transformation and Human Cancers Yanyan Zheng and Jing-hsiung James Ou; 1. Introduction and Historical Aspects; 2. Human Oncogenic Viruses; 2.1. Hepatitis B Virus (HBV); 2.2. Hepatitis C Virus (HCV); 2.3. Human Papilloma Virus (HPV); 2.4. Epstein-Barr Virus (EBV); 2.5. Kaposi's Sarcoma-associated Herpesvirus (KSHV); 2.6. Human T-cell Leukemia Virus-1 (HTLV-1); 3. Mechanisms of Virus-induced Cellular Transformation; 3.1. Perturbation of Signaling Pathways; 3.1.1. Mimicking the signaling ligands 3.1.2. Mimicking the cellular signaling receptors3.1.3. Mimicking the intracellular signaling adaptors; 3.1.4. Activation of cell surface receptors; 3.2. Deregulation of the Cell Cycle; 3.2.1. Abrogation of the RB function; 3.2.2. Enhancement of CDK activities; 3.2.3. Targeting of cyclin; 3.3. Escape of Apoptosis; 3.3.1. Inactivation of the "gatekeeper" p53; 3.3.2. Expression of viral version of Bcl-2 (vBcl-2); 3.4.

Immortalization of Cells; 3.5. Induction of Genetic Instability; 3.6. Insertional Mutagenesis; 3.7. Induction of Chronic Inflammation; 4. Concluding Remarks; Bibliography
Chapter 2 Hepatitis B Virus and Hepatocellular Carcinogenesis T. S. Benedict Yen1. Hepatitis B Virus and Hepatocellular Carcinoma; 2. Biology and Epidemiology of HBV; 3. HBV Virology; 4. Prevention and Treatment of HBV; 5. Mechanisms of HBV Carcinogenesis; 5.1. Overview; 5.2. HBV-specific Factors; 5.2.1. X protein; 5.2.1.1. Function of X protein in the HBV life cycle; 5.2.1.2. Effect of X protein on transcription; 5.2.1.3. Effect of X protein on viral replication; 5.2.1.4. X protein and DDB1; 5.2.1.5. X-protein carcinogenesis; 5.2.2. Other viral proteins potentially involved in carcinogenesis
5.2.2.1. Truncated MSP5.2.2.2. preS2 mutants; 5.2.2.3. Core gene mutants; 5.2.3. Insertional mutagenesis; 5.3. Role of Liver Injury and Inflammation; 5.4. Dietary Carcinogens; 5.5. Other Aspects of HBV Oncogenesis; 5.5.1. Role of genotypes; 5.5.2. Role of sex hormones; 6. Summary; Note; References; Chapter 3 Molecular Mechanism of Hepatitis C Virus Carcinogenesis Keigo Machida, Jing-hsiung James Ou and Michael M. C. Lai; 1. Introduction; 2. Molecular Carcinogenesis of HCV; 2.1. Induction of Mutator Phenotype; 2.2. Chromosome Translocation; 2.3. Reactive Oxygen Species (ROS); 2.4. Nitric Oxide 2.5. Inhibition of DNA Damage Repair2.6. Oncogenic Activities of the HCV Core Protein; 2.6.1. TNF-; 2.6.2. MAPK and AP-1; 2.6.3. NF-B; 2.6.4. Oxidative stress; 2.6.5. Insulin resistance; 2.6.6. PPAR; 2.6.7. Proteasome activator PA28; 2.6.8. SOCS-1; 2.6.9. p53; 2.7. Oncogenic Activities of the HCV NS5A Protein; 3. Other Causative Factors in HCV-associated HCC; 3.1. Chronic Liver Inflammation; 3.2. Alcohol; 4. Endoplasmic Reticulum Stress and HCV Pathogenesis; 5. Gene Expression Profile of HCC; 6. HCV and Lymphomagenesis; 6.1. Induction of IgHypermutation by HCV
6.2. Induction of DNA Translocation between IgGenes and Proto-Oncogenes by HCV

Sommario/riassunto

Viruses are the causes of approximately 25% of human cancers. Due to their importance in carcinogenesis, there is a desperate need for a book that discusses these viruses. This book is therefore timely, providing a comprehensive review of the molecular biology of oncogenic viruses and the cancers they cause. Viruses that are discussed in the individual chapters include hepatitis B virus, hepatitis C virus, human papilloma viruses, Epstein-Barr virus, Kaposi's sarcoma virus and human T-cell leukemia virus type 1. This book provides up-to-date information for graduate students, postdoctoral fe