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First published in 1932, Annual Reviews are considered as authoritative set of publication in disseminating knowledge for Science and human culture. The focus of the fourth edition is to impart the broad concept and emerging theme in pathogenesis of cancer. Importantly, because of funding support by Robert Wood Johnson Foundation, this Cancer Biology series are available freely under a Creative Commons (CC) by license. The carefully chosen twenty-four chapter, written by eminent Scientists, focuses on the five areas of cancer metabolism, roles of immune cell, cell signaling, drug resistance, and therapeutics.

Recent studies show that metabolic changes within tumor drive oncogenesis and may provide novel strategies for detection and therapeutic targeting of cancer. Heiden lab gleaned the metabolic aspect of stromal cells and their role within tumor micro-environment. Sciacovelli and colleagues discussed the effect of germline mutation of mitochondrial enzyme succinate dehydrogenase and fumarate hydratase promotes oncogenic activation in hereditary cancer. Another review examined the molecular details and regulatory mechanism of PTEN-induced putative kinase 1 (PINK1) dependent and BNIP3/NIX-dependent (stress-induced) mitochondrial autophagy on cellular senescence, stemness, survival, metabolic homeostasis and progression of cancer. Another fascinating review focused on fermentative glycolysis and provided the mechanistic insight on the lactate induced acidity in tumor progression. Further, they revealed how lactate dehydrogenases and transporter (MCT4) serve as novel therapeutic agent and biomarker for patient survival, metastasis, and therapeutic resistance in various cancers. Moreover, they also showed the potential use of metabolic bioluminescence imaging (imBI) to differentiate between normal and cancer cell.

Several articles cover recent advancement of immune cells in cancer progression. Plitas and Rudensky elaborated the regulatory mechanisms of tumor specific T-regulatory (T-reg) cells in

the inhibition of anti-tumor immune response, survival of transformed cell, and limiting response to immunotherapeutic drug. Given the great success of T- cell in leukemic cancer, one reviews summarized the synthetic biology methods of engineering T-cell for efficient and controlled delivery to target solid tumor. Following similar theme, one article delves in to recent development and hurdle of immune checkpoint inhibitor, tumor specific monoclonal antibody, antibody drug conjugates, and chimeric antigen receptor (CAR) T cells to treat pediatric solid tumor. Ganesan and Mehnert urged the pressing need to develop efficient biomarker for immune checkpoint blockage with antibodies and discussed the vivid account of target assessment, tumor intrinsic factors, and host factor to measure the response post targeting with PD-1, PD-L1 or CTLA-4 in category of cancer.

Furthermore, few reviews provided the key insight into mechanisms, therapeutic targeting, and challenges of well-established targets genes such as Myc, Mcl-1, Wnt- β -catenin, and epithelial-mesenchymal transition in tumor progression. Importantly, they also discussed how recently developed knockout mouse model along with multi-color lineage tracing and dynamic intra-vital imaging will be an important tool for determining dissemination and metastasis of breast, and pancreatic cancer. Herlyn lab emphasized the existence of heterogeneous population within tumor microenvironment with varying metabolic, metastatic potential, and lineage plasticity that poses a further challenge to target these cells. Focusing on the drug development, a review highlighted the molecular mechanisms of epacadostat, an immunotherapeutic small molecule that inhibit Indoleamine-2, 3 dioxygenase 1 (IDO1) activity and degrade tryptophan in melanoma patient. They further discussed the cause of its failure in phase III clinical trials and lesson learned to use for treatment of cancer. Moreover, Biswas and colleagues pointed out the mechanisms and role of cancer-associated cachexia (a devastating condition causing irreversible

loss of skeletal muscle mass and function) as a major cause to organ failure and death in cancer.

A must read article in this volume covers the role of non-coding repetitive elements (such as LTR, LINEs, and SINEs) in perturbing genomic integrity, cell cycle regulation, and generation of therapy resistant cells.

An additional paper provides the signaling nexus of AMPK isoform which act like tumor suppressor in early stage and later switches into oncogenic role and promotes survival of tumor cell under stress. Post-transcriptional modification furnish another level of complexity in cancer development. One article discussed the dynamic modification of eukaryotic mRNA transcript control the various hallmarks of cancer. Another article described the mechanisms and pathways of the activation and deactivation through post-translational modification of spindle assembly checkpoint protein components in chromosome segregation during cell division.

In summary, this volume presented the most recent progress in cancer biology such as retrotransposon reactivation, perineural invasion (PNI), kinome rewiring as Cancer resistance marker in lung cancer, drug tolerant persister cell, and immunotherapy based targeting in cancer. Further, it also highlighted how oncogene addiction may be an attractive target for personalized therapy in non-small-cell lung cancer. I missed the past-time in cancer biology article which was populated in earlier volume. To enhance the readability, I request authors to define the keywords as snippet within the article. I also suggest including a summary statement and future perspective in all reviews which will help reader to grasp the key point. Given the rapid growth of precision medicine, I solicit editors to include an annual summary of approved oncology drug. I am overwhelmed with the quality material of this must-read annual series. Overall, this elegantly illustrated invaluable on-line resource will continue to inspire, and inculcate the next generation of cancer biologist.

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94 The author confirms the sole contributor of this work and has approved it for publication.

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99 **Conflict of Interest Statement**

100 The author declares that he conducted the research without any commercial or financial
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