The Cell Cycle and Cancer
Study Guide/Answer Key

1. What is the cell cycle?
   The regular sequence of growth and division cells undergo.

2. What are the three stages of cell division?
   Interphase – Mitosis - Cytokinesis

3. What is mitosis?
   The stage of cell division in which the nucleus divides into two nuclei; in this stage one copy of the DNA is copied (replicated) and distributed to each new daughter cell.

4. List the four phases of mitosis?
   Prophase – Metaphase – Anaphase - Telophase

5. In which stage does the cell spend most of its time?
   Interphase

6. What happens in prophase?
   The chromatin condenses (thickens) to form chromosomes; spindle fibers form; the nuclear membrane breaks down;

7. What happens in metaphase?
   The chromosomes line up in the center of the cell; each chromosome attaches to the spindle fiber at the centromere;

8. What happens in anaphase?
   The centromere splits and the two chromatids separate to opposite ends;

9. What happens in telophase?
   The chromosomes stretch and lose their rod like appearance; a nuclear membrane forms around each region of chromosomes;

10. What is cytokinesis?
    The cell membrane pinches in around the middle of the cell; each daughter cell ends up with the same number of identical chromosomes as the parent cell;

11. What is replication?
    Replication is when the cell makes a copy of the DNA in its nucleus during interphase;

Cell Cycle And Cancer Webquest Answers

Yueming Sun,Junhui Hu,Mingyan Zhu

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The Cell Cycle and Cancer Renato Baserga,1971 *Cell Cycle Deregulation in Cancer* Greg H. Enders,2010-03-10 Cancer is fundamentally a disease of abnormal cell proliferation Cancer cells multiply when and where they should not This proliferation entails escape from normal bounds imposed by the tissue environment the internal biology of the cell DNA damage chromosomal imbalances disorganized mitotic spindles and the proliferative history of the cell normal generational times Some of the key oncogenic events in cancer directly perturb proteins that regulate progression through the cell division cycle others alter cell cycle progression indirectly through effects on signaling pathway that impinge on the cell cycle This biology is fundamentally important in cancer therapy Many of the workhorse treatments for cancer rely on killing proliferating cells Furthermore there is growing recognition that stem cell transit amplifying cell hierarchies may persist or be generated during tumorigenesis generating important functional heterogeneity in cell cycle control among tumor cells with far reaching scientific and clinical implications This volume outlines major cell cycle perturbations that drive tumorigenesis and considers the prospects for using such knowledge in cancer therapy Cell Cycle Regulators in Cancer Philip W. Hinds,1997-01-01 *Cell Cycle Checkpoints And Cancer* Mikhail V. Blagosklonny,2001-01-01 *Cell Cycle Regulators in Cancer* Kiran Musunuru,Philip W. Hinds,1997 Cancer can be tersely yet accurately described as improper cell proliferation To understand cancer we must first understand the genetic and biochemical mechanisms responsible for proper cell proliferation The last five years have witnessed the characterization of several families of novel proteins involved in cell cycle regulation and the clarification of the biochemical processes in which they participate This book illuminates the roles of various cell cycle regulators cyclins cyclindependent kinases CDKs and CDK inhibitors and describes the connections between these proteins and oncogenesis Possible ways of clinical intervention that might be developed into potent cancer therapies are also explored By chronologically documenting the discovery of cell regulators and providing clear brief synopses of current findings this work offers an easily accessible guide for both students and experienced researchers An extensive list of excellent reviews for further reading rounds off the reference value of this timely publication The cell cycle in cancer prognosis Arthur B. Pardee,1991 **Tumour Suppressor Genes, the Cell Cycle and Cancer** Arnold Jay Levine,1992-01-01 Cell Cycle Checkpoints in Cancer Mikhail V. Blagosklonny,2002 This book addresses mechanisms of normal and cancer cell cycling checkpoint control the link of mitogenic signaling and cell cycle machinery Considerable attention is devoted to the analysis of checkpoint mechanisms from yeast to man allowing us to understand the logic of the cell cycle Emerging Molecular Mechanisms of Cell Cycle Regulation in Cancer: Functions and Potential Applications Yueming Sun,Junhui Hu,Mingyan Zhu,2022-03-07 One Renegade Cell Robert A. Weinberg,1998-10-08 Cancer research has reached a major turning point The quality and quantity of information gathered about this disease in the past twenty years has revolutionized our understanding of its origins and behavior No one is better qualified to comment on these dramatic leaps forward than molecular biologist Robert A Weinberg director of one of the leading cancer research centers in the world In One Renegade Cell Weinberg presents an accessible and state of the art account of how the disease begins and how one day it will be cured Weinberg tells how the roots of cancer were uncovered in 1909 and when the first cancer causing virus was discovered He then moves forward to the discovery of the role of chemical carcinogens and radiation in triggering cancer and relates the remarkable story of the discoveries of oncogenes and tumor suppressor genes the master controllers of normal and malignant cell proliferation This book which presumes little prior knowledge of biology describes the revolution in biomedical research that has finally uncovered the forces driving malignant growth Drawing on insights that simply were not available until recently the discoveries presented in One Renegade Cell have already begun to profoundly alter the way that we diagnose and treat human cancers *Dynamics of Kv10.1 Expression Through the Cell Cycle of Cancer and Non-cancer Cells* Diana Elizabeth Urrego-Blanco,2014 **CANCER AND QUIESCENCE** ,2022 Abstract An estimated 1 9 million people in the United States will be diagnosed with cancer in 2022 Cancer is characterized by uncontrolled cell growth resulting from loss of function of key cell cycle regulatory proteins The retinoblastoma protein pRb and p130 are two proteins that regulate cellular entry into the cell cycle Both pRb and p130 repress expression of cell cycle genes with pRb interacting with and suppressing E2F DP transcriptional activators and p130 assembling in the DREAM transcriptional repressor complex When normal cells receive signals to enter the cell cycle cyclin and cyclin dependent kinase CDK complexes phosphorylate pRb and p130 causing both to release from their respective complexes Recently CDK inhibitors have been proven to be effective chemotherapeutics as they effectively lock pRb and p130 in their cell cycle repressive functions Unfortunately many ovarian tumors remain resistant to CDK inhibitor treatment However to date no study has systematically evaluated how ovarian cancer affects both pRb and p130 function Using flow cytometry we confirmed that the SKOV3 ovarian cancer cell line responds to CDK inhibition but the OVCAR3 ovarian cancer cell line does not respond to CDK inhibition Using western blot and expression analysis we evaluated pRb and p130 response to CDK inhibition in SKOV3 cells as compared to normal human foreskin fibroblasts HFFs We observed that although both pRb and p130 protein remain similarly expressed throughout the cell cycle repression of 2 cell cycle genes CCNB2 and MCM5 in CDK inhibited cells appears to be perturbed in the SKOV3 cell line as compared to HFF cells This study will lay the groundwork for future studies aimed to differentiate whether pRb or p130 dysfunction is causing the cell cycle gene misregulation we observed in the SKOV3 cell line **Metabolic Checkpoints in Cancer Cell Cycle** Mahesh Saqcena,City University of New York. Biochemistry,2014 Growth factors GFs as well as nutrient sufficiency regulate cell division in metazoans The vast majority of mutations that contribute to cancer are in genes that regulate progression through the G1 phase of the cell cycle A key regulatory site in G1 is the growth factor dependent Restriction Point R where cells get permissive signals to divide In the absence of GF instructions cells enter the quiescent G0 state Despite fundamental differences between GF signaling and nutrient sensing they both have been confusingly referred to as R and therefore by definition considered to be a singular event in G1 Autonomy from GF signaling is one of the hallmarks in cancer however cancer cells also have metabolic rewiring enabling them to engage in anabolic biosynthetic pathways In the absence of GF instructions and nutrients cells commonly undergo apoptotic cell death Thus it is of importance to elucidate the differences between GF and nutrient deregulation in cancer to develop novel strategies in targeting tumor cell proliferation and survival *Cell Cycle Regulation and Cancer* Nicholas B. La Thangue,1995 **The Role of Human Emi-1 in the Cell Cycle and Cancer** Jerry Y. Hsu,2004 **PATH - Proliferation-Accumulation-Tumor-Hypothesis** Thomas Wascher,2018-08-15 What is or what does a gene mutation do A gene mutation i e the damage of a gene leads to a gene expressing more less or modified proteins These altered proteins alter the behaviour of the cell And this is how cancer is ultimately to develop Changed proteins due to changes in the genes But what would happen if these changes in the proteins were not caused by mutations in the genes but by direct damage to the proteins Then the result cancer would be the same but otherwise everything would be different And cancer could be curable We cannot reverse a mutation but we can reverse a mechanical process such as protein accumulation At least we could considerably influence such a carcinogenesis The proliferation accumulation tumour hypothesis describes how this might be possible According to PATH cancer is not a mutation disease but a protein accumulation disease But a protein accumulation protein oxidation protein aggregation in immortal stem cells And a protein accumulation that affecting 400 protein types mainly cell cycle proteins and millions of protein copies simultaneously A protein accumulation in which autophagocytosis in the G0 phase as the most important proteolysis factor is no longer sufficiently effective PATH is based on the latest findings on the ubiquitin proteasome system cyclins in particular Cyclin D CDK4 6 and autophagy according to Ohsumi s findings and their role in the development of tumours And PATH also shows why certain forms of treatment and substances e g acetylsalicylic acid aspirin metformin 2 deoxy 2 glucose methadone terfenadine etc have a certain effect in studies And how cancer could possibly be hindered in its development to such an extent that a lethal effect is omitted For example PATH also explains better why a person has a 50% chance of developing cancer or at least a 25% chance of dying from it The most important basis for the development of PATH was the rigorous detachment from the cancer multimutation principle which has not been effective to date and the exclusive reorientation towards the most probable and logical facts and conclusions And the realization What is the cause of death for at least 25% of all people cannot be something that has to be caused by a rare undirected coincidence mutation **Prediction of Combination Efficacy in Cancer Therapy** Jie Yang,2013 The cell cycle is an essential process in all living organisms that must be carefully regulated to ensure successful cell growth and division Disregulation of the cell cycle is a key contributing factor towards the formation of cancerous cells Understanding events at a cellular level is the first step towards comprehending how cancer manifests at an organismal level Mathematical modelling can be used as a means of formalising and predicting the behaviour of the biological systems involved in cancer In response cell cycle models have been constructed to simulate and predict what happens to the mammalian cell over a time course in response to variable parameters Current cell cycle models rarely account for certain precursors of cell growth such as energy usage and the need for non essential amino acids as fundamental building blocks of macromolecules Normal and cancer cell metabolism differ in the way they derive energy from glucose In addition normal and cancer cells also demonstrate different levels of gene expression Two versions of a mammalian cell cycle and metabolism model based on ordinary differential equations ODEs that respond to fluctuations in glucose concentration levels have been developed here for the normal and cancer cell scenarios Sensitivity analysis is performed for both normal and cancer cells using these cell cycle and metabolism models to investigate which kinetic reaction steps have a greater effect over the cell cycle period Detailed analysis of the models and quantitatively assessing metabolite levels at various stages of the cell cycle may offer novel insights into how the glycolytic rate varies during the cell cycle for both normal and cancer cells The results of the sensitivity analysis are used to identify potential drug targets in cancer therapy Combinations of these individual targets are also investigated to compare the different effects of single and multiple drug compounds on the time it takes to complete a cell division cycle Cancer Metabolism: Molecular Targeting and Implications for Therapy Shanmugasundaram Ganapathy-Kanniappan,2017-11-03 Development of an effective anticancer therapeutic necessitates the selection of cancer related or cancer specific pathways or molecules that are sensitive to intervention Several such critical yet sensitive molecular targets have been recognized and their specific antagonists or inhibitors validated as potential therapeutics in preclinical models Yet majority of anticancer principles or therapeutics show limited success in the clinical translation Thus the need for the development of an effective therapeutic strategy persists Altered energy metabolism in cancer is one of the earliest known biochemical phenotypes which dates back to the early 20th century The German scientist Otto Warburg and his team Warburg Wind Negelein 1926 Warburg Wind Negelein 1927 provided the first evidence that the glucose metabolism of cancer cells diverge from normal cells This phenomenal discovery on deregulated glucose metabolism or cellular bioenergetics is frequently witnessed in majority of solid malignancies Currently the altered glucose metabolism is used in the clinical diagnosis of cancer through positron emission tomography PET imaging Thus the deregulated bioenergetics is a clinically relevant metabolic signature of cancer cells hence recognized as one of the hallmarks of cancer Hanahan and Weinberg 2011 Accumulating data unequivocally demonstrate that besides cellular bioenergetics cancer metabolism facilitates several cancer related processes including metastasis therapeutic resistance and so on Recent reports also demonstrate the oncogenic regulation of glucose metabolism e g glycolysis indicating a functional link between neoplastic growth and cancer metabolism Thus cancer metabolism which is already exploited in cancer diagnosis remains an attractive target for therapeutic intervention as well The Frontiers in Oncology Research Topic Cancer Metabolism Molecular Targeting and Implications for Therapy emphases on recent advances in our understanding of metabolic reprogramming in cancer and the recognition of key molecules for therapeutic targeting Besides the topic also deliberates the implications of metabolic targeting beyond the energy metabolism of cancer The research topic integrates a series of reviews mini reviews and original research articles to share current perspectives on cancer metabolism and to stimulate an open forum to discuss potential challenges and future directions of research necessary to develop effective anticancer strategies Acknowledgment I sincerely thank the Frontiers for providing the opportunity and constant support throughout the process of this research topic and eBook production I gratefully acknowledge all the authors for their valuable contributions Finally I would like to thank my brother Saravana Kumar G K whose personal sacrifices and unflinching encouragement made my career in science possible References Hanahan D Weinberg RA 2011 Hallmarks of cancer The next generation Cell 144 5 646 74 Warburg O Wind F Negelein E 1926 ber den stoffwechsel der tumoren in k rper Klinische Wochenschrift 5 829 32 Warburg O Wind F Negelein E 1927 The metabolism of tumors in the body J Gen Physiol 8 6 519 30 **Cancer** David M. Prescott,Abraham S. Flexer,1982 *Regulation of the Cell Cycle* Mihajlo Krsmanovic,Columbia University. Biotechnology Program,2005

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