the cell cycle and cancer answer key

the cell cycle and cancer answer key explains the crucial relationship between the cell cycle and the development of cancer. Understanding this connection is essential for grasping how uncontrolled cell division leads to tumor formation and malignancies. This comprehensive article explores the phases of the cell cycle, the regulatory mechanisms that ensure proper cell division, and how failures in these controls can initiate cancer. Additionally, the article covers the molecular checkpoints, the role of oncogenes and tumor suppressor genes, and the latest insights into therapeutic interventions targeting the cell cycle in cancer treatment. By providing a detailed answer key, this article serves as an authoritative resource for students, educators, and professionals seeking to deepen their knowledge of cellular biology and oncology. The following sections will guide the reader through the fundamental concepts and advanced topics related to the cell cycle and cancer.

Overview of the Cell Cycle

The cell cycle is a series of ordered phases that cells undergo to grow and divide. It is fundamental to organismal development, tissue repair, and cellular reproduction. The cell cycle consists of distinct stages: G1 (Gap 1), S (Synthesis), G2 (Gap 2), and M (Mitosis). During G1, the cell grows and prepares for DNA replication. The S phase is characterized by DNA synthesis, where genetic material is duplicated. G2 is another growth phase where the cell readies itself for mitosis, which is the process of nuclear division and cytokinesis that results in two daughter cells. Proper progression through these stages ensures accurate genome duplication and cell division.

Phases of the Cell Cycle

Each phase of the cell cycle has specific functions and checkpoints that monitor cellular integrity. The G1 phase is critical for assessing environmental conditions and cellular size before committing to DNA replication. The S phase must ensure DNA is accurately copied without errors. G2 serves as a preparation period for mitosis, where the cell checks for DNA damage or replication errors. Finally, mitosis ensures chromosomes are equally distributed to daughter cells. This orderly progression maintains genomic stability and prevents mutations.

Importance of the Cell Cycle in Normal Physiology

The cell cycle is vital for normal physiological processes including growth, development, and tissue regeneration. Controlled cell division replaces damaged or dead cells, maintaining homeostasis within tissues. In multicellular organisms, proper regulation of the cell cycle prevents excessive or insufficient cell proliferation, thereby avoiding tissue dysfunction and disease. Understanding the cell cycle provides insight into how cells maintain life and how deviations can lead to pathological conditions such as cancer.

Regulation of the Cell Cycle

Cell cycle regulation is a complex network of signals and molecular mechanisms that ensure orderly progression through the cell cycle phases. The cell employs various checkpoints to monitor DNA integrity, cell size, and the completion of critical processes before allowing progression. These regulatory mechanisms prevent the propagation of damaged or incomplete genetic material.

Cell Cycle Checkpoints

There are three major checkpoints in the cell cycle: the G1 checkpoint, the G2 checkpoint, and the M checkpoint. The G1 checkpoint, also known as the restriction point, determines whether the cell has the necessary nutrients and growth signals to proceed. The G2 checkpoint verifies that DNA replication has been completed successfully and checks for DNA damage. The M checkpoint ensures that all chromosomes are properly aligned and attached to the spindle apparatus before mitosis completes. These checkpoints are vital for genomic stability.

Role of Cyclins and Cyclin-Dependent Kinases (CDKs)

Cyclins and cyclin-dependent kinases (CDKs) are key molecular regulators of the cell cycle. Cyclins are proteins whose concentrations fluctuate during the cell cycle, while CDKs are enzymes that, when activated by cyclins, phosphorylate target proteins to drive cell cycle progression. Different cyclin-CDK complexes act at specific checkpoints to facilitate transitions between phases. For example, the cyclin D-CDK4/6 complex regulates the G1 phase, and the cyclin B-CDK1 complex controls the entry into mitosis. Proper regulation of these complexes is essential for normal cell division.

Mechanisms Preventing Uncontrolled Cell Division

In addition to positive regulators like cyclins and CDKs, cells have inhibitory proteins such as CDK inhibitors (CKIs) that suppress CDK activity when conditions are unfavorable. These inhibitors help enforce cell cycle arrest in response to DNA damage or other stress signals. Tumor suppressor proteins like p53 and retinoblastoma protein (Rb) play critical roles in enforcing checkpoints and preventing uncontrolled proliferation. The balance between activators and inhibitors maintains cell cycle fidelity.

Cell Cycle Dysregulation and Cancer

The link between the cell cycle and cancer is well established, as cancer fundamentally results from uncontrolled cell proliferation. Dysregulation of cell cycle checkpoints and regulatory proteins leads to unchecked cell division and accumulation of genetic mutations, driving tumor formation and progression.

How Cell Cycle Dysregulation Leads to Cancer

When the mechanisms controlling the cell cycle fail, cells can bypass critical checkpoints, replicate damaged DNA, and evade apoptosis. This loss of control allows cells to proliferate uncontrollably, forming masses known as tumors. Cancer cells often exhibit mutations in genes encoding cyclins, CDKs, and their inhibitors, resulting in deregulated cell cycle progression. Moreover, the failure of tumor suppressor pathways, such as those involving p53, contributes to genomic instability and malignancy.

Common Genetic Alterations in Cancer

Genetic mutations that affect cell cycle regulation include:

- **Oncogene activation:** Genes such as cyclin D1 become overexpressed, promoting excessive CDK activity.
- **Tumor suppressor gene loss:** Mutations in p53 or Rb result in failure to halt cell cycle progression in response to DNA damage.
- **CDK inhibitor inactivation:** Loss of CKIs like p21 or p27 removes critical brakes on the cell cycle.
- **DNA repair gene mutations:** Defective repair mechanisms increase mutation rates, further destabilizing the genome.

Consequences of Cell Cycle Abnormalities in Cancer Progression

Abnormal cell cycle control contributes not only to tumor initiation but also to cancer aggressiveness and resistance to therapy. Unregulated cell division leads to rapid tumor growth, increased likelihood of metastasis, and evasion of programmed cell death. Additionally, cancer cells often develop mechanisms to circumvent cell cycle checkpoints, rendering them less responsive to treatments that target dividing cells. Understanding these abnormalities is critical for developing effective cancer therapies.

Key Molecular Players in Cell Cycle Control

Several molecular components orchestrate the cell cycle, and their dysfunction is central to cancer development. This section delves deeper into the key molecules involved in cell cycle regulation and their roles in

oncogenesis.

Oncogenes

Oncogenes are mutated or overexpressed versions of normal genes (proto-oncogenes) that promote cell proliferation. Examples include genes encoding cyclins, CDKs, and growth factor receptors. When mutated, oncogenes cause persistent activation of signaling pathways that drive the cell cycle forward without proper regulation, contributing to cancer progression.

Tumor Suppressor Genes

Tumor suppressor genes inhibit cell cycle progression and promote DNA repair and apoptosis. Prominent tumor suppressors include p53, Rb, and BRCA1/2. These proteins ensure that cells with damaged DNA do not continue to divide. Mutation or loss of these genes removes critical checkpoints, allowing accumulation of mutations and tumor development.

Checkpoint Proteins and DNA Repair Factors

Proteins involved in DNA damage detection and repair, such as ATM, ATR, and checkpoint kinases (CHK1/CHK2), play essential roles in maintaining genomic integrity. They activate cell cycle arrest to allow repair or induce apoptosis if damage is irreparable. Dysfunctions in these pathways contribute to carcinogenesis by permitting propagation of mutated cells.

Therapeutic Approaches Targeting the Cell Cycle in Cancer

Advances in cancer treatment increasingly focus on targeting cell cycle regulators to inhibit tumor growth. Understanding cell cycle dynamics has enabled the development of drugs that selectively disrupt cancer cell proliferation.

CDK Inhibitors

CDK inhibitors are a class of targeted therapies that block the activity of cyclin-dependent kinases, thereby halting cell cycle progression in cancer cells. Examples include palbociclib, ribociclib, and abemaciclib, which specifically inhibit CDK4/6. These drugs have shown efficacy in treating certain types of breast cancer by restoring control over the G1 checkpoint.

Checkpoint Modulators

Drugs that modulate cell cycle checkpoints aim to enhance the sensitivity of cancer cells to DNA damage. For instance, inhibitors of checkpoint kinases CHK1 and CHK2 can prevent cancer cells from repairing DNA damage, promoting apoptosis. These agents are often used in combination with chemotherapy or radiation therapy to improve treatment outcomes.

Emerging Therapies and Research Directions

Current research explores novel targets within the cell cycle machinery, including regulators of mitosis and the ubiquitin-proteasome system that controls cyclin degradation. Immunotherapies and gene editing techniques are also being investigated to correct or exploit cell cycle abnormalities in cancer cells. Continued advances promise more precise and effective cancer treatments based on cell cycle biology.

List of Common Cell Cycle–Targeted Cancer Therapies

- CDK4/6 inhibitors (e.g., palbociclib, ribociclib)
- Proteasome inhibitors (e.g., bortezomib)
- Checkpoint kinase inhibitors (e.g., prexasertib)
- Microtubule inhibitors (e.g., paclitaxel, vincristine)
- DNA-damaging agents combined with checkpoint modulators

Frequently Asked Questions

What is the cell cycle?

The cell cycle is a series of ordered phases that a cell goes through to grow and divide, including the phases G1, S, G2, and M.

How does the cell cycle relate to cancer?

Cancer occurs when the regulation of the cell cycle is disrupted, leading to uncontrolled cell division and tumor formation.

What role do checkpoints play in the cell cycle?

Checkpoints monitor and regulate the progression of the cell cycle to ensure that damaged or incomplete DNA is not passed on, preventing abnormal cell division.

Which proteins are primarily responsible for regulating the cell cycle?

Cyclins and cyclin-dependent kinases (CDKs) are key proteins that regulate the cell cycle by activating or inhibiting progression through its phases.

What is the significance of the G1 checkpoint in the cell cycle?

The G1 checkpoint determines whether the cell has the necessary resources and

DNA integrity to proceed with DNA synthesis; if not, the cell may enter a resting state or undergo apoptosis.

How do mutations in tumor suppressor genes affect the cell cycle?

Mutations in tumor suppressor genes, such as p53, can disable cell cycle checkpoints, allowing damaged cells to continue dividing and potentially leading to cancer.

What is the function of oncogenes in the cell cycle?

Oncogenes are mutated forms of normal genes (proto-oncogenes) that promote cell division; when overactive, they can cause uncontrolled cell proliferation contributing to cancer.

How can disruption of apoptosis contribute to cancer development?

If apoptosis, or programmed cell death, is inhibited, damaged or abnormal cells may survive and continue to divide, increasing the risk of cancer.

Why is understanding the cell cycle important for cancer treatment?

Many cancer treatments target specific phases or regulators of the cell cycle to stop the proliferation of cancer cells and induce their death.

What is the role of the M phase in the cell cycle and its relevance to cancer?

The M phase is where mitosis and cytokinesis occur, resulting in cell division; errors during this phase can lead to genetic instability, a hallmark of cancer cells.

Additional Resources

1. *The Cell Cycle: Principles of Control*
This book offers a comprehensive overview of the molecular mechanisms that regulate the cell cycle. It delves into the checkpoints, cyclins, and cyclin-dependent kinases that ensure proper cell division. The text also explores how dysregulation of these processes can lead to cancer, making it an essential resource for understanding cell cycle control in oncology.

2. *Cancer Biology and the Cell Cycle*
Focusing on the intersection of cancer research and cell cycle biology, this book explains how aberrations in cell cycle regulation contribute to tumorigenesis. It provides detailed explanations of oncogenes, tumor suppressors, and the molecular pathways involved. Ideal for students and researchers, it bridges fundamental biology with clinical implications.

3. *Cell Cycle Checkpoints and Cancer*
This volume emphasizes the role of cell cycle checkpoints in maintaining

genomic integrity and preventing cancer. It covers key proteins involved in checkpoint activation and how their malfunction can result in uncontrolled cell proliferation. The book also discusses therapeutic strategies targeting these checkpoints in cancer treatment.

4. *Molecular Biology of Cancer: The Cell Cycle Connection*
A detailed exploration of how molecular changes in the cell cycle machinery drive cancer development. The book integrates molecular biology techniques with cancer research, highlighting how mutations in cell cycle regulators contribute to malignancy. It is suitable for advanced students and professionals seeking in-depth knowledge.

5. *Regulation of the Cell Cycle in Cancer Cells*
This text focuses on the regulatory mechanisms that govern cell cycle progression specifically in cancer cells. It addresses how cancer cells bypass normal regulatory controls and the impact of this on tumor growth. The book also reviews current and emerging therapies aimed at correcting these regulatory defects.

6. *Cell Cycle Dysregulation and Cancer Therapy*
This book examines how understanding cell cycle dysregulation has led to novel cancer therapies. It provides an overview of drugs targeting cyclin-dependent kinases and other cell cycle components. Case studies and clinical trial results are included to demonstrate the practical applications of this knowledge.

7. *The Role of p53 in Cell Cycle and Cancer*
Dedicated to the tumor suppressor protein p53, this book discusses its critical function in cell cycle arrest and apoptosis. It explains how mutations in p53 contribute to cancer progression and resistance to therapy. The text is valuable for readers interested in molecular oncology and targeted treatments.

8. *Cell Cycle and Cancer: An Answer Key for Students*
Designed as a study guide, this book provides clear explanations and answers to common questions about the cell cycle and its relationship to cancer. It includes diagrams, review questions, and detailed answer keys to facilitate learning. Perfect for students preparing for exams or needing a concise reference.

9. *Targeting the Cell Cycle in Cancer Treatment*
This book explores therapeutic approaches aimed at manipulating the cell cycle to combat cancer. It discusses inhibitors of cyclins, CDKs, and other regulatory proteins, highlighting their clinical development and use. The text also addresses challenges and future directions in cell cycle-targeted therapies.

The Cell Cycle And Cancer Answer Key

The Cell Cycle and Cancer: Answer Key

Unravel the mysteries of cell division and its connection to cancer—and finally understand how this deadly disease develops.

Are you struggling to grasp the complex intricacies of the cell cycle and its devastating role in cancer development? Do textbooks leave you feeling more confused than enlightened? Are you overwhelmed by the sheer volume of information, leaving you unable to connect the dots between cellular processes and oncogenesis? Do you need a clear, concise, and accessible guide to understand this critical area of biology and medicine?

This ebook provides the answers you've been searching for. It cuts through the jargon and delivers a clear, easy-to-understand explanation of the cell cycle, its regulation, and the ways in which disruptions lead to cancer.

Author: Dr. Evelyn Reed (Fictional Author)

Contents:

The Cell Cycle and Cancer: Answer Key – A Detailed Exploration

Introduction: The Cell Cycle – A Fundamental Process

The cell cycle is a fundamental biological process that governs the growth and reproduction of cells. It’s a tightly regulated series of events leading to cell division, resulting in two daughter cells inheriting identical genetic material. Understanding the cell cycle is crucial, particularly in the context of cancer, as uncontrolled cell division is a hallmark of this devastating disease. Disruptions in this intricate process can lead to uncontrolled cell proliferation, a defining feature of cancer. This introduction sets the stage for a comprehensive exploration of the cell cycle's phases, regulation, and its crucial link to cancer development.

Chapter 1: Phases of the Cell Cycle

The cell cycle is conventionally divided into four main phases: G1 (Gap 1), S (Synthesis), G2 (Gap 2), and M (Mitosis). Each phase plays a distinct role in preparing the cell for division.

G1 Phase: This is the initial growth phase where the cell increases in size, synthesizes proteins and organelles, and prepares for DNA replication. It's a critical point for cell cycle control, as the cell assesses its environment and decides whether to proceed to S phase. Specific checkpoints in G1 ensure sufficient resources and optimal conditions before DNA replication commences.

S Phase: This is the DNA synthesis phase where the cell replicates its entire genome. Precise duplication is essential to ensure each daughter cell receives a complete set of chromosomes. Errors during this phase can lead to mutations, some of which can contribute to cancer development.

G2 Phase: The second gap phase allows the cell to grow further, synthesize proteins necessary for mitosis, and prepare for chromosome segregation. Another critical checkpoint in G2 ensures the integrity of the replicated DNA before proceeding to mitosis. DNA damage detection and repair mechanisms are highly active during this phase.

M Phase (Mitosis): This is the actual cell division phase, consisting of several sub-stages: prophase, prometaphase, metaphase, anaphase, telophase, and cytokinesis. Mitosis ensures accurate segregation of chromosomes into two daughter nuclei, followed by the division of the cytoplasm (cytokinesis), resulting in two genetically identical daughter cells. Errors in chromosome segregation during mitosis can lead to aneuploidy (abnormal chromosome number), a common characteristic of cancer cells.

Chapter 2: Cell Cycle Regulation

The cell cycle is not a passive process; it's meticulously regulated by a complex network of proteins, primarily cyclins and cyclin-dependent kinases (CDKs). These proteins work together to drive the cell cycle forward and ensure its proper progression through various phases.

Cyclins: These proteins are regulatory subunits that fluctuate in concentration throughout the cell cycle. Different cyclins associate with specific CDKs at different phases, activating them and driving

the cell cycle forward. For instance, cyclin D is crucial for G1 progression, while cyclin B is essential for mitosis.

Cyclin-Dependent Kinases (CDKs): These are enzymes that phosphorylate target proteins, triggering crucial events in each cell cycle phase. CDKs require cyclin binding for activation. The precise timing and activity of cyclin-CDK complexes are crucial for regulating the cell cycle's progression.

Checkpoints: These are surveillance mechanisms that monitor the integrity of the genome and the cell's internal state. They act as brakes, halting cell cycle progression if errors or damage are detected. The major checkpoints are located at the G1/S transition, the G2/M transition, and during mitosis itself.

Signaling Pathways: A variety of signaling pathways, such as the p53 pathway and the retinoblastoma (Rb) pathway, play crucial roles in regulating the cell cycle. These pathways integrate signals from the environment and the cell's internal state to influence cell cycle progression, ensuring appropriate cell growth and division.

Chapter 3: Cell Cycle Checkpoints

Cell cycle checkpoints are crucial for maintaining genome integrity and preventing the propagation of damaged cells. These checkpoints function as surveillance mechanisms that monitor the cell's internal state and halt cell cycle progression if errors or damage are detected. Failure of these checkpoints can lead to the accumulation of mutations and contribute to cancer development.

G1 Checkpoint: This checkpoint assesses the cell's environment and its internal state to ensure optimal conditions for DNA replication. It checks for DNA damage, nutrient availability, and growth factors. The p53 tumor suppressor protein plays a critical role in this checkpoint, inducing cell cycle arrest or apoptosis (programmed cell death) if DNA damage is detected.

G2 Checkpoint: This checkpoint ensures that DNA replication is complete and accurate before the cell proceeds to mitosis. It verifies that the genome is undamaged and that all DNA has been replicated without errors. Similar to the G1 checkpoint, p53 plays a crucial role in this checkpoint.

M Checkpoint (Spindle Checkpoint): This checkpoint ensures that all chromosomes are correctly attached to the mitotic spindle before anaphase begins. It prevents premature chromosome segregation and ensures accurate distribution of genetic material to daughter cells. Errors in this checkpoint can lead to aneuploidy, a common feature of cancer cells.

Chapter 4: Cancer and the Cell Cycle

Cancer is characterized by uncontrolled cell growth and division. This uncontrolled proliferation

arises from disruptions in the normal cell cycle regulation. Mutations in genes that control the cell cycle, such as cyclins, CDKs, and tumor suppressor genes (e.g., p53, Rb), can lead to uncontrolled cell division and tumor formation.

Oncogenes: These are mutated genes that promote cell growth and division. They often arise from mutations in proto-oncogenes, which normally regulate cell cycle progression. Activated oncogenes can drive the cell cycle forward even in the presence of damage or unfavorable conditions.

Tumor Suppressor Genes: These genes normally inhibit cell growth and division, preventing uncontrolled proliferation. Mutations or inactivation of tumor suppressor genes can lead to loss of cell cycle control and contribute to cancer development. p53 and Rb are prime examples of tumor suppressor genes crucial for cell cycle regulation.

Genetic Instability: Cancer cells often exhibit genomic instability, characterized by an increased rate of mutations and chromosomal abnormalities. This instability arises from defects in DNA repair mechanisms and cell cycle checkpoints, further contributing to uncontrolled cell proliferation and tumor development.

Chapter 5: Cancer Treatments Targeting the Cell Cycle

Many cancer treatments aim to disrupt the cell cycle and prevent uncontrolled cell growth. These therapies exploit the differences between normal and cancer cells in their cell cycle regulation.

Chemotherapy: This involves the use of drugs that interfere with various aspects of the cell cycle, preventing DNA replication, mitosis, or other essential processes. Many chemotherapeutic agents target specific phases of the cell cycle or enzymes involved in cell cycle regulation.

Targeted Therapy: These therapies specifically target molecules that are crucial for cancer cell growth and division. They can inhibit specific kinases, cyclins, or other proteins involved in cell cycle regulation, offering a more precise approach compared to traditional chemotherapy.

Radiation Therapy: This uses high-energy radiation to damage the DNA of cancer cells, leading to cell cycle arrest or apoptosis. Radiation therapy can disrupt the cell cycle at various points, depending on the dose and type of radiation used.

Conclusion: Synthesizing Key Concepts

Understanding the cell cycle and its intricate regulation is critical for comprehending cancer development and treatment. Disruptions in this fundamental process, stemming from genetic mutations, signaling pathway dysregulation, and checkpoint failures, lead to uncontrolled cell proliferation and tumor formation. Modern cancer therapies increasingly target specific aspects of

the cell cycle, offering novel approaches to combat this devastating disease. Continued research into the precise mechanisms of cell cycle control and its dysregulation in cancer is crucial for developing more effective and targeted therapies.

FAQs:

1. What is the role of p53 in the cell cycle? p53 is a tumor suppressor protein that plays a vital role in cell cycle checkpoints, inducing cell cycle arrest or apoptosis in response to DNA damage.

2. How do cyclins and CDKs regulate the cell cycle? Cyclins bind to and activate CDKs, which then phosphorylate target proteins, driving the cell cycle forward.

3. What are oncogenes? Oncogenes are mutated genes that promote cell growth and division, often arising from mutations in proto-oncogenes.

4. What are tumor suppressor genes? Tumor suppressor genes normally inhibit cell growth and division; their inactivation contributes to cancer development.

5. What are the main cell cycle checkpoints? The main checkpoints are at the G1/S, G2/M transitions, and during mitosis.

6. How does chemotherapy target the cell cycle? Chemotherapy drugs interfere with various aspects of the cell cycle, preventing DNA replication, mitosis, or other essential processes.

7. What is aneuploidy? Aneuploidy is an abnormal number of chromosomes, a common feature of cancer cells, often resulting from errors in chromosome segregation during mitosis.

8. What is the significance of genomic instability in cancer? Genomic instability, characterized by high mutation rates and chromosomal abnormalities, drives tumor development and progression.

9. How does radiation therapy affect the cell cycle? Radiation therapy damages DNA, leading to cell cycle arrest or apoptosis.

Related Articles:

1. The Role of p53 in Cancer Development: This article explores the crucial role of the p53 tumor suppressor gene in preventing cancer.

2. Cyclins and CDKs: Key Regulators of Cell Cycle Progression: A detailed examination of the cyclin-CDK complexes and their regulatory mechanisms.

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8. Genomic Instability and Cancer Progression: This article discusses the role of genomic instability in driving cancer development and resistance to therapy.

9. The Cell Cycle and Aging: An exploration of the relationship between cell cycle regulation and the aging process.

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exploding field. Outstanding contributors reviewed, not only knowledge on the most important classes of cell cycle regulators, but also summarized the various processes in which cell cycle control plays a pivotal role. The central role of the cell cycle makes this book an absolute must for plant molecular biologists.

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assemblies. This book is thus designed for laboratory use by graduate students, technicians, and researchers in many molecular and cellular disciplines. - Describes modern tools and techniques used to study nuclear pore complexes and nucleocytoplasmic transport in diverse eukaryotic model systems (mammalian cells, Xenopus, C. elegans, yeast) - Chapters are written by experts in the field - Cutting-edge material

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book's second half demonstrates recent applications of cytogenomic techniques, such as characterizing 3D chromosome structure across different tissue types and insights into multilayer organization of chromosomes, role of repetitive elements and noncoding RNAs in human genome, studies in topologically associated domains, interchromosomal interactions, and chromoanagenesis. This book is an important reference source for researchers, students, basic and translational scientists, and clinicians in the areas of human genetics, genomics, reproductive medicine, gynecology, obstetrics, internal medicine, oncology, bioinformatics, medical genetics, and prenatal testing, as well as genetic counselors, clinical laboratory geneticists, bioethicists, and fertility specialists. - Offers applied approaches empowering a new generation of cytogenomic research using a balanced combination of classical and advanced technologies - Provides a framework for interpreting chromosome structure and how this affects the functioning of the genome in health and disease - Features chapter contributions from international leaders in the field

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that have roles in force production, motion and shape change that occur in other phases of the biology of the cell. The localization of the force-producing mechanism to a restricted linear part of the subsurface is caused by the mitotic apparatus, the same cytoskeletal structure that insures orderly mitosis.

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