

<<美国医师执照考试>>

图书基本信息

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### 内容概要

《美国医师执照考试:High-Yield细胞与分子生物学(第3版)》内容高度概括,重点突出有利于读者快速掌握学科的核心知识。

编排新颖、既有基础知识要点的介绍,又有以疾病为核心的综合归纳,并体现了相关学科的横向联系。

语言规范、地道,既有利于读者快速掌握专业词汇,又有利于医学英语思维的培养。

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书籍目录

Preface Abbreviations 1 Chromosomal DNA .The Biochemistry of Nucleic Acids .Levels of DNA Packaging .Centromere .Heterochromatin .Euchromatin .Studying Human Chromosomes .Staining of Morphology .DNA Melting Curve 2 Chromosome Replication .General Features .The Chromosome Replication Process .DNA Topoisomerases .The Telomere .DNA Damage .DNA Repair .Clinical Considerations .Summary of Chromosome Replication Machinery 3 Meiosis and Genetic Recombination .Meiosis .Genetic Recombination 4 The Human Nuclear Genome .General Features .Protein-Coding Genes .RNA-Coding Genes .Epigenetic Control .Noncoding DNA 5 The Human Mitochondrial Genome .General Features .The 13 Protein-Coding Genes .The 24 RNA-Coding Genes .Other Mitochondrial Proteins .Mitochondrial Diseases 6 Protein Synthesis .General Features .Transcription .Processing the RNA Transcript into mRNA .Translation .Clinical Considerations 7 Control of Gene Expression .General Features .Mechanism of Gene Expression .The Structure of DNA-Binding Proteins .Other Mechanisms of Gene Expression .The Lac Operon .The trp Operon 8 Mutations of the DNA Sequence .General Features .Silent (Synonymous) Mutations .Non-Silent (Nonsynonymous) Mutations .Loss of Function and Gain of Function Mutations V. Other Types of Polymorphisms 9 Proto-Oncogenes, Oncogenes, and Tumor-Suppressor Genes .Proto-Oncogenes and Oncogenes .Tumor-Suppressor Genes .Hereditary Cancer Syndromes 10 The Cell Cycle I. Mitosis .Control of the Cell Cycle 11 Molecular Biology of Cancer .The Development of Cancer (Oncogenesis) .The Progression of Cancer .Signal Transduction Pathways ... .. 12 Cell Biology of the Immune System 13 Molecular Biology of the Immune System 14 Molecular Biology Techniques 15 Identification of Human Disease Genes 16 Gene Therapy Appendix 1: The Genetic Code Appendix 2: Amino Acids Appendix 3: Chromosomal Locations of Human Genetic Diseases Figure Credits Index

## 章节摘录

版权页：插图：C. FRAMESHIFT MUTATIONS (Figure 8-6). Frameshift mutations are point mutations where either a deletion or insertion of nucleotides (not a multiple of three) alters the codon so that a premature STOP codon is formed or the reading frame is shifted. Frameshift mutations produce either unstable mRNAs which are rapidly degraded or nonfunctional ("garbled") proteins because all of the amino acids after the deletion or insertion are changed, respectively. In-frame mutations are point mutations where either a deletion or insertion of nucleotides (a multiple of three) alters the codon but does not shift the reading frame. In-frame mutations produce compensated proteins. Clinical examples of frameshift and in-frame mutations are Duchenne muscular dystrophy (DMD) and Becker muscular dystrophy (BMD).

1. Duchenne muscular dystrophy a. DMD is an X-linked recessive genetic disorder caused by various mutations in the DMD gene on chromosome Xp21.2 for dystrophin which anchors the cytoskeleton (actin) of skeletal muscle cells to the extracellular matrix via a transmembrane protein (α-dystrophin and β-dystrophin), thereby stabilizing the cell membrane. The DMD gene is the largest known human gene. b. DMD is caused by small deletion, large deletion, deletion of the entire gene, insertion, duplication of one or more exons, or single-based change mutations. The deletion or insertion of nucleotides (not a multiple of three) results in a frameshift mutation. These mutations result in either the absence of dystrophin protein or a nonfunctional ("garbled") dystrophin protein which causes severe clinical features (more severe than BMD). c. Serum creatine phosphokinase (CK) measurement. The measurement of serum CK is one of the diagnostic tests for DMD ([serum CK] = >10 times normal is diagnostic). d. Skeletal muscle biopsy. A skeletal muscle biopsy shows histological signs of fiber size variation, loci of necrosis and regeneration, hyalinization, and deposition of fat and connective tissue. Immunohistochemistry shows almost complete absence of the dystrophin protein. e. Clinical features include symptoms appear in early childhood with delays in sitting and standing independently; progressive muscle weakness (proximal weakness > distal weakness) often with calf hypertrophy; progressive muscle wasting; waddling gait; difficulty in climbing; wheelchair bound by 12 years of age; cardiomyopathy by 18 years of age; death by 30 years of age due to cardiac or respiratory failure.

2. Becker muscular dystrophy a. BMD is an X-linked recessive genetic disorder caused by various mutations in the DMD gene on chromosome Xp21.2 for dystrophin which anchors the cytoskeleton (actin) of skeletal muscle cells to the extracellular matrix via a transmembrane protein (α-dystrophin and β-dystrophin) thereby stabilizing the cell membrane. b. BMD is caused by the deletion or insertion of nucleotides (a multiple of three) which results in an in-frame mutation. The in-frame mutation results in a compensated dystrophin protein which causes less severe clinical features compared with DMD.

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### 编辑推荐

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