<<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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章节摘录

 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. Ouchenne 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 of 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

《美国医师执照考试:High-Yield细胞与分子生物学(第3版)》是参加美国医师执照考试的必备辅导用书，也可作为我国医学院校从事双语教学的教材和参考用书，对教师进行英语授课，学生学习、参加考试具有重要的参考价值。

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