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PHARMACOLOGY

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   6.2. Interleukin-1 Receptor Antagonist (Anakinra)
   6.3. Interleukin-6 receptor antagonist (tocilizumab)
7. Antagonist of Immunoglobulin E (Omalizumab)
8. Interleukin therapy in oncology
   8.1. Interleukin-2
   8.2. Interleukin-2/diphtheria toxin conjugate (Ontak)
   8.3. Interferon-γ and Interleukin-12
9. Perspectives and future developments

**Pain Pharmacology and Analgesia**

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1. Pain definitions
   1.1. Pain – According to Duration
      1.1.1. Acute Pain
      1.1.2. Chronic Pain
   1.2. Pain – According to Type
      1.2.1. Nociceptive pain
      1.2.2. Inflammatory pain
      1.2.3. Neuropathic pain
   1.3. Emotional Response to Pain
2. Pain signaling system
   2.1. Pain detection
   2.2. Functional Characteristics of the Pain Signaling Apparatus
      2.2.1. Nociceptors
      2.2.2. Primary Sensory Neurones
2.2.3. Primary Sensory Neurons and the Spinal Cord
2.2.4. Spinal Cord Neurons
   2.2.4.1. Projection Neurons and the Spinal Cord
   2.2.4.2. Spinal Interneurons
2.3. Pain characteristics
2.4. Animal Models of Nociception/Pain
2.5. Neurochemical Characteristics of the Nociceptive Signaling System
   2.5.1. Transmission of Nociceptive Information from the Periphery to the Spinal Cord
2.6. Nociceptive Neurotransmitters and their Target Receptors
   2.6.1. Excitatory Amino Acids and their Receptors
      2.6.1.1. Glutamate Receptors
      2.6.1.2. NMDA Receptors
      2.6.1.3. AMPA Receptors
      2.6.1.4. Kainate Receptors
      2.6.1.5. Metabotropic Glutamate Receptors (mGlRs)
      2.6.1.6. Excitatory Amino Acid Transporters (EETs)
      2.6.1.7. Glycine Transporters (GlyTs)
   2.6.2. Co-containment of Neurotransmitters in Nerve Terminals
2.7. Descending Modulation of Nociception
3. Neural plasticity and pain
   3.1. Inflammation
      3.1.1. Inflammation and Peripheral Sensitization
      3.1.2. Inflammation and Post-translational Changes
      3.1.3. Inflammation-Induced Transcriptional Changes: Effects on Peripheral Sensitization
      3.1.4. Inflammation and Central Sensitization
   3.2. Peripheral Nerve Injury and Neuropathic Pain
   3.3. Central Sensitization and Neuropathic Pain
      3.3.1. Dysfunction of Central Inhibition
   3.4. Non-Neuronal Cells in the Dorsal Root Ganglia and the CNS
      3.4.1. Satellite Glia Cells in Dorsal Root Ganglia
      3.4.2. Activated Microglia and Astrocytes in the CNS
4. Endogenous pain relief system
   4.1. Processing of low-intensity stimuli
   4.2. Processing of high-intensity stimuli
   4.3. Opioids
      4.3.1. Heterodimeric Opioid Receptors
   4.4. Tolerance to Opioids
      4.4.1. Innate Tolerance
      4.4.2. Acquired Tolerance
         4.4.2.1. Pharmacodynamic Tolerance
         4.4.2.2. Pharmacokinetic Tolerance
      4.4.3. Tolerance to Opioid-Related Side-Effects
      4.4.4. Prevention of the Development of Analgesic Tolerance
5. Strategies for producing pain therapeutics
   5.1. Modulation of the NMDA Receptor-Nitric Oxide Synthase Cascade
      5.1.1. NMDA receptor antagonists
      5.1.2. Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)
      5.1.3. NOS inhibitors
      5.1.4. Substance P antagonists
   5.2. Inhibition of Pro-nociceptive Neurotransmitter Release
      5.2.1. N-type Calcium Channel Blockers
      5.2.2. Gabapentinoids
   5.2. Voltage-Gated Sodium Channels as Potential Drug Targets in Persistent Pain States
      5.2.1. Nav1.8
      5.2.2. Nav1.9
      5.2.3. Sodium Channels in Inflammatory Pain
      5.2.4. Sodium Channels in Neuropathic Pain
   5.3. Transient Receptor Potential Vanilloid Receptor-1 (TRPV1) as a Potential Drug Target
5.4. Purinergic Receptors
5.5. Nerve Growth Factor (NGF) and trkA Receptors
5.6. Brain-Derived Neurotropic Factor (BDNF) and trkB Receptors
5.7. Nicotinic Cholinergic Agonists
5.8. Neuronal Nicotinic Cholinergic Antagonists
5.9. Anti-opioid Peptides
   5.9.1. CCK-8
   5.9.2. Dynorphin A
5.10. Adenosine
5.11. Cannabinoids
6. Pain assessment and pain assessment tools
7. Pharmacological treatment of pain: analgesics and adjuvants
   7.1. Treatment of Pain in the Clinical Setting
   7.2. Major Aims of the Treatment of Clinical Pain
   7.3. Pharmacological management of pain: current treatment guidelines
   7.4. Controlled or Sustained Release Oral Formulations
8. Analgesic Agents
   8.1. Non-opioid Analgesics
      8.1.1. Paracetamol
      8.1.2. Non-steroidal anti-inflammatory drugs, (NSAIDs)
   8.2. Opioid Analgesics
   8.3. Commonly Prescribed Opioid Analgesics
      8.3.1. Strong Opioid Analgesics
         8.3.1.1. Morphine
         8.3.1.2. Oxycodone
         8.3.1.3. Methadone
         8.3.1.4. Hydromorphone
         8.3.1.5. Fentanyl
      8.3.2. Weak Opioid Analgesics
         8.3.2.1. Codeine
         8.3.2.2. Pethidine (Meperidine)
         8.3.2.3. Tramadol
      8.3.3. Partial Opioid Agonists/Antagonists: Buprenorphine
   8.4. Opioid-Related Adverse effects
   8.5. Adjunct analgesics
   8.5.1. Tricyclic Antidepressants
   8.5.2. Anticonvulsants
   8.5.3. Anti-arrhythmics
   8.6. Topical Agents
      8.6.1. Lignocaine (lidocaine)
      8.6.2. Capsaicin
   8.7. NMDA receptor antagonists
      8.7.1. Ketamine
      8.7.2. Dextromethorphan
   8.8. Alpha(2)-Adrenergic Receptor Agonists
   8.9. Invasive Procedures
      8.9.1. Neurolytic Celiac Plexus Blockade
      8.9.2. Implantable Intrathecal Drug Delivery
      8.9.3. Spinal Cord Stimulation
9. Conclusion

Anesthetics
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PHARMACOLOGY

1. Inhalation Agents
   1.1. Introduction
   1.2. Pharmacokinetics and Pharmacodynamics
   1.3. Specific Agents
      1.3.1. Diethyl Ether (Ether)
      1.3.2. Chloroform
      1.3.3. Cyclopropane
      1.3.4. Trichloroethylene
      1.3.5. Halogenated Alkanes and Ethers
      1.3.6. Nitrous Oxide.
      1.3.7. Xenon

2. Neuromuscular Blocking Agents (Muscle Relaxants)
   2.1. Introduction
   2.2. Non-Depolarising Muscle Relaxants
      2.2.1. Tubocurarine (1935)
      2.2.2. Metocurine (dimethyl tubocurarine chloride/bromide)
      2.2.3. Alcuronium (1961)
      2.2.4. Gallamine (1948)
      2.2.5. Pancuronium (1968)
      2.2.6. Vecuronium (1983)
      2.2.7. Atracurium (1980s)
      2.2.8. Cis-atracurium (1995)
      2.2.9. Mivacurium (1993)
      2.2.10. Rocuronium (1994)
      2.2.11. Sugammadex (2003)
      2.2.12. Rapacuronium
   2.3. Reversal Drugs (Anticholinesterase)
   2.4. Depolarising Muscle Relaxants (Suxamethonium or Succinylcholine)

3. Local Anesthetics
   3.1. Introduction
   3.2. Pharmacokinetics and Pharmacodynamics
   3.3. Toxicity
   3.4. Specific Agents
      3.4.1. Cocaine
      3.4.2. Procaine
      3.4.3. Chloroprocaine
      3.4.4. Tetracaine (Amethocaine)
      3.4.5. Lidocaine
      3.4.6. Prilocaine
      3.4.7. Mepivacaine
      3.4.8. Bupivacaine
      3.4.9. Ropivacaine
      3.4.10. Eutectic Mixture of Local Anesthetics (EMLA)

4. Intravenous Induction Agents
   4.1. Introduction
   4.2. Actions and Mechanisms of General Anesthetics
   4.3. Specific Agents
      4.3.1. Barbiturates
      4.3.2. Propofol
      4.3.3. Ketamine
      4.3.4. Etomidate
      4.3.5. Benzodiazepines

Drug Discovery
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1. Introduction
2. The Modern Drug Discovery Process
   2.1. Target Selection
   2.2. Compound/Biological Identification
   2.3. Compound/Biological Optimization for Efficacy
   2.4. Pharmacokinetic Profiling
   2.5. Safety Assessment
3. Clinical Development
   3.1. Phase I Clinical Studies - Investigational New Drug (IND) Application
   3.2. Phase II Clinical Studies
   3.3. Phase III Clinical Studies - New Drug Application (NDA)
   3.4. Phase IV and Life Cycle Management
4. Translational Medicine
   4.1. The Role of Translational Medicine in Drug Discovery
       4.1.1. Disease Biomarkers
       4.1.2. Compound-Target Interaction Biomarkers
       4.1.3. Pharmacodynamic Biomarker
       4.1.4. Surrogate Endpoints (Biomarkers)
5. The Role of Experimental Animal Models in Drug Discovery and Development
6. The Use of Imaging Technology in Drug Discovery and Development
7. Personalized Medicine and Drug Discovery and Development
8. Conclusion

Gene Therapy
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1. Introduction
2. Relation of Gene Therapy to Other Biotechnologies
3. Gene Therapy Technologies
   3.1. Classification of Gene Therapy Techniques
   3.2. Physical Methods of Gene Transfer
       3.2.1. Electroporation
       3.2.2. Particle Bombardment
       3.2.3. Ultrasound-mediated Transfection
       3.2.4. Molecular Vibration
       3.2.5. Gene Transfection using Laser Irradiation
       3.2.6. Photochemical Transfection
       3.2.7. Chemical Methods of Gene Transfer
   3.3. Ex vivo and In vivo Gene Therapy
       3.3.1. Ex vivo Gene Therapy
       3.3.2. In vivo Gene Therapy
   3.4. Gene Repair and Replacement
       3.4.1. Gene Repair by Single-stranded Oligonucleotides
   3.5. Spliceosome-mediated RNA Trans-splicing
   3.6. Vectors for Gene Therapy
       3.6.1. Use of Genes as Pharmaceuticals
       3.6.2. The Ideal Vector for Gene Therapy
       3.6.3. Viral Vectors
       3.6.4. Non-viral Vectors for Gene Therapy
   3.7. Concluding Remarks about Vectors
   3.8. Cell-mediated Gene Therapy
       3.8.1. Stem Cell Gene Therapy
   3.9. Routes of Administration for Gene Therapy
   3.10. Targeted Gene Therapy
       3.10.1. Controlled Induction of Gene Expression
       3.10.2. Controlled Gene Therapy
3.10.3. Technologies for Gene Suppression
3.10.4. Locked Nucleic Acid
3.11. Clinical Applications
  3.11.1. Strategies for Cancer Gene Therapy
  3.11.2. Gene Therapy of Neurological Disorders
  3.11.3. Gene Therapy of Cardiovascular Disorders
4. Concluding Remarks