

1. Record Nr.	UNINA9910842283503321
Autore	Mishra Awanish
Titolo	Drug Delivery Strategies in Neurological Disorders
Pubbl/distr/stampa	Singapore : , : Springer, , 2024 ©2023
ISBN	981-9968-07-0
Edizione	[1st ed.]
Descrizione fisica	1 online resource (457 pages)
Altri autori (Persone)	KulhariHitesh
Disciplina	616.80461
Lingua di pubblicazione	Inglese
Formato	Materiale a stampa
Livello bibliografico	Monografia
Nota di contenuto	<p>Intro -- Foreword -- Foreword 1 -- Preface -- Contents -- Editors and Contributors -- Part I: Anatomy and Physiology of Human Nervous System -- 1: Nanocarriers as an Emerging Tool for Drug Delivery to Combat Neurodegenerative Diseases -- 1.1 Introduction -- 1.2 Barriers in Delivery of Therapeutic Agent to CNS -- 1.2.1 BBB -- 1.2.2 BCSFB -- 1.3 Problems Associated with Conventional Drug Delivery -- 1.4 Nano Strategies to Enhance Drug Delivery Across the BBB -- 1.4.1 Liposomes -- 1.4.2 Solid Lipid Nanoparticles (SLNs) -- 1.4.3 Polymeric Nanoparticles -- 1.4.4 Polymeric Micelle -- 1.4.5 Dendrimers -- 1.4.6 Inorganic Nanoparticles -- 1.4.7 Quantum Dots -- 1.4.8 Nanocrystals -- 1.4.9 Nanotubes -- 1.4.10 Nanoemulsion -- 1.4.11 Natural Polymer-Based Nanoparticles -- 1.5 Application of Nanotechnology in Neurological Conditions -- 1.5.1 AD -- 1.5.2 PD -- 1.5.3 Huntington's Disease -- 1.5.4 Stroke -- 1.5.5 Brain Tumors -- 1.6 Conclusion and Future Perspectives -- References -- 2: Challenges in Drug Development for Neurological Disorders -- 2.1 Introduction -- 2.2 Barriers to CNS Drug Development -- 2.2.1 Barriers Related to the Biological Aspect -- 2.2.1.1 Complexity of Blood-Brain and Blood-Spinal Cord Barrier -- 2.2.1.2 Incomplete Understanding of Disease Biology -- 2.2.1.3 Inferior Reproducibility and Predictive Value of Preclinical Animal Models -- 2.2.1.4 Lack of Pharmacodynamic Biomarkers and Reliable Target Engagement -- 2.2.2 Barriers Related to the Drug Development Aspect -- 2.2.2.1 Difficulty in Target Identification and Validation -- 2.2.2.2 Imprecise Clinical Outcome</p>

Measures -- 2.2.2.3 Shortage of Trial-Ready Patients -- 2.2.2.4 Variability in the Clinical Population -- 2.2.2.5 Faulty Regulatory Approval Process -- 2.3 Conclusion -- References -- 3: Transporter Systems and Metabolism at the Blood-Brain Barrier and Blood-CSF Barrier.

3.1 Introduction -- 3.1.1 Barrier to CNS Drug Delivery -- 3.1.1.1 The Blood-Brain Barrier (BBB) -- 3.1.1.2 The Blood-Cerebrospinal Fluid (B-CSF) Barrier -- 3.2 Transport Mechanisms of Drug-Loaded Nanocarriers Across the Barriers -- 3.2.1 Receptor-Mediated Transcytosis (RMT) -- 3.2.2 Carrier-Mediated Transcytosis (CMT) -- 3.2.3 Adsorptive-Mediated Transcytosis (AMT) -- 3.3 Nanocarriers for Drug Delivery Across Barriers: Special Emphasis on Neurodegenerative Disease -- 3.3.1 Liposomes -- 3.3.1.1 Transferrin-Modified Liposomes -- 3.3.1.2 Glutathione-Modified Liposomes -- 3.3.1.3 PEG-Modified Liposomes -- 3.3.1.4 Multifunctional Liposomes -- 3.3.2 Nanoparticles (NPs) -- 3.3.2.1 Gold Nanoparticles (Gold NPs) -- 3.3.2.2 Iron Oxide Nanoparticles (IONPs) -- 3.3.2.3 Cerium Oxide Nanoparticles (CEONPs) -- 3.3.2.4 Molybdenum Nanoparticles -- 3.3.2.5 Silica Nanoparticles (SiNPs) -- 3.3.2.6 Organic Nanoparticles -- 3.3.2.7 Nanoemulsion (NE) and Nanosuspension (NS) -- 3.3.2.8 Dendrimers -- 3.3.2.9 Nanogels (NGs) -- 3.3.3 Nanomicelles -- 3.3.4 Exosomes -- 3.3.5 Carbon Dots (CDs) -- 3.4 Nanocarriers in Clinical Trials -- 3.5 Potential Risk of Nanocarriers -- 3.6 Future Perspectives and Conclusions -- References -- Part II: Pathophysiology and Management of Neurological Disorders -- 4: Pathophysiology and Management Approaches in Alzheimer's Disease -- 4.1 Introduction -- 4.2 Pathophysiology of Alzheimer's Disease -- 4.2.1 Abeta in Alzheimer's Disease -- 4.2.2 Cholinergic Dysregulation -- 4.2.3 Metal Ion Toxicity -- 4.2.4 Neuroinflammation -- 4.2.5 Tau Hyperphosphorylation -- 4.2.6 Oxidative Stress -- 4.2.7 Mitochondrial Dysfunction -- 4.2.8 Miscellaneous -- 4.3 Management Approaches in Alzheimer's Disease -- 4.3.1 Targeting Amyloid-Beta (Abeta) Protein -- 4.3.1.1 Targeting Secretase -- 4.3.2 Targeting Tau-Hyperphosphorylation.

4.3.3 Targeting Intracellular Signaling Cascades -- 4.3.4 Targeting the Neurotransmitters -- 4.3.4.1 Modulation of GABAergic Neurons -- 4.3.5 Targeting Mitochondrial Dysfunction -- 4.3.6 Targeting Oxidative Stress -- 4.3.7 Targeting Neuroinflammation -- 4.4 Conclusion -- References -- 5: Pathophysiology and Management Approaches for Parkinson's Disease -- 5.1 Introduction -- 5.2 Epidemiology -- 5.3 Transition of the Brain: Biology to Pathology -- 5.3.1 Neuroanatomical Changes in PD -- 5.3.2 Neuronal Circuitry Changes in PD -- 5.4 Neuropathology -- 5.4.1 LB Formation and Neuronal Loss -- 5.4.2 Genetics at the Interplay -- 5.4.3 Microtubule Malfunctioning -- 5.4.4 Mitochondrial Dysfunction and Oxidative Stress -- 5.4.5 ER Stress/UPR -- 5.4.6 Neuroinflammation -- 5.4.7 Autophagy Impairment -- 5.5 Symptomatic Targeting: A Conventional Approach -- 5.5.1 Symptomatic Dopaminergic Agents -- 5.5.1.1 Levodopa -- 5.5.1.2 Dopamine Agonists (DAAs) -- 5.5.1.3 Monoamine Oxidase-B Inhibitors (MAO-BIs) -- 5.5.1.4 Catechol-O-Methyltransferase Inhibitors (COMTIs) -- 5.5.2 Symptomatic Nondopaminergic Agents -- 5.5.2.1 Acetylcholine (ACh)-Based Therapeutics -- 5.5.2.2 5-HT-Based Therapeutics -- 5.5.2.3 Glutamate and GABA-Based Therapeutics -- 5.5.2.4 NA (Noradrenaline)-Based Therapeutics -- 5.5.2.5 Adenosine-Based Therapeutics -- 5.6 Pathological Targeting: Disease-Modifying Approach -- 5.6.1 Agents Targeting Specific PD Pathological Hallmark -- 5.6.1.1 Proteinopathy in PD -- 5.6.1.2 Targeting LRRK-2 -- 5.6.1.3 Targeting Glucosylceramide Beta 1 (GBA) -- 5.6.1.4 Targeting PINK-1/Parkin -- 5.6.2 Agents Rescuing Neurons -- 5.6.2.1 Calcium

Targeting Therapies -- 5.6.2.2 Iron Targeting Therapies -- 5.6.2.3 Neuroinflammation Targeting Agents -- 5.6.2.4 Mitochondria Targeting Agents -- 5.6.3 Gene Therapy -- 5.6.4 miRNAs as a Novel Therapeutic Approach.

5.7 Alternative Approaches for PD Management -- 5.7.1 Cellular Therapy -- 5.7.1.1 Fetal Ventral Mesencephalic Tissue -- 5.7.1.2 Stem Cell Therapy -- 5.7.2 Nanotechnology -- 5.7.3 Invasive Brain Stimulation -- 5.7.3.1 Deep Brain Stimulation (DBS) -- 5.7.4 Brain Connectomic Studies -- 5.7.4.1 Repetitive Transcranial Magnetic Stimulation (rTMS) -- 5.7.4.2 Transcranial Direct Current Stimulation (tDCS) and Transcranial Alternating Current Stimulation (tACS) in PD -- 5.7.4.3 Transcranial Random Noise Stimulation (tRNS) -- 5.7.4.4 Transcranial Pulsed Current Stimulation (tPCS) -- 5.7.4.5 Focused Ultrasound (FUS)-Based Gene Therapy -- 5.8 Key Roadblocks and Pitfalls in PD Management -- 5.9 Conclusion -- References -- 6: Pathophysiology and Management Approaches for Epilepsy -- 6.1 Introduction -- 6.2 Antiepileptic Drugs -- 6.3 Epilepsy Surgery -- 6.4 Neuromodulatory Devices -- 6.4.1 Vagus Nerve Stimulation -- 6.4.2 Deep Brain Stimulation -- 6.4.3 Responsive Neurostimulation -- 6.5 Diets -- 6.6 Immunotherapy -- 6.7 Conclusion -- References -- 7: Pathophysiology and Management Approaches for Traumatic Brain Injury -- 7.1 Introduction -- 7.2 Pathophysiology -- 7.2.1 Primary Brain Injuries -- 7.2.2 Secondary Brain Injuries -- 7.2.2.1 Excitotoxicity -- 7.2.2.2 Mitochondrial Dysfunction -- 7.2.2.3 Neuroinflammation -- 7.2.2.4 Cell Death Process Following TBI -- 7.2.2.5 Long-Term Consequence -- 7.3 Management Approaches for Traumatic Brain Injury -- 7.3.1 Antithrombotics and Thrombolytics -- 7.3.1.1 Antiplatelet Drugs -- 7.3.1.2 Nonsteroidal Anti-Inflammatory Drugs -- 7.3.1.3 Thienopyridines -- 7.3.1.4 GpIIb/IIIa Inhibitors -- 7.3.2 Anticoagulants -- 7.3.2.1 Heparin -- 7.3.2.2 Direct Thrombin Inhibitors -- 7.3.3 Thrombolytics -- 7.3.4 Recombinant Tissue Plasminogen Activator (tPA) -- 7.3.5 Neuroprotectants -- 7.3.5.1 Glutamate Antagonists.

7.3.5.2 Magnesium Sulphate -- 7.3.6 Cannabinoids and Other Analogues -- 7.3.7 Hormone-Based Agents -- 7.3.7.1 Progesterone -- 7.3.7.2 Estrogen -- 7.3.7.3 Erythropoietin -- 7.3.7.4 Glyburide -- 7.3.7.5 Synthetic Insulin-like Growth Factors -- 7.3.8 Antioxidants -- 7.3.8.1 ROS-Free Radical Scavengers -- 7.3.8.2 Lipid Peroxidation Inhibitors -- 7.3.8.3 Endogenous Superoxide Dismutase (SOD) -- 7.3.9 Immunomodulators and Immunosuppressants -- 7.3.10 Antiepileptics and Sedatives -- 7.3.11 Statins -- 7.4 Conclusion -- References -- 8: Pathophysiology and Management Approaches for Huntington's Disease, Multiple Sclerosis, and Other Neurological Disorder -- 8.1 Introduction -- 8.2 Pathophysiology -- 8.3 Management Approaches for HD, MS, and ALS -- 8.3.1 Management Approaches for ALS -- 8.3.2 Disease-Modifying Treatment of ALS -- 8.4 Advanced Therapies -- 8.5 Conclusion -- References -- 9: Genes Encoding Ion Channels in Neurotherapeutics: Opportunities and Challenges -- 9.1 Introduction -- 9.1.1 Genes Encoding Ion Channels and Neurotherapies in Neurological Diseases -- 9.1.1.1 Epilepsy -- 9.1.1.2 Parkinson Disease -- 9.1.1.3 Schizophrenia -- 9.2 Conclusion -- References -- 10: Herbal Approaches for the Management of Neurological Disorders -- 10.1 Introduction -- 10.2 Secondary Metabolites in Neurological Disorders -- 10.2.1 Role of Flavonoids in Neurological Disorders -- 10.2.1.1 Role of Flavonoids in Epilepsy -- 10.2.1.2 Role of Flavonoids in Alzheimer's Disease -- 10.2.1.3 Role of Flavonoids in Parkinson's Disease -- 10.2.2 Role of Alkaloids in Neurological Disorders -- 10.2.2.1 Role of Alkaloids in Epilepsy -- 10.2.2.2 Role of Alkaloids in

Alzheimer's Disease -- 10.2.2.3 Role of Alkaloids in Parkinson's
Disease -- 10.2.3 Role of Glycoside in Neurological Disorder --
10.2.3.1 Role of Glycoside in Epilepsy -- 10.2.3.2 Role of Glycoside in
Alzheimer's Disease.
10.2.3.3 Role of Glycoside in Parkinson's Disease.
