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Hormones in Neurodegeneration, Neuroprotection, and Neurogenesis **The Pharmacology of Neurogenesis and Neuroenhancement** *Neuroprotection* Neuroprotection in Alzheimer's Disease **Neural Stem Cell Transplantation** Neuroprotection **Neural Progenitor Cell Transplantation and Proneurogenic Compound Administration Improve Outcomes After Trauma** *Neuroprotection in Autism, Schizophrenia and Alzheimer's disease* Neurogenesis and Neuroprotection in Huntington Disease *Experimental Stroke* **Melatonin Breathing, Feeding, and Neuroprotection** Effect of Ketamine on Neurogenesis and Neuroprotection Following Lithium-pilocarpine Induced Status Epilepticus in Rats **New Perspectives in Neurosteroids action: a Special Player Allopregnanolone Neural Stem Cells in Health and Disease** *Estrogen Effects on Traumatic Brain Injury Stem Cells and Progenitor Cells in Ischemic Stroke - Fashion or Future? Molecular Aspects of Neurodegeneration, Neuroprotection, and Regeneration in Neurological Disorders* **The Neuroprotective Effects of Z-bisdehydrodoisynolic Acid Following Traumatic Brain Injury** **Molecular Aspects of Neurodegeneration and Neuroprotection** **Brain Neurotrauma** *Melatonin, Neuroprotective Agents and Antidepressant Therapy* **Never-resting microglia: physiological roles in the healthy brain and pathological implications** *Mechanisms of Neuroinflammation and Inflammatory Neurodegeneration in Acute Brain Injury Study of the Effects of Docosahexaenoic Acid (DHA) and a Structured Phospholipid Containing DHA on Physiological and Pathological Conditions of Neurogenesis in Vitro Neuroprotective Effects of Physical Exercise on Stressed Brain Alpha-linolenic Acid Confers Neuroprotection and Improves Behavioral Deficits After Soman Exposure: Involvement of Neurogenesis Through an MTOR-mediated Pathway* **Implications of Pgrmc1 Regulation of Kit Ligand Synthesis in the Hippocampus** **Neuroimmunity** *The Neuroprotective Compound P7C3-A20 Promotes Neurogenesis and Improves Functional Outcomes After Focal Cerebral Ischemia* To Investigate Neuroprotective Mechanism in Female Brain *The Hippocampus* The Rat Brain in Stereotaxic Coordinates **Microglia in Health and Disease** **Chemokines and Chemokine Receptors in Brain Homeostasis** Neuroprotective Effects of Physical Exercise on Stressed Brain Single Administration of Fluoxetine Improves Memory Function Without Neuroprotection After Cardiac Arrest/cardiopulmonary Resuscitation in Mice **Neurodegenerative Diseases** Issues in Neuroscience Research and Application: 2011 Edition **Neurogenesis**

and Neural Plasticity

Neuroprotective Effects of Physical Exercise on Stressed Brain Mar 10 2021 This dissertation, "Neuroprotective Effects of Physical Exercise on Stressed Brain: Its Relationship to Hippocampal Neurogenesis and Dendritic Remodeling" by Suk-yu, Yau, ???, was obtained from The University of Hong Kong (Pokfulam, Hong Kong) and is being sold pursuant to Creative Commons: Attribution 3.0 Hong Kong License. The content of this dissertation has not been altered in any way. We have altered the formatting in order to facilitate the ease of printing and reading of the dissertation. All rights not granted by the above license are retained by the author. DOI: 10.5353/th_b4322376 Subjects: Cell proliferation Stress physiology - Molecular aspects Exercise - Pathophysiology Brain - Physiology Protein kinases Corticosterone Developmental neurobiology

Hormones in Neurodegeneration, Neuroprotection, and Neurogenesis May 04 2023 As life expectancy increases and population ages, the already enormous impact of neurodegeneration on society will become even larger without better prevention and treatment. Developing strategies to prevent degeneration of neurons and to promote a healthy nervous system is, thus, critical. The development of pharmacological agents that would increase production of new neurons was recently facilitated by the identification of the hormonal regulators of various steps of adult neurogenesis. The proposed book is writen by a group of top world experts involved in the study of the mechanisms of hormonal control of brain damage and repair. The effects of thyroid and steroid hormones (estrogens, androgens, progestins, gluco-mineralo-corticoids, various neurosteroids) or polypeptide hormones (CRF, urocortins, somatostastin, GH/IGF, leptin, prolactin, PACAP, erythropoetin) on neuronal survival and neurogenesis in various neurodegenerative conditions and in brain aging will be discussed in detail. The proposed book is unique because it gives a comprehensive account of the neuroprotective and neurogenic effects of steroid and polypeptide hormones. Furthermore, new pharmacological approaches for treatment of neurodegenerative conditions are presented, based on the neuroprotective and neurogenic properties of natural and synthetic hormones.

Neural Stem Cells in Health and Disease Feb 18 2022 This book is a comprehensive guide on neural stem cell behavior in health and disease. The book confers the altered behavior of endogenous neural stem cells in neurodegenerative disease conditions and the prospects of neural stem cell therapy for alleviating brain dysfunction in a variety of neurodegenerative disorders. Neural stem cell activity and neurogenesis in the adult brain is now confirmed in virtually all mammalian species including humans. Hence, a series of chapters in the first half of the book discusses the current knowledge on mechanisms of neural stem cell activity, the extent and functional significance of neurogenesis in the adult brain under normal, aged and disease environments, the susceptibility of neural stem cells and the plasticity of neurogenesis to alcohol, drugs of abuse and anesthetic agents, and advanced techniques that trigger neurogenesis in non-neurogenic regions. A second series of chapters in this book are focused on discussing the promise and efficacy of grafting of neural stem cells and/or other stem cells for treating neurological disorders such as Parkinson's disease, stroke, temporal lobe epilepsy, Alzheimer's disease and spinal cord injury. The final chapter confers on advances that are made in manufacturing a

variety of neural cell types from human pluripotent stem cells that can be used as donor cells for cell transplantation.
Neuroprotection in Alzheimer's Disease Feb 01 2023 Neuroprotection in Alzheimer’s Disease offers a translational point-of-view from both basic and clinical standpoints, putting it on the cusp for further clinical development with its emphasis on nerve cell protection, including the accumulation of knowledge from failed clinical trials and new advances in disease management. This book brings together the latest findings, both basic, and clinical, under the same cover, making it easy for the reader to obtain a complete overview of the state-of-the-field and beyond. Alzheimer's disease is the most common form of dementia, accounting for 60 to 80 percent of dementia cases. It is a progressive brain disease that slowly destroys memory, thinking skills, and eventually, even the ability to carry out the simplest tasks. It is characterized by death of synapses coupled to death nerve cells and brain degeneration which is manifested by loss of cognitive abilities. Understanding neuroprotection in Alzheimer’s disease will pave the path to better disease management and novel therapeutics. Comprehensive reference detailing neuroprotection in Alzheimer’s Disease, with details on nerve cell protection and new advances in disease management Combines the knowledge and points-of-view of both medical doctors and basic scientists, putting the subject at the forefront for further clinical development Edited by one of the leading researchers in Alzheimer’s Disease
Neural Stem Cell Transplantation Dec 31 2022
Effect of Ketamine on Neurogenesis and Neuroprotection Following Lithium-pilocarpine Induced Status Epilepticus in Rats Apr 22 2022
Experimental Stroke Jul 26 2022 "This Ebook compiles the efforts of 20 experts in the field to review the latest advances in experimental stroke, with its strong emphasis on neurogenesis, angiogenesis and neuroprotection after cerebral ischemic stroke. It also provides current data for p"
Issues in Neuroscience Research and Application: 2011 Edition Jan 26 2020 Issues in Neuroscience Research and Application: 2011 Edition is a ScholarlyEditions™ eBook that delivers timely, authoritative, and comprehensive information about Neuroscience Research and Application. The editors have built Issues in Neuroscience Research and Application: 2011 Edition on the vast information databases of ScholarlyNews.™ You can expect the information about Neuroscience Research and Application in this eBook to be deeper than what you can access anywhere else, as well as consistently reliable, authoritative, informed, and relevant. The content of Issues in Neuroscience Research and Application: 2011 Edition has been produced by the world’s leading scientists, engineers, analysts, research institutions, and companies. All of the content is from peer-reviewed sources, and all of it is written, assembled, and edited by the editors at ScholarlyEditions™ and available exclusively from us. You now have a source you can cite with authority, confidence, and credibility. More information is available at http://www.ScholarlyEditions.com/.
Neuroprotection Nov 29 2022 In this first book to cover model systems, molecular mechanisms and clinical trials all in one volume internationally renowned scientists and clinicians provide a comprehensive treatment of neuroprotective strategies for all important

neurological disorders. Following an overview of neurodegenerative, traumatic, and ischemic disorders, the book goes on to cover in vitro and animal model systems as well as cellular and molecular mechanisms. An extremely helpful analysis of clinical studies explains reasons for their success and failure, and the whole is rounded off with a look at the current challenges and hopes for the development of effective treatment strategies in the future.

To Investigate Neuroprotective Mechanism in Female Brain Oct 05 2020 It is well known that gender differences exist in the experimental or clinical stroke with respect to the tissue damage and the loss of functional outcome. We have previously reported neuroprotective properties of Ginkgo biloba/EGb 761® (EGb 761) in transient and permanent mouse models of brain ischemia in male mice and the mechanism of neuroprotection was attributed to the upregulation of the HO1/Wnt pathway. Here, we sought to investigate the novel hypothesis, whether neuroprotection by EGb 761 in ovariectomized (OVX) female mice is mediated by HO1/Wnt upregulation in a model of permanent cerebral ischemia. The OVX mice pretreated with EGb 761 for 7 days were subjected to permanent ischemia at day 8 and sacrificed on day 14. Infarct volume analysis showed that EGb 761 pretreated OVX mice had lower infarct volume, lower neurological deficits and improved motor skills as compared to OVX female vehicle mice. Protein analysis studies demonstrated that neuroprotection in EGb 761 pretreatment OVX group is not mediated by HO1/Wnt pathway. Furthermore EGb 761 pretreatment group demonstrated overexpression of vascular endothelial growth factor (VEGF) and endothelial nitric oxide (eNOS). In addition, increased expression of TNF-a in the vehicle group was observed to be comparatively lower in EGb 761 pretreated OVX group. These results suggest that the neuroprotective effect of EGb 761 in females is not associated with the activation and upregulation of HO1/Wnt pathway. To further understand the cell death mechanism involved, we studied expression levels of apoptotic protein cleaved caspase-3 and caspase-8 which were found to be significantly elevated in the Veh/OVX group as compared to the EGb 761/OVX group. We previously showed that EGb 761 post and pretreatment enhances neurogenesis by increasing the number of neural stem progenitor cells (NSPC's) in male mice 7 days following permanent ischemia, similarly in this study we demonstrated that in absence of ovarian hormone estrogen, EGb 761 increased NSPC's 7 days after ischemia. To test whether EGb 761 in absence of estrogen activates another gonadal steroid androgen, luciferase assay and immunofluorescent studies suggested that EGb 761 significantly binds and results in activation of androgen receptor (AR) in female brain. Taken together these results suggest that the possible mechanism of EGb 761 mediated neuroprotection in females following permanent ischemia is independent of HO1/Wnt signaling and via caspase dependent pathway and neurogenesis.

Neurogenesis and Neural Plasticity Dec 27 2019 This volume brings together authors working on a wide range of topics to provide an up to date account of the underlying mechanisms and functions of neurogenesis and synaptogenesis in the adult brain. With an increasing understanding of the role of neurogenesis and synaptogenesis it is possible to envisage improvements or novel treatments for a number of diseases and the possibility of harnessing these phenomena to reduce the impact of ageing and to provide mechanisms to repair the brain.

Mechanisms of Neuroinflammation and Inflammatory Neurodegeneration in Acute Brain Injury May 12 2021 Mechanisms of brain-immune interactions became a cutting-edge topic in systemic neurosciences over the past years. Acute lesions of the brain parenchyma, particularly, induce a profound and highly complex neuroinflammatory reaction with similar mechanistic properties between differing disease paradigms like ischemic stroke, intracerebral hemorrhage (ICH) and traumatic brain injury (TBI). Resident microglial cells sense tissue damage and initiate inflammation, activation of the endothelial brain-immune interface promotes recruitment of systemic immune cells to the brain and systemic humoral immune mediators (e.g. complements and cytokines) enter the brain through the damaged blood-brain barrier. These cellular and humoral constituents of the neuroinflammatory reaction to brain injury contribute substantially to secondary brain damage and neurodegeneration. Diverse inflammatory cascades such as pro-inflammatory cytokine secretion of invading leukocytes and direct cell-cell-contact cytotoxicity between lymphocytes and neurons have been demonstrated to mediate the inflammatory ‘collateral damage’ in models of acute brain injury. Besides mediating neuronal cell loss and degeneration, secondary inflammatory mechanisms also contribute to functional modulation of neurons and the impact of post-lesional neuroinflammation can even be detected on the behavioral level. The contribution of several specific immune cell subpopulations to the complex orchestration of secondary neuroinflammation has been revealed just recently. However, the differential vulnerability of specific neuronal cell types and the molecular mechanisms of inflammatory neurodegeneration are still elusive. Furthermore, we are only on the verge of characterizing the control of long-term recovery and neuronal plasticity after brain damage by inflammatory pathways. Yet, a more detailed but also comprehensive understanding of the multifaceted interaction of these two supersystems is of direct translational relevance. Immunotherapeutic strategies currently shift to the center of translational research in acute CNS lesion since all clinical trials investigating direct neuroprotective therapies failed. To advance our knowledge on brain-immune communications after brain damage an interdisciplinary approach covered by cellular neuroscience as well as neuroimmunology, brain imaging and behavioral sciences is crucial to thoroughly depict the intricate mechanisms.

New Perspectives in Neurosteroids action: a Special Player Allopregnanolone Mar 22 2022 Early in the 80’s date the first observations on the existence of hormonal steroids that may be synthesized and act in the nervous system. In order to refer to these endogenous steroids, proved important to control both central and peripheral nervous system, it was proposed the term “neurosteroids” (NSs). Over the years, their importance in regulating the physiological functions of neuronal and glial cells increased progressively. These steroids can be involved in several pathophysiological conditions such as depression, anxiety, premenstrual syndrome (PMS), schizophrenia and Alzheimer disease. Among the different classes of NSs, the progestagens revealed particularly important. The progesterone metabolite 5a-pregnan-3a-ol-20-one, also named tetrahydroprogesterone or allopregnanolone (ALLO) was one of the first most important steroid that was originally shown to act as neurosteroid. ALLO is synthesized through the action of the 5aR-3a-HSD, which converts P into DHP and subsequently, via a bidirectional reaction, into ALLO. NSs exert complex effects in the nervous system through ‘classic’, genomic, and ‘non-classic’, non-genomic actions. ALLO displays a rapid ‘non-genomic’ effect, which mainly

involves the potent modulation of the GABA type A (GABA-A) receptor function. Recently a membrane receptor has been identified as target for ALLO effects, i.e. the membrane progesterone receptors (mPRs) that are able to activate a signalling cascade through G protein dependent mechanisms. By these ways, ALLO is able to modulate several cell functions, acting as neurogenic molecule on neural progenitor cells, as well as by activating proliferation and differentiation of glial cells in the central and peripheral nervous system. In this topic, we review the most recent acquisitions in the field of neurosteroids, focusing our attention on ALLO because its effects on the physiology of neurons and glial cells of the central and peripheral nervous system are intriguing and could potentially lead to the development of new strategies for neuroprotection and/or regeneration of injured nervous tissues and for the treatment of neuropsychiatric disorders.

Brain Neurotrauma Aug 15 2021 Every year, an estimated 1.7 million Americans sustain brain injury. Long-term disabilities impact nearly half of moderate brain injury survivors and nearly 50,000 of these cases result in death. Brain Neurotrauma: Molecular, Neuropsychological, and Rehabilitation Aspects provides a comprehensive and up-to-date account on the latest developments in the area of neurotrauma, including brain injury pathophysiology, biomarker research, experimental models of CNS injury, diagnostic methods, and neurotherapeutic interventions as well as neurorehabilitation strategies in the field of neurotraum research. The book includes several sections on neurotrauma mechanisms, biomarker discovery, neurocognitive/neurobehavioral deficits, and neurorehabilitation and treatment approaches. It also contains a section devoted to models of mild CNS injury, including blast and sport-related injuries. Over the last decade, the field of neurotrauma has witnessed significant advances, especially at the molecular, cellular, and behavioral levels. This progress is largely due to the introduction of novel techniques, as well as the development of new animal models of central nervous system (CNS) injury. This book, with its diverse coherent content, gives you insight into the diverse and heterogeneous aspects of CNS pathology and/or rehabilitation needs.

Neurogenesis and Neuroprotection in Huntington Disease Aug 27 2022 "In conclusion, our study confirmed the involvement and the importance of neurogenesis in influencing learning and spatial memory, and demonstrated that a combination of FGF6 with HDAC inhibitor CHDI-3, have the ability to enhance two different neurogenesis signaling pathways, through the activation of pERK1/2 and the activation of BDNF, leading to an increase in learning and memory. These results suggest that FGF6 combined with HDAC inhibitor CHDI-3, can be utilized for future therapeutic treatment to enhance learning and memory for HD patient."--Page 19.

Single Administration of Fluoxetine Improves Memory Function Without Neuroprotection After Cardiac Arrest/cardiopulmonary Resuscitation in Mice Mar 29 2020 Background and Goal of study: Several clinical studies have indicated that serotonin re-uptake inhibitor (SSRI) administration after acute ischemic stroke can improve clinical recovery. Fluoxetine (FLX: one of the SSRIs) has several mechanisms which contribute to relieve the ischemic brain damage. We have previously reported that FLX had neuroprotective effects on day3 after cardiac arrest and cardiopulmonary resuscitation (CA/CPR) in mice. However, the neuronal loss after global ischemia is suspected to continue up to 14 days after insults. The goal of this study is to evaluate the neuroprotective effect of FLX for

a longer observation period. Material and methods: PROTOCOL 1: Global cerebral ischemia was induced in male C57BL/6 mice for 7 minutes of CA. Thirty minutes after recovery of spontaneous circulation, the mice were randomly assigned to 2 groups and administered either FLX 10 mg/kg (Group F: n = 12) or 0 mg/kg (Group C: n = 12). Six and seven days after CA/CPR, behavioral tests (passive avoidance test) were conducted and brains were removed for histological evaluation. PROTOCOL 2: Methods of global ischemia and behavioral test were same as protocol 1. Fourteen days after CA/CPR, behavioral tests and histological evaluations were similarly conducted. (Group F: n=24, Group C: n=24) Data are presented as meanu00b1SEM, and the groups were compared by unpaired t test or Mann-Whitney U test. The u03c72 test was used for survival analysis. Results and discussion: Seven days after CA/CPR, there was a significant difference in number of surviving neurons (Neu-N positive cell) between the groups. On day14, the difference disappeared. However, Group F showed a significantly greater number of immature neurons (Doublecortin positive cell) than Group C. No differences were found in number of microglia (IBA-1 positive cell). At this time point, memory function significantly improved in Group F. The previous study has shown that the neurogenesis in hippocampus modulates the hippocampus-dependent period of memory, without loss of memory. The functional recovery in this study may have been induced by neurogenesis in hippocampus.Conclusion: Fluoxetine improved memory function after cardiac arrest. This recovery may be due to neurogenesis in hippocampus, but not due to surviving neurons. Further investigation is needed to clarify the interaction of memory function and neurogenesis after fluoxetine administration.

Microglia in Health and Disease Jul 02 2020 These past few years have witnessed a revolution in our understanding of microglia, especially since their roles in the healthy central nervous system (CNS) have started to unravel. These cells were shown to actively maintain health, in concert with neurons and other types of CNS cells, providing further insight into their involvement with diseases. Edited by two pioneers in the field, Marie-Ève Tremblay and Amanda Sierra, Microglia in health and disease aims to share with the broader scientific community some of the recent discoveries in microglia research, from a broad perspective, with a collection of 19 chapters from 52 specialists working in 11 countries across 5 continents. To set microglia on the stage, the book begins by explaining briefly who they are, what they do in the healthy and diseased CNS, and how they can be studied. The first section describes in more details their physiological roles in the maturation, function, and plasticity of the CNS, across development, adolescence, adulthood, neuropathic pain, addiction, and aging. The second section focuses on their implication in pathological conditions impairing the quality of life: neurodevelopmental and neuropsychiatric disorders, AIDS, and multiple sclerosis; and in leading causes of death: ischemia and stroke, neurodegenerative diseases, as well as trauma and injury.

Melatonin Jun 24 2022 There is growing interest in the field of melatonin research regarding its neurobiological mechanisms as well as its repercussions in clinical practice. Melatonin: Therapeutic Value and Neuroprotection explores melatonin’s neuroprotective effects and discusses the therapeutic potential of melatonin and melatonin agonists in treating neurodegenerative diseases and other ailments. Topics include: The basic aspects of melatonin’s physiology, including its production, bioavailability, and metabolism The

functional importance of melatonin receptors and their role in mediating the therapeutic effectiveness of melatonin in cancer Melatonin's effect on the regulation of blood pressure, sleep, and circadian rhythms The cardioprotective role of melatonin The neuroprotective role in glaucoma, Alzheimer's disease, Parkinson's disease, and neurodegenerative diseases Use as a therapeutic agent for treating epilepsy and degenerative discs Treatment for obesity, diabetes mellitus, and other metabolic disorders Protective role in peri-natal hypoxic-ischemia The contributors also examine the discovery of a number of melatonergic agonists, their potential role as antioxidants, and their therapeutic applications in treating glaucoma, Parkinson's disease, Alzheimer's disease, primary insomnia, and psychiatric disorders. Opening new vistas in our understanding of etiology, pharmacotherapy, and treatment, the book is a significant milestone in our knowledge about advances in melatonin's physiology and its therapeutic application in a number of disorders.
Neuroimmunity Dec 07 2020 Pathbreaking research offers new hope for treating brain diseases and injuries and for maintaining brain health even into old age In the past, the brain was considered an autonomous organ, self-contained and completely separate from the body's immune system. But over the past twenty years, neuroimmunologist Michal Schwartz, together with her research team, not only has overturned this misconception but has brought to light revolutionary new understandings of brain health and repair. In this book Schwartz describes her research journey, her experiments, and the triumphs and setbacks that led to the discovery of connections between immune system and brain. Michal Schwartz, with Anat London, also explains the significance of the findings for future treatments of brain disorders and injuries, spinal cord injuries, glaucoma, depression, and other conditions such as brain aging and Alzheimer's and Parkinson's diseases. Scientists, physicians, medical students, and all readers with an interest in brain function and its relationship to the immune system in health and disease will find this book a valuable resource. With general readers in mind, the authors provide a useful primer to explain scientific terms and concepts discussed in the book.
Breathing, Feeding, and Neuroprotection May 24 2022 This book presents a detailed analysis of current understanding of breathing. Chapters on neuroprotection examine the functional significance of the blood – brain barrier; other chapters examine health and disease in relation to the hypothalamic and limbic systems. The book reviews research on the human brain, focusing on the recently developed EEG/dipole tracing method, as well as animal experiments. The book is a useful reference for researchers in neuroscience and related fields.
Estrogen Effects on Traumatic Brain Injury Jan 20 2022 This book demystifies, deconstructs, and simultaneously humanizes the field of estrogen-mediated neuroprotection following TBI, making the subject approachable to both researchers and advanced students. Bringing together leading researchers and practitioners to explain the basis for their work, methods, and their results, chapters explore what is known about the role of estrogens following damage to the brain. With topics covering induction of estrogen response, consequences of estrogen action, and mechanisms underlying estrogen mediated neuroprotection, Estrogen Effects on Traumatic Brain Injury is of great importance to teachers, researchers, and clinicians interested in the role that estrogens play following traumatic brain injury. Written to provide a foundational view of estrogens as neuroprotectors in TBI, appropriate for both researchers and advanced

students Data Analysis boxes in each chapter help with data interpretation and offer guidelines on how best to understand results. A multidisciplinary approach to the methods, issues, empirical findings in the field of estrogen mediated neuroprotection. Detailed focus on how studies relate and build upon each other and the ways different methods of analysis inform each other. Written to provide clinicians with new and developing treatment options for patients in their field.

Stem Cells and Progenitor Cells in Ischemic Stroke - Fashion or Future? Dec 19 2021 Stroke remains one of the most devastating diseases in industrialized countries. Recanalization of the occluded arterial vessel using thrombolysis is the only causal therapy available. However, thrombolysis is limited due to severe side effects and a limited time window. As such, only a minority of patients receives this kind of therapy, showing a need for new and innovative treatment strategies. Although neuroprotective drugs have been shown to be beneficial in a variety of experimental stroke models, they ultimately failed in clinical trials. Consequently, recent scientific focus has been put on modulation of post-ischemic neuroregeneration, either via stimulation of endogenous neurogenesis or via application of exogenous stem cells or progenitor cells. Neurogenesis persists within the adult brain of both rodents and primates. As such, neural progenitor cells (NPCs) are found within distinct niches like the subventricular zone (SVZ) of the lateral ventricles and the subgranular zone of the dentate gyrus. Cerebral ischemia stimulates these astrocyte-like progenitor cells, upon which NPCs proliferate and migrate towards the site of lesion. There, NPCs partly differentiate into mature neurons, without significantly being integrated into the residing neural network. Rather, the majority of new-born cells dies within the first weeks post-stroke, leaving post-ischemic neurogenesis a phenomenon of unknown biological significance. Since NPCs do not replace lost brain tissue, beneficial effects observed in some studies after either stimulated or protected neurogenesis are generally contributed to indirect effects of these new-born cells. The precise identification of appropriated cellular mediators, however, is still elusive. How do these mediators work? Are they soluble factors or maybe even vesicular structures emanating from NPCs? What are the cues that guide NPCs towards the ischemic lesion site? How can post-ischemic neurogenesis be stimulated? How can the poor survival of NPCs be increased? In order to support post-ischemic neurogenesis, a variety of research groups have focused on application of exogenous stem/progenitor cells from various tissue sources. Among these, cultivated NPCs from the SVZ and mesenchymal stem cells (MSCs) from the bone marrow are frequently administered after induction of stroke. Although neuroprotection after delivery of stem/progenitor cells has been shown in various experimental stroke models, transplanted cells are usually not integrated in the neural network. Again, the vast amount of grafted cells dies or does not reach its target despite profound neuroprotection, also suggesting indirect paracrine effects as the cause of neuroprotection. Yet, the factors being responsible for these observations are under debate and still have to be addressed. Is there any “optimal” cell type for transplantation? How can the resistance of grafted cells against a non-favorable extracellular milieu be increased? What are the molecules that are vital for interaction between grafted cells and endogenous NPCs? The present research topic seeks to answer - at least in part - some of the aforementioned questions. Although the research topic predominantly focuses on experimental studies (and reviews alike), a current outlook towards clinical relevance is given as well.

Neurodegenerative Diseases Feb 27 2020 Neurodegenerative diseases are widespread disorders, with prevalence increasing with age. The first chapter of this book discusses autophagy, a cellular tool for the elimination of proteins that accumulate in neurodegenerative disorders and have pathogenetic significance. Parkinson's disease (PD) is one of the most common degenerative diseases of the central nervous system. Chapter Two examines evidence on impairment of social cognition in patients with PD. Chapter Three discusses how finding the molecular cause of the PD could contribute to early diagnosis, and facilitate the development of effective neuroprotective or modulatory drugs. The final chapter reviews the functional role of adult hippocampal neurogenesis in learning and memory, and how this form of structural plasticity is altered in neurodegenerative diseases known to involve cognitive impairment such as Alzheimer's disease, Parkinson's disease, and Huntington's disease.

The Hippocampus Sep 03 2020 The hippocampus is an important brain region, a true central hub for memory of various kinds and other processes. Neuropsychiatric disorders such as Alzheimer's disease, drug addiction, and schizophrenia are characterized by hippocampal alterations. The dentate gyrus of the hippocampus is a site exhibiting adult neurogenesis. This book covers the topic of the hippocampus from various perspectives. It discusses adult neurogenesis, effect of enriched environments on hippocampal plasticity, and long-term potentiation-associated gene expression. The book also addresses multiscale representations of complex environments and strategies in the hippocampus-dependent spatial tasks. Finally, insight into the hippocampus as a link between negative affect and relapse to psychostimulants is provided. The book collects evidence of various hippocampal functions in healthy and disordered brain.

Implications of Pgrmc1 Regulation of Kit Ligand Synthesis in the Hippocampus Jan 08 2021 The mammalian hippocampus is responsible for many crucial brain functions such as learning, memory, and neurogenesis in adults. Its degeneration is a pathology associated with the early stages of Alzheimer's disease. A variety of genes have been associated with both neuroprotection and neurogenesis in the brain, some of which include progesterone membrane component 1 (Pgrmc1) and kit ligand (KitL). Pgrmc1 is recognized for mediating hormonal functions in both the ovary and neuroendocrine regions such as the anteroventral periventricular nucleus (AVPV), but its functions in the hippocampus are not well known. Both Pgrmc1 and KitL share downstream targets, the most strongly supported being genes in the Janus kinase (Jak)/signal transducer and activator of transcription (Stat) pathway. I hypothesized that Pgrmc1 regulates neural targets through KitL/c-Kit signaling. To investigate this hypothesis I used a variety of in vivo and in vitro techniques. These techniques included mapping both KitL and receptor c-Kit in the adult female rat brain using in situ hybridization. I used Pgrmc1 silencing with siRNA in hippocampal-derived mHe-18 cells and Pgrmc1/2 double conditional knock out mouse brains to study Pgrmc1 regulation of KitL synthesis. To determine common downstream targets of KitL and Pgrmc1 I then treated mHe-18 cells with soluble KitL protein. Finally, to determine whether c-Kit mediated effects of Pgrmc1, I treated cells with both Pgrmc1 siRNA and AG-1296, a c-Kit inhibitor. The results show that Pgrmc1 regulates KitL expression, as well as downstream targets Pias1, 2, 3, and 4. However, AG-1296 did not abrogate Pgrmc1 regulation of the downstream targets, demonstrating regulation independent of KitL signaling. Taken together, these results suggest that while Pgrmc1 alters KitL expression and regulates the same genes as KitL/c-Kit,

the mechanism of action likely differs. Considering that these two genes are involved in neurogenesis and neuroprotection, as well as memory and learning, a better understanding of the pathways may help lead the way in treating neurodegenerative diseases in the future.

Molecular Aspects of Neurodegeneration, Neuroprotection, and Regeneration in Neurological Disorders Nov 17 2021 Molecular Aspects of Neurodegeneration, Neuroprotection, and Regeneration in Neurological Disorders presents readers with comprehensive and cutting-edge information on the neurochemical mechanisms of various types of neurological disorders. The book covers information on signal transduction processes associated with neurochemistry of neurological disorders, including neurodegenerative, neurotraumatic, and neuropsychiatric disorders. The book also discusses risk factors, symptoms, pathogenesis, biomarkers, and the potential treatments of neurological disorders. The comprehensive information in this monograph may not only help in early detection of various neurological disorders, but will also promote the discovery of new drugs. Provides a comprehensive overview of the molecular aspects of neurodegeneration, neuroprotection, and neuro-regeneration, along with therapeutic strategies for various types of neurological disorders Provides cutting-edge research information on the signal transduction processes associated with the neurochemistry of neurological disorders Discusses risk factors, symptoms, pathogenesis, biomarkers, and the potential for treatments of neurological disorders

Study of the Effects of Docosahexaenoic Acid (DHA) and a Structured Phospholipid Containing DHA on Physiological and Pathological Conditions of Neurogenesis in Vitro Apr 10 2021 Docosahexaenoic acid (DHA, 22:6n-3) is an essential omega-3 polyunsaturated fatty acid (PUFA). It is specifically enriched in the brain and the retina and it is required for visual acuity, proper brain development and cerebral functions. While DHA deficiency in the brain was shown to be linked to the emergence of cerebral diseases (i.e. Alzheimer's disease or Parkinson's disease), studies showed that a dietary intake of omega-3 PUFA could prevent or attenuate neurologic disturbances linked with ageing or neurodegenerative diseases. In this context, it is primary to deliver DHA efficiently to the brain. Targeting the brain with DHA might offer great promise in developing new therapeutics for neurodegenerative diseases. The French host laboratory previously synthesized a stabilized form of lysophosphatidylcholine-DHA, which is main vector of DHA transportation to the brain, of structure 1-acetyl,2-docoshexaenoyl-glycerophosphocholine, patented and named AceDoPC®. Injection of AceDoPC or DHA after experimental ischemic stroke showed that both molecules also had neuroprotective effects. These potential neuroprotective effects are expected to be due, in part, to DHA conversion into oxygenated metabolites. This study aims to investigate the beneficial effects of DHA and its derived metabolites either unesterified or esterified within structured phospholipids on a model of neurogenesis in vitro under physiological or pathological conditions. The first objective of this work was then to synthesize the DHA-containing structured phospholipid AceDoPC®, DHA oxygenated derivative protectin DX (PDX) and a novel structured phospholipid of protectin: 1-acetyl,2-protectinDX-glycerophosphocholine (AceDoxyPC). The second objective was to investigate the effects of DHA, AceDoPC and PDX on neurogenesis using an in vitro model of neurogenesis, namely cultures of neural stem progenitor cells

(NSPCs) derived from the adult mouse brain under physiological or pathological conditions (ischemic conditions). Following this, the third objective of this work was to identify the mechanisms involved in such response to stress induced under pathological conditions. Synthesis of the novel structured phospholipid AceDoxyPC was successfully performed by double enzymatic lipoxygenation of AceDoPC and identification of the product was possible using advanced techniques of liquid chromatography (LC)/electrospray ionization (/ESI)/mass spectrometry (/MS). Future studies on this potential neuroprotective molecule transporter are to be investigated in the near future. Neurogenesis study of cell cultures with AceDoPC showed enhanced neurogenesis compared to addition of unesterified DHA or vehicle control, especially under pathological conditions. Preliminary studies of the potential mechanisms involved in neuroprotection hinted that AceDoPC neuroprotective and regenerative effects might be due in part to its anti-oxidative effects.

Never-resting microglia: physiological roles in the healthy brain and pathological implications Jun 12 2021 Microglia are largely known as the major orchestrators of the brain inflammatory response. As such, they have been traditionally studied in various contexts of disease, where their activation has been assumed to induce a wide range of detrimental effects. In the last few years, a series of discoveries have challenged the current view of microglia, showing their active and positive contribution to normal brain function. This Research Topic reviewed the novel physiological roles of microglia in the developing, mature and aging brain, under non-pathological conditions. In particular, this Research Topic discussed the cellular and molecular mechanisms by which microglia contribute to the formation, pruning and plasticity of synapses; the regulation of adult neurogenesis as well as hippocampal learning and memory; among other important roles. Because these novel findings defy our understanding of microglial function in health as much as in disease, this Research Topic also summarized the current view of microglial nomenclature, phenotypes, origin and differentiation, and contribution to various brain pathologies. Additionally, novel imaging approaches and molecular tools to study microglia in their non-activated state have been discussed. In conclusion, this Research Topic seeked to emphasize how the current research in neuroscience is challenged by never-resting microglia.

Chemokines and Chemokine Receptors in Brain Homeostasis May 31 2020 Virtually involved in all pathologies that present an inflammatory component, it is now evident that, in the central nervous system, chemokines and chemokine receptors possess pleiotropic properties beyond chemotaxis: costitutive brain expression of chemokines and their receptors on endothelial cells, but also on neurons and glia, suggests a role for such molecules in mediating homeostatic cross-talk between cells of the brain perenchyma. Cross-talk between neurons and glia is determinant to the establishment and maintenance of a brain enviroment that ensure normal function, and in particular glial cells are active players that respond to enviromental changes and act for the survival, growth, differentiation and repair of the nervous tissue: in this regard brain endogenous chemokines represent key molecules that play a role in brain development, neurogenesis, neurotransmission and neuroprotection. As important regulators of peripheral immune response, chemokines are molecules of the immune system that play a central role in coordinating communication between the nervous and the

immune systems, in the context of infections and brain injury. Indeed, in phatological processes resulting from infections, brain trauma, ischemia and chronic neurodegenerative diseases, chemokines represent important neuroinflammatory mediators that drive leucocytes trafficking into the central nervous system, facilitating an immune response by targeting cells of the innate and adaptive immune system. The third edition of the international conference "Chemokines and Chemokine Receptors in the Nervous System", hold in Rome in October 2013, represented an exciting platform to promote discussion among researchers in different disciplines to understand the role of chemokines in brain homoestasis. This Frontiers Research Topic arises from this conference, and wants to be an opportunity to further discuss and highlight the importance of brain chemokines as key molecules that, not only grant the interplay between the immune and the nervous systems, but in addition drive modulatory functions on brain homeoastasis orchestrating neurons, microglia, and astrocytes communication.

Neuroprotection in Autism, Schizophrenia and Alzheimer's disease Sep 27 2022 Neuroprotection in Autism, Schizophrenia and Alzheimer's Disease provides an up-to-date overview on recent clinical studies and the similarities discovered in the most prevalent brain disorders. The book's content will help shed light on basic mechanisms and provide new avenues for early diagnosis toward disease prevention and disease modification. It is written for researchers, clinicians and medical physicians in neuroscience, neurology and psychiatry. Sections discuss the shared pathophysiological mechanisms that underlie autism, schizophrenia/mood disorders and Alzheimer’s disease, i.e. neurodevelopmental disorders, neuropsychiatric diseases and neurodegenerative disorders. Offers an up-to-date overview of basic and clinical studies concerning similarities in the most prevalent brain disorders Helps the reader become familiar with novel neuroprotective mechanisms and experimental treatment modalities in these difficult to treat disorders Written for researchers, clinicians and medical physicians in neuroscience, neurology and psychiatry

The Neuroprotective Compound P7C3-A20 Promotes Neurogenesis and Improves Functional Outcomes After Focal Cerebral Ischemia Nov 05 2020 Ischemic stroke is the second leading cause of death worldwide and the leading cause of adult long-term disability in the United States. Despite its prevalence, there are few therapeutic interventions available. The neuroprotective compound P7C3-A20 (A20) has been shown to reduce mature neuronal cell death while also increasing the net magnitude of postnatal neurogenesis in models of neurodegeneration and acute brain injury. A20 compounds demonstrate protection by enhancing the flux of nicotinamide adenine dinucleotide (NAD) in mammalian cells, a proposed therapeutic approach to treating cerebral ischemia. The studies carried out in this dissertation sought to investigate the effectiveness of A20 treatment after focal cerebral ischemia by assessing subacute and chronic histopathological and behavioral outcomes, as well as ischemia-induced neurogenesis. In the first series of experiments, rats underwent a weeklong course of A20 or vehicle treatment, beginning immediately after a 90 minute unilateral transient middle cerebral artery occlusion (tMCAO). A20-treated rats performed significantly better than vehicle-treated controls in sensorimotor cylinder and grid-walk tasks, and in a chronic test of spatial learning and memory. These behavioral improvements with A20 treatment were correlated with significantly decreased cortical and hippocampal atrophy as well as increased neurogenesis in the subventricular zone

and hippocampal dentate gyrus subgranular zone. Furthermore, cerebral ischemia significantly depleted NAD in the cortex, but treating with A20 restored cortical NAD levels. After demonstrating efficacy of A20 treatment at an early, post-ischemic timepoint, we then sought to examine A20's treatment window of opportunity. Due to a limited therapeutic window, current stroke pharmacological treatment is rarely administered to ischemic patients. Therefore, we investigated a more clinically relevant time point and again treated tMCAO rats for one week with A20, beginning either immediately (iA20) or at a delayed point (dA20) 6 hours post-reperfusion. dA20 treatment significantly reduced ischemia-induced sensorimotor deficits in motor coordination and limb-use asymmetry as well as cognitive deficits in hippocampal-dependent spatial learning, memory retention, and working memory. In the cerebral cortex, dA20 treatment significantly increased tissue sparing 7 weeks after stroke and reduced infarct volumes 48 hours after reperfusion compared to vehicle-treated animals. At 48 hours after injury, there was no change in striatal infarct volumes between tMCAO groups. However, when tissue volume was reassessed at 7 weeks, A20-treated animals had a significant increase in striatal tissue volume, suggesting that A20's protection in the ischemic striatum requires an extended treatment regimen. In the hippocampus, only iA20-treated animals had a significant increase in tissue sparing compared to vehicle-treated stroke animals. This translated into minimal hippocampal-dependent behavioral improvements with dA20 treatment. However, all rats treated with dA20 did demonstrate a significant improvement in both sensorimotor tasks compared to vehicle controls, suggesting a somatosensory driven recovery. Overall, our studies show that A20 treatment is an effective strategy against focal cerebral ischemia by mitigating chronic neurodegeneration, enhancing repair, and rescuing stroke-induced behavioral deficits when treated at a clinically relevant time point. Therefore, treatment with A20 compounds represent a novel therapeutic approach to safely augment NAD tissue levels, promoting two independent processes critical to protecting the brain from ischemic stroke; mature neuron survival and postnatal hippocampal neurogenesis throughout the post-ischemic brain.

The Neuroprotective Effects of Z-bisdehydrodoisynolic Acid Following Traumatic Brain Injury Oct 17 2021 Experiment 3 attempted to determine if Z-BDDA treatment reduced edema following CCI injury in young male rats. Analysis of percent brain water 50 hours following injury revealed a Z-BDDA induced decrease in hippocampal edema ipsilaterally in comparison to controls. No other differences were found.

Neuroprotective Effects of Physical Exercise on Stressed Brain Apr 30 2020

Alpha-linolenic Acid Confers Neuroprotection and Improves Behavioral Deficits After Soman Exposure: Involvement of Neurogenesis Through an MTOR-mediated Pathway Feb 06 2021

The Rat Brain in Stereotaxic Coordinates Aug 03 2020 This completely revised edition of The Rat Brain in Stereotaxic Coordinates, the second most cited book in science, represents a dramatic update from the previous edition. Based on a single rat brain, this edition features an entirely new coronal set of tissue cut in regular 120 micron intervals with accompanying photographs and drawings of coronal, horizontal and sagittal sections of this new set. The use of the single brain allows for greater consistency between sections,

while advances in histochemistry techniques provides increased refinement in the definition of brain areas, making this the most accurate and detailed stereotaxic rat atlas produced to date. The atlas will also include a CD-ROM featuring all of the graphics and text. Every lab working with the rat as an experimental animal model will want to use this book as their atlas of choice. This book is also available in a softcover spiral binding at the same price. * Includes twice as many coronal sections, nissl plates, and sagittal plates as the previous edition * Uses a single rat brain allowing for better consistency and better delineations in the line drawings of structures * Provides improved stereotaxic coordinates at a higher level of detail * Accompanying CD-ROM features graphics and text * Now available as hardcover version and softcover version with a spiral binding at the same price.

Melatonin, Neuroprotective Agents and Antidepressant Therapy Jul 14 2021 This work is a guidebook for clinicians who are involved in treating depressive patients and also serves the research scientists who are working on the psychopharmacological mechanisms of antidepressant actions and psychopathological mechanisms underlying mood disorders. Mood disorders such as major depressive disorder (MDD), bipolar disorder (BPD) and seasonal affective disorder (SAD) are the most disabling disorders that are among the most expensive of all medical illnesses. The pathophysiology of mood disorders is very complex and involves many mechanisms like circadian rhythm disruption, sleep abnormalities, melatonin rhythm abnormalities and alterations in melatonin receptor mechanisms, abnormalities in monoaminergic neurotransmitter mechanisms, glutamatergic release mechanisms, hippocampal neurogenesis, and abnormal immune and cytokine release mechanisms. Many antidepressants that are in clinical use today including the recently introduced novel agents like agomelatine or other antidepressants cause clinical remission by resynchronizing disrupted circadian rhythms and melatonin receptor functions, enhancing monoaminergic neurotransmission, promoting hippocampal neurogenesis, and regulating immune mechanisms. This book explains various etiological factors that are involved in the pathogenesis of mood disorders and the mechanisms of therapeutic actions of antidepressants including the recently introduced agomelatine and other antidepressants that exhibit rapid onset of action with greater efficacy and fewer side effects. .

Molecular Aspects of Neurodegeneration and Neuroprotection Sep 15 2021 "Neurodegenerative diseases are a complex heterogeneous group of diseases associated with site-specific premature and slow death of certain neuronal populations in brain and spinal cord tissues. For example, in Alzheimer disease, neuronal degeneration occu"

Neural Progenitor Cell Transplantation and Proneurogenic Compound Administration Improve Outcomes After Trauma Oct 29 2022 Traumatic brain injury (TBI) represents a serious public health problem as there are no clinically-available treatments to mitigate the functional complications and societal burdens endured by patients and their caregivers. In addition to the primary mechanical insult, deleterious secondary injuries contribute to the progressive atrophy and long-term histopathological changes that impair functional and cognitive outcomes. The studies carried out in this dissertation project assessed two treatment strategies designed to engage and enhance endogenous neurorestorative responses in the injured brain. We postulated that the protection of vulnerable cortical neurons and perilesional parenchyma together with the promotion of endogenous hippocampal neurogenesis would confer

histological and behavioral improvement after brain injury. The first series of experiments evaluated the effects of transplanting syngeneic neural progenitor cells (NPCs) with or without genetic modification to secrete a synthetic multineurotrophin (MNTS1) with multifunctional, multitargeting, neurotrophic capacity. NPCs were obtained from Sprague Dawley fetuses at embryonic stage E15 and transduced with either MNTS1 and GFP constructs (MNTS1-NPCs) or with GFP and blue fluorescent protein (BFP) constructs (control GFP-NPCs). Adult Sprague Dawley rats received a moderate fluid percussion-induced insult over the right parietal cortex or underwent sham surgery. Animals were transplanted pericontusionally 1 week later with either control GFP-NPCs, MNTS1-NPCs, or injected with saline (vehicle). Five weeks after surgery, groups were evaluated for hippocampal-dependent spatial memory and then sacrificed for immunohistochemical analyses. Six weeks after TBI (5 weeks after transplantation), there was significant survival and neuronal differentiation of MNTS1-transduced NPCs, as well as injury-activated targeted migration towards contused brain regions. NPCs displayed long processes with spine-like formations that extended into many cortical and subcortical brain structures, including the hippocampus and contralateral hemisphere. All transplanted NPCs, irrespective of transduction profile, conferred significant preservation of pericontusional host tissues and enhanced hippocampal neurogenesis in the posttraumatic brain. Furthermore, NPC transplantation significantly improved spatial memory capacity on the hippocampal-dependent Morris water maze (MWM) cognitive task. Transplant recipients exhibited escape latencies approximately half that of injured vehicle controls, performing on par with sham uninjured animals. The second set of experiments was conducted to assess histological and functional outcomes with administration of a recently-discovered proneurogenic compound, the highly-active aminopropyl carbazole, P7C3-A20. Sprague Dawley rats were subjected to moderate fluid percussion brain injury or sham surgery. Treatment with 10 mg/kg of P7C3-A20 or vehicle was initiated intraperitoneally 30 min post surgery, and twice per day everyday thereafter for 7 days. Administration of P7C3-A20 significantly reduced overall contusion volume, preserved vulnerable NeuN-positive pericontusional cortical neurons, and improved sensorimotor function 1 week after trauma. P7C3-A20 treatment also significantly increased both 5-bromo-2'-deoxyuridine (BrdU)-positive and doublecortin (DCX)-positive cells within the subgranular zone of the ipsilateral hippocampus 1 week after TBI. Five weeks after TBI, animals treated with P7C3-A20 showed significantly increased BrdU/neuronal nuclei (NeuN) double-labeled neurons in the ipsilateral dentate gyrus and improved cognitive function in the MWM compared to TBI-vehicle animals. These results suggest that P7C3-A20 is neuroprotective and promotes endogenous reparative strategies, such as hippocampal neurogenesis, after brain trauma. The chemical scaffold represented by P7C3-A20 may provide a basis for developing new pharmacological agents for protecting patients against the early and chronic consequences of TBI. Neural progenitor cell transplantation and treatment with a highly-active proneurogenic compound both resulted in significant neuroprotection, enhanced hippocampal neurogenesis, and preservation of cognitive capacity in an experimental model of TBI. Collectively, the experiments carried out in this dissertation project suggest that exogenous interventions that target and strengthen endogenous reparative processes, such as NPC-mediated trophic support and enhanced hippocampal neurogenesis, may be effective at restoring and protecting histological and functional outcomes after traumatic brain

injury.
The Pharmacology of Neurogenesis and Neuroenhancement Apr 03 2023 Currently, few drugs are available for the effective treatment of neurodegenerative diseases and neurodevelopmental disorders. Recent advances in neuroscience research offer hope that future strategies for treating these brain disorders will include neurogenesis and neuroenhancement as therapeutic endpoints. This volume reviews cutting-edge findings related to the pharmacological aspects of neurogenesis and neuroprotection. A broad range of topics are covered from basic lab bench research to drug discovery efforts and important clinical issues. This collection of reviews is a perfect way to become acquainted with these exciting new fields in the space of a single volume. Chapters are written with a general audience in mind, but with enough high-level discussion to appeal to specialists and experts as well. The authors have done an excellent job of challenging current paradigms and pushing the boundaries of exploration in keeping with the pioneering spirit that gave rise to these emerging areas of research. Consequently, this will be an indispensable resource for many years to come. Provides state-of-the-art reviews spanning significant emerging fields Discusses future directions and questions for future studies Includes informative illustrations
Neuroprotection Mar 02 2023 Neurological disease affects nearly 25%–30% of the world's population, exerting enormous financial strain on the healthcare system. Estimated current costs are around $800 annual billion, and this number is expected to increase exponentially as the global population ages. As such, new and alternative neuroprotective strategies are urgently needed. This book examines some of the most promising approaches in neuroprotection as well as discusses current goals and prospects. Organized into three sections, chapters cover such topics as the use of cannabinoids, medicinal plants, and essential oils in Alzheimer's and Parkinson's; protein misfolding and the neuroprotective potential of vitamin E in cerebral ischemia; and potential new neurological treatments and their mechanisms of action.

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