DOWNLOAD PDF NEURODEVELOPMENTAL DISORDERS (JOURNAL OF NEURAL TRANSMISSION. SUPPLEMENTA)

Chapter 1 : Neurology Resources | NYU Health Sciences Library

Series: Journal of Neural Transmission. Supplementa, Supplement di Giovanni, Parkinson's Disease and Related Disorders. Series: Journal of Neural Transmission.

Open in a separate window Dopaminergic hypothesis Dopamine is the most extensively investigated neurotransmitter in schizophrenia. The dopaminergic hypothesis came about from the observation that drugs that antagonized dopamine were found to be effective in the treatment of schizophrenia. This theory dominated the scene for nearly fifteen years. The evidence for the role of dopamine in the pathogenesis of schizophrenia comes from the fact that there are abnormalities in genes involved in dopamine synthesis, receptors and transporters, functional neuroimaging studies SPECT and PET , and the efficacy of antidopaminergic agents in treating schizophrenia. Dopamine has also been implicated in mediating aberrations in i developmental processes such as neuronal proliferation and migration as well as pruning, and ii degenerative processes such as oxidative stress and excitotoxicity. In adolescence, the onset of psychosis may be related to an excessive elimination of synapses and secondarily, phasic dopaminergic overactivity. This hypothesis is consistent with central characteristics of schizophrenia such as premorbid manifestations, adolescent onset, functional decline early in the illness, cognitive impairments, the role of dopamine, and the role of genes and environment in pathophysiology. Such dysregulation of the tonic-phasic DA system has been proposed to account for the positive and negative symptoms of schizophrenia that emerge during adolescence. Is it the bridge? It has been postulated that glutamate could be the link between neurodevelopment and neurodegeneration. It plays a role in several stages of neurodevelopment neuronal migration, survival, and plasticity. In adolescence, it is involved in plasticity and pruning, and in old age, it is implicated in neurodegeneration through excitotoxicity. The effects of second generation antipsychotic medications are linked to their ability to modulate glutamatergic neurotransmission also. Molecules that enhance glutamatergic transmission, e. Critical analysis Why do we need to know this? If schizophrenia is a purely developmental disorder, our focus will be limited to understanding its etiology and refining preventive strategies. If a neurodegenerative element is present, then we will focus on prevention, early intervention, and treatment strategies. The label of developmental disorder also conveys a sense of therapeutic nihilism which may be detrimental. The presence of neurodevelopmental anomalies does not rule out neurodegeneration as a significant presence in schizophrenia and vice versa. Direct comparisons of the theories are not possible but the strengths and weaknesses of each theory can be examined. Neurodevelopmental hypothesis There is evidence of aberrations in the same process from various perspectives etiological, genetic, histopathological, neuroanatomical, and clinical. This hypothesis claims that schizophrenia is a disorder of brain development. Hence, by definition, the disease should be early-onset not late-onset, untreatable not treatable, and static not progressive. Neurodevelopmental hypothesisâ€”Onset of illness Arguments that go against a neurodevelopmental hypothesis of schizophrenia are many: The hypothesis is able to address these issues by using three convincing arguments: There is clear evidence that antipsychotic agents work. It has also been seen that early institution of antipsychotics leads to a less malignant course of the illness. There is also evidence to show that a sizeable proportion of patients recover completely. Neurodegenerative hypothesis This hypothesis proposes that schizophrenia is a disorder caused due to the degeneration of the brain. By definition, the disease should have characteristic histopathological features and progression. Neurodegenerative hypothesisâ€”histopathology Absence of gliosis and of any other histological evidence of degeneration such as inclusion bodies, is the strongest argument against neurodegeneration. A genetic defect in bcl-2 has been seen in schizophrenia which supports pathological apoptosis. The sensitivity of methods to detect gliosis has been questioned. Subcellular biochemical evidence of degeneration in the form of oxidative damage has been seen in schizophrenia. Neurodegenerative hypothesisâ€”progression Longitudinal neuroimaging studies have been equivocal and generally, no progressive deterioration has been seen in cognitive functions. However,

subgroups with clinical, cognitive, and neuroimaging evidence of progression are present. Thus, evidence from histopathology is mostly against neurodegeneration but clinical and biochemical evidence of degeneration is undeniably present in certain groups of patients with schizophrenia. Progressive neurodevelopmental disorder This term and concept are new and other disorders that may be classified in the same group are few. It may be said that it has been specially created to accommodate findings in the study of schizophrenia. The theoretical bases of this hypothesis are evolving and its biggest strength is that the theory emerges from research findings and not vice versa. It may be said that the disorder is unique and hence, requires a unique biological explanation. Research evidence exists for degeneration as well as development, although at present, evidence for the latter appears to be stronger. There is considerable heterogeneity in clinical findings; there may be different subgroups with different contributions of various processes towards disease manifestation. Of late, there are theoretical proposals such as the glutamatergic hypothesis that bridge the gap between development and neurodegeneration. Evidence for this proposition at present is minimal. Finally, it should be remembered that viewing schizophrenia as having both components of development and degeneration is therapeutically more optimistic. Footnotes Conflict of Interest: Clinical study of schizophrenia children. Patterns of childhood social development in adult schizophrenics. In Developmental model of schizophrenia. Comprehensive Textbook of Psychiatry. Lippincot Williams and Wilkins; On the topographical distribution of cortical lesions and anomalies in dementia praecox, with some account of their functional significance. Schizophrenia as a long term outcome of pregnancy, delivery, and perinatal complications: A 28 year follow up of the North Finland general population birth cohort. Association of schizophrenia with maternal body mass index, small size at birth and thinness during childhood. Do hypertension and diuretic treatment in pregnancy increase the risk of schizophrenia in offspring? Obstetric complications and schizophrenia: Historical and meta-analytic review. The problem of obstetrical complications and schizophrenia. Schizophrenia and complications of pregnancy and labour: An individual patient data meta-analysis. Adult schizophrenia following prenatal exposure to an influenza epidemic. Maternal influenza in the aetiology of schizophrenia. Schizophrenia following in utero exposure to the influenza epidemics in Japan. Prenatal exposure to influenza does not cause schizophrenia. Evidence against maternal influenza as a risk factor for schizophrenia. No relation between risk of schizophrenia and prenatal exposure to influenza in Holland. Schizophrenia after prenatal famine: Minor physical anomalies in schizophrenia. The anthropometric assessment of dysmorphic features in schizophrenia as an index of its developmental origins. Dysmorphic features in schizophrenia. The neurodevelopmental basis of schizophrenia. Is reduced dermatoglyphic a-b ridge count a reliable marker of developmental impairment in schizophrenia? Murray MR, Bramon E. Developmental model of schizophrenia. A review of MRI findings in schizophrenia. Altered distribution of nicotamine-adenine dinucleotide phosphate-diaphorase cells in frontal lobe of schizophrenics implies disturbances of cortical development. Increased density of microtubule associated protein 2 â€” immunoreactive neurons in the prefrontal white matter of schizophrenic subjects. Jacob H, Beckmann H. Prenatal developmental disturbances in the limbic allocortex of schizophrenics. Variability in the human entorhinal region may confound neuropsychiatric diagnosis. Acta Anat Basel ; Akil M, Lewis DA. Cytoarchitechture of the entorhinal cortex in schizophrenia. Morphometric studies of the entorhinal cortex in neuropsychiatric patients and controls: A review of disrupted in schizophrenia -1 disc1: Neurodevelopment, cognition and mental conditions. A decrease of reelin expression as a putative vulnerability factor in schizophrenia. Characterization of human cleaved N-CAM and association with schizophrenia. Decreased expression of the embryonic form of the neural cell-adhesion molecule in schizophrenic brains. Proc Natl Acad Sci U. Caused by a fault in programmed synaptic elimination during adolescence? Alterations in brain high-energy phosphate and membrane phospholipid metabolism in first-episode, drug-naive schizophrenics. Jernigan TL, Tallal P. Late childhood changes in brain morphology observable with MRI. Dev Med and Child Neurol. Positron-emission tomography study of human brain functional development. Delta sleep deficits in schizophrenia:

Chapter 2 : Journal of Pediatric Neurological Disorders- Open Access Journals

Supplementa) by W. Wolfgang Fleischhacker, David J. Brooks (ISBN:) from Amazon's Book Store. Everyday low prices and free delivery on eligible orders. Neurodevelopmental Disorders (Journal of Neural Transmission.

This article has been cited by other articles in PMC. Abstract Neurodevelopmental disorders include a wide range of diseases such as autism spectrum disorders and mental retardation. Mutations in several genes that regulate neural development and synapse function have been identified in neurodevelopmental disorders. Interestingly, some affected genes and pathways in these diseases are associated with the autophagy pathway. Autophagy is a complex, bulky degradative process that involves the sequestration of cellular proteins, RNA, lipids, and cellular organelles into lysosomes. Despite recent progress in elucidating the genetics and molecular pathogenesis of these disorders, little is known about the pathogenic mechanisms and autophagy-related pathways involved in common neurodevelopmental disorders. Therefore, in this review, we focus on the current understanding of neuronal autophagy as well as recent findings on genetics and the roles of autophagy pathway in common neurodevelopmental disorders. It is considered to be important for cellular homeostasis, especially under nutrient-deficient or stress conditions, by degrading cytosolic materials in order to either supply the components required for alternate energy metabolism pathways or remove toxic components for cell survival. However, a growing body of evidence has suggested that autophagy is constitutively activated during normal nutrient conditions in a cell-type specific manner. Autophagy has been implicated in various cellular processes such as protein and organelle quality control, development and differentiation, ageing, and immunity. Therefore, alteration of autophagy is associated with several cellular pathologies and diseases, including tumor formation, infectious diseases, liver diseases, myopathy, diabetes, and several neurodegenerative diseases [1 , 2]. Autophagy can be generally classified as microautophagy, chaperone-mediated CMA , or macroautophagy [2 - 4]. Microautophagy delivers the cytoplasmic contents by invagination of the lysosomal membrane into its lumen. Macroautophagy referred to as autophagy is the well-characterized form of autophagy that involves the sequestration of cytosolic components into lysosomes in a non-selective manner. Although autophagy is mostly a non-specific degradative process, there are some selective forms of autophagy in terms of cargo selectivity e. Although these several forms of autophagy are important for physiology and pathology, in this review, we discuss the general form of autophagy, macroautophagy, in neurodevelopment and neurodevelopmental disorders. In this section, the current understanding of the roles of molecular components and autophagy signaling in neurodevelopment are described. A thorough understanding of the molecular mechanism by which autophagy is regulated in neurodevelopment might provide potential targets for the novel therapeutic intervention of common neurodevelopmental disorders associated with autophagy. Autophagy machinery and signaling pathway Understanding of the molecular pathway of autophagy has been achieved by identifying several autophagy genes ATG from yeast to mammals. Autophagy requires several essential steps for lysosomal degradation: Several ATG are involved in each step of autophagy, and mutations in these genes have been found in several human diseases [2 - 4]. Consequently, inactivation of mTORC1 by nutrient deprivation or rapamycin treatment could activate the autophagy pathway. Once autophagy is induced, activated ULK1 phosphorylates AMBRA1, leading to translocation of the PIK3C3 complex from the microtubule network to the endoplasmic reticulum ER , which is considered as the most important source of autophagosome formation. Beclin1 regulates PIK3C3 kinase activity through interactions with multiple modulators. Elongation The elongation step for efficient expansion of the phagophore requires two ubiquitin-like conjugation systems: Despite the extensive involvement of these two ubiquitin-like conjugation systems in autophagy, the presence of ATG5-, ATG7-, and LC3-independent autophagy pathways has been reported [9].

DOWNLOAD PDF NEURODEVELOPMENTAL DISORDERS (JOURNAL OF NEURAL TRANSMISSION. SUPPLEMENTA)

Chapter 3 : Neurodevelopmental disorders (Book,) [calendrierdelascience.com]

The Journal of Neural Transmission aims to establish an interface between basic sciences and clinical neurology and psychiatry. It intends to put a special emphasis on translational publications of the newest developments in the field from all disciplines of the neural sciences that relate to a better understanding and treatment of neurological.

Submit manuscript at https: This enables learners and academicians to gain access to instant and quick reference for the development of research in the field of Pediatric Neurology. The peer-review process is designed to ensure the consistency of the submitted manuscripts in line with the set guidelines for a standard open access case report or research paper. The Journal of Pediatric Neurological Disorders has been acquired by OMICS International for dissemination of knowledge in the field of Neurology with provision of free sharing and transmission of articles under the norms of the Bethesda Statement. Epilepsy and Seizures Seizures, abnormal movements or behavior due to unusual electrical activity in the brain, are a symptom of epilepsy. Epilepsy is defined as a brain disorder characterized by an enduring predisposition to generate epileptic seizures and by the neurobiologic, cognitive, psychological, and social consequences of this condition. Genetics play a part in many types of epilepsy. The concept of genetic epilepsy is that the epilepsy is, as best as understood, the direct result of a known or presumed genetic defect s in which seizures are the core symptom. Related Journals for Genetic Epilepsies: Journal of Neurophysiology, Progress in Neurobiology, Current Opinion in Neurobiology Headaches and Migraines Migraines are painful headaches often accompanied by nausea, vomiting, and sensitivity to light. Migraines and other types of headaches, such as tension headache and sinus headache, are painful. Migraine symptoms are treated with anti-nausea drugs and abortive or preventive medications. Headache remedies include pain relievers. Related Journals for Headaches and Migraines: A metabolic disorder occurs when the metabolism process fails and causes the body to have either too much or too little of the essential substances needed to stay healthyRelated journals for Genetic and Metabolic Dissorders: Brazilian Journal of Medical and Biological Research: Diagnosis of perinatal stroke requires a careful assessment of both clinical factors and brain imaging by a specialist familiar with perinatal stroke. Some babies have symptoms in immediate time period hours to days after birth. These are most often seizures though other neurological concerns may be present. This in turn leads to imaging being performed that reveals the stroke Related Journals for Stroke and Perinatal Injuries: A narrower use of the term refers to a disorder of brain function that affects emotion, learning ability, self-control and memory and that unfolds as the individual grows. Related Journals for Neurodevelopmental Disorders: Congenital brain defects are abnormalities in the brain that are present at birth. The defects typically affect the bone and soft tissue in the head and spine. There are many different types of these malformations. They can vary greatly from mild to severe conditions. A congenital brain defect usually occurs due to an interruption in the normal growth of the nervous system. The brain begins to form in the first month after conception. Development of the brain begins from a small, special plate of cells on the surface of the embryo. These cells grow and form the different regions of the brain. When this process is disturbed, it can cause structural defects in the brain and skull. Related Journals for Congenital Brain Defects: Abnormal fluency or speed of movement called dyskinesia may involve excessive or involuntary movement hyperkinesia or slowed or absent voluntary movement hypokinesia. Common dystonias include spasmodic torticollis, which affects muscles of the head, face, and neck, and blepharospasm, which causes involuntary closing of the eyelids. Related Journals for Movement Disorders: The peripheral nervous system includes muscles, the nerve-muscle neuromuscular junction, peripheral nerves in the limbs, and the motor-nerve cells in the spinal cord. Patients with neuromuscular diseases can have weakness, loss of muscle bulk, muscle twitching, cramping, numbness, tingling, and a host of other symptoms. Problems with the nerve-muscle junction can also cause droopy eyelids, double vision, and weakness that worsen with activity. Some neuromuscular disorders can also cause difficulty with swallowing and sometimes with breathing. Related Journals for Neuromuscular Disease:

DOWNLOAD PDF NEURODEVELOPMENTAL DISORDERS (JOURNAL OF NEURAL TRANSMISSION. SUPPLEMENTA)

Pavlova Pediatric Brain Tumour A brain tumor is a collection or mass of abnormal cells in the brain. The skull is very rigid and the brain is enclosed, so any growth inside such a restricted space can cause problems. Brain tumors can be cancerous malignant or non-cancerous benign. When benign or malignant tumors grow they can cause the pressure inside the skull to increase. This can cause brain damage and even death. Brain tumors are categorized as primary or secondary. Primary brain tumors originate in the brain. According to the University of Maryland Medical Center, about half of primary brain tumors are benign. Secondary brain tumors occur when cancer cells spread to the brain from another organ such as the lung or breast. Related Journals for Pediatric Brain Tumor: Journal of Vestibular Research: Equilibrium and Orientation, Acta Neurobiologiae Experimentalis, Autism Research, Neural Processing Letters, Neuroinformatics Chiari malformation Chiari malformation is a condition in which brain tissue extends into your spinal canal. It occurs when part of your skull is abnormally small or misshapen, pressing on your brain and forcing it downward. Chiari malformation is uncommon, but improved imaging tests have led to more frequent diagnoses. Chiari malformation type I develops as the skull and brain are growing. As a result, signs and symptoms may not occur until late childhood or adulthood. The most common pediatric form, called Chiari malformation type II, is present at birth congenital. Treatment of Chiari malformation depends on the form, severity and associated symptoms. Regular monitoring, medications and surgery are treatment options. In some cases, no treatment is needed. HIV does not appear to directly invade nerve cells but it jeopardizes their health and function, causing symptoms such as confusion, forgetfulness, behavioral changes, severe headaches, progressive weakness, loss of sensation in the arms and legs, stroke, cognitive motor impairment, or damage to the peripheral nerves. Other complications that can occur as a result of HIV infection or the drugs used to treat it include pain, seizures, shingles, spinal cord problems, lack of coordination, difficult or painful swallowing, anxiety disorder, depression, fever, vision loss, gait disorders, destruction of brain tissue, and coma. Even relatively common disorders may go undiagnosed and untreated by clinicians who are not familiar with the range of "atypical" cognitive or behavioral symptoms possible in an affected child. Recent research in genetics and brain development has altered the phenotypic description of various disorders, but this new knowledge is not readily available to practitioners. This collection provides a single resource that will help clinicians, pediatricians, neuropsychologists, educators, and others use the latest research to identify and treat a variety of genetic disorders as early as possible. Related Journals for Neurogenetic Disorders: Not only do pediatric sleep problems affect child health, but they can impact family dynamics and parental or sibling sleep. Children may suffer from problems falling or staying asleep; physiological problems such as obstructive sleep apnea abnormal or disruptive behaviors during sleep such as sleepwalking or other parasomnias symptoms that occur near sleep onset such as restless legs syndrome, and daytime symptoms such as excessive sleepiness, cataplexy and others. While adults may suffer from the same problems, the etiology, presentation, and associated findings in children may be very different than those seen in adults. In addition, developmental aspects of childhood play an important role in pediatric sleep, such as in the cases of early childhood insomnias and adolescent delayed sleep phase syndrome. Related Journals for Pediatric Sleep Disorders: Activitas Nervosa Superior Rediviva, Clinical and Experimental Neuroimmunology, Journal of Nanoneuroscience, Journal of Neurosciences in Rural Practice, Journal of Pediatric Neurosciences Neonatal encephalopathy Neonatal encephalopathy is a clinically defined syndrome of disturbed neurologic function in the earliest days of life in an infant born at or beyond 35 weeks of gestation, manifested by a subnormal level of consciousness or seizures, and often accompanied by difficulty with initiating and maintaining respiration and depression of tone and reflexes. This expanded clinical definition must be put into use based on measures that can be reliably and accurately implemented by trained staff. The first mandatory step in an assessment of neonatal encephalopathy is to confirm whether a specific infant meets the case definition. Related Journals for Neonatal encephalopathy:

Chapter 4 : PsycINFO Neuroscience Coverage

DOWNLOAD PDF NEURODEVELOPMENTAL DISORDERS (JOURNAL OF NEURAL TRANSMISSION. SUPPLEMENTA)

Journal of Neural Transmission Translational Neuroscience, Neurology and Preclinical Neurological Studies, Psychiatry and Preclinical Psychiatric Studies.

Chapter 5 : Neurodevelopmental disorders (eBook,) [calendrierdelascience.com]

Journal of Neural Transmission. Supplementa. Hypokinetic disorders, such as Parkinson's disease, are hypothesized to result from a complex series of changes in the activity of striatal.

Chapter 6 : Neuronal Autophagy and Neurodevelopmental Disorders

Current Topics in Extrapyramidal Disorders (Journal of Neural Transmission. Supplementa) (Volume 16) Softcover reprint of the original 1st ed. Edition.