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Research Article **Published Date:-2019-11-11 00:00:00**

Immunohistochemical expression of Nestin as Cancer Stem Cell Marker in gliomas

Background: Gliomas represent the most frequent primary tumors of central nervous system (CNS), contributing to more than half of the incidence of brain tumors. Cancer stem cell markers (CSC) identify a group of patients at high risk for progression. Nestin is an intermediate filament (IF) protein was first described as a neural stem cell/progenitor cell marker. Nestin-positive neuroepithelial stem cells are detected in the subventricular zone of the human adult brain and they remain mitotically active throughout adulthood. The expression of Nestin in gliomas has been suggested to be related to dedifferentiation, improved cell motility, invasive potential and increased malignancy. This study aims to investigate Nestin immunohistochemical expression in different types of glioma and its correlation with different clinicopathological parameters.

Materials and Methods: Nestin immunostaining was studied in 60 specimens of glioma using avidin-biotin peroxidase method.

Results: Nestin was strongly expressed in 11/60 (18.33%), moderately expressed in 29/60 (48.33%) and weekly expressed in 15/60 (25%) of studied gliomas. A significant positive correlation was found between Nestin expression and histologic type ($p < 0.001$) and increasing grade of gliomas ($p < 0.001$).

Conclusion: Increased Nestin expression is correlated with tumor progression, increasing grade and poor prognostic parameter of glioma. Nestin is a useful marker for detection of CSC in high-grade glioma which is responsible for resistance to chemo-radiotherapy and may serve as a predictor for patient outcomes.

Review Article **Published Date:-2019-11-04 00:00:00**

Protection from the Pathogenesis of Neurodegenerative Disorders, including Alzheimer's Disease, Amyotrophic Lateral Sclerosis, Huntington's Disease, and Parkinson's Diseases, through the Mitigation of Reactive Oxygen Species

The biological changes caused by oxidative stress (OS) are known to be involved in the etiology of neurodegenerative disorders, including Alzheimer's disease, amyotrophic lateral sclerosis, Huntington's disease, and Parkinson's disease. The brain is particularly vulnerable to OS due to its high lipid content and extensive consumption of oxygen. OS processes, particularly the excessive production of reactive oxygen species (ROS), play a critical role in how neurodegenerative disorders develop. This is evidenced by in vivo studies investigating various biomolecules related to OS, such as products of lipid and DNA oxidation. Accordingly, ROS can also cause oxidative-related damage in neurodegenerative disorders, including dopamine auto-oxidation, mitochondrial dysfunction, glial cell activation, ?-synuclein aggregation, excessive free iron, and changes in calcium signaling. Furthermore, excessive levels of cellular oxidants reduce antioxidant defenses, which in turn propagate the cycle of OS. As such, it is increasingly important to determine the linkage between a high intake of antioxidants through dietary interventions and a lower risk of developing neurodegenerative diseases. Indeed, in addition to modulating the immune system, optimal nutritional status is capable of changing various processes of neuroinflammation known to be involved in the pathogenesis of neurodegeneration. Accordingly, a better understanding of the role ROS plays in the etiology of neurodegeneration is needed, along with the identification of dietary interventions that may lead to improved therapeutic strategies for both the treatment and prevention of neurodegenerative disorders. Therefore, this review presents a comprehensive summary of the role of ROS in the pathogenesis of neurodegenerative disorders. In addition, nutrients believed to be useful for mitigating and counteracting ROS are discussed.

Research Article **Published Date:-2019-10-01 00:00:00**

Tamsulosin and Dementia in old age: Is there any relationship?

Tamsulosin is used to treat Benign Prostatic Hyperplasia (BPH), prescribed annually to about 12.6 million patients worldwide. It is an alpha-adrenergic antagonist that reduces the tone of the prostate smooth muscle involved in the pathophysiology of BPH. By acting on alpha 1A receptors, predominant in the prostate, tamsulosin also acts on receptors present in the brain. This study consisted of a literature review aimed at disseminating scientific knowledge about the relationship between the use of tamsulosin and the onset of dementia. PubMed, Scopus, Scielo, Embase, and Web of Science studies involving dementia in patients using tamsulosin in the last five years were selected. The review showed a risk correlation and a higher incidence of dementia in treated patients. The risk ratio, when compared to other medicines, approached 1.20. In conclusion, it was identified the need for clinical trials with higher sampling power to increase relational significance due to the high prevalence of BPH and the extensive use of tamsulosin in elderly patients with the disease.

Research Article **Published Date:-2019-08-01 00:00:00**

Carbonic Anhydrase I modifies SOD1-induced motor neuron toxicity in Drosophila via ER stress pathway

Background: Drosophila models of amyotrophic lateral sclerosis (ALS) have been widely used in understanding molecular mechanisms of ALS pathogenesis as well as discovering potential targets for therapeutic drugs. Mutations in the copper/zinc superoxide dismutase (SOD1) cause ALS by gain of toxic functions and induce toxicity in fly motor neurons.

Results: In this study, we have determined that human carbonic anhydrase I (CA1) can alleviate mutant SOD1-induced motor neuron toxicity in the transgenic fly model of ALS. Interestingly, we found that motor neuron expression of CA1 could independently induce locomotion defect as well as decreasing the survival rate. In addition, CA1-induced toxicity in motor neurons is anhydrase activity-dependent. Mechanistically, we identified that both SOD1- and CA1-induced toxicity involve the activation of eIF2? in the ER stress response pathway. Downstream activation of the JNK pathway has also been implicated in the induced toxicity.

Conclusion: Our results have confirmed that SOD1-induced toxicity in fly motor neuron also involves endoplasmic reticulum (ER) stress pathway. More importantly, we have discovered a new cellular role that CA1 plays by antagonizing mutant SOD1-induced toxicity in motor neurons involving the ER stress pathway. Such information can be potentially useful for further understanding disease mechanisms and developing therapeutic targets for ALS.

Research Article **Published Date:-2019-07-31 00:00:00**

The turing machine theory for some spinal cord and brain condition, A toxicological - antidotic depurative approach

Aim of this work is to produce a general theory related an new depurative strategy to be devalued for reduce or delay some spinal cord and brain degenerative and inflammatory chronic disease or acute traumatic condition. It is used and informatics approach in order to set correct the problem and the process. Scope of this project is to submit to the researcher a new therapeutic strategy (under a depurative- toxicological-pharmacological) in this complex kind of disease. A Turing machine theory say us a method to TRASLATE the need of a strategy in a practical hypotesys of work. A global conceptual map can help in this field.

Research Article **Published Date:-2019-07-24 00:00:00**

Comparative study of carboxylate and amide forms of HLDF-6 peptide: Neuroprotective and nootropic effects in animal models of ischemic stroke

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Aim:The work was to perform a comparative study of the neuroprotective and nootropic activities of two pharmaceutical substances, the HLDF-6 peptide and its amide form (HLDF-6-NH2).

Materials and Methods: We used in the study healthy adult male Wistar rats aged 180–200 days weighing 280–300 g. We modelled ischemic stroke in rats by chronical occlusion of carotid arteries. Solutions of the HLDF-6-NH2 and HLDF-6 peptides were administered intranasally. Cognitive functions we assessed with Novel object recognition test and Morris maze.

Results: The amide form of HLDF-6 peptide is more efficient: the neuroprotective activity of HLDF-6-NH2, evaluated by improvement of cognitive functions in animals, surpassed that of the native HLDF-6 peptide. A dose of 250 μg/kg of HLDF-6-NH2 peptide resulted in practically complete restoration of the disturbed functions. In the model of ischemic stroke, the amide form of the peptide significantly excelled the reference substance mexidol both in the effective dose and biological activity.

Conclusion: The results of study of the agent allow hoping for its success in further clinical investigation. In view of high demand for the agent and in case of successful clinical trials, it will surely become widely used in clinical practice in treatment of IS.

Case Report **Published Date:-2019-07-09 00:00:00**

Mimicking multiple sclerosis - Ghost tumor that comes and goes in different parts of the brain without any treatment

Lesions that spontaneously come and go in central nervous system without any treatment at different time points and at different locations (CNS) usually lead ones to think of the possibilities of multiple sclerosis. However, sometimes there are exceptions. Surgical biopsy remains an important tool for definitive diagnosis in difficult cases. We report a case of intracranial diffuse large B cell lymphoma that spontaneously disappeared without any treatment and then reappeared at different time points and different locations.
