ABSTRACT BOOK

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6

Integrative Genomic Analysis of Schizophrenia in the Basal Ganglia, and the Frontal lobe

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Abstract

Schizophrenia is a mental disorder which results in disordered thinking, hallucinations and behavior that impair daily functions which affects multiple brain regions such as basal ganglia, frontal lobe and many more. Earlier studies have shown that COMT is linked to schizophrenia, COMT when deleted results in a complex syndrome, the psychiatric manifestations of which include schizophrenia and other psychoses and so I decided to take COMT as a reference gene for doing genomic analysis. The purpose of this study was to identify possible candidate genes for Schizophrenia by doing gene search of COMT correlates in Basal ganglia and Frontal lobe and do comparative analysis. Gene expression data of COMT gene correlates in the Basal Ganglia, and the Frontal lobe was obtained from Allen Brain Atlas. A bioinformatics approach was used to analyze gene expression profiles in order to identify candidate genes that have an effect on Schzophrenia. Bioinformatics tools used were DAVID, STRING database, and Gene Ontology knowledgebase. Total 16 genes found linked to schizophrenia were CBS, DDR1, GSTPI, GSTTI.GSTT2. HOMER3. HLA-A, MAP4, PHGDH, PLXNB1, PSEN1, SMPD1, SREBFI, TSPO, SOX10. More genes linked to schizophrenia were found in the Basal ganglia as compared to the Frontal lobe. Some genes linked to schizophrenia were found directly or indirectly interacting with each other whereas some were not found interacting at all. These gene interaction study can help us to further on finding the other gene linked to them and may also results in some possible early detection method of schizophrenia.

Keywords

SCHIZOPHRENIA, Alzheimer, COMT, BASAL GANGLIA, FRONTAL LOBE

Theme(s) of the abstract

Computational Neuroscience

Category of Submission

Original Research

7

Managing Tourette Syndrome using Artificial Intelligence

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Abstract

Background: Tourette Syndrome is a common neurological disorder in the Indian Subcontinent which accounts for more than 1 million cases per annum. Tics are distinctive features of this syndrome which may be motor or vocal. Clinical manifestations of simple motor tics include frequent blinking, darting of eyes, shrugging of shoulders. With no cure for this syndrome, behavioral adaptations are known to prove better in its management.

Purpose: This study aims at developing an AI-powered model which could detect the frequency of eye blinking, thereby making the patient aware of the severity of his/her condition. This could help the patient in managing the Tourette Syndrome better.

Methods: A dedicated Android application has to be installed in the smartphone of the patient. This application would be running in the background. Whenever the patient uses his mobile, the front camera of the smartphone records his face. The patient can customize the period during which the camera records, thereby ensuring privacy. The AI model then processes these images to classify them into normal blinking or characteristic motor tics using deep learning techniques. The application displays the frequency of occurrence of motor tics, if any. The patient would also be given a provision to compare the frequency of tics and the work he/she does on mobile.

Results: The app has been able to distinguish the tics accurately from normal blinking. This also provides the frequency of occurrence of tics along with timestamp. This could enable the patient to know better about his severity and take necessary corrective measures to effectively manage the syndrome. Also, by correlating the frequency of tics with activities like reading and gaming, the patients could get an idea of the activities which could possibly aggravate their condition. This could let the patients refrain from such activities.

Conclusion: The Tourette Syndrome doesn’t bother much initially. However, these may be progress with age, particularly when young children turn into teens. This could have a negative impact on their social and personal life making them feel depressed. This model would help the patients manage the manifestations of the syndrome. Usage of such applications should be encouraged. More research needs to be undertaken in this domain, so that we could improve the lifestyle of patients affected with Tourette Syndrome.

Keywords

Artificial Intelligence (AI), Neurological Disorder, Tics, Tourette Syndrome

Theme(s) of the abstract

Translational and Clinical Neuroscience, Cognitive and Behavioral Neuroscience, Psychology, Other

Category of Submission

Original Research

15

Sequential activation of Notch and Grainyhead gives apoptotic competence to Abdominal-B expressing larval neuroblasts in Drosophila Central nervous system

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Abstract

Neural circuitry for mating and reproduction resides within the terminal segments of central nervous system (CNS) which express Hox paralogous group 9–13 (in vertebrates) or Abdominal-B (Abd-B) in *Drosophila*. Terminal neuroblasts (NBs) in A8-A10 segments of *Drosophila* larval CNS are subdivided into two groups based on expression of transcription factor Doublesex (Dsx). While the sex specific fate of Dsx-positive NBs is well investigated, the fate of Dsx-negative NBs is not known so far. Our studies with Dsx-negative NBs suggests that these cells, like their abdominal counterparts (in A3-A7 segments) use Hox, Grainyhead (Grh) and Notch to undergo cell death during larval development. This cell death also happens by transcriptionally activating *RHG* family of apoptotic genes through a common apoptotic enhancer in early to mid L3 stages. However, unlike abdominal NBs (in A3-A7 segments) which use increasing levels of resident Hox factor Abdominal-A (Abd-A) as an apoptosis trigger, Dsx-negative NBs (in A8-A10 segments) keep the levels of resident Hox factor Abd-B constant. These cells instead utilize increasing levels of the temporal transcription factor Grh and a rise in Notch activity to gain apoptotic competence. Biochemical and *in vivo* analysis suggest that Abdominal-A and Grh binding motifs in the common apoptotic enhancer also function as Abdominal-B and Grh binding motifs and maintains the enhancer activity in A8-A10 NBs. Finally, the deletion of this enhancer by the CRISPR-Cas9 method blocks the apoptosis of Dsx-negative NBs. These results highlight the fact that Hox dependent NB apoptosis in abdominal and terminal regions utilizes common molecular players (Hox, Grh and Notch), but seems to have evolved different molecular strategies to pattern CNS.

Keywords

Apoptosis, Neural Stem Cells, Enhancer, Notch signalling, Hox genes

Theme(s) of the abstract

Developmental Neuroscience, Cellular and Molecular Neuroscience

Category of Submission

Original Research

17

Eliciting Experiential Knowledge through Neuro Signature profiling

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Abstract

Emotional experiences leave vivid memories that last in an individual for life time. The emotions facilitates the memory has been attributed or trigger the brain regions and those signals are long lasting impact. In memory systems, ‘remembering’ is more attributed to ‘experiential knowledge’ (EK), while ‘knowing’ is related to mere recognition. Brain signatures specific to remembering as distinct from knowing will have enormous applied value including its forensic use. BEOS is based on EEG and its principle is neuro-psychology. Neuro-psychological criteria of BEOS system contains Experiential Knowledge (EK), Primary processing (PP), Encoding (EN), Emotional Response (ER), Activation Suppression (AS), Inattention (IN) ; etc. “32” electrodes EEG cap to be attached to a suspect and REM electrodes for rotatory eyes moment to be attached. An EEG gel to be fill for better conductivity. The aim of the study to elicit experiential knowledge in boys and girls through their positive and negative experiences. The results showed that girls have high impact of positive experiences than negative experiences and the boys have high impact negative experiences. Sample were consisted of 17, girls were 10 and boys were 7. EEG was recorded while each participant had a series of auditory presentations of short verbal statements related to the previous ‘activity session’.

Keywords

EEG, Remembrance, Knowing, EK, Encoding++, Encoding, Activation Suppression, Inattention.

Theme(s) of the abstract

Cognitive and Behavioral Neuroscience, Social Neuroscience

Category of Submission

Original Research

18

Aberrant expression and toxic gain of function of netrin1 in Amyotrophic Lateral Sclerosis(ALS) astrocytes contribute to motor neurodegeneration

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Abstract

Mutations in the ubiquitously expressed superoxide dismutase-1 (SOD1) gene cause ~20% of inherited amyotrophic lateral sclerosis (ALS). The underlying molecular mechanisms in mutant SOD1 linked ALS involves a combination of direct motor neuron and glia-induced toxicity. Although, the contribution of astrocytes towards motor neuron death in ALS has been well established using a variety of in vitro and in vivo approaches, the identity of the mediators of this toxicity is not well understood. We previously have shown in the SOD1-G93A mouse model of ALS (ALS mice) that caspase-3 cleaves the astroglial glutamate transporter GLT-1 (EAAT2), releasing a sumoylated C-terminal fragment (CTE-SUMO1), which accumulates in the nucleus of astrocytes over disease. Astrocytes harboring CTE-SUMO1 initiate extrinsic, non-cell-autonomous motor neuron toxicity. Microarray analysis of these CTE-SUMO1 astrocytes revealed a higher expression of netrin-1. While in the normal adult CNS only oligodendrocytes and neurons express and release netrin-1 to regulate axon homeostasis and synaptic maintenance, spinal cord astrocytes have not been shown to express netrin-1. We provide here the first evidence of netrin-1 expression in the astrocytes of the symptomatic SOD1-G93A mice. Interestingly, netrin-1 is also elevated in neurons localized to the ventral horn of the presymptomatic and disease onset stages of the SOD1-G93A mice suggesting a neuron-astrocyte cross-talk during disease progression. Expression of Netrin-1 was also detected in astrocytes in spinal cord post-mortem tissue and iPS cells-derived astrocytes of sporadic ALS patients. Super ecliptic pHluorin tagged netrin1 expression in ALS astrocytes showed a higher frequency of exocytosis events in comparison to control astrocytes signifying increased secretion. Intrathecal injection of astrocyte targeted netrin-1 expressing AAV5 in mice decreased the compound muscle action potential (CMAP) signifying motor dysfunction. Neutralization of netrin-1 in human iPSC derived astrocyte-conditioned media (ACM) using anti-netrin-1 antibodies significantly rescued the motor neuron directed toxicity. Acute stimulation of mature primary motor neurons with netrin-1 (25 nM) elicits a robust Ca2+ influx response. Microelectrode analysis of motor neurons treated with 25 nM netrin-1 showed changes in the frequency of firing both in the short term and long term. Aberrant production and release of netrin-1 by astrocytes may, therefore, be one of the pathogenic, non-cell-autonomous mechanisms of motor neuron toxicity occurring over ALS progression and, therefore, be a potential novel molecular target for intervention.

Keywords

non-cell autonomous toxicity, netrin1, amyotrophic lateral sclerosis, induced pluripotent stem cell astrocytes, microelectrode analysis

Theme(s) of the abstract

Cellular and Molecular Neuroscience, Neurogenetics, Translational and Clinical Neuroscience

Category of Submission

Original Research

20

Dementia's Protein-Protein Interaction Network's Stability lies in Motif-localized Hubs where Heat Shock Proteins/Genes and EGFR gene act as Centers of High Influence

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Abstract

Objective: To find high-impact core regulating processes and factors involved in dementia’s protein-protein interaction network and explore various aspects related to its stability and signal propagation.

Methodology: Using disease query for dementia, high-confidence experimentally verified data from STRING – a principal dementia network (PDN) was constructed in GeneMANIA plugin of CytoScape which was analyzed for topology (probability of degree distribution, clustering coefficient, neighborhood connectivity distribution; betweenness, closeness and eigenvector centrality) using NetworkAnalyzer tool, gene ontology (GO) categories using Database for Annotation, Visualization and Integrated Discovery (DAVID) and presence of communities (using Louvain method of modularity maximization). High degree hubs from PDN were traced through these communities present at different level of hierarchy up to the level of motifs to find key-regulators. Motif-localized hubs present at every level were knocked out from PDN (one level at a time) to assess their contribution towards network's resilience, and were made to interact with filtered-for-noise (using MCODE algorithm) drug-associated genes (obtained from GWAS, Medic and OMIM databases) at high-confidence score setting to identify busy hubs among these interactions.

Results: PDN exhibited hierarchical scale-free topology with assortativity and consisted of 881 genes with 59085 interactions. It was enriched in various GO categories and KEGG pathways like 'negative and positive regulation of apoptotic processes', 'macroautophagy', 'aging', 'response to drug', 'protein binding', 'cytosol', 'neurotrophin signaling pathway' etc. A number of communities were found at different levels of hierarchy in PDN consisting of 95 motif localized hubs out of which 7 were present at deepest level and hence were key regulators (KRs) of PDN (HSP90AA1, HSP90AB1, EGFR, FYN, JUN, CELF2 and CTNNA3). Knock-out of motif-localized hubs changed network’s topology from hierarchical scale-free to scale-free with increased assortativity.

Further, as a result of druggable genome – motif-localized hubs high confidence interactions a hierarchical-scale free disassortative resultant network (RN) consisting of 109 genes and 2433 interactions was obtained where UBC, EGFR, APP, CTNNB1, NTRK1, FN1, HSP90AA1, MDM2, VCP, CTNNA1 and GRB2 were identified as busy hubs based on degree and betweenness centrality.

Conclusion: Stability and resilience of PDN highly rely on motif-localized hubs (especially those present at deeper levels) making them important therapeutic intervention candidates. HSP90AA1 involved in heat shock response (and its master regulator i.e. HSF1) and EGFR are most important genes in pathology of dementia apart from KRs given to their presence as KRs in PDN as well as hubs in RN.

Keywords

Protein-protein interactions in Dementia, Network Medicine in Dementia, Heat shock proteins in dementia, Epidermal Growth Factor Receptor in Dementia's Pathology

Theme(s) of the abstract

Neurogenetics, Systems Neuroscience, Cellular and Molecular Neuroscience

Category of Submission

Original Research

23

Antagonistic actions of CART and NPY tune the excitability of specific group of dorsal telencephalic neurons to maintain hunger-satiety bistable states.

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Abstract

Consuming food to fulfil energy requirements of the body is critical for survival of an organism. In order to establish robust control over food intake, the neural circuits regulating feeding are hardwired. One of the highly conserved elements of feeding circuits is the population of interoceptors which sense energy levels of a body and accordingly release orexigenic (E.g. NPY) or anorexigenic (E.g. CART) neuropeptides. These neuropeptidergic signals, providing information about the energy status of a body, are integrated at the downstream circuit nodes and further processed to dictate appropriate behavioural output. In mammals, energy sensing mechanisms of the brain have been extensively studied but there is a very little knowledge about integration and processing of this information which results in nutritional state dependent regulation of food intake. Our research focuses on investigation circuit level mechanisms of integration of neuropeptidergic signals which are essential for maintenance of bistable states of feeding behaviour- Hunger and Satiety. Using adult zebrafish as a model system, we have identified a specific group of dorsal telencephalic neurons which shows significant change in activity under alternative nutritional states of body as well as upon exogenous administration of orexic/anorexic neuropeptides. Interoceptory regions in zebrafish brain (Entopeduncular Nucleus and Hypothalamus) project to this region and release orexic neuropeptides (e.g.- Neuropeptide Y; NPY)/anorexic neuropeptides (e.g.- Cocaine and amphetamine regulated transcript; CART) depending upon the energy status of the body. Antagonistic actions of CART and NPY differentially modulate the activity of these neurons. CART facilitates activation of this region which leads to decrease in the feeding drive; on the other hand, NPY leads to decreased activation of this region resulting in increased feeding drive. We have further investigated mechanistic details of these pathways, which suggest that antagonistic actions of CART and NPY modulate the excitability of a group of dorsal telencephalic neurons by modulating potentiation of NMDA receptor. CART treatment leads to increased phosphorylation NMDARs by PKA-PKC activation. On the other hand, NPY leads to activation of calcineurin (a protein phosphatase) and down regulation of PKA levels which together results in decreased phosphorylation NMDA receptors. Based on these results, we propose that specific group of dorsal telencephalic neurons acts as integrators of energy-status information, possibly relaying this processed information to motor output centres to maintain hunger-satiety bistable states.

Keywords

Feeding, Neuromodulation, Bistability

Theme(s) of the abstract

Cellular and Molecular Neuroscience, Systems Neuroscience

Category of Submission

Original Research

29

Deleting Neurons : A closer look at Synaptic Pruning

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Abstract

The overproduction of neural elements, including neurons, axons, and synapses, is a tool commonly used in developmental neuroscience to reconstruct nervous systems. This generation and maturation of these neuronal synapses are accompanied by a so-called "pruning" process, marking a peak in synapse elimination (synaptic pruning) which is a final stage in the development of the human brain. Analogous to “cleaning up” of the brain, synaptic pruning eliminates extra neurons and synaptic connections to increase the efficiency of neuronal transmissions along with eliminating weaker synaptic contacts while stronger connections are kept and strengthened. Brain plasticity is a result of this neural "pruning"; neurons that are more frequently activated are preserved, while those forming weaker synaptic contacts are "trimmed away." Research in zebrafish and rat models have shown pruning occurring in about 80 percent of the synapses, barring the largest ones. These larger synapses were found to be associated with the most stable and crucial memories residing in the brain. Recent studies indicate that glial cells play a critical role in synaptic pruning, mediated by a set of signaling pathways between neurons and glia, identifying and removing unnecessary neural connections. This loss of redundant pathways may explain the arduous task of recovering from a traumatic brain injury; eliminating synaptic redundancies diminishes our ability to develop alternative pathways to bypass the damaged regions. Brain imaging and postmortem anatomical studies have pointed to insufficient or excessive synaptic pruning that may underlie several neurodevelopmental disorders, including autism, schizophrenia, and epilepsy. In this review, we explore the brain’s innate “delete” button and present current data on the mechanisms of glial-cell-dependent synaptic pruning by outlining their potential contribution to neurodevelopmental disorders.

Keywords

Neural Plasticity , Synaptic Pruning , Neuron -Glia Interactions , Neurodegeneration , Neurodevelopmental Disorders

Theme(s) of the abstract

Developmental Neuroscience, Cellular and Molecular Neuroscience, Cognitive and Behavioral Neuroscience, Translational and Clinical Neuroscience

Category of Submission

Systematic Review

30

Brain proposes, spinal cord disposes: Organization and function of cerebellospinal neurons

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Abstract

Seamless execution of a broad repertoire of motor skills, from dexterous movement of the arm to walking, is accomplished by the concerted action of diverse motor areas, including the cortex, basal ganglia, red nucleus, brainstem, cerebellum, and spinal cord. While there have been great strides toward defining the neural computations of each of these areas and their contribution to motor control, to truly understand the neural basis of behavior, it is essential to reveal how these individual motor areas are bound into coordinated networks to accomplish purposeful movement. Here we used a circuit-based approach to define and investigate the anatomical organization and function of a small population of deep cerebellar neurons that link two important motor control areas, the cerebellum and the spinal cord. First, we performed an anatomical characterization of CerebelloSpinal (CeS) neurons, identifying their distribution, organization, and neurotransmitter status. Next, we used an intersectional genetic strategy to specifically silence CeS neurons, and identified distinct roles for the contralateral and ipsilateral CeS neurons in motor control. While silencing CeS neurons did not affect basic features of locomotion, we found that silencing the contralaterally-projecting CeS neurons affected rotarod learning and that silencing the ipsi-laterally projecting CeS neurons affected skilled reaching. Finally, to understand the circuit mechanisms underlying the role of CeS neurons in skilled locomotor learning, we sought to define the specific spinal cord populations that receive inputs from this pathway. We found that the CeS neurons target the medial ventral horn of the spinal cord – the neuronal hub for interlimb coordination in the spinal cord. Within this domain, CeS neurons synapse onto long descending propriospinal neurons that link together the cervical and lumbar regions of the spinal cord, together providing a likely anatomical substrate for cerebellar control of movement. Together, we (a) show that a small sub-population of neurons in the deep cerebellar nuclei, defined by their projection to the spinal cord, is critical for normal motor control and learning, and (b) provide a link between motor control networks in the cerebellum and specific circuits for movement execution in the spinal cord, thereby establishing cerebellospinal neurons as important players in the descending control of movement.

Keywords

Cerebellum, spinal cord, circuits, motor control, neuroanatomy

Theme(s) of the abstract

Systems Neuroscience

Category of Submission

Original Research

33

Brain implants - An overview on the advancements and neuroethics. A literature review from a bio-electronics standpoint.

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Abstract

Abstract—The use of artificial devices to control the functioning of various parts in a human being has seen an upsurge in the last decade, which has led to voluminous deliberations and skepticism. This paper presents an exhaustive discussion of several experiments conducted on brain implants, their results, research gaps, possible future advancements and the neuro-ethical vantage point for continued usage of brain implants in patients.

Keywords

Brain implants, deep brain stimulation, Parkinson's disease, Alzheimer's disease, neuroethics

Theme(s) of the abstract

Developmental Neuroscience, Computational Neuroscience, Neuroethics, Systems Neuroscience

Category of Submission

Systematic Review

2

The Translocator protein (TSPO) receptor agonist XBD173 alleviates cognitive deficits in a mouse model of Alzheimer's disease

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Abstract

Accumulation of β-amyloid peptide is a characteristic pathophysiological feature of Alzheimer's (AD). Depression has been considered as a common antecedent of AD and may be an early manifestation of dementia before the cognitive decline becomes apparent. Pharmacologically targeting these overlapping cellular mechanisms would allow for a novel therapeutic strategy. The translocator protein (18 kDa) (TSPO) also known as peripheral benzodiazepine receptor controls the translocation of cholesterol from the outer to the inner mitochondrial membrane and is therefore essential in neurosteroidogenesis. XBD173 which has high selective affinity for TSPO exerts rapid anxiolytic effects and doesn’t have any tolerance or withdrawal effects. Since TSPO activation promotes the synthesis of active neurosteroids, we hypothesized that the application of XBD173 restores the Aβ-induced deficits on LTP and improves cognitive performance.

The different species of Aβ such as Aβ1-42, Aβ1-40, 3NTyr10-Aß, and AßpE3 antagonize LTP induction and exert potent synaptotoxicity. Application of XBD173 restores the Aß-induced deficits on LTP. Similarly incubation of hippocampal slices with Aβ1-42 results in a significant decrease in the spine density which is bettered by the application of the TSPO ligand XBD173. Our current findings from the water cross maze experiments suggest that chronic application of XBD173 alleviates the cognitive response in Alzheimer's modeled mice while the acute treatment with XBD173 is not sufficient to improve cognitive performance. The difference in the performance of the tasks is based on the individual perceiving of the tasks. One of the interesting aspects from the amyloid plaques staining suggests that there is a decrease in the plaque load in the XBD treated animals which hints towards either a clearance mechanism of the plaques or a neuroprotection mechanism which prevents the formation of plaques at the first place.

Keywords

Alzheimer's, Long term Potentiation, Translocator protein, Learning and Memory

Theme(s) of the abstract

Cognitive and Behavioral Neuroscience, Translational and Clinical Neuroscience, Systems Neuroscience

Category of Submission

Original Research

3

Differential Identification Of Biomarkers Between Normal Neural Stem Cells And Glioblastoma Stem Cells Using RNA Seq Analysis

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Abstract

Glioblastoma multiforme (GBM) is the most malignant and heterogenous primary brain tumor with an overall survival of only 15 months after therapy. There are two main hypothesis that suggest the formation of GBM tumor in the brain- one suggests that it arises from dysregulated neural stem cells (NSCs) that transform to form the tumor, and the other theory suggests that cancer or glioma stem cells (GSCs) initiate and cause progression of GBM. To understand the gene expression differences between these two types of stem cells, RNA Seq analysis was carried out on 20 RNA sequence samples of NSCs and GSCs selected from the SRA study SRP200400 on NCBI database. Using the T-Bioinfo® platform, gene expression data in FPKM (Fragments Per Kilobase of transcript per Million mapped reads) units was obtained which was normalized and log scaled and further used to perform principal component analysis (PCA), differential expression analysis (DGE), hierarchical clustering and gene ontology (GO) studies on DAVID. DGE analysis showed that there were 192 significantly differentially expressed genes in GSCs (padj. value <0.05, $\log_2$fold change >3). and 156 significantly differentially expressed genes in NSCs (padj. value <0.05, $\log_2$fold change <-3). Many novel cufflink IDs were also obtained. Hierarchical clustering showed the grouping of NSC and GSC samples in two separate clusters. An outlier of GSC sample was found in the NSC cluster (SRR9200898_PE). On removing the outlier while performing PCA, a PC1 of 80.03% and PC2 of 2.13% was obtained. GO studies showed involvement of both gene sets in tumorigenesis pathways. In all, a significant difference between gene expression was seen in the two types of stem cell samples suggesting a substantial genetic shift between normal NSCs and GSCs and these could be potential biomarkers to assess the prognosis of GBM tumors and be essential targets for its precision therapy.

Keywords

Glioblastoma Multiforme, RNA Seq Analysis, Machine Learning, Cancer Stem Cells, Biomarkers

Theme(s) of the abstract

Translational and Clinical Neuroscience, Cellular and Molecular Neuroscience, Neurogenetics

Category of Submission

Original Research

4

Effect of Ultra-Small Chitosan Nanoparticles Doped with Brimonidine on the Ultra-Structure of the Trabecular Meshwork of Glaucoma Patients

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Abstract

Glaucoma is recognised as a multifactorial progressive neurodegenerative disorder characterised by the acquired death of retinal ganglionic cells and loss of their axons as well as optic nerve atrophy due to consistent increase in intraocular pressure caused by the accumulation of aqueous humour due to obstruction of the Schlemm's canal which is guarded by trabecular meshwork tissue. Brimonidine, an anti-glaucoma medicine, acts as an adrenergic agonist which decreases the synthesis of aqueous humour and increases the amount of drainage through Schlemm's canal and trabecular meshwork, but shows dose-dependent (0.2% solution thrice daily) toxicity. To reduce the side effects and improve the efficacy, brimonidine was nanoencapsulated on ultra-small-sized chitosan nanoparticles (nanobrimonidine) (28 ± 4 nm) with 39% encapsulation efficiency, monodispersity, freeze–thawing capability, storage stability, and 2% drug loading capacity. This nanocomplex showed burst, half, and complete release at 0.5, 45, and 100 h, respectively. Nanobrimonidine did not show any in vitro toxicity and was taken up by caveolae-mediated endocytosis. The nanobrimonidine-treated trabeculectomy tissue of glaucoma patients showed better dilation of the trabecular meshwork under the electron microscope. This is direct evidence for better bioavailability of nanobrimonidine after topical administration. Thus, the developed nanobrimonidine has the potential to improve the efficacy, reduce dosage and frequency, and improve delivery to the anterior chamber of the eye.

Keywords

brimonidine, glaucoma, nanobrimonidine, nanoencapsulation, ultra-small chitosan nanoparticles

Theme(s) of the abstract

Translational and Clinical Neuroscience

Category of Submission

Original Research

5

Nanotechnology as a tool for better delivery across Blood Brain Barrier for treatment of neurological disorders

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Abstract

Blood brain barrier (BBB) is the major obstacle for the delivery of drugs which are designed to be delivered to brain to show their therapeutic effect. It is reported that almost 98% of the drugs are unable to cross BBB. To overcome this barrier, so many approaches have been utilized so far for better delivery of drugs to treat neurological disorders. Among them, nanotechnology based delivery systems have evolved as a wonderful tool. Several types of nanoparticles have been utilized, like polymeric nanoparticles, metallic nanoparticles, magnetic nanoparticles, etc. for the same. This work is a systematic meta-study to showcase the importance of this emerging tool and the mechanism of overcoming BBB for the better treatment of neurological diseases. Also, the major limitations of the use of nanotechnology, specially the toxicity, is the concern. So, this work will systematically present the overview of nanotechnology based approaches which have been utilized so far for the treatment of neurological disorders.

Keywords

nanotechnology; chitosan nanoparticles; magnetic nanoparticles, blood brain barrier

Theme(s) of the abstract

Other

Category of Submission

Original Research

8

The Silence of the guys: The neuroscience behind the unheard stories of emotional abuse of men.

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Abstract

In the present global climate of discussions around gender, there are instances when men's experiences of being psychologically or emotionally abused are underrepresented. The various behavioral and sociopolitical reasons aside, there is a possibility for certain neuropsychological factors among men that might have contributed to the same. The present review attempts to explore some of these factors, focusing on those pertaining to a relatively lower sensitivity towards threat and abuse as well as to the low ability of men in terms of articulating about such experiences when compared to women. This way, the review attempts to bridge neuroscience to the sociopolitical atmosphere and tries to understand human behavior in that context.

Keywords

men,emotion,abuse,mentalhealth, gender

Theme(s) of the abstract

Social Neuroscience, Psychology

Category of Submission

Systematic Review

9

Towards a neural explanation of managerial decision making under uncertainty

Alireza Valyan[1], Hossein Rahmanseresht[2]

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Abstract

With over and over uncertainties in the business world, managers have to make decisions under more uncertainty. Conventional explanations of managerial decisions due to the several shortcomings have failed to empower managers in real-world situations. Cognitive neuroscience approaches have brought some hopes to inform the context of management with more accurate explanations and opportunities to modulate managerial decision making. However, the field lacks mural targets required to assess and intervene in managerial decision-making under uncertainty. Intending to find neural correlates of managerial decision making under uncertainties, we have reviewed studies addressing neural correlates of decision making and decision making under uncertainties as well as managerial decision making. We have defined managerial decision making and it's differentiating features from at a neural level and used these features to limit neural correlates of decision making under uncertainty. Results showed that specific areas can be assigned to managerial decision making.To assess managerial decision-making under uncertainty we need the right neural targets. Our study will help scholars in the field to design and implement assessment paradigms as well as intervention strategies.

Keywords

Neuro-strategy, Managerial Decision-Making, Uncertainty

Theme(s) of the abstract

Social Neuroscience

Category of Submission

Systematic Review

10

Occurrence Patterns of Reported Gastrointestinal Cancers and Its Correlation with Psychological Issues Due to Cancer Diagnosis and Its Treatments – An Epidemiological Study.

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Abstract

Background: Cancer is regarded as the leading cause of mortality and disabilities worldwide. Gastrointestinal cancer is the fifth highest cancer accounting for 5.7% of all cancers worldwide. Cancer diagnosis and its treatment has an impact on the patients' physical and psychological wellbeing.

Methods: This epidemiological study was conducted at KLE Hospital and Medical Research Centre in Belagavi city, Karnataka, India for a period of 3 months. Data of sixty-one (61) patients was collected using Zung Depression Scale, General Anxiety Disorder-7, FACT-Cog and FCSI for depression, anxiety, cognition and quality of life respectively.

Results: The mutual risk factor of 61 patients was tobacco consumption, cigarette smoking and alcohol consumption (p=0.001) with majority of the cases being carcinoma of buccal cavity (n=14). A poor correlation between the type of gastrointestinal cancers, stage and treatment to depression, anxiety and cognition was noted.

Conclusion: This study concludes that there is an association between the risk factor and the occurrence pattern and an overall poor correlation of the cancer diagnosis and treatment with the psychological issues such as depression, anxiety and cognition.

Keywords

gastrointestinal cancers, depression, anxiety, cognition, pattern of occurrence.

Theme(s) of the abstract

Psychology, Cognitive and Behavioral Neuroscience

Category of Submission

Original Research

11

Ditching the questionnaire. How much can we trust ourselves with the booklet of questions.

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Abstract

Abstract:

Introduction:

Research into the pathology Major Depressive Disorder (MDD) has been heavily reliant on several theories sharing the goal of determining a biological explanation for depressive symptoms. By successfully deciphering the pathology of MDD, questionnaires may no longer be the means by which individuals are diagnosed with the disorder. Ideally, a more biological approach to testing would be used by virtue of more quantifiable and reliable data. This, however, involves the implication by which one may be denied the diagnoses of MDD despite experiencing two weeks' worth of symptoms. Questionnaires, on the other hand, directly question the user about the symptoms of the disorder, and as such, make them more relevant in the diagnoses. This study aims to investigate the validity of questionnaires by analyzing some of its major disadvantages. These disadvantages including

1. The effects of time spent on each question
2. The effects of personal interpretation (One may have differing opinions on what 'good' is vs what 'poor' is)
3. The effects of survey fatigue
4. The effects of lacking accessibility features

Methods:

The listed effects will be analyzed through a variety of methods. The effects of time will be analyzed by comparing the results of questionnaire's where the user had an unreasonably short time constraint with the results of users with an abundance of time. Further, the second disadvantage will be analyzed through delivering the same case to numerous test subjects, to examine fluctuations between interpretation of circumstance. Additionally, the users will be required to complete two questionnaires in total, in which half will do them in an opposite order of the other. This will test the variation in results of those fatigued after completing the initial questionnaire against the subjects who completed that questionnaire first. The effects of accessibility will be examined by analyzing various papers on MDD, with criteria that specifies the involvement of questionnaires. Analysis will be performed by analyzing the specific P values associated with the features listed.

Anticipated Results:

I hypothesize the overall effect of these features will have significant ($p<5\%$) effects on the overall data.

Conclusion:

Questionnaires are a powerful tool in data collection, especially when analyzing heterogenous disorders such as MDD. Despite this, there are glaring disadvantages that weaken their viability in a scientific environment.

Theme(s) of the abstract

Neuroethics, Psychology, Cognitive and Behavioral Neuroscience

Category of Submission

Original Research

12

THE EFFECT OF INTERPERSONAL COMMUNICATION COMPETENCE AND QUALITY OF FRIENDSHIP ON SOCIAL CONNECTEDNESS AMONG EMERGING ADULTS

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Abstract

Emerging adulthood is the stage of life between the ages of 18 to 25 when adolescents navigate the process of becoming young adults by becoming more independent and discovering much about themselves, which also sets the course for the rest of their life. Interpersonal communication competence is the skill to effectively exchange ideas with others in interpersonal relationships. It is an important soft skill which is essential in the present social structure. The friendships one has in emerging adulthood also significantly influence one's experience of life, with positive, supportive and meaningful friendships adding self-worth and happiness to a person's life. Social connectedness refers to one's sense of belongingness, where they feel comfortable and confident within a larger social context of family, friends and other acquaintances. A person struggling to feel connected begins to feel lonely and frustrated, becoming ostracized and distant from other people. This study explored the role of interpersonal communication competence and the quality of friendships in determining the perceived social connectedness in emerging adults. The variables were measured using Interpersonal communication competence (Rubin & Martin, 1994), McGill Friendship Questionnaire–Friend's Functions (Mendelson & Aboud, 1999), and Social Connectedness Scale- Revised (Lee & Robbins, 1995) among a sample of 128 emerging adults. Results indicate a strong correlation between the variables, with interpersonal communication competence being a significant predictor of social connectedness.

Keywords

interpersonal communication competence, quality of friendship, social connectedness, emerging adults

Theme(s) of the abstract

Social Neuroscience, Psychology

Category of Submission

Original Research

13

A study on handedness

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Abstract

A basic study was conducted to understand the nuances of 'handedness'. Factors that are commonly associated with handedness were studied. A total of 156 participants were selected from different parts of India. The sample included 106 females and 50 males. Some of the aspects considered were the relation of handedness with the sex of the individual, birth stress experienced by mother, brain damage suffered by the individual and handedness of family members. In addition, a handedness questionnaire by Annett (1970) was used, along with a few added questions. 83.96% of females were right-handed, 11.32% were left-handed and 3.77% were mixed handers. 78% of males were right-handed, 14% were left-handed and 10% were mixed handers. It was found that 22.2% of mixed handers individuals suffered from some kind of brain damage (as reported), 33.3% suffered from birth stress and 44.4% have an immediate family member who is left-handed. In the case of left-handers, 17.85% suffered from birth stress, and 39.3% had an immediate family member who is left-handed. 14% of right-handers suffered from birth stress and 14% had an immediate family member who is left-handed. Fisher exact test found that there is no significant association between sex and handedness, and between birth stress and handedness. However, a significant association was found between brain damage and handedness, and between handedness of family members and the individual. After observing the percentages of people doing each task with the left hand, right hand or either, we can understand that handedness is not discrete or concrete. It is fluid.

Keywords

Handedness, sex, birth stress, brain damage

Theme(s) of the abstract

Psychology

Category of Submission

Original Research

14

Computational Analysis of MicroRNA based Gene Regulation in Alzheimer's Disease

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Abstract

In the era of post-genomics, computational analysis of genomics and transcriptomics to understand the non-coding microRNA (miRNA) based regulated networks in neurodegenerative diseases like alzhimer's disease is an uphill task. We have herein approached the challenge on the basis of four computational approaches: Top-down **- miRNA based associated regulation**; Bottom-up **- miRNA based expressed and co expressed regulation**; Direct **- miRNA based Pharmacogenomic regulation**; Indirect **- miRNA based pharmacovarient regulation.** The outcomes of the present study on the identification of a specific miRNA as a potential biomarker for treating psoriasis will be discussed in detail in the presentation.

Keywords

Alzheimer's Disease, Computational Analysis, MicroRNA and Pharmacogenomics

Theme(s) of the abstract

Systems Neuroscience, Computational Neuroscience, Cellular and Molecular Neuroscience

Category of Submission

Original Research

16

Mutational Analysis of Exon 3 and Exon 4 of *PINK1* Gene.

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Abstract

The project objective was to analyze Exon 3 and Exon 4 of *PINK* 1 gene for investigating the presence of mutations causing autosomal recessive early onset of Parkinson's Disease (PD). PD is the second most abundant neurodegenerative disorder after Alzheimer's Disease. It is clinically characterized by a classic tetrad of symptoms: low frequency resting tremors, rigidity of skeletal muscles, bradykinesia, and postural instability. The cardinal symptoms of PD result from progressive and pro-found loss of neuro-melanin containing dopaminergic neurons present in the substantia nigra pars compacta area of the midbrain, associated with eosinophilic, intracellular proteinaceous inclusions, termed Lewy bodies, and dystrophic Lewy neurites in the brain stem and cortical areas. PD has been the subject of intense investigation to understand its etiology and physiopathology. Environmental factors were long thought to be the predominant cause of PD. However, recently, thorough study of certain rare families revealed a clear Mendelian inheritance of Parkinsonism. Genetic mutations are the cause of 10% of total PD cases worldwide. Several causative genes have been identified, the *PINK1* gene being one of them. *PINK1 gene* encodes for PTEN-induced kinase 1 (PINK1). PINK1 protein protects cells from mitochondrial dysfunction by causing parkin protein to bind to the depolarized mitochondria, inducing its autophagy. To investigate the genetic cause behind early onset of Parkinson's Disease, a sample size of 25 patients suffering from Young Onset Parkinson's Disease at Institute of Medical Science, Banaras Hindu University, Uttar Pradesh was taken. DNA was isolated from the 5ml peripheral blood samples collected from the patients. Quality assessment of the diluted form of DNA samples collected was done by NanoDrop. Amplification of Exon 3 and 4 of *PINK1* gene was carried out by Polymerase Chain Reaction. Quantitative analysis of PCR products was done by Gel Electrophoresis. ExoSAP was carried out to enzymatically decontaminate PCR products from excess *Taq* polymerase, buffer, primers and nucleotides. Cycle sequencing was carried out to obtain their nucleotide sequence, which then underwent computational analysis using NCBI-BLAST to detect any point mutations. Result obtained was that an Adenosine deletion was found in exon 4 of *PINK 1* gene. Due to this deletion, frameshift mutation occurs resulting in STOP codon 'UGA'. Due to the STOP codon, pre-translated truncated protein forms which might disrupt the PINK and PARKIN pathway leading to disease phenotype.

Keywords

Parkinson's Disease, PINK1 gene, genetic mutations, Polymerase Chain Reaction, Lewy bodies

Theme(s) of the abstract

Neurogenetics, Cellular and Molecular Neuroscience

Category of Submission

Original Research

22

PERCIEVED STRESS AND EMOTIONAL EATING DURING COVID -19: AN EXPLORATORY STUDY FROM INDIA

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Abstract

Abstract

Background:

Across the globe, multifaceted dimensions of the COVID crisis and its socio-economic and psychological impact have unleashed uncertainty in the life and livelihood of individuals. It is known that stress may cause overeating in certain individuals. Obesity due to inappropriate eating habits, including overeating has recently been discussed during this COVID pandemic crisis in the context of stress. Emotional over-eating (EOE) is sometimes considered as a strategy for emotion regulation (ER). Considering this fact, this study used a snowball technique to analyze the prevalence of emotional overeating caused due to stress during this pandemic situation.

Methods:

The present cross-sectional online study of 607 respondents across all regions of the India attempts to assess the perceived stress levels during COVID-19, evaluate their tendency to engage in emotional eating and examine the relationship between perceived stress and emotional eating during COVID 19 situation.

Result:

Stress perception is significantly associated with emotional over eating(r=0.44; p=1.365e-30) and such phenomenon is marginally more prominent in males(r=0.46; p=4.779e-17) than females (r=0.37; p=2.197e-11). A negative association of age, though weak (r= -0.34,p=3.17e-18), has been found with both perceived stress levels as well as emotional over eating, signifying that the youngsters are more prone to stress and over eating than older persons, during these COVID times. Study also evidenced that people with psychiatric conditions are significantly more affected than others with their average perceived stress scores (PSS) being significantly higher at 22.57 (SD=6.99) as compared with the overall average PSS of 17.39 (SD=7.38). Similarly, the average emotional over eating(EOE) scores for such persons was also higher at 15.36(SD=4.75) as compared with the overall average EOE score of only 9.49 (SD=5.57) for the entire population indicating that they may be more vulnerable to emotional eating. Whereas, medically ill persons are only marginally more affected than overall population the study shows.

Conclusion:

Emotional over eating is significantly associated with perceived stress; the association is more prominent in females. Younger people and people with mental illness display significantly more emotional eating behaviour during stress.

Keywords

Emotional over eating, Perceived stress, COVID-19, Mental health.

Theme(s) of the abstract

Psychology

Category of Submission

Original Research

24

Multi-session Transcranial Direct Current Stimulation(tDCS) for cognitive enhancement and motor learning in healthy subjects: A Systematic Review

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Abstract

Introduction:

tDCS is a non-invasive brain stimulation method which can be used to stimulate the cortical regions of interest. tDCS enhances cognitive abilities and motor skills, which can have potential utility in critical and high-demanding fields like defense. Despite uniformity in the stimulation parameters, the outcome variability is very high among the single session tDCS studies. In contrast, multisession tDCS would be more predictable and can have longer-lasting effects. Hence, we systematically reviewed the multi-session tDCS studies to understand its impact on the cognitive and motor abilities in healthy individuals.

Methods:

We searched articles on electronic databases using terms such as “Multi-session” OR “Multiple sessions” AND “tDCS” OR “Transcranial Direct current stimulation” AND “Healthy”. Studies published in English language with healthy participants aged above 18 years, from all races and sex who were administered tDCS for more than two consecutive days were included in the review.

Results:

After removing the duplicates, protocol paper and those not meeting inclusion criteria, 14 articles were identified to fulfil the criteria among 38 articles identified in the search. All of the reviewed papers were randomized controlled studies. They used anodal tDCS at the site of stimulation with cathode being the reference electrode at the contralateral supraorbital area(10/14), cheek(2/14), or the deltoid region(2/14). The intensity of stimulation ranged from 1-2 mA, where the majority of the Working Memory(WM) domain studies were using 1mA (6/10); the Motor domain studies were using 2mA of current (2/3). The studies which targeted improvement in WM predominantly stimulated the Left Dorsolateral Prefrontal cortex, whereas the studies targeting verbal memory and language learning targeted Broca’s area. The studies in the motor learning domain targeted the primary motor cortex and supplementary motor area. The number of sessions varied from 3-15, and most of the studies used Online stimulation, where tDCS is delivered during the training of the task. Most of the published studies showed an intended positive impact of anodal tDCS. The cumulative dose of current and online task performance seemed to be associated with the studies showing positive effects.

Conclusion:

tDCS is safe, tolerable, and seems to be a promising modality to enhance cognition in healthy individuals with multi-session stimulation. Further studies evaluating the optimal parameters and understanding the biological mechanisms with multi-session tDCS in healthy individuals is warranted. These studies would provide leads towards its utility in cognitive/motor skill training in health and disease.

Keywords

Transcranial Direct Current Stimulation (tDCS), Multisession, Cognition, motor learning, healthy subjects.

Theme(s) of the abstract

Cognitive and Behavioral Neuroscience, Translational and Clinical Neuroscience

Category of Submission

Systematic Review

25

Combinatorial therapy against Alzheimer's disease using herbal nano-drug delivery system

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Abstract

Alzheimer’s disease (AD) is a common neurodegenerative disorder and is majorly characterized by symptoms such as the decline of cognitive function coupled with progressive memory loss. AD is detected by the presence of several molecular lesions that lead to inflammatory and oxidative damage due to the presence of misfolded proteins accumulated in the aging brain, amounting to synaptic dysfunction and energy loss. AD and dementia have gained major public health concern owing to their lack of treatment. AD is a neurodegenerative disorder that progresses day by day. Presently, none of the FDA approved drugs to treat AD can neither completely cure the disease nor enhances the patient's cognitive or memory skills as they are not designed to improve the neural functions. Such drugs increase the efficacy of the drug at the cost of the hosts’ tolerance to it in a dose-dependent manner causing several adverse effects at higher doses. Hence, a need for an alternate source of medication to cure Alzheimer's disease arises.

Combination drugs are gaining importance and progress in pharmaceutical technology as they act on multi-mode targets responsible for the cause of AD. These products increase clinical effectiveness, reduce administrative costs and enhance the patients' adherence capacity. The plants with the potential neuroprotective property may provide for an efficient yet sustainable pharmaceutical practice if considered for combinational therapies of AD after a detailed mechanistic evaluation. The HAp (hydroxyapatite) nanoparticles synthesized from plant extracts are well known for their excellent biocompatibility and bioactivity effects. After achieving good reasonable activity, Hap nanoparticles are selected as the drug delivery system to degrade the disease-causing proteins using the Drosophila model. Drosophila is well suited for AD research, as it provides numerous approaches to investigate defects in neuronal morphology and function along with the possibility to assay cognitive processes, hence the Hap drug delivery systems are evaluated against AD using Drosophila model.

Keywords

FDA, hydroxyapatite, neuroprotective, inflammatory, cognitive

Theme(s) of the abstract

Other

Category of Submission

Original Research

26

The glibenclamide effect on dopamine and norepinephrine content in rat brain in endotoxemia

Alexander Zubov[1], Tatiana Tuituinnik[1,2], Viktoriya Maystrenko[1], Marina Karpenko[1,3]
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Abstract

Introduction. Type 2 diabetes mellitus mainly affects people in older age groups, who usually have a history of comorbidities associated with the development of a chronic inflammatory process. The purpose of this research was to study the dose-dependent effect of chronic administration of glibenclamide on the content of dopamine (DA) and norepinephrine (NA) in the striatum and rat hippocampus in normal and endotoxemia.

Materials and methods. Four groups of Wistar rat males were used in experiments: group 1 (daily intraperitoneal (i.p.) GD administration in a dose of 10 μg/kg for 10 days, n=10), group 2, 3 (GD in a dose of 25 μg/kg or 50 mg/kg, respectively, n=10), and group 4 (daily i.p. administration of sodium chloride in a dose of 1 ml/rat for 10 days). On the eighth day of the experiment, 5 animals from each group were administered i.p. LPS in a dose of 1 mg/kg. At the end of the experiment the animals were decapitated and the striatum and hippocampus were removed. The concentration of norepinephrine and dopamine in samples was measured with HPLC-ED. The data were expressed as ng/mg protein in the sample and presented as m ± SEM, MANOVA c post hoc Tukey criterion was used.

Results. It was found that introduction of GD according to the chosen scheme leads to a dose-dependent increase of NA content in the striatum cells from 12.5 ± 2.0 in group 2 (+ LPS) to 3.3 ± 1.0 in the same group 2 (- LPS), p=0.022. The level of NA in the striatum of rats of group 1 (+ LPS) was 2. 7 ± 1.0, p=0.028 vs 5.7 ± 1.50 in rats of group 1 (- LPS), p=0.049. DA level in the striatum of rats of group 3 (+ LPS) on the 8th day was 117.2±14.5, which is more than that of the control group (- LPS) 57.6±11.4, p=0.022. In the hippocampus, the level of norepinephrine in the 3rd group of rats (+LPS) on day 8 was 1. 7±0.4, higher than the level of NA in the same group 3 (-LPS) 0.7±0.2, p=0.016, as well as increasing in comparison with the control group (-LPS) 1.7±0.3, p=0.042 and with the control group (+LPS) 0.9±0.2, p=0.015.

Conclusions. Thus, the introduction of GD leads to an increase in the content of dopamine and norepinephrine in the striatum, norepinephrine in the hippocampus, and the effects of GD are preserved under endotoxemia conditions.

Keywords

CNS, glibenclamide, lipopolysaccharide, dopamine, norepinephrine

Theme(s) of the abstract

Cellular and Molecular Neuroscience

Category of Submission

Original Research

28

The Anomalous Memory Consolidation Model of Dreaming: A Robust Model of Dreaming mechanism that accounts for both continuities and incongruities of dream content

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Abstract

Psychologists like Freud exclaimed that repressed wishes accommodated in the subconscious determine what we see in our dreams. While, this notion seen neurobiologically, is the other way around, this study would deal with the neuroscientific aspect of composition of dream content. Since it is now widely accepted that sleep, and especially nonrapid eye movement (NREM) sleep plays an active role in learning and memory consolidation, [5] it becomes important to find out what kind of memories are these. The different (neurobiological) perspective about dream content comes from the 'anomalous' information transfer that occurs during the neocortical-hippocampal dialogue during memory consolidation (while sleeping), the two areas are known to communicate during the NREM stage of sleep and are believed to break off their communication just when switching to the REM stage of sleep [1] characterized by elevated levels of Acetylcholine. The consolidated information already transferred to the neocortex during NREM stage must be made available to the dreaming brain but as newly consolidated memories evoking related old memories for consolidation as new cortical circuits. Hypothetically, the 'bits' of information present in neocortex may come together very arbitrarily to constitute the dream content which can be supported by Retro-selection theory (Blackmore, 2004). According to some studies, the automatically-activated forebrain synthesizes the dreams by contrasting the information generated in specific brain stem circuits with information stored in memory [9]

Keywords

Memory Consolidation, Model of Dreaming, Dreaming mechanism, Sleep, REM Dreams

Theme(s) of the abstract

Cognitive and Behavioral Neuroscience

Category of Submission

Systematic Review

31

Development of Proteomic biomarkers for the detection of Mild Traumatic brain inury .

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Abstract

Traumatic Brain Injury (TBI) is a significant health concern and affects a wide cross section of today's society. Current diagnostic criteria and modalities, such as brain imaging and subjective measures of consciousness such as the Glasgow Coma Scale (GCS) score, are insufficient to properly diagnose the full spectrum of head injuries. Assessment of injury severity and the outcome are further complicated by the vast array of symptoms, many of which mimic those displayed by other disorders. It is important to possess a better diagnostic tool for head injury triage and outcome prediction. One current line of inquiry seeks to discover traumatic brain injury biomarkers, in order to differentiate the healthy patient from the concussed individual at an early stage of detection to avoid unnecessary neuroimaging and harmfully associated radiation exposure to the patients. There are a few TBI indicators already discovered, however, biomarkers that could be used clinically are yet to be established. The objective of this study was to investigate the proteomics associated with TBI and was attempted to understand better with in-vitro, in-vivo, as well as in silico techniques.

For in-vitro experiments, Neuro-2a cell lines were used and Sprague Dawley rats were treated with blunt traumatic brain injury for in-vivo studies. The main proteomic techniques that assisted in this investigation were Bradford Assay, SDS-PAGE, and 2-D PAGE. An in silico characterization of the top four potential TBI biomarker candidates was also performed using ProtParam, GOR IV, Swiss Model, and STITCH tools on the ExPASy server.

Keywords

TBI , biomarker , detection , proteomics

Theme(s) of the abstract

Translational and Clinical Neuroscience, Cellular and Molecular Neuroscience, Computational Neuroscience

Category of Submission

Original Research

32

AUTISM OR AUTISM SPECTRUM DISORDERS (ASD) : Autism or autism spectrum disorder(ASD) is a neurological and developmental disorder characterized by social impairment and communicative behaviour.

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Abstract

Autism or Autism spectrum disorders is a neurological and developmental disorder characterized by social impairment and restricted interactive and communicative behaviour. It may occur as an isolated disorder or in the context of other neurological, psychiatric developmental and genetic disorder with a singular pathway and has a rather complex etiology. It is interested to note that perturbation in synaptic growth, developmental and stability underlie a variety of neuropsychiatric disorder including ASD, Schizophrenia, Epilepsy and intelluctual disability.

Autism spectrum disorders (ASD) are a complex set of heterogeneous neuro developmental disorders categorized by a triad of key behavioural anomalies. Characteristics behavioural abnormalities consists of restricted interests accompanied by repetitive behaviour, deficits in language and communication and the inability to engage in reciprocal social interaction. Autism is not a singular disease entity. The disorder encompasses a spectrum of wide ranging phenotypic manifestation which span from debilitating impairments to mild behavioural and personality traits. Therefore, autism is rightfully referred to as "autism spectrum disorders (ASD)."

Autism spectrum disorder or autism appear to be involved in early brain development.

Obvious sign and symptoms show early onset within the first 3 year of life and persist into adult-hood. According to the recent report from the centre for disease Control an estimated 1 in 88 children has been identified with Autism spectrum disorders. Interesting, these disorders show a gender bias where male are affected almost five time more than females.

Research finding from animal model of Autism spectrum disorder suggest that disruption of synapse formation and stabilization processes is a key underlying feature in Autism spectrum disorders etiology. Dysfunction in the assembly on structure of transmembrane and scaffolding protein needed for building and maintaining synapses and disruption in cellular signalling pathways controlling Synaptogenesis are major contribution factor in Autism spectrum disorder(ASD).

Autism is an incurable disease but treatment can make a big difference. The sooner treatment start, the better.

* Speech therapy to help with talking and language skills.

* Occupational therapy to help with everyday tasks like dressing and playing.

*Behavioural therapy to help improve behaviour.

*Social skill training to help with relating to other.

*Special education to help learning

*Medicine to help with things like sleeps, paying attention and hyperactivity.

Diagnosis of Autism spectrum disorders now includes several conditions that used to be diagnosed separately - autistic disorder, Perusive developmental disorder not otherwise specified (PDD - NOS) and Asperger syndrome. These conditions are now called Autism spectrum disorders (ASD).

Keywords

Autism, ASD, neuro, neuropsychiatric disorders, autistic, brain development and neurological development, perusive developmental disorders not otherwise specified, Asperger syndrome, Etiology

Theme(s) of the abstract

Developmental Neuroscience, Cognitive and Behavioral Neuroscience, Social Neuroscience, Ecological Neuroscience

Category of Submission

Systematic Review

34

Effects of targeted activation of microglia using monoclonal antibodies on Aβ42 clearance mechanisms

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Abstract

Alzheimer's disease (AD) is a chronic neurodegenerative disorder, which affects the memory and cognitive functions due to the degeneration of neurons. While various susceptibility and risk factors are known, the exact cause or trigger of this disease is still under investigation. AD affects a large number of the elderly population in humans, and this number is charted to rise in the upcoming years. One of the hallmarks of AD is the Amyloid Beta Plaque, which is formed due to the accumulation of the Aβ42 peptides. Aβ42 is produced when the Enzymes β Secretase and γ Secretase cleave the Amyloid Precursor Protein (APP). Aβ42 is usually produced in regulated amounts, and hence doesn't escalate like in the case of AD, due to the clearance mechanisms that preserve the homeostatic conditions. These clearance mechanisms involve two different approaches: Enzymatic and Non-enzymatic. Among the Non-enzymatic plaque clearance mechanisms, microglia are one of the predominant cells involved, due to their phagocytic properties. While the other plaque clearance mechanisms are biomolecules and signal-inducing chemicals that may have singular functions, microglia are also particularly involved in another aspect of Alzheimer’s Disease: exacerbation of AD in later stages. When amyloid plaque accumulates, the microglia get activated, which cascades into serious neuroinflammation and a strong immune response leading to T-cells invading the brain, resulting in the eventual neurodegeneration. Targeted activation of microglial cells may provide a nuanced and prophylactic solution to prevent AD exacerbation and neuronal damage. Recent studies report that Anti-Tau antibodies enhance microglial activity. This study proposes to test such potential monoclonal antibody candidates that specifically activate microglial cells during the early stage of plaque accumulation, to help clear amyloid beta effectively and prevent the progression of Alzheimer’s, and elucidate the mechanism of action. This investigation can also help reduce the sensitivity and susceptibility of the 65 and above populations to AD. The study proposes to conduct a two-week experiment that exposes the HMC3 cell culture samples to the potential MAb candidates. The microglial cell samples are actively monitored and analysed on a regular basis until they reach their respective activation stages. The efficiency of microglial activation is calculated and the morphological side-effects of the MAbs on the samples are observed. The study will provide important data that can help understand the effects of monoclonal antibody dependent activation of microglia as a precursor event that cascade into an efficient plaque clearance mechanism.

Keywords

Alzheimer’s Disease, monoclonal antibodies, microglia, Aβ42, activation

Theme(s) of the abstract

Cellular and Molecular Neuroscience, Translational and Clinical Neuroscience, Other, Cognitive and Behavioral Neuroscience

Category of Submission

Original Research

35

Antidiabetic and anti-Alzheimer activity of eugenol – an in silico analysis

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Abstract

Introduction

Eugenol (4-Allyl-2-methoxyphenol) is an aromatic oil present abundantly in *Syzgium aromaticum, Cinnamomum verum, Ocimmum gratissimum*, and various other medicinal plants. This phenylpropene compound is known to exhibit antidiabetic, anti-amnestic activity, and other nootropic benefits. Given to the close-knit relation between Alzheimer's disease (AD) and type 2 diabetes (T2D), the speculate that a common target must exist which could alleviate diabetes as well as AD. Therefore, in the present study, computational approaches have been employed to identify common protein targets of eugenol and to discern its mechanism of action.

Method

Using literature and publicly available databases such as DisGeNET, Malacards, NCBI-Gene and KEGG, AD and T2D associated genes were identified. Genes common to both the diseases were collected for protein-protein interaction analysis. Using databases like chEMBL, STITCH, BindingDB, and DrugBank potential targets of eugenol were identified. Further, these targets were analyzed using computational tools such as STRING, GeneMania. Finally, eugenol-target protein docking studies was conducted to predict the interaction of eugenol with a target to discern the mechanism of action of eugenol

Results

From literature and freely accessible database, 39 genes were identified to be common to both AD and T2D. On the other hand, 181 AD-associated and 48 T2D associated proteins were identified as targets of eugenol. Among the genes of AD and T2D, 39 genes were identified to be involved in both the diseases. Of the common genes, TRPV1 and ERBB2 are the common targets of eugenol. Further docking studies showed the interaction mechanism of action of eugenol with TRPV1, thus ameliorating AD and T2D.

Conclusion

The mode of action of eugenol in ameliorating T2D and AD was predicted to be via TRPV1. In addition, better blood-brain barrier penetration and gastrointestinal permeation capability of eugenol show the prospective of this phytochemical in drug development against AD and T2D.

Keywords

Docking, Eugenol, Alzheimer's Disease, Network Analysis, Type 2 diabetes

Theme(s) of the abstract

Systems Neuroscience, Computational Neuroscience, Cognitive and Behavioral Neuroscience, Cellular and Molecular Neuroscience

Category of Submission

Original Research

37

Evaluation of Anti-Parkinson’s activity of Herbomineral on Rotenone induced Parkinson’s disease in Drosophila melanogaster.

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Abstract

Background and rationale: The study was designed to validate the claims in Ayurveda regarding the efficacy of Ayurvedic drugs in neurodegenerative disorders. It was decided to conduct an efficacy study of two herbomineral drugs (*Abhrak bhasma*) on Parkinson’s disease induced chemically in Drosophila melanogaster (fly). Parkinson’s disease has symptom descriptions in Ayurveda and modern medicine. The latter offers only the symptomatic therapy by replacing dopamine, the neurotransmitter involved, but does not slow down or reverse the loss of dopaminergic neurons.

Methods: Rotenone, in a final concentration of 125 µM, was induced for inducing Parkinson’s disease in Drosophila melanogaster. Table concentration of bhasma samples were selected on the basis of viability assays carried out in our lab. The group serving as negative control will not have the study drugs in the cornmeal medium and the flies from positive control will be fed with L-dopa dissolved in the medium in the concentration of 1 mM. The flies in the bottle will be maintained for a period of 7 days at 25^{O}C. On the 8^{th} day, they will be subjected to climbing assay. The brain was dissected out and was checked for Malondialdehyde, Glutathione and Protein estimation.

Results: Climbing assays showed a significant reduction in the climbing/ motor ability between the control and disease control groups. There was a significant improvement in the climbing ability of flies fed with l-dopa and study drugs. Malondialdehyde and Glutathione levels showed an improvement in the test group indicating a potential anti-inflammatory property. There was a significant improvement in the protein levels in the Protein levels in the Drosophila test group.

Conclusion: Abhrak bhasma was found to be improve the motor activity and reduce the inflammation in Drosophila brain.

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Keywords

Drosophila, Parkinsons disease, Rotenone, motor activity, inflammation

Theme(s) of the abstract

Other

Category of Submission

Original Research

38

Microglia mediated Synaptogenesis and Synaptic Pruning in cultured Hippocampal Neurons.

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Abstract

Microglia are primary innate immune cells in the central nervous system. There is a lot of understanding of how microglia maintain immune homeostasis by being activated by pathogenic stimulus and how they resolve injuries to the brain but very less work is aimed at understanding their function in a healthy brain. Microglia aren't just mere pathologic sensors they play a multifaceted role in synapse formation and the elimination of unwanted synapses via processes termed synaptogenesis and synaptic pruning respectively during early brain development and maturation. Synapses play a very important role in higher cognitive functions in the human brain. This study points out that microglia play a key role in synaptogenesis and synaptic pruning of cultured hippocampal neurons and have identified a set of critical signaling pathways between microglia and hippocampal neurons. This review highlights the most recent findings demonstrating how the dynamic interactions between hippocampal neurons and microglia shape the synaptogenesis and synaptic pruning in a healthy brain and how alterations in microglia-hippocampal neurons signaling could potentially contribute to neurodevelopmental disorders such as schizophrenia, autism and epilepsy.

Keywords

Microglia, synaptogenesis, synaptic pruning, hippocampal neurons, synapse

Theme(s) of the abstract

Developmental Neuroscience, Cellular and Molecular Neuroscience, Cognitive and Behavioral Neuroscience, Neurogenetics

Category of Submission

Systematic Review

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