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Abstracts

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Neurological Disorders, Stroke and CNS

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PROGNOSTIC VALUES OF PERIPHERAL GLUTAMATE AND TNF-α LEVELS IN PATIENTS WITH INTRACEREBRAL HAEMORRHAGE

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Intracerebral hemorrhage (ICH), a detrimental disease, is associated with one month fatality in 40% of patients and worst neurological outcome among survivors. In the past several years, emerging evidence suggests that inflammatory and excitotoxic mechanisms are in the core of the pathophysiological processes, leading to neurological deterioration and secondary brain injury after ICH, thus being tied up to the patient's outcome. Following ICH, tumor necrosis factor-α (TNF-α) signalling exerts an acute detrimental role, being also argued as the main driver for increase in the blood brain barrier permeability and formation of the perihematomal edema. Elevated perihematomal glutamateinduced excitotoxicity have also been associated with the blood brain barrier disruption and neuronal death, severely affecting patient prognosis. In this lecture, we discuss the results from our working group which support the idea that peripheral TNF-α and glutamate levels can reflect CNS inflammation and excitotoxicity following ICH, as well as their utility as biomarkers for prognostication and clinical decision making between conservative or surgical treatment in patients with ICH.

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MEMTIN, A NOVEL PREVENTATIVE AND THERAPEUTIC FOR ALZHEIMER'S DISEASE

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Our research is focused on the identification and characterization of factors relevant to obesity and/or the metabolic syndrome that modulates brain aging or can manipulate the conversion of normal brain aging to that hampered with Alzheimer's disease (AD). To achieve that, we have used biochemical, molecular, cell biology and *in vivo* models to screen, identify and characterize our lead product under development, Memtin (a leptin product). Leptin is an adipocyte hormone, thought to control energy homeostasis and known to have pleiotropic activities. Data suggest that leptin can modulate memory and cognition through receptors in the hippocampus, where it is expressed at high density, following transport from the periphery via a natural saturable transporter in the blood-brain-barrier. We further have accumulated data in support of leptin's disease modifying potential utilizing cell-based and animal models. Further, epidemiological studies in humans involving thousands of subjects demonstrated an association of low leptin and a high risk for Alzheimer's disease and a positive correlation between leptin levels and hippocampal volume. Interventional studies are also in agreement. Thus, our approach involving repurposing an approved drug as a replacement therapy for MCI/prodromal AD characterized by hypoleptinemia represents a novel solution for a huge unmet medical need.

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EXPRESSION OF SIGNALING PROTEINS IN ISCHEMIC PENUMBRA AFTER PHOTOTHROMBOTIC STROKE

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In ischemic stroke, vascular occlusion and energy deficit rapidly induce tissue infarct. Cell damage propagates to surrounding tissues for several hours. This therapeutic window provides time to save neuronal cells in penumbra. To determine proteins involved in neurodegeneration and neuroprotection in penumbra, we studied protein expression profile in 2 mm ring around photothrombotic infarct core induced in rat cerebral cortex by local laser irradiation after rose bengal administration. Histological and ultrastructural studies showed edema and degeneration of neurons, glia and capillaries, which decreased gradually across penumbra. Expression profile of 224 signalling proteins, 1, 4 or 24 hours after photothrombotic infarct comparing with untreated contralateral cortex was studied with antibody microarrays. Diverse cellular subsystems were involved in penumbra response. Proteomic analysis showed concerted upregulation of diverse proteins that initiate, regulate and execute apoptosis (Par4, E2F1, p75, p38, JNK, p53, GADD153, GAD65/67, NMDAR2a, c-myc, Bcl-10, AIF, SMAC/DIABLO, PSR, caspases 3, 6 and 7). Different anti-apoptotic (Bcl-x, p63, p21WAF-1, MDM2, ERK5, MKP-1, NEDD8) and signalling proteins that regulate cell metabolism, functions and survival (calmodulin, CaMKIIα, CaMKIV, ERK1/2, MAKAPK2, PKCα, PKCβ, PKCμ, RAF1, protein phosphatase 1α, ATF2, estrogen and EGF receptors) were simultaneously overexpressed. Bidirectional changes in adhesion and cytoskeleton proteins were associated with penumbra destruction or remodelling. Proteins that regulate actin cytoskeleton (cofilin, actopaxin, p120CTN, α-catenin, p35, myosin Va, pFAK) were up-regulated, whereas others (ezrin, tropomyosin, spectrin (α+β), βIV-tubulin, polyglutamated β-tubulin, cytokeratins 7 and 19) were downregulated. Downregulation of syntaxin, AP2β/γ, and adaptin β1/2 indicated impairment of vesicular transport and synaptic processes. Downregulation of Cdk6, Cdc7 kinase, Trf1, and topoisomerase-1 showed suppression of proliferation. APP, nicastrin and β-amyloid were upregulated. These data provide integral view on neurodegeneration or neuroprotection processes in penumbra. Some of these proteins may be potential targets for anti-stroke therapy.

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ROLE OF CALCIUM BINDING PROTEINS IN THE EARLY DEVELOPMENT OF THE ZEBRAFISH CNS

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Ionic Calcium (Ca^{+2}) plays an important role in controlling many physiological processes, such as muscle contractions, nerve signaling, membrane permeability, cell division and hormone release. It is well known that in nerve cells, the excess intracellular concentration of Ca^{+2} causes cell death. In recent years, many studies have reported that certain intracellular calcium binding proteins (CaBPs) act as calcium buffers, endogenous neuroprotectants to prevent neuronal death from excess Ca^{+2} influx, plays role in neuronal Ca^{2+} signaling and Ca^{+2} homeostasis, prevent or delay Ca^{+2} related excitotoxicity and are involved in neurotransmission. Besides, they are excellent markers for the neuroanatomical studies to identify unknown neuronal populations and pathways in CNS. We have investigated the role of calb2a and calb2b genes that are expressed in the CNS and multiple other tissues during early embryonic development of zebrafish. We have adopted individual and combined morpholino mediated inactivation approach to investigate the functions of calb2a and calb2b. Morpholino inactivation of *calb2a/calb2b* alone failed to generate an obvious phenotype. Morphological inspection of *calb2a/calb2b* combined knockdown morphants showed abnormal neural plate folding in midbrain-hindbrain region. The loss of mRNA leads to severe hydrocephalus, axial curvature defect, and yolk sac edema. Knockdown of *calb2a/calb2b* showed an impaired touchdown and swimming performance. Co-injection of the *calb2a/calb2b* morpholino oligonucleotide cocktail with human mRNA leads to the rescue of the strong morphant phenotype. This study provided the first comprehensive analyses of the zebrafish Calb2a and Calb2b proteins that are highly conserved and are originated from the same ancestral gene in evolution. Homology modeling and docking with the similar structure and Ca^{2+} binding sites for both proteins provide the evidence that they may have similar function and one can compensate for the loss of other. It confirms the unique and essential functions of *calb2a /calb2b* genes in the early development of the zebrafish.

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CHINESE MEDICINE FOR STROKE, BREAKING THROUGH BLOOD BRAIN BARRIER AND PROMOTING NEUROGENESIS

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Stroke is great challenge to human health. Blood brain barrier and nerve regeneration is the key factor for stroke treatment. Wu Shen Xing Nao Fang (WSXN) is composed of ginseng polygoni multiflori radix and gingko leaf, with the function of promoting blood circulation to remove blood stasis and awakening brain function has been made into dropping pill available for clinical stroke.

Breaking the blood-brain barrier and brain targeting

WSXN could reversibly open the blood-brain barrier, increasing the bioavailability of ginseng saponins, distyrene and ginkgolide, through the blood brain barrier into the brain tissue, to increase the concentration of within the brain tissue as much as 2~10 times. Oral dosage of 40, 80 mg/kg of WSXN for 7 days or 14 days can significantly reduce ischemic infarction area to more than 35% in MCAO and 4 VO cerebral ischemia models of rats, inhibit the neuroinflammation and mortality of the hippocampus CA1 area neurons >60%; and also improve the learning and memory of damaged rats; increase the number of BrdU positive cells in the ischemic tissue by 300% and promote the nerve regeneration. 0.2~5 μg/ml of WSXN can promote the proliferation and directional differentiation of neural stem cells, reduce neuronal injury and apoptosis of the neuron induced by hypoxia oxidative stress and Aß 25-35, whose efficacy index is superior to that of ginkgo biloba extract (EGB761 ®). Oral administration of WSXN for 90 days showed no obvious liver and kidney toxicity in rats. Clinical volunteers orally taking WSXN pill 0.35 g a time, three times a day, for 2~6 months, can eliminate sequela hemiplegia and movement disorder of the stroke patients and muscle tremor symptom of Parkinson's disease, resulting in independent living with effective rate > 80%

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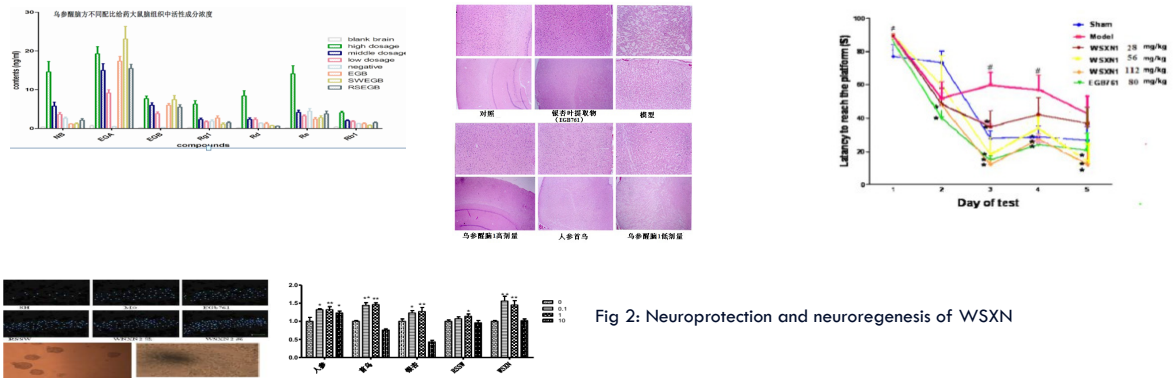

Fig 2: Neuroprotection and neuroregenesis of WSXN

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DIAGNOSTIC CHALLENGES OF AUTO MUTILATIONS: A CASE REPORT

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Introduction: Automutilation is a common psychiatric behavioral disorder. However, it could be a revealing sign of a rare neurological disease such as neuroacanthocytosis.

Case Report: We report a case of a 39 year-old patient, without previous neurological history. She was born to consanguineous parents. She has developed insidious oral automutilations. She was examined and followed by dermatologists and psychiatrists. No etiology was retained. Few months' later, she developed movement disorders. Neurological examination has confirmed choreic movements in the head and upper limbs, tendon areflexia and cognitive impairement. Blood smear revealed the presence of acanthocytes (9%). Biological assessment showed a high level of muscular enzymes (CPK=1000 IU/L, LDH=600 IU/L). Cerebral MRI showed an atrophy of caudal nuclei. The EMG concluded sensitivo-motor axonal neuropathy, predominant in inferior limbs. Genetic assessment for Huntington disease was negative. We retained the diagnosis of neuroacantocytosis and the treatment was mainly symptomatic (neuroleptics and vitamins). Evolution and prognosis were poor.

Discussion: Neuroacanthocytosisis is an autosomal recessive affection, with a progressive evolution. It appears often in young male adults and rarely among women such in our case. Clinical symptoms include movement disorders (chorea, oro-facial dyskinesia and oro-lingual automutilations), peripheral neuropathy and frontal dementia. Research of acanthocytes in blood smear is important for diagnosis, showing a percentage above 5%. Persistent elevation of CPK is usual.. MRI and genetic research showed that an atrophy of caudal nuclei can cause mutation in chromosome 9q21-22 gene. Treatment is mainly based on vitamins, neuroleptics and psychotherapy.

Conclusion: Neuroacanthocytosis is a rare affection with a polymorphous clinical expression. We should consider psychiatric symptoms as an inaugural form of its revelation.

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DIFFERENTIAL SEX RESPONSE TO ASPIRIN IN DECREASING ANEURYSM RUPTURES IN HUMANS AND MICE

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We previously found that aspirin decreases the risk of cerebral aneurysm rupture in humans. We aim to assess whether a sex differential exists in the response of human cerebral aneurysms to aspirin and confirm these observations in a mouse model of cerebral aneurysm. A nested case-control analysis from the international study of unruptured intracranial aneurysms was performed to assess whether a sex differential exists in the response of human cerebral aneurysms to aspirin. A series of experiments were subsequently performed in a mouse model of cerebral aneurysms. Aneurysms were induced with hypertension and elastase injection into mice basal cisterns. We found that aspirin decreased the risk of aneurysm rupture more significantly in men than in women in the International Study of Unruptured Intracranial Aneurysms. In mice, aspirin and cyclooxygenase-2 inhibitor did not affect cerebral aneurysm formation but significantly decreased the incidence of rupture. The incidence of rupture was significantly lower in male versus female mice on aspirin. Gene expression analysis from cerebral arteries showed higher 15-hydroxyprostaglandin dehydrogenase levels in male mice. The rate of cerebral aneurysm rupture was similar in male mice receiving aspirin and 15-hydroxyprostaglandin dehydrogenase inhibitor compared with females receiving aspirin and 15-hydroxyprostaglandin dehydrogenase agonist, signaling a reversal of the sex-differential response to aspirin. Aspirin decreases aneurysm rupture in human and mice, in part through cyclooxygenase-2 pathways. Evidence from animal and human studies suggests a consistent differential effect by sex. 15-hydroxyprostaglandin dehydrogenase activation in females reduces the incidence of rupture and eliminates the sex-differential response to aspirin.

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INVOLVEMENT OF D1/D2 DOPAMINE RECEPTORS WITHIN THE NUCLEUS ACCUMBENS AND VENTRAL TEGMENTAL AREA IN THE DEVELOPMENT OF SENSITIZATION TO ANTINOCICEPTIVE EFFECT OF MORPHINE

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The nucleus accumbens (NAc) and the ventral tegmental area (VTA) are two major areas for the mesolimbic dopaminergic system which are strongly involved in the development of behavioral sensitization. In the present study, we investigated the role of D1/D2 dopaminergic receptors within the NAc or VTA in response to sensitization to morphine by the tail-flick test as a model of acute pain. Sensitization was induced by subcutaneous (SC) injection of morphine (5 mg/kg), once daily for three days followed by 5 days free of drug. After the sensitization period, antinociceptive responses induced by an ineffective dose of morphine (1 mg/kg; SC) were obtained by the tail-flick test, and represented as maximal possible effect (%MPE). In experimental groups, D1 and D2 receptor antagonists, SCH-23390 and sulpiride (0.25, 1 and 4 µg/rat), were separately microinjected into the NAc or VTA, 10 min before morphine administration during the sensitization period, respectively. Results showed that injection of morphine during the sensitization period (development of sensitization) increased %MPE of the ineffective dose of morphine from 2.43±1.4% in naive to 47.75±4.01% in sensitized animals (P<0.001). Unilateral microinjections of different doses of the D1/D2 receptor antagonists, SCH-23390 and sulpiride, into the NAc dose-dependently decreased %MPEs in morphine-sensitized animals. Nonetheless, %MPEs were only affected by intra-VTA administration of SCH-23390 in morphine-sensitized animals (P<0.05). Our findings suggest that both the D1/D2 dopamine receptors in the NAc and the D1 receptors in the VTA may be of more important in the development of sensitization to in rats.

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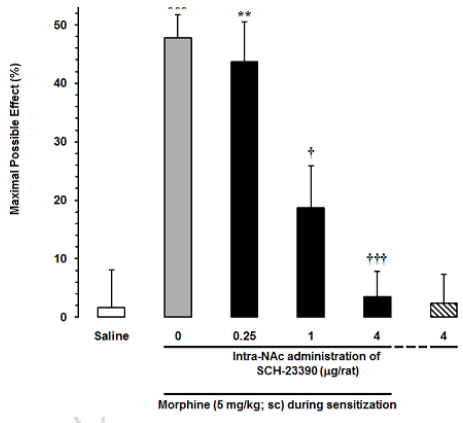

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RIBOFLAVIN: BENEFICIAL EFFECTS ON NEUROLOGICAL MOTOR DISABILITY IN MURIN MODEL OF MULTIPLE SCLEROSIS

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This experimental study assessed the effects of riboflavin on motor disability, spatial learning, and memory in experimental autoimmune encephalomyelitis (EAE). The C57BL/6 female mice (n=56) were assigned into 7 groups: sham operated 1 (SO1), healthy mice received PBS (phosphate buffer saline); sham operated 2 (SO2), healthy mice received PBS and riboflavin; sham treatment1 (ST1), EAE mice received water; sham treatment 2 (ST2), EAE mice received sodium acetate buffer; treatment 1 (T1), EAE mice received interferon beta-1a (INF-β1a); treatment 2 (T2), EAE mice received riboflavin; treatment 3 (T3), EAE mice received INFβ-1a and riboflavin. After EAE induction, scoring was performed based on clinical signs. By detecting score 0.5, riboflavin at 10 mg/kg of body weight and/or INFβ-1a at 150 IU/g of body weight, administration were started for two weeks. The brain and spinal cord levels of brain-derived neurotrophic factor (BDNF) were studied using real-time PCR and ELISA methods. Spatial learning and memory were assessed through the Morris water maze (MWM). BDNF mRNA expression and BDNF levels increased significantly in the brain of T3 group compared to the T2 or T1 groups (P<0.01 and P<0.05, respectively). Clinical scores were reduced in riboflavin treated groups compared to others. The EAE mice performed similarly compared to the healthy controlled mice in MWM test. However, T2 and T3 mice swam faster than the ST2 (*P<0.05*), T1 (*P<0.05*), and ST1 (*P<0.05*) mice. The results concluded that riboflavin has beneficial effects on neurological motor disability mediated by BDNF in EAE model of MS.

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NEUROLOGICAL STROKE REHABILITATION AND RECOVERY BY WAY OF MEDITATION

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Desire or excessive attachment suffer from depression, discouragement, hatred, resentment, fear, and anger leads to the path of intoxicants consumption. Thus generate more and more violence, road rage leads to traffic injuries and deaths. Those are on spiritual paths have a tremendous challenge if we are to counterbalance this negativity or face the multiple strokes. Stroke rehabilitation may be by physical therapy, occupational therapy, speech therapy along with optimal medical care continues to be viewed as advantageous. Coordination by a physician with expertise in neurorehabilitation is also an important aspect of successful stroke rehabilitation. Such expertise takes the form of realistic expectations about recovery based upon performance of serial functional assessment scales, knowledge about the size and location of the stroke. The care plan must also institute effective measures to protect against recurrent stroke, as stroke patients are generally at significant risk for further ischemic or hemorrhagic events. Stroke rehabilitation might be last stage should begin during the patient's hospital admission. Obviously, the degree of recovery is impacted by the age of the patient, the magnitude of the initial deficit, the condition of the patient, the motivation and family members, the risk of recurrent stroke as well as the quality of the stroke rehabilitation process. Many meditation systems available to stroke patients that can help them adjust to their new post-stroke lifestyle. All these systems are based on the path of Dhamma that created and teach by Gautam the Buddha. Thus whether you're coping with the aftermath of a stroke or working to overcome functional limitations created by multiple sclerosis, Parkinson's disease, brain injury, balance disorders or other neurological conditions, to restore your capacity for full, active living as quickly as possible.

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RIBOFLAVIN: BENEFICIAL EFFECTS ON NEUROLOGICAL MOTOR DISABILITY BUT NOT SPATIAL LEARNING AND MEMORY CONSOLIDATION IN MURINE MODEL OF MULTIPLE SCLEROSIS

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This experimental study assessed the effects of riboflavin on motor disability, spatial learning, and memory in experimental autoimmune encephalomyelitis (EAE). The C57BL/6 female mice (n=56) were assigned into 7 groups: sham operated 1 (SO1), healthy mice received PBS (phosphate buffer saline); sham operated 2 (SO2), healthy mice received PBS and riboflavin; sham treatment1 (ST1), EAE mice received water; sham treatment 2 (ST2), EAE mice received sodium acetate buffer; treatment 1 (T1), EAE mice received interferon beta-1a (INF-β1a); treatment 2 (T2), EAE mice received riboflavin; treatment 3 (T3), EAE mice received INFβ-1a and riboflavin. After EAE induction, scoring was performed based on clinical signs. By detecting score 0.5, riboflavin at 10 mg/kg of body weight and/or INFβ-1a at 150 IU/g of body weight, administration were started for two weeks. The brain and spinal cord levels of brain-derived neurotrophic factor (BDNF) were studied using real-time PCR and ELISA methods. Spatial learning and memory were assessed through the Morris water maze (MWM). BDNF mRNA expression and BDNF levels increased significantly in the brain of T3 group compared to the T2 or T1 groups (*P<0.01* and *P<0.05*, respectively). Clinical scores were reduced in riboflavin treated groups compared to others. The EAE mice performed similarly compared to the healthy controlled mice in MWM test. However, T2 and T3 mice swam faster than the ST2 (P<0.05), T1 (*P<0.05*), and ST1 (*P<0.05*) mice. The results concluded that riboflavin has beneficial effects on neurological motor disability mediated by BDNF in EAE model of MS.

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EXCESSIVE INCIDENCE OF STROKE IN IRAN: EVIDENCE FROM THE MASHHAD STROKE INCIDENCE STUDY (MSIS), A POPULATION-BASED STUDY OF STROKE IN THE MIDDLE EAST

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Background & Purpose: The epidemiology of stroke and its subtypes in the Middle East is unclear. Most previous studies have been performed in Western countries, and incidence rates are unlikely to apply in the Middle East. We aimed to determine the incidence of stroke in Mashhad, North-eastern Iran.

Methods: During a 12-month period (2006–2007), we prospectively ascertained all strokes occurring in a population of 450 229. Multiple overlapping sources were used to identify people with stroke. A large number of volunteers assisted in finding stroke patients not admitted to hospital. Potential cases were reviewed by a group of stroke experts before inclusion.

Results: A total of 624 first-ever strokes occurred during the study period, 98.4% undergoing imaging. Despite a relatively low crude annual incidence rate of first-ever stroke FES (139; 95% CI, 128 to 149) per 100 000 residents, rates adjusted to the European population aged 45 to 84 years were higher than in most other countries: 616 (95% CI, 567 to 664) for ischemic stroke, 94 (95% CI, 75 to 113) for intracerebral hemorrhage, and 12 (95% CI, 5 to 19) for subarachnoid hemorrhage. Age-specific stroke incidence was higher in younger patients than is typically seen in Western countries. Comparison of age-specific incidence rates between regions revealed that stroke in Mashhad occurs approximately one decade earlier than in Western countries.

Conclusions: The results of this study provide evidence that the incidence of stroke in Iran is considerably greater than in most Western countries, with stroke occurring at younger ages. Ischemic stroke incidence was also considerably greater than reported in other regions.

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INSTRUCTIONAL TEACHING STRATEGY CAN BOOST AN ACTIVATOR OF STUDENTS

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A teacher has a person who teaches in school rather than, especially helps others to shows knowledge, competence and value, playing a role of half mother and father. Concept mapping instructional approach as an activator of students' performance in the teaching and learning of excretion has investigated. The quasi experimental design has employed. Purposive sampling technique has used to select three intact biology classes of Senior High School 2 students with a total sample size of 108. The two main instruments used for data collection has General Knowledge in Biology Performance Test (GKBPT) and Students' Performance Test in Excretion (SPTE) with K-R 20 reliability coefficient of 0.812 and 0.866 respectively. Point Bi-serial Correlation, Wilcoxon Signed Rank test, effect size, chi-square and Kruskal-Wallis H test has employed to analyse the quantitative data collected using the students' achievement scores. The study showed that the effect size of the students' performance in the concept mapping of the post-test scores has better than that of the pre-test scores. The instructional approach did not only improve students' achievement in the biology course but also helped the students to retain the concept learned for longer period. Based on the result, recommendations have been made.

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BEHAVIOUR ADOPTED AT THE FIRST SYMPTOMS OF STROKE

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Introduction: Stroke is the second cause of death in Colombia. The level of knowledge of stroke within the population can determine its ability to recognize and make the decision to go to an emergency room.

Objective: Study the attitude and/or behaviour adopted by the patient and/or the family before a stroke in patients, diagnosed as ischemic stroke in 4 different hospital centers of Bucaramanga, Colombia.

Methods: Information on 348 patients was included; they were followed in a cohort from Nov' 2015 to Dec' 2017. Information was collected from patients and/or relatives.

Results: The mean age of the patients was 69.2 years. 75% of them came from urban areas. During the acute presentation of the event (stroke), 69% of patients or families did not recognize or think of stroke as a possibility. 8.9% did not initially consider that it was an important illness and 6.3% thought that it would recover spontaneously. In the sample analyzed, only 8% underwent vascular recanalization therapies.

Conclusion: Knowledge about stroke is still very poor in the general population and this affects the out of window arrival of these patients to hospital centers, and the low percentage of patients gain benefit from the recanalization therapy.

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PREVALENCE OF INHERITED NEUROLOGICAL DISORDERS IN AZAD JAMMU AND KASHMIR

Sadia , Zeeshan Anjum, Naheed Bashir Awan, Farhat Rafique Kiani, Syeda Ain ul Btool, Fauzia Saeed, Qudir Javed, Dahood Ahmad Qurashi, Mohsan Hanif, Shabnum Hameed, Tooba Khanum and Ghazanfar Ali

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Background & Objectives: Large scale epidemiological studies on inherited neurological diseases are rare in Pakistan and in Azad Jammu and Kashmir. Therefore, we conducted a population based cross sectional epidemiological study on a stratified randomly selected sample from the four major districts of AJ&K, to study the four major inherited neurological disorders which include: intellectual disability, microcephaly, neuromuscular dystrophy and Parkinson's disease.

Methods: Families with inherited neurological disorders were randomly recruited through door to door survey. Approval for this study was obtained from the Institutional Review Board of University of Azad Jammu and Kashmir. A simple easy-to-use questioner was developed to collect information from the affected individuals.

Results: Total 10,000 individuals were selected for interview among which 9711 (97.11%) took part in the study while 289 (2.89%) refused to take part in this study. Among responsive, the total number of affected individuals with different neurological disorders was 466 (4.80%) with an age range of 1-60 years. According to this data, prevalence of neurological disorders in males: 272 (5.67%) was more than the females: 194 (3.94%). The highest prevalence was recorded in age <18 years 253 (6.40%).

Conclusions: Among the total positive cases, intellectual disability was most frequent: 337 (72.31%), followed by microcephaly: 64 (13.7%), neuromuscular dystrophy: 51 (10.90%) and Parkinson: 14 (3. 00%). These data suggested that intellectual disability is the most frequent among all neurological disorders. Therefore, further exploration of the remaining districts and genetic counseling is necessary.

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THE NEUROPROTECTIVE AND NEUROREGENERATIVE ACTIONS OF HYDROGEN SULPHIDE DONOR, INTRACEREBRAL MSCS, GINKO BILOBA AND KEFIR IN ATTENUATING NEUROPATHOLOGICAL HALLMARKS OF ALZHEIMER'S DISEASE INDUCED BY LIPOPOLYSACCHARIDE IN RATS

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Neurocognitive disorders have been characterized by being a devastating long term neurological disease with a huge social and economic impact. Alzheimer's disease (AD) is the most common type of dementia accompanied with decline in cognitive functions.

Objectives: **The main aim of this study is to suggest therapeutic protocol having the potentials for restoring and modifying neuropathological deposited hallmarks including both positive and negative lesions such as amyloid plaques, tau protein and synaptic loss.**

Materials & Methods: **Rats were divided into nine groups: (G1) control; (G2) rats received lipopolysaccharide (LPS) as a method of inducing non-genetically manipulated neuroinflammatory AD type; (G3) AD rats received NaHS; (G4) AD rats received mesenchymal stem cells (MSCs) intracerebrally; (G5) AD rats received MSCs+NaHS; (G6) AD rats received kefir+Ginko Biloba (GB); (G7) AD rats received MSCs+ kefir+GB; (G8) AD rats received NaHS+ kefir+GB; (G9) AD rats received MSCs+NaHS+kefir+GB.**

Results: **The induction of AD resulted in downregulation of cystathionine β-synthase enzyme (CBS) relative gene expression and glutathione (GSH) brain tissue level accompanied with overexpression in amyloid-β protein, mitogen-activated protein kinases (MAPK), tau protein, ACAT (Acyl-CoA cholesterol acyl transferase) relative gene expression and malondialdehyde (MDA) brain tissue level in addition to elevated caspase-3 serum activity level.**

Conclusion: **The present study clearly proved the beneficial role of NaHS as exogenous H2S donor in attenuating AD drawbacks mainly through CBS/H2S overexpression, enhancing the degradation of Aβ deposited plaques in addition to the ability of MSCs administrated locally to develop into different types of neural cells to compensate damaged ones. The role of administrating Kefir and GB in maintaining normal brain functions suggested being as a result of their synergistic antioxidant and antiapoptotic actions.**

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NOVEL PHENOXAZINONES AS POTENT AGONIST OF PPAR-α: DESIGN, SYNTHESIS, MOLECULAR DOCKING AND IN VIVO STUDIES

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Background: Treatment of dyslipidemia impacts directly on the cardiovascular health. The use of statin, a 3-hydroxy-3-methylglutaryl coenzyme, a reductase inhibitor for the treatment of dyslipidemia has been associated with dose limiting hepatoxicity, myototoxicity and tolerability due to myalgias thereby necessitating the synthesis of new drug candidates for the treatment of lipid disorder.

Methods: The reaction of appropriate benzenesulphonamide with substituted phenoxazinone in the presence of phenylboronic acid gave the targeted compounds. The molecular docking study were carried out using autodock tool against peroxisome proliferator activated receptor alpha. The *in vivo* lipid profiles were assayed using conventional methods. The kidney and liver function test were carried out to assess the effect of the derivatives on the organs. The LD50 of the most active derivatives were determined using mice.

Results: The targeted compounds were successfully synthesized in excellent yields and characterized using spectroscopic techniques. The results of the molecular docking experiment showed that they were good stimulant of peroxisome proliferator activated receptor alpha. Compound 9f showed activity at K_i of 2.8 nM and 12.6 kcal/mol of binding energy. All the compounds tested reduced triglyceride, total cholesterol, low density lipoprotein cholesterol and very low density lipoprotein cholesterol level in the mice model. Some of the reported compounds also increased high density lipoprotein cholesterol level in the mice. The compounds did not have appreciable effect on the kidney and liver of the mice used. The LD_{50} showed that the novel compounds have improved toxicity profile.

Conclusion: The synthesis of 15 new derivatives of carboxamides bearing phenoxazinone and sulphonamide were successful. The compounds possessed comparable activity to gemfibrozil. The reported compounds had better toxicity profile than gemfibrozil and could serve as a replacement for the statins and fibrate class of lipid agents.

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DNA/RNA-LIPID COMPLEXES IN NORME AND PATHOLOGY OF NERVE CELLS

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The ternary complexes (TC): DNA-phosphatidylcholine (PC) liposomes-divalent metal cations unlike lipoplexes have received attention lately. We proposed their involvement in the nuclear pore assembly. The formation of TC accompanied by the aggregation and fusion of PC liposomes was shown by freeze etching and cryo-TEM technique. At the same time, double helix of DNA unwinds in the region of liposomes fusion that enhances initiation of DNA transcription. Membrane vesicles forming the nuclear pores in a cell are analog of PC liposomes. In our last nuclear pore model, TC arises in the chromatin areas with three-stranded hybrids: DNA-small nuclear RNA (snRNA) and their interactions with two small membrane vesicles (~70 nm in diameter). The thermo stability of DNA/snRNA triple helix is considerably lower than the same sequence of double-stranded DNA. That specifies preferential attachment of triple-stranded hybrids to membrane vesicles. The triple helical hybrid unwinding during fusion of two membrane vesicles results in pre-pore formation: double-stranded DNA/snRNA hybrid and a single-stranded DNA (ssDNA) located on the outer diameter of fused big vesicle. This vesicle can form channel between membranes during interaction with double nuclear membrane. During this fusion of ssDNA and hybrid, DNA/snRNA shifts to pore annulus center. The ssDNA in pore annulus is the reason for the enhanced transcriptional activity of the genes neighboring nuclear pore. The number of pores in a nucleus specifies chromosome territory and number of chromosome loops. Nuclear pores serve as sites of the initiation of transcriptions in a cell, because ssDNA is the best site of transcription initiation. Binding of many toxic substances to ssDNA can prevent transcription initiation in area of nuclear pores. The beta-amyloid and tau protein aggregates may irreversibly binds to ss DNA and decrease transcription of many genes in a cell. Using TCs, we can find toxins inducing Alzheimer's disease and drugs for treatment and prevention of this and possible other neurodegenerative diseases.

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