

Centre for Physiology and Biochemical Research (CPBR),
International Stress and Behavior Society (ISBS), The Russian Society
for BioPsychiatry (RSBP), Ukrainian Society for Biological Psychiatry (USBP),
Institute of Experimental Medicine (IEM RAMS)

Conference Proceedings



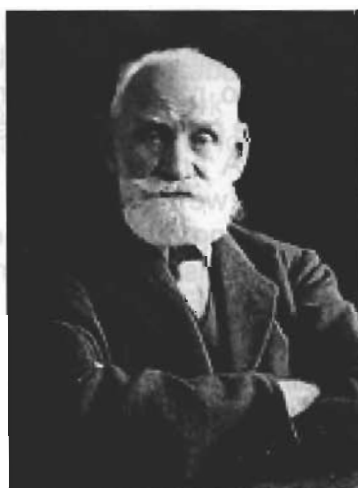
St-Petersburg, Russia
May 16-20, 2011

Centre for Physiology and Biochemical Research (CPBR),
International Stress and Behavior Society (ISBS), The Russian Society
for BioPsychiatry (RSBP), Ukrainian Society for Biological Psychiatry (USBP),
Institute of Experimental Medicine (IEM RAMS)

**15th Multidisciplinary International Conference
on Neuroscience and Biological Psychiatry**

“Stress and Behavior”

Dedicated to 120th anniversary of I. Pavlov's Department
of Physiology (Institute of Experimental Medicine, St Petersburg)



**St-Petersburg, Russia
May 16-20, 2011**

reactive oxygen species. Neuronal membrane phospholipids are especially vulnerable to damage, and injury leads to receptor-mediated signal transduction and, furthermore, information processing disorders. Indeed, there are difficulties in rating and interpreting data, due to inhomogeneous factors, including gender, race, age, nutritional, deployment factor (e.g., reservists or regular personnel), and different stressful military experiences in various Peace Support Missions (PSM). Our research aim was to assess PTSD and OS levels, and analyze their correlation in CIO.

METHODS: A total of 143 participants were assessed in this study: Latvian CIO, regular personnel, male Europeans, average age of 27.4, before and after the same PSM in Afghanistan were examined. The Latvian language "military" version of the PCL-M questionnaires were used for PTSD evaluation. Activity of AOE – Glutathione peroxidase (GPx) and intensity of lipid peroxidation – Malondialdehyde (MDA) as OS indicators in blood were determined. Data were processed using SPSS 15.0.

RESULTS: Before PSM, response rate (RR) 97.9% of study participants corresponded to PTSD diagnosis necessary criterions, with constituent 1.4%, GPx level decreased in 33.0%, and MDA level increased in 75.5% of samples. After PSM: RR 93.8%, PTSD 6.7%, GPx level decreased in 51.7%, MDA level increased in 80.0%. There was correlation between increase of OS and PTSD levels in CIO, further study required.

RESEARCH SUPPORT: Supported by the European Social Foundation co-financing: Project for Doctorants support in Riga Stradins University. The views expressed in this abstract do not reflect the official policy or position of the Latvian National Armed Forces, the Latvian government, or any of the institutions with which the authors are affiliated.

EXAMINATION OF THE TOXIC RAT MODELS OF PARKINSON'S DISEASE

IV Miliukhina, OS Veselkina, IN Abdurasulova, VN Mukhin, DE Korzhevsky, VM Klimenko, Institute for Experimental Medicine RAMS, St. Petersburg, Russia

INTRODUCTION: Parkinson's disease (PD) is a chronic pathology characterized by degeneration of dopaminergic neurons of the nigrostriatal system. Absence of valid animal models causes complication of diagnostic and treatment of this disease. Nowadays, there are two types of experimental animal models of Parkinson's disease: genetic and toxic. There are two variants of toxic models. One of them is inducible by injection of a toxin into the substantia nigra or nigrostriatal pathway of rat's brain and the other one is based on i.p. treatment with a toxin. Mostly, Parkinson's disease is caused by toxins. Therefore, toxic models of this disease are preferable. Two toxic impacts in rats cause symptoms of Parkinson's disease: single injection of 6-OHDA and protracted administration of rotenone. Unlike 6-OHDA effect of rotenone on dopaminergic neurons accompanied by intracellular accumulation of protein, that is immunoreactive for ubiquitin and alpha-synuclein. Valid model is considered to demonstrate basic symptoms of Parkinson's disease; which are hypokinesia, tremor, and postural disorders. This study was aimed to a comparative evaluation of effectiveness of modeling of the basic symptoms of Parkinson's disease in 6-OHDA and rotenone models at different stages after injection of neurotoxins.

METHODS: The study was carried out in male Wistar rats. 5 µl of 6-OHDA hydrobromide (Tocris Bioscience, UK) was injected into nigrostriatal pathway in accordance to the

stereotaxic coordinates: AP – 5,5 mm posterior to bregma, L – 2,0 mm lateral to the midline, V – 8,0 mm below the surface. Before this, 120 rats (270-290 g) were treated with 15 mg/kg Anafranil for nerve ending protection. 20 rats of the control group were injected with physiological saline solution. The rotenone model was replicated in rats weighing 300-490 grams. They were daily administrated intraperitoneal with rotenone for 35 days (2.75 mg/kg). Rotenone was dissolved in the mixture of DMSO:5mygliol (2:98). The control group of 10 rats was administrated with saline solution or vehicle. Neurological impairment and weight were assessed daily observing muscle tone, posture, motor activity, postural stability, salivation, width of palpebral fissures, and tremor. At 0, 7, 14, 21, 28, 35 days after neurotoxin administration behavior of rats was assessed with set of tests such as open field test, adjusting steps test, foot print test, beam-walking test, and rail-walking test. After behavioral tests morphological study was carried out. Selective detection of substantia nigra neurons was performed using monoclonal antibodies to tyrosine hydroxylase at the serial brain sections.

RESULTS: The basic symptoms of parkinsonism such as muscle rigidity, hypokinesia and postural disorders were observed 72 h after 6-OHDA administration and gradually became marked 7 days after rotenone administration. Most marked symptoms were observed 2 hours after a neurotoxin injection. Both models showed marked postural impairment, instability of posture, and muscle rigidity. But hypokinesia was more marked (sometimes even catalepsy by Morpurgo) in the rotenone model. One of the behavioral manifestations of one-side impairment of the nigrostriatal pathways with 6-OHDA was rotation (frequently spontaneous). Some rats had tremor in 6-OHDA model but not in the rotenone one. Salivation was a typical symptom of the rotenone model but not the 6-OHDA one. Both neurotoxins caused decrease of horizontal and vertical activity in the open field test. In the rail-walking test 6-OHDA administration caused more marked impairment than rotenone one. Most of rats were not be able to move on rails, and number of downfalls increased. In addition to that in rotenone model was also postural impairment, speed of rail walking decrease, but amount of downfalls and episodes of freezing was less than in the case of 6-OHDA administration. In the beam walking test was reduction of distance that rat was able to overcome without any mistakes. And this reduction associated with intensity of locomotor dysfunctions. 6-OHDA injection into substantia nigra caused ipsilateral neuron death in the same region of the brain. Immunohistochemical staining of tyrosine hydroxylase was not overt at the ipsilateral side unlike the contralateral one. The rotenone treatment also caused decrease in the number of neurons, and some animals had more loss of DA-neurons at the right side. Thus, 6-OHDA model of PD is effective in symptoms modeling of hypokinesia, postural disorders and postural instability. But this is a model of hemiparkinsonism and as such, is not entirely valid because Parkinson's disease is characterized by bilateral though asymmetrical symptoms. The advantage of the rotenone model is a gradual progression of symptoms of Parkinson's disease, which makes it possible to investigate the mechanisms of neurodegeneration DA-neurons in order to find early markers of neuropathological process and identify effective neuroprotectors.

RESEARCH SUPPORT: Supported by pharmaceutical company "Vertex Ltd".

significant increase of N-acetyl-aspartate concentration and regression of the lactate peak. Our data suggests that: 1) compared to HIV positive patients, PML could be a treatable disorder if correct and prompt imaging recognition in patients under immunosuppressive treatment is done; 2) the presence of huge lactate peak in white matter processes could be additional supporting information to include PML in differential diagnosis; and 3) Increase of N-acetyl-aspartate peak after clinical improvement additionally confirms the fact that reduction of this neuronal marker could be consistent not only with destruction of neurons but also with a reversible neuronal dysfunction.

DEPRESSION AND ITS ASSOCIATION TO NEGATIVE AUTOMATIC THOUGHTS AMONG UNDERGRADUATE MEDICAL AND HEALTH SCIENCES STUDENTS IN MALAYSIA

F Mukhtar, MH Mukhtar, LY Meng, CY Tong, MBM Pauzi, Z Ahmad, University Putra Malaysia, Malaysia

INTRODUCTION: Depression is currently one of the leading causes of disability among medical doctors and health related workers. However, few related studies have been reported on negative automatic thoughts and their relationship to symptoms of depression among medical and health science students in Malaysia. Thus, the objectives of this study are to determine the prevalence and relationship of negative automatic thoughts and depression.

METHODS: A cross sectional study was conducted among 281 first-year students from Faculty Medicine and Health Sciences (FMHS), University Putra Malaysia. Health sciences programs consist of four groups: Biomedical Science, Environmental and Occupational Health, Nutrition and Community Health and Dietetic. Two sets of validated questionnaires: Automatic Thoughts Questionnaire-Malay (ATQ-Malay) and Beck Depression Inventory-Malay (BDI-Malay) were administered. Data were analysed using SPSS (version 17.0), including descriptive, Pearson correlation and 2-sample T-test.

RESULTS: The prevalence for negative automatic thoughts and depression were higher among medical than health sciences students. Overall, there is a significant relationship between negative automatic thoughts and depression ($r = 0.622$). However, there was no effect of ethnicity on negative automatic thought, as well as no significant difference for gender or ethnicity on depression among medical and health sciences students.

RESEARCH SUPPORT: The study was not been funded by any authority or grant. It is part of research requirement by the university and the ethical approval is sought from the University Putra Malaysia with absolute voluntarily consent from the participants.

LEARNING IMPAIRMENT IN AMYLOID-BETA RAT MODEL OF ALZHEIMER DISEASE

VN Mukhin, VM Klimenko, Institute for Experimental Medicine, St. Petersburg, Russia

INTRODUCTION: Amyloid-beta peptide is a part of the physiological mechanism of memory. On the other hand, accumulation of this peptide in the form of plaques is a principal attribute of Alzheimer's disease. Moreover, it is considered that amyloid-beta is involved in pathogenesis of Alzheimer's disease. In line with this, injections of amyloid-

beta in different sites of the animal brain cause memory deficits. For this reason such injections could be animal models of Alzheimer disease for applied studies. However, there is not clarity of what kind of learning impairment should be observed depending on the kind of amyloid-beta peptide (fragment and solubility), injection site, and time between injection and testing. The aim of this study was to investigate behavioral markers of memory deficits in rats due to intracerebroventricular injection of the soluble form of amyloid-beta peptide.

METHODS: Three groups of adult Wistar rats (220 \pm 20 g) were used in this study: experimental group, saline solution control group and intact group. Amyloid-beta fragment 25-35 was dissolved in water and injected into the right brain ventricle (5 μ l, 1.2 μ l/min) of the experimental group. After 14 days behavioral testing of learning and memory started, in which rats were subjected to passive avoidance test and learning in the TSE PhenoMaster system.

RESULTS: Complex cognitive impairments in rats, including impairment of long-term memory and confusion-like loss of behavioral certainty were discovered. These results confirmed the pathogenetic significance of amyloid-beta 25-35, and made it possible to discriminate amyloid-beta rats with behavioral testing.

DO WOMEN WITH SHOULDER AND NECK PAIN PRESENT A DYSREGULATION OF THE HYPOTHALAMIC-PITUITARY-ADRENAL AXIS WITH INCREASED RISK OF DEVELOPING FIBROMYALGIA?

R Riva, PJ Mork, RH Westgaard, U Lundberg, Department of Psychology, Stockholm University, Sweden; Department of Human Movement Science, Department of Industrial Economics and Technology Management, Norwegian University of Science and Technology, Trondheim, Norway

INTRODUCTION: Shoulder and neck pain (SNP) and fibromyalgia syndrome (FMS), two musculoskeletal conditions of unknown pathogenesis, share common features in terms of altered neuroendocrine responses, pain and stress perception. However, the pain distribution in SNP is localized, whereas FMS is more widespread. To investigate whether SNP showed a dysregulated hypothalamic-pituitary-adrenal (HPA) axis and represent an intermediate stage between FMS and pain free conditions, we compared free salivary cortisol levels in women with SNP, FMS patients, and healthy controls (HC) in a controlled hospital-hotel setting, in which the participants' compliance was high and a number of potential confounders were analyzed.

METHODS: We recruited 22 women with SNP, 29 female FMS patients, and 29 female HC. Cortisol samples were collected to measure the cortisol waking response: upon waking, 30 and 60 minutes later. Questionnaires measuring pain levels, sleeping problems, perceived stress and personality were administered to the participants.

RESULTS: Compared with HC, women with SNP had a tendency towards higher cortisol levels, whereas FMS had lower cortisol levels. The potential confounders analyzed did not influence the results. Women with SNP and FMS patients reported more health complaints, pain, and perceived stress than the HC, but women with SNP were less affected than the FMS patients. Women with SNP showed a tendency towards an elevated HPA axis activity compared with HC. Overall, our data on circulating cortisol