The International Stress and Behavior Society (ISBS)
Guangdong Ocean University, China
Psychoneuroimmunology Research Society, USA
China Medical University, Taiwan

Program and Proceedings

THE JOINT MEETING:
9th International Regional Biomedical and Neuroscience STRESS AND BEHAVIOR (ISBS) Conference
6th Mind-Body Interface (MBI) International Symposium
1st Marine Drugs and Nutrition for Brain Diseases Symposium
Psychoneuroimmunology Research Society US-China Workshop

BRAIN AND IMMUNE INTERACTION: BEHAVIOR, STRESS, BRAIN DISEASES, DRUGS AND NUTRITION

October 27-29, 2016
Zhanjiang, China

ORGANIZING COMMITTEE

Conference Co-Chairs:

Prof. Allan V. Kalueff, PhD (USA, China and Russia), ISBS President
Prof. Cai Song, MD, PhD (Taiwan and Mainland China), LOC Chair
Prof. Kuan-Pin Su, MD, PhD (Taiwan), The 6th MBI Symposium Chair

International Program Committee:

Prof. Cai Song, MD, PhD (Taiwan and Mainland China), Co-Chair
Prof. Allan V. Kalueff, PhD (USA, China and Russia), Co-Chair
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Prof. Viktor M. Klimenko, PhD, MD (Russia)
Prof. Brian E Leonard, PhD (Ireland)
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Prof. Kuan-Pin Su, MD, PhD (Taiwan)
Prof. Bin Zhao, MD (China)

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Prof. Can Chen，MD (China), Co-Chair
Prof. Chao-Hua Zhang, PhD (China), Co-Chair
Prof. Bin Zhao, MD (China)
Prof. Peng-Zhi Hong, PhD (China),
Prof. Hong Wu Ji, PhD (China),
Prof. Xiao-Ming Qin, PhD (China)

CONFERENCE VENUE

Main campus of Guangdong Ocean University (GDOU)

Day 1. Thursday, October 27, 2016

Venue: Guangdong Ocean University Main Campus, Zhanjiang, China

08.00-17.00 **Registration**

09.00-09.30 **OPENING AND WELCOMING ADDRESSES**

- The Office of Zhanjiang City Major and the City Council
- Office of the President and CCP Committee of Guangdong Ocean University
- ISBS President Prof. AV Kalueff
- Conference Co-Chair and LOC Chair Prof. C Song
- MBI Symposium Chair Prof. K-P Su
- Psychoneuroimmunology Research Society and PNIRS-China Chair Prof. CL Coe

09.30-10.10 **CONFERENCE OPENING PLENARY LECTURE: INFLAMMATION, BRAIN GLUCOSE METABOLISM AND DEPRESSION: IS THERE A LINK?** BE Leonard, National University of Ireland, Galway, Ireland

10.10-10.40 **SPECIAL KEYNOTE LECTURE:** JR Hibbeln, Section of Nutritional Neuroscience, Laboratory of Membrane Biochemistry and Biophysics, National Institute on Alcohol Abuse and Alcoholism (NIAAAA), NIH, Rockville, MD, USA

OMEGA-3/ OMEGA-6 FATTY ACID IMBALANCES IN ADDICTIONS: FISH CONSUMPTION AND RISK OF SMOKING IN PREGNANCY. Joseph R. Hibbeln [1], John Paul SanGiovanni[1], John M. Davis[2], Rachel V. Gow[1] Jon Heron [3].

1. Section of Section of Nutritional Neurosciences, LMBB, NIAAA, Bethesda, MD 20892.

2. University of Illinois at Chicago, Chicago, Illinois, USA

3. School of Social and Community Medicine, University of Bristol, UK

10.40-11.20 **ISBS SPECIAL PLENARY LECTURE 1: STRATIFICATION AND OUTCOME PREDICTION OF DEPRESSED PATIENTS USING PANELS OF INFLAMMATION AND KYNURENINE BIOMARKERS.** A Halaris, Loyola University Chicago Stritch School of Medicine, Chicago, IL, USA

11.20-11.50 **COFFEE BREAK**

11.50-12.25 **ISBS/ISBN SPECIAL PLENARY LECTURE 2: CONVERGENT PATHWAYS UNDERLYING GENE-ENVIRONMENT INTERACTION IN SCHIZOPHRENIA.** M Pletnikov, Johns Hopkins University School of Medicine, Baltimore, MD, USA

12.25-13.00 **ISBS SPECIAL PLENARY LECTURE 3: EMERGING PHARMACOLOGY OF TRACE AMINE-ASSOCIATED RECEPTOR 1 (TAAR1).** RR Gainetdinov, Institute of Translational Biomedicine, St. Petersburg State University, St. Petersburg, Skolkovo Institute of Science and Technology (Skoltech), Skolkovo, Moscow, Russia

13.00-14.00 **LUNCH BREAK**

14.00-17.00 **SYMPOSIUM I: PSYCHONEUROIMMUNOLOGY RESEARCH SOCIETY PNIRS-CHINA WORKSHOP. Chairs:** CL Coe, KW Kelley (USA)

14.00-14.20 **THE HISTORY OF THE PSYCHONEUROIMMUNOLOGY RESEARCH SOCIETY (PNIRS) AND THE EMERGENCE OF PNI RESEARCH IN CHINA.** KW Kelley, University of Illinois-Urbana, IL, USA

14.20-14.45 **THE SIGNIFICANCE OF THE MICROBIOME AND GUT/BRAIN AXIS FOR DEVELOPMENTAL HEALTH AND PSYCHONEUROIMMUNOLOGY RESEARCH.** CL Coe, University of Wisconsin-Madison, Madison, WI, USA

14.45-15.10 **OBESITY, BRAIN INFLAMMATION AND COGNITION: AN ESSENTIAL ROLE FOR MICROGLIA IN PERINATAL PROGRAMMING OF THE OBESE BRAIN**. SJ Spencer, School of Health and Biomedical Sciences, RMIT University, Melbourne, Australia

15.10-15.35 **HMGB1 AND ITS REDOX STATE PLAYS A KEY ROLE ON THE INDUCTION OF DEPRESSIVE-LIKE BEHAVIOR.** Y-J Lian, H Gong, T-Y Wu, Y-X Wang, Laboratory of Stress Medicine, Department of Psychology and Mental Health, Second Military Medical University, Shanghai, China

15.35-16.00 **WHAT YOUR MONOCYTES SAY ABOUT YOUR MIND AND BODY AND WHY WE SHOULD LISTEN: IMPAIRED β2-ARs AND GRs MAY EXPLAIN LOW-GRADE INFLAMMATION, CARDIOVASCULAR DISEASE RISK AND DEPRESSIVE MOOD.** S Hong, S Dimitrov, T Cheng, F Shaikh, JM Green, C Pruitt and N Beg**,** Departments of Psychiatry, Family Medicine and Public Health, University of California San Diego, CA, USA

16.00-16.25 **ANGIOGENIC AND IMMUNE SIGNATURES IN PSYCHOSIS RISK AND EARLY PSYCHOSES.** JK Yao, PL Lizano and MS Keshavan, VA Pittsburgh Healthcare System, Medical Research Service, Department of Psychiatry, University of Pittsburgh School of Medicine, Pittsburgh, PA, Department of Psychiatry, Beth Israel Deaconess Medical Center, Boston, MA, USA

16.25-16.50 **COFFEE BREAK**

16.50-18.00 **SYMPOSIUM II: ZOFIA ZUKOWSKA SYMPOSIUM ON TRANSLATIONAL NEUROSCIENCE OF STRESS. Chairs:** AV Kalueff (USA, Russia, China), R Gainetdinov (Russia), M Pletnikov (USA)

16.50-17.10 **THE MILD ENCEPHALITIS HYPOTHESIS OF SEVERE PSYCHIATRIC DISORDERS.** K Bechter, Ulm University, Ulm, Germany

17.10-17.30 **MULTIFACETED CONTRIBUTIONS BY DIFFERENT REGIONS OF THE ORBITOFRONTAL AND MEDIAL PREFRONTAL CORTEX TO PROBABILISTIC REVERSAL LEARNING.** GL Dalton, NY Wang, AG Phillips, SB Floresco, University of British Columbia, Vancouver, Canada

17.30-17.45 **PLASMINOGEN ACTIVATOR INHIBITOR-1 (PAI-1) 4G/5G GENE POLYMORPHISM IS ASSOCIATED WITH THE CARDIOVASCULAR DISEASE OVERREPRESENTED IN SCHIZOPHRENIA.** HF Yang, Psychiatry Department, Affiliated Hospital of Guangdong Medical University, Zhanjiang, China

17.45-18.00 **THE EFFECTS OF G-PROTEIN-COUPLED RECEPTOR KINASE 5 POLYMORPHISMS ON ALZHEIMER'S DISEASE.** M Yin, J Zhao, Y Cai, L Cui, B Zhao, Guangdong Key Laboratory of Age-related Cardiac and Cerebral Diseases, Affiliated Hospital of Guangdong Medical University, Zhanjiang, Guangdong Province, China

19.00-21.00 ZHANJIANG CITY COUNCIL-SPONSORED WELCOMING RECEPTION AT HAIBING HOTEL

Day 2. Friday, October 28, 2016

Venue: Guangdong Ocean University Main Campus, Zhanjiang, China

08.00-17.00 **Registration**

09.00-09.40 **WORKSHOP: HOW TO PUBLISH YOUR BEST SCIENCE IN RECOGNIZED INTERNATIONAL JOURNALS: INSIGHTS FROM THE EDITOR-IN-CHIEF OF "BRAIN, BEHAVIOR, AND IMMUNITY".** KW Kelley, University of Illinois-Urbana, IL, USA

09.40-10.15 **ISBS SPECIAL PLENARY LECTURE 4: AGE-RELATED CHANGES IN LEARNING AND MEMORY IN MOUSE MODELS OF ALZHEIMER'S DISEASE.** RE Brown, Department of Psychology and Neuroscience, Dalhousie University, Halifax, Nova Scotia, Canada

10.15-10.50 **ISBS SPECIAL PLENARY LECTURE 5: EFFECT OF PRO-INFLAMMATORY CYTOKINES ON THE FORMATION OF THE COGNITIVE FUNCTIONS IN RATS IN EARLY POSTNATAL ONTOGENESIS.** VM Klimenko, OE Zubareva, Federal State Research Institution "Institute for Experimental Medicine", St. Petersburg, Russia

10.50-11.20 **OMEGA-3 ON QUALITY OF LIFE AND DURATION OF LIFE: A FEW LATEST EXAMPLES.** W Zhang, DSM Nutritional Products, Human Nutrition & Health, China

11.20-11.40 **COFFEE BREAK**

11.40-16.30 **SYMPOSIUM III. THE 6TH MIND-BODY INTERFACE (MBI) INTERNATIONAL SYMPOSIUM. Chair:** K-P Su (Taiwan)

11.40-12.20 **PERSONALIZED MEDICINE WITH OMEGA-3 FATTY ACIDS FOR DEPRESSION.** KP Su, Graduate Institute of Neural and Cognitive Sciences, Mind-Body Interface Laboratory (MBI-Lab), China Medical University and Hospital, Taichung, Taiwan

12.20-12.50 **EXPOSURE TO A MATERNAL N-3 FATTY ACID-DEFICIENT DIET DURING THE BRAIN DEVELOPMENT ENHANCE THE ACTIVITY AND DYSREGULATION OF HYPOTHALAMIC-PITUITARY-ADRENAL AXIS RESPONSES TO STRESS IN RAT OFFSPRING LATER IN LIFE.** YJ Hsieh and HM Su, Physiology Department, College of Medicine, National Taiwan University, Taipei, Taiwan

12.50-13.20 **DISTINGUISHING NEUROPROTECTIVE EFFECTS OF DIFFERENT OMEGA (N)-3 FATTY ACIDS ON LPS-INDUCED BEHAVIORAL CHANGES AND MICROGLIA-MEDIATED NEUROINFLAMMATION.** YY Mei, S Gaikwad and C Song, Neuroimmunology and Behavior Lab, China Medical University Hospital, Taichung, Taiwan; Research Institute for Marine Drugs and Nutrition, Guangdong Ocean University, Zhanjiang, China

13.20-13.50 **EFFECTS OF N-3 POLYUNSATURATED FATTY ACID SUPPLEMENTATION ON COGNITIVE FUNCTION IN PATIENTS WITH LATE-LIFE DEPRESSION - A PRELIMINARY STUDY.** CC Chiu, CJ Chang, WC Chiu, IW Sun, SI Liu, ML Lu, CH Chen, SY Huang, Taipei City Psychiatric Center, Cathay General Hospital, Mackay Memorial Hospital Taipei, Taipei Medical University, Taipei, Taiwan

13.50-15.00 **LUNCH BREAK**

15.00-15.30 **ASSOCIATION BETWEEN A FUNCTIONAL VARIANT IN A GENE ENCODING AN ENZYME METABLLIZING DOPAMINE (CATECHOL-O-METHYLTRANSFERASE) AND POST CANNABIS PSYCHOSIS: A REPLICATION IN CANADIAN CAUCASIANS.** KJ Aitchison, R Lodhi, Y Wang, C Crocker, A Loverock, A Dimitrijevic, H Ren, D Bugbee, G Macintyre, P Tibbo, SE Purdon, Departments of Psychiatry and Medical Genetics, University of Alberta, Edmonton Early Psychosis Intervention Clinic, Department of Psychiatry, Dalhousie University, Nova Scotia Early Psychosis Program, Halifax, NS, Neuropsychology Department, Alberta Hospital Edmonton, Department of Medicine, University of Alberta, Edmonton, AB, Canada

15.30-16.00 **FATTY ACIDS THAT ARE PRESENT AND NOT PRESENT IN THE BRAIN: IMPLICATIONS FOR TARGETING THE BRAIN IN PSYCHIATRY.** R Bazinet, University of Toronto, Toronto, Canada

16.00-16.30 **ANTIDEPRESSANT-LIKE EFFECTS OF WATER EXTRACT OF GASTRODIA ELATA BLUME IN RATS EXPOSED TO UNPREDICTABLE CHRONIC MILD STRESS VIA MODULATION OF MONOAMINE REGULATORY PATHWAYS.** YE Lin, SH Lin, WC Chen, CT Ho, YS Lai, S Panyod, and LY Sheen, China Medical College, Taichung, Taiwan

16.30-16.50 **COFFEE BREAK**

16.50-18.00 **SYMPOSIUM IV. LAPIN SYMPOSIUM ON TRANSLATIONAL BIOMEDICINE. Chairs:** VM Klimenko (Russia), BE Leonard (Ireland), RE Brown (Canada)

16.50-17.10 **THE EFFECT OF POLYMORPHISM OF 5-HTTLPR GENE ON AFFECTIVE TEMPERAMENT, DEPRESSION AND COGNITION IN OBESITY.** A Borkowska, M Bieliński, Department of Clinical Neuropsychology, Collegium Medicum of Nicolaus Copernicus University Torun, Bydgoszcz, Poland

17.10-17.30 **EVALUATION OF COGNITIVE DEFICITS IN A MOUSE GENETIC MODEL OF PARKINSON'S DISEASE WITH OVEREXPRESSION OF A-SYNUCLEIN.** TG Amstislavskaya, YJ Ho, MA Tikhonova, Scientific Research Institute of Physiology and Basic Medicine, Novosibirsk, Russia; Department of Psychology, Chung Shan Medical University, Taipei, Taiwan

17.30-17.45 **A NOVEL, MULTI-TARGET NATURAL DRUG CANDIDATE, MATRINE, IMPROVES COGNITIVE DEFICITS IN ALZHEIMER'S DISEASE.** L Cui, Y Cai, W Cheng, G Liu, J Zhao, H Cao, H Tao, Y Wang, M Yin, T Liu, Y Liu, P Huang, Z Liu, K Li, B Zhao, Guangdong Key Laboratory of Age-Related Cardiac and Cerebral Diseases, Affiliated Hospital of Guangdong Medical University, Zhanjiang, China

17.45-18.00 **CO-TARGETING MEK AND PIM-1 FOR THE INTERVENTION OF MALIGNANT TUMORS.** Y Cui, Shantou University Medical College Cancer Hospital, Shantou, Guangdong, China

Day 3. Saturday, October 29, 2016

Venue: Guangdong Ocean University Main Campus, Zhanjiang, China

08.00-13.00 **Registration**

09.00-09.40 **GDOU SPECIAL LECTURE 6: TBN.** AG Phillips, University of British Columbia, Vancouver, BC, Canada

09.40-10.10 **RIMND SPECIAL LECTURE 7: NEUROINFLAMMATORY AND NEUROPROTECTIVE ROLES OF GLIA CELLS IN CELLULAR MODELS OF ALZHEIMER'S AND PARKINSON'S DISEASES.** C Song, YS Wu, XF Wang, YY Thao, ZL Peng, YP Zhang, AV Kalueff, KP Su, Research Institute for Marine Drugs and Nutrition, Guangdong Ocean University, Zhanjiang, China

10.10-16.10 **SYMPOSIUM V: INTERNATIONAL SYMPOSIUM ON SCREENING AND DEVELOPMENT OF MARINE AND RELATED DRUGS. Chair:** C Song (China)

10.10-10.30 **CHEMICAL ANALYSIS FROM ECOLOGICAL NICHES TO MICROBIAL BIOACTIVE AGENTS AS BIOCHEMICAL TOOLS.** M Konnerth, NA Schilling, A Zipperer, F Zubeil, D Weisbrod, F Surup, A Peschel, B Krismer, S Grond, Institute of Organic Chemistry, Interfaculty Institute of Microbiology and Infection Medicine, Eberhard Karls Universität Tübingen, Tübingen, Helmholtz Center for Infection Research (HZI), Department of Microbial Drugs, Braunschweig, Germany

10.30-11.00 **CHEMICAL DIVERSITY OF BIOACTIVE NATURAL PRODUCTS FROM MARINE-DERIVED ENDOPHYTIC FUNGI.** B Wang, Laboratory of Marine Biology and Biotechnology, Qingdao National Laboratory for Marine Science and Technology, Key Laboratory of Experimental Marine Biology, Institute of Oceanology, Chinese Academy of Sciences, Qingdao, China

11.00-11.15 **SHRIMP EXTRACTED BY-PRODUCTS PREVENTS SH-SY5Y CELLS FROM NEUROTOXICITY INDUED BY Aβ 25-35.** YP Zhang, S Gu, J Gagnon, L Ma, P Zhang, RE Brown, C Song, Research Institute for Marine Drugs and Nutrition, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang, China

11.15-11.30 **THREE NOVEL PEPTIDES FROM ANTARCTIC KRILL (EUPHAUSIA SUPERBA) PROTEIN HYDROLYSATE AS A DIPEPTIDYL PEPTIDASE IV INHIBITOR IMPROVE GLYCEMIC CONTROL IN DIABETIC ZEBRAFISH.** W Ji, C Zhang, H Ji, AV Kalueff, W Su, College of Food Science and Technology, Guangdong Ocean University, Guangdong Provincial Key Laboratory of Aquatic Products Processing and Safety, Key Laboratory of Advanced Processing of Aquatic Products of Guangdong Higher Education Institution, Zhanjiang, China

11.30-11.50 **COFFEE BREAK**

11.50-12.20 **RIMDN SPECIAL LECTURE 8: ANTI-INFLAMMATORY TRITERPEN PLANT EXTRACTS AND COGNITIVE FUNCTION IN OBESITY AND DIABETES.** XF Huang, University of Wollongong, New South Wales, Australia

12.20-12.35 ***CLOSTRIDIUM BUTYRICUM* TO-A ALLEVIATE ANXIETY IN APP/PS1 ALZHEIMER'S DISEASE MICE.** C Jiang, K Li, X Peng, D Chen, L Hu, S Zhu, L Cui, Z Liu, B Zhao, Guangdong Key Laboratory of Age-Related Cardiac and Cerebral Diseases, Affiliated Hospital of Guangdong Medical University, Zhanjiang, China

12.35-12.50 **THE IMPACT OF HIGH SALT DIET ON GUT MICROBIOTA AND COGNITIVE FUNCTION IN C57BL/6J MICE.** X Peng, K Li, C Jiang, D Chen, L Hu, S Zhu, L Cui, Z Liu, B Zhao, Guangdong Key Laboratory of Age-Related Cardiac and Cerebral Diseases, Affiliated Hospital of Guangdong Medical University, Zhanjiang, China

12.50-13.05 **THE PROTECTIVE EFFECT OF FECAL MICROBIOTA TRANSPLANTATION ON EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS MICE.** K Li, C Jiang, X Peng, D Chen, L Hu, S Zhu, L Cui, Z Liu, B Zhao, Guangdong Key Laboratory of Age-Related Cardiac and Cerebral Diseases, Affiliated Hospital of Guangdong Medical University, Zhanjiang, China

13.05-13.20 **INFLAMMATORY ENDOTHELIAL MICROVESICLE MODULATE THE PROLIFERATION, MIGRATION AND APOTOSIS OF BRAIN VASCULAR SMOOTH MUSCLE CELLS VIA THEIR CARRIED RNAS ASSOCIATED WITH THE MEK1/2/ERK1/2 AND CASPASE-3/BCL-2 PATHWAYS.** Q Pan, X Liao, Y Wang, Y Chen, B Zhao, X Ma, Guangdong Key Laboratory of Age-Related Cardiac and Cerebral Diseases, Institute of Neurology, Affiliated Hospital of Guangdong Medical University, Zhanjiang, China; Department of Pharmacology and Toxicology, Boonshoft School of Medicine, Wright State University, Dayton, OH, USA

13.20-13.35 **ZINC MODULATION ON GLYCINE RECEPTOR ALPHA 1 SUBUNIT IS ASSOCIATED WITH HUMAN HYPEREKPLEXIA.** DC Wu, University of British Columbia, Vancouver, Canada

13.35-13.50 **THE STUDY ON ANTI-ALZHEIMER RELATED BIOACTIVE CONSTITUENTS OF MARINE FUNGI.** Y Zhang, H Bao, Y Nie, Research Institute for Marine Drugs and Nutrition, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang, China

13.50-15.00 **LUNCH BREAK**

15.00-15.15 **GRK5 DEFICIENCY ACCELERATED THE HYPER-PHOSPHORYLATION OF TAU THROUGH GSK-3B ACTIVATION IN THE EARLY STAGE OF ALZHEIMER'S DISEASE.** J Zhao, M Yin, Y Cai, L Cui, B Zhao**,** Guangdong Key Laboratory of Age-Related Cardiac and Cerebral Diseases, Affiliated Hospital of Guangdong Medical University, Zhanjiang, China

15.15-15.30 **THE EFFECTS OF RETRIEVAL-EXTINCTION PARADIGM DURING SLEEP ON ALCOHOL CRAVING.** R Tao, J Zhu, M Ma, C Wang, H Sun, Peking University Sixth Hospital, Peking University Institute of Mental Health, Key Laboratory of Mental Health, Ministry of Health, Peking University, Beijing, Department of Psychiatry, the Second Affiliated Hospital of Xinxiang Medical University, Xinxiang, China

15.30-16.10 MODERATED POSTER SESSION
AND COFFEE BREAK

SEX DIFFERENCES IN THE CHRONIC UNPREDICTABLE MILD STRESS RESPONSE IN SD RATS. HY Wang, C Zhang, HY Xue, YY Li, BP Liu, KW Li, YP Zhang, ZL Peng, C Song, Research Institute for Marine Drugs and Nutrition, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang , Guangdong, China

EFFECT OF CHRONIC NEUROINFLAMMATION INDUCED BY INTERLEUKIN-1β ON PERIPHERAL IMMUNITY. HS Xue, YP Zhang，C Zhang, YY Li, BP Liu, C Song, Research Institute for Marine Drugs and Nutrition, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang, Guangdong, China

EFFTS OF EPA ON CHRONIC STRESS-INDUCED CHANGE IN THE FUNCTION OF PERIPHERAL MACROPHAGE AND THE UNSTURATED FATTY ACIDS COMPOSITION OF CELLULAR MEMBRANE. YY Li, C Zhang, HS Xue, BP Liu, CYP Zhang, C Song, Research Institute for Marine Drugs and Nutrition, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang, Guangdong, China

FAT-1 TRANSGENIC MICE ALLEVIATE DEPRESSION-LIKE BEHAVIOR AFTER CENTRAL ADMINISTRATION OF LIPOPOLYSACCHARIDE. BP Liu, KW Li, HS Xue, YY Li, YP Zhang, C Song, Research Institute for Marine Drugs and Nutrition, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang , Guangdong, China

EFFECT OF GU HONG INJECTION ON THE FUNCTION OF CEREBRAL CORTEX MITOCHONDRIA IN RATS WITH CEREBRAL ISCHEMIA. L Feng, M Wang, J Liu, N Yang, Y Liu, P Zuo, Department of Pharmacology, Institute of Basic Medical Sciences, Peiking Union Medical School, Chinese Academy of Medical Sciences, Beijing, China

ZEBRAFISH ON BATH SALTS: NOVEL INSIGHTS FOR DRUG ABUSE RESEARCH. AV Kalueff, TO Kolesnikova, SL Khatsko, YuYu Morzherin, Ural Federal University, Ekaterinburg, Institute of Translational Medicine, St. Petersburg State University, St. Petersburg, Russia; Research Institute for Marine Drugs and Nutrition, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang, China; ZENEREI Research Center and the International Zebrafish Neuroscience Research Consortium (ZNRC), New Orleans, LA, USA

AFTERNOON TALKS

16.10-16.30 THE GGA FAMILY PROTEINS MODULATE THE CELL SURFACE TRANSPORT OF ALPHA2B-ADRENERGIC RECEPTOR. M Zhang, G Wu, Department of Pharmacology and Toxicology, Medical College of Georgia, Georgia Regents University, Augusta, GA, USA

16.30-16.50 EFFECT OF TOTAL FLAVONOIDS EXTRACT FROM *EUPATORIUM ODORATUM* ON IMMUNITY AND INTESTINAL FLORA IN BROILER. JJ Chen, XN Wang, HY Lin, FH Nie, JQ Zheng, DL Gong, YP Yu, XH Ju, Y Ma, QH Zhang, Guangdong Ocean University, Zhanjiang, China

16.50-17.10 EFFECT OF POLYSACCHARIDES FROM *CORDYCEPS SINENSIS* CAMT 63341 ON SOME BEHAVIOR AND INMMUNITY ORGAN INDEX OF MICE. D Xu, S Li, J Hao, Guangdong Provincial Key Laboratory of Aquatic Products Processing and Safety, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang, China

17.10-17.30 **ISBS PRESIDENTIAL CLOSING LECTURE 8: ZEBRAFISH MODELS OF NEUROPSYCHIATRIC AND COMORBID METABOLIC AND IMMUNE DISORDERS.** AV Kalueff, Research Institute for Marine Drugs and Nutrition, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang, China; Institute of Translational Medicine, St. Petersburg State University, St. Petersburg; Ural Federal University, Ekaterinburg, Russia; ZENEREI Research Center and the International Zebrafish Neuroscience Research Consortium (ZNRC), New Orleans, LA, USA

17.30-17.45 **ISBS OUTREACH COLLABORATION: AN ARTIST'S LOOK AT MENTAL DISORDERS.** D Raytchev, Daniela Raytchev Art, London, UK

17.45-18.00 **OFFICIAL CLOSING OF THE CONFERENCE**

Day 4. Sunday, October 30, 2016

10.00-15.00 **POST-CONFERENCE TOUR TO THE VOLCANO LAKE NATIONAL PARK**

Meeting point: Central gates of Guangdong Ocean University Main Campus, Zhanjiang

ABSTRACTS

Day 1. Thursday, October 27, 2016

Venue: Guangdong Ocean University Main Campus, Zhanjiang, China

OPENING AND WELCOMING ADDRESSES

- The Office of Zhanjiang City Major and the City Council
- Office of the President and CCP Committee of Guangdong Ocean University
- ISBS President Prof. AV Kalueff
- Conference Co-Chair and LOC Chair Prof. C Song
- MBI Symposium Chair Prof. K-P Su
- Psychoneuroimmunology Research Society and PNIRS-China Chair Prof. CL Coe

CONFERENCE OPENING PLENARY LECTURE: INFLAMMATION, BRAIN GLUCOSE METABOLISM AND DEPRESSION: IS THERE A LINK? BE Leonard, National University of Ireland, Galway, Ireland

The role of the endocrine and immune systems in the pathophysiology of depression, and the link to physical ill-health which result from the metabolic syndrome, has been a subject of considerable interest in recent years. Chronic low grade inflammation, a frequent feature of major depression, contributes to changes in glucose and lipid metabolism which are associated with the increase in type-2 diabetes and heart disease and which may also contribute to increased neurodegeneration which frequently occurs in the elderly depressed patient. An important mechanism which acts as a prelude to these metabolic sequels arises from the decrease in insulin receptor sensitivity and the subsequent reduction in the transport of glucose to neurons and neuroglia. Mitochondrial dysfunction is also an important outcome of chronic major depression which results in a reduction in the synthesis of ATP and other high energy substrates. These are essential for the maintenance of neuronal function. In addition, the synthesis of an important cofactor for the electron transport system in mitochondria, NAD+, from the tryptophan – kynurenine pathway is decreased further contributing to the energy deficiency in neurons. Ultimately the metabolic changes which arise from inflammation and cortisolaemia, contribute to the increased neuronal apoptosis and degeneration which characterise chronic major depression.

SPECIAL KEYNOTE LECTURE: JR Hibbeln, Section of Nutritional Neuroscience, Laboratory of Membrane Biochemistry and Biophysics, National Institute on Alcohol Abuse and Alcoholism (NIAAAA), NIH, Rockville, MD, USA

OMEGA-3/ OMEGA-6 FATTY ACID IMBALANCES IN ADDICTIONS: FISH CONSUMPTION AND RISK OF SMOKING IN PREGNANCY. Joseph R. Hibbeln [1], John Paul SanGiovanni[1], John M. Davis[2], Rachel V. Gow[1] Jon Heron [3].

1. Section of Section of Nutritional Neurosciences, LMBB, NIAAA, Bethesda, MD 20892.

2. University of Illinois at Chicago, Chicago, Illinois, USA

3. School of Social and Community Medicine, University of Bristol, UK

Our central hypothesis is that dietary excesses of omega-6 and deficiencies of omega-3 fats change the tissue status of bioactive lipids which, in turn, causes disturbances to the homeostasis of multiple integrated biological processes underlying addictive and affective illnesses. Our proposed cascade links dietary imbalances in omega-3 and omega-6 fats to excessive immunological reactivity and to neurotransmitter abnormalities relevant to addiction vulnerabilities and depressive disorders. Peripheral macrophage and microglial activation of the TLR receptor activates NFkB nuclear hormone induced transcription of a coordinated inflammatory cascade reaction of Il-1B, IL-6, TNF cytokines, chemokines and cyclooxygenase (COX 1-2) transcription. Dietary omega-6 lipids elevate tissue levels of arachidonic acid (AA) elevating metabolism by COX 1-2 to prostaglandin E2 (PGE2). PGE2 binds EP-1 receptors inducing indolamine 2-3 dioxygenase in serotonergic (5-HT) neurons degrading 5-HT to kynurenic and quinolenic acids lowering serotonin levels and inducing depression. In dopaminergic (DA) neurons, PGE2 binds EP-1 receptors reducing firing and initiating apoptosis. DA levels are reduced by 50% in omega-3 deficient diets. Depletion of DA and reward responses are characteristic of chronic addiction. In the hypothalamus, PGE2 binds to EP-1 receptors and IL-6 activates transcription of corticotrophin releasing hormone (CRH) mRNA

three-fold. CRH is an anxiogenic peptide. Additionally, excessive CRH stimulates hyperactive stress responses including the sympathetic nervous system and the hypothalamic-pituitary-adrenal (HPA) axis. Thus, the central neurotransmitters systems implicated in addictions are all adversely affected by dietary excesses of omega-6 and deficiencies of omega-3 fats. Thus, we posited that lower fish consumption would be associated with greater risks perinatal smoking among n=9,640 mothers enrolled in the Avon Longitudinal Study of Parents and Children (ALSPAC). We examined relationships between self-reported prenatal dietary intakes of omega-3 HUFA-rich foods (fish and shellfish) and maternal smoking including cessation and the number of cigarettes smoked per day in univariable and multivariable regression models. Both before and during pregnancy, there was evidence ($p < 0.001$) of a large protective associations. Compared to never/rare consumption, some fish consumption (0-340g/wk) was associated with an approximately 25% lower risk of smoking, and high fish consumption (340g+/wk) was associated with a 50% lower risk, in unadjusted analyses. Following adjustment for confounders there remained fairly strong evidence (p values in range 0.011 – 0.001) of a moderate beneficial effect of fish consumption, in particular for mothers consuming 340g or more per week where risk of smoking was approximately 20% lower. These observations suggest that greater fish or n-3 HUFA consumption should be evaluated to reduce smoking in pregnancy in randomized trials.

ISBS SPECIAL PLENARY LECTURE 1: STRATIFICATION AND OUTCOME PREDICTION OF DEPRESSED PATIENTS USING PANELS OF INFLAMMATION AND KYNURENINE BIOMARKERS. A Halaris, Loyola University Chicago Stritch School of Medicine, Chicago, IL, USA

INTRODUCTION: Depressive disorders are highly heterogeneous disorders. This heterogeneity contributes greatly to diagnostic inaccuracies, a very low percentage of remitters after the initial antidepressant drug trial, and poor predictability of suicidality, treatment response and requirement for prolonged maintenance treatment. **METHODS:** Males and females (20-65 years of age) meeting DSM-IV criteria for primary major depressive disorder (MDD) and were physically healthy were considered. Thirty patients met inclusion criteria and were enrolled. After baseline assessments they were started on escitalopram on an open label basis. Healthy control subjects were enrolled throughout the period the MDD subjects were recruited. Rating scales were used to assess depression, state and trait anxiety, state and trait anger, stress perception, somatosensory amplification, quality of life. Inflammation biomarkers and growth factors were measured by "Evidence InvestigatorTM" (Randox Technologies). Pentraxin-3 and hsCRP were measured by ELISA. Tryptophan and kynurenines were measured by HPLC. Quinolinic acid was analyzed using GCMS. SPSS version 20 was used for the analyses. Distributions for all biomarkers were analyzed for normality, skewedness, kurtosis, and homoscedasticity of the residuals prior to analysis. Biomarkers found to have skewed distributions were analyzed through non-parametric methods. **RESULTS:** We observed correlations between specific symptoms of MDD and some of the biomarkers studied. Depressed mood correlated significantly with IFNγ while weight loss correlated significantly with TNFα levels. QUIN correlated significantly with guilt and QUIN/3HK correlated significantly with guilt and psychomotor agitation. Pentaxin-3 correlated with anxiety. VEGF and hsCRP predicted treatment outcome. **DISCUSSION:** To our knowledge, this is the first report that in MDD patients correlations are observed between specific depressive symptom scores and inflammation, growth factors and kynurenine metabolites. These results once confirmed in a larger cohort can lead to patient stratification and predict treatment outcome. **RESEARCH SUPPORT:** Intramural grant support

ISBS/ISBN SPECIAL PLENARY LECTURE 2: CONVERGENT PATHWAYS UNDERLYING GENE-ENVIRONMENT INTERACTION IN SCHIZOPHRENIA. M Pletnikov, Johns Hopkins University School of Medicine, Baltimore, MD, USA

Multiple adverse environmental factors contribute to the pathogenesis of schizophrenia via complex interactions with genetic risk factors in susceptible individuals. We have been modeling complex etiologically relevant gene-environment interactions in mice that selectively express mutant Disrupted-In-Schizophrenia 1 (DISC1) in neurons or astrocytes and are exposed to prenatal immune activation or environmental toxins, or cannabis during adolescence. We evaluated the neurobehavioral, histopathological, neuroimmune, and molecular phenotypes in adult mice. We found that mice with neuronal expression of mutant DISC1 developed the brain and behavioral alterations consistent with aspects of mood disorders following prenatal immune activation, and schizophrenia-like abnormalities

treatable with D-serine treatment following developmental exposure to low doses of Pb2+. Chronic adolescent cannabis (tetrahydrocannabinol) exposure of mice expressing mutant DISC1 in neurons exacerbated deficient fear conditioning and synergistically decreased c-Fos expression induced by cue-dependent fear memory retrieval. The similar adolescent cannabis exposure in mice expressing mutant DISC1 in astrocytes led to lasting cognitive impairment and decreased number of parvalbumin-positive neurons in adult mice. These behavioral and neuroanatomical changes were restored in DISC1 mice after doxycycline treatment to shut down expression of mutant DISC1. Our studies suggest that both shared and cell type specific alterations could explain variable phenotypic manifestations of gene-environment interactions consistent with symptom heterogeneity of psychotic disorders.

ISBS SPECIAL PLENARY LECTURE 3: EMERGING PHARMACOLOGY OF TRACE AMINE-ASSOCIATED RECEPTOR 1 (TAAR1). RR Gainetdinov, Institute of Translational Biomedicine, St. Petersburg State University, St. Petersburg, Skolkovo Institute of Science and Technology (Skoltech), Skolkovo, Moscow, Russia

G protein-coupled Trace Amine-Associated Receptor 1 (TAAR1) is emerging as a promising new target for psychiatric disorders. Recent progress in identifying selective ligands for TAAR1 led to the possibility of evaluation of the functional consequences of stimulation/ blockade of TAAR1. By using these compounds in an experimental paradigm developed in our laboratory that involves dopamine transporter knockout mice, a novel model of acute dopamine deficiency, and mice lacking TAAR1 (TAAR1-KO mice), we explored the role of TAAR1 in modulation of dopaminergic and glutamatergic transmission. Pharmacological or genetic targeting of TAAR1 revealed that stimulation of TAAR1 suppressed dopamine-dependent behaviors, while TAAR1 deficiency potentiated them. TAAR1-selective ligands have shown potential antipsychotic, antidepressant, and pro-cognitive effects in experimental animal models; however, it remains unclear whether TAAR1 can affect PFC-related processes and functions. Recently, we documented a distinct pattern of expression of TAAR1 in the PFC, as well as altered subunit composition and deficient functionality of the glutamate N-methyl-D-aspartate (NMDA) receptors in the pyramidal neurons of layer V of PFC in mice lacking TAAR1. The dysregulated cortical glutamate transmission in TAAR1-KO mice was associated with aberrant behaviors in several tests, indicating a perseverative and impulsive phenotype of mutants. Conversely, pharmacological activation of TAAR1 with selective agonists reduced premature impulsive responses observed in the fixed-interval conditioning schedule in normal mice. This study indicates that TAAR1 plays an important role in the modulation of NMDA receptor-mediated glutamate transmission in the PFC and related functions. Furthermore, these data suggest that the development of TAAR1-based drugs could provide a novel therapeutic approach for the treatment of disorders related to aberrant cortical functions.

SYMPOSIUM I: PSYCHONEUROIMMUNOLOGY RESEARCH SOCIETY PNIRS-CHINA WORKSHOP.
Chairs: CL Coe, KW Kelley (USA)

THE HISTORY OF THE PSYCHONEUROIMMUNOLOGY RESEARCH SOCIETY (PNIRS) AND THE EMERGENCE OF PNI RESEARCH IN CHINA. KW Kelley, University of Illinois-Urbana, IL, USA

Dr. Kelley will serve as the discussant for this PsychoNeuroImmunology Research Society US-China Workshop. Dr. Kelley is a former President of the PNIRS, and the current Editor of the premier journal in this field: Brain, Behavior and Immunity. Dr. Kelley is a founding father of the PNI discipline, and conducted pioneering research on the brain-immune axis as well investigated neuroendocrine influences on the immune system. Dr. Kelley also made significant contributions to our understanding of the relationship between proinflammatory cytokines and the behavioral malaise syndrome that is now described as ‘sickness behavior’. Dr. Kelley will discuss the presentations from this workshop and also talk about the growing interest in PNI among Chinese researchers, including the establishment of a new scientific organization, PNIRSchina.

THE SIGNIFICANCE OF THE MICROBIOME AND GUT/BRAIN AXIS FOR DEVELOPMENTAL HEALTH AND PSYCHONEUROIMMUNOLOGY RESEARCH. CL Coe, University of Wisconsin-Madison, Madison, WI, USA

We have come to appreciate that the commensal microbiota in the gut, as well as the bacteria on many other body surfaces, play an important role in promoting our health. Many disease conditions have now also been associated with a microbial dysbiosis. In addition, the bacterial symbionts in the gut are able to signal and communicate with the brain via the enteric nervous system and gut-associated lymphoid tissue. Our research focuses specifically on the importance of the microbiome for the young infant, and the influence of the mother, diet and early life experiences on the bacteria taxa that become established as the resident microbial community in the gut. This presentation will review recent advances in microbiome research, and new perspectives on the significance of the gut-brain axis for behavior, emotion, and health.

OBESITY, BRAIN INFLAMMATION AND COGNITION: AN ESSENTIAL ROLE FOR MICROGLIA IN PERINATAL PROGRAMMING OF THE OBESE BRAIN. SJ Spencer, School of Health and Biomedical Sciences, RMIT University, Melbourne, Australia

Around two thirds of adults and one quarter of children in developed countries are obese or overweight. Caloric intake and dietary composition have large and lasting effects on metabolism and are responsible for the increasing incidence of metabolic disease, but they can also influence our ability to conduct cognitive tasks. The brain is particularly vulnerable to the negative effects of poor diet on cognition during sensitive windows of development such as in early postnatal life. However, the mechanisms for these effects are not well understood. Our research shows microglia are likely to be a key player. Thus, the diet consumed in early life can permanently alter the brain's neuroinflammatory profile. Obesity that is established in early life by perinatal over-eating can lead to microgliosis that extends beyond the hypothalamus and feeding-related regions into brain regions involved in cognitive processing, including hippocampus. These microglia are hyper-responsive to challenging stimuli, with negative consequences for cognitive and immune function throughout life.

HMGB1 AND ITS REDOX STATE PLAYS A KEY ROLE ON THE INDUCTION OF DEPRESSIVE-LIKE BEHAVIOR. Y-J Lian, H Gong, T-Y Wu, Y-X Wang, Laboratory of Stress Medicine, Department of Psychology and Mental Health, Second Military Medical University, Shanghai, China

WHAT YOUR MONOCYTES SAY ABOUT YOUR MIND AND BODY AND WHY WE SHOULD LISTEN: IMPAIRED β2-ARs AND GRs MAY EXPLAIN LOW-GRADE INFLAMMATION, CARDIOVASCULAR DISEASE RISK AND DEPRESSIVE MOOD. S Hong, S Dimitrov, T Cheng, F Shaikh, JM Green, C Pruitt and N Beg**,** Departments of Psychiatry, Family Medicine and Public Health, University of California San Diego, CA, USA

INTRODUCTION: Hyperactive sympathoadrenal and hypothalamic-pituitary-adrenal systems and elevated inflammation are observed in relation to chronic stress, but the link between those systems and its clinical risk remain unclear. Subclinically elevated blood pressure (BP), pre-hypertension (PHT), is related to future cardiovascular disease (CVD) and greater mortality. One of the major risk factors for PHT is obesity. We report that low-grade systemic inflammation is associated with compromised immunocompetence in obese individuals. We also show that an extended recombinant human-TNF treatment of monocytes results in decreased immune response, indicating contribution of chronic inflammation in compromised immunity in obesity. **METHODS:** We aimed to further investigate a cellular mechanism of obesity-related inflammation and hypothesized that the responsivity of monocytes' beta 2 adrenergic (β2-ARs) and glucocorticoid receptors (GR) to agonists is impaired among the obese. β2-AR and GR responsivity was examined by assessing intracellular TNF expression in monocytes upon ex vivo LPS-stimulation by flow cytometry with and without isoproterenol (Iso; 10-10-10-8 M) and cortisol (0.1-1μM), respectively. Plasma IL-1β, IL-6, TNF, and cortisol levels were determined by immunoassay. Asymptomatic individuals with normal BP or PHT participated. CV risk was calculated using Framingham risk score (FRS) for CVD. **RESULTS:** β2-AR responsivity to Iso in TNF inhibition was smaller among the obese compared to lean individuals (p's < .05). It was also impaired among the PHT compared to normal BP individuals and related to FRS. Furthermore,

β2-AR responsivity was negatively associated with plasma levels of IL-1β, IL-6 and TNF (p's < .05). Monocytes' GR sensitivity to cortisol in regulating TNF production was associated with both obesity and somatic depressive mood after controlling for demographic variables (p's < .05). **DISCUSSION:** We propose investigating monocytes as a meaningful and reliable tool in examining the neurohormone-immune link in obesity-related pathology and CV risk. **RESEARCH SUPPORT:** Grants from National Institutes of Health, USA

ANGIOGENIC AND IMMUNE SIGNATURES IN PSYCHOSIS RISK AND EARLY PSYCHOSES. JK Yao, PL Lizano and MS Keshavan, VA Pittsburgh Healthcare System, Medical Research Service, Department of Psychiatry, University of Pittsburgh School of Medicine, Pittsburgh, PA, Department of Psychiatry, Beth Israel Deaconess Medical Center, Boston, MA, USA

Evidence continues to mount demonstrating impaired antioxidant defense system (AODS) and presence of oxidative stress in patients with schizophrenia (SZ). The body of evidence yields a complex picture that shows pathological alterations in multiple pathways involving homeostatic balance of AODS in schizophrenia patients, both prior to treatment initiation and after long-term treatment. However, it is not clear whether such alterations occur in schizophrenia before the onset of illness. Recent evidence suggests that abnormalities in inflammatory, neurotrophic, and angiogenic processes may play a role in the etiology of SZ. The identification of molecular biomarkers early in the course of disease is crucial to transforming diagnostic and therapeutic avenues. We investigated 14 molecular analytes focusing on inflammatory, neurotrophic and angiogenic pathways from the plasma of antipsychotic-naïve familial high risk for SZ (FHR) and first-episode psychosis (FEP) subjects, in comparison to healthy controls (HC). We identified distinct alterations in molecular signatures in young relatives at FHR for SZ prior to psychosis onset and FEP subjects. Our findings indicated that the expression of soluble fms-like tyrosine kinase (sFlt-1) was significantly increased in the FHR group compared to HC, but not in FEP. At baseline, sFlt-1 was also significantly correlated with soft neurologic signs, but not symptomatology, cognition or MTL structure. Using the MRI, longitudinal examination in the FHR group demonstrated that high levels of sFlt-1 were significantly associated with worsening schizotypal symptoms and reduced left parahippocampal cortical thickness when compared to low levels of sFlt-1 expression. sFlt-1 is an anti-angiogenic factor that binds vascular endothelial growth factor (VEGF). Network analysis revealed a positive correlation between sFlt-1 and VEGF, suggesting an activation of the angiogenic cascade in the FHR group, which persists in FEP. Collectively, our findings suggest an association between sFlt-1 and structural abnormalities in the parahippocampus of subjects at familial high risk for psychosis. The presence of an angiogenesis and immunological dysfunction early in the course of disease shifts the balance towards anti-angiogenesis and inflammation.

SYMPOSIUM II: ZOFIA ZUKOWSKA SYMPOSIUM ON TRANSLATIONAL NEUROSCIENCE OF STRESS.

Chairs: AV Kalueff (USA, Russia, China), R Gainetdinov (Russia), M Pletnikov (USA)

INTRODUCTION: PROF. ZOFIA M. ZUKOWSKA. This regular ISBS symposium is dedicated to Professor Zofia Zukowska (1949-2012). Professor Zukowska received her M.D. and Ph.D., trained in cardiovascular medicine at the Warsaw Medical Academy (Poland). She pursued post-doctoral training at the NIH, working with such renowned scientists as Irwin I. Kopin, Scientific Director of NINDS, and Julie Axelrod, Nobel Laureate. It was during this research period when her interest in stress and neuropeptides became galvanized. For the 25 years, she was a professor (and, recently, Chair) of the Department of Physiology and Biophysics at Georgetown University, before moving to the University of Minnesota as the Director of Stress Physiology Center. Her research examined how stress affects cardiovascular and metabolic health and diseases, and the

role of peptides, in particular neuropeptide Y (NPY), a sympathetic neurotransmitter and a stress mediator. She was the first to determine that NPY mediates stress-induced prolonged vasoconstriction and vascular mitogenic and pro-atherosclerotic effects (via Y1 receptors) and potent angiogenic actions (via Y2 receptors), establishing the role of NPY in ischemia, retinopathy, tumors and obesity. Professor Zukowska was a strong supporter of the ISBS and a regular plenary speaker at our conferences. Her scientific vision, extraordinary creativity, kindness to colleagues, and the talent to be daring, continue to inspire all her ISBS colleagues and their research.

THE MILD ENCEPHALITIS HYPOTHESIS OF SEVERE PSYCHIATRIC DISORDERS. K Bechter, Ulm University, Ulm, Germany

Based on continued Research Projects on severe psychiatric disorders including virological and Imaging studies and psycho-neuro-immunological methods the mild Encephalitis (ME) hyopothesis was developed (Bechter 2001) , and more and more supported (Bechter 2013). A considerable subgroup of cases of severe psychiatric disorders including schizophrenia and severe depression and bipolar disorders may be causally related to ME. Such is especially supported by CSF studies demonstrating minor but definite CSF patholgies in more than 70 % of therapy resistant cases of this spectrum (Bechter et al 2010, maxeiner et al 2009& 2014, Kuehne etal 2013, Review in Bechter 2016).These findings are relevant for developing improved(psycho-neuro-immunological) treatments.

MULTIFACETED CONTRIBUTIONS BY DIFFERENT REGIONS OF THE ORBITOFRONTAL AND MEDIAL PREFRONTAL CORTEX TO PROBABILISTIC REVERSAL LEARNING. GL Dalton, NY Wang, AG Phillips, SB Floresco, University of British Columbia, Vancouver, Canada

Different subregions of the prefrontal cortex (PFC) contribute to the ability to respond flexibly to changes in reward contingencies, with the medial versus orbitofrontal cortex (OFC) subregions contributing differentially to processes such as set-shifting and reversal learning. To date, the manner in which these regions may facilitate reversal learning in situations involving reward uncertainty remains relatively unexplored. We investigated the involvement of five distinct regions of the rat OFC (lateral and medial) and medial PFC (prelimbic, infralimbic, and anterior cingulate) on probabilistic reversal learning wherein "correct" versus "incorrect" responses were rewarded on 80% and 20% of trials, respectively. Contingencies were reversed repeatedly within a session. In well trained rats, inactivation of the medial or lateral OFC induced dissociable impairments in performance (indexed by fewer reversals completed) when outcomes were probabilistic, but not when they were assured. Medial OFC inactivation impaired probabilistic learning during the first discrimination, increased perseverative responding and reduced sensitivity to positive and negative feedback, suggestive of a deficit in incorporating information about previous action outcomes to guide subsequent behavior. Lateral OFC inactivation preferentially impaired performance during reversal phases. In contrast, prelimbic inactivation caused an apparent improvement in performance by increasing the number of reversals completed. This was associated with enhanced sensitivity to recently rewarded actions and reduced sensitivity to negative feedback. Infralimbic inactivation had no effect, whereas the anterior cingulate appeared to play a permissive role in this form of reversal learning. These results clarify the dissociable contributions of different regions of the frontal lobes to probabilistic learning.

PLASMINOGEN ACTIVATOR INHIBITOR-1 (PAI-1) 4G/5G GENE POLYMORPHISM IS ASSOCIATED WITH THE CARDIOVASCULAR DISEASE OVERREPRESENTED IN SCHIZOPHRENIA. HF Yang, Psychiatry Department, Affiliated Hospital of Guangdong Medical University, Zhanjiang, China

INTRODUCTION: PAI-1 4G/5G polymorphism is associated with coronary artery disease. However, its impact on the cardiovascular disease overrepresented in schizophrenia remains unknown. **OBJECTIVE:** To investigate the potential association between PAI-1 4G/5G polymorphism and the cardiovascular disease overrepresented in schizophrenia. **METHODS:** This was a case-control study involving 138 patients with schizophrenia and 142 unrelated healthy controls. PAI-1 genotyping was done by polymerase chain reaction-allele specific amplification. **RESULTS:** PAI-1 4G/4G was prevalent in schizophrenia group when compared with controls (P = 0.002) and associated with increased risks of schizophrenia (OR = 2.1,

95% confidence interval (CI) = 1.19-3.73). Participants with PAI-1 4G/4G have significantly higher BMI and glucose compared with women without PAI-1 4G/4G. **CONCLUSION:** PAI-1 4G/5G genetic variations are associated with the cardiovascular disease overrepresented in schizophrenia.

THE EFFECTS OF G-PROTEIN-COUPLED RECEPTOR KINASE 5 POLYMORPHISMS ON ALZHEIMER'S DISEASE. M Yin, J Zhao, Y Cai, L Cui, B Zhao, Guangdong Key Laboratory of Age-related Cardiac and Cerebral Diseases, Affiliated Hospital of Guangdong Medical University, Zhanjiang, Guangdong Province, China

INTRODUCTION: The G-protein-coupled receptor kinases (GRKs) are important regulators in AD. Reported that the functional GRK5 deficiency in hippocampal cholinergic neuron leads to decreased ACh release, further increases β-amyloid accumulation and exaggerates tau hyperphosphorylation in the hippocampus. However, the effects of GRK5 polymorphisms in AD were not clearly. This study characterized single nucleotide polymorphisms (SNPs) in the GRK5 gene affect GRK5 functions in AD. **METHODS:** We distribution the two SNPs of GRK5 (Q41L and R304H) on 292 AD patients and 300 controls in case-control study. Moreover, we constructed plasmids carrying the two GRK5 polymorphisms and transfected into SH-SY5Y cells to analyze genetic effects on GRK5 and P-tau expression. We further used the protein modeling and docking analysis to study the effect of the two mutant proteins on GRK5 function. **RESULTS:** The allele distributions were significantly different between the cases and controls for the GRK5 Q41L in the LOAD subgroup. Haplotype analysis show that Q41L A-T may be the protective factor in risk of AD. The expression of P-tau was significantly increased in R304H group compared with WT. However, it showed obviously reduced in Q41L group. **DISCUSSION:** The GRK5 Leu41 allele protects from AD might by affecting the stabilizing of GRK5 on the membrane. On the contrast, the His304 allele might induced defective in the catalytic domain of GRK5. **RESEARCH SUPPORT:** National Natural Science Foundation of China (No. 81271214).

Day 2. Friday, October 28, 2016

Venue: Guangdong Ocean University Main Campus, Zhanjiang, China

WORKSHOP: HOW TO PUBLISH YOUR BEST SCIENCE IN RECOGNIZED INTERNATIONAL JOURNALS: INSIGHTS FROM THE EDITOR-IN-CHIEF OF "BRAIN, BEHAVIOR, AND IMMUNITY". KW Kelley, University of Illinois-Urbana, IL, USA

ISBS SPECIAL PLENARY LECTURE 4: AGE-RELATED CHANGES IN LEARNING AND MEMORY IN MOUSE MODELS OF ALZHEIMER'S DISEASE. RE Brown, Department of Psychology and Neuroscience, Dalhousie University, Halifax, Nova Scotia, Canada

We examined age-related changes in spatial learning and memory, procedural learning and memory, and olfactory memory in male and female APP/PSEN1 double transgenic, 3x-Tg AD and 5XFAD mouse models of AD and their appropriate control strains between 3 and 18 months of age. The 5xFAD mouse has the APP transgene with the Swedish (K670N/M671L), Florida (1716V) and London (V7171) mutations and a presenilin transgene with M146L and L286V mutations. The controls are wild-type litter mates. The 3xTg-AD mouse has the human amyloid precursor protein (APPswe), a mutated mouse presenilin-1 (PS1M1461), and a transgene associated with tau pathology (Tau301L). The B6129S1F2 mice are used as controls. The APP+PS1dE9 mouse has APP with a Swedish mutation (K670N/M671L) and mutant human presenilin (dE9) and has the B6C3F1/J mice as controls. The data presented examines the concept of multiple memory systems and their changes with age, the neuropathology of each mouse strain and the relationship of this neuropathology with the age-related decrements in visuo-spatial, motor and

olfactory learning and memory. The data examines genotype, age, and sex differences on each memory system to examine the effects of the transgenes on different memory systems. There were no genotype or age-related deficits in olfactory memory. The 5xFAD mice had age-related deficits in procedural learning. There were also age-and genotype-related deficits in visuo-spatial memory. Confounding effects of background strain, behavioural measures and housing conditions will be discussed.

ISBS SPECIAL PLENARY LECTURE 5: EFFECT OF PRO-INFLAMMATORY CYTOKINES ON THE FORMATION OF THE COGNITIVE FUNCTIONS IN RATS IN EARLY POSTNATAL ONTOGENESIS. VM Klimenko, OE Zubareva, Federal State Research Institution "Institute for Experimental Medicine", St. Petersburg, Russia

Proinflammatory cytokines interleukin 1 beta, -6 (IL-1 beta, IL-6) and tumor necrosis factor α (TNFα) are considered as key mediators of neuroimmune interactions. Raising the level of proinflammatory cytokines in the blood and the brain occurs when many pathological conditions (infectious diseases, trauma, hypoxia, etc.) and leads to the development of a central component of prodromal syndrome: increased body temperature, decreased motor activity, the suppression of motivations. In recent years, it has been shown evidence on the effects of proinflammatory cytokines in brain mechanisms of neuroplasticity, learning and memory, but questions remain insufficiently studied the actions of IL-1 beta, IL-6 and TNFα on the development of cognitive functions in early postnatal ontogenesis.Meanwhile, it is shown that the violation of attention and memory often occur in children, adolescents and adults with perinatal hypoxia or infectious diseases at an early age, the severity of neurological disorders correlates with increased levels of proinflammatory cytokines. Revealed are the effects of IL-1 beta and TNF- alpha on development and maturation of glial cells and neurons in the early postnatal period. The injections of bacterial lipopolysaccharide - the inductor of synthesis of proinflammatory cytokines - to experimental rats in early postnatal ontogenesis leads to short-term and long-term changes of behaviour and memory violations. In particular, our research has shown that a moderate increase in the level of IL-1 beta during certain critical periods of early postnatal ontogenesis, leads to delayed (detected in adult animals), changes in exploratory behavior and different kinds of memory. Experimental animals have a high stress-reactivity and impairments of the genes expression of the brain cells involved in the neuroplasticity regulation (FGF2). The treatment of rats with bacterial lipopolysaccharide (an inductor of proinflammatory cytokine synthesis) during early postnatal ontogenesis induces the short-term and long-term changes in the gene expression of subunits of AMPA и NMDA glutamate receptors in the cells of hippocampus and of medial prefrontal cortex. Violations caused by increased levels of proinflammatory cytokines in early postnatal ontogenesis can be associated with the formation of neuropsychiatric problems specific to children and adults with attention deficit disorders and other cognitive dysfunctions.

OMEGA-3 ON QUALITY OF LIFE AND DURATION OF LIFE: A FEW LATEST EXAMPLES. W Zhang, DSM Nutritional Products, Human Nutrition & Health, China

The scientific research has advanced our understanding about the importance of marine-derived omega-3 long chain polyunsaturated fatty acids (LCPUFA) in health and in disease pathogenesis. Here a few examples of the latest progress in the field are highlighted. Firstly LCPUFA supplementation in early life (i.e. during pregnancy) improves birth outcomes including increased birth weight and decreased rate of preterm birth, which may lead to reduced renal, cardiovascular and metabolic burden in adult life. Secondly the increased LCPUFA intake or the elevation of docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) in the circulation is associated with lower incidence or prevalence of some non-communicable diseases (NCDs) including cardiovascular disorders, type-II diabetes and some cancer. Fish oil administration also minimizes some harmful responses to the exposure of air pollution (i.e. PM2.5). Thirdly, the increased intake of LCPUFA or the elevation of DHA and EPA in the circulation reduces all-cause mortality in general population, as demonstrated recently by our meta-analysis: from 11 prospective studies involving 371,965 participants of general population, 31,385 death events were recorded. The summary RR of all-cause mortality for high-versus-low LCPUFA intake is 0.91 (95% CI: 0.84–0.98). The summary RR for EPA and DHA intake is respectively 0.83 (95% CI: 0.75–0.92) and 0.81 (95% CI: 0.74–0.95). In the dose-response analysis, each 0.3 g/d increment in LCPUFA intake is associated with 6% lower risk of all-cause mortality (RR = 0.94, 95% CI: 0.89–0.99); and each 1% increment in the proportions of circulating EPA and DHA in total fatty acids in blood is accompanied by 20% (RR = 0.80, 95% CI: 0.65–

0.98) and 21% (RR = 0.79, 95% CI: 0.63–0.99) decreased risk of all-cause mortality, respectively (Sci Rep 2016;6:28165). In conclusion, the research results from us and others suggest that both dietary and circulating LCPUFA hold great promise in promoting human health. The health benefits of LCPUFA supplementation are seen as early as neonates are born, which may favorably have long term impact. In general population, those who take more LCPUFA or have higher circulating levels of EPA and DHA have lower risk in developing some NCDs, resulting in better health and longer lifespan.

SYMPOSIUM III. THE 6TH MIND-BODY INTERFACE (MBI) INTERNATIONAL SYMPOSIUM.

Chair: K-P Su (Taiwan)

PERSONALIZED MEDICINE WITH OMEGA-3 FATTY ACIDS FOR DEPRESSION. KP Su, Graduate Institute of Neural and Cognitive Sciences, Mind-Body Interface Laboratory (MBI-Lab), China Medical University and Hospital, Taichung, Taiwan

Depression is one of the leading causes of morbidity and mortality in medicine. Current available treatments clearly do not meet clinical needs, while clinicians and researchers are facing the huge challenge of developing effective depression treatments despite of the advance of neurosciences. As detailed in our Consensus Statements in the Lancet Psychiatry and World Psychiatry, nutritional medicine is a promising strategy for the crisis of under-effectiveness in depression treatment (1,2). Omega-3 polyunsaturated fatty acids (PUFAs), including eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), have a range of neurobiological activities in modulation of neurotransmitters, anti-inflammation, anti-oxidation and neuroplasticity, by which could contribute to the antidepressant effects (3-5). Evidence from epidemiological, pre-clinical, and clinical studies have revealed that omega-3 PUFAs play an important role in the treatment and prevention of certain subgroups of clinical depression (6-8). According to biological specificity and safety consideration, omega-3 PUFAs is a potential antidepressant treatment for pregnant women, children, adolescents, and inflammation-related depression. Omega-3 PUFAs are well tolerated and accepted by general populations for health promoting (9). Thus, more research on stratifying depression is needed to justify the clinical application of omega-3 PUFAs as one of the first-line antidepressant treatments in specific populations with depression.

EXPOSURE TO A MATERNAL N-3 FATTY ACID-DEFICIENT DIET DURING THE BRAIN DEVELOPMENT ENHANCE THE ACTIVITY AND DYSREGULATION OF HYPOTHALAMIC-PITUITARY-ADRENAL AXIS RESPONSES TO STRESS IN RAT OFFSPRING LATER IN LIFE. YJ Hsieh and HM Su, Physiology Department, College of Medicine, National Taiwan University, Taipei, Taiwan

Brain docosahexaenoic acid (DHA, 22:6n-3) accumulates rapidly during brain development and is essential for normal neurological function. The aim of this study was to examine whether DHA deficiency during brain development leads to dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis in response to stress later in life. Rats were exposed to an n-3 fatty acid-deficient or n-3 fatty acid-adequate diet from embryo to weaning at 3-weeks-old via maternal intake throughout the pregnancy and lactation, and then were changed to chow diet till sacrificed at 10-weekold. We found that the maternal rats fed n-3 fatty acid-adequate diet showed significant higher licking/grooming and arch-back nursing behavior than the maternal rats fed n-3 fatty acid-deficient diet. Exposure to the maternal n-3 fatty acid-deficient diet during the brain development resulted, at postnatal day 21, in a significant decrease in hypothalamic DHA levels and a reduced male offspring body weight. DHA deficiency during the brain development significantly increased and prolonged restraint stressinduced changes in colonic body temperature and serum corticosterone levels, caused a significant increase in sensitivity to dexamethasone negative feedback regulation, and enhanced depressive-like behavior in the forced-swimming test and anxiety-like behavior in the plus-maze test in later life. These results suggest that DHA deficiency during brain development leads to excessive HPA responses and blunted HPA negative regulation to stress and elevated behavioral indices of depression and anxiety in adulthood.

DISTINGUISHING NEUROPROTECTIVE EFFECTS OF DIFFERENT OMEGA (N)-3 FATTY ACIDS ON LPS-INDUCED BEHAVIORAL CHANGES AND MICROGLIA-MEDIATED NEUROINFLAMMATION. YY Mei, S Gaikwad and C Song, Neuroimmunology and Behavior Lab, China Medical University Hospital,

Taichung, Taiwan; Research Institute for Marine Drugs and Nutrition, Guangdong Ocean University, Zhanjiang, China

Neuroinflammation plays a critical role in development and deterioration of psychiatric and neurodegenerative diseases, including major depression. Recently, polyunsaturated omega (n)-3 fatty acids (FA), such as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), serve as anti-inflammatory candidates due to their multiple functions in cell membranes such as regulation of neurotransmission, immune protein binding and membrane-bound enzyme activity. However, the effect of different omega (n)-3 FAs on the behavior and neuroinflammation in depression are still unclear. In the present study, C57BL/6 mice were fed with ad libitum food powder mixed with 1% coconut oil, 1% EPA or 1% DHA for 12 weeks before signal injection of lipopolysaccharide (LPS) (5ug intracerebroventricully, ICV). After ICV injection 1week, mice given LPS injection did not show any significant differences in locomotor activity, but had significantly increased anxiety-like behavior in the elevated plus maze, which were reversed by pre-fed with EPA or DHA diets. At the protein level, EPA pretreatment significantly elevated peroxisome proliferator-activated receptor gamma (PPARγ) level and reversed LPS-induced glial cell line-derived neurotrophic factor (GDNF) and transforming growth factor beta 1 (TGF-β1) decreases, but DHA pretreatment only prevented LPS-induced decrease in TGF-β1 in the hippocampus. At the mRNA level, both EPA and DHA pretreatments significantly decreased LPS-induced corticotropin-release factor (CRF), CD11b and GFAP activation, and further elevated GDNF expression both in control and LPS groups. In addition, mRNA expressions of TGF-β1 and PPARγ were elevated in DHA and EPA groups without LPS administration, respectively. In a cellular model of neuroinflammation, similar to in-vivo findings, pretreatment with EPA or DHA for 24 hours not only inhibited LPS-induced microglial activation but also elevated cell viability in differentiated SH-SY5Y cells. Different from proliferative effects of EPA pretreatments, high-dose DHA pretreatment became neurotoxic to differentiated SH-SY5Y cells. Previously TGF-β1 and PPARγ have been known to involve in neuroprotection and neurogenesis. The present study suggests that EPA seems to be more effective than DHA in neuroprotection and neurogenesis especially related to PPARγ activation. Keywords: EOC-20 cells, major depression disorder (MDD), neuroinflammation, omega (n)-3 FAs, PUFAs, SH-SY5Y cells

EFFECTS OF N-3 POLYUNSATURATED FATTY ACID SUPPLEMENTATION ON COGNITIVE FUNCTION IN PATIENTS WITH LATE-LIFE DEPRESSION - A PRELIMINARY STUDY. CC Chiu, CJ Chang, WC Chiu, IW Sun, SI Liu, ML Lu, CH Chen, SY Huang, Taipei City Psychiatric Center, Cathay General Hospital, Mackay Memorial Hospital Taipei, Taipei Medical University, Taipei, Taiwan

INTRODUCTION: N-3 polyunsaturated fatty acid (PUFAs) supplementation may be beneficial in cognitive function in older people. Older people with late-life depression (LLD) are a high-risk sample for cognitive impairment. The aim of this study was to investigate the effects of n-3 PUFA supplementation on cognitive decline in patients with LLD by a 48-week randomized double-blind placebo-controlled study. **METHODS:** People≧60 years with previous major depression and without dementia or Mini-Mental State Examination (MMSE) score < 17 were randomly assigned to 3 gm n-3 PUFAs or placebo added on their ordinary treatment. Cognitive function was evaluated using a series of cognitive tests at week 0 and week 48. Cognitive decline was defined as decrease of MMSE score ≧ 2 points. Which cognitive test parameter is more sensitive to n-3 PUFAs supplementation was also explored. **RESULTS AND DISCUSSION:** Seventy cases, 50 female and 20 male with average age of 67.3±6.8 years, had completed 48-week study. After adjustment for possible confounders, the Odds Ratio (OR) for cognitive impairment in n-3 PUFA group compared to placebo group was 1.22 (p=0.88). There was no difference in the decline of any representative individual cognitive test parameter between groups. In spite of good tolerability, n-3 PUFA supplementation was not found to be beneficial in decrease of cognitive impairment in older people with LLD compared to placebo. **RESEARCH SUPPORT:** Taipei City Hospital and the National Science Council of Taiwan.

ASSOCIATION BETWEEN A FUNCTIONAL VARIANT IN A GENE ENCODING AN ENZYME METABLLIZING DOPAMINE (CATECHOL-O-METHYLTRANSFERASE) AND POST CANNABIS PSYCHOSIS: A REPLICATION IN CANADIAN CAUCASIANS. KJ Aitchison, R Lodhi, Y Wang, C

Crocker, A Loverock, A Dimitrijevic, H Ren, D Bugbee, G Macintyre, P Tibbo, SE Purdon, Departments of Psychiatry and Medical Genetics, University of Alberta, Edmonton Early Psychosis Intervention Clinic, Department of Psychiatry, Dalhousie University, Nova Scotia Early Psychosis Program, Halifax, NS, Neuropsychology Department, Alberta Hospital Edmonton, Department of Medicine, University of Alberta, Edmonton, AB, Canada

INTRODUCTION: Caspi et al. (2005) observed that COMT rs4680 genotype predisposed to subsequent schizophreniform disorder after adolescent cannabis use. Since then an association between lifetime use of cannabis and this COMT variant has been described (Costas et al., 2011; Ermis et al., 2015). Our aim was to investigate these associations in Canadian Caucasians. **METHODS:** 175 patients of self-reported Caucasian ethnicity with psychosis were recruited in Edmonton, and Halifax (Canada). Age of onset of psychosis (AoP), defined as age of DSM-IV diagnosis (schizophrenia, schizoaffective disorder, schizophreniform disorder, delusional disorder, brief psychotic episode, psychosis not otherwise specified, and substance-induced psychosis) was established using the Structured Clinical Interview for DSM-IV. Data on cannabis use were collected using a self-report computerized questionnaire. DNA was extracted from saliva collected using Oragene kits. The COMT Val158Met variant (rs4680) was genotyped by SNaPshot and TaqMan assays. **RESULTS AND DISCUSSION:** COMT genotype was associated with lifetime cannabis use (β=2.144, p=0.047), and with age of first cannabis use (β=2.188, p=0.044, after adjusting for gender). In those who had used cannabis before 20 years of age, COMT genotype had a significant effect on AoP (median AoP: Val/Val<Val/Met<Met/Met 19.70, 20.95, 21.90 years respectively; log rank test p=0.037). Our data are consistent with those of Caspi et al (2005), which may reflect factors such as Caucasian ethnicity (stringently defined), and other sociodemographic factors. **RESEARCH SUPPORT:** Bebensee Schizophrenia Research Unit, Canadian Institutes of Health Research (grant 200810), Nova Scotia Health Research Foundation, Canadian Foundation for Innovation, Alberta Innovation and Advanced Education, Government of Alberta (Alberta Centennial Addiction and Mental Health Research Chair to KJA).

FATTY ACIDS THAT ARE PRESENT AND NOT PRESENT IN THE BRAIN: IMPLICATIONS FOR TARGETING THE BRAIN IN PSYCHIATRY. R Bazinet, University of Toronto, Toronto, Canada

The brain is especially enriched with the polyunsaturated fatty acids (PUFA) docosahexaenoic acid (DHA) and arachidonic acid, while being virtually devoid of other PUFA such as eicosapentaenoic acid (EPA). It has been suggested that the plasma supply to the brain regulates brain PUFA levels and replace PUFA consumed in the brain. Candidate plasma pools that supply the brain with PUFA include the plasma unesterified pool, PUFA esterified to lysophosphatidylcholine or the uptake of PUFA-containing lipoproteins via lipoprotein receptors into endothelial cells of the blood brain barrier. This paper will present recent studies that have examined the role of lipoprotein receptors and the kinetics of candidate plasma pools which supply the brain. Upon presenting evidence that the plasma unesterified pool is a major source of brain PUFA, especially for DHA, I will describe how rapid metabolism also maintains very low levels of certain PUFA, such as EPA. Because fatty acid uptake into the brain can be imaged in humans, we can estimate brain PUFA, including DHA, requirements. A better understanding of brain DHA requirements has implications for food choices to maintain brain DHA levels.

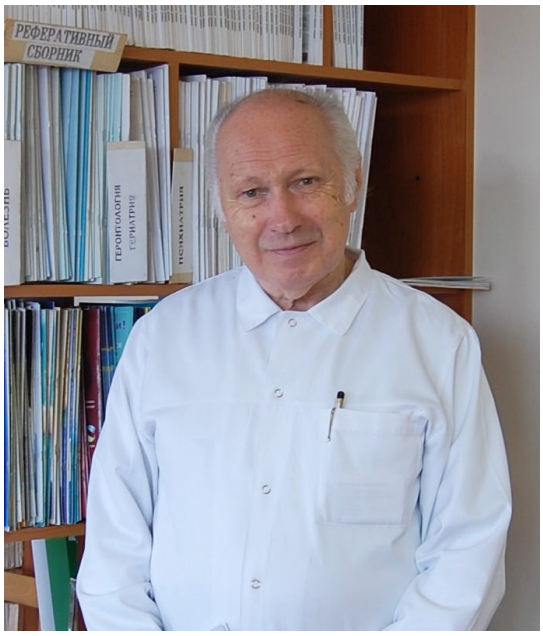
ANTIDEPRESSANT-LIKE EFFECTS OF WATER EXTRACT OF GASTRODIA ELATA BLUME IN RATS EXPOSED TO UNPREDICTABLE CHRONIC MILD STRESS VIA MODULATION OF MONOAMINE REGULATORY PATHWAYS. YE Lin, SH Lin, WC Chen, CT Ho, YS Lai, S Panyod, and LY Sheen, China Medical College, Taichung, Taiwan

ETHNOPHARMACOLOGY RELEVANCE: Gastrodia elata Blume (GE) is a traditional herbal medicine belonging to the Orchidaceae family, and has been used to manage neurological disorders for centuries. We have previously reported that its water extract (WGE) could improve the depressive-like behaviours in the forced swimming test (FST), an animal model of depression. **AIM OF THE STUDY:** To investigate the

antidepressant-like effects of WGE in rats exposed to unpredictable chronic mild stress (UCMS) model, and to explore its possible molecular mechanisms. **MATERIALS AND METHODS:** UCMS rats were orally administered with WGE (0.5g/kg body weight) daily within the 4 weeks UCMS procedure. The sucrose preference test and the open field test were conducted to assess anhedonia and spontaneous behaviours, respectively. The cerebral turnover rates of monoamine neurotransmitters and the serum corticosterone levels were measured. In vitro direct and indirect monoamine oxidase A (MAO-A) inhibitory assays were employed to assess the possible antidepressant-like mechanisms of WGE (0.5mg/mL) and its major component, gastrodin (GAS, 15, 30 and 60μg/mL). Western blot was used to examine the expression of protein related to monoamine regulation, such as MAO-A and tyrosine hydroxylase (TH). **RESULTS:** WGE significantly reversed the sucrose preference and other abnormal behaviours induced by 4 weeks of UCMS. WGE significantly restored the cerebral turnover rates of serotonin and dopamine and decreased serum corticosterone levels. WGE and gastrodin inhibited the activity and protein expression of MAO-A, and increased TH levels in PC12 cells. **CONCLUSION:** The antidepressant-like effects of WGE and gastrodin might be mediated by the regulation of monoamine neurotransmitters, and therefore were beneficial in depression treatment as a complementary approach.

SYMPOSIUM IV. LAPIN SYMPOSIUM ON TRANSLATIONAL BIOMEDICINE.

Chairs: VM Klimenko (Russia), BE Leonard (Ireland), RE Brown (Canada)



INTRODUCTION: PROF. IZYASLAV P. LAPIN. This regular ISBS symposium is dedicated to Professor Izyaslav 'Slava' P. Lapin (1930-2012), one of the true pioneers of experimental neuropsychopharmacology and biological psychiatry. Slava Lapin graduated from Pavlov Medical School in St. Petersburg, and shortly after receiving PhD, was invited in 1960 to establish the first psychopharmacology laboratory at the Bekhterev Psychoneurological Institute. The most important scientific contribution of Prof. Lapin was establishing the link between serotonin levels and mood-elevating (thymoleptic) action of antidepressants. He suggested that enhanced central serotonergic tone is essential for the mood-elevating effects of antidepressants. Lapin's serotonin hypothesis of antidepressant action, published in *Lancet* in 1969, became one the most cited papers published in this journal in the last 50 years. Lapin's studies have contributed greatly to the development of newest serotonergic antidepressants, such as SSRIs, currently representing the most prescribed group of psychotropic drugs in the world. Prof. Lapin was also the first to report the neuroactive effects of kynurenine and its derivatives – a discovery that opened another

rapidly expanding area of glutamatergic psychopharmacology. A talented professional musician, prolific writer, painter, and an enthusiastic athlete, Prof. Lapin was a strong supporter of ISBS, and generously shared his knowledge with colleagues and students at our "Stress and Behavior" conferences and ISBS summer schools. His enthusiasm, friendship, generous support of junior colleagues, and the deep knowledge as both a clinical and experimental neuropharmacologist ('humanists' and 'animalists', as he called them), made a long-lasting impact on his colleagues and students.

THE EFFECT OF POLYMORPHISM OF 5-HTTLPR GENE ON AFFECTIVE TEMPERAMENT, DEPRESSION AND COGNITION IN OBESITY. A Borkowska, M Bieliński, Department of Clinical Neuropsychology, Collegium Medicum of Nicolaus Copernicus University Torun, Bydgoszcz, Poland

BACKGROUND: Obesity is one of serious disorder caused with higher pre4dispozistion for somatic diseases and higher rate of mortality. This is growing problem across the world, especially in countries with high socioeconomic development. Current studies pointed significant associations between obesity and affective disturbances, such as depression, bipolarity, loose of impulse control. Affective temperament is a subclinical manifestation of such conditions. The 5-HTTLPR gene encoding the serotonin transporter may be involved in both mood and eating dysregulation. On other hand obesity is associated with cognitive disturbances especially executive frontal lobe dysfunctions. The aim of this study was to investigate the associations between polymorphism of the 5-HTTLPR gene and the five affective temperament dimensions (depressive, cyclothymic, hyperthymic, irritable and anxious), depressive symptoms, Body Mass Index (BMI) and working memory in obese patients. **METHODS:** This study involved 390 patients (237 females, and 153 males) with obesity. The TEMPS-A questionnaire was applied to evaluate affective temperaments. The Beck Depression Inventory (BDI) and Hamilton Depression Rating Scale (HDRS) were used for evaluation of intensity of depression. Working memory and executive functions were evaluated by Wisconsin Card Sorting test. Genomic DNA was extracted from 7–10 ml of peripheral blood. DNA was obtained for serotonin transporter gene-linked polymorphism (5-HTTLPR) genotyping. **RESULTS:** In obese patients S/S genotype was associated with depressive and L/L with cyclothymic temperament. Subjects with L/L genotype presented significantly higher BMI and greater intensity of depressive symptoms in BDI and HDRS. Patients with morbid obesity (BMI>40)he show greater level of depression and lower results on WCST, however no association between the WCST results and intensity of depression were observed. L/L genotype of serotonin transporter gene was associated with worse results on WCST, especially in morbid obesity group. Conclusions: In obese patients S allele of 5-HTTLPR gene was associated with depressive temperament on TEMPS-A, L allele corresponded with greater obesity higher prevalence of depression and cognitive frontal dysfunction.

EVALUATION OF COGNITIVE DEFICITS IN A MOUSE GENETIC MODEL OF PARKINSON'S DISEASE WITH OVEREXPRESSION OF A-SYNUCLEIN. TG Amstislavskaya, YJ Ho, MA Tikhonova, Scientific Research Institute of Physiology and Basic Medicine, Novosibirsk, Russia; Department of Psychology, Chung Shan Medical University, Taipei, Taiwan

INTRODUCTION: Parkinson's Disease (PD) is a severe neurodegenerative disease caused primarily by degeneration of dopamine neurons, its incidence is increasing due to the aging of population. In addition to motor dysfunction, in 20-40% of patients with PD dementia occurs including emotional changes, memory and recognition deficits. We will present the methods for evaluation of affective and cognitive functions in mice. Mice of B6.Cg-Tg(Prnp-SNCA*A53T)23Mkle/J strain express the familial Parkinson's disease-associated A53T missense mutant form of human alpha-synuclein and develop adult-onset neurodegenerative disease with a progressive motoric dysfunction. This model is widely used to study the mechanisms of PD and screening for neuroprotective agents. However, cognitive deficits have not yet been assessed in this PD model. Modern research tools of advanced behavioral phenotyping allows accurate monitoring the behavioral impairments in rodent models of different neurologic and psychiatric disorders. The aim of this study was to compare cognitive and affective characteristics of mice of B6.Cg-Tg(Prnp-SNCA*A53T)23Mkle/J strain with those of mice of control C57Bl/6J strain. **METHODS:** We conducted the following behavioral testing in 5-months old mice: open-field test, Barnes test, T-maze, forced swim test, sucrose preference test, IntelliCage. **RESULTS AND DISCUSSION:** Mutant B6.Cg-Tg(Prnp-SNCA*A53T)23Mkle/J mice showed higher horizontal locomotor activity and a tendency to increase in vertical locomotor activity in the open-field test. No significant difference was found in T-maze,

sucrose preference and IntelliCage performance between mutant and wild-type mice. In forced swim test, mutant mice demonstrated higher activity and a decrease in the immobility time that correlated with their higher locomotion in open field test. However, the goal box latency in the Barnes test was significantly increased while exploratory activity was significantly decreased and learning was retarded in B6.Cg-Tg(Prnp-SNCA*A53T)23Mkle/J mice that might be regarded as an early marker of cognitive disturbances in this PD model. Hence, at the young age only light cognitive alterations can be detected in B6.Cg-Tg(Prnp-SNCA*A53T)23Mkle/J mice using Barnes test. **RESEARCH SUPPORT:** This work was partially supported by grants: No. 15-54-52029_HHC-a from the Russian Foundation for Basic Research (Russia) and No. MOST 104-2923-H-040-001-MY3 from the Ministry of Science and Technology (Taiwan, R.O.C.).

A NOVEL, MULTI-TARGET NATURAL DRUG CANDIDATE, MATRINE, IMPROVES COGNITIVE DEFICITS IN ALZHEIMER'S DISEASE. L Cui, Y Cai, W Cheng, G Liu, J Zhao, H Cao, H Tao, Y Wang, M Yin, T Liu, Y Liu, P Huang, Z Liu, K Li, B Zhao, Guangdong Key Laboratory of Age-Related Cardiac and Cerebral Diseases, Affiliated Hospital of Guangdong Medical University, Zhanjiang, China

INTRODUCTION: The therapeutics of AD is a puzzle that fascinated researchers for a long time. Although many factors were responsible for AD formation, Aβ was still considered to be the most important target for the cure of AD. In this study, we attempted to explore multiple targets for AD treatments with the aim of identifying a qualified compound that could both inhibit the aggregation of Aβ and block the RAGE/Aβ axis. **METHODS:** MTT analysis was used to determine the most optimal incubating concentration of matrine (MAT).The images of Aβ42 oligomers in different incubating time were captured by transmission electron microscope (TEM). Morris water maze (MWM) was applied to evaluate the effect of Mat on the spatial cognition of the AD mice .Western blotting and Elisa assay was adopted to detect related proteins expression. Molecular docking was adopted to analyze the crystal structures of Aβ42 monomer and detect the extracellular domain of RAGE. **RESULTS AND DISCUSSION:** The high doses of Mat significantly increased the viability of cells treated with Aβ42 monomers in 24 and 72 h ($p=0.0236$ and $p=0.0369$, respectively). It suggested that Mat could inhibit Aβ42-induced cytotoxicity and suppress the Aβ/RAGE signaling pathway in vitro. Additionally, the results of in vivo evaluations of the effects of Mat on the two targets were consistent with the results of our in vitro studies. Furthermore, Mat reduced proinflammatory cytokines and Aβ deposition and attenuated the memory deficits of AD transgenic mice. **RESEARCH SUPPORT:** Support for this work includes funding from the National Nature Science Foundation of China (81271214, 31171219 and 81401061).

CO-TARGETING MEK AND PIM-1 FOR THE INTERVENTION OF MALIGNANT TUMORS. Y Cui, Shantou University Medical College Cancer Hospital, Shantou, Guangdong, China

Finding new survival factors deriving from crosstalk between different signaling pathways and suppressing them purposely is essential for the future clinical development of targeted therapies. Here, we found that conventional MEK inhibitor (AZD6244) upregulated Pim-1, a serine/threonine kinase, in ERK-activated human colon cancer cell lines. Mechanistic studies revealed that targeting MEK could transcriptionally upregulate Pim-1 through activating the IRS1/AKT axis. Although administration of Pim-1 inhibitor (Pim-1 1) or AZD6244 alone showed very low efficacy, they could synergize in suppressing the proliferation of cancer cells (coefficient of drug interaction, CDI<1). Collectively, Pim-1 is a prosurvival factor, as well as a therapeutic target for MEK inhibition. Co-targeting MEK and Pim-1 is identified as an efficient strategy for overcoming cancer cells resistance to MEK-targeted therapy.

Day 3. Saturday, October 29, 2016

Venue: Guangdong Ocean University Main Campus, Zhanjiang, China

GDOU SPECIAL LECTURE 6: TBN. AG Phillips, University of British Columbia, Vancouver, BC, Canada

RIMND SPECIAL LECTURE 7: NEUROINFLAMMATORY AND NEUROPROTECTIVE ROLES OF GLIA CELLS IN CELLULAR MODELS OF ALZHEIMER'S AND PARKINSON'S DISEASES. C Song, YS Wu, XF Wang, YY Thao, ZL Peng, YP Zhang, AV Kalueff, KP Su, Research Institute for Marine Drugs and Nutrition, Guangdong Ocean University, Zhanjiang, China

Activated microglial and dysfunction of astrocytes may contribute to the etiology of Alzheimer's (AD) and Parkinson's diseases (PD). However, the direct interaction between neurons and glial cells in the aspects of neuroinflammation and neurodegeneration is unclear. In a cellular AD model, glutamate decreased neuronal cell viability and the expressions of BDNF, BDNF receptor TrKB and GDNF, while increased expressions of p75NTR and TNF-α. After adding glutamate-stimulated astrocytes-conditioned medium (ACM), glutamate-decreased cell viability and BDNF, increased GDNF and TNF-α were reversed at mRNA level. Similarly, at protein expression level, glutamate-decreased BDNF and TrkB FL receptor, but increased pro-BDNF, TrKB T1 receptor, p75NTR, GDNF and TNF-which were also attenuated by ACM. In a PD model, 1-methyl-4-phenylpyridinium (MPP+) mildly decreased SH-SY5Y cell viability, but not lipopolysaccharides (LPS) which only activated microglia EOC20. However, MPP+ and activated microglia synergistically promote signaling of inflammation and apoptosis in differentiated SH-SY5Y cells, such as much pronouncedly decreased SH-SY5Y cell viability and caused strong synergistic increases in the bax:bcl¬2 and bad:bcl-xl ratios , which was associated with activated NF-KB signaling. Other targets, including JNK and p38 were also upregulated during MPP+ and LPS interaction, but less synergistically. These effects were attenuated by microglia inhibitor minocycline, a microglial inhibitor. Altogether, the study demonstrated that astrocytes exerted some neuroprotective effects on neurons, which mechanism may be via the modulation of neurotrophins and TNF-α expressions. However, neuroinflammation may be required to induce a pro-apoptotic state in dopaminergic neurons exposed to relatively low doses of parkinsonian toxin.

SYMPOSIUM V: INTERNATIONAL SYMPOSIUM ON SCREENING AND DEVELOPMENT OF MARINE AND RELATED DRUGS. Chair: C Song (China)

CHEMICAL ANALYSIS FROM ECOLOGICAL NICHES TO MICROBIAL BIOACTIVE AGENTS AS BIOCHEMICAL TOOLS. M Konnerth, NA Schilling, A Zipperer, F Zubeil, D Weisbrod, F Surup, A Peschel, B Krismer, S Grond, Institute of Organic Chemistry, Interfaculty Institute of Microbiology and Infection Medicine, Eberhard Karls Universität Tübingen, Tübingen, Helmholtz Center for Infection Research (HZI), Department of Microbial Drugs, Braunschweig, Germany

The primary goal of our natural products research is to investigate the innovative potential of the chemistry and biology of new microbial chemical structures to discover new biochemical tools. We present further insights into the secondary metabolism of microorganisms: We screen for natural products with new chemical structures which might act as microbial bioactive agents ('mibactents') in biochemistry and, as a long-term goal, in medicine or agriculture in the future. Regulation activities and the biosynthesis of metabolites are of our interest. Actinomycetes are well known producers of microbial bioactive agents with diverse biological functions. Yet overlooked microbial producers are also presented. We discuss our focus on natural products from ecological niches of selected microbe-animal/plant habitats and screening methods. Effective dereplication approaches are mandatory to identify novel compounds. Our in-house HPLC-HR MS (chromatography-high resolution mass spectrometry) database "Tadeus" as well as a custom dereplication software enables both, targeted screening through scheduled precursor lists as well as untargeted screening in crude extracts. Furthermore, micro-fractionation in 96-well plates ideally gives direct correlation of the observed bioactivity to the sum formula of the compounds. Chemical synthesis

adds on the versatile availability of 'mibactents' and derivatives thereof for biological profiling to pave the road to novel ideas for drug development.

CHEMICAL DIVERSITY OF BIOACTIVE NATURAL PRODUCTS FROM MARINE-DERIVED ENDOPHYTIC FUNGI. B Wang, Laboratory of Marine Biology and Biotechnology, Qingdao National Laboratory for Marine Science and Technology, Key Laboratory of Experimental Marine Biology, Institute of Oceanology, Chinese Academy of Sciences, Qingdao, China

China has a rich source of marine organisms covering from temperate to tropical zones. We focused our interests in recent years to search for diversified bioactive natural products from marine-derived endophytic fungi. As a result, a wide range of natural occurring compounds including alkaloids, quinones, terpenoids as well as other kinds of compounds have been isolated and identified. These compounds displayed various biological activities. Among them, tetranorditerpenoids isolated from algal-derived endophytic Aspergillus wentii EN-48 displayed potent cytotoxicity against eight tumor cell lines. Further experiments indicated the in vivo activity against human cell lung carcinoma cell line (NCI-H460) and human small cell lung carcinoma cell line (NCI-H446). The antitumor activity, both in vitro and in vivo, the toxic experiment and the in vitro metabolism as well as the mode of action will be presented in the presentation.

SHRIMP EXTRACTED BY-PRODUCTS PREVENTS SH-SY5Y CELLS FROM NEUROTOXICITY INDUED BY Aβ 25-35. YP Zhang, S Gu, J Gagnon, L Ma, P Zhang, RE Brown, C Song, Research Institute for Marine Drugs and Nutrition, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang, China

INTRODUCTION: Neuroinflammation and oxidative stress, which reduces neurogenesis and induces neuron apoptosis, play important roles in amyloid-beta (Aβ)-related pathological progress of AD. Increased evidence suggests that marine by-products may exert neuroprotective effects against A□-induced neurodegeneration. The present study investigated the anti-inflammatory and anti-oxidative effects of 4-2A, an extract from Pandalus borealis shrimp wastes, in the cellular model of AD induced by Aβ25-35. **METHODS:** After differentiation，SH-SY5Y cells were treated with Aβ25-35 and/or 4-2A, cell viability and cytotoxicity were measured by MTT assay and LDH assay respectively. The changes of oxidative stress then were detected. Quantitative PCR and Western blotting were used to study the expression of several genes, including neurotrophins and their receptors, pro-inflammatory cytokines and apoptosis. **RESULTS:** Exposure of SH-SY5Y cells to 20 μM Aβ25-35 for 24 h significantly reduced cell viability, expression of never growth factor (NGF) and its tyrosine kinase receptor (Trk A), and the content of anti-oxidant glutathione (GSH), while increased reactive oxygen species, the proinflammatory cytokine tumor necrosis factor-alpha (TNF-α) and brain derived neurotrophic factor (BDNF) and its receptor TrkB. Furthermore, Aβ25-35 increased the Bax : Bcl-2 ratio and Caspase-3 expression. 4-2A treatment significantly attenuated the above changes induced by Aβ25-35, except for nitric oxide, BDNF and TrkB. **CONCLUSION:** Based on these results, it is concluded that 4-2A effectively protects SH-SY5Y cells against Aβ-induced neuronal apoptosis/death by suppressing inflammation and oxygen stress, regulating neurotrophin and receptor expressions. Further studies in animal models of AD should be carried out. **RESEARCH SUPPORT:** Atlantic Innovation Fund of Canada, National Natural Science Foundation of China.

THREE NOVEL PEPTIDES FROM ANTARCTIC KRILL (EUPHAUSIA SUPERBA) PROTEIN HYDROLYSATE AS A DIPEPTIDYL PEPTIDASE IV INHIBITOR IMPROVE GLYCEMIC CONTROL IN DIABETIC ZEBRAFISH. W Ji, C Zhang, H Ji, AV Kalueff, W Su, College of Food Science and Technology, Guangdong Ocean University, Guangdong Provincial Key Laboratory of Aquatic Products Processing and Safety, Key Laboratory of Advanced Processing of Aquatic Products of Guangdong Higher Education Institution, Zhanjiang, China

Diabetes mellitus is a metabolic disorder disease considered as one of the major health problems worldwide, with type 2 diabetes representing approximately 90-95% of the diagnosed cases. Dipeptidyl peptidase-IV (DPP-IV) is strongly implicated in type 2 diabete, and its inhibitors show potential for antidiabetic therapy. In this study, Antarctic krill (Euphausia superba) protein was hydrolyzed using animal proteolytic enzymes. This hydrolysate obtained was purified sequentially by ultrafiltration, gel filtration

chromatography and reversed phase high-performance liquid chromatography (RP-HPLC) to prepare DPP-IV inhibitory peptides. Three novel DPP-IV inhibitory peptides, Ala-Pro (AP), Ile-Pro-Ala (IPA) and Ile-Pro-Ala-Val-Phe (IPAVF), with IC50 values of 0.53, 0.37 and 0.73 mg/ml respectively were identified by hybrid quadrupole time-of-flight mass spectrometry (QTOF-MS). In order to evaluate the hypoglycemic effects of 3 DPP-IV inhibitory peptides, zebrafish model was established by using combination of high cholesterol diet and high glucose exposure method. The results showed that after 15 days peptides treatment, total insulin level increased, glucagon and cholesterol levels decreased by physicochemical detection, accompanied by significant hypoglycemic effects corresponding relative genetic expressions in the insa, glucagon and pck1 contents by RT-PCR tests. Three peptides all showed hypoglycemic effects, IPAVF was the best among them. Therefore, results suggest the potential of Antarctic krill protein as a functional food for the management of type 2 diabetes mellitus. **Key words:** Antarctic krill, DPP-IV inhibitory peptide, purification, zebrafish model, anti-diabetes.

RIMDN SPECIAL LECTURE 8: ANTI-INFLAMMATORY TRITERPEN PLANT EXTRACTS AND COGNITIVE FUNCTION IN OBESITY AND DIABETES. XF Huang, University of Wollongong, New South Wales, Australia

***CLOSTRIDIUM BUTYRICUM* TO-A ALLEVIATE ANXIETY IN APP/PS1 ALZHEIMER'S DISEASE MICE.** C Jiang, K Li, X Peng, D Chen, L Hu, S Zhu, L Cui, Z Liu, B Zhao, Guangdong Key Laboratory of Age-Related Cardiac and Cerebral Diseases, Affiliated Hospital of Guangdong Medical University, Zhanjiang, China

INTRODUCTION: Evidence suggest that Probiotics have a potential role in brain function and behaviour, especially decreasing anxiety and depression. In this study, we test the efficacy of Clostridium butyricum TO-A(CBTA) in reducing anxiety-like behavior in animal model of Alzheimer's disease. **METHODS:** Ten four-month-old male APP/PS1 transgenic mice randomly assigned to two groups were inoculated daily with CBTA (1x107CFU/0.5 ml for each mouse) or saline by oral gavage for 6 weeks. The effect of CBTA on anxiety-like behavior was measured by the elevated plus maze test. **RESULTS AND DISCUSSION:** CBTA treatment of APP/PS1 mice significantly increased the percentage of the total time spent in the open arms, compared with the APP/PS1 mice (13.36 %± 5.27 % vs 2.18% ± 0.45%, $p<0.05$). Open-arm entries of treatment group were almost 3 times as much as controls (5.00 ± 1.35 vs 1.75 ± 0.25, $p< 0.05$). This result indicate that CBTA may have a positive impact on anxiety. We are studying the underlying mechanisms of CBTA on anxiety. In addition, Alzheimer's disease concomitant with anxiety is not uncommon in clinical practice. Clinical validation of the role of CBTA on anxiety or even cognition is also warranted, which may provide us new insights into novel therapeutic strategies. **RESEARCH SUPPORT:** This work was supported by grants from the NSFC (No.81400986, No. 81271214), by the Natural Science Foundation of Guangdong Province (2014A0303135), by the Key Lab Funding from Guangdong Province (2012A061400019), by the Science and Technology Project of Zhanjiang (No. 2014A01031), and by the Doctoral Starting up Foundation of Affliated of Guangdong Medical University.

THE IMPACT OF HIGH SALT DIET ON GUT MICROBIOTA AND COGNITIVE FUNCTION IN C57BL/6J MICE. X Peng, K Li, C Jiang, D Chen, L Hu, S Zhu, L Cui, Z Liu, B Zhao, Guangdong Key Laboratory of Age-Related Cardiac and Cerebral Diseases, Affiliated Hospital of Guangdong Medical University, Zhanjiang, China

INTRODUCTION: High-salt diet(HSD) increase the risk of cognitive impairment, However, whether HSD changing gut microbiota(GM) and GM change lead to cognitive impairment are unclear. In this study, we verify the HSD can affect GM and cognition. **METHODS:** Ten Four-month-old C57BL/6J mice were divided into two groups randomly. Normal control group (NCG) were given free access to normal diet and the HSD group (HSDG) were fed with HSD for two months. GM populations were analyzed using 16S rRNA sequencing. Cognitive spatial ability was evaluated by the Morris water maze. **RESULTS AND DISCUSSION:** During the probe trial, the time and frequency, the percentage of total distance spent in the target quadrant, the number of platform crossings in HSDG had significantly decreased compared with the NCG (22.6 ± 3.9 vs. 34.9 ± 1.9, $p = 0.008$; 0.29±0.05 vs 0.46 ±0.02, $p=0.002$; 3.5 ± 0.87 vs. 6.6 ± 0.81,$p = 0.03$, respectively). Furthermore, there were significant different in GM diversity and population. 405 OUTs

were in common in both groups, 23 OUTs in NCG and 29 OUTs in HSDG were different from each other. HSDG had higher relative abundance of Firmicutes (41.55±10.58% vs 19.16±5.74% p=0.008) and lower that of Bacteroidetes (51.64±10.43% vs 71.45±7.24% p=0.003). PC1 accounted for 68.8% of the variability. LEfSe identified several differentially abundant taxons between two groups. Our results indicate that HSD influence GM and cognitive fuction.Whether the GM changed by HSD would exert an impact on cognition and the possible mechanic are our next research focus. Therefore, modulation of GM to control the negative effects of HSD, could be a new treatment strategy to reduce risk of dementia. **RESEARCH SUPPORT:** This work was supported by many grants, which have been mentioned in the abstract of "CLOSTRIDIUM BUTYRICUM …"

THE PROTECTIVE EFFECT OF FECAL MICROBIOTA TRANSPLANTATION ON EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS MICE. K Li, C Jiang, X Peng, D Chen, L Hu, S Zhu, L Cui, Z Liu, B Zhao, Guangdong Key Laboratory of Age-Related Cardiac and Cerebral Diseases, Affiliated Hospital of Guangdong Medical University, Zhanjiang, China

INTRODUCTION**: Several studies have report that multiple sclerosis(MS) patients have a distinct gut microbiota**(GM) compared to healthy controls. Large number of researches have proven that probiotics can improve experimental autoimmune encephalomyelitis (EAE) mice behavior. Fecal microbiota transplantation (FMT) has a 1700-year history, is efficacious for inflammatory bowel disease. But limited information on FMT for MS. In this study, we investigated whether FMT could ameliorate severity and progression on EAE mice. **METHODS:** Twenty-seven mice were induced by MOG35–55 peptide and assigned to two groups. One group was transplanted healthy intestinal fecal bacteria for 43 days. Clinical scores and incidence of EAE were recorded. **RESULTS:** FMT resulted in an alleviation of clinical symptoms remark, including lower clinical score and cumulative score of each mouse (0.29±0.18 vs 1.03±0.14, p< 0.004; 11.70±12.86 vs 38.60±31.76,p=0.02, respectively), delay onset of disease (14.00±4.45 d vs 28.75±4.69 d, p< 0.0001), incidence of EAE in 30 d (94.1% vs 20%, p=0.0002). **DISCUSSION:** Case report of FMT on multiple sclerosis patient for constipation reported that quality of life improved. In this study we have shown FMT alleviated the severity, reduced the incidence of EAE obviously, delayed onset of EAE. Dysbiosis, an altered microbial composition is being explored in the context of different autoimmune conditions. Lots of researches have found that FMT can improve intestinal microbial diversity and regulate immune response. FMT implementation is an easy and cheap treatment method, which could be a new treatment options for MS. But further investigation is necessary. **RESEARCH SUPPORT:** This work was supported by many grants, which have been mentioned in the abstract of "CLOSTRIDIUM BUTYRICUM …"

INFLAMMATORY ENDOTHELIAL MICROVESICLE MODULATE THE PROLIFERATION, MIGRATION AND APOTOSIS OF BRAIN VASCULAR SMOOTH MUSCLE CELLS VIA THEIR CARRIED RNAS ASSOCIATED WITH THE MEK1/2/ERK1/2 AND CASPASE-3/BCL-2 PATHWAYS. Q Pan, X Liao, Y Wang, Y Chen, B Zhao, X Ma, Guangdong Key Laboratory of Age-Related Cardiac and Cerebral Diseases, Institute of Neurology, Affiliated Hospital of Guangdong Medical University, Zhanjiang, China; Department of Pharmacology and Toxicology, Boonshoft School of Medicine, Wright State University, Dayton, OH, USA

INTRODUCTION: Microvesicles (MVs) can modulate the function of recipient cells by transferring their contents. In this study, we investigated the potential effects of endothelial MVs on proliferation, migration and apoptosis of human brain vascular smooth cells (HBVSMCs). **METHODS:** Endothelial MVs (EMVs) were prepared from human brain microvascular endothelial cells (HBMECs) cultured in a TNF-α plus SD medium. RNase-EMVs were made by treating EMVs with RNase for RNAs depletion. The proliferation, apoptosis and migration abilities of HBVSMCs were determined after co-cultured with EMVs or RNase-EMVs. Mek1/2 inhibitor PD0325901 was used for pathway analysis. Western blot was used for analyzing Mek1/2, Erk1/2, p-Erk1/2, activated caspase-3 and Bcl-2. The level of miR-146a-5p was measured by qRT-PCR. **RESULTS:** 1) EMVs promoted the proliferation and migration of HBVSMCs. The effects were accompanied with the increase of Mek1/2 and p-Erk1/2, which could be abolished by PD0325901; 2) EMVs decreased the apoptosis of HBVSMCs, which was accompanied with cleaved caspase-3 down-regulation and Bcl-2 up- regulation; 3) EMVs increased the miR-146a-5p level in HBVSMCs; 4) The RNase -EMVs were less effective as EMVs on HBVSMCs activities and miR-146a-5p expression. **DISCUSSION:**

EMVs generated under inflammation challenge can modulate HBVSMCs function and fate via their carried RNAs associated with Mek1/2/Erk1/2 pathway and caspase-3/Bcl-2 regulation, during which miR-146a-5p may play an important role. **RESEARCH SUPPORT:** National Natural Science Foundation of China (No. 81400360).

ZINC MODULATION ON GLYCINE RECEPTOR ALPHA 1 SUBUNIT IS ASSOCIATED WITH HUMAN HYPEREKPLEXIA. DC Wu, University of British Columbia, Vancouver, Canada

The divalent cation zinc modulates several types of synaptic receptors, including glycine receptors, GABAA receptors, and NMDA receptors. In physiological condition, zinc also modulates synaptic transmission. GlyR mediated fast synaptic transmission is involved in motor control and other physiological processes. Deficit of GlyR a1 channel function is linked to human hyperekplexia. However, it is still not clear whether zinc modulation on synaptic receptors is involved in human diseases. Recently, we reported that a single mutation (W170S) in glycine receptor alpha1 subunit, which was identified from Omani families with hyperekplexia and mild mental retardation, caused almost complete loss of zinc-mediated potentiation and enhanced zinc-mediated inhibition without changes on other electrophysiological properties of glycine receptors in both HEK cells and cultured neurons. The impairment of zinc-mediated potentiation was observed in glycine receptor currents mediated by three different endogenous agonists, including glycine, taurine, and beta-alanine. We also found that the micro structural environment changes of W170 may lead to reduce the surface expression and channel modulation on GlyRs. Interestingly, Connection between β8 and β9 strands mediated GlyRs surface expression and zinc mediated allosteric modulation. Moreover, we confirmed that the W170S knockin mice showed the similar startle responses as human hyperekplexia. Taken together, our study has discovered a new zinc potentiation site on glycine alpha 1 receptors and also revealed a strong link between synaptic zinc modulation and human disease.

THE STUDY ON ANTI-ALZHEIMER RELATED BIOACTIVE CONSTITUENTS OF MARINE FUNGI. Y Zhang, H Bao, Y Nie, Research Institute for Marine Drugs and Nutrition, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang, China

Alzheimer's disease (AD) is a severe global challenge to public health. The anti-Alzheimer related activities and the diversity of active constituents were investigated for forty-four marine fungal strains. The acetylcholinesterase (AChE) inhibitory activity was tested by 5,5-dithiobis-2-nitrobenzoic acid (DTNB) method and the anti-oxidant activity was assessed by 1,1-Diphenyl-2-picrylhydrazyl radical (DPPH) method. The bioautographies and specific color developing reagents were applied to investigate the diversity of bioactive substances and their structural types, respectively. HPLC-Q-TOF-ESI-HRMS was used to characterize the fungal bioactive metabolites. During the study, the fermentation extracts of 20 strains in marine potato-sucrose-peptone culture medium and of 22 strains in marine malt extract culture medium, showed remarkable dual bioactivity against AChE and DPPH. These dual-bioactive strains are originated from different fungal taxa. Bioautographies displayed their diverse bioactive constituents. Chemical colorization further showed that the active components of three strains may have the structural characteristics of nitrogenated compounds (alkaloids), and phenols, while the other strains may produce active compounds belonging to other types. By HPLC-Q-TOF-ESI-HRMS analysis, a metabolite with molecular formula $C_{16}H_{13}NO_2$ of a strain was found to display relatively strong inhibition against AChE. The present study reveals that marine fungi are important resource of diverse anti-Alzheimer's active substances and lays the foundation for future work on the isolation and elucidation of bioactive compounds.

GRK5 DEFICIENCY ACCELERATED THE HYPER-PHOSPHORYLATION OF TAU THROUGH GSK-3B ACTIVATION IN THE EARLY STAGE OF ALZHEIMER'S DISEASE. J Zhao, M Yin, Y Cai, L Cui, B Zhao, Guangdong Key Laboratory of Age-Related Cardiac and Cerebral Diseases, Affiliated Hospital of Guangdong Medical University, Zhanjiang, China

INTRODUCTION: The specific pathway of over phosphorylation of Tau then contributed to the pathogenesis of Alzheimer's disease (AD) is still not clear. GSK-3β has been reported that was one of the most efficient kinases for the hyperphosphorylation of Tau, our previous research suggest that G protein-

coupled receptor kinase 5 (GRK5) deficiencies may regulate the GSK3β and finally contribute to the early AD. In this research, we focus on reveal the pathological relations betweenGRK5, GSK3β and Tau, looking forward to explain the biological role of GRK5 in different stage of AD. **METHODS:** In aged APP/SP1 double transgene mice (6, 9 and 14 months) and GRK5 knock out (KO) mice (6, 9 and 14 months), we detected the expression level of GRK5, GSK3β and tau by Western Blot and Immunofluorescence. Morris Water Maze (MWM) was also used to detect changes in learning and memory function. **RESULTS AND DISCUSSION:** Our results showed that the cytosolic level of GRK5 was increased meanwhile the membrane (functional) levels of GRK5 were reduced in the hippocampi of aged APP/SP1 double transgene mice (6, 9 and 14 months), and GSK3β and P-Tau were corresponding positive regulated by GRK5 in SH-SY5Y cell, Moreover, the GSK3β inhibitor could also block the GRK5-GSK3β-Tau signal pathway. In GRK5 KO mice, both Phosphorylated of Tau and GSK3β in the hippocampus of aged GRK5 KO mice (9 and 14 months) were increased and GRK5 KO mice also displayed selective working memory impairment. Taken together, these findings suggest that GRK5 could accelerated the hyperphosphorylation of Tau through GSK-3β activation in the early stage of Alzheimer's disease. **RESEARCH SUPPORT:** This work was supported by funding from the National Natural Science Foundation of China (grant number 81271214).

THE EFFECTS OF RETRIEVAL-EXTINCTION PARADIGM DURING SLEEP ON ALCOHOL CRAVING.
R Tao, J Zhu, M Ma, C Wang, H Sun, Peking University Sixth Hospital, Peking University Institute of Mental Health, Key Laboratory of Mental Health, Ministry of Health, Peking University, Beijing, Department of Psychiatry, the Second Affiliated Hospital of Xinxiang Medical University, Xinxiang, China

INTRODUCTION: Alcohol dependence is an easy recurrence of chronic brain disease. Craving plays an important role in the process. On the prevention of relapse, it is significant to reduce craving among the patients diagnosed with alcohol dependence. Addiction memory retrieval involved the process of cue induced craving. Retrieval-extinction paradigm can eliminate addiction memory. Therefore, the aim of the study was to observe the effects of retrieval-extinction paradigm during sleep on alcohol cues-induced craving in the patients with alcohol dependence. **METHODS:** Alcohol dependence patients formed associated memory that tones served as the Conditioned Stimulus (CS) and an alcohol smell served as the Unconditioned Stimulus (US). Then, they were randomly assigned to four groups（sleep- CS, sleep-CTR, Wake-CS, Wake-CTR). During the experimental period, all of participants monitored by polysomnography (PSG). Ensuring that the part of participants were in the sleep state, the participants of sleep- CS group exposed the CS (sound associated with alcohol) and the participants of sleep-CTR group exposed sound which was nothing with alcohol .Whereas in the wake state, the participants of Wake-CS exposed the CS (sound associated with alcohol), the participants of Wake-CTR exposed sound which was associated with water. Then the indicators or scales related with craving were measured, including skin conductance response (SCR), blood pressure, heart rate, Alcohol Urgency Questionnaire, (AUQ), Craving-visual analogue scale (CVAS). **RESULTS:** One-Way ANOVA analysis was applied to four experiments related demographic indicators. There were no statistical differences. Compared with the wake group, the subjective craving degree of the sleep group was reduced more obviously, and the sleep-CS group was reduce more than the sleep-CTR group. DISCUSSION: We found that retrieval-extinction paradigm could suppresses craving of alcohol in alcohol addicts and sleep groups is more obviously than awake group. Fear memory study found that exposed to the voice for such frequency and decibel will not be effected the sleep structure. **RESEARCH SUPPORT:** Natural Science Foundation of China.

MODERATED POSTER SESSION

SEX DIFFERENCES IN THE CHRONIC UNPREDICTABLE MILD STRESS RESPONSE IN SD RATS.
HY Wang, C Zhang, HY Xue, YY Li, BP Liu, KW Li, YP Zhang, ZL Peng, C Song, Research Institute for Marine Drugs and Nutrition, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang , Guangdong, China

INTRODUCTION: Women have a higher risk than men to suffer depression. The hyperactive hypothalamic–pituitary–adrenal (HPA) axis, including the elevation of glucocorticoid (GC)

level, and increased inflammatory response in both the periphery and the brain was found in depressed patients. However, whether male and female have different response to chronic unpredictable mild stress (CUMS), a trigger of depression, especially in the neuroinflammation is unclear. Thus, this study aimed to explore gender difference in response to chronic CUMS. **METHODS:** Rats were divided into four groups. Behavior tests were performed after CUMS. mRNA expression was measured by qPCR in the amygdale. The activity of peritoneal macrophage and proliferation of splenic monocytes were tested to measure the changes of immune function. **RESULTS:** Compared to control, increased anxiety-like behavior in the elevated-plus and immobility times in the forced-swimming, while down-regulated sucrose preference and exploration in the open-field were found in both female and male CUMS rats. Female CUMS rats were more anxious compared with male CUMS rats. Furthermore, the reduced expressions of BDNF and GDNF, while increased TrK B and microglia maker CD11b expressions after CUMS in both female and male. However, down-regulation of GFRα-1 and α-2 expression only found in female CUMS rats, and the expression of CD11b was higher in female CUMS rats than in male CUMS rats. Higher macrophage activity and lower lymphocyte proliferation were found in both female and male CUMS rats. Conclusions: Females showed more pronounced inflammatory response than male in amygdala under CUMS, which may reveal the mechanism that more women suffer depression than men. **RESEARCH SUPPORT:** National Natural Science Foundation of China to C Song（81171118, 81471223）.

EFFECT OF CHRONIC NEUROINFLAMMATION INDUCED BY INTERLEUKIN-1β ON PERIPHERAL IMMUNITY. HS Xue, YP Zhang, C Zhang, YY Li, BP Liu, C Song, Research Institute for Marine Drugs and Nutrition, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang, Guangdong, China

INTRODUCTION: Currently, the etiology of depression is still unclear. Stress, anxiety, and depressive behavior, as well as dysfunction of neurotransmitter and endocrine system induced by sub-acute administration of pro-inflammatory cytokines are similar to the symptoms and pathology of depressed patients. However, how chronic neuroinflammation affect the peripheral immune system has not been reported. Here, we hypothesized that neuroinflammation may decrease peripheral immunity. **METHODS:** 20 male SD rats were divided into two groups: normal saline (NS) and interleukin-1β (IL-1β) treated groups. The rats in IL-1β group received lateral ventricle injection（icv）IL - 1 β for 14 days to induce chronic neuroinflammation. MTT assay was used to measure the proliferation of lymphocytes and activity of peritoneal macrophages. The levels of serum tumor necrosis factor（TNF-α）、hydrogen peroxide（H2O2）、glucocorticoid（GC）were detected by commercial kits. **RESULT:** Compared with control group, IL - 1β reduced lymphocyte proliferation, TNF-α concentration, exploration in open-field and macrophage phagocytosis，while increased the levels of H2O2、GC and anxiety-like behavior in elevated plus maze.. **CONCLUSION:** chronic IL-1-induced neuroinflammation may suppress the peripheral immune function but enhance oxidative stress. **Keywords:** IL-1β; GC; behavior, lymphocytes; macrophages; TNF-α; H2O2. **RESEARCH SUPPORT**：national science foundation of china（81171118，81471223）.

EFFTS OF EPA ON CHRONIC STRESS-INDUCED CHANGE IN THE FUNCTION OF PERIPHERAL MACROPHAGE AND THE UNSTURATED FATTY ACIDS COMPOSITION OF CELLULAR MEMBRANE. YY Li, C Zhang, HS Xue, BP Liu, CYP Zhang, C Song, Research Institute for Marine Drugs and Nutrition, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang, Guangdong, China

INTRODUCTION: Major depression is usually accompanied by inflammation response, increased stress hormones and change in cellular membrane polyunsaturated fatty acids (PUFAs) composition. PUFAs are important components of cellular membranes. Eicosapentaenoic acid (EPA) is a natural anti-inflammatory active substance that can improve depression. However, how EPA changes the peripheral inflammatory response and the fatty acids composition of cellular membrane PUFAs in depression model induced by chronic stress is unclear. **METHODS:** Chronic unpredictable mild stress was used to induce depression model, sucrose preferences and open field were tested as depressive behavior;corticosterone concentrations in serum and phagocytosis of macrophages were measured to analyze the function of peripheral immune. Finally, gas chromatography was used to detect the composition of f n-3 and n-6 fatty

acids in the brain. **RESULTS:** Compared to controls, stress significantly decreased sucrose intake, horizontal movement and vertical movement of open field test, while the ability of macrophage phagocytosis increased significantly. Furthermore, a decreased EPA, DPA and n-3/n-6 PUFAs were found in the brain of stress group. However, feeding EPA significantly improved the above abnormal changes caused by chronic stress. **CONCLUSION:** Chronic stress induces the changes of immune and PUFAs composition in rats. EPA can improve the depression and the mechanism may be rebalance of immune activation and composition of n-3 and n-6 fatty acids in the brain. **Key words:** EPA; polyunsaturated fatty acids; depression; chronic unpredictable mild stress; inflammatory. **RESEARCH SUPPORT:** National Science Foundation Of China (81471223,81171118).

FAT-1 TRANSGENIC MICE ALLEVIATE DEPRESSION-LIKE BEHAVIOR AFTER CENTRAL ADMINISTRATION OF LIPOPOLYSACCHARIDE. BP Liu, KW Li, HS Xue, YY Li, YP Zhang, C Song, Research Institute for Marine Drugs and Nutrition, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang , Guangdong, China

INTRODUCTION: A number of studies have showed that pro-inflammatory cytokines can directly stimulate hypothalamus-pituitary-adrenaline (HPA) axis to secrete adrenal cortical hormone, activate glial cells in brain and release pro-inflammatory cytokines. Excessive produced pro-inflammatory cytokines could cause the neuronal death, resulting neurodegeneration and depression. Epidemiological and clinical studies have shown that essential fatty acid n-3 in the food can improve depression. However, little is known about the exact mechanisms by which n-3 PUFA modulates depression. **OBJECTIVE:** To investigate the effect of n-3 PUFA on depression-like behaviors and the function of peripheral immune in brain of animal models of depression. **METHODS:** Fat-1 transgenic mice and their brood-born negative C57BL/6 mice were used in the present study. After 24h central administration of lipopolysaccharide (LPS) and saline, animal's depression-like behavior was tested. Then, animals were sacrificed and macrophages were obtained for MTT assay. **RESULTS:** In the elevated plus maze, the ratio numbers and times of open arm to close arm were both decreased in C57BL/6 mice after LPS administration, and immobility time in tail suspension test was increased ($P<0.05$), while LPS-induced change was reversed in Fat-1 mice($P<0.05$). In open field test, numbers in central zone and rearing was reversed in Fat-1 mice after LPS administration, but not up to statistical significance, which is similar with the forced swim test and MTT assay. However, there is no statistical significance in above tests between saline and LPS in Fat-1 mice. **CONCLUSION:** omega-3 fatty acid can alleviate depression-like behavior induced by inflammation, further study should be conducted to illuminate specific mechanism. **RESEARCH SUPPORT:** National Natural Science Foundation of China（81171118）.

EFFECT OF GU HONG INJECTION ON THE FUNCTION OF CEREBRAL CORTEX MITOCHONDRIA IN RATS WITH CEREBRAL ISCHEMIA. L Feng, M Wang, J Liu, N Yang, Y Liu, P Zuo, Department of Pharmacology, Institute of Basic Medical Sciences, Peiking Union Medical School, Chinese Academy of Medical Sciences, Beijing, China

OBJECTIVE: To observe the effect of Gu Hong Injection on the function of cerebral cortex mitochondria in rats with cerebral ischemia reperfusion injury. **METHODS:** The model of middle cerebral artery occlusion (MCAO) in SD rats was prepared by the method of thread embolism. The drug was administered immediately after the operation, and the effect was evaluated by 14 d after continuous administration. The expression changes of mitochondrial autophagy related protein Beclin1, Parkin, KIFC2, SNAP-25 and UCP3 were detected by Western blot method. **RESULTS:** Compared with the model group, the Guhong injection can significantly reduce the symptoms of the neurological deficit in ischemia and reperfusion rat model and recovery of sensorimotor function, and effectively reverse the lack of changes in mitochondria and the autophagy related protein expression in cerebral cortex of the model rats. **CONCLUSION:** It is possible that the action mechanism of Gu Hong Injection on improving cerebral ischemic injury may be related to the activation of mitochondrial autophagy. **Key words:** Gu Hong injection; Cerebral ischemia reperfusion; Rat cerebral cortex; Mitochondrial autophagy.

ZEBRAFISH ON BATH SALTS: NOVEL INSIGHTS FOR DRUG ABUSE RESEARCH. AV Kalueff, TO Kolesnikova, SL Khatsko, YuYu Morzherin, Ural Federal University, Ekaterinburg, Institute of Translational

Medicine, St. Petersburg State University, St. Petersburg, Russia; Research Institute for Marine Drugs and Nutrition, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang, China; ZENEREI Research Center and the International Zebrafish Neuroscience Research Consortium (ZNRC), New Orleans, LA, USA

AFTERNOON TALKS

THE GGA FAMILY PROTEINS MODULATE THE CELL SURFACE TRANSPORT OF ALPHA2B-ADRENERGIC RECEPTOR. M Zhang, G Wu, Department of Pharmacology and Toxicology, Medical College of Georgia, Georgia Regents University, Augusta, GA, USA

INTRODUCTION: Molecular mechanisms governing the cell surface transport of G-protein-coupled receptors (GPCRs) remain poorly elucidated. Here, we used alpha2B-adrenergic receptor (alpha2B-AR) as a model to determine the role of three Golgi-localized, gamma-adaptin ear domain homology, ADP ribosylation factor-binding proteins (GGAs), and one family of adaptor proteins for clatherin-coated vesicles involved in the trans-Golgi network (TGN) to endosome transport. **METHODS:** It has been well demonstrated that the function of GGAs is tightly controlled by specific interactions via their VHS domain with the acidic LL motifs presented in C-terminus of cargo molecules. **RESULTS:** We found that shRNA-mediated depletion of individual GGAs strongly arrested alpha2B-AR in the TGN and significantly reduced the cell surface expression and signaling of receptor. We further demonstrated the GGAs physically associated with alpha2B-AR through specific domains. The GGA-binding domains were mapped to the C-terminal regions in the third intracellular loop of alpha2B-AR, whereas alpha2B-AR-binding domains were identified to the hinge domain of GGA1, the GAE domain of GGA2 and VHS domain of GGA3. **CONCLUSION:** These data show novel functions of GGA family in the TGN-to-plasma membrane transport of alpha2B-AR which is likely mediated through a non-conventional mechanism. These data also suggest that the GGA adaptor proteins are important mediators of GPCR cell surface targeting. **RESEARCH SUPPORT:** This work was supported by the National Institutes of Health grants GM076167.

EFFECT OF TOTAL FLAVONOIDS EXTRACT FROM *EUPATORIUM ODORATUM* ON IMMUNITY AND INTESTINAL FLORA IN BROILER. JJ Chen, XN Wang, HY Lin, FH Nie, JQ Zheng, DL Gong, YP Yu, XH Ju, Y Ma, QH Zhang**,** Guangdong Ocean University, Zhanjiang, China

INTRODUCTION: Eupatorium odoratum Linn., a perennial herb of Eupatorium in Compositae, grows invasively and intensively in South China. It has been used by Chinese folk as an antipyretic and a detoxicating herbal medicine against bruises, swelling, inflammation and microbial infections. Its active components are mainly flavonoids. However, research on antimicrobial feed additive of E. odoratum has not been reported yet. **METHODS:** The total flavonoids from E. odoratum (TFEO) were prepared by ultrasonic assisted solvent extraction with 70% ethanol (v∶v). The effects of TFEO on immune organ indexes and serum immunoglobulins in AA broilers were studied for 42 days. And the intestinal bacterial 16S rDNA fragments were analyzed by Miseq high-throughput sequencing platform with the applications of bioinformatics such as classification unit cluster analysis, taxonomic analysis to explore the diversity and abundance of the intestinal microflora. **RESULTS AND DISCUSSION:** Adding 400 mg/kg of TFEO significantly increased the broilers' thymus index, bursal index, IgG, and SIgA, which meant TFEO promoted broilers' immunity. And 95% of the intestinal microbes were categorized into phyla of Firmicutes and Bacteroidetes. During experiment day 1 to day 21, in A1 (control), A2 (low-dose of TFEO), A3 (medium dose of TFEO), A4 (high dose of TFEO) the percent of genus Bacaeroides in the microbial community were 11.98%, 14.44%, 16.13%, and 13.14%, respectively. That is, in groups A2, A3, A4 genus Bacaeroides microbial communities were higher than that in control group by 2.46%, 4.15%, and 1.16%. From day 22 to day 42, in B1 (control), B2 (low-dose of TFEO), B3 (medium dose of TFEO), B4 (high dose of TFEO) the percent of the genus Bacaeroides were 17.63%, 16.64%, 16.86%, 32.44%, respectively, and in the B4 group it was significantly higher by 14.81%, compared to the B1 value. Thus, TFEO could enhance the abundance of intestinal beneficial microbes, i.e., Bacaeroides. **ACKNOWLEDGEMENT:** This research was supported by Guangdong Province Zhanjiang Financial Foundation of Science and Technology Special Competitive Allocation Project (No. A14031).

EFFECT OF POLYSACCHARIDES FROM *CORDYCEPS SINENSIS* CAMT 63341 ON SOME BEHAVIOR AND INMMUNITY ORGAN INDEX OF MICE. D Xu, S Li, J Hao, Guangdong Provincial Key Laboratory of Aquatic Products Processing and Safety, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang, China

OBJECTIVE: To evaluate the effect of polysaccharides which was precipitated from the liquid fermentation by a strain Cordyceps sinensis CAMT 63341 isolated from Cordyceps sinensis in Tibetan areas of China, on the growth, emotion, cognition and immunity properties of rats. **METHOD:** Totally 60 healthy male mice were averagely divided into blank control group, Ganoderma lucidum polysaccharides control group and Cordyceps sinensis polysaccharides groups. The mice in blank control group were feed with saline solution and those in Ganoderma lucidum group were feeding with Ganoderma lucidum polyaccriades of 5mg/mL. In Cordyceps sinensis groups, the mice were feed with the Cordyceps sinensis CAMT 63341 polysaccharides in the concentration of 5mg/mL, 3mg/mL and 1mg/mL, corresponding to high-, middle-, and low-dose groups, respectively. After continuous feeding for 30 days elevated plus maze test, shuttle box test, delayed type hypersensitivity test were conducted and the body weight and the index of thymus and spleen organ to body weight were recorded. **RESULTS:** In comparison with blank control groups, the mice feeding with Cordyceps sinensis CAMT 63341 polysaccharides exhibited acceleration in growth, enhancement in times of entering the open arm and staying time in maze text, improvement in active-avoiding capacity and passive-avoiding latent period of mice in shuttle box test, elevation in the index of thymus and spleen organ to body weight. These effects were found with a dose and response manner and the significant or very significant different level were observed in high-dose groups, almost equal to those feeding with Ganoderma lucidum polysaccharides. **CONCLUSION:** The polysaccharides in liquid fermentation by Cordyceps sinensis CAMT 63341 possess some promotions in growth, emotion, recognition and immunity. **Keywords:** Cordyceps sinensis CAMT 63341; polysaccharides; liquid fermentation; emotion; recognition.

ISBS PRESIDENTIAL CLOSING LECTURE 8: ZEBRAFISH MODELS OF NEUROPSYCHIATRIC AND COMORBID METABOLIC AND IMMUNE DISORDERS. AV Kalueff, Research Institute for Marine Drugs and Nutrition, College of Food Science and Technology, Guangdong Ocean University, Zhanjiang, China; Institute of Translational Medicine, St. Petersburg State University, St. Petersburg; Ural Federal University, Ekaterinburg, Russia; ZENEREI Research Center and the International Zebrafish Neuroscience Research Consortium (ZNRC), New Orleans, LA, USA

ISBS OUTREACH COLLABORATION: AN ARTIST'S LOOK AT MENTAL DISORDERS. D Raytchev, Daniela Raytchev Art, London, UK

OFFICIAL CLOSING OF THE CONFERENCE

Guangdong Ocean University (GDOU) and Research Institute for Marine Drugs and Nutrition

Guangdong Ocean University (GDOU) is a key institution jointly established by the Guangdong Province and State Oceanic Administration 80 years ago. GDOU's 4 beautiful campuses include 18 colleges in the fields of fishery, food, agriculture, engineering, economics, navigation, information, science, humanities, law, foreign studies, political science, public administration, sports and arts. GDOU has 3 first-class disciplines for doctoral degree and 22 disciplines for master's

degree. Its teaching buildings and 66 research centers are well-quipped with advanced facilities, including one state-level metric attestation center, 11 provincial key labs and 4 model experimental teaching centers. The University has made great strides in scientific research. 1,744 scientific research projects, carried out at GDOU, include national “973” and “863” programs, national Sci-Tech support plan, Natural Sciences Foundation of China and National Social Science Fund, with a total research budget of over 316 million yuan.

Research Institute of Marine Drugs and Nutrition (IMDN) was established at GDOU in 2014, aiming at the development of new treatments for psychiatric and neurodegenerative diseases. The institute consists of three main laboratories.

The chemistry laboratory focuses on the searching for anti-psychiatric and anti-neurodegenerative natural products, such as polyunsaturated fatty acids (PUFAs), bioactive oligopeptides, and small bioactive molecules belonging to other structural types, from marine fungi, seaweeds, and aquatic byproducts. Its focus also includes structural elucidation, structure-activity relationship investigation, and the mechanisms of related secondary metabolic synthetic pathways and regulation.

The pharmacology and toxicology laboratory focuses on 1) studying neuroinflammation in psychiatric and neurodegenerative diseases; 2) development and use of cellular and animal models of human diseases, and 3) pharmacological screening and study of marine bioactive compounds. The lab operates 3 research platforms: 1) Behavioral platform in rodents and zebrafish for testing learning and memory, motor activity, mental health (stress, anxiety and depression), social interaction, vision, hearing, olfaction and cognitive function；2) Cellular model platform for studying cancer cells, neuron and glial cell lines and white cell lines; 3) Molecular platform. In addition, several marine products are currently being developed.

The marine bio-resource laboratory studies marine organisms in the South China Sea which possess the potential to be developed into bio-products for pharmaceuticals or healthy foods. It primarily works on the biotechnologies and engineering techniques of microalgae breeding, large scale cultivation, harvest and oil extract for the production of very long chain unsaturated fatty acids.

IMDN International Symposium

1st GDOU Symposium “International Symposium on Screening and Development of Marine and Related Drugs” – October 28, 2016 at Guangdong Ocean University, Zhanjiang, China.

THE INTERNATIONAL “STRESS AND BEHAVIOR” SOCIETY (ISBS)

Established in 2007

www.stress-and-behavior.com
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ISBS is the international society of experts working with a wide range of topics in the field of translational neuroscience, neurobehavioral sciences, biopsychology and bio-psychiatry, with a particular focus on

stress, stress-related neurobehavioral phenotypes, their neural, molecular and genetic mechanisms, as well as stress-evoked neuropsychiatric disorders.

Anyone with an interest in stress-related human or animal behaviors, neurobehavioral disorders and their mechanisms, wishing to join the International Stress and Behavior Society can do so by paying dues of USD $100.00 regular member or $60.00 student member for a three-year term. Payment can be made following sending the e-mail form and payment request to the ISBS Secretariat at info@stressandbehavior.com. Once the form and the payment have been received, you will receive a membership confirmation.

Membership:
Regular membership dues are $ 100.00 for the period of three years, or $ 60.00 for the period of one year. Student (undergraduate and graduate) membership dues are $ 60.00 for the period of three years. Membership period starts January 1 of each year.

Regular membership benefits include a $ 50.00 discount for registration for any of the ISBS Conferences, symposia, workshops and summer schools. Student members will benefit from a $ 25.00 discount for registration for any of the ISBS Conferences, symposia, workshops and summer schools.

ISBS Membership application form (please fill in and send by e-mail to the ISBS Secretariat at info@stressandbehavior.com, with the subject 'ISBS Membership request')

Name, Family name:
Position/Title:
Institute/Company:
Category: please select one:

- Regular member, 3-year term ($ 100.00)
- Regular member, 1-year term ($ 60.00)
- Student member, 3-year term ($ 60.00)

Address (affiliation):
City:
Postal code:
Country:
Phone, Fax:
E-mail address:
www:

The Mind-Body Interface Laboratory (MBI-Lab)

Established in 2006

sites.google.com/site/mindbodyinterface/
mindbodyinterface@gmail.com

The **MBI-Lab** vigorously promotes a global agenda of translational medicine by promoting interdisciplinary research, and integrating work from the bench and bedside, which aim to provide better care and service in the field of mental health. Based at the China Medical University, Taichung, Taiwan, the MBI-Lab is host to education, research, and outreach programs in medical and basic research. Our goal is to promote research and teaching that leads to a better understanding of mental illnesses. We aim to create a stimulating environment for graduate students, clinicians and researchers, by understanding the molecular mechanisms that underlie basic biological processes and how they go wrong in psychiatric disease. The research centre focuses a wide range of medical and basic research topics by using the analytical tools of biochemistry, molecular biology, functional neuroimaging, and genetics techniques. Furthermore, academic cooperation is engaged and supported domestically and internationally with the research interest and specialty of our faculty.

MBI International Symposia

1. 5th MBI Symposium "**Nourishing the Mind and Body: From Nutrition to Neuroscience**" Oct. 20-21, 2015 at China Medical University Hospital, Taichung, Taiwan.
   https://sites.google.com/site/5thmindbodyinterfacesymposium/welcome-message

2. MBI Workshop "**Experts' Secrets about Neuroscience Research**" Oct 29, 2014 at China Medical University Hospital, Taichung, Taiwan.
   https://sites.google.com/site/mindbodyinterfaceworkshop2014/Home

3. 4th MBI Symposium "**One Step Ahead: Prevention and Prediction in Mental Health**" Feb 21-22, 2014 at China Medical University Hospital, Taichung, Taiwan.
   https://sites.google.com/site/mindbodyinterface/academicactivity/2014

4. 3rd MBI Symposium "**The Spirit of Health: Mastering the Mind-Body Interface**" Jan. 18, 2013 at China Medical University Hospital, Taichung, Taiwan.
   https://sites.google.com/site/mindbodyinterface/academicactivity/2013

5. 2nd MBI Symposium "**Mind-Body Interface: New Concept and Promising Treatment**" Nov. 2, 2011 at China Medical University Hospital, Taichung, Taiwan.
   https://sites.google.com/site/mindbodyinterface/academicactivity/2011

6. 2nd MBI Symposium "**Mind-Body Interface - From Cells to Clinics**" Nov. 5, 2010 at China Medical University Hospital, Taichung, Taiwan.
   https://sites.google.com/site/mindbodyinterface/academicactivity/2010

Please join our 2016-2017 ISBS conferences:

International Neuroscience and Biological Psychiatry ISBS Symposium "TRANSLATIONAL NEUROSCIENCE OF STRESS", November 10-11, 2016, San Diego, CA, USA

10th International Neuroscience and Biological Psychiatry ISBS Regional (S. America) Conference "NEUROSCIENCE OF STRESS" December 1-3, 2016, Rio de Janeiro, Brazil

4th Caribbean Biomedical Research Days CBRD-2017 January 16-18, 2017, Rodney Bay, St. Lucia

24th International Neuroscience and Biological Psychiatry Conference "STRESS AND BEHAVIOR" May 16-19, 2017, St. Petersburg, Russia

11th International Regional Neuroscience and Biological Psychiatry Conference "STRESS AND BEHAVIOR" (North America) June 22-24, 2017, Miami Beach, FL, USA

12th International Regional Neuroscience and Biological Psychiatry Conference "STRESS AND BEHAVIOR" (Asia) July 24-25, 2017, Yokohama, Japan

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