



13th Annual Psychiatry Research Day

Abstract Book

Presented by
The Department of Psychiatry

Wednesday, May 14th, 2014

WELCOME TO THE 13TH ANNUAL RESEARCH DAY OF THE DEPARTMENT OF PSYCHIATRY AT THE UNIVERSITY OF ALBERTA!

Our department has an internationally recognized record of research going back to the early days of Dr. Bill Dewhurst's chairmanship. A psychiatrist and neurochemist, Dr. Dewhurst recognized the importance of having basic science researchers included in clinical departments and of encouraging collaborative research between basic science and clinical researchers. Dr. Dewhurst was a pioneer in the area of "trace amines" and a founding Co-Director of the Neurochemical Research Unit. In recent years, the number of basic science and clinical researchers in our Department has grown significantly, and we now have active programs in many areas relevant to psychiatry including neurochemistry, neuropsychopharmacology, psychotherapy, neuroimaging, neuropsychiatry and translational neuroscience, with newer programs evolving in other areas. Many of these programs involve collaborative research with colleagues from other departments and institutions locally, nationally and internationally.

Over the years, the Department of Psychiatry has developed very strong MSc and PhD programs to complement our excellent residency program, with some of our residents enrolled in our MSc program. Our graduate students and postdoctoral fellows represent our department exceedingly well by winning many national, provincial, university and faculty scholarships and awards and by disseminating their research at scientific conferences throughout the world.

We will begin our research day with talks by graduate students in our department, followed by faculty presentations in the afternoon. We thank graduate students James Benoit, Huining Guo, and Yanlin Wang for volunteering to present their research this year, as well as Drs. Christopher Power and Junhui Wang for their valuable faculty presentations. We will also feature two poster session presentations by our research trainees and collaborators. The top presentations by research trainees will be acknowledged with awards, namely the Geoff Hopkinson Award, the Gordon King Award, and the Glen Baker Research Award. We thank Drs. Jacqueline Cummine, Esther Fujiwara, Dawn Kingston, and Rebeccah Marsh for agreeing to serve as judges for these awards.

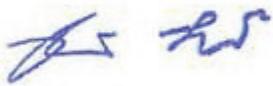
Lunchtime is reserved for our keynote lecture, sponsored by the W.G. Dewhurst Memorial Fund. Our keynote speaker for this year's event is **Dr. Rémi Quirion**, Quebec's first Chief Science Officer, a member of the FRSQ Board of Directors, and an internationally recognized expert on Alzheimer's Disease and neurodegeneration. We are extremely excited to host Dr. Quirion at our event and anticipate that his talk will be substantial, interesting, and informative both for members of our faculty and external attendees. Dr. Quirion's keynote presentation at Psychiatry Research Day is entitled, "*A Brain's Traveler: From Basic Neuroscience to Psychiatry and Landing as Quebec's Chief Science Officer.*"

We are grateful to all our research trainees and their supervisors for their overall contribu-

tion to the vital research component of our department, and to Tara Checknita for providing excellent ongoing administrative support for our research program. Special thanks this year go to Jiyun Chung and Dimitar Ourdev (our graduate student representatives), Dr. Esther Fujiwara (our graduate program director) and Tara Checknita for their tireless efforts in organizing this Research Day. We also gratefully acknowledge financial support from several sources for this important venture, as indicated in the book of abstracts.

Thank you for joining us in celebrating our research accomplishments for the past year.

Best wishes,



Xin-Min Li, Professor
Chair, Department of Psychiatry



Andrew Greenshaw, Professor
Associate Chair of Research, Dept. of Psychiatry

ACKNOWLEDGEMENTS

The Department of Psychiatry is grateful to the following for their financial support*:

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UNIVERSITY OF ALBERTA
FACULTY OF MEDICINE & DENTISTRY
Department of Psychiatry

13TH PSYCHIATRY RESEARCH DAY ITINERARY

8:45AM - 9:00AM - OPENING REMARKS

9:00AM - 10:00AM - GRADUATE STUDENT PRESENTATIONS

JAMES BENOIT: *Imaging high-risk adolescents: Assessing myelin deficits with DTI*

HUINING GUO: *Desvenlafaxine prevents anxiety/depression-like behaviors in an unpredictable chronic mild stress mouse model of depression: The role of myelin and oligodendrocytes*

YANLIN WANG: *Roles of insulin-like growth factor 2 receptor and lysosomal enzymes in Alzheimer's disease pathology*

10:15AM - 11:45AM - STUDENT POSTER SESSION

12:00PM - 1:00PM - W.G. DEWHURST LECTURE

RÉMI QUIRION: *A brain's traveler: From basic neuroscience to psychiatry and landing as Quebec's chief science officer.*

1:00PM - 2:00PM - FACULTY PRESENTATIONS:

DR. CHRISTOPHER POWER (DEPT. OF MEDICINE/DIV. OF NEUROLOGY): *HIV/AIDS and neuropsychiatry: An evolving leopard's coat*

DR. JUNHUI WANG (DEPT. OF PSYCHIATRY): *Regulation of astrocyte pathology by fluoxetine prevents the deterioration of Alzheimer phenotypes in an APP/PS1 mouse model*

2:00PM - 3:00PM - STUDENT POSTER SESSION

3:00PM - 4:00PM - AWARDS PRESENTATION & RECEPTION

W.G. DEWHURST MEMORIAL LECTURE

12:00PM - 1:00PM

Rémi Quirion
OC, CQ, PhD,FRSC

Dr. Rémi Quirion is the Chief Scientist of Quebec and an internationally recognized authority on neurodegenerative disorders. Prior to accepting this position in 2011, Dr. Quirion was a full professor of Psychiatry at McGill University, the director of the Douglas Mental Health University Institute Research Centre, and the executive director of the International Collaborative Research Strategy for Alzheimer's Disease of the Canadian Institutes of Health Research. Under his leadership, the Douglas Hospital Research Centre became a premier research facility in Canada in the fields of neurosciences and mental health. During his tenure, he has trained over 70 graduate students and post-doctoral fellows, published 5 books and over 650 articles in prominent scientific journals such as Nature, including seminal works on cholinergic communication in the central nervous system and its relation to neuropathology. Additionally, Dr. Quirion has made significant contributions in elucidating the roles of neuropeptide Y in depression, learning, and memory, and the calcitonin gene-related peptide (CGRP) in pain and opiate tolerance. His major interest lies in the training of the next generation of scientists.

ww

A BRAIN'S TRAVELER: FROM NEUROSCIENCE TO PSYCHIATRY TO LANDING AS QUEBEC CHIEF SCIENTIST.

For close to 35 years, I had great fun working as an active neuroscientist based in a large psychiatric institution, the Douglas, affiliated to McGill University. In partnership with a family of graduate students and fellows, we studied basic mechanisms involved in diseases of the brain such as schizophrenia, mood disorders, chronic pain and dementia. Collaboration with psychiatrists and neurologists ensured that we were continuously questioning ourselves as to the potential clinical relevance of our research findings. Our major findings were in the field of neuropeptides and their roles in mood disorders and pain, as well as interactions between phenotypes of the Alzheimer's brain suggesting a multi-targeted approach toward the development of truly effective therapies. In parallel to these activities, I played major roles in the administration of sciences both at McGill and at the Canadian Institutes of Health Research (CIHR). More recently, I was appointed as the first Chief Scientist of Quebec overseeing all aspects of research and innovation for the province. Certainly most challenging as covering all fields from music and religion to mathematics, engineering and health. During my talk, I will briefly summarize my current activities focusing on the rather unique opportunities offered by a career in science.

GRADUATE STUDENT PRESENTATIONS

9:00AM - 10:15AM

PRESENTER <i>Supervisor</i>	PROGRAM	TITLE	PAGE
James Benoit <i>Dr. Andrew Greenshaw & Dr. Serdar Dursun</i>	Ph.D.	<i>Imaging high-risk adolescents: Assessing myelin deficits with DTI</i>	11
Huining Guo <i>Dr. Xin-min Li & Dr. Glen Baker</i>	M.Sc.	<i>Desvenlafaxine prevents anxiety/depression like behaviors in an unpredictable chronic mild stress mouse model of depression: the role of myelin and oligodendrocytes</i>	12
Yanlin Wang <i>Dr. Satyabrata Kar</i>	Ph.D.	<i>Roles of insulin-like growth factor 2 receptor and lysosomal enzymes in Alzheimer's disease pathology</i>	13

FACULTY PRESENTATIONS

1:00PM - 2:00PM

PRESENTER <i>Department</i>	POSITION	TITLE	PAGE
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Dr. Junhui Wang <i>Department of Psychiatry</i>	Post-Doctoral Fellow	<i>Regulation of astrocyte pathology by fluoxetine prevents the deterioration of Alzheimer phenotypes in an APP/PS1 mouse model</i>	15

STUDENT POSTER PRESENTATIONS 1&2

10:15AM - 11:45AM

2:00PM - 3:00PM

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2	Jiyun Chung <i>Dr. Satyabrata Kar</i>	M.Sc.	<i>Effect of endosomal cholesterol accumulation on the metabolism of amyloid precursor protein in cultured N2A cells</i>	17
3	Ericson Dametto <i>Dr. Andrew Greenshaw & Dr. Serdar Dursun</i>	Ph.D.	<i>Risk behavior in adolescents and the anterior cingulate</i>	18
4	Bahram Foroutanpay <i>Dr. Satyabrata Kar</i>	M.Sc.	<i>Effects of Ab treatment on APP processing in cultured N2A cells</i>	19
5	Farrel Greenspan <i>Dr. Peter Silverstone</i>	M.Sc.	<i>What treatments are available for childhood sexual abuse, and how do they compare?</i>	20
6	Deena Hamza <i>Dr. Peter Silverstone & Dr. Andrew Greenshaw</i>	M.Sc.	<i>A comparative review of treatment outcomes for alcohol use disorders in inpatient and outpatient settings</i>	21
7	Michal Juhas <i>Dr. Andrew Greenshaw & Dr. Serdar Dursun</i>	Ph.D.	<i>Alcohol use disorder-related differences in resting state functional brain connectivity</i>	22
8	Yasmeen Krameddine <i>Dr. Peter Silverstone</i>	Ph.D.	<i>Changing behavior and attitudes: Police and the homeless mentally ill</i>	23
9	Marnie MacKay <i>Dr. Serdar Dursun</i>	Ph.D.	<i>Translational behavioral analysis of acute L-argininine and antipsychotic treatment in a phencyclidine model of psychosis</i>	24

Poster #	Presenter <i>Supervisor</i>	Program	Title	Page
10	Manoj Malik <i>Dr. Andrew Greenshaw</i>	Ph.D.	<i>Machine learning analysis of neuroimaging datasets in Alzheimer's disease</i>	25
11	Dmitriy Matveychuk <i>Dr. Glen Baker</i>	Ph.D.	<i>Elevation of rat brain levels of L-tyrosine by phenelzine, an effect mediated by its metabolite β-Phenethylidinedihydrochloride</i>	26
12	Michael Mazo <i>Dr. Kathy Hegadoren & Dr. Esther Fujiwara</i>	Ph.D.	<i>Delineating the relationship between self-trust and generalized self-efficacy</i>	27
13	Dimitar Ourdev <i>Dr. Satyabrata Kar</i>	M.Sc.	<i>Effects of Ab treatment on APP processing in cultured N2A cells</i>	28
14	David Rossolatos <i>Dr. Katherine Aitchison</i>	M.Sc.	<i>Exploring interactions between COMT, BDNF, AKT1 and cannabis consumption in the genesis of psychosis</i>	29
15	Anna Sasse <i>Dr. Satyabrata Kar</i>	M.Sc.	<i>Increased levels and activity of cathepsin B and D in kainate-induced toxicity</i>	30
16	Sudhakar Sivapalan <i>Dr. Katherine Aitchison</i>	M.Sc.	<i>Cognitive function due to metabolic syndrome</i>	31
17	Zohaib Siddiqi <i>Centre for Neuroscience</i>	Ph.D.	<i>The effect of chronic pain on cognition and mental health</i>	32

DEPARTMENT OF PSYCHIATRY AWARDS

GENERAL DEPARTMENTAL AWARDS

Roger C. Bland Graduate Award in Psychiatric Research

This award is offered annually to a graduate student who has demonstrated excellence in psychiatric research. This award consists of \$1000 and a certificate bearing the recipient's name.

Hassan F.A. Azim Award

The Azim award is offered annually to a graduate student (including psychiatric residents enrolled in the M.Sc./Ph.D. program) who has demonstrated excellence in psychotherapy or psychopharmacology research with direct implications for clinical practice. The award will consist of \$750.00 and a certificate bearing the recipient's name.

RESEARCH DAY AWARDS FOR PRESENTATIONS BY RESEARCH TRAINEES

Gordon King Research Prize

This award is named in honour of Dr. Gordon King, a child psychiatrist with a strong interest in psychopharmacology. Preference will be given to research trainee presentations in child psychiatry or psychopharmacology, but any area of research is eligible. The award will consist of \$500 and a certificate bearing the recipient's name.

Geoff Hopkinson Memorial Award

This award is named in memory of Dr. Geoff Hopkinson, a long-time member of the Department of Psychiatry. Dr. Hopkinson was particularly interested in the training of psychiatry residents and graduate students. Preference for the award will be given to research trainee projects on the neurobiology of psychiatric disorders, but any area of psychiatric research is eligible. The award will consist of \$500 and a certificate bearing the recipient's name.

Glen Baker Award

This award is named after Dr. Glen Baker, a former Chair of Psychiatry and a former Canada Research Chair who is currently a University of Alberta Distinguished University Professor. The award can be given for any area of psychiatric or neurologic research conducted by a research trainee and will consist of \$500 and a certificate bearing the recipient's name.

JUDGES PANEL

Dr. Jacqueline Cummine

Dr. Esther Fujiwara

Dr. Dawn Kingston

Dr. Rebecca Marsh

IMAGING HIGH-RISK ADOLESCENTS: ASSESSING MYELIN DEFICITS WITH DTI.

James R.A. Benoit¹ Andrea T. Shafer² Matthew R.G. Brown¹ Andrew J. Greenshaw¹ Serdar M. Dursun¹ Sunita Vohra³ Florin Dolcos⁴ Anthony Singhal⁵

¹ Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

² Department of Neuroscience, University of Alberta, Edmonton, AB, Canada

³ Department of Pediatrics, University of Alberta, Edmonton, AB, Canada

⁴ Department of Psychology, University of Illinois, Champaign, IL, USA

⁵ Department of Psychology, University of Alberta, Edmonton, AB, Canada

Adverse childhood experiences slow the development of the diameter of white matter axons and microtubule structure, and decrease the ratio of myelinated to unmyelinated fibers in the brain's white matter tracts. The brain is becoming viewed more as a complex system of networks, with each network relying on white matter integrity to function optimally. Diffusion MRI provides a means of measuring the integrity of these white matter tracts. In this study, Diffusion Tensor Imaging (DTI) scans were used to investigate whether complex psychiatric symptoms are correlated with impaired white matter development. Single-scan DTI data from 20 adolescent inpatients from CASA House Edmonton were compared to 20 pair-matched (age/gender/handedness) controls. Patients were diagnosed with at least two Axis-I (DSM-IV TR) disorders; patients with any neurological disorder (e.g., FAS, Tourette's) at the time of scanning were excluded from analysis. Tract integrity analysis was carried out using Tract-Based Spatial Statistics. This approach extracts Fractional Anisotropy (FA) maps from the brain, which indicate white matter tracts. These are combined by group and compared for differences at the core of each tract. Differences between groups suggest a significant difference in tract integrity. Following statistical corrections and confirmation of tract locations from a neurologist, we saw decreases in patient white matter integrity, localized to three tracts. Adolescent inpatients had significantly impaired white matter development in three tracts compared to control subjects. This suggests underdeveloped white matter could act as a marker for complex psychiatric disorders, and potentially as a target for assessing long-term treatment response.

DESVENLAFAXINE PREVENTS ANXIETY/DEPRESSION-LIKE BEHAVIORS IN AN UNPREDICTABLE CHRONIC MILD STRESS MOUSE MODEL OF DEPRESSION: ROLE OF MYELIN AND OLIGODENDROCYTES

Huining Guo¹, Junhui Wang¹, Xin-Min Li¹

¹ Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

Serotonin-norepinephrine reuptake inhibitors (SNRIs) are a class of antidepressant drugs that exert their primary effects by rapidly increasing the serotonin and norepinephrine in synaptic clefts. Studies show it takes at least 2-3 weeks for the mood-enhancement despite the immediate increase of the monoamine neurotransmitter levels, indicating other mechanisms underlying their therapeutic effects. Here, we investigated the role of white matter in the pathogenesis and treatment of depression in an unpredictable chronic mild stress (UCMS) mouse model. Desvenlafaxine (DVS), a relatively new SNRI drug, was used in this study to explore the specific relevance of white matter and depression. We found DVS prevented UCMS induced anxiety/depression-like behavioural changes in elevated plus maze test, sucrose preference test, tail suspension test and forced swimming test. No significant changes were found in protein markers of neurons and astrocytes between control and UCMS groups. However, myelin basic protein (MBP), 2,3-cyclic nucleotide 3-phosphodiesterase (CNPase) and platelet-derived growth factor alpha-receptors (PDGFR α), all of which are myelin and oligodendrocyte markers, were significantly reduced in UCMS group. And DVS could attenuate these proteins decrease in mouse brain. These findings suggest that DVS may alleviate the depressive endophenotype in UCMS mouse by ameliorating myelin damages. While providing evidence for novel mechanism of DVS, our study may also contribute to the understanding of the important role of myelin and oligodendrocytes in the pathogenesis of depression.

INSULIN-LIKE GROWTH FACTOR-II RECEPTOR AND LYSOSOMAL ENZYMES: RELEVANCE TO ALZHEIMER'S DISEASE PATHOLOGY

Yanlin Wang^{1,2}, David Westaway² and Satyabrata Kar^{1,2}

¹ Department of Psychiatry; University of Alberta, Edmonton, Alberta, Canada,

² Centre for Prions and Protein Folding Disease, University of Alberta, Edmonton, Alberta, Canada

The insulin-like growth factor-II (IGF-II) receptor is an important regulator of the endosomal-lysosomal (EL) system involved in the transport of newly synthesized lysosomal enzymes cathepsins B and D from the trans-Golgi network to endosomes. Evidence suggests that up-regulation of certain lysosomal enzymes within lysosomes can prevent sub-lethal damage, whereas sustained release of the enzymes into cytosol can induce cell death via cytochrome c release from mitochondria. However, very little is known about functional interrelationship between the IGF-II receptor and lysosomal enzymes and their significance in Alzheimer's disease (AD). Since EL system is critical in the generation of β -amyloid ($A\beta$) peptides, which play important role in the degeneration of neurons and development of AD pathology, we hypothesize that release/activation of lysosomal enzymes may participate in $A\beta$ -mediated toxicity and development of AD pathology.

We used human $A\beta$ 1-42-induced primary mouse cortical neuronal death model to evaluate the levels/activities and subcellular distributions of IGF-II receptor and lysosomal enzymes cathepsin B and D during neurodegeneration. The levels of cathepsins and IGF-II receptor were increased with time during $A\beta$ 1-42-induced neuronal death. The cytosolic levels of cathepsins were associated with enhanced levels of Bcl-2-associated X protein and cleaved caspase-3. Inhibitors of lysosomal enzymes were able to protect neurons against $A\beta$ -induced toxicity. Our experiments with transgenic mice overexpressing $A\beta$ also showed an increased levels of IGF-II receptor and cathepsins in the vulnerable cortical region of the brain compared to control mice. These results, taken together, suggest a direct role for cathepsins B and D in AD pathogenesis.

HIV/AIDS AND NEUROPSYCHIATRY: AN EVOLVING LEOPARD'S COAT

Christopher Power¹

¹ Division of Neurology, Department of Medicine, University of Alberta, Edmonton, AB, Canada

HIV-1 infects the brain immediately after primary infection although the neuropsychiatric manifestations of HIV-1 infection are widely assumed to be apparent only in the later stages of disease, particularly after the development of AIDS. However, more recent studies suggest that the HIV-Associated Neurocognitive Disorder (HAND) becomes evident prior to AIDS, particularly Asymptomatic Neurocognitive Impairment in adults and developmental delay in children. In the Alberta NeuroAIDS Cohort of HIV/AIDS patients, symptomatic HAND represents ~20% of infected persons, evident as cognitive, motor and behavioural impairments. Data from our laboratory and others indicate the mechanisms underlying the development of HAND include both the direct effects of HIV-1 proteins including Vpr and Nef as well as chronic neuroinflammation. Diagnostic tools for HAND include neuroimaging and neuropsychological testing although new technologies such as microRNA levels in blood and CSF are emerging. Combination antiretroviral therapy is the cornerstone for treating HIV/AIDS although the exact combination of antiretroviral drugs for HAND treatment remains unclear. Other neuroprotective therapies are being developed in our laboratory and are in early clinical trials.

Disclosures: none

Funding: CIHR

REGULATION OF ASTROCYTE PATHOLOGY BY FLUOXETINE PREVENTS THE DETERIORATION OF ALZHEIMER PHENOTYPES IN AN APP/PS1 MOUSE MODEL

Junhui Wang^{1,2}, Hongxing Wang³, Yu Song³, Kelly Hartle², Huining Guo², Jiming Kong³, Qingjun Huang¹ and Xin-Min Li¹

¹ Mental Health Center, Shantou University, Shantou, Guangdong, China

² Department of Psychiatry, Faculty of Medicine and Dentistry, University of Alberta, Edmonton, AB, Canada

³ Department of Anatomy and Cell Science, Faculty of Medicine, University of Manitoba, Winnipeg, MB, Canada

Animal studies and postmortem brain analyses have implicated astrocytic dysfunction in Alzheimer disease (AD) pathophysiology. However, roles of astrocyte in the pathophysiology and treatment of the disease are poorly characterized. Here we identified astrocyte as an independent key factor involved in several Alzheimer disease-like phenotypes in an APP/PS1 mouse model, including impaired memory and cognition, altered neuronal, synaptic properties and deteriorated amyloid pathology. We observed notable plaque deposition, astrocyte accumulation and synaptic protein loss in the mouse brain. After isolated and incubated in in vitro condition, astrocytes from the APP/PS1 mice caused synaptotoxicity and reduced dendritic complexity and axon branching of hippocampal neurons. Furthermore, we revealed these astrocytes produced high levels of soluble β -amyloid (A β 40 and A β 42) which can be significantly inhibited by an antidepressant, fluoxetine (FLX), via activating serotonin 5-HT₂ receptors. Consequently, FLX protected the cultured hippocampal neurons against the astrocyte-induced synaptotoxicity and neurotoxicity. Moreover, in the same APP/PS1 mouse, FLX inhibited activation of astrocytes, lowered A β products, ameliorated neurotoxicity and improved the behavioral performances. These results highlight the important roles of astrocytes in producing neurotoxic A β and offer a potential avenue for AD treatment.

POST CRITICAL-PERIOD RELATIONAL INTERVENTION FOR ATTACHMENT RELATED TRAUMA

Chandra Kirn Ashton¹, Peter H. Silverstone¹

¹ Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

Advances in neuroscience, molecular biology, and genomics have converged on three compelling conclusions. Firstly, that our bodies are constructed by early experience, secondly, that the body's stress response systems, cardiovascular and immune systems, and metabolic regulatory controls, are all significantly disrupted by early adversity, and lastly, that these disruptions can lead to life-long impairments in physical and mental health. Drawing on these multiple streams of investigation, this study seeks to gain support from widespread evidence of the ubiquitous impacts of early attachment related stressors, offering intriguing insights into mechanisms that link early adversity to later impairments in learning, behaviour, and both physical and mental well-being. What we hope to understand goes beyond the long-term impact of early adverse experience and considers interventions that strengthen caregiving relationships, reduce parent/child stress, and teach new strategies for relational mediation of behaviour. We plan to examine their effectiveness in rebuilding the developing brain, subsequent attachment relationships, and over all mental health. Though emphasis has traditionally been placed on early intervention, this research looks to examine if meeting the needs of children at any age can have the long-term benefit of eradicating the neurological and relational effects of maltreatment and toxic stress. Our hypothesis is that by strengthening protective relationships we help to mitigate the effects of toxic stress caused by attachment trauma on the developing brain, even after the deleterious effects have occurred. We hope to understand what makes intervention effective with this population in order to guide future research.

EFFECT OF ENDOSOMAL CHOLESTEROL ACCUMULATION ON THE METABOLISM OF AMYLOID PRECURSOR PROTEIN IN CULTURED N2A CELLS

Jiyun Chung¹, Mahua Maulik², Satyabrata Kar^{1,2}

¹ Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

² Centre for Prions and Protein Folding Diseases, University of Alberta, Edmonton, AB, Canada

Alzheimer's Disease is thought to be initiated by abnormal aggregation of β -amyloid ($A\beta$)-related peptides. Connection between cholesterol and $A\beta$ synthesis has been suggested by a number of studies. Some have reported that a rise in cholesterol level and/or redistribution influences AD pathology by increasing the production of $A\beta$ -related peptides from the β - and γ -secretase-mediated processing of Amyloid Precursor Protein (APP).

Our study investigates the role of cholesterol in APP processing using cultured N2a cells treated with U18666A, a class II amphiphile which triggers redistribution of cholesterol to the endosomal-lysosomal system. Differential influence of U18666A treatment on three variations of N2a cell lines – wild type (N2awt) or transfected with either wild type human APP (APPwt) or human APP containing Swedish mutation (APPsw) – in media containing 0%, 5%, or 10% fetal bovine serum is examined. Our results, obtained so far, indicate that U18666A treatment differentially increases the levels of APP, APP-CTFs, α -secretase ADAM10, and components of γ -secretase (Nicastrin, PEN 2, and PS1) in the three cell lines. Changes in the levels of β -secretase, β -secretase activity, and extracellular $A\beta$ are currently under investigation. These results suggest that redistribution of cholesterol into the endosomal-lysosomal system increase the levels and possibly the processing of APP, leading to increased production of $A\beta$ -related peptides

RISK BEHAVIOR IN ADOLESCENTS AND ANTERIOR CINGULATE.

Ericson Dametto¹, Matthew Brown¹, Marnie MacKay¹, James Benoit¹, Manoj Malik¹, Michal Juhas^{1,2}, Serdar Dursun^{1,2}, Andrew Greenshaw¹

¹ Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

² Alberta Health Services, Edmonton, AB, Canada

The Youth Risk Behavior Surveillance System monitors six categories of priority health-risk behaviors among youth and young adults: 1) behaviors that contribute to unintentional injuries and violence; 2) tobacco use; 3) alcohol and other drug use; 4) sexual behaviors that contribute to unintended pregnancy and sexually transmitted diseases (STDs), including human immunodeficiency virus (HIV) infection; 5) unhealthy dietary behaviors; and 6) physical inactivity. Adolescence is characterized by the rapid development of biological and psychological systems. Interactions with social agents (family and peers) allow a transition to the adult life. Peers promote positive or negative influences, according to the characteristic of the group and the personal decisions of the adolescent. In early adolescence (10-14 years), the amount of time spent with peers increases and peer relationships typically become the primary social context that influences social development. Peer relationships during this period provide companionship and they act as primary venues for gaining status, as well as providing opportunities for self-disclosure while seeking independence from family. The hypothesis is that risk behavior in adolescents is linked to sadness-related brain regions rather than to imbalanced in the reward-related brain regions. The Anterior Cingulate Cortex is related to detection of errors and depression. It is an important brain area in risk behavior according to studies in: neuroimaging, ontogenesis and phylogenesis. The literature review of the Anterior Cingulate Cortex supports the role of this brain area in processing sadness and decision making and suggests that behavior risk in adolescents may be associated to depression.

EFFECTS OF A β TREATMENT ON APP PROCESSING IN CULTURED N2A CELLS.

Bahram V. Foroutanpay^{1,2}, Tim J. Revett², David Vergote², David Westaway², and Satyabrata Kar^{1,2}

¹ Department of Psychiatry, University of Alberta, Edmonton, Alberta, Canada

² Centre for Prions and Protein Folding Diseases, University of Alberta, Edmonton, AB, Canada

Alzheimer's disease (AD), the most common cause of senile dementia and a growing global medical concern, is characterized by neuronal loss, tau-positive neurofibrillary tangles and amyloid- β (A β) containing neuritic plaques in selected brain regions. A β peptides are a family of proteins that are considered to contribute to AD pathogenesis. These peptides are derived from Amyloid Precursor Protein (APP) via sequential processing by β - and γ -secretases. Although much work has been focused on the interactions between A β and neurons, the potential role of A β in APP processing is relatively unclear. Here we aimed to elucidate the effects of A β peptide on APP processing using cultured neuronal N2a cells. Through use of RT-PCR, we have shown that APP transcription is increased after A β treatment. Further analysis indicates an increase in APP and APP-CTFs levels in a time- and dose-dependent manner, however, the levels of secretases do not appear to alter following A β treatment. Similarly, our ELISA results have shown an increase in A β levels both in cell lysates and conditioned media. As a follow up, we will examine activity of β - and γ -secretases, as well as the subcellular localization of the various components involved in the cellular processing of APP and generation of A β . These experiments will provide an insight into the molecular mechanisms by which A β can induce a feed-forward pathway for the proteolytic processing of APP, which may have relevance in the degeneration of neurons and subsequent development of AD pathology.

WHAT TREATMENTS ARE AVAILABLE FOR CHILDHOOD SEXUAL ABUSE, AND HOW DO THEY COMPARE?

Farrel Greenspan¹, Andreia G. Moretzsohn¹, Peter H. Silverstone¹

¹ Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

Child sexual abuse (CSA) is frequent, with rates for significant abuse estimated at 15-20% of the female population and 8-10% of the male population. Such CSA frequently leads to significant short-term and long-term sequelae including a multitude of psychiatric conditions such as post-traumatic stress disorder, anxiety disorder, and depression. However, treatment of CSA remains uncertain, with even the most widely recommended types of treatment, cognitive behavioural therapy (CBT) and trauma-focused cognitive behavioural therapy (TF-CBT), having not always been found to be statistically beneficial in studies of adult survivors. Furthermore, treatment of children and youth has been even less well researched. Many types of treatment have been recommended, including CBT, TF-CBT, eye movement desensitization and reprocessing (EMDR), play therapy, art therapy, and pet therapy. The aim of this review is to examine the various treatments recommended for CSA to date, and determine whether one specific treatment or a combination of treatments may be the most appropriate therapeutic approach for child and youth victims of CSA.

A COMPARATIVE REVIEW OF TREATMENT OUTCOMES FOR ALCOHOL USE DISORDERS IN INPATIENT AND OUTPATIENT SETTINGS

Deena M. Hamza¹, Peter H. Silverstone¹

¹ Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

Substance use disorders (SUDs), including alcohol use, have serious implications for individuals and the general public including increased risk of violence, motor vehicle collisions and crime. The treatment of these conditions can occur through inpatient and outpatient settings. Treatment programs vary in duration, care setting, goal intensity, and psychoeducation; however, research into differences between inpatient and outpatient care has produced conflicting results. Outpatient care offers variable intensity and duration and may produce similar outcomes as intense inpatient programs, yet there is evidence that individuals with concomitant psychiatric conditions and unstable environments require rigorous monitoring and structure to be successful. Based on existing literature, a “one-size-fits-all” approach to SUDs is unlikely to be effective, and program characteristics and patient characteristics must both be considered when planning treatment.

ALCOHOL USE DISORDER-RELATED DIFFERENCES IN RESTING STATE FUNCTIONAL BRAIN CONNECTIVITY

Michal Juhas¹, Matthew Brown¹, Marnie MacKay¹, James Benoit¹, Timothy Gillese¹, Ericson Dametto¹, Allan Aubry², Glenn Walmsley², Blayne Blackburn², Cindy King², Liana Urichuk², Mark Loowell², Serdar Dursun^{1,2}, Andrew Greenshaw¹

¹ Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

² Alberta Health Services, Edmonton, AB, Canada

Alcohol use disorder (AUD) is a global health problem affecting over 140 million people worldwide. Dynamic brain changes associated with alcohol dependence, recovery, and different treatments are only partially understood. We compared changes in functional connectivity of resting state networks in patients undergoing treatment for AUD. Our objective was to identify changes in the resting state networks caused by chronic alcohol abuse. 20 male patients (age 24-63) with AUD (DSM-IV TR) were recruited 5-10 days after detoxification and scanned (4.7 Tesla Varian system) before and after a 21-day residential treatment. 10 healthy volunteers matched for age, handedness, and education level were scanned for comparison. Both scanning sessions included an anatomical scan, a resting-state functional magnetic resonance scan, and a diffusion tensor scan. The functional data was analyzed using an independent component and region of interest analyses with SPM8, GIFT, MarsBaR and a custom Matlab code. Our analysis revealed significant changes in several resting state networks including the core and frontal networks. In comparison to controls, patients had significant differences in functional connectivity between anterior cingulate cortex and different somatosensory, motor, visual, and association regions. These findings suggest changes in functional connections of anterior cingulate cortex in AUD patients before and after supervised treatment. Due to the role of anterior cingulate cortex in modulating execution of appropriate and suppression of inappropriate responses in reward anticipation and impulse control, these results could help us better understand dynamic changes in functional connectivity which are closely associated with addiction, craving, and internal conflict resolution.

CHANGING BEHAVIOUR AND ATTITUDES: POLICE AND THE HOMELESS MENTALLY ILL

Yasmeen I. Krameddine¹, Peter H. Silverstone¹

¹ Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

Police officers constantly interact with individuals exhibiting varying forms of mental illness. This interaction lacks understanding by both parties and is poorly researched. For these reasons a novel training program was created in close collaboration with the Edmonton Police Service focusing on respect, empathy, communication and de-escalation techniques during interactions with individuals suffering from different psychiatric disorders. Measurements of police attitudes (n=108) and behaviour at baseline, 6 month post training and 2.5 years post training have been collected as well as surveys concerning vulnerable and homeless persons attitudes towards the police, 2 years post training (n= 213). Results of police data show no change in attitudes but significant improvements in behaviour of police officers including increases in empathy, communication, verbal de-escalation, proficiency, decrease in negative outcomes and cost-savings 6 months post training. Results of homeless and vulnerable data show the overall level of satisfaction after police interactions was positive in 31%, neutral in 7%, and negative in 62% of cases with the large majority of these individuals suffering from self-reported mental illness (78%). Quantitative analysis shows that being arrested leads to increased negative attitudes towards police while qualitative analysis demonstrates a significant factor leading to the level of satisfaction is the portrayed level of respect individuals receive from officers. Given these findings of increases in police efficiency, decrease in costs related to mental health calls, and need for respect, empathy, communication and de-escalation training, we advocate that this training should be broadly and continuously implemented in law enforcement services.

TRANSLATIONAL BEHAVIORAL ANALYSIS OF ACUTE L-ARGININE AND ANTI-PSYCHOTIC TREATMENT IN A PHENCYCLIDINE MODEL OF PSYCHOSIS

Marnie B. MacKay¹, Hugh A. Semple², John C. Lind³, Andrew J. Greenshaw¹, Glen B. Baker¹, Serdar M. Dursun¹

¹ Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

² Alberta Innovates-Technology Futures, Vegreville, AB, Canada

³ Centre for Psychiatric Assessment and Therapeutics (CPAT), Alberta Hospital Edmonton, Alberta Health Services, Edmonton, AB, Canada

Schizophrenia is a complex illness that often requires combined treatments to control its symptoms. Unfortunately, many patients remain symptomatic. The opportunity exists to identify safe and well-tolerated treatments that can be used with antipsychotic medications that will improve efficacy. Glutamate/nitric oxide (NO)-based therapies may offer an alternative approach as a target for drug development.

The phencyclidine (PCP) animal model of psychosis is an established pharmacological model of schizophrenia. PCP produces a behavioral response that is considered relevant to the clinical presentation of the illness. Acute PCP administration in animals increases locomotor activity, a useful indicator of the ability of PCP to produce psychosis in humans.

In this experiment, we examined the dose-response relationship of L-arginine (a NO precursor) to PCP-induced hyperlocomotion in rats, as well as the dose-response of clozapine and risperidone. The effect of synergistic augmentation between these antipsychotic medications and L-arginine on PCP-induced hyperactivity was also measured.

Acute dosing of L-arginine alone and synergistic augmentation between L-arginine and antipsychotic medications did not decrease PCP-induced hyperlocomotor activity in animals, an index of positive symptoms of schizophrenia. Consistent with results from a previous clinical trial, we also found no significant changes in the Positive and Negative Syndrome Scale (PANSS) positive symptoms scores at the end of augmenting L-arginine treatment in patients with schizophrenia.

The translation of findings from preclinical and clinical data will increase our understanding of the role that NO may play in psychosis and the validation of its potential as a target for future drug development in schizophrenia.

MACHINE LEARNING ANALYSIS OF NEUROIMAGING DATASETS IN ALZHEIMER'S DISEASE:

Manoj K. Malik¹, Michal Juhas¹, James R.A. Benoit¹, Ericson Dametto¹, Matthew R.G. Brown¹, Serdar M. Dursun¹, Andrew J. Greenshaw¹

¹ Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

Alzheimer's disease (AD) is a progressive neurodegenerative disease, clinically predominantly presenting with progressive deficits in memory, and cognition. The precise etio-pathogenesis of AD remains unclear. According to the estimates of the Alzheimer society of Canada, about 0.5 million Canadians suffer from AD or related dementia. AD imposes not only a significant financial burden on our health care system but also an immeasurable human suffering to the patients and their families. For my research project, I am intending to utilize large-scale databases of advanced neuroimaging datasets including structural magnetic resonance imaging, diffusion tensor magnetic resonance imaging, and positron emission tomography brain scans. I am planning to apply machine learning to compare and contrast brains of healthy controls, patients suffering from mild cognitive impairment, and patients with AD. Using multi-voxel pattern analysis with the help of SPM8, Weka, and custom Matlab code, I intend to devise advanced computer algorithms with diagnostic and prognostic potential. The diagnostic tool will classify brain imaging data into healthy, mild cognitive impairment, or AD. The prognostic tool will focus on predicting the progression of mild cognitive impairment to full syndromal AD.

My research project has the potential to elucidate key morphological and functional brain changes associated with progression of AD and related dementias. The machine learning tools I will develop will help clinicians make faster, more accurate diagnoses and prognostic estimates, which should improve the treatment outcomes for patients suffering from AD.

ELEVATION OF RAT BRAIN LEVELS OF L-TYROSINE BY PHENELZINE, AN EFFECT MEDIATED BY ITS METABOLITE β -PHENYLETHYLIDENEHYDRAZINE (PEH)

Dmitriy Matveychuk¹, Emerson Nunes¹, Nasir Ullah², Fahad S. Aldawsari², Carlos A. Velázquez-Martínez² and Glen B. Baker^{1,2}

¹ Neurochemical Research Unit, Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

² Faculty of Pharmacy and Pharmaceutical Sciences, University of Alberta, Edmonton, AB, Canada

The antidepressant and anxiolytic drug phenelzine is an inhibitor of monoamine oxidase (MAOI) as well as a substrate for the enzyme, resulting in the formation of the metabolite β -phenylethylenedihydrazone (PEH). PEH is a weak MAOI but shares phenelzine's effects on brain amino acids, increasing levels of GABA, alanine and ornithine and decreasing levels of glutamine. When animals are pre-treated with another MAOI, the effects on the aforementioned amino acids are abolished for phenelzine, but not for PEH, suggesting that they are mediated by the MAO-catalyzed formation of PEH. We are reporting that phenelzine and PEH also increase rat whole brain concentrations of L-tyrosine, an effect that is mediated by PEH.

We employed HPLC with electrochemical detection to quantitate rat whole brain levels of L-tyrosine following administration of phenelzine and E- and Z- geometric isomers of PEH (30 mg/kg i.p.). To determine whether this effect on L-tyrosine was MAO-dependent, rats were pre-treated with the MAOI tranylcypromine prior to administration of phenelzine or racemic PEH.

Rat whole brain levels of L-tyrosine were significantly elevated by phenelzine and both geometric isomers of PEH at 3 and 6 hours following drug administration, reaching approximately 265-305% of vehicle-treated controls at 3 hours. Pre-treatment with tranylcypromine reversed the increase in L-tyrosine for phenelzine, but not for PEH, suggesting that PEH is responsible for the tyrosine-elevating property of phenelzine. Since L-tyrosine is the precursor for synthesis of the catecholamines dopamine and noradrenaline, PEH may be a useful adjunctive drug in a number of neurological and psychiatric disorders where there is a functional deficiency of catecholamines.

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DELINEATING THE RELATIONSHIP BETWEEN SELF-TRUST AND GENERALIZED SELF-EFFICACY

Michael Mazo¹, Kathleen M. Hegadoren², Esther Fujiwara¹

¹ Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

² Faculty of Nursing, University of Alberta, Edmonton, AB, Canada

Generalized self-efficacy (GSE) comprises a stable and trait-like belief that one can manage the challenges one is faced with, irrespective of the domain in which these challenges are presented. The association between GSE and outcomes has been studied extensively in the realm of mental and physical health, stress and quality of life. Low GSE is correlated with risk factors for mental illness such as high state and trait anxiety as well as depressive symptoms.

While the consequences of high or low GSE have been studied in various domains, less is known about personality traits underlying GSE. For that reason, I explore whether self-trust might be a component of self-efficacy. Self-trust is defined here as the unquestioned acceptance of one's thoughts, feelings, and emotions as valid indicators of the individual's subjective experience. The relationship between the two constructs is delineated in the context of 1) person variables such as age, gender, ethnicity and year in university 2) trait-anxiety, an indicator of mental health

The primary purpose of my thesis is to determine whether GSE and self-trust are distinguishable psychological constructs, and to clarify whether self-trust is psychologically relevant when considering variations in GSE. Should self-trust emerge as a component of GSE and predict risk factors of mental illness (trait-anxiety here) similarly to GSE, my findings may help inform the development of targeted interventions to increase self-trust. Knowing which person variables influence the link between self-trust and self-efficacy may further help identifying people in whom such interventions might be most/least effective.

EFFECTS INDUCED BY AMYLOID BETA 42 TREATMENT ON AMYLOID PRECURSOR PROTEIN PROCESSING IN U373 ASTROCYTES

Dimitar Ourdev^{1,2}, Bahram Foroutanpay^{1,2}, Satyabrata Kar^{1,2}

¹ Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

² Centre for Prions and Protein Folding Diseases, University of Alberta, Edmonton, AB, Canada

Amyloid- β ($A\beta$) peptides are a family of proteins that are considered to be a principal aspect of Alzheimer's Disease (AD), the most common cause of senile dementia, and a growing global medical concern. These peptides result from the proteolytic processing of Amyloid Precursor Protein (APP) by the sequential cleavage of enzymes known as secretases. Once these peptides are released into the brain parenchyma, they aggregate into soluble oligomers and subsequently form $A\beta$ plaques, which potentiate a variety of neurotoxic effects that lead to the disease state. Although much work has been focused on the various interactions between $A\beta$ plaques and neurons, the relationship between soluble $A\beta$ peptides and astrocyte cells has received relatively little attention. Through the use of human astrocytoma cell line U373, we investigated the effects induced by $A\beta_{42}$ treatment on the cellular expression of APP and its proteolytically generated products CTFs, $A\beta_{40}$, and $A\beta_{42}$. In conjunction with these experiments, we are examining the relative cellular levels and activity of secretase enzymes, as well as the localization of the various components involved in the cellular processing of APP via immunostaining. We report here that $A\beta_{42}$ treatment increased the expression of APP and cleaved products within astrocytes in a time-dependent manner. These experiments will provide insight into the effect of soluble $A\beta_{42}$ treatment on astrocytes, thus further contributing to the understanding of how AD potentiates its progressive neurodegenerative effects.

EXPLORING INTERACTIONS BETWEEN COMT, BDNF AND AKT1 AND CANNABIS CONSUMPTION IN THE GENESIS OF PSYCHOSIS

David Rossolatos,¹ Yabing Wang¹, Brodie Heywood¹, Beatriz Carvalho Henriques¹, Darren Bugbee,² Aleksandra Dimitrijevic,² Alexandra Lovelock,³ Carol Bolt⁴ Georgina Macintyre^{1,2,6} Phil Tibbo,⁵ Katherine J Aitchison,^{1,3,6,7} Scot E Purdon,^{1,3,4}

¹ Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

² Department of Medicine, University of Alberta, Edmonton, AB, Canada

³ Edmonton Early Psychosis Intervention Clinic, Edmonton, AB, Canada

⁴ Neuropsychology, Alberta Hospital Edmonton, Edmonton, AB, Canada

⁵ Nova Scotia Psychosis Research Unit, Dalhousie University, Halifax, NS, Canada

⁶ Department of Medical Genetics, University of Alberta, Edmonton, AB, Canada

⁷ King's College London, Institute of Psychiatry, MRC Social Genetic and Developmental Psychiatry Centre, London, UK

Background: Although psychosis is a relatively treatable condition, the mechanisms that trigger psychotic symptoms are still being elucidated. For example, it is known that the consumption of substances such as cannabis can induce psychosis, but how this interacts with other factors such as genetic vulnerability still requires further exploration and replication in a variety of samples. If genetic vulnerabilities to cannabis exposure were better understood, appropriate measures in public health education could be taken. It is known that COMT, BDNF, and AKT1 are amongst candidates for genes leading to susceptibility to psychosis following cannabis use (Decoster et al, 2012; van Winkel et al, 2011; Di Forti et al, 2012).

Methods: In this study, we are seeking to explore the role of markers in the above candidate genes and cannabis use in a sample of patients with psychosis recruited in Edmonton and Halifax. The markers are rs4680 (Val158Met) in COMT, rs2494732 in AKT1, and rs6265 (Val66Met) in BDNF. Data on substance use and other relevant variables including cognition have been collected.

Results: The proportion of our sample that has used cannabis in their lifetime is approximately 70%, with a smaller proportion having problematic substance use. Genotyping of patients for the COMT, AKT1, and BDNF variants is complete.

Conclusions: It will be interesting to see whether findings identified in European Caucasians are reproducible in our Albertan and Nova Scotia samples, while the analysis of genetic moderation of cognitive dysfunction in the context of cannabis use represents a novel approach.

INCREASED LEVELS AND ACTIVITY OF CATHEPSINS B AND D IN KAINATE-INDUCED TOXICITY

Anna Sasse¹, Mayukh Banerjee¹, Yanlin Wang¹, Mahua Maulik^{1,2} and Satyabrata Kar^{1,2}

¹ Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

² Centre for Neuroscience, University of Alberta, Edmonton, AB, Canada

Administration of kainic acid induces acute seizures that result in the loss of neurons, gliosis and reorganization of mossy fiber pathways in the hippocampus resembling those observed in human temporal lobe epilepsy. At present, mechanisms underlying the degeneration of neurons following administration of kainic acid remain unclear. Since lysosomal enzymes cathepsins B and D are known to be involved in the loss of neurons we evaluated their potential roles in kainic acid-treated rats. We also measured the levels/expression of insulin-like growth factor-II (IGF-II) receptors, which mediate the trafficking of these enzymes, in kainic acid-treated rats. Our results showed that administration of kainic acid evoked loss of neurons along with hypertrophy of astrocytes and microglia in the hippocampus of the adult rat brain. The levels and activity of cathepsins B and D increased in the hippocampus of kainic acid-treated rats compared to control rats. The expression of both cathepsins was also markedly increased in activated astrocytes and microglia of the treated rats. Additionally, the cytosolic levels of cathepsins were enhanced along with cytochrome c and to some extent Bax in the hippocampus of kainic acid-treated rats. These changes were accompanied by the appearance of cleaved caspase-3-positive neurons in the hippocampus of treated rats. The levels of IGF-II receptors were not significantly altered, but these receptors were present in a subset of reactive astrocytes kainic acid-treated rats. These results suggest that enhanced levels/expression and activity of lysosomal enzymes may have a role in the loss of neurons in kainic acid-treated rats.

COGNITIVE FUNCTION DUE TO METABOLIC SYNDROME

Sudhakar Sivapalan¹, Katherine J. Aitchison,^{1,2} Scot E. Purdon^{1,3}

¹ Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

² Department of Medical Genetics, University of Alberta, Edmonton, AB, Canada

³ Neuropsychology Service, Alberta Hospital - Edmonton, Edmonton, AB, Canada

There are many factors that contribute to the development of cognitive dysfunction. Dyslipidemia, hypo- and hyperglycemia, obesity, and hypertension are a few of the factors that are individually associated with cognitive dysfunction. Elements of these together also form the criteria for metabolic syndrome. Metabolic syndrome is present in about 24% of the general population, and its prevalence increases with age. It is directly associated with an increased risk of developing cardiovascular disease and diabetes, both of which in turn again impact cognitive function.

The relevance of metabolic syndrome to the psychiatric population has also been established, especially amongst those being treated with antipsychotic medications. However, more recent studies seem to indicate that simply having severe mental illness(SMI) may also put one at increased risk of developing metabolic syndrome. Given this association, it is important to consider the cognitive impact on these individuals. Executive functioning, attention and memory have been implicated as affected areas of cognitive functioning. Deficits in these areas affect the ability of the individual to maintain their own mental well-being. Individuals presenting with SMI are usually relatively young, and these complications are life-long, leading to increased mortality and morbidity. A review of the current literature looking at the cognitive impact due to metabolic syndrome will be presented.

THE EFFECT OF CHRONIC PAIN ON COGNITION AND MENTAL HEALTH

Zohaib Siddiqi^{1,2}, Camille Olechowski^{1,2}, Michelle Verrier², Saifee Rashiq², Bruce D. Dick^{1,2}

¹ Centre for Neuroscience, University of Alberta, Edmonton, AB, Canada

² Department of Anaesthesiology and Pain Medicine, University of Alberta, Edmonton, AB, Canada

Objective: Chronic Intractable Pain patients are often diagnosed with anxiety and depression. Some studies show that these patients have a cognitive disruption as well, manifesting as a deficiency in attention and memory. Our objective was to investigate the link between chronic pain and cognitive dysfunction while also looking at relationships with depression, anxiety, and sleep.

Methods: 29 adult chronic pain patients have been examined. The Test of Everyday Attention (TEA), Reading Span Test, and Spatial Span Test were used to examine cognition. A battery of questionnaires was administered to examine other aspects of life. A regression analysis was used to compare the scores of the cognitive tests to those of the questionnaires.

Results: Out of all participants, we didn't find any relationship between pain level and cognition. However, when analyzing only the 11 participants with impaired scores, we found that the best predictor of performance on the spatial span orientation task was pain chronicity in years (-0.723) and on the spatial span mirror task was the sensory score on the MPQ (-0.621).

Discussion: We did not find a correlation between pain and cognition in the total number of participants but we found a relationship in those participants with disabled cognitive scores. This replicates previous results that have shown that chronic pain patients perform the same as controls on easy tasks but become greatly deficient on difficult tasks.

Conclusion: Our preliminary results show a link between pain and cognition only in patients with scores in the disabled range in the TEA.