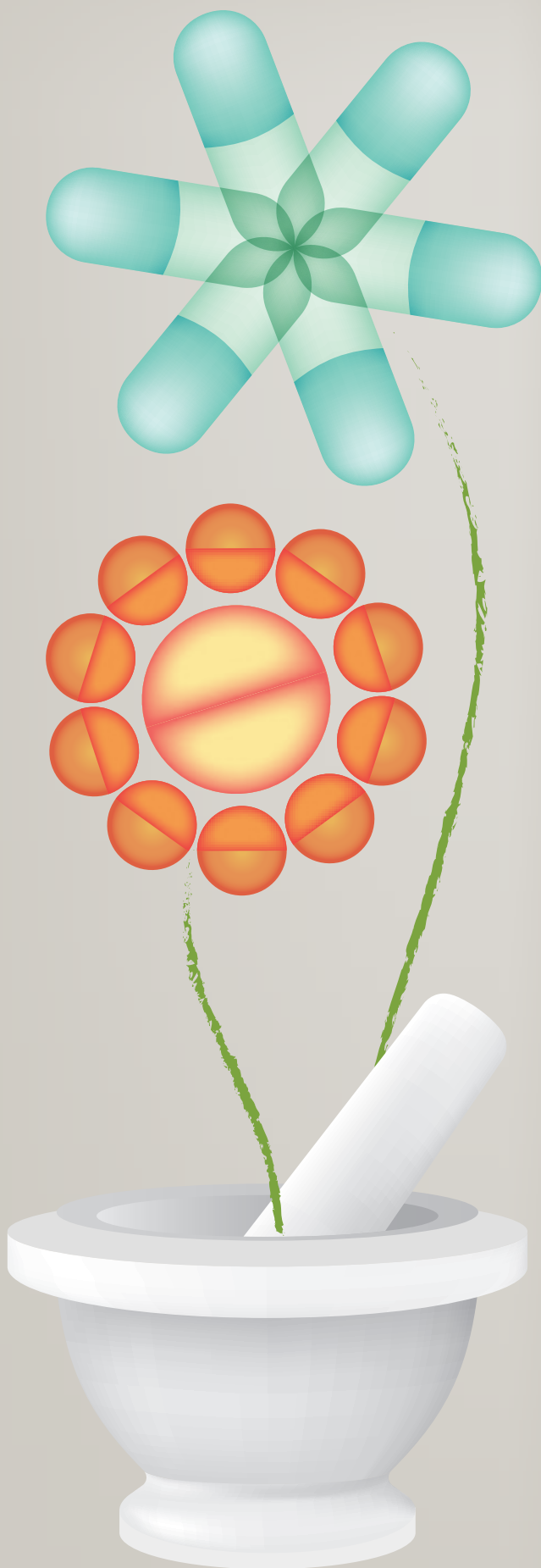




Collaboration

Across Pain Science & Practice

APRIL 11 • 2019

4th Annual Translational Neuroscience Center (CTNT) Symposium

Neurobiological Disease Mechanisms

Keynote: Endogenous Opioids in Chronic Pain and Pleasant Touch

M. Catherine Bushnell, Ph.D.

Scientific Director, National Center for Complementary and Integrative Health at NIH



APRIL 12 • 2019

2nd Annual Integrative Medicine Symposium

Combating the Opioid Crisis: Integrative Approaches for Pain Management

Keynote 1: Integrative Health and Medicine: A New Essential Partner in Effective Pain Management

Margaret A. Chesney, Ph.D.

Distinguished Professor and Past Director of the Osher Center for Integrative Medicine, University of California San Francisco

Keynote2: Natural Products and Chronic Pain

Bruce A. Watkins, Ph.D.

Research Professor, University of California Davis
Emeritus Professor, Purdue University



Panel Discussion

Herbal Tea and Essential Oil Stations

Art Exhibit and Posters

Afternoon Interactive Interprofessional Workshops

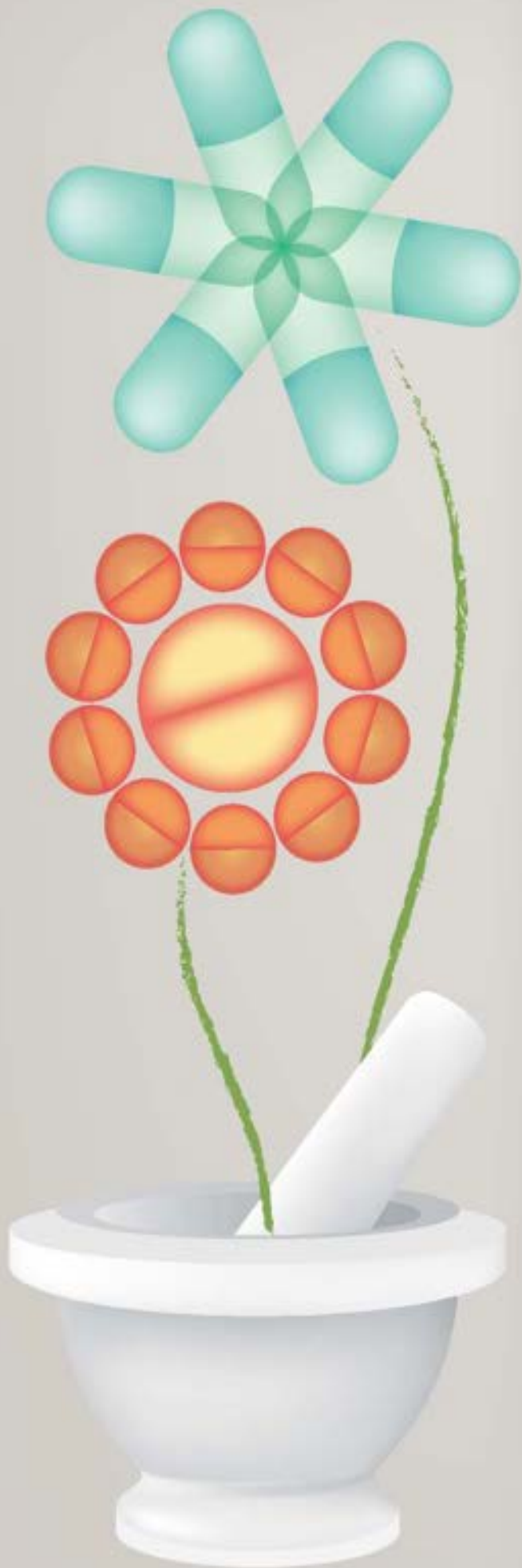
To Register: <https://ttuhsc-integrative-medicine-2019.eventbrite.com>

Sponsored by

Division of Integrative Medicine, Center of Excellence for Integrative Health, Office of Interprofessional Education, Laura W. Bush Institute for Women's Health and Center for Excellence for Translational Neuroscience and Therapeutics

For more information, please contact us at ipe@ttuhsc.edu or call 806-743-2028





Program Content Information

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Thank you to all participants and attendees.



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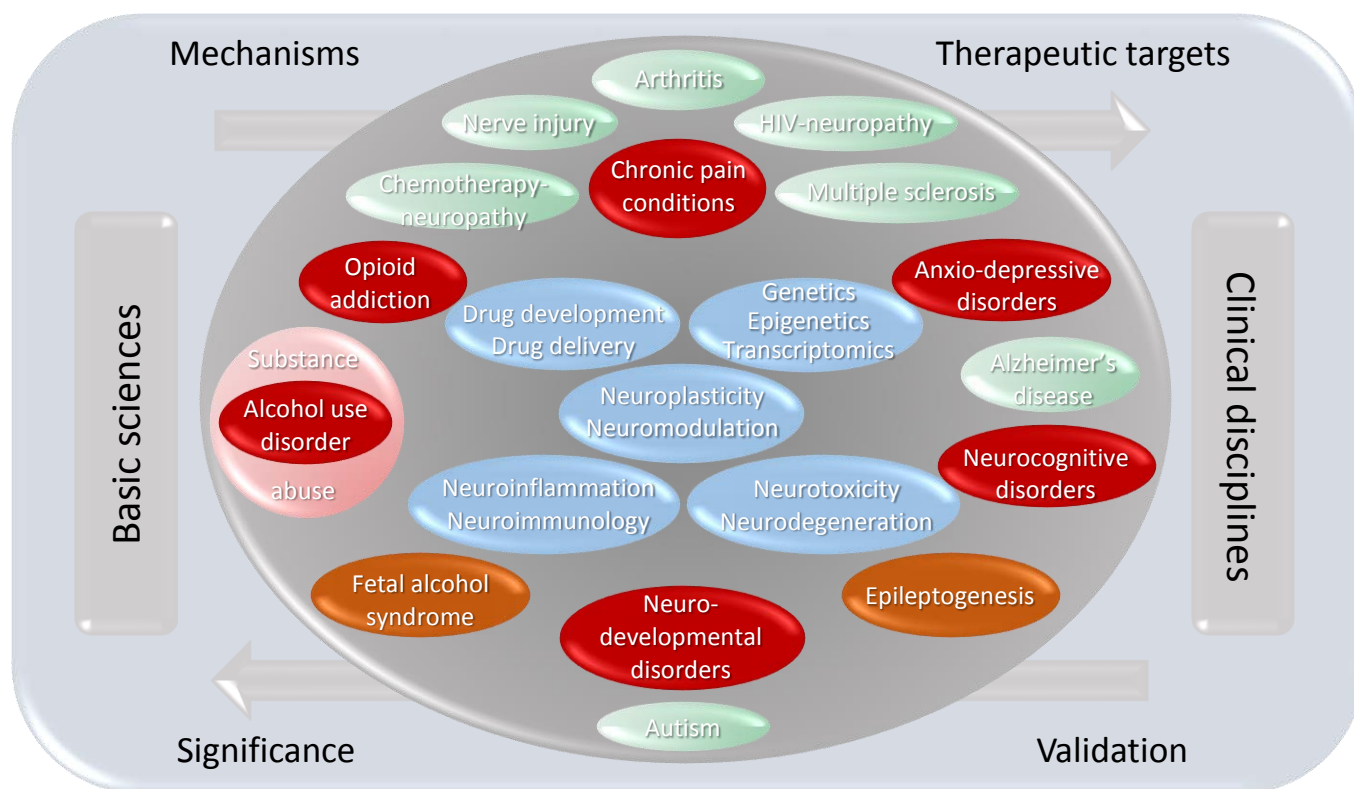
TEXAS TECH UNIVERSITY
HEALTH SCIENCES CENTER™

Center of Excellence for Translational
Neuroscience and Therapeutics

4th Annual Research Symposium

Thursday, April 11, 2019

Translational Research



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April 11, 2019

Welcome to the *Fourth Annual Symposium of the Center of Excellence for Translational Neuroscience and Therapeutics (CTNT)*. The Center continues to bring together basic scientists and clinicians to stimulate scholarly activities, facilitate collaborations, and generate translational research projects.

Through regular meetings and resources available to collaborative research teams, CTNT members have successfully developed clinically relevant research projects and grant applications in the areas of chronic pain, alcohol use disorder, neurodegenerative disorders, multiple sclerosis, epileptogenesis, autism and other conditions related to nervous system plasticity and dysfunctions.

Commitment and support from Dr. Steven Berk, Dean of the School of Medicine and Executive Vice President and Provost, TTUHSC, Drs. Jannette Dufour and Leslie Shen, former and current Associate Deans for Research, School of Medicine, TTUHSC continue to drive our collaborative efforts bridging basic science and clinical entities and are greatly appreciated. We would also like to thank Dr. Tedd Mitchell, TTUHSC President, for enabling us to acquire a state-of-the-art multiphoton system for in vivo and ex vivo imaging studies; based in our *Neurophysiology Core* it is available for collaborative research projects.

This year's theme on pain science and practice is timely in the face of the current opioid crisis, and we are excited about teaming up with the *Center of Excellence for Integrative Health* to join their *Annual Integrative Medicine Symposium*. The key note lecture, translational research presentations by CTNT teams of basic scientists and clinicians, and posters by trainees in basic science and clinical disciplines provide unique opportunities for faculty and trainees to interact and learn about ongoing translational research and scholarship activity in our Center.

It is a great pleasure to welcome this year's *Key Note Speaker*, Dr. Catherine Bushnell, Scientific Director Division of Intramural Research National Institutes of Health/NCCIH, who has kindly agreed to share with us recent advances in the field of opioid research in her lecture and in meetings with basic science and clinical CTNT teams.

A special thank you goes to our CTNT Coordinator, Tiffany Hancock, for creating the program and organizing the event with valuable support from the Integrative Medicine Symposium Organizing Team. We are also very thankful to our colleagues who generously agreed to serve as judges for the poster session.

We hope that the symposium activities will stimulate new collaborative efforts. Thank you for your interest and participation in our Center activities!



Volker Neugebauer, M.D., Ph.D.

Director, Center of Excellence for Translational Neuroscience and Therapeutics

CTNT Opportunities

The Center of Excellence for Translational Neuroscience and Therapeutics (CTNT) serves to bridge preclinical “basic science” research and the clinical setting to generate and disseminate new knowledge, tools and strategies for the diagnosis and treatment of nervous system disorders (<http://www.ttuhschool.edu/centers-institutes/translational-neuroscience-therapeutics>).

Founded in 2015 and based in the Department of Pharmacology and Neuroscience, School of Medicine, TTUHSC, our Center has grown to 40 members from 17 different departments or institutes at TTUHSC and TTU, including faculty doing basic preclinical research in 7 departments and working with patients or human subjects in 10 departments (<http://www.ttuhschool.edu/centers-institutes/translational-neuroscience-therapeutics/leadership-members.aspx>).

In 2018, our members published >60 articles, gave >40 invited talks, submitted 1 provisional patent application and 27 grant applications, and received \$9.7 Mio. (\$4.7 Mio. direct costs) in funding for 15 grants.

CTNT provides the expertise and infrastructure for multidisciplinary translational research projects through the Annual Symposium, CTNT Research Meetings and Grant Development Program (Translational Research Club), the Translational Neuroscience and Pharmacology Lecture Series, Seed Funds for collaborative translational research and scholarly activity by teams of basic science and clinical faculty. A Neurophysiology Core Laboratory based in the Department of Pharmacology and Neuroscience includes electrophysiology systems for in vivo and ex vivo preparations, a battery of state-of-the-art behavioral assays, optogenetic tools for in vivo and ex vivo studies, and a multiphoton system for in vivo and ex vivo imaging.

CTNT support and core facilities are available for collaborative research projects by teams of basic scientists and clinicians to generate translational scholarly activities and external funding to support the mission of the Center and TTUHSC. CTNT maintains and is developing close relationships with other Centers and Institutes such as the Garrison Institute on Aging and the new Texas Tech Mental Health Institute.

CTNT Organization and Membership

Director

Volker Neugebauer, M.D., Ph.D.
Professor and Chair, Dept. of Pharmacology & Neuroscience, TTUHSC
Executive and Chief Scientific Officer, Garrison Institute on Aging

Steering Committee

Susan Bergeson, Ph.D.
Associate Professor, Dept. of Pharmacology & Neuroscience, TTUHSC

Vadivel Ganapathy, Ph.D.
Professor and Chair, Dept. of Cell Biology and Biochemistry

George Henderson, Ph.D.
Professor, Dept. of Pharmacology & Neuroscience, TTUHSC

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Terry McMahon, M.D.
Professor and Chair, Dept. of Psychiatry, TTUHSC

Office

Tiffany Denton
CTNT Coordinator, Unit Manager, Dept. of Pharmacology & Neuroscience
Office: 3601 4th Street, Room 5C192, Lubbock, TX 79430- 6592
Phone: 806-743-4008

Members

Karina Alvina, PhD
Assistant Professor, Dept. of Biological Sciences, TTU

Mirla Avila, MD
Associate Professor, Dept. of Neurology, TTUHSC

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Luis Cuello, PhD

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Nadezhda German, PhD

Assistant Professor, Department of Pharmaceutical Sciences, TTUHSC - Amarillo

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Josee Guindon, DVM, PhD

Assistant Professor, Dept. of Pharmacology and Neuroscience, TTUHSC

Petar Grozdanov, Ph.D., M.B.(ASCP)

Research Assistant Professor, Dept. of Cell Biology and Biochemistry, TTUHSC

George Henderson, PhD

Professor, Dept. of Pharmacology and Neuroscience, TTUHSC

Byungkwan Hwang, MD

Resident, Dept. of Neurology, TTUHSC

Michaela Jansen, PhD

Associate Professor, Dept. of Cell Physiology and Molecular Biophysics, TTUHSC

Guangchen Ji, PhD

Research Assistant Professor, Dept. of Pharmacology and Neuroscience, TTUHSC

Andrey Karamyshev

Assistant Professor, Dept. of Cell Biology and Biochemistry, TTUHSC

Shin Ye Kim, PhD

Assistant Professor, Dept. of Psychology, TTU

Irene La-Beck, PharmD

Assistant Professor, Dept. of Immunotherapeutics & Biotechnology, TTUHSC-Abilene

Hongjun Liang, Ph.D.

Assistant Professor, Dept. of kinesiology and Sports Management, TTU

Hui Ying Luk, Ph.D., CSCS*D

Professor, Dept. of Pharmacology & Neuroscience, TTUHSC

Terry McMahon, MD

Professor and Chair, Dept. of Psychiatry, TTUHSC

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Director Emeritus, Texas Tech Neuroimaging Institute (TTNI), Professor, Dept. of Electrical and Computer Engineering, TTU

J. Josh Lawrence, PhD

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Volker Neugebauer, MD, PhD

Professor and Chair, Dept. of Pharmacology and Neuroscience, TTUHSC

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Professor, Dept. of Human Development and Family Studies, Associate Dean for Research, College of Human Science, TTU

Jie Pan, MD

Resident, Dept. of Neurology TTUHSC

Arubala Reddy, PhD

Research Assistant Professor, Dept. of Internal Medicine, TTUHSC

Hemachandra Reddy, PhD

Professor, Dept. Internal Medicine, TTUHSC

Natalia Schlabritz-Lutsevich, MD, PhD

Associate Regional Dean for Research, Associate Professor Dept. Obstetrics and Gynecology, TTUHSC - Permian Basin

Adam Schmidt, Ph.D.

Assistant Professor, Dept. of Psychological Sciences, TTU

Leslie Shen, PhD,

Professor, Dept. of Pathology, Director, Center of Excellence for Integrative Health, TTUHSC

Heather Vellers, PhD, RCEP

Professor, Dept. of Kinesiology and Sport Management, TTU

Henrik Wilms, MD, PhD

CH-Foundation Chair in Parkinson's Disease, Research Associate Professor, Dept. of Neurology. TTUHSC

Time	Activities	Location (TTUHSC ACB, Lubbock)
10:00-11:30	Poster Presenter Registration	ACB Lobby
12:00-12:30	Opening Remarks and Introduction of ADK and Keynote Speaker: Drs. Quentin Smith, Tom Tenner and Volker Neugebauer (Pizza lunch provided)	ACB 100
12:30-1:30	Keynote Lecture: Dr. M. Catherin Bushnell “Endogenous Opioids in Chronic Pain and Pleasant Touch”	ACB 100
01:30-02:30	CTNT Research Group Presentations	ACB 100
01:30-01:50	Research Group Presentation 1: Dr. Volker Neugebauer “Novel opioid actions in the amygdala”	ACB 100
01:50-02:10	Research Group Presentation 2: Dr. Susan Bergeson “Neuro- inflammation as a target for alcohol use disorder”	ACB 100
02:10-02:30	Research Group Presentation 3: Dr. Natalia Schlambitz- Lutsevich “Developmental programming of pain-related spectrum disorders: focus on endogenous cannabinoids”	ACB 100
02:30-04:30	Poster Session	ACB Lobby
04:30-05:00	Refreshment Break	ACB Lobby
05:00-05:30	Awards Ceremony: Dr. Leslie Shen	ACB 100



M. Catherine Bushnell, Ph.D.

Scientific Director

Division of Intramural Research

National Institutes of Health/NCCIH

Dr. Bushnell is the Scientific Director of the National Center for Complementary and Integrative Health at the National Institutes of Health in Bethesda, Maryland. She holds a Ph.D. in Experimental Psychology from the American University and received postdoctoral training in neurophysiology at the NIH. She was the Harold Griffith Professor of Anesthesia at McGill University before returning to NIH in 2012. Among her honors are the Lifetime Achievement Award from the Canadian Pain Society and the Frederick Kerr Basic Science Research Award from the American Pain Society. Her research interests include forebrain mechanisms of pain processing, psychological modulation of pain, and neural alterations in chronic pain patients.

CTNT Collaborative Research Presenters



Susan Bergeson, Ph.D.

Associate Professor, Pharmacology and Neuroscience
Texas Tech University Health Sciences Center, Lubbock

Dr. Bergeson received her Ph.D. in Biochemistry and Molecular Biology from the Oregon Health and Science University in 1998. She began studies on Alcohol and Substance Use Disorders as an undergraduate student and has dedicated her career to better understanding the addiction process. With the availability of high throughput genomic analyses, Dr. Bergeson's research contributed to a previously underappreciated role for the innate immune system. She and her collaborators tested compounds known to inhibit neuroinflammation, which showed reductions in alcohol consumption levels, the severity of alcohol withdrawal, and alcohol-mediated pain sensitization. Her current work includes investigational new drug development for pharmacotherapeutic treatment of AUD.

Neuroinflammation as a target for alcohol use disorder

A role for innate immune system dysfunction in myriad neuroinflammatory and neurodegenerative disorders has recently garnered attention. Much excitement revolved around numerous reports of minocycline 'repurposed' to improve conditions in a wide variety of clinically relevant illnesses. Minocycline, a second-generation semi-synthetic derivative of tetracycline, is commonly prescribed as an antibiotic for numerous bacterial infections, and has now been shown to possess anti-apoptotic, anti-inflammatory, anti-oxidant and immunomodulatory effects through a variety of mechanisms, including in the CNS. We completed structure-function tests and used the crystal structures of tetracycline analogs bound to the A-site in the bacterial ribosome to design a new compound, which was predicted to retain its anti-inflammatory action with loss of antimicrobial properties. The purpose was to reduce GI disturbance and risk of viral-mediated disease severity for long-term use to treat Alcohol Use Disorder (AUD) and other inflammatory disease processes. Our chemically modified minocyclines (CMM) were tested for antimicrobial activity. CMM1 has been tested in both our murine and porcine AUD models and a clear retention of efficacy to reduce risky alcohol consumption was shown.



Volker Neugebauer, MD, Ph.D.

Professor and Chair, Pharmacology and Neuroscience
Texas Tech University Health Sciences Center

Dr. Neugebauer is Professor and Chair of the Department of Pharmacology and Director of the Center of Excellence for Translational Neuroscience and Therapeutics (CTNT) at the Texas Tech University Health Sciences Center (TTUHSC). He was recently appointed as the Executive Director and Chief Scientific Officer of the Garrison Institute on Aging (GIA). Dr. Neugebauer received broad training in physiology, pharmacology, neuroscience and neurology at the University of Würzburg, Germany and the University of Texas Medical Branch in Galveston, TX; he joined TTUHSC in 2014. Dr. Neugebauer directs a research program on higher brain functions and dysfunctions in chronic pain and related neuropsychiatric conditions that has been continuously funded by NIH for the past two decades. The analysis of emotional-affective and cognitive brain mechanisms of pain (centered on the amygdala) is a key contribution of his work to the field of pain research and neuroscience. Collaborative research projects explore brain mechanisms of neurodegenerative disorders including Alzheimer's disease, alcohol use and addiction disorders, comorbidities with depression and anxiety disorders, epileptogenesis, and aging-related health issues in general through innovative research, education, and community outreach. The goal is the better understanding of disease mechanisms and development of novel therapeutic strategies to improve quality of life and healthy aging.

Novel opioid actions in the amygdala

Volker Neugebauer, Matthew Hein, Vadim Yakhnitsa, Takaki Kiritoshi, Guangchen Ji, Edit Navratilova, Frank Porreca Opioids are generally powerful analgesics but have undesirable side effects such as tolerance and addiction. Opioid use disorder has emerged as a critical health care issue. Opioids act on different receptors in pain modulatory systems.

Recent evidence suggests that kappa- and mu-opioid receptors (KOR and MOR) have opposing effects on pain control and reward systems in the brain. The amygdala, a brain area critically involved in negative emotions, has also emerged as an important pain modulatory center and is rich of KOR and MOR. Our new hypothesis is that in the amygdala activation of KOR promotes pain by disinhibiting amygdala output. Conversely, blockade of KOR in the amygdala with antagonists inhibits amygdala-dependent pain sensory and affective consequences of pain. Behavioral and electrophysiological data support the novel concept that KOR antagonists represent an effective analgesic option in chronic pain without liabilities of MOR agonists such as morphine and could actually inhibit addictive effects of MOR agonists. This is because in contrast to opioid agonists, antagonists are only effective where and when the receptors are pathologically activated.

Supported by NIH grants R01NS038261 and R01NS106902; Pain Research Challenge Award - Virginia Kaufman Endowment Fund and Clinical & Translational Science Institute, Univ. Pittsburgh; Crofoot Presidential Endowment in Epilepsy; Giles McCrary Endowed Chair in Addiction Medicine; Mildred and Shirley L. Garrison Chair in Aging; South Plains Foundation; Center of Excellence for Translational Neuroscience and Therapeutic



Natalia Schlabritz-Lutsevich, MD, Ph.D.

Associate Professor, Obstetrics and Gynecology

Texas Tech University Health Sciences Center, Permian Basin

Dr. Schlabritz-Lutsevich received her medical degrees (MD) from Belarus state medical University (Belarus) and University of Kiel (Germany). She received her PhD in Medicine in 1997. Her post-doctoral Fellowships included Institute of Hormone and Fertility Research (Germany) and University of Tennessee Health sciences Center (USA). Her research interests are comparative reproductive physiology in normal and pathological pregnancies; mechanism of developmental and maternal health programming in maternal and environmental adverse conditions. Dr. Schlabritz-

Lutsevitch research goal is to connect basic

and clinical sciences and to create interdisciplinary approaches to solve medical problems. Her research accomplishments include discovery of melatonin receptors in human uterus, development of novel models of maternal alcohol binge drinking and pre-eclampsia, discovery of novel *Brucella* subspecies in non-human primates, application of RAMAN spectroscopy and optoacoustic as point of care diagnostic tools and description of fetal endocannabinoid deficiency in maternal obesity

Developmental Programming of Pain-Related Spectrum Disorder: Focus on Endogenous Cannabinoids.

Cannabinoids have been used for treatment of chronic pain for millennia with documented results of numerous clinical trials. Exogenous cannabinoids act through the mechanism of “kick-starting” the components of the *endogenous cannabinoid system (endocannabinoid (eCB) system, ECS)*. ECS is a pharmacological target for the treatment of obesity, inflammation, cardiovascular and neuronal damage and pain. First described by Russo in 2004, the concept of Clinical Syndrome of Endocannabinoid Deficiency (CECD) spectrum disorders has been developed applied first to such conditions as irritable bowel syndrome (IBS) and fibromyalgia. CECD is linked to numerous pain-related conditions. IBS involves chronic constipation/diarrhea with painful abdominal spasms. Based on the presence of both cannabinoid receptors and their ligands in the gut, it has been suggested that cannabinoid derivatives could be used to treat IBS. Additionally such chronic pain conditions as migraines, chronic pelvic pain, and anxiety are part of this syndrome. All these conditions as well as depression and PTSD are treatable with cannabinoids. The clinical definition of the syndrome is important, since it leads to the therapeutic application of the cannabinoids derivatives to its treatment. Remarkably, diseases, related to CECD are documented in the offspring of obese mothers (effect, known as a developmental programming). We hypothesized, that maternal obesity will be associated with the decreased level of fetal endogenous cannabinoids. We believe, that while the spectrum of adverse offspring health conditions programmed by MO is likely to be broad, it includes a spectrum of conditions that are defined as the clinical endocannabinoid deficiency syndrome. In addition, circulating eCB concentrations are increased in obese individuals. We have shown that in contrast to maternal values fetal circulating eCB concentrations are reduced in MO in human pregnancy and placental eCB content is decreased in a baboon over-feeding MO model. So that fetal conditions could be termed as fetal endocannabinoid deficiency. Animal models represent an opportunity to dissect specific mechanisms related to the various dietary patterns that result in MO and provide important data for the development of interventional strategies in humans.

Poster Judges

Michael Blanton, Ph.D.
Pharmacology and Neuroscience
TTUHSC, Lubbock

Hui Ying Luk, Ph.D.
Kinesiology and Sport Management
TTU, Lubbock

Gaungchen Ji, Ph.D.
Pharmacology and Neuroscience
TTUHSC, Lubbock

Clint MacDonald, Ph.D
Cell Biology and Biochemistry
TTUHSC, Lubbock

Michael O'Boyle, Ph.D.
Development and Family Studies
TTU, Lubbock

Josee Guindon, DVM, Ph.D
Pharmacology and Neuroscience
TTUHSC, Lubbock

Michaela Jansen, Ph.D.
Cell Physiology and Molecular Biophysics
TTUHSC, Lubbock

Andrey Karamyshev, Ph.D.
Cell Biology and Biochemistry
TTUHSC, Lubbock

Volker Neugebauer, MD, Ph.D
Pharmacology and Neuroscience
TTUHSC, Lubbock

Natalia Schlabritz-Lutsevich, M.D., Ph.D Human
Obstetrics and Gynecology
TTUHSC, Permian Basin

Poster Presentation Times

1. Basic Science – Graduate/Undergraduate

Posters	Time	Presenter	Judges
1	2:45-3:00	Afroz, Kazi Farhana	Drs. Michael Blanton/Hui Ying Luk
2	2:45-3:00	Blanton, Henry	Drs. Guangchen Ji/Michael O'Boyle
3	2:45-3:00	Edwards, Hunter	Drs. Josee Guindon/Andrey Karamyshev
4	2:45-3:00	Hein, Matthew	Drs. Michaela Jansen/Natalia Schlabritz-Lustevich
5	2:45-3:00	Jodeiri-Farshbaf, Mohammad	Drs. Clint MacDonald/Volker Neugebauer
6	3:00-3:15	Hernandez, Sarah	Drs. Michael Blanton/Hui Ying Luk
7	3:00-3:15	Mazzitelli, Mariacristina	Drs. Michaela Jansen/Natalia Schlabritz-Lutsevich
8	3:00-3:15	Raut, Snehal	Drs. Guangchen Ji/Michael O'Boyle
9	3:00-3:15	Songhe, Li	Drs. Josee Guindon/Andrey Karamyshev

2. Basic Science – Medical Student

Posters	Time	Presenter	Judges
10	3:15-3:30	D'Souza, Preston	Drs. Michael Blanton/Hui Ying Luk
11	3:15-3:30	Parmar, Kanak	Drs. Guangchen Ji/Michael O'Boyle
12	3:15-3:30	Presto, Peyton	Drs. Josee Guindon/Andrey Karamyshev

3. Basic Science – Postgraduate

Posters	Time	Presenter	Judges
13	3:00-3:15	Castro-Piedras, Isabel	Drs. Clint MacDonald/Volker Neugebauer
14	3:15-3:30	Kumar, Subodh	Drs. Clint MacDonald/Volker Neugebauer
15	3:15-3:30	Fowzia, Selina	Drs. Clint MacDonald/Volker Neugebauer
22	3:45-4:00	Rahman, Mizanur	Drs. Guangchen Ji/Michael O'Boyle

4. Clinical – Undergraduate/Graduate Student

Posters	Time	Presenter	Judges
23	3:45-4:00	Adifei-Mosi, Jenniger	Drs. Josee Guindon/Andrey Karamyshev

5. Clinical – Medical Student

Posters	Time	Presenter	Judges
16	3:30-3:45	Aldrete, Jonathan	Drs. Michael Blanton/Hui Ying Luk
17	3:30-3:45	Keith, Hanson	Drs. Guangchen Ji/Michael O'Boyle
18	3:30-3:45	Kopacz, Avery	Drs. Josee Guindon/Andrey Karamyshev
19	3:30-3:45	Lee, Suheng	Drs. Clint MacDonald/Volker Neugebauer
20	3:30-3:45	Merugumala, Praveen Dev	Drs. Michaela Jansen/Natalia Schlabritz-Lutsevich

6. Faculty Posters *(Will not be judged)*

Posters	Presenter
22	Dr. Khalid Benamar
23	Dr. Clint MacDonald
24	Dr. Igor Ponomarev
25	Dr. Betsy Jones

1. Basic Science – Graduate/Undergraduate

Afroz, Kazi Farhana

Parental Elevated Salt Consumption in Mice and the Development of Autism Spectrum Disorder (ASD)-like Behavior in the Offspring

Kazi Farhana Afroz, Kajal Parikh, Varsha Mishra, Shree Patel, Noah Reyes, Karina Alvina

Department of Biological Sciences

Texas Tech University, Lubbock, TX USA

Autism Spectrum Disorder (ASD) is a prevalent neurodevelopmental disorder with no known etiology or cure. Several possible contributing factors, including both genetic and environmental, are being currently investigated. Amongst the environmental factors, recent studies have identified parental immune dysregulation as being potentially involved in promoting ASD in the offspring. Indeed, animal studies using the maternal immune activation (or MIA) mouse model have shown ASD-like behavior in the offspring, being mediated by maternal immune dysregulation. Higher ASD offspring were reported to the fathers who used have any disorder related to immune imbalance. Moreover, maternal dietary habits and gut microbiota have also been included as factors behind ASD. However, these studies have been mostly limited to high fat and high sugar diet. Several recent studies show that elevated salt consumption has a significant effect on the gut microbiota and immune system, facilitating gut dysbiosis and induction of pro-inflammatory pathways. Considering all these variables, we hypothesize that parental high-salt diet (HSD) induces gut dysbiosis and concomitant immune dysregulation, and ultimately contribute to the development of ASD in the offspring. To test our hypothesis, we fed male and female mice with high-NaCl chow and NaCl in their drinking water for 8- weeks while Control groups (CD) were fed with low NaCl chow and regular water. Then we paired HSD- or CD- fed males and females. The offspring from CD and HSD breeding pairs were then weaned and kept for behavioral analysis at 8 weeks-old. Our results show that only male mice from HSD-fed parents showed less social interaction, exploration, along with increased repetitive behaviors in comparison with the offspring from CD-fed mice, all signs indicative of ASD-like behavior in male mice. Interestingly, female mice did not show significant changes in social behavior. These results support the idea that parental HSD might increase the chances of ASD-like behaviors in the offspring, in a sex-dependent manner. We are currently investigating mechanisms underlying these findings, focusing on inflammatory markers and gut microbiota.

Blanton, Henry

Cannabinoid Receptor Agonists in Rodent Models of Chemotherapy Induced Peripheral Neuropathy and Ovarian Cancer

Henry Blanton, Jennifer Lilley, Jennifer Brelsfoard, Isabel Castro, Kevin Pruitt, and Josee Guindon

Department of Pharmacology and Neuroscience, Center of Excellence for Translational Neuroscience and Therapeutics, Department Immunology and Molecular Microbiology

Tech University Health Sciences Center, Lubbock, TX.

The search for novel, efficacious analgesics with minimal side effects and tolerance development has prompted research interest into the endocannabinoid system. At the center of the endocannabinoid system are two G-protein coupled receptors named CB1 and CB2. The CB1 receptor is expressed on presynaptic neurons in the CNS, while the CB2 receptor is expressed on peripheral tissue, immune cells, and glia. Activation of these receptors has been demonstrated in numerous pre-clinical studies to produce a analgesic effects. The analgesic effect mediated by CB1 receptor activation is limited in its potential clinical efficacy due

to its psychoactivity, and the development of tolerance to the analgesic effects. CB2 selective cannabinoids eschew these downsides associated with CB1 selective and mixed agonists, and are a promising novel treatment avenue for conditions including pain. In this study we compared CB1 selective and CB2 selective cannabinoid receptor agonists in mouse models of chemotherapy induced peripheral neuropathy and ovarian cancer in female mice. Our findings suggest that the clinical utility of CB2 selective compounds as analgesics, specifically in treatment of certain cancers, may be limited due to alterations in the female hormonal system.

Edwards, Hunter

*Mechanical compression in a microfluidic environment, a novel method for the study of traumatic nervous system injury in *Caenorhabditis elegans**

Hunter Edwards¹, Mizanur Rahman², Taslim Anupom³, Marton Toth², Hannah Jackson¹, Karina Alvina¹, Monica Driscoll⁴, and Siva Vanapalli²

¹Department of Biological Sciences, Texas Tech University, Lubbock, TX, ²Department of Chemical Engineering, Texas Tech University, Lubbock, TX, ³Department of Electrical Engineering, Texas Tech University, Lubbock, TX, ⁴Department of Molecular Biology and Biochemistry, Rutgers University, Piscataway, NJ

Traumatic brain injuries (TBIs) have become a leading health concern, with injuries often resulting in debilitating neurodegeneration and psychological disorders. TBI research, however faces many unique experimental challenges. TBIs vary greatly between each individual event, thereby making it difficult for researchers to develop reliable, reproducible methods to study the mechanisms of TBI related neurodegeneration at the cellular and whole organism levels. Moreover, growing evidence suggests that TBIs have far reaching whole body effects that may directly contribute to severity of TBI outcomes. *In vivo* studies using animal models are limited in sample size and in their ability to detect small scale molecular changes in real time. Likewise, *in vitro* studies utilizing neuronal cell cultures lack the capacity to capture holistic physiological effects from complex injury. Consequently, relatively little is known about the critical nature of underlying secondary physiological changes that follow primary head impact.

Here we explore the dose-dependent consequences of severe mechanical compression in *Caenorhabditis elegans* using a novel, microfluidic-based injury platform. Using our platform, we can track acute and long-term behavioral and mechanistic changes across the entire lifespan of worms, following a single episode or a repetitive injury. In a structured microenvironment, *C. elegans* undergo distinct, but temporary locomotory changes including but not limited to, paralysis and hyper-activity immediately following the delivery of a compressive stimulus. Importantly, we observe lasting physiological changes in animals subject to increasing severity of compression including a high frequency of matricidal hatching events, a marked increase in the presence of ALM exophers, and exophers-genesis at the PLM touch receptor neurons. Our results indicated that *C. elegans* has the potential to be used as a genetic model for studies of nervous system trauma and may directly inform translational efforts to decipher the mechanisms of TBI-related neurodegeneration.

Hein, Matthew A.

Kappa opioid receptor mediated disinhibition of amygdala CRF neurons

Matthew A. Hein¹, Vadim Yakhnitsa¹, Guangchen Ji^{1,2}, Edita Navratilova⁴, Frank Porreca⁴, Volker Neugebauer^{1,2,3} ¹Department of Pharmacology and Neuroscience, Texas Tech University Health Sciences, Lubbock, TX, USA

²Center of Excellence in Translational Neuroscience and Therapeutics, Texas Tech University Health Sciences Center, Lubbock, TX, USA

³Garrison Institute on Aging, Texas Tech University Health Sciences Center, Lubbock, TX, USA

⁴Department of Pharmacology, University of Arizona, Tucson, AZ, USA

Neuroplastic changes in the central nervous system have been implicated not only in pain conditions associated with an identifiable injury, but also in functional pain syndrome (FPS), in which the pain cannot be attributed to tissue pathology. Mechanisms of FPS remain to be determined, but these conditions are typically triggered by stress, which can create a chronic pain condition. Corticotropin releasing factor and its CRF1 receptor in the amygdala have been linked to emotional-affective behaviors and pain modulation. The amygdala is also a major site of opioid receptors, including $G_{i/o}$ -coupled kappa opioid receptors (KOR). KOR activation by its endogenous ligand dynorphin or agonists can have adverse effects and oppose mu-opioid receptor-mediated actions. Here we tested the hypothesis that KOR activation, through an action on inhibitory interneurons, disinhibits CRF neurons in the central nucleus (CeA) in naive rats. CeA serves major amygdala output functions.

Brain slice electrophysiology was used to determine the effects of a KOR agonist (U-69,593) on CRF-CeA neurons. To visualize these neurons, AAV-EF1a-DIO-mCherry was injected into the right CeA of transgenic CRF-Cre rats (4 weeks old). AAV5-ChR2-CaMKII-eYFP was injected into the lateral parabrachial area (LPB) to allow optical activation of glutamatergic synaptic input. Animals were allowed to recover for five to six weeks for viral expression. Whole-cell patch-clamp recordings of CRF-CeA neurons were used. In current clamp, neuronal excitability (frequency-current F-I relationship) and synaptically evoked spiking (E-S coupling) were measured. In voltage clamp, excitatory and inhibitory synaptic currents (EPSCs and IPSCs), paired pulse facilitation evoked by optical activation of LPB terminals in the CeA or by electrical stimulation in the basolateral amygdala, another source of synaptic input, and spontaneous and miniature excitatory and inhibitory currents were measured. U-69,593 decreased glutamate driven IPSCs (feedforward inhibition) and E-S coupling but had no effect on EPSCs and on F-I relationships. The data suggest that KOR activation under normal conditions leads to synaptic disinhibition of CRF-CeA neurons, which could result in increased pain- and anxiety-like behaviors.

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Hernandez, Sarah

alpha-Synuclein Regulation in Parkinson's Disease

Sarah Hernandez^{1,2}, Kristen Baca^{1,3}, Elena B. Tikhonova¹, Andrey L. Karamyshev^{1,2}

¹Department of Cell Biology and Biochemistry,

²Graduate School of Biomedical Sciences, Texas Tech University Health Sciences Center,

³CISER (Center for the Integration of STEM Education and Research), Texas Tech University, Lubbock, TX, USA

Parkinson's disease (PD) is a neurodegenerative disorder that is associated with the formation of Lewy bodies (LBs) in the human brain. The major component of LBs is aggregated alpha-Synuclein (aSyn). Despite many studies on aSyn, the mechanism by which it aggregates is still unknown. Our hypothesis is that alterations of interacting partners during translation leads to misfolding and aggregation of aSyn, causing disease. In PD, this alteration of interacting partners can be due to a mutation in aSyn itself (familial PD) or by defects in the interacting partners (sporadic PD). The major goal of this study is to use a candidate approach to identify possible interacting partners during translation for both wild-type and mutated aSyn. Candidates include proteins or complexes that are involved in translation at the ribosomal level, such as the signal recognition particle (SRP), ribosome-associated chaperones, and chaperonins. SRP is also involved in the Regulation of Aberrant Protein Production (RAPP) Pathway, where it functions with its counterpart, Ago2, to control the expression of misfolded proteins. Here we demonstrate that knock down of SRP54, the nascent-chain binding

subunit of SRP, affects both mRNA and protein expression of aSyn. Our results suggest that the targeting factor, SRP, and the RNA-silencing factor, Ago2, are involved in aSyn regulation, possibly at the level of translation. Determining co-translational interacting partners of aSyn is key in discerning the causes of aggregation and developing new pharmacological treatments of neurodegenerative diseases.

Jodeiri-Farshbaf, Mohammad

Neurobehavioral dysfunction induced by acute stress is ameliorated by the myokine irisin in male mice

Mohammad Jodeiri-Farshbaf¹, Daniel Cherkowsky¹, Nabeela Manal², Dominica Moussoki¹ and Karina Alviña¹

¹Department of Biological Sciences, Texas Tech University, Lubbock, TX

²Honors College, Texas Tech University, Lubbock, TX.

The myokine irisin, obtained after cleavage of its precursor protein fibronectin type III domain-containing 5 (FNDC5), has a variety of physiological roles such as regulation of body weight, temperature and glucose homeostasis. Irisin is also secreted during physical exercise and upon entering the peripheral circulation it can impact several organs including the brain. In fact, it has been shown that irisin levels are decreased in patients with disturbed mood as well as post-stroke depression. However, the overall effects of irisin on brain function, in normal and pathological conditions, are largely unknown.

Exercise can ameliorate certain aspects of stress-induced mental illness. Therefore, we hypothesized that irisin may indeed be involved in mediating the observed positive effects of exercise on brain function. We focused on the hippocampus as a vulnerable region to stress that is also associated with emotional regulation, and learning and memory, all functions heavily modulated by stress. Using both female and male adult C57Bl/6 mice, we first implemented an acute 3-hours restraint stress test that increased anxiety-like behaviors in the open field test and impaired novel object recognition, effects that were significantly more robust in male mice. Then using stereotaxic injections, we bilaterally injected 1 ng of irisin in the hippocampi of male mice and subjected them to 3 h of acute stress followed by open field and novel object recognition tests.

Irisin-injected stressed mice showed reduced anxiety-like behavior and improved working memory compared to non-injected or vehicle-injected stressed mice. In addition, we measured mice's skin temperature and irisin injection in the hippocampus markedly reversed the decrease in temperature triggered by acute stress, which is consistent with the well-known thermogenic function of irisin. Mechanisms underlying irisin effects are currently being investigated, emphasizing the role of brain-derived neurotrophic factor and mitochondrial function. Overall our findings suggest a possible role of the exercise-induced protein irisin in counteracting the anxiogenic effects of acute stress. Furthermore, our results may provide novel insights into the neurobiological mechanisms of stress-induced brain dysfunction and suggest putative therapeutic avenues.

Mazzitelli, Mariacristina

Group II metabotropic glutamate receptors, particularly mGluR2, in the amygdala regulate sensory and affective responses in a rodent model of arthritis pain

M. Mazzitelli¹, and V. Neugebauer^{1,2}

¹Pharmacology and Neuroscience, Texas Tech University Health Sciences Center, Lubbock, TX

²Center of Excellence for Translational Neuroscience and Therapeutics, Texas Tech University Health Sciences Center, Lubbock, TX

Pain is a multidimensional experience with an important aversive-affective dimension. The amygdala plays a critical role in the emotional-affective aspects of behaviors and in pain modulation. The central nucleus of

amygdala (CeA) serves major output functions, and neuroplasticity in the CeA is mechanistically linked to pain-related behaviors in different pain conditions. The activation of Gi/o-coupled group II metabotropic glutamate receptors (mGluR2 and mGluR3) can decrease neurotransmitter release and regulate synaptic plasticity. Evidence from preclinical studies suggests that mGluR2/3 may be a target for neuropsychiatric disorders and they can inhibit pain-related processing and behaviors. The contribution of mGluR2 and mGluR3 in the amygdala to pain-related behaviors remains to be determined.

Audible and ultrasonic vocalizations, and mechanical withdrawal thresholds were measured in normal and arthritic rats (5-6 h after induction of a mono-arthritis in the left knee joint with intra-articular kaolin and carrageenan). Systemic application (30 min before behavioral testing) of a group II mGluR agonist (LY379268 disodium salt) decreased the vocalizations and increased the spinal reflex thresholds of arthritis rats. To determine the contribution of mGluRs in the amygdala, a group II mGluRs antagonist (LY341495 disodium salt), a positive allosteric modulator for mGluR2 (PAM, LY487379 hydrochloride), or a negative allosteric modulator for mGluR2 (NAM, VU6001966) was applied stereotactically into the right CeA by microdialysis. Blockade of mGluR2 with LY341495 or VU6001966 in the CeA reversed the effects of a systemically applied group II mGluR agonist. Activation of mGluR2 with LY487379 in CeA mimicked the effect of the systemically applied group II mGluR agonist in arthritis rats.

These results suggest that group II mGluRs, and particularly mGluR2, in the amygdala can regulate pain-related behaviors and play a major role in the effects of systemic group II agonists.

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Raut, Snehal

Effect of presenilin (PS) mutations on the blood-brain barrier function in vitro using patient-derived induced pluripotent stem cells.

Snehal Raut; Ronak Patel; Dr. Abraham Al-Ahmad

Department of Pharmaceutical Sciences, School of Pharmacy, Texas Tech University Health Sciences Center, Amarillo, TX

Background and aims: Alzheimer's disease (AD) is the common neurodegenerative disease characterized by the progressive loss of hippocampal and cortical neurons, A key element of AD pathophysiology is characterized by the presence of senile plaques (formed by Abeta peptides aggregations) and neurofibrillary tangles (formed by hyperphosphorylated Tau protein). Although Abeta and Tau have been target for several drug candidates, none of the current approaches were capable to yield into clinically validated treatment, therefore an important shift in paradigm is needed. More recently, non-neuronal approaches have been evoked, in particular the contribution of the Blood-Brain Barrier (BBB) in the pathophysiology of the disease. In this study, we investigated the effects of mutations on PSEN genes (a gene associated with familial form of Alzheimer) against a healthy control on the blood-brain barrier phenotype using induced pluripotent stem cells (iPSCs).

Methods: In this study, we have used two PSC lines from patients with familial form of AD and mutations in PSEN genes (*PSEN1*, *PSEN2*) iPSCs were differentiated into BMEC monolayers. We assessed the BMEC phenotype by immunofluorescence, barrier function by measuring transendothelial electrical resistance (TEER) and permeability, as well as changes in glucose metabolism and vesicle trafficking.

Results: Notably, both the control and PSEN2 BMECs showed tight monolayers (TEER>1000 $\Omega \cdot \text{cm}^2$) whereas PSEN-1 BMECs showed loose monolayers (TEER 150-200 $\Omega \cdot \text{cm}^2$). Similar outcomes were observed for fluorescein permeability. We have also noted alteration in drug efflux transporter activity and glucose uptake, as well as abnormal lysosomal pH in PSEN1 BMECs compared to control and PSEN2 BMECs.

Discussion: Our study constitutes the first report of the presence of an BMEC phenotype associated with PSEN

mutations at the BBB, in particular between PSEN1 and PSEN2 mutant carriers. We are currently further investigating such differences by increasing the number of iPSC lines to ensure such effects observed are directly correlating with mutations associated with PSEN1.

Songhe, Li

Do Categorically Distinct Stressors Affect Visual Attention to Food in Humans

Li Sk (1), Keene JR (2), Harris BN (1), Carr JA (1)

(1) Department of Biological Sciences, Texas Tech University, Lubbock, Texas, USA, (2) College of Media and Communication, Texas Tech University, Lubbock, Texas, USA.

Neuroanatomical and physiological studies support the idea of two (or more) pathways relaying stressor information to CRF neurons in the paraventricular nucleus (PVN); anticipatory stressors that reach the PVN through limbic system pathways and so-called reactive stressors that ascend from the brainstem via ventral noradrenergic pathways. While both anticipatory and reactive stressors have been reported to modulate food intake, there has been little work comparing how anticipatory and reactive stressor influence behavior. We examined the influence of an anticipatory stressor (Trier-social stress test, TSST) and a reactive stressor (cold-pressor test, CPT) on visual attention to food images in human participants. Participants (n = 60) were divided equally between control, TSST, or CPT groups. We measured salivary cortisol before and after stressor exposure. Following stressor exposure participants performed an eye tracking test using a standardized picture database. We analyzed three metrics in balanced pairs of food and non-food images: saccade latency, gaze duration, and saccade bouts. Missing data were replaced using harmonic means. Salivary cortisol was elevated over baseline in both stressor groups. Linear mixed model with repeated measures revealed main effects of image type for all three eye tracking variables, with initial saccades of shorter latency to food images and longer gaze duration and more saccade bouts with food images. There were no main effects of stressor group on any eye tracking variable. There was a statistically significant interaction between image type and stressor group for gaze duration ($p = 0.03$), both stressors significantly reduced gaze duration on food images relative to controls. There was a trend ($p=0.051$) for an interaction of the two independent variables on saccade bouts, with CPT tending to reduce the number of gaze bouts on food images. We conclude that both anticipatory and reactive stressors decrease time spent looking at food, but not non-food, images. These data are partly consistent with the idea that stressors adaptively reduce attention to non-critical visual signals.

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2. Basic Science – Medical Student

D'Souza, Preston

Assessing the Activity of CRF Neurons in the Central Amygdala following application of Kappa Opioid Receptor Agonist: A novel network in pain relief

Preston D'Souza, Takaki Kiritoshi, Vadim Yakhnitsa, Volker Neugebauer

Texas Tech Health Science Center Department of Pharmacology and Neuroscience

Chronic pain is a major health care issue with limited treatment options outside of prescribing opioids. Our previous work on pain mechanisms has identified the amygdala, a limbic brain region known for its role in

emotions and memory, as a key player in emotional-affective aspects of pain and pain modulation. Specifically, the main output region of the amygdala (central nucleus, CeA) has been linked to the processing of nociceptive information and to pain behaviors through numerous interneuron networks including cells like corticotropin-releasing factor (CRF) neurons. Recent evidence suggests that activation of kappa-opioid receptors (KOR), Gi coupled metabotropic receptors, has opposing effects to mu-opioid receptor agonists, such as morphine, on CeA output. Thus, a KOR antagonist could potentially mitigate pain. This study tested the effects of a KOR agonist (U-69,593) on CeA output neurons, specifically CRF cells, using in-vitro calcium imaging.

A genetically encoded calcium indicator (GCaMP6f) was injected stereotaxically into the right CeA of transgenic CRF-Cre rats. After 4-6-weeks to allow sufficient time for expression, brain slices containing the amygdala were obtained and CRF cells were visualized with multiphoton microscopy. Trains of electrical stimuli (5Hz) were delivered to the dorsomedial fiber tract providing nociceptive input from the parabrachial nucleus (PB) to the CeA. Calcium signals were measured in CRF neurons continuously before, during and after agonist perfusion. We found that CRF neurons displayed synaptically-evoked activity following PB stimulation. Synaptically evoked activity increased during agonist perfusion.

These findings support the novel concept that KOR in the amygdala contribute to pain amplification and open an avenue for KOR antagonists as novel pharmaceuticals to mitigate pain.

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Parmar, Kanak

Variations in EEG with mobile phone usage in medical students

Kanak Parmar, visiting MS4 student

King George's Medical University

Background Electromagnetic fields (EMF) emitted by cellular telephones may cause neurological ill-effects like cognitive dysfunction, emotional instability, and even brain tumors. Slowing of brain activity on electroencephalography (EEG) has been shown. However, these findings need further validation.

Aims EEG changes and adverse effects experienced following cell-phone use were studied.

Settings and Design Study was conducted in the Department of Neurology, King George's Medical University, Lucknow on North Indian students of the University, from August 2017 to October 2017.

Materials and Methods Twenty one students underwent video-EEG recording before and after application of Samsung GT-56312 dual SIM smart phone in switched off, switched on and switched on mode with conversation.

Statistical analysis used Average EEG frequencies and amplitudes were calculated for different brain regions. Chi-square tests and t-tests were used for comparison between variables.

Results Mean age of 7 (33.3%) males and 14 (66.7%) females was 20.76 ± 1.48 years. Average EEG frequencies following mobile phones application with conversation were higher and the amplitudes lower than baseline values. Frequencies were greater on right side. Frontal slow waves were detected in 38.1%, parietal in 33.3%, occipital and temporal in 19.1% and generalized slow waves in 9.5% students. During experiment, 23.8% experienced headache, 19% experienced irritation and 9.5% felt drowsy. Headache and loss of concentration (33.3%), sleep disturbances (28.6%) and fatigue (19%) were frequent in daily life.

Conclusions Experimental application of mobile phones may lead to some EEG changes and certain ill effects on the well being. Hence, prolonged use of these gadgets warrants caution.

Presto, Peyton

Sex differences in fear extinction learning ability predict pain behaviors

P. D. PRESTO¹, G. JI^{1,2}, V. NEUGEBAUER^{1,2};

¹Department of Pharmacology and Neuroscience, ²Center of Excellence for Translational Neuroscience and Therapeutics, Texas Tech University Health Sciences Center, Lubbock, TX

Sex differences in pain and disorders such as depression and anxiety are now being recognized. Pain and fear may share neurobiological mechanisms such as plasticity in emotional networks that include the amygdala. The amygdala plays a key role in fear conditioning and has emerged as an important node of emotional- affective aspects of pain modulation. Impaired fear extinction learning, which involves prefrontal cortical control of amygdala processing, has been linked to conditions such as posttraumatic stress disorder (PTSD).

Here we tested the hypothesis that fear extinction learning ability can predict certain aspects of pain-related behaviors of rats and that these may be different in female and male rats. We correlated fear extinction learning in adult male and female rats with behavioral outcome measures (sensory thresholds, vocalizations, and anxiety-like behaviors) before and >6h after induction of an arthritis pain model (kaolin/carrageenan- induced knee joint arthritis). Auditory fear conditioning, extinction, and extinction retention tests were conducted using two chambers. On Day 1 rats were habituated to context A followed by fear conditioning (2 US-CS pairs). On Day 2, rats were habituated to context B followed by extinction training (30 CSs). On Day 3, rats were habituated to context B followed by extinction retention measurement (5 CSs). There was no difference in fear learning between male and female rats. The majority of rats (78% male, 73% female) showed a quick decline of freezing level during extinction training and retention (FE+) whereas a smaller group of rats (22% male, 27% female) maintained a high freezing level (FE-). Male and female FE- rats had lower open-arm preferences in the elevated plus maze (EPM) or shorter center duration in the open field test (OFT) than FE+ rats, reflecting anxiety-like behavior, but there were no significant differences in sensory thresholds and vocalizations between FE+ and FE- types under normal conditions. In the arthritis pain model, male and female FE- rats developed higher levels of vocalizations and anxiety-like behavior than FE+ rats, but there were no differences in mechanical reflex thresholds. Female FE- rats had stronger vocalizations than FE- males.

The data may suggest predictive value of fear extinction ability for emotional-affective pain aspects in male and female rats, and greater vulnerability of female than male rats with lower extinction ability.

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3. Basic Science – Postgraduate

Isabel Castro-Piedras, Edgar G. Martinez, Jennifer Brelsfoard and Kevin Pruitt

Department of Immunology & Molecular Microbiology. Texas Tech University Health Sciences Center. School of Medicine. Lubbock, TX.

Estrogens play an essential role in brain structure and function, in addition to behavior. It was believed that the presence of estrogens in the brain is due to passive diffusion from the peripheral circulation. Remarkably, more recent studies have shown that all the enzymes necessary for estradiol (E2) production are present in the brain and support region-specific estrogen production. Estrogens are produced from androgens by the enzyme aromatase (CYP19A1). The presence of different tissue-specific aromatase promoters complicates the understanding of its transcriptional regulation. One of these poorly studied aromatase promoters is the brain-specific promoter (1.F).

We recently reported that Dishevelled (DVL) regulates aromatase transcription. DVL proteins are key mediators

of the Wnt pathway. Wnt signals are active in numerous contexts, initially in early development and later during the growth and maintenance of various tissues. As currently understood, Wnt proteins bind to receptors on the cell surface. Through several cytoplasmic relay components like DVL, the signal is transduced to β -catenin which enters the nucleus to activate transcription of Wnt target genes.

We found that DVL itself translocates to the nucleus and acts as a regulator of transcription of the placental aromatase transcript. Interestingly, new reports not only show that aromatase is expressed in the brain, but also that DVL translocates to the nucleus in glioblastoma. Hence, we sought to investigate whether DVL participates as a new regulator of the brain-specific 1.F aromatase transcript. The regulation of the 1.F aromatase promoter may play a role in the local production of estrogen, which remains an essential question that is yet to be addressed.

Based on our preliminary data and the literature, we were able to demonstrate for the first time that DVL binds to the aromatase 1.F promoter. This binding of the DVL to the aromatase 1.f promoter establishes a robust scientific premise for our future mechanistic studies in the transcriptional regulation of aromatase and local production of estrogen in the brain.

Kumar, Subodh

Novel MicroRNA-455-3p and its Protective Effects Against Abnormal APP Processing and Amyloid Beta Toxicity in Alzheimer's Disease

Subodh Kumar¹ Arubala P Reddy², Xiangling Yin⁷ and P. Hemachandra Reddy¹⁻⁶

1.Internal Medicine Department, 2. Pharmacology & Neuroscience Department, 3. Cell Biology and Biochemistry Department, 4. Neuroscience & Pharmacology Department, 5. Speech, Language and Hearing Sciences Departments, 6. Neurology and Public Health Departments, 7. Garrison Institute on Aging, Texas Tech University Health Sciences Center, Lubbock, TX, USA

The purpose of our study is to understand the protective role of miR-455-3p against abnormal amyloid precursor protein (APP) processing, amyloid beta ($A\beta$) formation, defective mitochondrial biogenesis/dynamics and synaptic damage in AD progression. In-silico analysis of miR-455-3p has identified the APP gene as a putative target. Using mutant APP cells, miR-455-3p construct, biochemical and molecular assays, immunofluorescence and transmission electron microscopy (TEM) analyses, we studied the protective effects of miR-455-3p on – 1) APP regulation, amyloid beta ($A\beta$)₍₁₋₄₀₎ & ₍₁₋₄₂₎ levels, mitochondrial biogenesis & dynamics; 3) synaptic activities and 4) cell viability & apoptosis. Our luciferase assay confirmed the binding of miR-455-3p at the 3'UTR of APP gene. Immunoblot, sandwich ELISA and immunostaining analyses revealed that the reduced levels of the mutant APP, $A\beta$ ₍₁₋₄₀₎ & $A\beta$ ₍₁₋₄₂₎, and c-terminal fragments of APP (C99, C83) by miR-455-3p. We also found the reduced levels of mRNA and proteins of mitochondrial biogenesis (PGC1 α , NRF1, NRF2, and TFAM) and synaptic genes (synaptophysin and PSD95) in mutant APP cells; on the other hand, mutant APP cells that express miR-455-3p showed increased mRNA and protein levels of biogenesis and synaptic genes. Additionally, expression of mitochondrial fission proteins (DRP1 and FIS1) were decreased while the fusion proteins (OPA1, Mfn1 and Mfn2) were increased by miR-455-3p. Our TEM analysis showed a decrease in mitochondria number and an increase in the size of mitochondrial length in mutant APP cells transfected with miR-455-3p. Based on these observations, we cautiously conclude that miR-455-3p regulate APP processing and protective against mutant APP-induced mitochondrial and synaptic abnormalities in AD.

Key words: microRNA-455-3p, Alzheimer's disease, Amyloid Precursor Protein, Amyloid Beta, Mitochondrial

Rahman, Mizanur

NemaLife Machine: An automated system for lifespan and healthspan studies in C. elegans

Taslim Anupom^a, Mizanur Rahman^b, Siddhartha Gupta^b, Hunter Edwards^c, Purushottam Soni^b

Matthew Le-Blanc, David Koblah, Christopher Gaffney, Timothy Etheridge, Nathaniel Szewczyk, Monica Driscoll and Siva A. Vanapali^{*b}

^a Department of Electrical Engineering, Texas Tech University, Lubbock, TX 79409, USA.

^b Department of Chemical Engineering, Texas Tech University, Lubbock, TX 79409, USA.

^c Department of Biology, Texas Tech University, Lubbock, TX 79409, USA.

Caenorhabditis elegans is a powerful animal model in aging research. Standard longevity assays on agar plates involve the tedious task of picking and transferring animals to prevent younger progeny from contaminating age-synchronized adult populations. Large-scale studies employ progeny-blocking drugs or sterile mutants to avoid progeny contamination, but such manipulations change adult physiology and alter the influence of reproduction on normal aging. Moreover, for some agar growth-based technology platforms, such as automated lifespan machines, reagents such as food or drugs cannot be readily added/removed after initiation of the study.

Here, we report an automated microfluidic system called NemaLife Machine (NLM) that addresses the current limitation of plate-based aging assays. NLM integrates: 1) a microfluidic device and flow control system for culturing *C. elegans* with programmed washing of progeny and delivery of food, 2) an illumination and smart-device imaging system for recording motion of a population of animals or individuals, and 3) data analysis software for scoring live/dead animals, behavior and their mobility. The machine is compact with a foot-print of 1 ft², amenable to integration with other microfluidic devices and can be operated via an on-board interactive display. We optimized the system parameters and developed an operational workflow that robustly yields lifespan and healthspan data on *C. elegans*. We validate the NLM with longevity studies of classical aging mutants and dietary restriction. Overall, the capacity of NLM system to generate reliable lifespan and physiological data underscores the potential of this automated machine for genetic and drugs screens, and fundamental investigations on lifespan/healthspan of *C. elegans*.

Selina, Fowzia

GABAergic Deficits Are Associated with Seizure Susceptibility in A Mouse Model of SLC13A5 Deficiency

Fowzia Selina¹, Toby Anderson¹, Rui Wang¹, Xiaobo Liu¹, Madison Hayes¹, Sabarish Ramshandran³, Vadivel Ganapathy^{2,3}, and J. Josh Lawrence^{1,2}

¹Department of Pharmacology and Neuroscience, Texas Tech University Health Sciences Centre-School of Medicine, Lubbock, USA, ²Center of Excellence for Translational Neuroscience and Therapeutics, Texas Tech University Health Sciences Center, Lubbock, TX USA, and ³Cell Biology & Biochemistry, Texas Tech University Health Sciences Centre-School of Medicine, Lubbock, USA

Loss of function mutations within the human SLC13A5 gene cause Early Infantile Epilepsy and Encephalopathy type 25 (EIEE-25). However, the mechanisms underlying disease pathogenesis remain poorly understood. SLC13A5 is a Na⁺-coupled citrate transporter (NaCT) that imports citrate into the neuronal cytoplasm from the extracellular space. Citrate is a key energy source for mitochondrial respiration and a building block in the synthesis of neurotransmitters, fatty acids, and steroids. Therefore, SLC13A5 deficiency may impact the availability of key neurotransmitters such as glutamate, GABA, and acetylcholine. These deficiencies in turn could alter synaptic function and excitation/inhibition imbalance by either weakening GABAergic function or enhancing glutamatergic function. Inhibitory neurons, especially parvalbumin-positive (PV) interneurons, are more active and require greater energy demands than principal neurons. In this study, we utilized SLC13A5 KO mice to test the hypothesis that SLC13A5 deficiency impairs GABAergic signaling. We tested the hypothesis that

SLC13A5 KO mice exhibit a lower seizure threshold to the chemoconvulsants pentylenetetrazole (PTZ) and pilocarpine (PILO) when administered intraperitoneally. Seizures were scored manually using a Racine scale (index of seizure severity, from 0 to 7). In the PTZ group (n=3), KO mice exhibited shorter latency to onset and increased seizure severity. In the PILO group (n=4), KO mice also exhibited increased seizure severity at 0-10 min and 10-20 min. Because cFos acts as a biomarker for neuron excitability, the seizure focus was investigated using cFos immunofluorescence. High cFos activation was present hippocampus (CA1, CA3, dentate gyrus), cortex and paraventricular nucleus of hypothalamus (PVH) of both genotypes. But cFos activation was highest in the dentate gyrus of PTZ-induced KO mice, suggesting that the dentate gyrus is the seizure focus in SLC13A5 KO mice. Our preliminary results from both PTZ and PILO converge on the finding that KO mice have lower seizure threshold compared to WT mice. In conclusion, these results are consistent with the hypothesis that SLC13A5 deficiency weakens GABAergic signaling. Future studies will determine whether these observations are consistent in the larger population.

Clinical – Undergraduate/Graduate Student

Adjei-Mosi, Jennifer

Title: Emotional Word Processing by Colombian Children with PTSD

Jennifer Adjei-Mosi¹, Breanna Chavez¹, Ivette Noriega¹, Kareem Al-Khalil¹, Elizabeth Trejos-Castillo¹, Liliana Calderon², Mauricio Barrera³, Guillermo Correa^{3,4}, Jon Duque⁴, Ximena Cardona², Michael O'Boyle¹

Post-Traumatic Stress Disorder (PTSD) is a neuropsychological condition caused by exposure to chronic stressors and extreme trauma (DSM-V). In the last decade Colombia, South America has experienced paramilitary violence and human/drug trafficking, which have created an environment of chronic stress. And, the latter is thought to have impaired the physical, emotional, and cognitive development of these children (Noriega, 2018). Currently, limited research exists on the effects of PTSD on the neuropsychological functioning of these children. In the present study, we used brain imaging technology (fMRI) along with a behavioral task performance measure to illuminate potential deficits in executive functioning of these Colombian children.

Twenty-one PTSD and 22 controls were asked to perform an emotional word task, which required them to determine the color of the ink in which a positive, negative or neutral word was printed. Based on our previous research we anticipated that PTSD children would commit a greater number of errors and react more slowly when determining the ink color of an emotional laden word, but particularly so when it expressed a negative emotion. We also expected an accompanying increase in frontal lobe activation, reflecting hyper-engagement of executive functioning. Our results showed no group differences in accuracy for determining ink color when presented as a positive or neutral word. However, PTSD children were reliably less accurate and notably slower at determining ink color when presented in the context of a negative word. Importantly, the latter performance was accompanied by an increase in frontal lobe activity, which may reflect compensatory executive functioning, induced perhaps as a by-product of their traumatic experiences.

4. Clinical – Medical Student

Aldrete, Jonathan

Subgaleal Osteolytic Pigmented Epithelioid Melanocytoma with Dural Infiltration

Jonathan Aldrete, Preston D'Souza, Pranati Pillutla, Avery Kopacz, Laszlo Nagy

Pigmented Epithelioid Melanocytoma (PEM) is a recently described rare, dermatological tumor that shares common histological features with the epithelioid blue nevus and animal-type melanoma. Preliminary findings have shown the PEM to be prevalent within adolescents and young adults, mean age 28 years, with no predilection towards ethnicity or sex, and various locations throughout distal extremities. PEMs are also not correlated with sun exposure and display an indolent clinical presentation. Additionally, the PEM has been associated with a familial syndrome, Carney Complex, that encompasses many tumors including melanomas and schwannomas. Yet, the true nature of this tumor is not fully understood, and current clinical regimens involves careful observation due to unknown prognosis. A few case studies have given slight indication that PEM may be benign, because it rarely spreads past regional lymph nodes. Currently, this entity is classified as a low-grade tumor with metastatic potential.

This case encompasses a unique presentation of PEM that is not described in the literature. A 14-month old female presented to the clinic by her mother who noticed two holes in the back of the child's head. Neuroimaging revealed a subgaleal lesion and an open biopsy was conducted. Following biopsy, a diagnosis of PEM was established, and further investigation revealed the PEM to be a lytic lesion of the posterior parietal calvarium. Intraoperative findings uncovered further infiltration into the dura. Partial resection of the tumor ensued, with most of the tumor removed. Intraoperative decision to leave a portion of the PEM that adhered to the dura was made to spare the dura from incision. Due to the ambiguity of PEM prognosis, meticulous observation of the patient is being conducted. This case is unique as the PEM has shown a subgaleal location and presented as a lytic lesion of the skull and dura which has not been described by the literature to date.

Hanson, Keith

Child Abuse and Deformational Plagiocephaly in a West Texas Hospital System

Keith A Hanson¹, Preston D'Souza¹, Peyton Presto¹, Pranati Pillutla¹, Brandon McCarty¹, and Laszlo Nagy² MD

¹Texas Tech University Health Sciences Center School of Medicine

²Texas Tech University Health Sciences Center, Department of Pediatrics

Intro

The aim of this study was to assess deformational plagiocephaly's (DP) predictive value in neglect and physical abuse (NAT) within the pediatric population. In addition, we sought to characterize the prevalence of DP and NAT for our hospital's mostly rural catchment area.

Methods

Data on hospitalized patients diagnosed with NAT and/or neglect between 2012-2018 was collected via retrospective chart review. All enrolled children were under the age of 4 years old at the time of diagnosis, and those without legible head CTs or MRIs during their initial hospitalization were excluded. Utilizing neuroimaging, we calculated the Cranial Vault Asymmetry Index (CVAI) and Cranial Index (CI) for each patient to assess for DP. Differences between the two groups were assessed using Wilcoxon rank sum test for continuous variables and Fisher's exact test for categorical variables. A p-value of 0.05 or less was considered statistically significant. All analyses were conducted using SAS 9.4 (Cary, NC, USA).

Results

The prevalence of DP within the combined cohort of NAT and Neglect patients is 21%, similar to that reported in the literature for the general population (20-50%). There was no significance between the prevalence of DP and a history of NAT or Neglect. Furthermore, there was no correlation between CVAI index and characteristics of initial presentation or history of trauma for either NAT (p-value: 0.359 and 0.250 respectively) or Neglect groups (p-value: 0.116 and 0.770 respectively).

Conclusion

While there are many limitations to this study, our results suggest that abused children are no more likely to have history of DP than the general population, and the degree of DP is not associated with severity of trauma history or initial presentation. We hope the results of this study promote future investigations for unique subtle predictive factors of child abuse/neglect.

Chavez, Alondra

Development of a premedical student volunteering/mentorship program designed to address the economic problem of the caregiver-patient ratio in skilled-care geriatric memory units

Willms J^a, Brown S^b, Chavez A^b, Zon A^b, Moseley K^b, Perez A^b, Wolpert J^b, Young K^b, Culberson J^a

^aGarrison Institute on Aging, Texas Tech University Health Sciences Center, 3601 4th Street, MS 9424, Lubbock, TX 79430, United States

^bTexas Tech University, 2500 Broadway, Lubbock, TX 79409

Unpaid caregivers provided 18.4 billion hours of care to patients with age-related dementias in 2017.¹ Despite this, the lifetime cost to a patient diagnosed with dementia for skilled care, medical attention, and housing is crippling, costing approximately \$341,840.¹ Our objective was to develop an innovative way to provide supportive care for dementia patients without increasing the financial burden. We created a platform at a nursing home for premedical students to perform dementia caregiving tasks including emotional support, providing respite for paid caregivers, managing behavioral symptoms (aggressive behavior, feeling lost), feeding, and entertainment. Students also implemented a wheelchair exercise program and a music therapy project. We recorded the number of volunteers who attended each week through photography and an online database, documented custom project outcomes, and recorded time spent on leadership outside of volunteer events. Premedical students received “pay” for their work through improvements to their applications to medical school (volunteer hours in a healthcare setting). We calculated the estimated value of the care provided to patients and documented the benefits for the premedical students. Over the past three years, our group contributed ~1200 hours of unpaid care to dementia patients. We estimated the value of this care to be about \$5046 per year. If similar groups were established at every US university, the value of this care would be \$20,889,000 per year. The sustainability of our work was made possible by the mutually beneficial relationship between pre-health students and dementia patients. Memory patients need supportive care, and pre-health students benefit from providing supportive care. Therefore, pre-health students are a currently untapped resource that if appropriately mobilized, could contribute 1,656,000 hours per year paid for by professional development, which does not increase the financial burden on either patients or the US economy.

1. Alzheimer's Association. "2018 Alzheimer's disease facts and figures." *Alzheimer's & Dementia* 14.3 (2018): 367-429.

Kopacz, Avery

Foramen size as a potential risk factor for febrile seizure development in the pediatric population

Peyton Presto, Keith Hanson, Mark Stephens, Nikki Tangella, Benjamin Elbersen, Preston D'Souza, Avery Kopacz

Texas Tech University Health Sciences Center School of Medicine, Department of Pediatrics

Febrile seizures have been shown to occur in 2-5% of children between the ages of 6 months to 5 years, making them the most common seizures of childhood. These seizures occur in young children who experience a fever but exhibit no evidence of intracranial infection or acute neurological illness. It has been shown that the likelihood of experiencing a febrile seizure increases with the child's temperature as opposed to the rate

of temperature rise. Febrile seizures can be classified as either simple or complex depending on length of the seizure and duration between seizure reoccurrence. Risk factors identified through review of relevant studies include male sex, developmental delay, family history, day care attendance, viral infections, certain vaccinations, and zinc and iron deficiencies. However, no investigation has been conducted to explore foramen size and associated venous drainage as a potential risk factor for experiencing febrile seizures. Of particular interest are the parietal foramen, which conducts the parietal emissary vein (PEV), and the condylar canal, which conducts the occipital emissary vein (OEV). Emissary veins lack valves, which allows them to play a crucial role in selective brain cooling via a bidirectional flow of cooler blood from the head's evaporating surface. If the cranial apertures conducting these veins are narrowed, the cerebral venous outflow is potentially reduced and therefore unable to cool the brain as rapidly as expected, leading to a febrile seizure. To explore this possibility, we conducted a retrospective chart review of all febrile seizure patient cases at the University Medical Center (UMC) and Covenant Medical Center (CMC) in Lubbock, Texas, over the past seven years. The area of the parietal foramen and condylar canal in febrile seizure patients is contrasted to those of similar-aged trauma patients. Our findings will help guide further work in the detection and prevention of febrile seizures.

Lee, Suheng

Hormonal Contraception in Vulvodynia Selected Pathway Analyses

Suheng Lee, Kushal Gandhi, Lilana Hsu, Natalia Schlabritz-Lutsevich, Gary Ventolini

Texas Tech Health and Science Center Department of Obstetrics and Gynecology, Odessa, TX, USA

Background. Vulvodynia (VD) is a high-prevalence, chronic, multifactorial condition that effects all women. Despite research efforts in pathogenicity, its etiology remains obscure. This study aims to evaluate expressions of biomolecules involved in angiogenesis, tissue growth, repair and inflammation in women with VD and determine effects of hormonal contraception (HC) on VD-related molecular pathways.

Methods and Findings. Patients (n=10) were selected according to approved IRB protocol (#L13-054, #PB19-020). All were diagnosed with provoked VD according to ISSVD guidelines. Samples were collected from the middle of vagina, using cotton swabs and analyzed, using Quantibody[®] Human Cytokine Antibody Array 4000, which quantifies 200 human biomolecules. Ingenuity Pathway analysis (IPA) was performed for identification of molecular pathways. Of 200 cytokines, 168 were expressed from our patients. Comparisons revealed 142 cytokines expressed in both groups (HC and non-HC), 24 cytokines expressed only in the contraceptive group, and 2 cytokines expressed exclusively in the patients without HC. The main area affected was the pathway of granulocyte activation adhesion and diapedesis. This cytokine pathway was linked to the main hormone-dependent molecules, such as Estrogen Receptor 1, Estrogen Receptor 2, Prostaglandin Receptor, and Androgen Receptors. Notably, the expression of MIP-3a (CCL20) levels was increased in patients with VD on contraceptives. In addition, the HC group demonstrated more enhancement of expression levels of GCP-2 (CXCL6), PF4 (CXCL4), OPN (Osteopontin), CTACK (CCL27), MIP-3a (CCL20), and IL-13R2, in comparison with the non-HC group.

Conclusion. Our data demonstrate an association of hormonal contraceptives with steroid-hormone-related pathways of painful vaginal conditions and reveals cytokines specific to those conditions.

Merugumala, Praveen Dev

neonatal Neurological Outcome in Pregnant Women with Type 1 Diabetes Mellitus on Continuous Insulin Pump: A Case Series

Praveen Dev Merugumala, Glen Bennion MD, Christophe A Enakpene, MD, Natalia Schlabritz-Lutsevich MD PhD, Elisa Brown, MD, Varuna Nargunan, MD;

Texas Tech Health and Science Center Department of Obstetrics and Gynecology, Odessa, TX, USA

According to the CDC in 2017, 23.1 million Americans have diabetes. Diabetes is the most common medical complication in pregnancy. Pregnancy complications that can arise from diabetes in general are spontaneous abortion, preterm delivery, malformations, altered fetal growth, hydramnios, and unexplained fetal demise. Continuous insulin delivery through insulin pump is used for effective management of type 1 diabetes. Preventing hypoglycemia and having tight control of blood glucose during the gestational period has significant impact in reducing neonatal neurological complication. Our case series is focused on well controlled type 1 diabetics on insulin pump who had successful pregnancy outcomes without any neonatal neurological deficits.

5. Faculty Presented Posters (not judged)

Benamar, Khalid (Poster #24)

New animal model for HIV-related neuropathic pain

Khalid Benamar

Department of Pharmacology and Neuroscience, Texas Tech University Health Sciences Center, Lubbock, TX, USA

Human Immunodeficiency Virus-1 (HIV)-related neuropathic pain is a common and debilitating condition and therefore a major health care problem affecting 55-67% of the 36.7 million infected individuals worldwide (<http://www.who.int/hiv/en/>). Despite the combination antiretroviral therapy (cART), neurologic complications and neuropathic pain continue to affect many individuals with HIV infection (Ellis et al., 2010). Peripheral neuropathy is currently the most common neurologic complication associated with HIV infection (Ellis et al. 2010). The most frequently reported clinical manifestation of HIV-associated peripheral neuropathy is distal sensory polyneuropathy (DSP). A progressive and often debilitating syndrome characterized by bilaterally symmetrical pain, numbness, and hypersensitivity that is most pronounced on the feet and lower legs (Pardo et al. 2001). The development of therapeutics strategies and better understanding of HIV-related neuropathic pain mechanisms has been hampered by the lack of a suitable animal model that mimics chronic HIV-related neuropathic pain condition.

Here we used adults male HIV transgenic rats (HIV-tg, University of Maryland) to establish an animal model that mimics this condition. Key advances of this model are: 1) Does not require invasive induction. 2) Continuously and constitutively expresses various HIV-associated proteins (in the absence of viral infection) mimicking the HIV chronic condition in the cART era. 3) The effects of 7 HIV viral proteins (gp120, tat, rev, vif, vpr, vpu, and nef), can be evaluated simultaneously. The animals were monitored weekly for 4 weeks for neuropathic pain-like behaviors using electronic von frey (mechanical allodynia) and Hargreaves (thermal hypersensitivity).

Compared to age-matched (wild-type), HIV-tg rats developed mechanical allodynia (electronic von Frey test) but not thermal sensitivity (Hargreaves test) consistent with clinical data from patients with HIV-related painful neuropathy (Martin et al., 2003). No impairment of physical function (e.g. motor abilities) that could interfere with paw withdrawal responses in the behavioral assays was observed in HIV-tg at this age, but we did observe differences in body weight.

These data indicate that continuous long-term exposure to HIV viral proteins in HIV-tg rats produces neuropathic pain-like behaviors, and suggest that HIV-tg can be used as a model of HIV chronic neuropathic pain-like behavior. These studies will significantly impact the field of HIV-associated neuropathic pain research and management by establishing an improved clinically relevant rodent model for chronic HIV-related neuropathic

pain condition that will serve as the basis for future mechanistic and interventional studies in this unique pain condition.

The proposed studies will test the novel hypothesis ***that continuous long-term exposure of HIV viral proteins results in a neuropathic pain-like condition with behavioral and pathological changes that mimic chronic HIV neuropathic pain condition.*** Male and female rats will be used in this proposal to determine any sex differences in neuropathic pain-like behaviors and DSP-associated pathology. The animals will be tested at 3 and 9 months old to determine if pain-like behavior and DSP-associated pathology are age-dependent. Because there is evidence for age-dependent differential expression of various HIV proteins (Peng et al., 2010) with consequent tissue injury over time.

Specific Aim 1. To analyze neuropathic pain-like behaviors in HIV-tg rats. Behavioral and pharmacological experiments will test the hypothesis that continuous long-term exposure to HIV viral proteins in HIV-tg rat results in neuropathic pain-like behaviors. Neuropathic pain-like behaviors will be determined in a battery of behavioral assays (sensory and affective). This include cold hypersensitivity (acetone test), heat hypersensitivity (Hargreaves test) and mechanical hypersensitivity (electronic von Frey test). Because pain related affective comorbidities (e.g. anxiety and depression) are known to be a feature of neuropathic pain in humans, HIV-tg neuropathic pain will be tested in elevated plus maze (common test for the study of anxiety) and forced swim test common test for the of depressive-like behavior). Conditioned place preference (CPP) will determine if HIV-tg experience spontaneous pain. This form of pain is the most common in patients with chronic neuropathic pain (Backonja and Stacey 2004; Campbell and Meyer 2006) including HIV (Ellis et al., 2010; Fuller et al., 1993).

Specific Aim 2. To analyze the peripheral nervous system (PNS) pathological in HIV-tg pain model. The main goal here is to determine if HIV-tg rats develop PNS alterations that mimic those found in HIV patients with DSP (Pardo et al., 2001). Immunohistochemistry studies will elucidate changes in intraepidermal nerve fiber (IENF) and macrophage infiltration in HIV-tg model. Skin biopsies from the plantar area of the hind paw will assess IENF density (a key clinical diagnostic tool for HIV-associate sensory neuropathy). Macrophages infiltration (a universal marker of peripheral nervous system) will be assessed in the dorsal root ganglia (DRG). We will also determine the role of neuroinflammation (e.g. gliosis and cytokine levels) that plays an important role in the induction and maintenance of chronic pain (Ellis and Bennett, 2013; Ji et al., 2014).

We predict that continuous long-term exposure to HIV viral proteins in HIV-tg rats will produce neuropathic pain-like behaviors and peripheral neuropathy.

MacDonald, Clint (Poster #25)

A Point Mutation in the RNA-Binding Domain of CSTF2 Results in Severe Intellectual Deficiency

Petar N. Grozdanov¹, Elahe Masoumzadeh², Kerri A. White¹, Michael P. Latham², Jamel Chelly³, Vera M. Kalscheuer⁴, and Clinton C. MacDonald¹

¹Department of Cell Biology & Biochemistry, Texas Tech University Health Sciences Center, Lubbock, Texas, USA; ²Department of Chemistry & Biochemistry, Texas Tech University, Lubbock, Texas 79409-1061 USA;

³Institut de Génétique et de Biologie Moléculaire et Cellulaire, Illkirch, France; ⁴Max Planck Institute for Molecular Genetics, Research Group Development and Disease, Ihnestr. 73, D-14195 Berlin, Germany

The *CSTF2* gene encodes CstF-64, an RNA-binding protein that is essential for mRNA cleavage and polyadenylation in all tissues including the brain. Previously, no deleterious mutations have been reported for *CSTF2*. We report here that a point mutation in the RNA recognition motif (RRM) of *CSTF2* that changes an aspartic acid at position 50 to an alanine (D50A) results in severe intellectual deficiencies in male patients from a family in Morocco. We hypothesized that the single amino acid change in *CSTF2* was sufficient to alter its functions during cleavage and polyadenylation, and that these functions were critical for brain functions in learning and memory. To test whether the D50A mutation in *CSTF2* affected mRNA polyadenylation, we used

a transfection-based reporter gene assay to measure polyadenylation efficiency. *CSTF2*^{D50A} consistently achieved 15% lower efficiency of C/P than wild type *CSTF2* in this assay, suggesting altered rates of polyadenylation in the patients. Interestingly, the D50A mutation resulted in a lower K_d indicating a greater affinity for RNA. These results suggest that D50A alters the features of *CSTF2* important for RNA binding. We expressed the *CSTF2* and *CSTF2*^{D50A} RRM in bacteria and examined their RNA-bound structures using nuclear magnetic resonance (NMR) spectroscopy. Overall, the polypeptide backbone of the wild type and *CSTF2*^{D50A} mutants were nearly identical. However, the mutation changed the side chain position within the structure to alter the electrostatic potential of the RRM, increasing the k_{on} rate for RNA. These changes were accompanied by changes in the stability of the D50A loop, allowing repositioning of the side chains of two RNA binding sites in the RRM, as well as repositioning of the fourth alpha helix. Male mice engineered to have the D50A mutation (*Cstf2*^{D50A}) tend to be smaller than wild types, and have other potential phenotypes. RNA-seq of RNA from brains of wild type and *Cstf2*^{D50A} mice showed altered expression of at least key mRNAs and altered polyadenylation of several genes. These results highlight the importance of mRNA processing in correct expression of genes important for brain plasticity and neuronal development.

Ponomarev, Igor (Poster #26)

Profiling Neuronal Epigenomes from Limited Tissue Samples

CR Bridges², O Ponomareva², CT Tulsia², T Larina², RA Harris², Igor Ponomarev¹

1. Department of Pharmacology and Neuroscience, Texas Tech University Health Sciences Center, Lubbock, TX USA, 2. Waggoner Center for Alcohol and Addiction Research and The College of Pharmacy, University of Texas at Austin, Austin, TX USA

Alcohol use disorder (AUD) is characterized by widespread changes in gene expression in humans and animal models. Mounting evidence points to a central role of chromatin (epigenomic) modifications in controlling gene expression and behavior in AUD, but there is a critical gap in our knowledge regarding AUD-associated epigenomic changes in specific neuronal populations in the addiction neurocircuits. Here, we tested two recently developed methods to obtain epigenomic profiles from limited number of cells. INTACT (Isolation of Nuclei TAGged in specific Cell Types) is based on the Cre-loxP system in mice to express a tagged nuclear membrane protein, allowing affinity purification of tagged nuclei from genetically defined cell populations. ATAC-Seq (Assay for Transposase-Accessible Chromatin using Sequencing) provides a comprehensive description of the epigenomic state in the cell, including chromatin accessibility (open chromatin), nucleosome positioning and occupancy of DNA binding proteins. We used INTACT to isolate nuclei from glutamatergic cortical neurons of mice expressing green fluorescent protein (GFP) in Camk2a-positive cells. We achieved >95% specificity and up to 70% yield of GFP+ neurons. We tested ATAC-Seq using various numbers of nuclei and were able to obtain reliable epigenomic profiles from as few as 5,000 nuclei. A pilot study examining effects of alcohol on epigenomes of GFP+ cortical neurons is underway. Identifying alcohol-induced, cell type-specific molecular changes is critical to our understanding of the roles of individual neuronal populations in alcohol actions, and a combination of INTACT and ATAC-Seq will be instrumental in studying neuronal epigenomes in a brain region – and neural circuit – specific manner.

Funding Support: NIH, NIAAA grants AA024586 (IP); Bruce/Jones Graduate Fellowship in Addiction Biology (CTT).

Jones, Betsy (Poster #27)*Student-Generated Videos: A Self-Directed Learning Project for Medical Students*

Betsy Goebel Jones EdD, Kelsey C. Walker MD, Simon Williams PhD

Despite expectations from accrediting bodies, faculties often struggle with how best to develop self-directed and active-learning activities, especially those that students enjoy and that faculty feel prepared to facilitate. Students also enter medical school with energy and creativity and often with extensive backgrounds in the arts. Failing to engage their imaginative expression thwarts both students' opportunity to manage stress and to synthesize complex information.

Design

This project is implemented during the MS1 medical students' first week of class in the doctoring course. Using scenarios that draw on themes related to communication, ethical challenges, collaborative team work and similar topics, student groups create videos by dramatizing the scenario, pointing out relevant challenges, and giving a brief series of teaching points germane to the scenario. Videos are posted to YouTube and shared as teaching tools.

Outcomes

Student evaluation of the project has been universally positive over 5 years of implementation. Students have embraced the creative challenge, taken seriously the goal of teaching their peers, and worked hard to produce polished products

Innovation's Strengths & Limitations

This activity engages students' creativity to create teaching products associated with key curricular themes such as doctor-patient communication, collaborative and interprofessional relationships, and ethical challenges. Opportunities for growth include longitudinal assessment of this experience on learners' behavior and the results of their collaborative interactions and performance in courses and clerkships.

Generalizability

This project could easily be adapted for other settings or learners such as to a systems-based basic sciences course, where the scenarios are related to clinical applications to foundational sciences content. It could be used in clinical clerkships, with scenarios focusing on higher-level ethical and communications challenges, for example the potential of domestic violence, the need for interpretation services, the importance of cultural competence, or the impact of social determinants of health on patient adherence and follow-up.



Combating the Opioid Crisis: Integrative Approaches for Pain Management

Texas Tech University Health Sciences Center,
Lubbock, Texas April 12, 2019

Program Book

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Symposium Goals

Welcome to the second TTUHSC Integrative Medicine Symposium. We are pleased to welcome you to this exciting inter-professional event in the field the integrative medicine and health.

Integrative Medicine (IM) is defined by the leading IM training center at University of Arizona as “healing-oriented medicine that takes account of the whole person, including all aspects of lifestyle. It emphasizes the therapeutic relationship between practitioner and patient, is informed by evidence, and makes use of all appropriate therapies”.

Interprofessional Education (IPE) is defined by the World Health Organization as when two or more professionals learn about, from, and with each other to enable effective collaboration and improve health outcomes.

The goals of the symposium are to:

- raise awareness of IM practice to combat the opioid crisis,
- demonstrate how IM may benefit pain management/practice,
- promote inter-professional collaborations among conventional health care professionals and trainees.

Students participating in the interprofessional workshop sessions in the afternoon will receive a **certificate for participation in a Registered IPE Activity**.

Sponsors

The 2019 Integrative Medicine Symposium is sponsored by:

- Division of Integrative Medicine,
- Center of Excellence for Integrative Health,
- Office of Interprofessional Education,
- Laura W. Bush Institute for Women’s Health
- Center for Excellence for Translational Neuroscience and Therapeutics
- Lubbock Arts Alliance (Art Contest Award)

Acknowledgement

We thank all speakers, instructors and volunteers who work hard for the symposium.

Organizing committee

Yan Zhang, Ph.D. L.Ac

Organizing committee Chair

Associate Professor, Division of Integrative Medicine, Department of Family Medicine
School of Medicine

Renée J. Bogschutz, Ph.D., CCC-SLP

Organizing committee Vice-Chair

Director, Office of Interprofessional
Education

Betsy Goebel Jones, EdD

Art Exhibit/Contest Chair

Chair and University Distinguished Professor, Department of Medical Education

Regional Director, Laura W. Bush Institute for Women's Health (LWBIWH)

School of Medicine

Leslie Shen

Associate Dean for Research

Professor of Pathology

Founding Director, Center of Excellence for Integrative Health

School of Medicine

Volker Neugebauer

Professor and Chair, Department of Pharmacology and Neuroscience

Giles McCrary Endowed Chair in Addiction Medicine

Director, Center of Excellence for Translational Neuroscience and Therapeutics

School of Medicine

Kathy Sridaromont

Associate Dean and Department Chair for the Traditional BSN Program

School of Nursing

Gary Kearns

Assistant Professor

School of Health Professions

Christie Beauregard, MSHS

Organizing committee member-event coordinator

Assistant Director, Office of Interprofessional Education

Tiffany Denton

CTNT Coordinator

Emily Iceland

Department of Family Medicine

Integrative Medicine Symposium Agenda

Time	Activities	Location (TTUHSC ACB, Lubbock)
08:00-09:00	Morning Registration	Lobby
08:30-12:15	Morning Sessions	ACB 100
08:30-08:45	Opening Remarks	
08:45-09:45	Keynote Presentation 1: Dr. Margaret Chesney- Integrative Health and Medicine-A New Essential Partner in Effective Pain Management	
09:45-09:50	Guided Meditation: led by Frances Kellerman Hanson MSII Class of 2022	
09:50-10:50	Keynote Presentation 2: Dr. Bruce Watkins- Natural Products and Chronic Pain	
10:50-11:05	Morning Break	
11:05-12:10	Panel Discussion: Pain Sciences and Practice Drs. Volker Neugebauer (Moderator), Miles Day, Jean-Michel Brismée, Nakia Duncan, and Josee Guindon	
12:10-12:15	Art exhibit awards: Dr. Betsy Jones	
12:15-01:30	Afternoon registration	Lobby
	Lunch	Lobby
	Art Exhibit and Poster Display	Lobby
	Herbal Tea and Essential Oil Stations	Lobby
12:30-01:15	Experiential Yoga Session (Limited yoga mats are provided, first come first serve): Rachelle Atkinson, Certified Yoga Therapist	ACB240
01:30-03:00	Afternoon Interactive Interprofessional	ACB 260 Suite
	Project Echo delivers Inter-professional Education to Remote Sites -Dr. Klein, Dr. Culberson and Dr. Lux	ACB 260A
	Dry Needling by Physical Therapists in the Medical Model by Dr. Gary Kearns	ACB 260B
	The Pain Neuroscience Paradigm by Dr. Brad Allen	ACB 260H
	Integrative Inter-professional Pain Management: A Holistic Approach by Dr. Kathy Sridaromont	ACB 260C
	Integrative Pain Management-Where Can We Go From Here? by Dr. Phillip S. Sizer	ACB 260 K
	Opioid Use Disorder: Medication Strategies for Prevention, Rescue and Treatment by Dr. Nakia Duncan	ACB 260J
	Mindfulness and Pain: Using Acceptance and Commitment Therapy as a tool to cope with physiological and psychological pain by Dr. David Trotter	ACB 250
03:30	Adjourn	

Morning Session Information

Keynote Presentation 1 (08:45-9:45, ACB100)

Title: Integrative Health and Medicine: A New Essential Partner in Effective Pain Management

Integrative Health and Medicine is a new field that is gaining increasing attention, in the United States and around the world. This presentation will begin by briefly describing the factors that have given rise to this new field, which emphasizes healthy lifestyles and self-care, provides information for personalized health-enhancing, disease-preventing strategies, focuses on the whole person, and provides a team approach that makes use of all evidence-informed treatments, to achieve optimal health and healing. The growth and acceptance of this new approach to health and wellness is evident, given the level of current interest in medical schools, the public, and most recently the Congress of the United States. The current opioid epidemic which has ravaged communities, leading to thousands of deaths and costing the nation well over 78 billion dollars a year is bringing attention to integrative health and medicine, with its numerous, evidence-based, nonpharmacological approaches to pain. This epidemic reflects the prevalence of pain as a public health priority throughout the country. Fortunately, integrative health and medicine is stepping forward as a new essential partner to provide effective alternatives for pain management in the United States.



Speaker: Margaret A. Chesney, PhD

Margaret Chesney is Professor of Medicine in the Department of Medicine at the University of California, San Francisco. From 2010 to 2015, she served as director of the UCSF Osher Center for Integrative Medicine. Taking leave from UCSF from 2003-2008, she served as the first Deputy Director of the National Center for Complementary and Integrative Health (NCCIH) at the National Institutes of Health (NIH).

Professionally, Margaret is engaged in research on interventions to reduce risk of chronic disease, promote effective pain management, and enhance health and wellness. She developed Coping Effectiveness Training, which focuses on nonpharmacological approaches to coping with chronic illness, particularly those characterized by pain. She is interested in translating research into health policies that will empower individuals, families and communities to adopt healthier ways of living, build resilience, prevent disease, and enhance wellbeing.

Margaret has won numerous awards throughout her career. In 2007, she was named one of the outstanding women leaders by the American Psychological Association. In 2011, she was awarded the Distinguished Scientist Award by the Society of Behavioral Medicine. She recently served as Chair of the Academic Consortium for Integrative Medicine and Health. She has been President of the Academy of Behavioral Medicine Research, the American Psychosomatic Society, and the Society for Health Psychology. She is author of over 350 scientific papers, and in 2001 was elected to the National Academy of Medicine.

Title: Natural Products and Chronic Pain

Chronic pain and its debilitating effect on the body has consequences that alter the emotional state and may lead to depression. Hence, research focusing on natural products to control pain has gained support from several new investigators and interest by funding agencies. Ethnobotany, the scientific study of the historical relationships between people and plants, spans thousands of years. Of the 28,000 plants for human use, about 100 have been studied to evaluate their potential in medicine. Natural products include sources from animals to plants. The actions of specific omega-3 polyunsaturated fatty acids found in cold water fish are known to alter pathways of COX and LOX production, lessening elaboration of pro-inflammatory compounds. Examples of successful natural products from plant sources used in models of pain research include flavonoids, terpenes, alkaloids, phenols, and carotenoids. However, investigations on natural products must consider the route of administration and delivery of the active compounds to the site where pain can be diminished. Pain perception is mediated by specific processes of nociceptive signals transported to the CNS along nerve fibers from the site of injury signals and modulated at synaptic sites in the CNS.

Neuroinflammation plays an important role in the induction and maintenance of chronic pain. Decreasing neuroinflammation through regulation of anti-inflammatory and inflammatory mediators is a hopeful pursuit for treating neuropathic pain. In this regard, targeting neuroinflammation is a promising analgesic approach for neuropathic pain and, in some cases, natural products may afford potential benefits to control pain. Thus, in the genesis of pain is the inflammatory process and the elaboration of inflammatory cytokines. The progression of pain involves multiple steps to test the feasibility of natural products to control and attenuate pain. The purpose of this presentation is to provide a concise overview of natural products that possess features to reduce pain and describe the actions of some specific pain-alleviating compounds.



Speaker: Bruce Watkins, PhD

Bruce A. Watkins is a Purdue University Emeritus Professor of Nutrition and adjunct Professor of Anatomy and Cell Biology in the Department of Anatomy and Cell Biology at Indiana University School of Medicine. He is the Editor-in-Chief of Nutrition Research and currently in the Department of Nutrition at the University of California, Davis. Bruce is affiliated with the Center on Aging at the University of Connecticut Health Center. His research emphasis is on endocannabinoids and their actions on cannabinoid receptors. The goal of his research is to understand the functions of the endocannabinoid system in muscle biology, systemic energy metabolism, and aging. Another area of his work is the study of dietary n-3 PUFA in kidney disease. Bruce received his PhD at the University of California, Davis. He is a member of many societies and has received several national awards, including the Babcock-Hart Award, IFT Research and Development Award, Bio-Serv Award, and PSA National Research Award. He was named one of the first University Faculty Scholars at Purdue University and member of the Academy of Teaching Scholars, Indiana University School of Medicine.

Bruce is a fellow of the ESCOP/ACOP Leadership Development Program, and the Committee on Institutional Cooperation Academic Leadership Program.

Panel Discussion (11:05-12:10, ACB100) Title: Pain Sciences and Practice

Drs. Volker Neugebauer (Moderator), Miles Day, Jean-Michel Brismée, Nakia Duncan, and Josee Guindon

Description: Engage the audience in a dynamic conversation between research, practitioner, and health provider about pain and pain management.

Panelists:



Volker Neugebauer

Volker Neugebauer, MD, PhD, is Professor and Chair of the Department of Pharmacology and Director of the Center of Excellence for Translational Neuroscience and Therapeutics (CTNT) at the Texas Tech University Health Sciences Center (TTUHSC). He was recently appointed as the Executive Director and Chief Scientific Officer of the Garrison Institute on Aging (GIA). Dr. Neugebauer received broad training in physiology, pharmacology, neuroscience and neurology at the University of Würzburg, Germany and the University of Texas Medical Branch in Galveston, TX; he joined TTUHSC in 2014. Dr. Neugebauer directs a research program on higher brain functions and dysfunctions in chronic pain and related neuropsychiatric conditions that has been continuously funded by NIH for the past two decades. The analysis of emotional-affective and cognitive brain mechanisms of pain (centered on the amygdala) is a key contribution of his work to the field of pain research and neuroscience. Collaborative research projects explore brain mechanisms of neurodegenerative disorders including Alzheimer's disease, alcohol use and addiction disorders, comorbidities with depression and anxiety disorders, epileptogenesis, and aging-related health issues in general through innovative research, education, and community outreach. The goal is the better understanding of disease mechanisms and development of novel therapeutic strategies to improve quality of life and healthy aging.

Jean-Michel Brismée, PT, ScD is Professor in the Doctor of Philosophy and Doctor of Science in Physical Therapy Programs at Texas Tech University Health Sciences Center (TTUHSC) in Lubbock, Texas. Dr. Brismée teaches at TTUHSC in the areas of Kinesiology and Orthopaedics and is involved in clinical research in Orthopaedics, Manual Therapy and Movement Sciences. He has instructed over 100 courses and conferences nationally and internationally in advanced Orthopaedic Medicine and Manual Therapy of the spine and extremities. He is the Editor-in-Chief of the Journal of Manual and Manipulative Therapy and maintains clinical practice in outpatient orthopedics at University Medical Center in Lubbock, Texas.





Dr. **Miles Day** is the medical director of The Pain Center at Grace Clinic, pain fellowship program director at Texas Tech, and the Traweek-Racz Endowed Professor in Pain Research in the Department of Anesthesiology at Texas Tech University Health Sciences Center in Lubbock. He is a diplomat of the American Board of Anesthesiology with subspecialty certification in Pain Medicine. He serves on the editorial boards of Pain Practice and Pain Physician. He is the past-president of the Texas Pain Society and past-chair of the Board of Examination for the World Institute of Pain. He has authored or co-authored numerous book chapters and peer-reviewed articles, and has presented nationally and internationally on various subjects in interventional pain medicine.

Nakia Duncan is an Assistant Professor of Pharmacy Practice at the Texas Tech University HSC SOP-Dallas. She is the Clinical Pharmacy Coordinator for the Palliative Care team at UT Southwestern; her focus is pain/symptom management. She earned her PharmD from Hampton University in 2010 and completed a PGY1, Geriatric pharmacotherapy PGY2, and Pain/Palliative Care Fellowship in 2013. Her research interests include palliative care/hospice, transitions of care, and opioid abuse potential pathways for older adults.



Dr. Josée Guindon is an Assistant Professor in the Department of Pharmacology and Neuroscience at TTUHSC. Dr. Guindon obtained her DVM and Ph.D. from Université de Montréal. Since 2006, she has published more than 35 manuscripts, first-authored 6 book chapters, gave more than 30 invited talks and won several prestigious awards. She just got recently awarded her first R01 from National Institute on Drug Abuse (NIDA). She is an expert in the behavioral, pharmacological, biochemical and transgenic analysis of pain mechanisms using various pain models in the cannabinoid field.

Afternoon Workshop Information

Each presented workshop has been designed to give students across each health profession a better understanding of the professional roles and responsibilities of each presented modality. Empirical exposure and personal interactions across all health profession disciplines has proven to be important in building effective interdisciplinary teams.

The afternoon workshops have been registered as an approved TTUHSC Interprofessional Education (IPE) learning activity. Students will receive a certificate upon completion of the workshops.

Students will be placed into interprofessional teams according to their workshop preferences and will participate with their team during the workshop learning activities and facilitator debrief. When registering for the symposium, please select your top workshop preferences. A confirmation email will be sent confirming your placement prior to the event.

PRACTITIONER-BASED CARE WORKSHOPS (1:30-3:00PM)

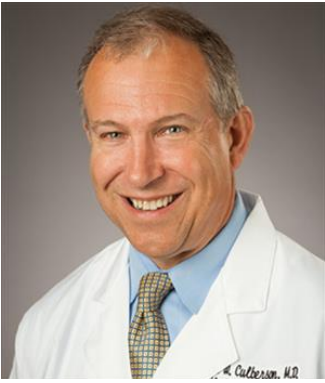
Project Echo: Delivering Interprofessional Education to Remote Sites

Overview: TTUHSC Project Echo for palliative medicine is a case-based videoconference bidirectional learning format. Project Echo involves 40 different worldwide disciplines in 16 countries on nearly every continent. Project Echo is a perfect format for bidirectional, case-based, interdisciplinary/inter-professional learning. This workshop will demonstrate Project Echo as an effective platform for inter-professional education using the concept of "move knowledge, not people". Participants will discuss its use with palliative and geriatric care patients.

Instructors:



Kelly Klein MD: Associate Professor, Department of Family & Community Medicine. After medical school and residency at TTUHSC Lubbock Department of Family Medicine, Dr. Klein practiced family medicine with obstetrics in my hometown of Littlefield TX for over 12 years, then joined the faculty in the Department of Family Medicine in 2011. She currently does full spectrum family medicine, including obstetrics, hospice and palliative medicine. She is the Program Director for the Hospice and Palliative Medicine Fellowship.



John Culberson MD: Associate Professor, Family & Community Medicine and Director, Garrison Institute on Aging, Clinical Geriatric Programs. Dr. Culberson received an MS in Toxicology and Pharmacology from Rutgers University, and an MD from New Jersey Medical School in 1992. He completed residency training in Family Medicine and a Fellowship in Geriatrics at Baylor College of Medicine in Houston, Texas where he continued as an Assistant Professor of Medicine from 2004-2014. Dr. Culberson is board certified in both Family Medicine and Geriatrics, is a Certified Medical Director with the Society of Post-Acute and Long Term Care, and has received two Geriatric Academic Career Awards from the Bureau of Health

Professions. He is currently an Associate Professor of Family and Community Medicine, and holds the Mitemeyer Endowed Chair for Excellence in Geriatric Medicine. Dr. Culberson is the Program Director of the Geriatrics Fellowship, and is a collaborator at the Garrison Institute on Aging. He has published on a wide variety of topics, including Alcohol and Substance Misuse in the Elderly and Medication Reduction in Long Term Care.

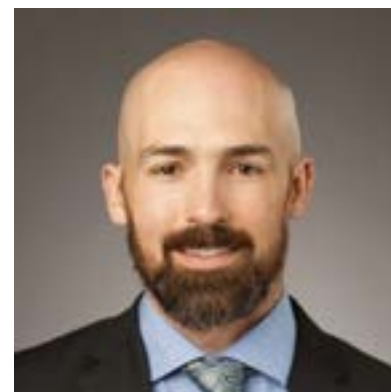


Louis Lux MD: Assistant Professor, Department of Family & Community Medicine. Dr. Lux is board certified in internal medicine and palliative medicine. He has long career in hospital medicine, palliative medicine and hospice. He is the Project Echo launch champion for palliative medicine at TTUHSC with interests in tele-medicine. He is the hospice medical director and educator and scientific counsel advisory board member for hospice.

Dry Needling by Physical Therapists in the Medical Model ACB260B

Overview: Dry needling is a skilled intervention that uses a thin monofilament needle to penetrate the skin and stimulate underlying myofascial trigger points, muscular, and connective tissues for the management of neuromusculoskeletal pain and movement impairments. This is a technique used to treat dysfunctions in skeletal muscle, fascia, and connective tissue, and diminish persistent peripheral nociceptive input, and reduce or restore impairments of body structure and function leading to improved activity and participation. This workshop will present a history of dry needling by physical therapists and discuss the mechanisms of pain relief, clinical application (technique and dosage) and appropriate screening to ensure patient safety and include a demonstration of the dry needling technique. Students will work in interprofessional teams to discuss the benefits of dry needling, discuss what patients would benefit from dry needling therapy, and how it can be used to improve patient care.

Instructor: Gary Kearns is a 2002 graduate from TTUHSC where he earned his Master's in Physical Therapy. He began his manual therapy training with the North American Institute for Orthopaedic Manual Therapy (NAIOMT) in 2005, becoming a Certified Orthopaedic Manipulative Therapist (COMT) in 2010. Graduating from the NAIOMT Fellowship Program, he was recognized as a Fellow in the American Academy of Orthopaedic Manual Physical Therapists (FAAOMPT) in 2010. He graduated with his Doctor of Science (ScD) in Physical Therapy through TTUHSC in 2015. He is currently an Assistant Professor in the Doctor of Physical Therapy Program at TTUHSC. Gary is a guest faculty member for NAIOMT, teaching Dry Needling. Most recently, he became a Board Certified Orthopaedic Specialist (OCS) in 2016.



The Pain Neuroscience Paradigm ACB250

Overview: This workshop will present the learner with an introduction to pain neuroscience including an explanation of pain as a means for the body to describe its safety in the environment instead of a measure of injury.

Instructor: Brad Allen PT, ScD, COMT is an Assistant Program Director and Assistant Professor in the Department of Rehabilitation Sciences at TTUHSC. His responsibilities are split between the Doctor of Science in Physical Therapy and the Doctor of Physical Therapy programs. His primary areas of teaching are related to curriculum design, orthopedic assessment of the spine, upper extremities, and lower extremities, therapeutic modalities and therapeutic exercise. He has almost 25 years of experience as a physical therapist and was the owner of a private outpatient physical therapy practice for 19 years. He completed his Doctor of Science (ScD) program through TTUHSC in 2010.



Integrative Interprofessional Pain Management: A Holistic Approach

Overview: The workshop will discuss the relevance of shared holistic assessment on interprofessional role competencies and communication about allopathic components, complementary and alternative components, and integrative components.

It will introduce several assessment tools for use across the lifespan.

Instructor: Kathy Sridaromont Kathy Sridaromont, RN, PhD, is an Associate Professor and serves as Associate Dean and Department Chair for the Traditional BSN Program. She is one of the founding faculty members of the School of Nursing with 35 years of teaching experience including didactic and clinical supervision, with a recent emphasis on simulation according to best practice. Accolades include Excellence in Teaching, TNA Nurse of the year for TTUHSC, and Distinguished Faculty Award. While serving in courses with populations across the lifespan, pain management has been a priority with contributions to several versions of the textbook on Clinical Application of Nursing Diagnoses. Dr. Sridaromont has a specialized focus in maternal-infant care to include service as founding nursing clinician of neonatal and pediatric critical care at St. Mary's Hospital, Affiliate of Mayo Clinic of Rochester, Minnesota. Her doctoral dissertation was entitled Interrater reliability of the Pediatric Infant Parent Exam: Nursing Screening as a Component of Well-baby Visits at Texas Woman's University. Kathy has been a strong advocate for interprofessionalism and has served as a facilitator for the IPE symposia for the past four years. Kathy is a strong supporter of the Values Based Culture and Global Health Initiatives of TTUHSC as well.



Integrative Pain Management: Where Can We Go From Here?

Overview: Each professional decade brings new treatment golden arrows for clinicians to embrace and implement when treating patients with persistent pain. Often these tools fall short as means for revolutionary results and are prematurely abandoned in response to misdirected utilization. This course will integrate orthopaedic manual therapy, sensorimotor control strategies, biopsychosocial skillsets, pain science education and medical intervention to explore multidisciplinary solutions for redirecting patients' persistent pain experiences. Management alternatives, case studies, demonstrations and practice will be included.

Instructor: Dr. Phillip S. Sizer PT, PhD, OCS, FAAOMPT is an Endowed Professor in Pain Science, TTUHSC President's University Distinguished Professor, and the Associate Dean for Research in the School of Health Professions at TTUHSC in Lubbock, TX, USA,

LECTURER-Phil has lectured at over 450 national and international courses, conferences, and symposia in musculoskeletal pathoanatomy, diagnostics and management; sensorimotor control; and pain science. He continues at TTUHSC as a lecturer, where he has coordinated or instructed in over 250 graduate courses deliveries.

RESEARCHER and SCHOLAR-Phil's research interests include: sensorimotor control and functional biomechanics of spine / extremities; clinical pathoanatomy; and tissue and movement screening. He has authored over 110 peer-reviewed articles, 3 monographs, and 24 editorial commentaries



in refereed journals. He has co-authored 12 international books or book chapters, and 12 Educational DVDs. He participated in over 170 research platform and poster presentations.

INNOVATOR-Phil is a co-founding partner and CEO of TKQuant, LLC and co-inventor of the patent-pending Tis-Kin™ technology. He is the Co-PI of the NSF I-Corps Team: Tissue Kinematics Quantification that conducted over 220 customer discovery interviews.

Opioid Use Disorder: Medication Strategies for Prevention, Rescue, and Treatment

Overview: This workshop will explore various approaches to combat the opioid crisis. Participants in interprofessional teams will learn how to administer naloxone products in an individual encountering an opioid overdose.

Instructor: Dr. Nakia Duncan is an Assistant Professor of Pharmacy Practice at the Texas Tech University HSC SOP-Dallas . She Clinical Pharmacy Coordinator for the Palliative Care team at UT Southwestern her focus is pain/symptom management. She earned her PharmD from Hampton University in 2010 and completed a PGY1, Geriatric pharmacotherapy PGY2, and Pain/Palliative Care Fellowship in 2013. Her research interest include palliative care/hospice, transitions of care , and opioid abuse potential pathways for older adults.



Mindfulness and Pain: Using Acceptance and Commitment Therapy as a Tool to Cope with Physiological and Psychological Pain

Overview: Mindfulness is the act of being aware of what you're sensing and feeling at every moment. Mindfulness-based clinical therapies are increasingly becoming well-known as effective interventions for a range of conditions. The benefits of mindfulness practices that are theorized and/or empirically supported include: improved emotional regulation, reduced rumination, stress reduction, reduced emotional reactivity, and reduced distractibility. This workshop will give a working definition of mindfulness and a description of its key components. Students will learn the simple steps of what constitutes mindfulness meditation with a demonstration of experiential exercises using guided discussion. Following the exercises, interprofessional teams of students will discuss the health benefits of mindfulness and share ways that mindfulness can be used in patient care.

Instructor: Dr. David Trotter is an Associate Professor at TTUHSC in the Departments of Family Medicine and Medical Education. He received his PhD in Clinical Psychology from Texas Tech University. Dr. Trotter completed his internship in Behavioral Medicine from the Alpert Medical School at Brown University and post-doctoral fellowship training in Primary Care Psychology and Motivational Interviewing from the University of Massachusetts Medical School. His clinical areas of expertise include mindfulness based interventions, health behavior change, tobacco cessation, and chronic pain. He also has extensive experience in medical education at the medical school and residency levels.

