The Annual Neuroscience Symposium at Kent State University:
The Neuroscience of Mental Health
April 29, 2013
Kent State University
Kent Student Center, Kiva
Dear Colleague,

It is my pleasure to welcome you to the inaugural Neuroscience Symposium at Kent State University: The Neuroscience of Mental Health. The purpose of this symposium is to bring together nationally renowned neuroscience researchers, clinical practitioners, students and the public to discuss leading-edge university research on mechanisms underlying mood and addiction, injury, disease and the brain.

I'd like to thank the Kent State University Department of Biological Sciences and Department of Psychology in the College of Arts and Sciences for co-sponsoring this event, as well as the members of the Symposium Organizing Committee, listed in the back of the program.

With nearly 40 neuroscientists working across multiple departments, Kent State has considerable strength in a broad range of the neurosciences, from molecular biology to behavior, producing interdisciplinary research on neurological diseases and conditions including: traumatic brain injury, post-traumatic stress disorder, the effects of aging, addiction and pain management. This symposium provides a venue for exchange of the scientific ideas that must take place to solve and treat these critical healthcare problems.

We are delighted to welcome our presenters today, including keynote speakers, Dr. Randy Nelson, Brumbaugh Chair in Brain Research and Teaching; professor and chair, Department of Neuroscience; and distinguished professor, College of Medicine, The Ohio State University Wexner Medical Center — and Dr. Kevin Guskiewicz, Kenan Distinguished Professor and director of the Matthew Gfeller Sport-Related Traumatic Brain Injury Research Center at The University of North Carolina at Chapel Hill.

I would like to thank you for attending and participating today, and I look forward to seeing you at our future neuroscience symposia on an annual basis. If you would like more information about our latest research initiatives in the neurosciences, please take a copy of our Kent State Neuroscience Researcher Directory which includes contact information, research interests, and major publications for each researcher.

If you would like more information about our other research programs or for partnering opportunities with us, please contact me at research@kent.edu or 330-672-3012.

Sincerely,

Grant McGimpsey, Ph.D.
Vice President for Research
Kent State University
EVENT PROGRAM

Monday, April 29, 2013
Kent State University,
Kent Student Center,
Kiva Auditorium

9 - 9:30 a.m. Registration in the Kiva Lobby
9:30 - 9:40 a.m. Welcome and Comments
   Lester Lefton, Ph.D.
   President, Kent State University
9:40 - 9:45 a.m. Overview
   Grant McGimpsey, Ph.D.
   Vice President for Research, Kent State University

First Session: Mechanisms Underlying Mood and Addiction – 9:45 a.m. - 2:30 p.m.
   Session Moderator: Eric Mintz, Ph.D. Associate Professor and Director,
   School of Biomedical Sciences, Kent State University

9:45 - 10:30 a.m. Morning Keynote Address:
   “Effects of Light at Night on Neuroinflammation, Metabolism and Mood”
   Randy Nelson, Ph.D., The Ohio State University
10:30 - 11 a.m. “Ethanol Disrupts Biological Clock Timing:
   Considering Alcoholism as a Circadian-based Disease”
   J. David Glass, Ph.D., Kent State University
11 - 11:30 a.m. “Circadian Genes, Rhythms and the Biology of Mood Disorders”
   Colleen McClung, Ph.D., University of Pittsburgh
11:30 a.m. - Noon “Adolescence: A Period of Increased Vulnerability to Cocaine Addiction”
   Michela Marinelli, Ph.D., Rosalind Franklin University of Medicine and Science

Noon - 1:30 p.m. Lunch
1:30 - 2:30 p.m. Panel discussion moderated by Eric Mintz

Second Session: Injury, Disease and the Brain, 2:45 - 7:15 p.m.
   Moderator: Douglas Delahanty, Ph.D., Professor
   Department of Psychology, Kent State University

2:45 - 3:15 p.m. “Strategies for the Preclinical Detection and Prevention of Alzheimer’s Disease”
   Stephen M. Rao, Ph.D., Cleveland Clinic
3:15 - 3:45 p.m. “Can Weight Loss Reduce Your Risk of Alzheimer’s Disease?”
   John Gunstad, Ph.D., Kent State University
3:45 - 4:15 p.m. “When Brain Injury and Psychological Trauma Co-Occur: Can we Disentangle the Consequences?”
   Jennifer Vasterling, Ph.D., Boston University
4:15 - 5:15 p.m. Reception, Ballroom Balcony, Kent Student Center
5:30 - 6:30 p.m. Evening Keynote/Public Address: “Making Sport Safer Through Innovative Science”
   Kevin Guskiewicz, Ph.D., University of North Carolina, Chapel Hill
6:30 - 7:15 p.m. Panel Discussion moderated by John Gunstad
Randy J. Nelson, Ph.D.
9:45 - 10:30 a.m.

“Effects of Light at Night on Neuroinflammation, Metabolism and Mood”

Bio:
Randy J. Nelson, Ph.D. holds the Brumbaugh Chair in Brain Research and Teaching at The Ohio State University Wexner Medical Center. He is professor and chair of the Department of Neuroscience and a member of the Institute for Behavioral Medicine at The Ohio State University Medical Center. He also holds joint appointments in the Department of Psychology and Department of Evolution, Ecology and Organismal Biology at Ohio State. Nelson earned his A.B. degree in psychology at the University of California at Berkeley. After receiving his M.A. in psychology, he began focusing on circadian rhythms and photoperiodism with Dr. Irving Zucker. He simultaneously earned a Ph.D. in psychology and a separate Ph.D. in endocrinology from University of California at Berkeley, then went on to complete a postdoctoral fellowship in reproductive physiology at the Institute for Reproductive Biology at the University of Texas at Austin. Nelson served on the faculty at The Johns Hopkins University for 15 years before moving to Columbus, Ohio. He has published more than 350 research papers and several books describing studies in seasonality, behavioral endocrinology, biological rhythms, immune function, sex behavior and aggressive behaviors. He is the recipient of The Ohio State University Alumni Distinguished Teaching, Distinguished Scholar and the University Distinguished Lecturer Awards. In 2012, he was appointed as Distinguished Professor of the Ohio State Wexner College of Medicine.

Abstract:
Technological advances, while providing many benefits, often create circumstances that differ from the conditions in which we evolved. With the widespread adoption of electrical lighting during the 20th century, humans became exposed to bright and unnatural light at night for the first time during their evolutionary history. Electrical lighting has led to the wide-scale practice of 24-hour shift-work and has meant that what were once just “daytime” activities run throughout the night; in many ways, western society now functions on a 24-hour schedule. Recent research suggests that this gain in freedom to function throughout the night may also come with significant repercussions. Disruption of our naturally evolved light and dark cycles can result in a wide range of physiological and behavioral changes with potentially serious medical implications. In this presentation, the disruptive effects of light at night on several core clock genes and the resulting neuroinflammation will be discussed. Increased brain inflammation may be a potent mechanism through which light at night affects mood and food intake.
J. David Glass, Ph.D.
10:30 - 11 a.m.

"Ethanol Disrupts Biological Clock Timing: Considering Alcoholism as a Circadian-based Disease"

Bio:
J. David Glass, Ph.D., is professor of biological sciences at Kent State University. Glass earned a B.S.C. in biology from the University of Victoria, British Columbia, a M.S.C. in zoology from the University of Alberta, Edmonton, and a Ph.D. in biology from Wesleyan University, Middletown. He was a postdoctoral fellow in the reproductive endocrinology program of the Department of Physiology and Biophysics at Colorado State University. His program of research at Kent State, continuously funded by federal agencies for more than 22 years, is focused on the neurobiology of mammalian biological rhythms. One aim of the research is to study how environmental information is integrated by the circadian clock to regulate the timing of daily behavioral, physiological and neuroendocrine rhythms. The second aim is to study the effects of ethanol on the circadian timing system.

Abstract:
Alcohol abuse leads to marked disruptions of sleep and circadian rhythms. These disturbances can promote excessive drinking, resulting in a cycle of circadian dysfunction and increasing alcohol intake. Here we summarize our complimentary studies demonstrating the actions of ethanol on critical systems that regulate the circadian clock of the suprachiasmatic nucleus (SCN). In vivo, in hamsters and mice, acute ethanol administration inhibits the phase-resetting effect of light on circadian behavioral rhythms. This treatment also impairs non-photic phase-shifting mediated by serotonergic stimulation. Intra-SCN administration of ethanol also blocks photic shifting. Ethanol also disrupts entrainment to a weak skeleton photoperiod, but not to a normal light-dark cycle. In vivo, Similar to the in vivo trials, ethanol treatment of SCN-containing brain slices blocks photic-like (glutamate-induced) phase shifts of clock-driven circadian neuronal activity. This effect is dose-dependent and is blocked by BDNF pre-treatment. Ethanol modulation of glutamatergic and serotonergic phase resetting exhibits acute tolerance, as 15-30 min ethanol pretreatment blocks these effects. Ethanol also modulates GABAergic phase-resetting in the SCN, likely through enhanced signaling of d subunit-containing GABA receptors. The SCN also exhibits rapid (24 h) ethanol tolerance, as assessed through glutamate phase resetting in tissue from ethanol-consuming mice. Together, these results confirm ethanol's significant disruption of SCN photic and non-photic phase-resetting responses. These effects involve direct actions of ethanol in SCN serotonergic, glutamatergic, BDNF, and GABAergic signaling. These results imply that alcohol abuse and alcoholism should be considered in the context of circadian-based pathology. Interventions that strengthen circadian rhythm structure (i.e. appropriately-timed exercise and/or bright light exposure) should be implemented alongside conventional treatments. Supported by NIH AA-015948 and AA-017898 grants.
Colleen A. McClung, Ph.D.
11 - 11:30 a.m.

"Circadian Genes, Rhythms and the Biology of Mood Disorders"

Bio:
Work in the McClung lab focuses on the molecular biology of mood disorders and drug addiction. McClung obtained her B.S. in biology and minor in chemistry at the University of North Carolina, Chapel Hill in 1994, where she worked with Dr. Bryan Kay on the mechanisms of transcriptional regulation using bacterial phage display. For her Ph.D. thesis at the University of Virginia in the lab of Dr. Jay Hirsh, she pioneered the use of drosophila as a model system to study the genes involved in cocaine sensitization. She went on to do her postdoctoral work with a leader in molecular psychiatry, Dr. Eric Nestler at University of Texas Southwestern Medical Center. There she studied the importance of transcription factors in the regulation of drug reward and mood-related behavior using mouse models. Through work as both a graduate student and a postdoc, she became interested in the role of genes that control circadian rhythms and central rhythm disruptions in the development of addiction and mood disorders. She became an assistant professor of psychiatry at University of Texas Southwestern in 2005 and her lab focused most of its attention on the regulation of dopaminergic reward circuitry by the circadian genes in the context of mood and addiction-related behavior. This work has been very successful and she has numerous publications and awards. Her lab moved to the University of Pittsburgh School of Medicine in the summer of 2011, where she is currently an associate professor of psychiatry.

Abstract:
Mood disorders are serious diseases that affect a large portion of the population. Nearly all people suffering from mood disorders have significant disruptions in circadian rhythms and the sleep/wake cycle. In fact, altered sleep patterns are one of the major diagnostic criteria for these disorders. Moreover, environmental disruptions to circadian rhythms including shift work, overseas travel and irregular social schedules tend to precipitate or exacerbate mood-related episodes. Recent studies have found that molecular clocks are found throughout the brain and body where they participate in the regulation of most physiological processes, including those thought to be involved in mood regulation. This seminar will summarize the evidence linking circadian rhythm disruptions with mood disorders, including the circadian rhythm abnormalities that tend to associate with specific disorders, as well as the effect of current treatments on rhythms. Moreover, I will summarize recent data which implicates the circadian system as a vital regulator dopaminergic signaling which is important in the regulation of mood and reward-related behavior.
Michela Marinelli, Ph.D.
11:30 a.m. - Noon

"Adolescence: A Period of Increased Vulnerability to Cocaine Addiction"

Bio:
Michela Marinelli (Micky) obtained her Ph.D. in pharmacology and neuroscience at the University of Bordeaux 2, France. After post-doctoral training in the United States, she was hired as an assistant professor by the French INSERM (the French equivalent to the American National Institutes of Health). Three years later, in 2003, she was recruited by the Department of Cellular and Molecular Pharmacology at the Rosalind Franklin University of Medicine and Science in North Chicago, where she currently works as an associate professor. Her lab seeks to understand how changes in dopamine neuron activity may contribute to drug addiction liability. Addiction is modeled in rodents using different behavioral paradigms such as voluntary drug-intake (intravenous self-administration) and drug-seeking tests. Dopamine neuron activity is studied with electrophysiological techniques such as in vivo extracellular and ex vivo patch clamp recordings. To study the circuits underlying addiction, electrophysiology is coupled with optogenetics.

Abstract:
In humans, adolescence is a period of heightened propensity for cocaine addiction. It is unknown if this is due to greater access/exposure to cocaine at this age, or if the adolescent brain is particularly susceptible to addiction. We determined if adolescent rats show elevated activity of midbrain dopamine neurons, a trait associated with heightened susceptibility to addiction. Dopamine neuron activity showed an inverted U-shaped curve from weaning to adulthood, with peak activity during adolescence. Heightened dopamine neuron activity during adolescence was observed both in vivo, with extracellular recordings in anesthetized rats, and ex vivo, with cell-attached recordings from midbrain slices. In adolescents, GABA-A receptor-mediated sPSCs occurred at lower frequency and smaller amplitudes, suggesting a possible mechanism underlying heightened dopamine neuron activity during adolescence. Elevated dopamine neuron activity during adolescence was associated with elevated addiction liability according to DSM-IV criteria, tested with intravenous cocaine self-administration. Increased cocaine self-administration in adolescents could be reversed by administering drugs that modify dopamine neuron activity, suggesting a causal relationship between these electrophysiological and behavioral determinants of cocaine addiction. In conclusion, these studies demonstrate that neurophysiological differences during development underlie the heightened addiction liability observed during adolescence.
Stephen M. Rao, Ph.D.
2:45 - 3:15 p.m.

"Strategies for the Preclinical Detection and Prevention of Alzheimer's Disease"

Bio:
Stephen M. Rao, Ph.D., ABPP-CN is the Ralph and Luci Schey Chair and director of the Schey Center for Cognitive Neuroimaging at the Cleveland Clinic and professor, Cleveland Clinic Lerner College of Medicine of Case Western Reserve University. He obtained his Ph.D. in clinical psychology from Wayne State University (Detroit) and completed a predoctoral internship at Rush-Presbyterian-St. Luke’s Medical Center (Chicago). He has authored over 155 scientific papers/book chapters and edited four books. His current research areas involve the application of advanced neuroimaging techniques (task-activated and resting-state fMRI; diffusion tensor imaging) to understand the disruption of brain circuits mediating memory (working, episodic and semantic), selective and sustained attention, motor control, temporal information processing and conceptual reasoning in patients with multiple sclerosis, individuals in the preclinical stage of Alzheimer’s and Huntington’s diseases, and military personnel with blast-related traumatic brain injury. He is the editor-in-chief of *Neuropsychology* (published by the American Psychological Association), has served as the associate editor of *Journal of the International Neuropsychological Society*, and has been a member of the editorial boards of eight other journals. He is the past president of the International Neuropsychological Society (INS) and has served board of directors of the American Board of Clinical Neuropsychology.

Abstract:
The neuropathological changes associated with Alzheimer’s disease (AD) are thought to begin decades prior to the advent of symptoms. By the time AD is diagnosed, patients have experienced widespread cognitive impairment and extensive brain atrophy. Interventions administered at this stage may be too late to alter the disease trajectory. This recognition has prompted investigators in the field to consider prevention trials involving cognitively intact individuals at genetic risk for AD. The first wave of such prevention trials will focus on persons with the autosomal-dominant mutation variant of AD, since it is possible to determine with certainty that the participant will develop AD. Should these prevention trials identify an agent that alters the disease course during the preclinical phase, the next step will be to enroll cognitively intact elders at risk for the more common, sporadic form of AD. Prior to conducting a prevention trial in sporadic AD, it is essential to validate biomarkers for purposes of accurately identifying persons at risk for AD during the preclinical phase and for monitoring treatment response since such individuals are, by definition, asymptomatic. Several candidate biochemical, anatomical and functional biomarkers have shown promise, but vary in their ease of administration, availability, safety, accuracy, reproducibility and invasiveness. Among these candidate biomarkers, task-activated functional magnetic resonance imaging (fMRI) is a promising approach that is noninvasive, carries little risk and offers a high potential for identifying persons who may eventually develop AD. My fMRI research program has examined brain circuits involved in semantic memory using a famous-name recognition. We have demonstrated that the presence of the apolipoprotein-E (APOE) E4 allele and a family history of dementia are associated with increased activation in several key AD-related brain regions (e.g., hippocampus, posterior cingulate, inferior parietal), suggesting its possible role as a marker for disease risk and progression during the preclinical stage. The value of task-activated fMRI for monitoring treatment efficacy will also be discussed in the context of a prevention trial involving cognitive training and aerobic exercise interventions.
John Gunstad, Ph.D.
3:15 - 3:45 p.m.

"Can Weight Loss Reduce Your Risk of Alzheimer's Disease?"

Bio:
John Gunstad obtained a B.A. in psychology from Moorhead State University and both his M.S. and Ph.D. in clinical psychology with concentrations in clinical neuropsychology and health psychology from Ohio University. He then completed internship and postdoctoral fellowship in clinical neuropsychology at Brown Medical School, where he began a line of work in the neurocognitive effects of medical conditions including obesity and cardiovascular disease. Gunstad is associate professor in the Department of Psychology at Kent State University. He has more than 125 publications and series of NIH grants, including projects examining the cognitive benefits of bariatric surgery and role of exercise in protecting the brain in heart failure. His research showing improved memory after weight loss surgery was featured on "World News with Diane Sawyer" and hundreds of print and digital news outlets. Gunstad received the Early Career Research Award from the International Neuropsychological Society and awards for both teaching and research from Kent State University. He is frequently asked to present on his research, including a recent event at the New York Academy of Sciences.

Abstract
Obesity is linked to many adverse brain changes, including accelerated cognitive decline and Alzheimer's disease. Less clear is the possibility that obesity-related cognitive dysfunction could be reversed through significant weight loss. Obesity is associated with many conditions with partly reversible cognitive deficits, including hypertension, type 2 diabetes, sleep apnea and depression. Our team is examining this possibility by prospectively assessing cognitive function in persons that undergo bariatric surgery. Interestingly, patients show improved memory function after bariatric surgery and these improvements persist over time. Future studies are much needed to determine whether these cognitive benefits persist at later follow-up (e.g. 60 months) when weight re-gain is common and the degree to which bariatric surgery may reduce risk for Alzheimer's disease or other neurological disorders in older adulthood.
Jennifer J. Vasterling, Ph.D
3:45 - 4:15 p.m.

"When Brain Injury and Psychological Trauma Co-Occur: Can we Disentangle the Consequences?"

Bio:
Jennifer J. Vasterling obtained her Ph.D. in psychology from Vanderbilt University in 1988, subsequently completing pre- and post-doctoral training in clinical neuropsychology at the Boston Veterans Affairs Medical Center. She currently serves as the chief of psychology at the Veterans Administration Boston Healthcare System and as a clinical investigator within the Behavioral Science division of the Veterans Administration National Center for Post-Traumatic Stress Disorder. Vasterling is a professor of psychiatry at Boston University School of Medicine and a Lecturer in Psychiatry at Harvard Medical School. Vasterling’s research has centered on further understanding the neurocognitive and emotional changes that accompany war-zone deployment and post-traumatic stress responses. She has edited several books, including a volume on comorbid PTSD and mild traumatic brain injury. Her recent work includes a longitudinal study examining neuropsychological and emotional outcomes of military deployment to Iraq.

Abstract:
The wars in Iraq and Afghanistan have highlighted the often complex clinical outcomes of traumatic brain injury events that occur within the context of exposure to extreme psychological stress. Traumatic brain injuries embedded within psychological trauma are not limited to military contexts, however, and also include such events as physical assault, industrial accidents and motor vehicle accidents. This presentation will discuss the potential contributions of traumatic brain injury and psychological trauma to neuropsychological outcomes, as well as their potential neural overlap. The presentation will emphasize mild brain injuries with data presented from a longitudinal study of neuropsychological outcomes of U.S. Army soldiers deployed to Iraq early in the war. A conceptual model of how psychological trauma, traumatic brain injury, and their consequences may adversely affect recovery will also be presented.
Kevin Guskiewicz, Ph.D.
5:30 - 6:30 p.m.

Making Sport Safer Through Innovative Science

Bio:
Kevin Guskiewicz is the Kenan Distinguished Professor, Co-Director of the Matthew Gfeller Sport-Related TBI Research Center, and Director of the Center for the Study of Retired Athletes at The University of North Carolina at Chapel Hill. Over the past 18 years, his clinical research program has investigated the effect of sport-related concussion on balance and cognitive function, the biomechanics of sport concussion and the long-term neurological effects of concussion in professional football players. Guskiewicz has received 24 funded research grants, and published more than 130 journal articles and 8 textbook chapters on sport concussion. Additionally, he has presented more than 250 national and international lectures on this topic. He teaches courses in sports medicine, anatomy, and research methods at UNC, and has been awarded fellowships in the American College of Sports Medicine, National Academy of Kinesiology and the National Athletic Trainers' Association. He serves on NCAA's Concussion Committee, NFL's Head, Neck and Spine Committee, and NFLPA's Mackey-White Committee. In 2011, Guskiewicz was awarded a prestigious MacArthur Fellowship.

Abstract:
Sport-related concussion is one of the most complex injuries facing sports medicine clinicians charged with making sound return to play decisions. There are many pieces to the concussion puzzle and until recently, the biomechanics of concussion has been largely ignored. Although recent studies of collegiate football players and youth ice hockey players indicate that the biomechanical threshold for concussive injury is still illusive, tracking head impacts in real time and translating the findings into models for injury prevention is improving safety. This presentation aims to summarize findings from these recently conducted studies investigating biomechanical relationships with various factors such as playing position, types of play, concussive versus sub-concussive impacts, location of impacts and clinical measures of concussion. Combining the biomechanical findings of active athletes with those of retired professional athletes may allow us to better understand the long-term consequences of concussive and sub-concussive impacts to the head.
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