

# Biopsychology

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# BIOPSYCHOLOGY AT THE UNIVERSITY OF MICHIGAN

## Preamble

The Biopsychology Area at the University of Michigan is a subdivision within the Department of Psychology that is committed to the belief that studies of behavior and biology complement each other, and that both are enhanced when they are combined in a common effort. The underlying philosophy of the Biopsychology Area is that there is a strong need for research at the interface of behavior, biology, and evolutionary theory. Students typically pursue graduate studies involving the investigation of *'Brain and Behavior Relationships'* (e.g., Physiological Psychology or Behavioral Neuroscience), or the *'Evolution of Behavior'* (e.g., Sociobiology or Comparative Animal Behavior), although students are encouraged to sample both of these approaches during their graduate career. In practice, research activities of the staff range from field observations of animal social behavior to recording the activity of single brain cells, and molecular and cellular manipulations and measure of brain mechanisms. The main research interests of the faculty are in one or more of the following sub-areas: learning and memory, attention, evolutionary basis and the adaptive significance of behavior, motivation and emotion, hormones and behavior, biological rhythms, cognitive neuroscience, stress neurobiology, neuropsychopharmacology, and social cognition and communication.

Each graduate student has a pre-candidacy advisory committee made-up of three Biopsychology faculty members to help them select courses and training appropriate to their goals. A synopsis of program requirements is given below, and more detailed information on available courses and training opportunities may be obtained by contacting the Chair of the Biopsychology Area. The Ph.D. Program in Biopsychology is strongly research-oriented, and students are required to initiate a research project (Psychology 619) in collaboration with a faculty member early in their first year of graduate study. They are also required to write a report and to give an oral presentation based on this project, typically by the Fall Term of their second year. Students continue research during their second year, along with course work, and take their Preliminary Examination at the end of the second year.

## Synopsis of Biopsychology Program Requirements

1. *First year research project and oral presentation* (Psychology 619). Students are expected to become involved in a research project early in their first semester in the program. Before the end of the fall semester of their second year they must give an oral presentation on their research project. Before the beginning of the third year, and advancement to candidacy, students must present a research paper describing their research for approval to their

research mentor and one other Biopsychology faculty member.

2. *Required introductory course:* All students are required to take the Advanced Seminar and Practicum in Physiological Psychology (Psy 731) in their first two years. Psy 731 cannot be used to replace one of the required, three advanced lecture and seminar courses (as detailed in 3).

3. A total of three *advanced lecture or seminar courses* relevant to biopsychology must be taken, and at least two of these must be at the '600-level' or above. The faculty advisors will assume the responsibility for assuring that the student's course selection is adequate preparation for their professional career. A signed approval note listing the three courses should be sent to the Biopsychology office.

Students are strongly encouraged to take at least one course in Neuroscience *and* one in Evolutionary Biology. There are a number of courses that meet these requirements, and the appropriate selection for a given student is determined by the student, in consultation with their advisory committee. Courses that have been approved in the past include: Biol 425/ NS 625 (Systems Neurobiology), CMB 422/ NS 622 (Cellular and Molecular Neurobiology), NS 570/571 (Human Neuroanatomy), NS 601 and 602 (Principles of Neuroscience I & II, modules can be taken independently for 1 credit each), Physiol 541/ Psych 532 (Mammalian Reprod. Endocrinology), Physiol/NS 693 (Nervous System Structure and Function), Biol 492 (Behavioral Ecology), Psych 530 (Advanced Comparative Animal Behavior), Anthropology 478 (Primate Behavioral Ecology and Sociobiology), Psych 831 (Seminar in Physiological Psychology). Students may also take advanced undergraduate courses, including Psych 433-438.

Other courses may be approved at the discretion of a student's advisory committee. [Note: Courses cannot be double counted between the categories; e.g., a Neuroscience course taken to meet a Biopsychology relevant course cannot also be counted as a Rackham cognate or Psychology breadth course.]

4. *Biopsychology Colloquium:* All students are expected to attend the weekly Area colloquium series.

5. *Departmental Breadth Requirement:* Graduate students are strongly encouraged to attend the monthly Departmental colloquium. In addition, all students must take *one* Psychology course in an area besides Biopsychology (i.e., a course not taught primarily by Biopsychology staff) sometime during their first two years, prior to candidacy. Students should seek the advice of their advisory committee in fulfilling this Psychology "breadth" requirement.

6. A one-year sequence of *statistics* (e.g., Psychology 613-614) or approved substitute must be taken.

7. Rackham requires a minimum of 4 credits of *cognate courses* outside of psychology. These courses should be related to the professional goals of the student and approved by advisors (e.g., neuroanatomy, biochemistry, “evolution courses” in biology or anthropology, etc.). Courses used to meet the Biopsychology advanced course or breadth requirement cannot be used to meet the Rackham cognate requirement.

8. *Preliminary Examination*: Normally, graduate students will take their Prelim Exam in May at the end of the second year. However, dates can be adjusted to accommodate research (especially field work) and class schedules.

The exam format will consist of students selecting one question from a list of questions prepared by the faculty. The purpose of the exam is to assess the ability of a student to think logically about a problem area and to formulate research questions, rather than assessing the amount of information they possess. Students will have 2.5 weeks to write a response in the format of a grant application in which they provide some background to the research area, generate experimental hypotheses, propose experiment(s) to test hypotheses, and discuss how results would be interpreted. The document is about 12 pages of double-spaced text. After the faculty has read the papers, an oral exam is held with a committee of 3 faculty. Students normally achieve Ph.D. candidate status by September of the third year in the program. After candidacy status is achieved, a Dissertation Committee is formed to advise on dissertation research and to evaluate the thesis when submitted.

To maintain full-time status, Pre-candidates must register for a minimum of 9 credits and a maximum of 18 credits. If they have been recommended for Candidacy, they should register for 8 credits of Psych 990. Once advancement to Candidacy has been approved by Rackham, the Registrar's Office will change the 990 enrollment to 995. For each Fall and Winter semester, Candidates will register for 8 credits of Psych 995; they also have the option of enrolling in one additional course per term. Students must enroll in each Fall and Winter term up to and including the semester in which they defend their dissertation.

At least two members of the dissertation committee must be core Biopsychology faculty and Rackham requires that one member be from a Department other than Psychology. The dissertation defense of students in the Biopsychology Area is a public talk given as part of the Biopsychology Colloquia Series followed by an oral exam.

## Application Procedures

All applicants must take the Graduate Record Examination. GRE registration forms may be obtained online at <http://www.gre.org>. This test should be taken early in the senior year.

The Rackham Graduate School application deadline is December 1. All application materials are to be submitted online. The application process can be started at

[http://www.rackham.umich.edu/admissions/prospective\\_students\\_degree/](http://www.rackham.umich.edu/admissions/prospective_students_degree/)

For further information about application procedures or any aspect of the graduate program in Biopsychology contact the Chair of Biopsychology:

Dr. Martin Sarter  
Chair, Biopsychology Program  
Department of Psychology  
The University of Michigan  
530 Church Street  
Ann Arbor, MI 48109  
Telephone: (734) 764-6392  
E-mail address: [msarter@umich.edu](mailto:msarter@umich.edu)

All applicants will be notified of admission decisions by April 15, and usually before March 1.

## Financial Support

The comprehensive financial support package the department offers typically provides five (5) academic years of stipend and tuition: two (2) academic years of support as a Research Fellow and three (3) academic years of support as a Graduate Student Instructor (GSI), all with health care and tuition. As a Research Fellow you will engage in your own research as well as work in collaboration with faculty in Psychology. The financial offer is equivalent for all students that are admitted into any of the areas of psychology.

The graduate program comprises two phases: 1) the first two years when a student is taking courses and acquiring specific intellectual and research skills necessary to become a candidate for the Ph.D., and completing their first research project; and 2) years 3-5 when a student is conducting their dissertation research and strengthening other skills. Similarly, the support package is in two phases. After the first year in the graduate program as Research Fellows, summer support is also provided to students for in the first summer. Some additional summer support in later years is provided by the department for an additional

two (2) summers of the student's choice. Students may be able to obtain summer support for additional years beyond these by winning fellowships, training grant slots. Also, mentors may sometimes be able to provide financial support during the summer months on research grants, and a student should discuss this with their mentor.

During years 2-4 students will typically begin developing their teaching skills with GSI support. Subject to acceptable performance, Psychology graduate students can count on six (6) semesters of support as a GSI. The department has a renewed emphasis on helping graduate students become excellent teachers, and the terms of GSI experience are a fundamental part of the student's professional development. A minimum of two (2) terms of teaching are required to fulfill the department's doctoral degree requirements.

During years 3-5, students conduct their dissertation research and have the opportunity to expand their teaching experiences. In the fifth year, students will be supported on a fellowship to facilitate the completion of their dissertation.

#### *Fellowships from External Agencies*

While support is guaranteed as described for 5 years, all eligible students are to apply for funding from NSF during their first year of graduate school or other sources of funding throughout their graduate career. If a student receives funding from an external foundation or institute (NSF, NIH, etc.) the department can combine funds in a way that will enhance the overall support package.

An undergraduate who considers applying to Ph.D. programs is well-advised to explore the many sources of individual fellowship support available to outstanding students, such as NSF fellowships (described below). Graduate students may also apply for NIH fellowships (NRSAs) or for slots on one of several NIH training grants held by the University. Other graduate students have sometimes served as Research Assistants, receiving support from research grants held by their mentors. Many of these fellowships provide several years of support.

Exceptional students are strongly encouraged to apply for a National Science Foundation predoctoral fellowship during their senior undergraduate year. These fellowships give three years of support. Information and application forms may be obtained either directly from the National Science Foundation web page (<http://www.fastlane.nsf.gov>) or from the Department of Psychology Student Academic Affairs Office.

We would like to encourage all prospective graduate students to become familiar with the Biopsychology staff

and their research interests. Following is a list of the members of the staff with a short description of their research interests and representative publications. Many Biopsychology faculty have web pages for their lab, which list current research projects and may have reprints of recent publications. If you would like to know more about the work of a particular staff member, feel free to email directly to that person.

**Biopsychology Staff, Research Interests and Representative Publications**

**J. Wayne Aldridge:** Our long-term goal is to understand how individual neurons and neuronal circuits in the basal ganglia contribute and process information related to movement and rewards. Our approach is to record electrical activity of individual nerve cells while animals respond to sensory cues and rewards and execute natural or learned movements. We also activate neural systems by the application of dopaminergic drugs that are known to affect motor behavior and motivational systems. This research is relevant to understanding normal brain function and neurological disorders such as Parkinson's disease, Huntington's disease, Tourette Syndrome, drug addiction and other disorders related to basal ganglia dysfunction.

**Representative Publications:**

Aldridge JW, Berridge KC: Coding of serial order by neostriatal neurons: a 'natural action' approach to movement sequence. *J. Neurosci* 1998; 18: 2777-2787.

Berridge KC, Aldridge JW: Super-stereotypy I: Enhancement of a complex movement sequence by systemic dopamine D1 agonists, *Synapse* 2000; 37: 194-204

Meyer-Luehmann M, Berridge KC, Thompson JF, Aldridge JW: Substantia nigra pars reticulata neurons code initiation of a serial pattern: Implications for natural action sequences and sequential disorders, *European J. Neurosci.* 2002; 16:1599-1608.

Tindell AJ, Berridge KC, Aldridge JW. Ventral pallidal representation of Pavlovian cues and reward: population and rate codes. *J. Neurosci* (in press).

Aldridge JW, Thompson JF, and Gilman S (1997). Unilateral striatal lesions in the cat disrupt well-learned motor plans in a GO/NO-GO reaching task. *Exp Brain Res.*, 113, 379-393.

**Brandon Aragona:** Evolution has favored brains that produce robust motivated behaviors that promote individual and species survival. This broad perspective serves as the foundation for my two specific lines of research. The first is focused on the neural regulation of a highly adaptive social behavior, monogamous pair bonding. The second is focused on the neural regulation of a maladaptive behavior, taking addictive drugs.

Prairie voles are a monogamous rodent species that form life-long pair bonds with their mates. My previous work has demonstrated that this behavior is controlled by brain circuitry that is essential for reward processing (including reward associated with addictive drugs). Most recently,

we have shown that abused drugs are less rewarding to prairie voles that are pair bonded. Prairie voles are therefore both an excellent model for studies of the neurobiology of social attachment and for investigation of interactions between social behavior and drug reward.

Addictive drugs powerfully control behavior because they target neural circuitry that controls motivated behavior essential for survival. I am very interested in how drugs, such as cocaine, alter this circuitry. In particular, I use state-of-the-art measurement technology (fast-scan cyclic voltammetry) to assess real-time dopamine transmission while rats receive drug infusions and learn that certain environmental cues predict drug delivery.

Questions to be addressed by future research include:

What changes in the brain control the formation and maintenance of a monogamous pair bond?

How is dopamine transmission altered during social interactions?

How is drug reward influenced by adaptive social behavior?

What mechanisms control increased dopamine signaling with drug intake?

What is the role of specific regions of the brain during learning and memory associated with drug taking?

What makes certain individuals highly susceptible to drug reward?

What protective steps (especially of a social nature) can be taken to lessen the development of drug addiction?

**Representative publications:**

Aragona B.J., Y. Liu, J.T. Curtis, F.K. Stephan, and Z.X. Wang (2003) A critical role for nucleus accumbens dopamine in partner preference formation in male prairie voles. *Journal of Neuroscience*, 23: 3483-3490.

Aragona B.J., Y. Liu, Y. Joy Yu, J.T. Curtis, J.M. Detwiler, T.R. Insel, and Z.X. Wang (2006) Nucleus accumbens dopamine differentially mediates the formation and maintenance of monogamous bonding. *Nature Neuroscience*, 9: 133-139.

Aragona B.J., J.M. Detwiler, and Z.X. Wang (2007) Amphetamine reward in the monogamous prairie vole. *Neuroscience Letters*, 418 (2): 190-194.

Cheer J.F., B.J. Aragona, M.L. Heien, A.T. Seipel, R.M. Carelli, R.M. Wightman (2007) Coordinated accumbal dopamine release and neural activity drive goal-directed behavior. *Neuron*, 54 (2): 237-244.

Aragona B.J., and Z.X. Wang (2007) Opposing regulation of pair bond formation by cAMP signaling within the nucleus accumbens shell. *Journal of Neuroscience*, 27 (48) 13352-6.

**Jill B. Becker:** Female and male brains differ. Differences begin early during development due to a combination of genetic and hormonal events and continue throughout the lifespan of an individual. Physiological sex differences account for marked differences in disease incidence, manifestation, prognosis and treatment observed between the sexes. Research in my laboratory integrates behavioral and neurochemical methodologies to investigate the relations between brain activity and sexually dimorphic behaviors. The current focus of the laboratory is on the nigrostriatal dopamine system. This area of the brain is the area damaged in Parkinson's Disease, and loss of this brain region results in loss of the ability to initiate movement. During normal function, this area of the brain is involved in the integration of sensory and motor information, learning and motivated behaviors. In my laboratory we are investigating how the ovarian hormones, estrogen and progesterone, act on this neural system to influence sexual behavior and drug abuse. We have shown that estradiol treatment of female rats, but not males, enhances the behavioral response to amphetamine and cocaine, and enhances the acquisition of cocaine self-administration. The laboratory is currently investigating how and where estradiol acts in the brain to produce these effects.

#### **Representative Publications:**

Jenkins WJ, Becker JB (2003) Dynamic increases in dopamine during paced copulation in the female rat. *Eur J Neurosci* 18: 1997-2001.

Hu M, Becker JB (2003) Effects of sex and estrogen on behavioral sensitization to cocaine in rats. *J Neurosci* 23: 693-699.

Becker, J. B., Arnold, A., Berkeley, K. J., Blaustein, J. D., Eckel, L. A., Hampson, E., Herman, J. P., Marts, S., Sadee, W., Steiner, M., Taylor, J., Young, E. *Strategies and Methods for Research on Sex Differences in Brain and Behavior.*, *Endocrinology*, 2005, 146:1650-1673

Jackson, L. R. Robinson, T. E. and Becker, J. B. Sex differences and hormonal influences on acquisition of cocaine self-administration in rats. *Neuropsychopharmacology*, 2005, (available online).

**Jacinta Catherine Beehner:** My research has centered on the physiology that underlies behavioral stress, aggression, social status, and mate choice in non-human primates. The short-term objectives of my research are to understand the causal connections between social conditions and individual variation in physiology. My long-term research goals are to identify some of the cognitive aspects of hormonal control for non-human primates. For example, what is the role of psychological vs. physical stressors in the lives of these primates? What sorts of cues do individuals use to size up a rival or choose a mate? What role do hormones play in these decisions? Most of my research has been conducted on wild baboons in Ethiopia and Botswana. A field-based approach allows me to observe the interactions of hormones and behavior within the selective environment under which the physiological responses evolved. I combine non-invasive methods of behavioral data collection with fecal hormone extraction from habituated, known individuals. Currently, I am investigating these questions on a group of gelada baboons (*Theropithecus gelada*) living in the Simiens Mountains National Park of Ethiopia. Gelada baboons are unique among cercopithecine primates because they live in extremely large social groups and have a diverse array of vocal and visual signals that may be mediated by steroid hormones.

#### **Representative Publications:**

Beehner JC, Bergman TJ, Cheney DL, Seyfarth RM, Whitten PL (2005a) The effect of new alpha males on female stress in free-ranging baboons. *Anim Behav* 69:1211-1221

Beehner JC, Bergman TJ, Cheney DL, Seyfarth RM, Whitten PL (in press) Testosterone predicts future dominance rank and mating activity among male chacma baboons. *Behav Ecol Sociobiol*

Beehner JC, Phillips-Conroy JE, Whitten PL (in press) Female testosterone, dominance rank, and aggression in an Ethiopian population of hybrid baboons. *Am J Primatol*

Beehner JC, Whitten PL (2004) Modifications of a field method for fecal steroid analysis in baboons. *Physiol Behav* 82:269-277

**Dr. Thore J. Bergman:** My research focuses on social behavior and social cognition from an evolutionary perspective. I am particularly interested in the adaptive nature of interactions (e.g., male-female bonds, coalition formation, mate choice, and sexual signaling) in complex social systems. What cognitive abilities underlie these

behaviors and is there a causal connection between sociality and cognitive capacity? What can the evolution of cognition in complex societies teach us about our own cognitive abilities? What are the selective pressures on these behaviors? What is the genetic basis of these behaviors and how flexible are they? Previously, I have studied baboons in Ethiopia and Botswana, augmenting behavioral observation with a variety of techniques (e.g. playback experiments, genotyping for paternity testing, and non-invasive hormone collection) that allow me to measure the cognitive correlates, fitness consequences, and physiological determinants of various behaviors. I am currently using these techniques to investigate social behavior in the gelada baboons of Ethiopia. Gelada live in extremely large, multi-level societies. I will be focusing on three elements of social behavior that may have evolved in relation to gelada's complex social system: social cognition, communication, and sexual signaling.

#### **Representative Publications:**

Bergman TJ, Beehner JC, Cheney DL, Seyfarth RM, Whitten PL (in press) Interactions in male baboons: the importance of both males' testosterone. *Behavioral Ecology and Sociobiology*.

Seyfarth RM, Cheney DL, Bergman TJ (2005) Primate social cognition and the origins of language. *Trends in Cognitive Sciences*, 9:264-266.

Bergman TJ, Beehner JC (2004) The social system of a hybrid baboon group (*Papio hamadryas anubis* x *P. h. hamadryas*). *International Journal of Primatology*, 25:1313-1330

Bergman TJ, Beehner JC, Cheney DL, Seyfarth RM (2003) Hierarchical classification by rank and kinship in baboons. *Science*, 302:1234-1236.

Bergman TJ & Beehner JC (2003) Hybrid zones and sexual selection: Insights from the Awash baboon hybrid zone (*Papio hamadryas anubis* x *P.h. hamadryas*). In CB Jones (ed.): *Sexual Selection and Reproductive Competition in Primates: New Perspectives and Directions*. American Society of Primatologists, Norman, OK, pp. 502-537.

**Joshua Berke:** My primary interests concern the role of basal ganglia circuits in the learning, selection and performance of actions, and how such neural mechanisms are altered in psychiatric and neurological disorders such as drug addiction and Parkinson's Disease. Current studies use chronic electrophysiological recording in

awake, freely-moving rats and transgenic mice to examine how subpopulations of striatal neurons encode information and interact with one another, and how these neural representations are changed by learning experiences and by dopaminergic manipulations. Some of the interrelated, long-term questions I address are:

How do neural circuits involving the basal ganglia mediate action selection and implicit learning?

By what mechanisms do neuromodulators such as dopamine affect these circuits to produce both acute and long-term changes in behavior?

How do alterations in the dynamic properties of basal ganglia circuits produce the key symptoms of human behavioral disorders such as Parkinson's Disease?

What differences in neural representations and dynamics distinguish deliberate from automatic actions? How does the prefrontal cortex suppress inappropriate habits to provide behavioral flexibility?

To what extent can we think of certain compulsive behaviors as disorders of learning/memory, arising from altered synaptic plasticity?

How do learning mechanisms in the basal ganglia differ from those in hippocampus? How do multiple memory systems interact during different types of associative learning?

#### **Representative Publications:**

Wiltchko AB, Pettibone JR, Berke JD. (2010) Opposite effects of stimulant and antipsychotic drugs on striatal fast-spiking interneurons. *Neuropsychopharmacology*. 2010 Jan 20.

Berke JD (2009) Fast oscillations in cortical-striatal networks switch frequency following rewarding events and stimulant drugs. *European Journal of Neuroscience*, Vol. 30: 848-859.

Berke JD, Breck JT, Eichenbaum H. (2009) Striatal versus hippocampal representations during win-stay maze performance. *J Neurophysiol*. 101:1575-87.

Berke JD, Okatan M, Skurski JA, Eichenbaum HB. (2004) Oscillatory entrainment of striatal neurons in freely-moving rats. *Neuron* 43: 883-896.

Berke JD (2003). Learning and memory mechanisms involved in compulsive drug use and relapse. In: Wang, J (ed.) *Drugs of abuse: analysis of neurological effects*. Humana Press, Totowa, NJ.

**Kent C. Berridge:** My research seeks answers to fundamental questions, such as:

How is pleasure generated in the brain?

What are the neural bases of wanting and liking?

How are rewards learned?

How do brain motivation systems work?

What causes addiction?

How does the brain distinguish pleasant from unpleasant?

How does fear relate to desire?

Can an emotion ever be truly unconscious?

How is real behavior produced by brains?

### **Representative Publications**

Tindell, A.J., Smith, K.S., Berridge, K.C. & Aldridge, J.W. Dynamic computation of incentive salience: 'wanting' what was never 'liked'. *Journal of Neuroscience*, 29(39), 12220-12228 (2009).

Zhang J, Berridge KC, Tindell AJ, Smith KS, Aldridge JW. A Neural Computational Model of Incentive Salience. *PLoS Computational Biology* 5(7): (2009). PMID: 2703828

Mahler, S.V. & Berridge, K.C. Which cue to 'want'? Central amygdala opioid activation enhances and focuses incentive salience on a prepotent reward cue. *Journal of Neuroscience*, 29(20), 6500-6513, (2009).

Berridge, K.C. 'Liking' and 'wanting' food rewards: brain substrates and roles in eating disorders. *Physiology & Behavior*, 97(5):537-50 (2009). PMID: 2717031

Berridge, K.C., Robinson, T.E. & Aldridge, J.W. Dissecting components of reward: 'liking', 'wanting', and learning. *Current Opinion in Pharmacology*, 9, 65-73 (2009). PMID: 2756052

Smith, K. S., Tindell, A.J., Aldridge, J.W., & Berridge, K.C. Ventral pallidum roles in reward and motivation. *Behavioral Brain Research*, 196, 155-167 (2009). PMID: 2606924

Faure, A. Reynolds, S.M., Richard, J.M. & Berridge, K.C. Mesolimbic dopamine in desire and dread: Enabling motivation to be generated by localized glutamate disruptions in nucleus accumbens. *Journal of Neuroscience*, 28, 7184-7192 (2008).

Reynolds, S.M. and Berridge, K.C. Emotional environments retune the valence of appetitive versus fearful functions in nucleus accumbens. *Nature Neuroscience*, 11(4), 423-425 (2008).

Berridge, K.C. & Kringelbach, M.L. Affective neuroscience of pleasure: reward in humans and animals. *Psychopharmacology*, 199, 457-480 (2008).

Robinson, T.E. & Berridge, K.C. The incentive sensitization theory of addiction: some current issues. *Philosophical Transactions of the Royal Society B*, 363, 3137-3146 (2008).

**Henry Buchtel:** Studies of brain and behavior in normal and brain-damaged human subjects: (1) hemispheric specialization in perception and memory (especially for faces and other difficult-to-verbalize stimuli); (2) language abilities before and after epilepsy surgery; and (3) imaging of brain changes in the context of radiation treatment for brain tumors. Testing facilities are located in the University Hospital and in the Neuropsychology Section Clinic.

### **Representative Publications:**

Buchtel, H.A. (2001) Left and right hemisphere contributions to physiognomic and verbal discrimination. *Neuropsychology*, 15:597-606.

Buchtel, H.A. & Selwa, L.M. (2009) Neuropsychological Aspects of Epilepsy. In I. Grant and K.M. Adams (Eds.) *Neuropsychological Assessment of Neuropsychiatric Disorders*. 3rd edition. Oxford University Press

Berlucchi, G and Buchtel, H.A. (2009) Neuronal Plasticity: Historical Roots and Evolution of Meaning. *Experimental Brain Research*. 192:307-320.

Cao, Y., Tsien, C.I., Sundgren, P.C., Nagesh, V., Normolle, D., Buchtel, H., Junck, L. and Lawrence, T.S. (2009) Dynamic Contrast-Enhanced Magnetic Resonance Imaging As a Biomarker for Prediction of Radiation-Induced Neurocognitive Dysfunction. *Clinical Cancer Research*, 15: 1747-1754.

**Geoffrey E. Gerstner:** My research has two current foci: (1) The neuroethology of the masticatory rhythm. Chewing rate among mammals is highly invariant, species-specific and individual specific. Through a combination of wild-mammal, dog-breed and lab-rodent studies, we have shown that the allometric scaling of chewing rhythm with body mass is almost exclusively a product of natural selection and has little to do with physiological or biochemical mechanisms operating in developmental time scales. Through a combination of field, motion analysis, neural ensemble recording and EMG methods, I am studying the sources of variation in the chewing rhythm within and between species. Chewing rhythm studies provide ideal means of tackling Tinbergen's four questions of animal behavior, and studies have relevance to neurobiology, ecology and

evolutionary thought. (2) Neural and psychophysical bases of chronic pain. I am collaborating with the Chronic Pain and Fatigue Research Center to study the central mechanisms associated with chronic pain. We use a pressure-pain testing paradigm in conjunction with psychometric tests and fMRI, fcMRI and H-SPECT neuroimaging methods to investigate the roles of central systems in perpetuating pain sensitivity and pain behaviors in humans suffering from chronic temporomandibular disorder pain. We are also studying the sensitivity in non-nociceptive sensory systems, e.g., auditory, in chronic pain patients, and whether there is a genetic bases for chronic pain susceptibility.

### **Representative Publications:**

Gerstner, G.E. and L.J. Goldberg. An analysis of mandibular movement trajectories and masticatory muscle EMG activity during drinking in the guinea pig. *Brain Res.* 479:6-15, 1989.

Gerstner, G.E. and L.J. Goldberg. Species-specific morphology of masticatory jaw movements. *Behaviour* 128:229-253, 1994.

Carvalho, T.C. and Gerstner, G.E. Licking rate adaptations to increased mandibular weight in the adult rat. *Physiol. Behav.* 82(2-3):331-337, 2004.

Dang, R., T. Carvalho and G.E. Gerstner. The effects of mandibular loading on rat craniofacial morphology: A new system for gravity studies. *Acta Astronautica* 56(3):357-366, 2004.

Gerstner, G.E. Chewing rate allometry among mammalian species. *J. Mammal.* 89, 2008 (in press)

Gerstner, G.E. and L.J. Goldberg. The process of mastication. In: (Y. Nakamura, ed.) *Neurobiology of mastication: from molecular to systems approach.* pp. 3-21, 1999.

Gerstner, G.E. Neuroethology. In: (M. Bekoff, ed.) *Encyclo. of Animal Behavior.* Greenwood Press: Westport, CT. pp. 806-809, 2004.

Gerstner, G.E. Discriminant analysis of mastication in temporomandibular disorder and control subjects. *IADR*, 1995.

Springstead, C., G. E. Gerstner, D. Clauw, R. Gracely. A psychophysiological model of endocrine response to chronic facial pain. Undergraduate Honors' Psychology Symposium, 2006.

Springstead, C., G. E. Gerstner, D. Clauw, R. Gracely. Neuroendocrine response to pain suggests differing etiology of temporomandibular disorder and other

chronic pain disorders. *Dental Research Symposium*, 2007.

**Theresa M. Lee:** My research examines the neural and behavioral features of circadian rhythms in the day-active *Octodon degus*, an animal model with circadian properties similar to humans. We are using training on a sustained attention task with rats during the rest phase to create a animal model of shift-work. This model is being used to assess the behavioral and neural changes that allow reorganization of the circadian rest/activity, as well as other rhythms. Currently, we are also interested in the role of steroid hormones in the development of circadian rhythms which results in phase delayed sleep/activity during adolescence. A variety of other studies examine the interaction of stressful environmental variables during development on adult circadian rhythms.

In addition to these circadian interests, the lab is involved in a large project examining the lifespan effects of prenatal steroid exposure on development of sex-specific behaviors. The sheep is used for behavioral and neuroanatomical analysis.

### **Representative Publications:**

Gritton, H.J., B.C. Sutton, V. Martinez, M. Sarter & T.M. Lee. Interactions between cognition and circadian rhythms: attentional demands modify circadian entrainment. *Behavioral Neuroscience*, 2009, 123:937-948.

Roberts, E.K., J.N. Flak, W. Ye, V. Padmanabhan, T.M. Lee. Juvenile rank can predict male-typical adult mating behavior in prenatally-androgenized female sheep. *Biol Reprod*, 2009, 80:737-742.

Hagenauer, M.H., J.I. Perryman, T.M. Lee, M.A. Carskadon. Adolescent changes in the homeostatic and circadian regulation of sleep. *Developmental Neurosci (Special Issue)*, 2009, Invited submission, 31:276-284.

Vosko, A.M., M.H. Hagenauer, D.L. Hummer & T.M. Lee. Period gene expression in the diurnal degu (*Octodon degus*) differs from the nocturnal laboratory rat (*Rattus norvegicus*). *Amer J Physiol*, 2009, 296:R353-R361.

Hummer, D.L., T.J. Jechura, M.M. Mahoney & T.M. Lee. Gonadal hormone effects on entrained and free-running circadian activity rhythms in the developing diurnal rodent, *Octodon degus*. *Am J Physiol*, 2007, 292:R586-R597.

**Stephen Maren:** The research in my laboratory is geared towards understanding the neurobiology of learning and memory, particularly memory for emotional events. From a clinical perspective, emotional memories are at the heart of a number of anxiety disorders in humans including post-traumatic stress disorder, panic disorder, and simple phobias. As a model system for studying emotional memory, we use Pavlovian fear conditioning in rats. Pavlovian fear conditioning is a simple form of associative learning that is rapidly induced and extremely long-lasting. Recent studies indicate that the hippocampus and amygdala play an essential role in fear conditioning, however the neurobiological mechanisms involved in encoding and storing fear memories in these structures is poorly understood. In addition, considerable interest has emerged in how fear memories can be either suppressed or erased after they have been learned. In this regard, we have become particularly interested in the neural mechanisms of extinction, and the role of context and time in regulating the retrieval of fear and extinction memories.

To study these mechanisms, we use brain lesions, immunohistochemistry, intracranial drug infusions, and single-unit electrophysiology in combination with sophisticated behavioral designs. These studies are directed at providing a more complete understanding of the neural circuitry underlying both fear conditioning and extinction and elucidating the nature of information processing within these circuits during the encoding and retrieval of emotional memories.

#### **Representative Publications:**

Maren, S. and Chang, C. H. (2006). Recent fear is resistant to extinction. *Proceedings of the National Academy of Sciences U.S.A.*, 103:18020-18025.

Maren, S. and Hobin, J. A. (2007). Hippocampal regulation of context-dependent neuronal activity in the lateral amygdala. *Learning & Memory*, 14:318-324.

Maren, S. (2007). The threatened brain. *Science*, 317:1043-1044.

Rabinak, C. A. and Maren, S. (2008). Associative structure of fear memory after basolateral amygdala lesions in rats. *Behavioral Neuroscience*, 122:1284-94.

Knapska, E. and Maren, S. (2009). Reciprocal patterns of c-fos expression in the medial prefrontal cortex and amygdala after extinction and renewal of conditioned fear. *Learning & Memory*, 16:486-493.

**Randy M. Nesse:** My core work is on the evolutionary origins and functions of emotions involved in psychopathology, in particular, how natural selection shaped the capacity for mood and how the regulation of normal high and low mood is related to clinical depression. This work now is based on new methods for eliciting comprehensive information on the incentive structures of individual lives with special emphasis on determining if a person is pursuing unreachable goals that cannot be given up. Related work investigates virtual foraging as a model for depression and ADHD. I am also conducting several studies that look at proximate mechanisms including whole blood serotonin and its relationship to status and mood, salivary cortisol and testosterone in community samples, and a large behavioral genetic study of personality characteristics as related to candidate genes and as they map onto a genome scan. Two additional projects involve determining the importance of commitment in relationships, and determining what factors account for how the sexual mortality ratio varies with time and culture.

#### **Representative Publications:**

Nesse RM, Stearns SC (2008) The great opportunity: Evolutionary applications to medicine and public health. *Evolutionary Applications* 1(1):28-48, 2008.

Nesse RM: Runaway Social Selection for Displays of Partner Value and Altruism, *Biological Theory* 2 (2): 1-13, 2007.

Nesse RM, Jackson ED (2006) : Evolution: Psychiatric nosology's missing biological foundation. *Clinical Neuropsychiatry* 3 (2):121-131.

Keller MC, Nesse RM(2006). The Evolutionary Significance of Depressive Symptoms: Different Adverse Situations Lead to Different Depressive Symptoms Patterns. *Journal of Personality and Social Psychology*, 91(2):316-30.

Nesse, RM: Natural selection and the regulation of defensive responses (2005) *Evolution and Human Behavior*, 26:88-105.

Nesse RM (2000) Is depression an adaptation? *Archives of General Psychiatry*, 57: 14-20.

Nesse RM (1999) Proximate and evolutionary studies of stress and depression: Synergy at the Interface. *Neuroscience and Biobehavioral Reviews* 23: 895-903.

**Bryan E. Pfingst:** My main research interest is in the perception and processing of auditory information by the ear and the brain. The focus of my current research is on psychophysical studies of hearing with electrical stimulation of the inner ear and central auditory nuclei. This work is done in operantly conditioned animals which are implanted with electrode arrays that can stimulate groups of auditory neurons to produce sensations of sound. Comparative studies are conducted in deaf or hearing-impaired human subjects implanted with auditory prostheses. Through measurements of the detection, loudness, and discrimination of patterned stimuli, including speech stimuli, we can better understand how the organisms process sensory information. This information is directly applicable to the design of prosthetic-hearing devices for profoundly deaf people.

### **Representative Publications:**

Su GL, Colesa DJ, Pfingst BE (2008) Effects of deafening and cochlear implantation procedures on postimplantation psychophysical electrical detection thresholds. *Hear Res.* [Epub ahead of print] doi: 10.1016/j.heares.2008.04.011.

Pfingst BE, Burkholder-Juhasz RA, Xu, L, Thompson CS (2008) Across-site patterns of modulation detection in listeners with cochlear implants. *J Acoust Soc Am* 123:1054-1062.

Pfingst BE, Burkholder-Juhasz RA, Zwolan TA, Xu (2007) Psychophysical assessment of stimulation sites in auditory prosthesis electrode arrays. *Hear Res* [Epub ahead of print] doi:10.1016/j.heares.2007.11.007.

Pfingst BE, Xu L, Thompson CS (2007) Effects of carrier pulse rate and stimulation site on modulation detection by subjects with cochlear implants. *J Acoust Soc Am* 121:2236-2246.

**Terry E. Robinson:** My research focuses primarily on the nature of long-term neuroplastic adaptations produced by repeated exposure to psychostimulant drugs and stress, and the role these neuroadaptations play in the development of psychopathology, especially addiction. Much of this research has focused on the phenomenon of psychomotor sensitization associated with the repeated intermittent administration of amphetamine and cocaine, or the effects of different patterns of drug self-administration behavior. These sensitization-related neuroadaptations are thought to be important in the development of compulsive behavioral disorders, such as addiction. We have shown that behavioral sensitization is accompanied by enduring alterations in a number of

neurobiological systems, especially mesotelencephalic dopamine systems, and they include an enhancement in the ability of psychostimulants to elevate extracellular dopamine concentrations in the nucleus accumbens and by structural changes in the morphology of dendrites in brain reward systems. A recent focus concerns the ability of environmental (nonpharmacological) factors to modulate the development and expression of sensitization and the neurobiological mechanisms by which environmental context gates drug responsiveness, including the ability to modulate gene expression. In addition, we are also interested in individual variation in the extent to which cues associated with rewards acquire incentive motivational properties, and the extent to which individual vary in their ability to resist such cues.

(See <http://sitemaker.umich.edu/terryrobinson> for more information).

### **Representative Publications:**

Ferrario, C.R., Gorny, G., Crombag, H.S., Li, Y., Kolb, B. and Robinson, T.E. Neural and behavioral plasticity associated with the transition from controlled to escalated cocaine use. *Biological Psychiatry*, 2005, 58, 751-759.

Jedynak, J.P., Uslaner, J.M., Esteban, J.A. and Robinson, T.E. Methamphetamine-induced structural plasticity in the dorsal striatum. *European Journal of Neuroscience*, 2007, 25, 847-853.

Robinson, T.E. and Berridge, K.C. The incentive-sensitization theory of addiction: some current issues. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 2008, 363, 3137-3146.

Briand, L.A., Flagel, S.B., Watson, S.M., Akil, H., Sarter, M. and Robinson, T.E. Persistent alterations in cognitive function and dopamine-related gene expression following extended (but not limited) access to self-administered cocaine. *Neuropsychopharmacology*, 2008, 33, 2969-2980.

Robinson, T.E. and Flagel, S.B. Dissociating the predictive and incentive motivational properties of reward-related cues through the study of individual differences. *Biological Psychiatry*, 2009, 65, 869-873.

Saunders, B.T. and Robinson, T.E. A cocaine cue acts as an incentive stimulus in some, but not others: implications for addiction. *Biological Psychiatry*, 2009, published online doi:10.1016/j.biopsych.2009.11.015.

**Martin Sarter:** My research is characterized by a systems neuroscience approach to the determination of the neuronal mechanisms mediating cognitive functions. Specifically, my research focuses on the regulation and function of the cortical cholinergic input system. Cholinergic neurons innervate all cortical areas and layers and thus modulate all cortical information processing. This ascending system is a component of the ‘anterior attention system’ that acts to optimize input processing. Abnormal regulation of the activity of cortical cholinergic inputs, and/or a decrease in the integrity of this neuronal system, mediate the manifestation of the cognitive symptoms of major neuropsychiatric disorders. Current experiments focus on the role of fast cholinergic transients, measured by using enzyme-selective microelectrodes, in mediating defined components of the cognitive operations underlying attentional performance, the regulation of glutamatergic and cholinergic signals by nicotinic acetylcholine receptors, the function of choline transporters and choline transporter trafficking, and the development of cognition enhancers for schizophrenia.

#### **Representative Publications:**

Sarter, M., Parikh, V., & Howe, W.M. (2009). Phasic acetylcholine release and the volume transmission hypothesis: time to move on. *Nature Reviews Neuroscience*, 10, 383-390.

Parikh, V., Kozak, R., Martinez, V., & Sarter, M. (2007). Prefrontal acetylcholine controls cue detection on multiple time scales. *Neuron*, 56, 141-154.

Parikh, V., Man, K., Decker, M.W., & Sarter, M. (2008). Glutamatergic contributions to nAChR agonist-evoked cholinergic transients in the prefrontal cortex. *Journal of Neuroscience*, 28, 3769-3780.

Parikh, V., Ji J., Decker, M.W., & Sarter, M. (2010). Prefrontal  $\beta 2$  subunit-containing and  $\alpha 7$  nAChRs differentially control glutamatergic and cholinergic signaling. *Journal of Neuroscience*, 30, 3518-3530.

**Barbara B. Smuts:** Primary interests: I study am interested in how nonhuman animals develop, maintain, and negotiate social relationships. I’ve studied social relationships in nonhuman primates, bottlenose dolphins, and, more recently, domestic dogs. Topics of interest include play, social reciprocity, cooperation, greetings, conflict resolution, and social cognition. Research in dogs examines the dynamics of social relationships by analyzing video-taped interactions in fine detail, using frame-by-frame and slow motion analysis. Questions

being addressed include: How do other animals develop trusting relationships in the absence of spoken language? What do animals understand about the beliefs and intentions of their social partners? How can understanding of nonhuman social relationships help us to better understand human behavior?

#### **Representative Publications:**

2008 Ward, C. & Smuts, B.B. Play partner preferences within litters of domestic dogs. *Animal Behaviour*, (in press)

2008, Smuts, B.B. Embodied communication in nonhuman animals,. In: *Human Development in the 21<sup>st</sup> Century: Visionary Policy Ideas from Systems Scientists*, Alan Fogel, Barbara King, and Stuart Shanker, eds. (publication of the *Council on Human Development*, Oxford: Oxford University Press)

2007, Bauer, E.B. & Smuts, B.B. Cooperation and competition during dyadic play in domestic dogs, *Canis familiaris*, *Animal Behaviour* 73: 489-499

2007 Ward, C. & Smuts, B.B. Quantity-based judgments in the domestic dogs (*Canis lupus familiaris*) *Animal Cognition* 10: 71-80

2001 Smuts, B.B. Encounters with animal minds. *Journal of Consciousness Studies* 8(5-7): 293-309.

1999 Smuts, B.B. *Sex and Friendship in Baboons*, second edition. Cambridge, MA: Harvard University Press.

Smuts, B., Cheney D.L., Seyfarth, R.M., Wrangham, R.W. and Struhsaker, T.T. (Eds.) (1987). *Primate Societies*. Chicago: The University of Chicago Press.

**Sari van Anders:** My research program centers on human social neuroendocrinology, sexuality, gender/sex, and evolution. I am particularly interested in social modulation of testosterone and other hormones via behavioral contexts related to partnering, sexuality, and nurturance. Here, the majority of my research focuses on the evolved physiology of pair bonding. In addition, I focus on bidirectional associations, and particularly how sexuality and testosterone are associated. I have been developing a theoretical framework – testosterone trade-offs – which is a supra-gender/sex framework positing trade-offs between high testosterone (and competitive behavioral contexts) and low testosterone (and bond-maintenance behavioral contexts). I am also interested androgen-immune trade-offs and social modulation of immune function. My program includes attention to

diverse populations, as well as the development of methods to conduct my research using inclusive research and feminist science practices. I have also become increasingly interested in biological rhythms (especially seasonality), both as methodological issues and evolutionary questions. My research is with humans; I currently employ salivary radioimmunoassays for endocrine and immune measures and fMRI to study correlations in endocrine and neural activity, and am looking to employ genetic techniques to look at hormone receptors. I maintain an active interest in broader research related to human sexuality, conceptualizations of gender/sex, and interpretations and implications of this research.

### **Representative Publications:**

van Anders, S. M. (in press). Gonadal steroids and salivary IgA in healthy young women and men. American Journal of Human Biology.

van Anders, S. M. & Dunn, E. J. (2009). Are gonadal steroids linked with orgasm perceptions and sexual assertiveness in women and men? Hormones and Behavior, 56, 206-213.

van Anders, S. M. & Gray, P. B. (2007). Hormones and human partnering. Annual Review of Sex Research, 18, 60-93. Invited contribution.

van Anders, S. M., Hamilton, L. D., & Watson, N. V. (2007). Multiple partners are associated with higher testosterone in North American men and women. Hormones and Behavior, 51, 454-459.

van Anders S. M., Hamilton, L. D., Schmidt, N., & Watson, N. V. (2007). Associations between testosterone secretion and sexual activity in women. Hormones and Behavior, 51, 477-482.

van Anders, S. M. & Watson, N. V. (2006). Relationship status and testosterone in North American heterosexual and non-heterosexual men and women: Cross-sectional and longitudinal data. Psychoneuroendocrinology, 31, 715-723.

van Anders, S. M. & Watson, N. V. (2006). Social neuroendocrinology: Effects of social contexts and behaviours on sex steroids in humans. Human Nature, 17(2), 212-237.

**James H. Woods:** My research interests are the behavioral pharmacology of drugs. For example, the manner in which activation of drug receptors is translated in behavioral change is studied. I am also interested in life-span primate models of drug abuse and the pharmacotherapy of drug abuse.

### **Representative Publications:**

Fantegrossi WE, Godlewski T, Karabenick RL, et al. (2003) Pharmacological characterization of the effects of 3,4-methylenedioxymethamphetamine ("ecstasy") and its enantiomers on lethality, core temperature, and locomotor activity in singly housed and crowded mice Psychopharmacology 166: 202-211.

Flory GS, Woods JH (2003) The ascending limb of the cocaine dose-response curve for reinforcing effect in rhesus monkeys. Psychopharmacology 166: 91-94.

Houshyar H, Galigniana MD, Pratt WB, Woods, JH. (2001) Differential responsivity of the hypothalamic-pituitary-adrenal axis to glucocorticoid negative-feedback and corticotropin releasing hormone in rats undergoing morphine withdrawal: Possible mechanisms involved in facilitated and attenuated stress responses. J Neuroendocrinol 13: 875-886.

Woods JH, Winger GD (2002) Observing responses maintained by stimuli associated with cocaine or remifentanyl reinforcement in rhesus monkeys. Psychopharmacology 163: 345-351.

Houshyar H, Cooper ZD, Woods JH (2001) Paradoxical effects of chronic morphine treatment on the temperature and pituitary-adrenal responses to acute restraint stress: A chronic stress paradigm J Neuroendocrinol 13: 862-874.

Williams KL, Kane EC, Woods JH (2001) Interaction of morphine and naltrexone on oral ethanol self-administration in rhesus monkeys. Behav Pharmacol 12: 325-333.

### **Emeritus Faculty in Residence:**

**Dr. Charles Butter**

**Dr. Elliot S. Valenstein**