

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: behavioral neuroscience, sensory processes, evolutionary basis and the adaptive significance of behavior, motivation and emotion, hormones and behavior, biological rhythms, cognitive neuroscience, stress neurobiology, psychopharmacology, neural plasticity/recovery of function, and neuropsychology.

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 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 semester.

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 568 (Primate Behavioral Ecology and Sociobiology). 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 exclusively 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 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 are 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. 8. 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.

Precandidates must enroll for at least 9 credit hours, and may enroll for up to 18 credits for 4 semesters. During the term that a student is waiting for Candidacy approval, he or she should register for 8 credits of Psych 990. When Candidacy is approved, the Registrar’s Office will change all 990 enrollments to 995 for the student. Candidates must enroll for 8 credit hours of Psychology 995 (dissertation research, and may enroll for one additional course. Students must enroll for a total of 8 semesters including the semester in which they defend their dissertation.

At least two members of the dissertation committee must be core Biopsychology faculty. 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. A departmental application is required in addition to the graduate school application by December 15. Applications may be obtained from:

Student Academic Affairs Office
Department of Psychology
The University of Michigan
530 Church Street
Ann Arbor, MI 48109
Telephone: (734) 764-2480
psych.saa@umich.edu

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

The application deadline is December 5. All applicants will be notified of admission decisions by April 15, and usually before March 1.

Financial Support

The department offers a financial package that funds the five years that a student is in the graduate program. This support package combines Research Assistant positions and Graduate Student Instructor (GSI) positions for a total of 5 years of support with health insurance. As a Research Assistant, students engage in their own research and research in collaboration with their faculty mentor in Psychology. The financial offer is equivalent for all students that are admitted into any of the seven areas of psychology with a few differences for the students in Clinical 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 2) years 3-5 when a student is conducting their dissertation research and strengthening

other skills. Similarly, the support package is in two phases. During the first one and a half years in the graduate program, a student is supported as a Research Assistant for three academic terms, as well as over the first summer, and they will be supported as a GSI for one term. Support for subsequent years in the program is contingent on having achieved candidacy.

During years 3-5, students begin their dissertation research and have the opportunity to expand their teaching experiences with five additional terms of GSI support. Mentors may be able to provide financial support during the summer months, and a student should discuss this with their mentor. The final semester students will be supported on a Psychology or Rackham 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 asked 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, NIMH, etc.) the department can combine funds in a way that will enhance the overall support package.

An undergraduate who is seriously contemplating study at the doctoral level is well advised to explore the many sources of individual fellowship support available to outstanding students. No later than the end of their junior year, students should consult counselors at their undergraduate colleges or universities regarding fellowships available from both federal and private sources. Students who receive an individual fellowship may use it at any university to which they have been admitted. 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 (NSF Graduate Research Fellowship Program, Oak Ridge Associated Universities, P.O. Box 3010, Oak Ridge, Tennessee 37831-3010) Phone:(423) 241-4300, Fax:(423) 241-4513, from the NSF web page: <http://www.fastlane.nsf.gov> or from the Department of Psychology Student Academic Affairs Office. Students who have entered the Biopsychology program are encouraged also to apply for NIH or NIMH predoctoral fellowships.

Some students have been supported on fellowships or Training Grants, and other graduate students have served as Research Assistants, receiving support from grants held by staff members.

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. If you would like to know more about the work of a particular staff member, feel free to write directly to that person. Copies of the articles listed are available from each individual, although you should be able to find most of them in your library.

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:

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.

Berke JD, Sgambato V, Lavoie B, Krause M, Hyman SE (2001) Dopamine and glutamate induce distinct striatal splice forms of Ania-6, an RNA polymerase II-associated cyclin. *Neuron* 32: 277-287.

Berke JD, Hyman SE (2000) Addiction, dopamine, and the molecular mechanisms of memory. *Neuron* 25: 515-532.

Kent C. Berridge: My research in affective neuroscience focuses on brain mechanisms of “liking” and “wanting” for sensory pleasures and related rewards. Our work aims to clarify mechanisms of natural motivation and of drug addiction. Most of our experimental work is on food reward and appetite, but also has implications for drugs and other rewards. Topics include the roles of specific brain structures and neurochemical systems in mediating specific affective building blocks such as 'wanting' and 'liking', and in differentiating positive reward emotions from negative emotions such as fear. In collaboration with Dr. Terry Robinson, some of this work has resulted in the 'incentive-sensitization' hypothesis of drug addiction. In collaboration with Dr. J. Wayne Aldridge, it has involved examination of how neuronal firing codes reward 'liking' and 'wanting' in limbic brain structures. Finally, in a separate line of research we also study the biopsychology of action syntax, or how brain systems generate and coordinate complex patterns of real action. Our focus in that action syntax line of research is on how sequences controlled, using multiple neural techniques applied to instinctive patterns of behavior.

In summary, our research seeks to understand 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 does the brain translate motivation/emotion into action?
- How is real behavior produced by brains?

Representative Publications

Smith, K.S. & Berridge, K.C. The ventral pallidum and hedonic reward: neurochemical maps of sucrose 'liking' and food intake. *Journal of Neuroscience*, 25, 2005.

Tindell, A.J., Berridge, K.C., Zhang, J., Peciña, S., and Aldridge, J.W. Ventral pallidal neurons code incentive motivation: Amplification by mesolimbic sensitization and amphetamine. *European Journal of Neuroscience*, 23, 2005.

Berridge, K.C., Aldridge, J.W., Houchard, K.R., Zhuang, X. (2005). Sequential super-stereotypy of an instinctive fixed action pattern in hyper-dopaminergic mutant mice: a model of obsessive compulsive disorder and Tourette's. *BMC Biology* 2005, 3:2.

Berridge, K.C. Motivation concepts in behavioral neuroscience. *Physiology & Behavior* 81(2), 179-209, 2004.

Tindell, A.J., Berridge, K.C., Aldridge, J.W. Ventral Pallidal Representation of Pavlovian Cues and Reward: Population and Rate Codes. *Journal of Neuroscience*, 24 (2), 2004.

Peciña S., Cagniard, B., Berridge, K.C., Aldridge, J.W. & Zhuang, X. Hyperdopaminergic mutant mice have higher 'wanting' but not 'liking' for sweet rewards. *Journal of Neuroscience* 23 (28), 9395-9402, 2003.

Berridge, K.C. & Robinson, T.E. Parsing reward. *Trends in Neurosciences*, 26 (9), 507-513, 2003

Berridge, K.C. Pleasures of the brain. *Brain & Cognition*, 52 (10), 106-128, 2003.

Robinson, T.E. & Berridge, K.C. Addiction. *Annual Review of Psychology*, v. 54, 25-53, 2003.

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 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:

Hagenauer, M.H. & T.M. Lee. Circadian organization of the diurnal Caviomorph rodent, *Octodon degus*. *Biol Rhythm Res*, 2008, 39:269-289.

Roberts, E.K., V. Padmanabhan & T.M. Lee. Differential effects of prenatal testosterone timing and duration on phenotypic and behavioral masculinization and defeminization of female sheep. *Biol Reprod*, 2008, Pre-publication online.

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.

Mohawk, JA, K. Cashen, TM Lee. Inhibiting cortisol response accelerates recovery from a photic phase shift. *Amer J Physiol*, 2005, 288:R221-R228.

Mohawk, J.A. & T.M. Lee. Restraint stress delays reentrainment in male and female diurnal and nocturnal rodents. *J Biol Rhythms*, 2005, 20:245-256.

Lee, T.M. *Octodon degus*: A diurnal, social and long-lived rodent. *ILAR*, 2004, 45:14-24.

Smale, L, TM Lee, AA Nunez. Mammalian diurnality: Facts and Gaps. *J Biol Rhythms*, 2003 18:356-366.

Stephen Maren: The research in my laboratory is geared towards understanding the neurobiological basis of emotional learning and memory. From a clinical perspective, emotional learning and memory 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 learning and 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. As an index of fear, we measure freezing behavior (immobility except for movement associated with breathing), a defensive response that rats have evolved to deter predation. 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.

To study these mechanisms, permanent or reversible brain lesions, intracranial drug infusion, and single-unit electrophysiology. These studies are directed at providing a more complete understanding of the neural circuitry underlying fear conditioning and elucidating the nature of information processing within these fear conditioning circuits.

Representative Publications:

Goosens, K. A. and Maren, S. (2001). Differential roles for the central, lateral, and basal amygdaloid in contextual and auditory fear conditioning in rats. *Learning & Memory*, 8:148-155.

Maren, S. (2001). Neurobiology of Pavlovian fear conditioning. *Annual Review of Neuroscience.*, 24:897-931.

Maren, S, Yap, S. A., and Goosens, K. A. (2001). The amygdala is essential for the development of neuronal plasticity in the medial geniculate nucleus during auditory fear conditioning in rats. *Journal of Neuroscience*, 21:RC135 (1-6).

Corcoran, K. A. and Maren, S. (2001). Hippocampal inactivation disrupts contextual retrieval of fear memory after extinction. *Journal of Neuroscience*, 21:1720-1726.

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) Cross-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 this latter line of research we hope to better understand how environmental and psychological factors interact with the neurobiological actions of drugs and stress to engender

the brain changes that result in psychopathology. (See <http://sitemaker.umich.edu/terryrobinson> for more information).

Representative Publications:

Robinson, T.E. and Berridge, R.C. Addiction. *Annual Review of Psychology*. 2003, 54, 25-53.

Samaha, A-N. and Robinson, T.E. Why does the rapid delivery of drugs to the brain promote addiction? *Trends in Pharmacological Sciences*, 2005, 26, 82-87.

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.

Uslaner, J.M., Acerbo, M.J., Jones, S.A. and Robinson, T.E. The attribution of incentive salience to a stimulus that signals an intravenous injection of cocaine. *Behavioural Brain Research*, 2006, 169, 320-324.

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.

Uslaner, J.M., Dell'Orco, J.M., Pevzner, A. and Robinson, T.E. The influence of subthalamic nucleus lesions on sign-tracking to stimuli paired with food and drug rewards: facilitation of incentive salience attribution?, *Neuropsychopharmacology*, advance online publication, December 5, 2007; doi:10.1038/sj.npp.1301653.

Briand, L.A., Fligel, 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, Feb 27; [Epub ahead of print] PMID: 18305460

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. (2005). Choline transporters, cholinergic transmission and cognition. *Nature Reviews Neuroscience*, 6, 48-56.

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.

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 21st*

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