Center for Neurobiology and Behavior

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Overview

The Center for Neurobiology and Behavior consists of 16 independent basic research laboratories in the Kolb Research Annex. The guiding research philosophy shared by the Center's faculty holds that an integrated approach, ranging from cellular and molecular biology to systems and behavioral analysis, is required to understand the basis of normal and abnormal human behavior. In this endeavor experimental approaches are complemented by a broad range of theoretical and computational techniques. The main foci of research in the Center are on basic science aspects of neural development and on the functions of the nervous system that underlie normal and abnormal behavior. The subjects investigated in these studies range from simple invertebrates to humans. Many of the studies carried out in the Center focus on processes such as learning and memory, attention, perception, and affective behavioral traits that can be affected by mental illness. Several ongoing projects may someday contribute to our understanding of the etiology of, and new therapeutic approaches to, addiction, anxiety, autism, benign-age-related memory loss, cerebral palsy, fragile-X syndrome, Rubinstein-Taybi Syndrome, schizophrenia, and spinal cord trauma.

The Center for Neurobiology and Behavior is also home to four more specialized academic entities:

Drs. Kandel and Siegelbaum are members of the **Howard Hughes Medical Institute**.

The **David Mahoney Center for Mind and Brain Research,** which is headed by Michael Goldberg, includes Drs. Abbott, Das, Ferrera, Gottlieb, Miller, Qian, and Salzman. The members in the Mahoney Center work in the area of cognitive and systems neuroscience.

The **Kävli Institute for Brain Science**, headed by Eric Kandel, includes Drs. Bailey, Siegelbaum, Goldberg, Salzman, Abbott, and Miller. This group focuses on the development of novel experimental and computational strategies for analyzing and deciphering how signaling in neural circuits controls behavior. The goal is to develop more powerful tools to enable one to move from the study of individual nerve cells to that of complex neural systems that underlie higher mental function.

The **Center for Theoretical Neuroscience**, directed by Drs. Abbott and Miller, includes Dr. Qian, as well as other faculty members on the Morningside Campus. The goals of this center are to determine how neurons encode and process information and to help extend our understanding from single-neuron to network-level analyses of neural systems.

Current Research

Systems and Cognitive Neuroscience

Three laboratories study visual cortex with the goal of gaining insights into general principles of cortical function:

The lab of Aniruddha Das uses optical imaging of primary visual cortex (V1) of monkeys and fMRI studies of humans to study the early stages of visual processing. They have observed strong nonlinearities in the optical signals generated by mid-level visual features (contours made of lines laid end to end). The geometry of these nonlinearities provide a measure of the intracortical interactions that could underlie cortical processing of such a feature. Moreover, the fMRI responses of human V1 to mid-level visual features have been found to match the physiological results from monkey and can be related to human psychophysics.

The Qian lab has developed a physiologically plausible theoretical model of the interactions within visual cortex that underlie daVinci stereopsis. This model provides the first computational understanding of a previously bewildering psychophysical phenomenon – the fact that an object viewed through an aperture evokes a perception of depth even though a significant portion of the object is viewed with one eye.

Ken Miller and colleagues are trying to understand the behavior of neural networks in which there is strong recurrent excitation that is stabilized by strong feedback inhibition. They have found that recent experimental results on surround inhibition in cat primary visual cortex (V1) can best be understood by assuming that V1 is such a network, and that observations of the patterns of spontaneous activity in V1 can be understood in this context. Such networks have many non-intuitive and seemingly paradoxical properties, and they are exploring their behavior both generally and in the context of specific problems of understanding V1.

Sensory responses arise from an interaction of evoked and spontaneous activity. In many sensory areas, spontaneous activity is similar in both magnitude and form to evoked responses. Embedding responses evoked by sensory stimuli in such strong and complex background activity seems like a confusing way to represent information about the outside world. However, modeling studies by Laurence Abbott and colleagues indicate that, contrary to intuition, information about sensory stimuli may be better conveyed by a network displaying chaotic background activity than in a network without spontaneous activity.

Three labs are engaged in studies examining higher cognitive functions such as selective attention and decision-making:
Michael Goldberg's lab has shown that neurons in the monkey parietal cortex show evidence of suppression of responses to an ordinarily salient popout stimulus when a monkey suppresses the response to the stimulus behaviorally. In a separate study they have discovered a previously unexpected proprioceptive representation of eye position in monkey somatosensory cortex.

Ken Miller and colleagues have found a robust and simple dynamical explanation, in terms of neural circuit properties, for the observation that single neuron responses in monkey visual cortical area LIP can predict the time over which a monkey's attention can be distracted from a behavioral goal by a behaviorally irrelevant stimulus.

Jacqueline Gottlieb's lab has discovered that attention-related activity of individual neurons in parietal cortex is sensitive to behavioral context, implying that the parietal cortex can be modulated by attention based on memory and prediction within specific contexts. These results argue that the parietal lobe participates in the higher-level decisions as to which of many sources of information is most attention-worthy at a particular moment.

Vincent Ferrera and colleagues have shown that categorical decision-making in humans involves a fronto-striatal cortical network different from the network involved in signal detection decisions. In ongoing research using fMRI in awake monkeys, they have found that monkeys have a categorical decision-making network similar to that in humans. This finding validates the hypothesis that the monkey can be used as a model system to understand the physiology of decision-making in humans.

Learning and Memory
Three labs use the giant neurons of the sea slug *Aplysia californica* as a model system to study the cellular and molecular mechanisms of learning and memory:
Craig Bailey and Eric Kandel have collaborated on studies of the molecular mechanisms that underlie learning-related synaptic growth. They have found that serotonin-induced regulation of the signaling pathways that are controlled by the small GTPases, Cdc42, and the consequent reorganization of the presynaptic actin network, appear to be a part of the initial molecular cascade required for the growth of new synaptic connections that mediate long-term learning and memory.

Sam Schacher's lab has found that formation of specific synapses during development shares common cellular and molecular mechanisms with long-term changes in those synapses during learning and memory in the adult. They have found that both target- and activity-dependent synthesis and release of a neurotrophin-like peptide from sensory neurons play a crucial role in both the formation of specific synapses and long-term facilitation of the synapses. Local translation of mRNA transported to synapses after binding with specific proteins is critical for synapse formation and synaptic plasticity underlying learning. The synthesis of the neurotrophin-like peptide near synaptic terminals and its subsequent release contributes to the initial steps associated with synapse formation and long-term plasticity. These studies suggest that a common response to external stimuli – secretion of a neuropeptide – regulates synapse formation both during development and when stimuli are sufficient to produce long-term memories in the adult.

Robert Hawkins' lab has continued their studies of neuronal mechanisms underlying long-term learning learning in *Aplysia* and in mammalian hippocampus. In *Aplysia* they have continued to study the relative importance of postsynaptic and presynaptic mechanisms for short-term facilitation at sensory-motor neuron synapses in isolated cell culture and for behavioral sensitization of siphon withdrawal in a semi-intact preparation. Their studies in both systems have shown that presynaptic mechanisms play important roles in the early stages of facilitation, and that postsynaptic mechanisms become important later on. More recently they have found that expression of the later stages of facilitation also involves modulation of the presynaptic machinery for transmitter release, and that expression of the later stages of facilitation may also involve a similar presynaptic mechanism. In parallel studies of the mammalian hippocampus they have determined the kinetics of specific molecular changes that contribute to synaptic growth during long-tern synaptic potentiation.

Steve Siegelbaum's laboratory investigates the role of dendritic integration and its control by ion channels in the function of the mammalian hippocampus. They focus on the hyperpolarization-activated HCN1 cation-channels, which are prominently expressed in a gradient of increasing density with distance along the apical dendrites of CA1 pyramidal neurons, the major output of the hippocampus. HCN1-knockout mice show an enhancement both in dendritic integration and long-term synaptic plasticity at the distal dendritic inputs, the site where HCN1 expression is normally greatest. In addition, the mutant mice show a surprising enhancement in learning and memory. Thus HCN1 channels normally act as an inhibitory constraint of synaptic transmission and learning and memory. Moreover, the HCN1 channels can be up-regulated or down-regulated as a function of neural activity, including during seizures. The Siegelbaum lab has been examining the molecular basis for the dendritic gradient of HCN1 expression. They have identified two alternatively spliced variants of a protein, termed TRIP8b, both of which interact with the HCN1 channels. One isoform causes up-regulation and the other down-regulation of the channel, and their expression patterns suggest that they may account for the birectional trafficking of the channel that underlies the variable dendritic gradients of HCN1.

Two laboratories focus on the role of the amygdala in encoding emotion:
The Salzman lab studies the neuronal basis of fear-conditioning in non-human primates. Using single neuron recordings from awake behaving monkeys they have showed that the amygdala has neurons that assign positive or negative values to conditioned stimuli, and that amygdalar activity correlates with the subject's learning

those associations. They have also discovered a value error signal in the amygdala that functions for both positive and aversive events – unexpected events have a greater effect on amygdala-activity than do expected events.

The Kandel Lab has focused on three aspects of amygdala functioning. In the first study they have identified stathmin, an endogenous protein inhibitor of microtubule formation, as being highly expressed in the lateral nucleus of the amygdala as well as in the thalamic and cortical structures that send information to the lateral nucleus about learned innate fearful stimuli. Their results indicate that stathmin is necessary for the long-term synaptic potentiation in the amygdala that is involved in recognizing danger in learned and innately aversive environments. In a separate study they examined the neurobiology of the mechanisms by which animals identify, develop, and exploit conditions of safety and security. They found that learning that a specific environment provides safety involves both a reduction of learned and instinctive fear as well as generation of positive affective responses. They observed neural signatures of neural activity for safety and danger in the amygdala and striatum of the mouse. In the third project – an fMRI study of humans – they found that emotional conflict is resolved through top-down inhibition of amygdalar activity by input from the rostral cingulate cortex.

Translational Research
John Martin's laboratory works in two areas of relevance to clinical problems. In the first set of experiments they study a process that is defective in cerebral palsy patients – the postnatal development of the corticospinal system, the principal system for controlling voluntary movements. Their results show that activity-dependent processes are critical for the normal development of the control of the final hand position during reaching. Interestingly, the same activity-dependent processes are not important for developing control of the trajectory of the hand to the target of a reach. This dissociation suggests that there are separate processing channels in the motor systems for controlling the trajectory and the terminal accuracy of a movement. In the second project they are attempting to translate a method for transmitting neural control signals around a CNS lesion in the spinal cord. Such neural bypasses together with brain machine interfaces hold great promise for improving motor control functions after spinal cord injury.

Cognitive processing requires precisely controlled signal transmission within and between brain regions. Earlier theoretical modeling of brain function in Laurence Abbott's lab has established basic principles of signal propagation but without any control of where or when signals were transmitted. They have now introduced a mechanism for gating signals that replies on balanced inhibition canceling incoming excitatory signals. In the "resting" state, neurons are unresponsive to these canceled signals. Transmission is gated on by modulation of excitatory and inhibitory gains to undo the detailed balance. They have modeled gating through detailed balance in large networks of integrate-and-fire neurons and have found that its failure modes produce effects reminiscent of clinically observed pathological states.

Three laboratories have used animal models to study various aspects of schizophrenia: Lorna Role's lab studies mechanisms that underlie the targeting, signaling, and activation of nicotinic-type acetylcholine-receptors (nAChR)s in neurons at developing circuits, and at mature pre, post and peri-synaptic locales. These processes elicit a broad range of cellular, circuit and behavioral responses. Role and colleagues examine the nAChR subunit determinants of synaptic modulation and the expression, targeting and signaling of nAChRs by taking a comprehensive cellular, electrophysiological, and behavioral approach to analyze select cortico-limbic circuits. The goal is to dissect the cellular mechanisms by which the expression and targeting of selected nAChRs participate in signaling in brain areas associated with natural reward signals and contribute to neuropsychiatric disorders such as schizophrenia.

The Siegelbaum lab, in collaboration with the laboratory of Joseph Gogos at Columbia, has found that mice with a deletion of the gene encoding proline-dehydrogenase, a risk factor for schizophrenia, exhibit abnormal transmitter release and synaptic plasticity in the hippocampus. These changes may in turn lead to the observed defect in learning and memory in these mice and perhaps contribute to the disease in humans.

Increased activity of D2 receptors (D2Rs) in the striatum has been linked to the pathophysiology of schizophrenia. To determine directly the behavioral and physiological consequences of increased D2R function in the striatum, the Kandel lab generated mice with reversibly increased levels of D2Rs restricted to the striatum. The deficit in the working memory task persisted even after the transgene had been switched off, indicating that it resulted not from continued overexpression of D2Rs but from excess expression during development. They found that D2R overexpression in the striatum impacts dopamine levels, rates of dopamine turnover, and activation of D1 receptors in the prefrontal cortex, measures that are critical for working memory. Current research in the lab addresses whether the negative symptoms of schizophrenia are related to defects in cognitive symptoms.

Education and Training

The faculty of the Center for Neurobiology and Behavior is actively involved in training medical, dental and pre-doctoral students and post-doctoral fellows and post-graduate clinical trainees. Together with their colleagues from throughout the university's neuroscience community, they teach the course in basic Neural Science for first year Medical and Dental students, as well as several graduate courses in neuroscience for predoctoral students. The pre-doctoral and postdoctoral trainees in the Center are supported by two training grants from NIH.

Awards/Honors

Laurence Abbott was elected to the American Academy of Arts and Sciences.

Michael Goldberg was elected to the American Academy of Arts and Sciences.

Eric Kandel received the Austrian Medal of Honor for Science and Art, a Doctor of Science, Honoris Causa, New York University and a Doctor of Science, Honoris Causa, Rockefeller University.

Lorna Role received both The Sidney R. Baer, Jr. Prize from NARSAD and the McKnight Neuroscience of Brain Disorders Award.

Research Highlights

Drs. Kandel and Joy Hirsch (Dept of Radiology) and colleagues have used fMRI techniques to show that in humans emotional conflict is resolved through top-down inhibition of activity in the amygdala by input from the rostral cingulate cortex.

Dr. Siegelbaum, in collaboration with Joseph Gogos (Dept. of Physiology & Cellular Biophysics), found that mice with a deletion of the gene encoding proline-dehydrogenase, a risk factor for schizophrenia, leads to alterations in transmitter release and synaptic plasticity in the hippocampus, potentially contributing to the defect in learning and memory in these mice.

Dr. Gottlieb has identified neurons in parietal cortex that integrate information about the location of a task-relevant object with information about the action instructed by that object and with information about behavioral context. This result suggests a mechanism by which attention-related areas of the brain can participate in linking sensory information with motor output in a flexible manner that is sensitive to task demands.

Dr. Kandel and colleagues found that stathmin, a peptide that is enriched in the amygdala, controls both learned and innate fear. Stathmin is required for the induction of long-term potentiation of synaptic inputs to the amygdala and is essential for regulation of both innate and learned fear.

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