Michael P. Sceniak

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Education

1994-2000 Ph.D. New York University Center for Neural Science.
Advisor: Robert Shapley.

1990-1994 BS Eng from Northwestern University, with a major in Biomedical Engineering

Awards

2004-present Stanford University Anesthesia Department Fellowship
2001-2003 Ruth L. Kirschstein National Research Service Award Fellowship
2000-2001 Hoffman Foundation Fellowship
1998 NYU, Deans's Travel Award

Research Experience

2004-present Postdoctoral research fellow in the laboratory of M. Bruce MacIver at Stanford University School of Medicine, Department of Anesthesia. Projects focusing on the synaptic organization of visual cortex and the cellular mechanisms of anesthesia on neocortical neurons, using visualized whole-cell patch clamp recording.

2001-2003 Postdoctoral research associate in the laboratory of Marty Usrey at the University of California, Davis. Research focused on whole-cell patch clamp recording *in vitro* of ferret primary visual cortical neurons. Other projects included investigating geniculocortical synaptic efficacy using multielectrode recordings in the LGN and primary visual cortex of paralyzed anesthetized primates.

2000-2001 Visiting researcher in the laboratory of Edward M. Callaway at the Salk Institute, La Jolla, CA. Research focused on extracellular recordings of geniculocortical afferents in macaque visual cortex using pharmacological techniques to inactivate cortical responses.

1994-2000 Predoctoral research under the guidance of Prof. Robert Shapley and Michael J. Hawken at the Center for Neural Science at New York University. Dissertation: Spatial summation and surround suppression in primate V1 neurons: contrast effects.

1992-1994 Undergraduate research projects under the direction of Prof. David Ferster, Northwestern University. Research assistant work focused on assisting with intracellular whole-cell patch recording experiments and constructing experimental electronics equipment.

Teaching Experience

2001-present Mentoring graduate students and technicians in laboratory procedures and scientific project design.

1995,1997 Teaching Assistant for Lab in Neural Science I, graduate level laboratory course on neuroanatomy and biophysics.

Scientific Community Activities

2006	Reviewer at several scientific journals.
2006	Grant reviewer for NIH SAT study section.
2003	Grant reviewer for National Science Foundation Learning Information Systems (LIS).
2001	Invited Speaker at Smith Kettlewell Eye Research Institute.
1998	ARVO 1998, Oral Presentation: Michael P. Sceniak, Dario L. Ringach, Michael J. Hawken and Robert Shapley. Spatial Summation in Macaque V1 Neurons Depends on Stimulus Contrast.
1998	Society for Neuroscience 1998, Oral Presentation: Michael P. Sceniak, Dario L. Ringach, Michael J. Hawken and Robert Shapley. Contrast-Dependent Area Summation in Macaque V1 Neurons.

Publications

Sceniak, M.P. and MacIver M.B. (in preparation) GABAa slow mediated synaptic current in primary visual cortex.

Sceniak, M.P. and Usrey W.M. (in preparation) Frequency Dependence of Spike-Rate Encoding in Ferrent Primary Visual Cortical Neurons *in vitro*.

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Sceniak, M.P. and Callaway, E.M. (2006) VisualSpatial Summation in Macaque Geniculocortical Afferents. *J Neurophysiol*. Aug 23; [Epub ahead of print]

Sceniak, M.P. and MacIver M.B. (2006) Anesthesia *in silico*. *Anesthesiology*. Mar;104(3):400-2.

Sceniak, M.P. and MacIver, M.B., (2006) Cellular actions of urethane on rat visual cortical neurons *in vitro*. *J Neurophysiol*. Mar 1.

Usrey W.M., **Sceniak, M.P.** and Chapman, B. (2003) Receptive Fields and Response Properties of Neurons in Layer 4 of Ferret Visual Cortex. *J. Neurophysiolog*, 89(2):100315.

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Mareschal, I. **Sceniak, M.P.**, and Shapley, R. (2001). Contextual influences on orientation discrimination: binding local and global cues. *Vision Res* 41(15): 1915-1930.

Hawken, M.J., Shapley R.M., **Sceniak, M.P.,** Ringach DL & Johnson EN (2000) "Contrast gain, Area summation and temporal tuning in the primate visual cortex. In Vision and Attention, ed LR Harris and M Jenkin, Springer-Verlag, NY.

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Abstracts

Sceniak, M.P. and MacIver M.B., (2006) $GABA_A$ slow synaptic responses in rat visual cortex.*Soc. Neurosci Abstr.*

Sceniak, M.P. and MacIver M.B., (2005) Urethane Anesthesia: A Novel and Specific Mechanism Of Action. *Anesthesiology*; 103: A141

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Sceniak, M.P., and Usrey W.M. (2003). Frequency Dependence of Spike-Rate Encoding in Ferret Primary Visual Cortical Neurons in vitro. *Soc. Neurosci. Abstr.*, **29**: 485.15.

Sceniak, M.P., Chatterjee, S., and Callaway, E.M. (2001). Spatial Analysis of Geniculocortical Afferents in Macaque V1. *Soc. Neurosci. Abstr.*, **27**: 619.44.

Mareschal, I., **Sceniak, M.P**., Shapley, R. (2000). Contextual influences on orientation judgments. *Invest Ophth Vis Sci. Suppl. S* 41(4): 188B188.

Hawken, M.J., Shapley, R., Ringach, D.L., **Sceniak, M.P**., Johnson, E.N., Mareschal, I. (2000). Laminar distribution of orientation selectivity of macaque V1 neurons. *Invest Ophth Vis Sci Suppl. S* 41(4): 276B276

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Hawken, M.J., Shapley, R., Mechler, F.M., Ringach, D.L., **Sceniak, M.P**., and Johnson, E.N. (1997). Temporal Tuning for Color and Luminance in Macaque V1. *Soc. Neurosci. Abstr.* **23**:405.6.

Research Statement —Michael P. Sceniak

Neocortical sensory inputs can be divided into 'drivers' and 'modulators' (Sherman and Guillery, 1998; Crick and Koch, 1998; Abbott and Chance, 2005). Driver inputs transmit information for basic receptive field properties while modulators alter the properties of certain aspects of transmission established by the drivers. Within the visual system, driver inputs transmit the information that leads to the classical receptive field structure, such as seen in simple and complex cells. Modulator circuits act to enhance or modify the classical receptive field properties. The role of modulator inputs within the visual cortex is clearly illustrated by recent studies that show effects of stimulation from 'beyond the classical receptive field' (Allman et al. 1985; Angelucci and Bullier, 2003). In these studies, it was shown that a stimulus placed in the region beyond the classical receptive field has no effect on the spike discharge when in isolation. However, when paired with stimulation of the classical receptive field region, the non-classical stimulation produces either facilitation or suppression of the center alone stimulation. Non-classical modulation has been linked to high-level processing for figure-ground segmentation. In awake-behaving preparations, the effects of attention have also been shown to modulate neuronal responses.

While much attention has been paid to defining the circuits of the driver inputs to the visual cortex, less is known about the circuitry and/or cellular mechanisms of the modulatory inputs. Recent studies have provided evidence for anatomical segregation of circuits that modulate versus those that provide direct driver input. Thalamocortical axons synapse in particular cortical layers and terminate near the apical dendrites of recipient neurons while intracortical synapses are distributed throughout the dendrite and long-range horizontal axons terminate distally (Douglas and Martin, 2004; Stettler et al., 2002). There are also differences in the inhibitory cell types and expression of inhibitory receptors located near the soma versus the distal dendrites of neocortical pyramidal neurons (Connors, 1992; Chu et al., 2003; Galarreta and Hestrin, 2002; Kasamatsu et al., 2005; Douglas and Martin, 2004). This segregation provides a valuable tool for dissection of the mechanisms mediating drive and modulation. By targeting the known cellular differences between proximal and distal inhibitory mechanisms *in vivo*, I will be able to determine the differential effects of these cellular targets on receptive field modulation. Synaptic and intrinsic membrane mechanisms related to modulation will be studied *in vitro*.

My overall goal is to explore the fundamental cellular mechanisms that govern visual perception. Given the interdisciplinary nature of neuroscience, it is essential to approach systems level questions from many angles, and this has motivated me to acquire complementary tools to approach these questions. My training in biomedical engineering as an undergraduate student and neural science as a graduate student has prepared me to explore these issues from the cellular to the psychophysical level. The postdoctoral training that I have received has allowed me to expand my range of techniques to include whole-cell patch clamp recording *in vitro*, *in vivo* pharmacological manipulations, and single cell labeling *in vitro* and *in vivo*.

Graduate Research

I received my Ph.D. under the supervision of Robert Shapley at NYU's Center for Neural Science. While at NYU, I studied receptive field properties of neurons in the primary visual cortex (V1) of anesthetized paralyzed macaque monkeys, using extracellular recording techniques. My thesis research focused on how stimulus context nonlinearly modulates receptive field properties. The size of the classical receptive field increases under low contrast conditions by 2.3 times, confirming that neurons in the primary visual cortex cannot be treated as passive stationary linear filters. Instead, V1 neurons alter their response properties based on the context of stimulation and, therefore, on the overall state of the network in which they are a member. This state dependence of spatial summation is not a result of antagonistic surround inhibition. Therefore, these contrast-dependent changes in spatial summation likely result from dynamic changes in the neural circuit.

My thesis work also revealed that the extent of spatial summation is larger than previously estimated and that this extent cannot be accounted for merely through convergent input from the thalamus into a single hypercolumn: receptive field properties must arise from recurrent feedback from intralaminar connections. The state dependence of spatial summation suggests that these intralaminar connections are also activity dependent and, therefore, influenced by modulator inputs.

Postdoctoral Research

After graduating from NYU's Center for Neural Science, I visited the Salk Institute in the laboratory of Ed Callaway. While at the Salk Institute, I investigated the properties of geniculocortical afferents in anesthetized paralyzed macaque monkeys. Bathing the cortex with muscimol, a $GABA_A$ receptor agonist, blocked cortical activity while preserving the activity of the axons of afferent lateral geniculate nucleus (*lgn*) inputs. Individual afferent inputs were isolated and characterized for their spatial and chromatic preference. Magno-cellular (M), parvo-cellular (P) and konio-cellular (K) inputs were identified both anatomically and based on wavelength preference. It was found that M, P and K afferent inputs exhibit a high degree of non-classical surround suppression. The extent of spatial summation was contrast-invariant, even in the absence of cortical activity. This supports the hypothesis that contrast-dependent spatial summation observed in the primary visual cortex arises from cortical modulator circuits.

My first postdoctoral position was in the laboratory of Dr. W. Marty Usrey at UC Davis' Center for Neuroscience. I was involved in several projects designed to investigate the information transfer from the LGN to the primary visual cortex. Specifically, I was involved in a project in collaboration with Dr. Barbara Chapman, where we characterized the receptive field properties of simple cells in the input layers of ferret visual cortex. Although the organization of geniculate inputs to layer 4 differs substantially between ferret and cat, our results demonstrate that, like in the cat, most neurons in ferret layer 4 are orientation-selective simple cells. In addition to in vivo studies of single unit responses in cortex, I also developed a whole-cell patch clamp preparation using ferret cortical brain slices. These experiments were focused on determining the role of noisy synaptic background activity on the frequency response function.

Currently, I am a postdoctoral fellow in the laboratory of M. Bruce MacIver in the department of Anesthesia at Stanford University School of Medicine. In the MacIver laboratory, I have continued to use the visual cortical brain slice preparation and the whole-cell patch clamp technique. My research has focused on the synaptic properties of rat neocortical neurons. A fundamental issue in animal research concerns the comparison of data acquired *in vitro* versus *in vivo* in whole animal anesthetized paralyzed preparations. Urethane anesthesia is widely used in rodent animal research, yet little was known about its cellular actions. I determined that urethane has no substantial impact on synaptic transmission for either GABAergic or glutamatergic responses, but depresses the responsiveness of neocortical neurons through a background leak conductance. This confirms the relatively minor anesthetic side effects seen under urethane anesthesia compared to volatile anesthestics.

Most recently I have been investigating a novel form of GABAergic transmission in the visual cortex, slow $GABA_A$. Robert Pearce and colleagues (Banks et al., 1998) demonstrated the existence of this unique form of GABAergic transmission in the hippocampus. The rise and decay time constants (5-10ms and 20-50ms respectively) are dramatically different from those of the fast $GABA_A$ spontaneous responses arising from fast spiking cells (FS) or low-threshold spiking inhibitory cells (LTS) previously described in neocortex. In hippocampus, slow $GABA_A$ responses are easily evoked through extracellular stimulation and occur infrequently spontaneously. In neocortex, spontaneous slow $GABA_A$ responses occur infrequently and are evoked only in superficial layers I/II. There is great diversity of inhibitory neurons in the neocortex, and it remains to be determined which subtype is the source of these slow $GABA_A$ responses. This slow form of $GABA_A$ synaptic transmission has relevance for modulatory effects in visual processing as well as for disease states and anesthesia.

Research Plans as an Independent Investigator

As a primary investigator, I hope to bring my expertise with *in vivo* extracellular recording together with intracellular techniques *in vitro* to understand better the microcircuits and biophysical mechanisms that contribute to response modulation in the neocortex. Understanding the state dependence of V1 receptive fields will require a detailed knowledge of the modulator inputs. Individual neurons *in vivo* display a great deal of nonlinearity in their response properties. These nonlinearities are expressed as context-dependent tuning for properties like receptive field size. There are also substantial nonlinear interactions in the temporal domain. By investigating the intrinsic properties of individual neurons *in vitro*, I will be able to determine the biophysical source of these nonlinear interactions.

Three general approaches will be used. Pharmacological manipulation of cellular targets will be tested *in vivo* to determine the role of these mechanisms on receptive field modulator inputs. Next, *in vitro* experiments will examine state-dependent modulation of synaptic and intrinsic membrane mechanisms for driver vs. modulator circuits. Lastly, anatomical analysis using juxtacellular or intracellular labeling *in vivo* will help correlate cell type specificity to particular sources of modulatory input. Studying both driver and modulator input neurons *in vivo* and *in vitro* will not only provide insight into the specific mechanisms of visual receptive field construction, but will also reveal the general neural mechanisms employed by the brain for communicating information from one region to another.

Objective 1— To investigate inhibitory modulation of receptive fields in visual cortex

Neurons in all cortical layers exhibit both $GABA_A$ and $GABA_B$ inhibitory post-synaptic potentials (IPSPs) (Douglas and Martin, 1991, 2004). Muscimol, a $GABA_A$ agonist, suppresses local field potential signals correlated to classical receptive field driver inputs while baclofen, a $GABA_B$ agonist, affects non-classical surround modulation signals (Kitano et al., 1994; Kasamatsu et al., 2005). $GABA_B$ receptors sensitive to baclofen are localized on the distal dendrites of excitatory pyramidal cells. Together, these findings argue for a differential effect of $GABA_A$ and $GABA_B$ receptors in targeting driver inputs versus modulatory inputs from the non-classical surround. These studies did not examine classical and non-classical receptive field properties at the single cell level and I propose to examine the differential role that these inhibitory inputs contribute to single unit receptive field properties.

One important approach is to consider the modulatory role that candidate cellular mechanisms convey to receptive field properties *in vivo*. Receptive field parameters are a direct measure of functional output of neurons in the visual cortex. Pharmacological manipulations *in vivo* combined with receptive field characterization will help tease apart the cellular mechanisms of modulation in the visual cortex. Receptive field characterization for orientation, temporal frequency, contrast, spatial frequency as well as stimulus diameter and bipartite field stimulation will be performed in control conditions and under drug application. Because the pyramidal neurons of the visual cortex project radialy toward the pial surface, it is possible to specifically target the distal dendrites with pharmacological blockers applied to the pial surface. Local application of the $GABA_B$ receptor agonist, baclofen, and the receptor antagonist, CGP, will permit a dissection of the differential impact of these inhibitory inputs on receptive field (driver) inputs. Local application of blockers *in vivo* can also be achieved through iontophoretic injection using multi-barrel pipettes. These experiments will be conducted in either rodents or carnivores such as ferrets and cats.

Objective 2 — To investigate state-dependence of intrinsic membrane and synaptic interactions *in vitro*

Inputs located near the distal dendrites of pyramidal cells originate from intracortical and long-range inputs and are thought to provide the basis of global modulation (Douglas and Martin, 2004; Kasamatsu et al., 2005). There is also specificity in the types of inhibitory neurons that synapse distally (i.e. LTS) or proximally (i.e. FS) with respect to pyramidal cell dendrites (Galaretta and Hestrin,

2002; Chu et al., 2003). Although non-classical surround modulation has been linked to long-range horizontal connections and normalization mechanisms, these conclusions are based primarily on speculation from conceptual models and extracellular recording. It is likely that additional mechanisms contribute to modulation, such as anatomically localized forms of inhibition, differences in the temporal integration of synaptic events and/or plasticity. The *in vitro* preparation allows simulation of activity levels to determine the state-dependence of these mechanisms and their possible roles in global modulation. It will also be important to determine the differential role of excitatory integration into the different inhibitory cell types located either distally or proximally to the pyramidal cell bodies. These experiments will be conducted in acute brain slices of either rodents or ferrets.

Contrast-dependent changes in receptive field size suggest that state-dependent changes in the cortical network can cause reorganization or modulation of driver inputs that establish receptive field structure. Changes in stimulus contrast are analogous to changes in the intensity of the input drive as well as the overall activity in the network. Therefore, dynamic changes in local inputs appear to depend on the overall global activity of the system. Because the acute brain slice is a quiescent circuit, it is amenable to experimental control and manipulation for testing these properties. The primary goal is to determine the effects of increased activity on synaptic integration. This will be accomplished by simulating background synaptic activity through the dynamic clamp system. Similar effects can be simulated through spatially localized focal uncaging of glutamate along the dendrites. In addition, these approaches can be combined with pharmacological targeting of particular modulator sites with specific blockers (e.g. acetycholine and noradrenalin, which have been linked to attention-dependent modulation *in vivo*).

Long-Term Objectives — To correlate physiological responses with cell subtypes

By recording *in vivo*, one can take advantage of the natural visual inputs to drive the system. Complex spatial interactions from regions beyond the classical receptive field can be studied in the *in vivo* preparation where the retinotopic map is preserved. Juxtacellular or intracellular biocytin filling can be used to correlate receptive field properties with particular anatomical or cell subtype classifications. Filled neurons can also be counter-stained with antibodies for further subclassification. For example, receptive field tuning properties can be related to whether the recorded neurons were FS or LTS cells, which target proximal and distal regions of pyramidal dendrites, respectively. These experiments will help identify which neurons contribute to specific forms of modulation observed *in vivo*.

References

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Teaching Statement — Michael P. Sceniak

A commitment to teaching is an important component to success as a professor. My undergraduate experience at Northwestern University emphasized the importance of a strong understanding of the fundamentals in science and mathematics. Teaching not only benefits the student, but also the professor. As we advance in our investigations of cutting edge science, it is important to be aware of this work as it relates to the big picture of the field. Teaching forces the professor to be aware of different perspectives and more fundamental issues in science.

During my graduate training at New York University's Center for Neural Science, I was a teaching assistant for the laboratory component of the core course in neural science twice. The course ran for one semester and consisted of a section on neuroanatomy, both gross and cellular, as well as a section on biophysics. I assisted the students in learning neuroanatomy by answering questions and designing and administering exams. The course covered human, sheep, cat, monkey and rat neuroanatomy. We also covered basic methods of anatomical preparation including rat perfusion, histology and microscope techniques. The latter portion of the course covered biophysics. During the course I gave lectures and assisted in the experiments. The students were required to perform intracellular recording of leech and frog neurons. I assisted the students in making the preparations and explaining and assisting in the actual recording. This included extensive explanation of the computer controlled data acquisition system.

My graduate and undergraduate experience has exposed me to subjects ranging from basic science and engineering to advanced subjects in neuroscience and psychology. I feel confident teaching courses ranging from biophysics and neuroanatomy to specialized courses within visual neurophysiology and psychophysics. I also have experience explaining and teaching others computational methods and analysis.

During my time as a graduate student and as a postdoc, I have given many public lectures on my work. I have given talks and poster presentations at the annual meeting for the Society of Neuroscience and the ARVO annual meeting. I was also an invited speaker at the Smith Kettlewell Eye Research Institute and Department of Physiology at Northwestern University. Recently I have given presentations at departmental seminars as well as the 2005 Annual Anesthesia Department Awards Dinner at Stanford University Medical Center. These experiences have given me exposure to lecturing in front of large audiences, teaching and seminar format lectures.

The success of any academic department ultimately depends both on the quality of its research and the quality of its teaching. As a Professor, I look forward to the challenges and rewards of research and teaching.

Teaching Experience:

1995	TA	Neural Science I — laboratory in neuroanatomy and biophysics
1997	TA	Neural Science I — laboratory in neuroanatomy and biophysics

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