Neuroscience Graduate Program
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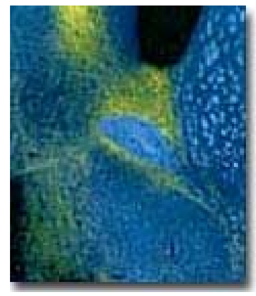

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Research Interests

The lab studies fundamental issues in systems neuroscience using the mammalian retina as a model. We focus on learning how a local circuit creates a neuronal receptive field and also on elucidating its multiple cellular mechanisms for plasticity. Such mechanisms predict the most likely future stimulus and use the prediction to optimize the neuron,s sensitivity (adaptation). We study these issues in retina because it is the only brain region that can be studied in vitro while presenting the natural stimulus it was designed to encode. For example, we can study how retinal neurons respond, in vitro, to videos of artificial stimuli or natural scenes presented on a miniature computer monitor.

Our studies have focused on the output neurons (ganglion cells), whose axons form the optic nerve. We start with observations from in vivo spike recordings and establish that the same paradigms work in vitro. Of course, the spike patterns are simply suprathreshold , readouts, of complex integrative processes. These processes we study by monitoring subthreshold events by whole-cell patch clamp recording. Various synaptic mechanisms are identified by pharmacological experiments and quantified with computational models.

Initially, we have focused on the cellular mechanisms for contrast adaptation. This plastic mechanism optimizes ganglion cell sensitivity to the level of contrast within a scene. We find that contrast adaptation arises beyond the photoreceptor synapse; thus it is created within the retinal circuit. One form of contrast adaptation occurs rapidly, reducing sensitivity within 100 ms of exposure to high contrast. Sensitivity changes arise at both the level of excitatory synaptic inputs and at the level of spiking outputs (Zaghloul et al., 2005; Beaudoin et al., 2007). Another form of contrast adaptation occurs slowly, causing prolonged membrane hyperpolarization following the offset of high contrast (Manookin and Demb, 2006). Unexpectedly, the synaptic basis for both of these adaptations arises from intrinsic properties of the excitatory interneuron, independent of synaptic inhibition. However, inhibition does drive other forms of adaptation. Ganglion cell spiking to local stimuli adapts to contrast present on a distant retinal region. The circuit requires a type of interneuron that relays contrast signals via long-distance inhibitory synapses. These synapses inhibit both the ganglion cell dendrites and the presynaptic excitatory axon terminal (Zaghloul et al., 2007).

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