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Barrels XXIII: Barrels by the shore

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Abstract
The 23rd annual Barrels meeting was held on the University of California, San Diego campus and highlighted the latest advances in the whisker-to-barrel pathway and beyond. The annual meeting brought together investigators from a dozen countries to present their data in posters and short talks. The meeting focused on several themes, first the barrel system was used as a model to study the consequences that result from alterations in the normal pattern(s) of development. A second session focused on what happens to whisker information once it leaves the layer IV barrel. A third session addressed issues of coding within the barrel system and a final session highlighted the latest advances in the engineering of transgenic mouse lines. The meeting highlighted the utility of the barrel system to study cortical circuitry in the normal and pathological state.

Keywords: *Whisker, vibrissa, barrel, meeting*

Forty years after the initial publication of the discovery of the cortical barrels, the 23rd annual Barrels meeting convened in the Calit2 Auditorium on the campus of the University of California, San Diego on 11 and 12 November 2010. The meeting welcomed 90 researchers from a dozen countries to discuss the latest advances in the rodent sensorimotor system (see Appendix 1 for the complete schedule).

The opening session focused on how the barrel system can serve as a model to investigate what can happen behaviorally, genomically, physiologically, and anatomically when the system is disrupted by developmental disorders. **Fan Wang** (Duke University, USA) introduced and moderated the morning session, reminding the audience of the devastating consequences on neurocognitive development in humans that result from Rett syndrome, Fragile X, and Reelin disorders.

Sacha Nelson (Brandeis University, USA) focused on the physiological genomics of Rett syndrome. Rett syndrome is the second most common genetic cause of mental retardation and is due to the specific mutation of the MeCp2 gene, in addition to the neurocognitive sequelae there are disruptions of the autonomic nervous system as well as sleep and respiratory abnormalities. He focused on determining the expression of genes found in specific cellular phenotypes and how their misregulation could lead to the disruption of neural circuits which directly leads to the resultant pathology. Mice who have had their MeCp2 gene deleted recapitulate the disease and it was found that cortical neurons from these animals showed a reduction of excitatory currents and an increase in inhibitory currents under spontaneous conditions and display reduced long-term potentiation and smaller excitatory post-synaptic potentials when synaptic inputs were stimulated. Interestingly, genomic analyses revealed that cell adhesion molecules that are important in the formation and maintenance of synapses were disrupted in the knockout animals.

Andreas Frick (University Bordeaux 2, France) focused his investigations on the most common

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inherited mental disorder, Fragile X syndrome, using mouse models where the Fmr1 gene had been deleted. Behavioral testing showed that knockout animals were able to complete a gap crossing task quicker, due to fewer whisks needed to make the decision to jump, and they were able to cross wider gaps compared to wild type animals. Voltage-sensitive dye experiments revealed more excitation following whisker stimulation in the Fragile X animals. A hallmark of Fragile X syndrome is dendritic alterations including increases in spine density. The FMRP protein (the product of the Fmr1 gene) targets many dendritic ion channels, in addition to cytoskeleton proteins, including K^+ (GIRK3, Kv 3.1, BkCa), Ca^{2+} (Cav1.3), and cation (hyperpolarization activate cation current (HCN)) channels whose misregulation was hypothesized to impact the neurons' computational abilities. Experiments revealed that neurons recorded in slices from knockout animals had less membrane ''sag'' in response to hyperpolarizing current pulses, were more excitable in response to depolarizing pulses, and showed lower resonance frequencies and greater ability to summate synaptic inputs due largely to a decrease in HCN currents. Due to a reduction in the large conductance Ca^{2+}-dependent K^+ current which normally would put the brakes on runaway excitation, there was increased excitability in the dendrites of knockout mice. These results confirmed the hypothesis that the neocortical pyramidal neurons of Fragile X mice have altered computational abilities which may underlie some of the neurocognitive deficits observed in afflicted individuals.

Jochen Staiger (George-August-Universitat, Germany) concluded the session describing his laboratories' investigations of the reeler mouse. The audience was reminded that in reeler animals the cortex develops ''inside out'' with neurons normally fated to the deep layers being found closer to the pial surface and the normally superficial neurons locating closer to the white mater. Although the ventral posterior medial nucleus of the thalamus is unaffected in these animals their thalamocortical projections are more widely distributed throughout the cortical column and do not aggregate into barrel-like units. Using *in situ* hybridization and indicators for supragranular, granular, and infragranular neurons it was seen that the thalamocortical afferents still targeted granular fated neurons even if they were not located in their normal position within the cortical column.

The session concluded with the realization that developmental disorders through their alterations in neural circuits directly lead to the observed neurocognitive and other pathological phenotypes confronted by clinicians. Only through careful dissection of the underlying neural circuits will we be able to understand how these alterations lead to the observed pathologies and stimulate the development of therapeutic interventions.

Following a brief coffee break the morning resumed with a series of short talks moderated by **Arthur Houweling** (Erasmus University, the Netherlands). Leading off, **Dudi Deutsch** (Weizmann Institute of Science, Israel) focused on the variability inherent to rat whisking. Utilizing a head-fixed preparation with all but three whiskers clipped (C1, C2, D1) he and his colleagues monitored free whisking and whisking in the presence of an object placed in the whisking path. It was found that whiskers on the same side of the face were very correlated and less correlated with whiskers on the opposite side of the face. In general, the amplitude of the whisking was more variable than the duration of the whisking cycle. When contacting an object there was a large increase of amplitude on the contralateral side with no significant impact on whisking duration. Detailed analysis of individual whisks revealed ''pumps'' wherein periods of negative velocity were observed during protraction in 25% of all contact whisks. They concluded that there were separate neural controllers for whisking amplitude and duration. From the same group, **Ehud Ahissar** (Weizmann Institute of Science, Israel) revealed the discovery of *Homo rehovotus whiskerus* a graduate student outfitted with whisker-like appendages attached to their fingers as a means of capturing all the relevant variables in regards to a sensory–motor task—''whisking'' and aperture width discrimination. The synthetic whiskers were outfitted with motion and force sensors. Increasing accuracy was correlated with more coordinated movement. It was found that changes in motor strategies by themselves directly led to increased perceptual resolution. The more ''whisking'' cycles (more sensory sampling) the smaller differences in aperture width which could be detected. In general, perception emerges from a convergence process including sensory and motor variables. The session concluded with **Jason Ritt** (Boston University, USA) arguing for creating standards for the measurement and reporting of whisker behaviors and their neural correlates. The key issue confronting researchers is how to measure the relevant variables given that little is known about the mechanics of the vibrissa follicle and how these variables might influence sensory transduction. It was pointed out that it is still unclear as to what behavioral tests should be conducted and in what reference frame whisker angles should be referenced. Finally, when using different tracking strategies to follow whisker movements it was suggested that estimation errors/assumptions should be highlighted in the relevant sections of the manuscripts.

After the annual business meeting (chaired by **Joshua C. Brumberg**, Queens College, CUNY, USA) and a scrumptious lunch, the afternoon session of short talks began under the direction of **Karel Svoboda** (Janelia Farms, USA). The session was started off by **Hiroshi Kawaski** (The University of Tokyo, Japan) who focused on the mechanism that governs barrel formation. He compared the rate of barrel formation in control animals vs those from mothers who were induced to deliver 1 day early. The size of the resultant litters and their body weights and their brains' cortical thickness were unaffected, but the barrels of the preterm animals developed earlier. The birthing process results in a decrease in serotonin levels which accounted for the early barrel development although the rate of development is the same as in control animals. Next, **Qian-Quan Sun** (University of Wyoming, USA) followed the progression of the balance of excitatory and inhibitory (EI) inputs onto neurons throughout the barrel column. Within the layer 4 barrel the EI ratio decreased from postnatal day (PND) 8 to PND18 and then remained stable. In the supragranular layers stable ratios were not obtained until PND30, layer 5 followed a similar developmental timeline to layer 4. The decrease in the EI ratio was largely accounted for by increases in inhibitory conductances. **Mary Ann Wilson** (Johns Hopkins University, USA) followed with a presentation on how perinatal exposure to lead can impact spine development within the barrel. Nursing mothers were exposed to lead in their drinking water for the first 10 days of their pups' lives which resulted in a smaller overall barrel field in the pups. Although there were no changes in the total number of neurons or in their dendritic architecture there was a decrease in the branching of the thalamocortical axons and an increase in spine density. Finally, **Rodrigo Kujis** (Encephalogistics, Inc., USA) reminded the audience of the devastating consequences of Alzheimer's disease and the lack of therapeutic interventions. Although primary sensory cortices had been thought to be spared during Alzheimer's disease, recent data show that this is not the case and that lesions can be found in the barrel cortex of mouse models of Alzheimer's disease suggesting that the barrel cortex can serve as a model system for studying the progression of the disease as well as for testing the efficacy of potential treatments.

The final short talk session of the day was led by **Qian-Quan Sun** (University of Wyoming, USA). Starting off was **Lu Li** (Helen Wills Neuroscience Institute, USA) who looked at rapid plasticity in the barrel cortex using whole cell *in vivo* recordings. The presentation highlighted two types of plasticity and their distinct mechanisms; hebbian plasticity which requires positive feedback vs homeostatic plasticity which requires negative feedback. To probe for these two types of plasticity in the barrel cortex, D row whiskers were trimmed for 1–10 days starting at PND20 and recordings were obtained *in vivo* from supragranular neurons under urethane anesthesia. It was found that there was a decrease in local field potentials following 3, 5, or 10 days of deprivation and following 3 days of deprivation there was an increase in the magnitude of the long latency response whereas after 5 days there was a decrease in the overall number of action potentials in response to principal whisker deflections. Whole cell recordings revealed that deprivation decreased overall synaptic conductance with inhibitory conductances decreasing more than excitatory which was consistent with a model of rapid homeostatic plasticity resulting from short-term deprivation in the supragranular layers. **Craig Brown** (University of Victoria, Canada) focused on the role $\alpha 4$ nicotinic receptors had on experience-driven cortical depression in the mouse somatosensory cortex using *in vivo* voltage-sensitive dye imaging. The C1 whisker was trimmed and then imaging commenced either 3, 7, or 21 days post-trimming. It was found that after 3 or 7 days post-trimming there was a decrease in the voltage-sensitive dye response to C1 deflections which returned to normal by 21 days post-trimming. It was determined that there was an increase in the $\alpha 4$ nicotinic receptor post-trimming which was colocalized with the post-synaptic scaffolding protein PSD95 and most GABAergic neurons had $\alpha 4$ puncta in apposition to their somata. Application of $\alpha 4$ agonists depressed responses and if following trimming $\alpha 4$ antagonists were continuously applied to the barrel cortex via an osmotic pump the trimming-induced depression was blocked. In sum, these results suggest that $\alpha 4$ receptors on GABAergic interneurons account for the majority of the trimming-induced depression in the voltage-sensitive dye response. The day ended with a stimulating poster session and then a barbecue dinner listening to the waves breaking on the shoreline in La Jolla, CA.

The second day of the conference opened with a session focused on understanding the barrel cortex as a part of a larger sensorimotor neural network. Specifically, the question was posed, how is peripheral sensory information conveyed and propagated beyond the layer IV barrel? **David Kleinfeld** (University of California, San Diego, USA) introduced and moderated this session, reminding the audience that instead of an isolated computational unit, the barrel cortex is strongly interconnected with the nearby primary motor cortex (M1) as well as the secondary somatosensory cortex (S2).

Carl Peterson (EPFL, Switzerland) focused on how intrinsic brain states can modulate the whisker-evoked responses of barrel cortex neurons in awake,

behaving mice. Voltage-sensitive dye (VSD) imaging showed that cortical voltage responses of a passive whisker stimulus were dramatically impacted by the behavioral state of the animal. In awake and quiescent animals, whisker stimulation elicited large depolarizations in the barrel cortex that spread almost immediately (30 ms post-stimulus) into both M1 and S2. In contrast, in animals that were actively whisking, passive stimulus elicited only small depolarizations that remained localized to the barrel cortex. The VSD imaging results were confirmed by intracellular patch-clamp recordings from layer 2/3 (L2/3) neurons. Peterson then investigated the responses of cortical neurons to active-touch, or object-contact during active whisking, using intracellular patch-clamp recording. Recordings form cortical neurons when rats where engaged in active-touch elicited mixed excitatory and inhibitory conductances which showed a large degree of variability and action-potential firing was unreliable. However, contact of object during active-touch increased the correlation of sub-threshold Vm between neighboring cells. The second part of the talk focused on the response of cortical neurons during a detection task. Mice were trained to detect a magnetic deflection of a single whisker for water rewards. Peterson showed that the voltage responses of cortical L2/3 neurons were highly dependent on the pre-stimulus EEG state of the animal. Specifically, when the EEG is in a slow-oscillatory state, the neurons show large stimulus-elicited depolarization in Vm and when the EEG is in a desynchronized state, neurons show very little Vm responses. The performance of the animal was only weakly correlated with the pre-stimulus brain state, where the miss trials showed slightly higher power in the low-frequency (1–5 Hz) oscillations.

Ron Frostig (University of California Irvine, USA) focused his investigation on using the spread of activity in barrel cortex as a general model for mammalian sensorimotor cortex activation. He first showed, using intrinsic imaging as well as extracellular recording, that the cortical area activated by a single-whisker periodic stimulus is about 15 mm^2, far larger than the area of a single barrel column ($\sim$0.5 mm^2). The spread of cortical activation is radially symmetrical, even when the center is near a border between different sensory areas (e.g., S1 and primary auditory cortex (A1)). To investigate the anatomical basis for this spread of excitation, a tracer was injected into L2/3 of individual barrels, and axons local to the injection site were labeled and were shown to radially spread several millimeters from the center. To further study the underlying mechanism of this non-specific local spread of excitation, Frostig performed a series of experiments where the gray matter between an array of eight extracellular electrodes was transected. Local field potential (LFP) signals in distant electrodes disappeared after transection, demonstrating that the spread of activation is mediated by intracortical connections. Tracer labeling was also attenuated by the transection. Additional support for the large symmetrical spread came from his study of cortical infarcts induced by permanent occlusion of the middle cerebral artery (pMCAO). When a single whisker was stimulated immediately or 1 h after pMCAO, the cortex was completely protected from the stroke. If whisker stimulation commenced 2 h after pMCAO, 80% of the animals showed complete cortical protection. However, when a single whisker was stimulated 3 h after pMCAO, the resulting infarct was larger than what would have been obtained without stimulation, and the resultant infarct matches the typical volume of cortical activation evoked by a single whisker. Imaging results similar to the ones obtained by Frostig have been shown in other sensory cortical areas such as A1 and V1 using either VSD or intrinsic imaging. The anatomical results have also been demonstrated in other regions of the sensorimotor cortex. Frostig therefore suggested that in response to a point sensory stimulus, a large, symmetrical sub-threshold spread of activation is a fundamental motif of mammalian sensorimotor cortex.

The session concluded with **Tim Murphy** (University of British Columbia, Canada) discussing his laboratory's investigation of widespread patterns of cortical activation under different conditions. Using VSD imaging, he found that spontaneous oscillation in Vm take the form of traveling waves in both anesthetized and awake mice. The traveling waves are highly synchronous between hemispheres and correspond to increases in the power of the low-frequency EEG. Areas along the midline typically initiate the traveling cortical depolarization, and are termed the ''core of spontaneous activity''. Recent sensory experiences can affect the pattern of spontaneous activity by initiating the traveling wave near the area recently activated by the sensory stimuli. Activation motifs found during spontaneous traveling waves were found to resemble the patterns of activity generated by sensory stimuli. Interestingly, sensory training stimuli can increase the amount of match between spontaneous motifs and evoked patterns of activity. Lastly, Murphy showed that, in transgenic animals whose cortical layer 5b neurons express channelrhodopsin (ChR), sensory stimuli-evoked patterns of activity can be mimicked by local light activation in the corresponding sensory cortical area.

The session concluded with the realization that the community must start viewing the barrel cortex as an integral part of a larger sensorimotor cortical network. Not only are there specific long-range

reciprocal connections between barrel cortex and motor cortex (as well as other higher sensory areas), but there are also non-specific local connections between primary sensory cortices of different modalities. Only when carefully considering these short- and long-range intracortical connections can the ultimate role of the barrel cortex in performing cortical computations be truly understood.

Following a brief coffee break the morning resumed with a talk by **Hongkui Zeng** (Allen Institute of Brain Sciences, USA) on the latest advances in the engineering of transgenic mouse lines. The speaker first reminded the audience of the importance of transgenic technologies as tools to dissect the function of specific cell types in complex circuits. The Allen Institute has created a cell-type selective mouse genetic toolkit system to facilitate the study of neural circuits. The toolkit system includes the generation of Cre-driver lines that target specific cortical cell types, Cre-responder lines with strong expression of fluorescent probes for neuronal tracing, calcium imaging (gCAMP3), and membrane proteins for photostimulation (Channelrhodopsin, Halorhopsin, and Arch). A set of corresponding AAV viral vectors will also be soon available, which can be used in conjunction with the transgenic mice. Experiments validating the functionality of these probes are still ongoing, but preliminary results suggest that investigators may soon be able to purchase lines of mice to study specific efferent and afferent pathways using optical means. Zeng also described the Mouse Brain Connectivity Atlas project, the aim of which is to create a whole-brain, mid-to-high-resolution, three-dimensional map of axonal projections in the mouse brain. The atlas will be created primarily using genetic tracing approaches that utilize a variety of cell-type specific Cre lines and Cre-dependent viral tracers. Both the raw data as well as the 3-D models will be presented on a public online database to create 2-D and 3-D searchable interactive tools and is hoped to be completed by the end of 2012.

The morning session concluded with a quick data blitz session moderated by **Randy Bruno** (Columbia University, USA). **Joshua C. Brumberg** (Queens College, CUNY, USA) presented a recent study investigating the physiological properties of two types of corticothalamic (CT) neurons: (1) the CT-VPM neurons and (2) the CT-POM neurons. CT neurons are identified by injections of retrograde tracers into either VPM or POM. CT-VPM neurons are typically located in L6 of barrel cortex while CT-POM neurons are found in L5b. Based on action potential (AP) half-width measured by patch-clamp recordings of retrogradely labeled neurons in thalamocortical slices, CT-VPM neurons have broader APs compared to CT-POM neurons. Both cell types receive thalamic inputs. In response to a pair of thalamic stimulations, CT-POM neurons showed response facilitation while CT-VPM neurons showed neither facilitation nor depression. Similarly, in response to a train of eight pulses, CT-POM neurons continued to show response facilitation, and CT-VPM neurons showed clear response depression. **Vincent Jacob** (Cardiff University, Wales) finished the data blitz session by showing sensory deprivation can differentially affect subtypes of pyramidal neurons in L5 of barrel cortex. After single-row whisker deprivation for 10 days in 6-week-old rats, responses to spared whisker potentiated in L5 regular spiking (RS) neurons but not in L5 intrinsically bursting (IB) neurons.

After lunch, the afternoon session started with a series of short talks moderated by **Jason Ritt** (Boston University, USA). The series started off with **Gaute T. Einevoll** (Norwegian University of Life Sciences, Norway) who created a thalamocortical network model of the barrel column using population firing data. Multi-unit activity (MUA) and local field potential (LFP) data were recorded with laminar electrodes in the barrel cortex, and MUA recordings were obtained with a single electrode in the thalamus. Responses of both thalamic and cortical neurons to a single-whisker stimulus with varying velocity and amplitude were recorded. Firing rates extracted from MUA recordings were in turn used to identify population firing-rate models. He found that the thalamus to L4 connection was best fitted by a model with fast feed forward excitation from VPM combined with a slower inhibitory process due to feed forward and/or recurrent connections. The model predicted that the thalamocortical circuit is optimally stimulated by rapid changes in the thalamic firing rate. Information transfer within the cortical column, on the other hand, was better fitted with a simple feed forward population firing-rate model combined with linear or mixed linear–parabolic activation function, which predicts that the intracortical circuits are low-pass filters that respond best to slow-varying inputs from cortical L4 neurons. Next **Robert Sachdev** (Yale University, USA) discussed the VSD response evoked in mouse barrel cortex by single-whisker stimuli. He showed that in urethane anesthetized mice, single-whisker stimulus elicits a wave of depolarization. A longer whisker stimulus caused a slower rise in the VSD signal and a persistent depolarization because the on-response is blending with the off-response. The persistent depolarization cannot be mimicked by a pair of whisker stimuli. Paired whisker stimuli at short intervals (8 ms) do not generate a second response but only evoke a larger spread of the initial cortical depolarization. At longer intervals (100–200 ms), the

second stimuli produced a VSD response that is slower to rise and slower to decay, but do not spread as far as the initial response. Lastly, the extent of the area of activation observed by VSD imaging is not dependent on direction of the whisker stimuli. Finally, **Alison L. Barth** (Carnegie Mellon University, USA) presented the work of her laboratory developing an unbiased method to assess the firing activity of individual neurons in the barrel cortex. They used a fos-GFP transgenic mouse to examine the properties of cells with a recent history of elevated activity. Intriguingly, their investigation has shown that neuronal responses are extremely heterogeneous *in vivo*. In barrel cortex, only layer 4 neurons and a very small proportion of layer 2/3 neurons show Fos-promoted GFP expression in response to sensory stimulation. *In vitro* patch-clamp recording of fos-GFP-positive neurons were not intrinsically more excitable than other neurons, but receive more synaptic inputs and are highly interconnected. She then performed dual *in vivo* targeted cell-attached recording, and demonstrated that fos-GFP-positive neurons spontaneously fire more and earlier than fos-GFP-negative neurons. Fos-GFP-positive neurons are also more likely to fire in bursts. Lastly, whisker-trimming did not affect the proportion of fos-GFP-positive neurons within the population.

The final session of the day focused on encoding and decoding of sensory inputs in the whisker–barrel system and other primary sensory cortical areas. **David Golomb** (Ben Gurion University, Israel) introduced the session by reminding the audience of the difficulties of interpreting sensory-stimulus-evoked physiological data, and the existing debates over the basis of cortical neural coding.

Garrett Stanley (Georgia Institute of Technology, USA) started off the session by discussing how adaptation affects thalamocortical transformations of peripheral somatosensory information. Using simultaneous single-unit recordings in the VPM and the barrel cortex in anesthetized rats, they discovered that adaptation differentially influences the thalamus and cortex. In response to repeated whisker stimuli (12 Hz) of various velocities, L4 cortical neurons adapt strongly in response amplitude. Adaptation in turn increases the slope of the velocity–sensitivity curve (angular velocity vs number of APs in 30 ms window) of cortical neurons, making it easier to discriminate different stimulus velocities based on firing rate alone. VPM neurons, on the other hand, were shown to adapt to repeated stimuli, but less than cortical neurons. While the velocity–sensitivity curve of VPM neurons does not change in the adapted state, there is a significant decrease in synchrony between VPM neurons. This decrease in synchrony could account for the change in the slope of the velocity–sensitivity curve of cortical L4 neurons. The second part of the talk involved his more recent work involving observer analysis of VSD imaging of cortex and anesthetized rats. In other words, if an ideal observer was present could they determine the nature of the sensory input based on the observed VSD signal. The area of activation detected by VSD imaging increases with whisker stimulus velocity, therefore, for an ideal observer to discriminate between stimulation of two neighboring whiskers, there is an optimal stimulus velocity which would still generate significant VSD signal but also minimal amount of spread outside of the stimulated barrel allowing for discrimination.

Next, **Brent Dorion** (University of Pittsburgh, USA) presented several strategies by which a neuronal network could be used to change output correlations when there is shared input correlation. Using a model of a network of neurons sharing a common input source, and several non-shared noise sources, he demonstrated that the background noise level of a neuron can change the firing-rate gain control of the cell, which in turn changes the output noise correlation of the neurons. He further showed that with high background noise, the output correlation of the network showed higher synchrony with a shorter window of analysis (3 ms) but lower synchrony with longer window of analysis (over 10 ms). *In vitro* recording from pairs of cortical L2/3 neurons while injecting different levels of noisy conductances using a dynamic clamp confirmed this finding. A second strategy is the recruitment of feed forward inhibitory circuits with higher input amplitudes, which could create a negative image of the input signal, which in turn decreases the amount of output synchrony of the principal neurons. Dorion further demonstrated that this strategy has been utilized by the electrosensory system of the weakly electric fish.

The meeting was concluded with a talk by **Ken Harris** (Imperial College London, UK) focused on the sensory-evoked population response of auditory corticalneurons. Using extracellular multi-unit recordings (MUA) from A1 of rats, he found that the population responses of A1 neurons to a single tune resembled spontaneous up-like events which occur in the absence of sensory stimuli. Both spontaneous and evoked events exhibited sparse, spatially localized activity in L2/3 pyramidal neurons, and densely distributed activity in larger L5 pyramidal neurons and putative interneurons. The only difference between spontaneous and evoked events was their laminar propagation: spontaneous events initiated in the deep layers and propagated upward while evoked activity initiated in the thalamo-recipient layers and then propagated throughout the column. In both un-anesthetized and urethanized

rats, global activity fluctuated between a ''desynchronized'' state characterized by a low-amplitude, high-frequency LFP and a ''synchronized'' state of larger, lower frequency waves. Computational studies done in his lab suggested that cortical neuronal responses could be modeled by a simple dynamical system model fitted to the spontaneous activity immediately preceding stimulus presentation. This result reflected a nonlinear self-exciting system in synchronized states and an approximately linear system in desynchronized states.

After 2 days of stimulating talks, posters, and discussions the 23rd annual Barrels meeting drew to a close with a commitment to reconvene in a year's time in Baltimore, MD. The meeting once again confirmed the utility of the barrel system as a model to study not only cortical development and functioning, but also as a model to study neuropathology and developmental disorders.

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Appendix 1. Barrels XXIII schedule

Thursday, 11 November	
9:00–9:05	**Welcome** Joshua C. Brumberg, *Queens College, CUNY*
	Introduction to local area David Kleinfeld, *UCSD*
9:05–11:15	**Development Gone Haywire**
9:05–9:15	Introduction/overview **Fan Wang**, *Duke University*
9:15–9:45	**Sacha Nelson**, *Brandeis University, USA*
	Physiological genomics of Rett syndrome
9:45–10:15	**Andreas Frick**, *University Bordeaux 2, France*
	Pathophysiology of information processing in neocortical circuits in Fragile X syndrome
10:15–10:45	**Jochen Staiger**, *Georg-August-Universität, Göttingen, Germany*
	Remodeling of (thalamo-)cortical circuits in reeler mice showing severe neuronal migration defects
10:45–11:15	Discussion
11:15–11:45	Coffee Break
11:45–12:45	**Short Platform Talks 1**
	Moderator: Arthur Houweling, *Erasmus MC, the Netherlands*
11:45–12:00	D. Deutsch[1*], M. Pietr[1], P. M. Knutsen[2], E. Ahissar[1] and E. Schneidman[1]
	[1]Weizmann Institute of Science, Rehovot, Israel, [2]University of California, San Diego, USA
	Closed loop whisking: effects of contacts
12:00–12:15	Avraham Saig*, Amos Arieli and Ehud Ahissar
	Weizmann Institute of Science, Rehovot, Israel
	Improving perceptual accuracy via motor and not sensory modifications
12:15–12:30	Jason Ritt[1*], Jason Wolfe[2], Mitra Hartmann[3], Ehud Ahissar[4], Michael Brecht[2] and Christiaan de Kock[5]
	[1]Boston University, [2]Humboldt University, Berlin, [3]Northwestern University, [4]Weizmann Institute of Science, [5]VU University, Amsterdam
	Establishing community standards for measurement and reporting of whisker behaviors and their neural correlates
12:30–12:45	Discussion
12:45–12:50	Business Meeting (Joshua C. Brumberg, *Queens College, CUNY*)
12:50–2:30	Lunch Break
2:30–3:45	**Short Platform Talks 2**
	Moderator: Karel Svoboda, *Janelia Farms*
2:30–2:45	Tomohisa Toda, Itaru Hayakawa and Hiroshi Kawasaki*
	The University of Tokyo, Japan
	The role of birth in somatosensory map formation in the mouse barrel cortex
2:45–3:00	Z. Zhang* and Q.-Q. Sun
	University of Wyoming
	A direct measure of E/I balance across different layers of somatosensory cortex during postnatal development
3:00–3:15	Mehdi Pedramfard, Manek S. Aulakh, Jong J. Park, Patrice D. Carr, Michael V. Johnston, Mary E. Blue and Mary Ann Wilson*
	Johns Hopkins University School of Medicine
	Effect of lead exposure on dendritic spine development in rodent barrel cortex
3:15–3:30	Rodrigo O. Kujis
	Encephalogistics, Inc.
	The barrel cortex as a novel assay for selective vulnerability and sparing in Alzheimer's disease

(*continued*)

3:30–3:45 Discussion
3:45–4:00 Coffee Break
4:00–5:00 **Short Platform Talks 3**
Moderator: Qian-Quan Sun, *University of Wyoming*
4:00–4:15 Lu Li* and Daniel E. Feldman
Helen Wills Neuroscience Institute
A novel and rapid component of whisker response plasticity in rat somatosensory (S1) barrel cortex
4:15–4:30 Craig E. Brown*, Dani Sweetnam, Maddie Beange, Thomas Watson and Raad Nashmi
University of Victoria
A role for $\alpha 4$* nicotinic receptors in experience-driven cortical depression in adult mouse somatosensory cortex *in vivo*
4:30–4:45 Peter W. Hickmott[1*] and Hubert Dinse[2]
[1]*University of California, Riverside,* [2]*Ruhr University Bochum, Germany*
Characteristics of thalamocortical EPSCs and IPSCs in hindpaw S1 in adult and aged rats *in vitro*
4:45–5:00 Discussion
5:00–5:30 Break
5:30 **Poster Session**
7:00 Dinner

Friday 12 November
9:00–11:00 **Beyond the Barrel**
9:00–9:10 Introduction/overview **David Kleinfeld**, *UCSD*
9:10–9:40 **Carl Peterson,** *EPFL, Switzerland*
Cortical sensorimotor integration
9:40–10:10 **Ron Frostig**, *University of California Irvine*
Activity spread beyond the barrel as a general model for mammalian sensory–motor cortex activation
10:10–10:40 **Tim Murphy**, *University of British Columbia, Canada*
Imaging widespread consensus patterns within spontaneous and sensory-evoked cortical depolarization *in vivo*, implications for normal function and recovery after injury
10:40–11:00 Discussion
11:00–11:15 Coffee Break
11:15–12:00 **Emerging Techniques**
11:15–11:30 **Hongkui Zeng**, *Allen Institute for Brain Sciences, USA*
Mouse genetic toolkit for probing neural circuits and mapping connectivity
11:30–11:45 Discussion
11:45–12:30 **Data Blitz**
Moderator: Randy Bruno, *Columbia University*
12:30–2:00 Lunch
2:00–3:00 **Short Platform Talks 4**
Moderator: Jason Ritt, *Boston University*
2:00–2:15 Gaute T. Einevoll[1*], Patrick Blomquist[1], Anna Devor[2,3], Ulf G. Indahl[1], Istvan Ulbert[4] and Anders M. Dale[3]
[1]*Norwegian University of Life Sciences, Norway,* [2]*Massachusetts General Hospital,* [3]*University of California, San Diego,* [4]*Hungarian Academy of Sciences, Budapest, Hungary*
Estimation of thalamocortical and intracortical network models for the rat barrel system from multielectrode recordings
2:15–2:30 Robert Sachdev[1], Douglas Davis[1,2] and Vincent Pleribone[1,2*]
[1]*Yale University School of Medicine,* [2]*The John B. Pierce Laboratory*
Traveling waves evoked in mouse barrel cortex by single whisker stimuli
2:30–2:45 L. Yassin*, B. L. Benedetti, J.-S. Jouhanneau, J. Wen, J. F. A. Poulet and A. L. Barth
Carnegie Mellon University
Unbiased methods to assess the firing activity of individual neurons in the neocortex have revealed that a large proportion of cells fire at extremely low rates
2:45–3:00 Discussion
3:00–3:15 Coffee Break
3:15–5:15 **Encoding and Decoding Sensory Inputs**
3:15–3:25 Introduction/overview **David Golomb**, *Ben Gurion University*
3:25–3:55 **Garrett Stanley** *Georgia Institute of Technology*
Decoding the thalamocortical circuit
3:55–4:25 **Brent Dorion** *University of Pittsburgh*
Long-term plasticity and sensory response modulation
4:25–4:55 **Ken Harris** *Imperial College London*
How do neurons work together? The view from auditory cortex
4:55–5:15 Discussion
5:15 Adjourn

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* indicates who is giving the presentation.