WORKSHOP ON
DS GANGLION CELL
6-8 October 2003 [By invitation only]

Supported by The Gatsby Foundation

Venue: **Seminar Room B10,** Gatsby Computational Neuroscience Unit, University College London, 17 Queen Square, Alexandra House, London WC1N 3AR
See map at http://www.gatsby.ucl.ac.uk/travel/index.html

Sunday evening (5 October) 6:00- 8:30pm
An informal welcome party will be held at the Gatsby Unit for Computational Neuroscience, Alexandra House, Queen's Square. Drop in if you can

We hope the moderators will take an active part in promoting discussion of the talks in their sessions and will explain their own views, with overheads if appropriate. The times indicated for each session include discussion and moderator's remarks. Names given are those of the presenter – all authors names are on the abstracts.

Monday Morning 9:00 – 12:30
Moderator Peter Sterling
9:00-9:10 - Welcome from Peter Dayan and Frank Werblin
9:10-9:50 - Horace Barlow: *Image motion and what you need to detect it*
9:50-10:30 - Bill Levick: *Two remarks on the interpretation of responses*

10:30-11:00 Coffee Break

11:00-11:45 - David Vaney: *Direction Selectivity in the Retina: Miracles and Mechanisms*
11:45-12:30 - *Moderator Frank Werblin:* Discussion: What are the key Open Questions?
Questions from Werblin, Euler, Barlow, Merwine, Denk , Levick and Massey are given at the beginning of "Abstracts"

12:30-2:00 Sandwich Lunch

Monday Afternoon 2:00 – 6:00
Moderator: Richard Masland DS Cell Studies
2:00-2:45 - Shelley Fried *Directional suppression of excitation and inhibition arriving at DS cells*
2:45-3:30 - Thomas Münch: *Cholinergic interactions between starburst cells and DS cells*

3:30-4:00 Tea Break

4:00-4:45 - Rowland Taylor: *The role of excitation in generating direction-selectivity in rabbit retina*
4:45-5:30 - Ray Dacheux: *Synaptic Inputs to DS Ganglion Cell*
5:30-6:15 - Lyle Graham: *What Synaptic Conductance Dynamics Tell Us about Directional Selectivity*

Evening free

Tuesday Morning 9:00 – 12:30

Moderator:Stephen Massey ST Cell studies
9:00-9:45 - Winfried Denk: *Combining 2-photon microscopy and light stimulation for retinal research*
9:45-10:30 - Thomas Euler: *Velocity tuning and active properties of starburst cell dendrites*

10:30-11:00 Coffee break

11:00-11:45 - Edward Famiglietti: *Functional Architecture of Directional Selectivity in the retina*
11:45-12:30 - Shigang He: *Co-fasciculation between ST amacrine cells and dendrites of ON-OFF and ON DS ganglion cells*

12:30-2:00 Sandwich Lunch

Tuesday Afternoon 2:00 – 5:45

Moderator: John Lisman DAPI III cells and Constraints on DS processing
2:00-2:45 - Stuart Mangel: *Chloride co-transporters mediate direction selectivity of starburst amacrine cell dendrites in the rabbit retina.*
2:45-3:30 - Charles Zucker: *Defining interactions with starburst amacrine cells: choline uptake transporter, $GABA_B$ receptors, muscarinic receptors and DAPI-3 cells*

3:30- 4:00 Tea Break

4:00-4:30 - David Merwine: *The responses of the DS-cell ensemble to "natural" stimuli*
4:30-5:00 - Dick Masland: *Responses of DS cells to (slightly) more natural stimuli*
5:00-5:30 - Frank Amthor: *Spikes in DS ganglion cells: what do they mean, where do they come from?*
5:30-6:00 - Simon Laughlin: *Placing models in their context – neural systems that extract useful information from image motion*

7:30 Workshop Dinner at

Ciao Bella Restaurant, 86-90 Lamb's Conduit Street, London WC1N 3LZ - T: 020 7242 4119

Wednesday Morning 9:00 – 1:30

Moderator: Peter Dayan Models of DS
9:00-9:45 - Robert Smith: *Morphology generates robust direction selectivity in the starburst amacrine cell*
9:45-10:30 - Martyn Mahaut-Smith: *Control of cytosolic Ca^{2+} during cellular signalling*

10:30-11:00 Coffee break

11:00-12:30 - *Moderator: Frank Werblin* Discussion on Models and Open questions
If you have a topic to suggest and would be prepared to speak about it for 5 minutes or so, please inform Frank Werblin in the preceding coffee break - or earlier.

12:30 Sandwich lunch and farewells

ABSTRACTS AND OPEN QUESTIONS

OPEN QUESTIONS

Monday 6 October – Morning
11:45-12:30

Werblin

a. Is DS in starburst cell processes a property of the process or the network?
b. What is the role of ACh in DS processing?
c. Are the proposed mechanisms and circuitry adequate to account for the observed DS properties, or are there additional components that must be incorporated?
d. How are variations in speed, contrast, size, and surrounding movement accounted for by the circuitry?

Euler

a Where are the GABA release sites in starburst cells? What is the GABA release mechanism?
b How and for what does the brain use the DS signals from the retina?
c How does retinal DS vary in different species? Can we expect the same purpose/circuitry e.g. in mice, rabbits and men?

Barlow

a What are the metabotropic glutamate receptors doing on the starburst amacrines?
b How do ON starbursts and DS ganglion cells differ from ON-OFF types?
c Any further ideas about how ΔS and ΔT (particularly ΔT) are mediated?
d What happens at a co-fasciculation?

Merwine

a How can null-side inhibition be so much larger than a starburst amacrine cell RF (particularly given that preferred-side facilitation measures ~1/2 a starburst RF, as predicted)?
b How does the DS ensemble respond to natural stimuli?
c Do DS cells contribute to any visual modality other than OKN?
d How does DS develop? (In particular, evidence in turtle shows that DS does not form until >30 days post-hatching, yet in rabbit DS responses can be recorded prenatally and DS cells with the same preferred direction are gap-junctionally coupled within a few days of eye-opening).
e How are the 4 preferred axes determined for On-Off DS cells?
f Why are On starburst ACs displaced to the GCL?

Denk

Wiring, development, is SBAC->DSGC push-pull or not?

Levick

a How is the He & Masland (Nature, 389, 378-382,1997) result on laser ablation of starburst amacrines to be explained if direction-selectivity in the peripheral dendrites of starburst amacrines is solely responsible for the direction-selectivity of ganglion cells?
b Has anyone tried to mark the neurons making inhibitory synapses on a single DS ganglion cell or starburst amacrine by using direct microinjection of LacZ-linked tetanus toxin into the cell body of either of the latter? (See: Coen et al., PNAS, 94, 9400-9405, 1997; Li et al., J. Biol. Chem. 276, 31394-31401, 2001).

Massey

Much data and many models suggest there must be asymmetric connections between Cholinergic amacrine cells and DS GCs. If there are asymmetric connections between these cells why can't we see them?

ABSTRACTS - [In programme order]

Monday 6 October - Morning

9:10-9:50
Horace Barlow
Image motion and what you need to detect it

I shall talk about three aspects of the problem of directional selectivity in the retina; 1) a brief account of the phenomenon itself, the types of DS retinal ganglion cells, and what becomes of the information they provide; 2) a rather general account of the changes in the image that motions cause, since these have implications for the mechanisms that detect them; and 3) a brief recap of how Bill and I thought it worked 40 years ago, partly because some of the properties that seem important for understanding the mechanism have been forgotten, partly for a good laugh at how wrong we were on some aspects, and partly to show that genuine progress has been made. I shall also outline a more recent model some aspects of which are still in the running

9:50-10:30
W. R. Levick
Two remarks on the interpretation of responses

The intention behind the first remark is to provoke some discussion about the adaptational state of the in vitro retina preparation. There does not appear to be much information about the effects of stepping the background to darkness. The adaptational characteristics of such a change may have significant spatiotemporal overlap with signals associated with directional inhibition in particular experimental configurations. This may indicate a need to explore ways of disentangling the respective processes. The second remark raises the issue of how to assign the components of motion responses to particular spatial locations. The problem is that a response elicited at a particular instant has latency and duration. So when a stimulus moves, the profile of response is no longer congruent with the spatially fixed sensitivity profile that gave rise to it. Yet it is the latter that is required for correlation with the structural layout of the participating morphological components.

11:00-11:45
David Vaney
Direction Selectivity in the Retina: Miracles and Mechanisms

Recent studies support a model of direction selectivity in which starburst dendrites pointing in different directions provide the null-direction inhibition to different subtypes of direction-selective ganglion cells (and perhaps also to the bipolar cells that drive them). This model is very attractive both for its elegant simplicity and for the insight that the beautiful symmetry of the starburst amacrine cell is a necessary prerequisite for generating the asymmetric responses of the ganglion cells. However before the model is canonized it deserves the scrutiny of a Devil's Advocate. David Vaney will take the audience on a graveyard tour of direction selectivity, looking at the monuments to the great, the unmarked plots of the still born, and the skeletons buried by both sides. Not for the faint hearted!

Monday 6 October – Afternoon

2:00-2:45
Shelley Fried, Thomas Münch, Frank Werblin
Directional suppression of excitation and inhibition arriving at DS cells

Recent studies have shown that both the excitatory and inhibitory inputs to DS cells are themselves directionally selective, suggesting that they are shaped at sites pre-synaptic to DS cells. Here we show that the inputs to DS cells become directionally selective because of asymmetric suppressive mechanisms. Inhibitory input becomes directionally selective because it is suppressed by activity on the preferred side of the cell while excitatory input is suppressed by activity on the null side of the cell. We used pharmacological dissection to identify several of the neural components along the synaptic pathways that mediate these suppressions.

2:45-3:30
Thomas Münch, Shelley Fried, & Frank Werblin
Cholinergic interactions between starburst cells and DS cells

Acetylcholine, released from starburst amacrine cells, has long been implicated in the generation of directionally selective responses. We analyzed the role of acetylcholine in shaping the inputs to DS cells, and found that it is not restricted to the excitatory pathway. We also found differences between the on and off systems. Direct recordings from starburst cells, combined with results from DS cell recordings, indicate how starburst cells might contribute to the overall DS circuitry.

4:00-4:45
Rowland Taylor
The role of excitation in generating direction selectivity in rabbit retina

Recent work has shown that both the inhibitory and excitatory inputs to DSGCs are directional. Excitatory inputs are thought to comprise cholinergic and glutamatergic components, from starburst amacrine cells and bipolar cells respectively. I will present results from experiments aimed at determining which of the excitatory components are directional.

4:45-5:30
Ray Dacheux
Synaptic Inputs to DS Ganglion Cell

A physiologically identified on-off directionally selective (DS) ganglion cell with its preferred-null axis defined was stained with horseradish peroxidase (HRP) and prepared for electron microscopy. A continuous series of thin sections were used to examine the cell's synaptology. Although the DS cell dendrite received the majority of its synaptic input from a heterogeneous population of amacrine cell processes, a frequently observed synaptic profile consisted of a DS cell dendrite receiving synapses from a cluster of several amacrine cell processes. These clusters of processes were assumed to be from a fascicle of amacrine cells, most of which probably belonged to several different cholinergic starburst amacrine cells. The most frequently observed presynaptic profile within the clusters consisted of a synaptic couplet in which two processes synapsed with each other before one of them finally synapsed with the DS ganglion cell dendrite; occasionally a chain of three serial synapses was seen. In addition, a specific microcircuit that has the potential to exert lateral feedforward inhibition was also observed. This microcircuit consisted of two cone bipolar cell terminal dyad synapses where one dyad contained an

amacrine cell process making a reciprocal synapse and a DS ganglion cell dendrite receiving direct excitation; the other dyad synapse, found lateral to the first dyad, contained two amacrine cell processes that both made reciprocal synapses, but one fed forward to make a putative inhibitory synapse with the DS cell dendrite.

Monday 6 October – Afternoon

5:30-6:15
Lyle J. Graham
What Synaptic Conductance Dynamics Tell Us about Directional Selectivity

The essential synaptic characteristics underlying the response of a direction selective (DS) neuron depend on whether excitation or inhibition (or both) is already DS, or whether there is a crucial direction-dependent correlation between excitation and inhibition. These alternatives are not mutually exclusive, and they can be evaluated by measuring the dynamics of evoked synaptic conductances, in contrast to the more classically measured synaptic potentials or synaptic currents. I will describe the methodology used to estimate these dynamics, which in particular allows a quantitative analysis of shunting inhibition. I will also describe whole-cell patch recordings of DS ganglion cells of the turtle retina - in the turtle at least, ganglion cell DS is due to an excitatory input that itself is DS, suggesting that the crucial inhibition implicated in this computation must act on cells presynaptic to the ganglion cell.

Tuesday 7 October - Morning

9:00-9:45
W. Denk, P.B. Detwiler, S.E. Hausselt, & T. Euler
Combining 2p microscopy and light stimulation for retinal research

When it comes to light stimulation conventional imaging techniques are of little use for retina research, because the excitation light interferes with the stimulus or even bleaches the photoreceptors irreversibly. With excitation wavelengths of about 930 nm multiphoton microscopy overcomes this limitation and allows optophysiological recordings of light-evoked responses at a high spatio-temporal resolution. In addition, fluorescent contrast staining can be used to visualize the tissue and greatly facilitate cell type selection and electrode positioning. In the talk, the design of a multiphoton microscope employing a miniature LCD display for light stimulation though the objective lens with will be presented.

9:45-10:30
S.E. Hausselt, P.B. Detwiler, W.Denk, & **T. Euler**
Velocity tuning and active properties of starburst cell dendrites

As previously shown circular wave stimuli not only evoke direction selective calcium responses in starburst cell dendrites but also asymmetrical voltage modulations in their soma. We used multiphoton microscopy and patch-clamp recordings to characterize these responses by varying stimulus parameters. We found that the responses show characteristic velocity tuning profiles. Further, somatic voltage responses to expanding and contracting motion differ not only in amplitude, but also in shape: during expanding motion the responses contain higher order frequency components that are absent during contracting motion. This suggests differential activation of voltage-gated channels by the two

motion directions. Interestingly, some non-starburst amacrine cells show similar somatic voltage responses, while others have no preference or even prefer contracting motion.

Tuesday 7 October - Morning

11:00-11:45
Edward Famiglietti
Functional Architecture of Directional Selectivity in the Retina

The anatomical organization of retinal neurons and their processes, likely to contribute to the mechanism of directional selectivity in rabbit retina, will be reviewed. The evidence to be examined will include that derived from light and electron microscopic study of starburst amacrine cells, directionally selective ganglion cells, and the cone bipolar cells that provide their excitatory drive. Circuits of excitatory and inhibitory neural connections that may explain the mechanism of directional selectivity in the retina will be considered in a 'bilayer' model, previously advanced (Famiglietti, 1993), and currently updated to accommodate recent experimental findings.

11:45-12:30
Shigang He
Cofasciculation between ST amacrines and dendrites of ON-OFF and ON DS ganglion cells

The relationship of DS cell dendrites and starburst cell processes were investigated. Dendrites of physiologically identified ON and ON-OFF DS cells were labeled with Neurobiotin and starburst processes were labeled with antibodies against VAChT. Both ON and OFF arbor of the ON-OFF DS cells and the ON arbor of the ON DS cells showed tight cofasciculation with starburst cell plexus, much higher than chance distribution and than the degree of cofasciculation of starburst plexus and dendrites of other types of RGCs. Analysis of different portions of dendritic arbors showed no statistical differences in degree of cofasciculation.

Tuesday 7 October – Afternoon

2:00-2:45
Stuart C. Mangel, Konstantin E. Gavrikov, Andrey V. Dmitriev, Kent T. Keyser, Tuesday am
Chloride cotransporters mediate direction selectivity of starburst amacrine cell dendrites in the rabbit retina

Because blockade of the Na-K-Cl and K-Cl cotransporters eliminates the directional responses of directionally-selective ganglion cells (Mangel et al., 2001, 2002; Gavrikov et al., 2003), we studied whether the chloride cotransporters mediate the directional responses of starburst amacrine cell (SAC) dendrites. Extracellular and whole-cell patch clamp recordings of DAPI-stained rabbit displaced SACs were obtained and the effects of blocking the chloride cotransporters assessed. The identity of SACs was confirmed with biocytin injections. We report that SACs depolarize and generate action potentials to stimuli that move from their somata to the periphery, but hyperpolarize to stimuli that move from the periphery to their somata. Moreover, the directional responses of SAC dendrites are highly sensitive to the polarity of their chloride gradients. Reducing the transmembrane chloride gradient by ion substitution or by blocking the K-Cl cotransporter with furosemide (25 microM) results in the SACs responding equally to light moving in opposite directions. Conversely, increasing the chloride gradient

by blocking the Na-K-Cl cotransporter with bumetanide (10 microM) eliminates responses to light moving in either direction although the cells still respond to stationary, flashing stimuli. These results indicate that the Na-K-Cl and K-Cl cotransporters play a key role in the generation of direction selectivity and suggest that the asymmetric distribution of the two cotransporters along SAC dendrites mediates their directional light responses.

Tuesday 7 October - Afternoon

2:45-3:30
Charles Zucker
Defining Interactions With Starburst Amacrine Cells: Choline Uptake Transporter, $GABA_B$ Receptors, Muscarinic Receptors and DAPI-3 Cells.

Starburst amacrine cells contain both acetylcholine and GABA, and are known to interact with ON-OFF directionally selective ganglion cells in such a way as to play prominently in the production of the physiological properties of these cells. Although there is still considerable debate, recent studies have suggested that coding of directional information may occur presynaptically to the ON-OFF directionally selective ganglion cell. Thus, a focus of attention is turning to the starburst amacrine cells themselves. Despite considerable efforts and progress toward understanding the function of starburst amacrine cells, the old adage that the more we know, the more we know we don't know, clearly holds true here. Basic features of starburst amacrine cell synaptic interactions are still poorly defined, as are their membrane receptor compliments. Additional classes of ganglion and amacrine cells, some of which may not be in direct synaptic contact with starburst amacrine cells, are also cholinoceptive. The nature of the interactions between starburst amacrine cells and their cholinoceptive targets has only been defined to a limited extent. Physiological and pharmacological data suggests that a glycinergic cholinoceptive amacrine cell type is involved in a $GABA_B$ mediated feedback loop with starburst amacrine cells that modifies the release of acetylcholine. We have shown that the so-called DAPI-3 cell is itself glycinergic and is cholinoceptive. In addition, we have also found that starburst amacrine cells, but not DAPI-3 cells, contain $GABA_B$ receptors. These $GABA_B$ receptors are localized to approximately 36% of boutons along the dendrites of individual Lucifer yellow-filled starburst amacrine cells. Because starburst amacrine cells are also thought to be GABAergic, our results suggest that their $GABA_B$ receptors function as presynaptic auto-receptors. Moreover, this $GABA_B$ receptor-localization pattern and the muscarinic-receptor localization on a bistratified glycinergic amacrine cell suggest an anatomical construct that may underlie a circuit involved in cholinergic modulation of retinal function. Since the majority of cholinergic boutons do not appear to express $GABA_B$ receptors, our data is supportive of the model proposed by Neal and Cunningham (1995) for a role of $GABA_B$ receptors in the facilitation of light-evoked acetylcholine release from the rabbit retina. We have also used an antibody directed to the hemicholinium-3 sensitive high-affinity choline uptake transporter to further explore the connectivity of starburst amacrine cells. Confocal microscopy of double-labeled filled cells shows that most labeling is restricted to dendritic spines and varicosities; virtually all of which are labeled. Only limited punctate labeling is seen on connecting processes. At the ultrastructural level, previously described connectivity (bipolar inputs, output to ganglion cells and other starburst amacrine cells) is readily observed. Additionally, we find that singleton profiles of starburst amacrine cells receive significant synaptic input from non-cholinergic amacrine cells. Though more limited, cholinergic synaptic output to non-cholinergic amacrine cell processes does occur at clusters of cholinergic processes. Further studies to define the identity of these synaptic inputs to, and targets from starburst amacrine cells will be needed in order to determine what constraints they may have on starburst amacrine cell contributions to retinal processing.

Tuesday 7 October – Afternoon

4:00-4:30
David Merwine and Norberto Grzywacz
The responses of the DS-cell ensemble to "natural" stimuli

Although DS cells are typically studied with high-contrast stimuli, statistics of natural images reveal that such stimuli are rare. We show that weak-stimulus responses carry significant information about motion direction, despite being more sensitive to noise. For instance, when preferred-direction motions produce a single spike on average, one can discriminate their responses from null ones in 80% of trials. Moreover, when considering the DS-cell population from a Bayesian perspective, low responses provide estimates of motion direction that are accurate within a few degrees. We show that glutamatergic bipolar synapses are necessary for sensitivity to weak motion signals. In turn, cholinergic synapses are necessary for directional selectivity under some of these conditions.

4:30-5:00
Richard Masland and C C Chiao
Responses of DS cells to (slightly) more natural stimuli

Many experiments have studied the behavior of the ON-OFF direction selective cell but most of them have focused on the possible mechanism of the directional discrimination. We have carried out a series of experiments designed instead to evaluate the overall behavior of the cells in response to complex stimuli, as a step toward understanding their role in a rabbit's natural vision.

In most of the experiments we used a standard, square wave grating stimulus to the receptive field center and varied the stimuli falling in the surround region of the receptive field center. As has been previously reported, extending the grating into the surround region reduces the cell's response, compared to the response to the center alone. However, if the center stimulus was out of spatial phase with the surround, this inhibition was substantially reduced. The same effect was observed for a stimulus consisting of a single long bar: phase shifting that portion of the bar that crosses the receptive field center increased the cell's response. If the spatial frequency, temporal frequency, or direction of movement of the surround stimulus was different from that of the center stimulus, the strength of the response to the center stimulus was preserved. Experiments using annular masks indicate that these effects are mediated by a local inhibitory subunit with a lateral extent of ~ 200 um.

These results may be summarized as suggesting that the ON-OFF DS cells are influenced not only by the presence or absence of movement in the surround (the requirement for "local motion") but by the higher-order characteristics of the surround stimulus and its relation to stimuli present in the center. They suggest that it will be important to test the responses of the ON –Off DS cells to natural scenes.

Tuesday 7 October 2003 - Afternoon

5:00-5:30
F. R. Amthor,
Spikes in DS ganglion cells: what do they mean, where do they come from?

Directionally selective (DS) ganglion cells exhibit greater spiking for preferred than null-direction motion. If, as generally assumed, these spikes are the result of a simple integrate and fire mechanism at the DS cell's soma, then the number of spikes primarily encodes the visual stimulus parameter of motion direction in the context of other visual parameters that affect the cell's discharge rate, such as contrast, velocity and stimulus pattern. However, recent evidence on both the origin of DS ganglion cell spikes and cooperative firing among DS ganglion cells suggests that spikes signal more than the one dimensional parameter of direction. Specifically, we have found evidence with respect to the origin of DS ganglion cell spikes that: (1) spikes in starburst amacrine cells may, under appropriate stimulus conditions, produce one-to-one spikes in DS ganglion cells, and (2) that active dendritic processes in DS ganglion cells, rather than the soma or initial segment, may provide the origin of some spikes, possibly translating spike inputs at distal dendrites from starburst amacrine cells into output spikes at the DS cell soma. We have also shown in multiple DS ganglion cell recordings that correlated firing (synchronous spiking) occurs in these cells that is stimulus dependent, and not obligatorily a product of gap junction coupling. Stimuli that are particularly effective in eliciting correlated spikes include large moving edges, even when such extended edges elicit smaller numbers of spikes than may be elicited by smaller moving spots. We hypothesize that one function of synchronous firing in these cells is to solve the aperture problem, by representing the fact that two cells have been stimulated by a congruent, extended edge in the pattern of their spiking outputs.

5:30-6:00
Simon Laughlin
Department of Zoology, University of Cambridge, UK
Placing models in their context – neural systems that extract useful information from image motion.

I review the status of the forerunner of modern models of movement detection, the correlation scheme deduced from observations of insect behaviour by Hassenstein and Reichardt in 1954. Their model has survived almost 50 years of intensive work on anatomy, molecular genetics, physiology, behaviour and information processing in the visual systems of flies. For technical reasons the cellular and molecular mechanisms responsible for motion detection by "Reichardt correlators" have not been identified, but circuits are on the horizon. The correlation model has been used to investigate the mechanisms that allow arrays of motion detectors to extract and code relevant signals under natural conditions. It has emerged that the correlation scheme is optimum for extracting motion signals from noise, and coding adapts to luminance. The conditioning of inputs to elementary detectors is important because the essential non-linear process, multiplication, easily saturates neural mechanisms. High-pass filtering of inputs, and subtractive and multiplicative mechanisms for adaptation and gain control reduce this problem. The fact that some of these adaptation mechanisms are sensitive to motion, irrespective of direction (i.e. speed not velocity) helps neurons extract ego-motion from optic flow. When it comes to coding image speed, the correlation scheme is not as bad as you might have thought. Filtering and adaptation enable neurons to extract reasonable measures of speed from natural images by reducing the sensitivity of correlators to contrast and spatial frequency. In summary, the application of Hassenstein and Reichardt's model to insect vision has demonstrates two points. First, molecular and cellular mechanisms for detection do not function in isolation. The neural coding of motion depends upon the properties of the circuits within which detection is embedded. Second, to understand the function of

these circuits, one has to know the natural inputs, the strengths and weaknesses of the elementary mechanisms and the purpose for which information on motion is extracted.

Wednesday 8 October – Morning

9:00-9:45
Robert G. Smith, Jan J. Tucker, and Rowland Taylor
Morphology generates robust direction selectivity in the starburst amacrine cell

In a compartmental model of a starburst amacrine cell that includes the cell's morphology, passive membrane properties, and appropriate synaptic inputs, wefound robust DS. We found that the addition of Q-type channels (thought to bepresent in the SBAC) can threshold the DS signal at the starburst dendritictips and amplify it, suggesting a mechanism for the DS measured in synaptic inputs to the DSGC. We have explored the DS behavior in the starburst for different stimuli and have identified what features of the cell's morphology and circuit are necessary to generate DS.

9:45-10:30
Martyn P. Mahaut-Smith
Control of cytosolic Ca^{2+} during cellular signalling

Ca^{2+}-induced Ca^{2+} release has been suggested to provide the basis for directional selectivity in neurons of the visual system (Barlow, Comp.Neur.Sys. 1996, 7, 251). Intracellular Ca^{2+} is certainly a versatile and ubiquitous second messenger involved in a wide range of physiological processes such as secretion, fertilisation, haemostasis, gene expression and contraction. Can Ca^{2+} mobilisation also account for directional selectivity? This talk will discuss the properties of different types of intracellular Ca^{2+} stores, including modulation by IP_3, Ca^{2+} and other cytosolic signals. The multiple mechanisms, both novel and emerging, by which intracellular Ca^{2+} stores can interact with the cell membrane potential will also be described.