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19. ABSTRACT (Continue on reverse if necessary and identify by block number) This four week course was offered for the second time at the Marine Biological Laboratory in Woods Hole, MA. Twenty students were selected from a pool of 52 highly qualified applicants. Students who were prepared in both computer science and neuroscience were selected because they could benefit immediately from the high level discussion without much additional training in either discipline. The course had two lectures per day plus a laboratory wherein students worked with GENESIS, the simulation software developed at Caltech. Once familiar with GENESIS, students undertook a simulation project. The reports of this project are attached. <i>Keywords: Neurology, computer education, Neurophysiology, computer program, Teaching methods, (EDC)</i>															
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MARINE BIOLOGICAL LABORATORY
Methods in Computational Neuroscience
August 6 - September 2, 1989
Course Report

James M. Bower and Christof Koch, Co-Directors
Computation and Neural Systems Program
California Institute of Technology
Pasadena, California

APOSR-TR. 89-1704

This four week course was offered for the second time at the Marine Biological Laboratory in Woods Hole, MA. Twenty students were selected from a pool of 52 highly qualified applicants. Students who were prepared in both computer science and neuroscience were selected because they could benefit immediately from the high level discussion without much additional training in either discipline. ~~Nine of the twenty accepted students were not residents of the U.S.A.~~

The course had two lectures per day plus a laboratory wherein students worked with GENESIS, the simulation software developed at Caltech. Once familiar with the GENESIS, students undertook a simulation project. ~~The reports of these projects are attached.~~ In a departure from last year, we scheduled six tutorials in the afternoon, lasting between 2 and 3 hours with a single lecturer. The tutorials covered very technical material in great detail e.g. the simulator GENESIS, Hodgkin-Huxley, numerical techniques, phase-space analysis. We feel that these were beneficial and plan to increase the number of tutorials.

The relationship of this course to a workshop on "Computational Neuroscience" organized by Terry Sejnowski was changed from last year when students attended these sessions. In 1989, a number of participants in this workshop lectured in the course and students who were interested in the subject matter attended workshop meetings. This change was successful and enabled the course curriculum to expand in scope to include more higher-level models. A participant list is attached.

A very successful one-day workshop on the "Biophysics of Computation" sponsored by the ONR and organized by Thomas McKenna of the ONR and Christof Koch was held at the MBL on August 12, 1989. Several of the course faculty participated in lively discussion with experts invited to explore the computational complexity of single cells. A participant list is attached.

Students and faculty were very enthusiastic about the course, about the quality of the lectures, the 25 excellent workstations (DEC 2100 and 3100) provided to the course by Digital Electronics Corporation, and the GENESIS software. The course assistants once again provided superb technical support to the Laboratory.

We feel confident that the course has a firm direction and that the Laboratory is working quite well. Nevertheless, we are constantly striving to refine the curriculum. In order to address the issues of concern to students we have asked them to comment on the course and to provide us with a written evaluation of their experience this summer. Based on these responses we have come to the following conclusions and suggestions for improvements for next year's course.

All the students were impressed by the variety and quality of the lectures (about 60 lectures *in toto*). In the space of four weeks, they received an extensive and diverse overview of all the themes and areas relevant to computational neuroscience. Given that the lectures lasted between 1.5 and 2 hours and that most of the lecturers were available for small group discussions after their presentations, students were exposed to an enormous amount of material and expertise. Students considered this aspect of the course to be of great value.

The four week duration of the course (an increase of one week from last year's three weeks) was an improvement and we will continue with this length of course in the future.

Much improved over last's years course was the existence of a a text: *Methods in Neuronal Modeling*, edited by Idan Segev and Christof Koch (MIT Press, 1989). This methodological handbook proved extremely useful to the students, since the authors of almost all of the chapters participated in our course. The situation with regard to textbooks will still be improved in the next year, due to the upcoming totally revised third edition of Gordon Shepherd's *The Synaptic Organization of the Brain* (Oxford University Press, 1990), which we plan to adopt as our secondary textbook.

However, a number of students remarked that the speakers overemphasized results, and in particular, results of their own research, instead of methods, assumptions, previous attempts at modeling which failed, etc. We take this criticism very seriously, because our course is primarily meant to teach methods and not so much results. We will thus coordinate carefully with next year's speakers, and try to impress upon them the need to stress both experimental as well as theoretical and computational methods.

Given the large number of students from an experimental background with an extensive knowledge of channels and biophysics, we will reduce the number of lectures devoted to ionic channels and basic biophysics (currently three 1.5 hour lectures) and add more lectures on perceptrons, learning, approximation, Bayesian estimation and other techniques of relevance to neural networks. A number of students explicitly requested additional lectures on these topics, because these subjects are new and not widely taught outside a handful of universities.

Finally, improvements in the use of GENESIS, our simulation software package are necessary if the course is to run smoothly. Our biggest obstacle is still complete documentation of this software even though members of Jim Bower's laboratory at Caltech wrote a 100 page manual specifically for the course. This document still, however, did not contain all the relevant technical information for running one own's code under GENESIS. This comes as no surprise given the power and versatility of GENESIS. Since one of us (Bower) is dedicating a significant amount of his effort toward improving GENESIS in order to release it to the general public, we feel this condition will stabilize by next year with the likely publication of a book-sized GENESIS manual.

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Marine Biological Laboratory
Computation at the Level of the Single Neuron
a one-day workshop held on August 12, 1989
sponsored by the Office of Naval Research

Participants:

Thomas McKenna
Office of Naval Research
Code 1142BI
800 N. Quincy St .
Arlington, VA 22217-5000

Paul Adams
Department of Neurobiology
SUNY, Stony Brook
Graduate Biology Building 576
Stony Brook, NY 11794

Thomas Brown
Department of Psychology
Yale University
P.O. Box 11A Yale Station
New Haven, CT 06520-7447

Barry W. Connors
Section Neurobiology
Division of Biology and Medicine
Box 6
Brown University
Providence, RI 02912

David A. McCormick
Section Neuroanatomy
Yale University School of Medicine
333 Cedar Street
New Haven, CT 06510

Peter C. Schwindt
Department of Physiology & Biophysics
University of Washington
Mail Stop SJ-40
Seattle, WA 98195

Jack Wathey
Salk Institute
P.O. Box 85800
San Diego, CA 92138

Idan Segev
Hebrew University

Lyle Borg-Graham
Department of Psychology
MIT, Room E25-201
Cambridge, MA 02139

Christof Koch
California Institute of Technology

1989 Workshop in Computational Neuroscience

Terrence Sejnowski, Director
Salk Institute
P. O. Box 85800
San Diego, CA 92138

Participants:

Ernst Poppel
Goethestr. 31
Institut Med. Psychol.
D-8000 Munchen 2
FRG

Dana H. Ballard
325 Yarmouth Rd.
Rochester, NY 14610

Pat Churchland
H52 Santa Helena
Solana Beach, CA 92075

Kevin Martin
Neurobiology Research Center
Volker Hall
UAB
University Station
Firmingham, AL 35294

Richard Durbin
Dept. Psychology
Stanford University
Stanford, CA 94305

Robert DeSimone
NIH
Bldg. 9, Room 1N107
Bethesda, MD 20892

Nikos Logothetis
MIT
E25-634
Cambridge, MA 02139

Pierre Baldi
JPL 198-330
California Institute of
Technology
Pasadena, CA 91109

Stan Schein
7 Upson Rd.
Wellesley, MA 02181-2233

John Allman
216-76
California Inst. of Technology
Pasadena, CA 91125

Alexandre Pouget
3803-A Miramar Street
La Jolla, CA 92037

Leif Finkel
Dept. of Bioengineering
University of Pennsylvania
220 So. 33rd St.
Philadelphia, PA 19104

Steven Zucker
338 Metcalf Ave.
Westmount, Quebec H3Z 2J3
CANADA

Richard A. Anderson
Dept. Brain & Cognitive Sciences
E25-236, MIT
Cambridge, MA 02139

David Van Essen
Biology Division, 216-76
California Inst. of Technology
Pasadena, CA 91125

Methods In Computational Neuroscience

Course Directors: James Bower and Christof Koch, Computation and Neural Systems Program, California Institute of Technology.

Lectures held in Whitman Auditorium weeks 1 and 2
Candle House Room 104 weeks 3 and 4

All lectures open to the MBL community

WEEK 1

Monday, August 7

- 9:15 am **JAMES BOWER**
Aims of the course; methods; requirements
CHRISTOF KOCH
Introduction to Computational Neuroscience
- 11:00 am **PAUL ADAMS**
Voltage- and Agonist-dependent ionic channels

Tuesday, August 8

- 9:15 am **IDAN SEGEV**
Introduction to cable theory; Rall's model of
neurons; $d^{3/2}$ law
- 11:15 am **CLAY ARMSTRONG**
Relating stochastic single channels to deterministic
macroscopic currents

Wednesday, August 9

- 9:15 am **PAUL ADAMS**
Hodgkin-Huxley nerve equations
- 11:00 am **MICHAEL MASCAGNI**
Numerical methods for neuronal modeling

Thursday, August 10

- 9:15 am **IDAN SEGEV**
Compartmental models of neurons; simulating
alpha-motoneurons

11:00 am **CHRISTOF KOCH**
Calcium dynamics; calcium dependent currents;
A typical vertebrate neuron: bullfrog sympathetic
ganglion cell

Friday, August 11

9:15 am **IDAN SEGEV**
Dendritic spines; anatomy; passive models; spines and
plasticity; spines and active currents

11:00 am **CHRISTOF KOCH**
Synaptic input; nonlinear interaction between synaptic
input; synaptic veto; NMDA receptors

WEEK 2

Monday, August 14

9:15 am **CHRISTOF KOCH**
Calcium diffusion; solving cable and diffusion equation
simultaneously; advanced non-linear cable theory

11:00 am **RUDOLFO LLINAS**
Bursting and oscillating cells: Purkinje cells and
cells in the inferior olive

Tuesday, August 15

9:15 am **DAN ALKON**
Learning in a small network: the seas snail *Hermisenda*

11:00 am **CHRISTOF KOCH**
The Hartline-Ratliff model of the *Limulus* lateral eye;
Recurrent and non-recurrent inhibitory networks

Wednesday, August 16

9:15 am **MARK NELSON**
Associative memory; Kohonen, Hopfield; associative
learning in *Limax*

11:00 am **CHRISTOF KOCH**
The correlation model of motion detection; physiology,
psychophysics and theory of motion detection in fly

Thursday, August 17

- 9:15 am **MARK NELSON**
Simulating neuronal networks on parallel computers
- 11:00 am **JAMES BOWER**
Parallel computer maps and brain maps

Friday, August 18

- 9:15 am **JAMES BOWER**
Oscillations in single cells, vertebrate and invertebrate
- 11:00 am **JAMES BOWER**
Neural networks for central pattern generators;
Tritonia and Getting model

WEEK 3

Monday, August 21

- 9:15 am **AVIS COHEN**
Lamprey spinal cord: early vertebrate locomotion
- 11:00 am **NANCY KOPELL**
Theory of neuronal oscillators in Lamprey
- 3:00 pm **JOHN HILDEBRAND**
The insect olfactory system

Tuesday, August 22

- 9:15 am **JAMES BOWER**
Olfactory Processing I
- 11:00 am **JAMES BOWER**
Olfactory processing II
- 3:00 pm **CHRISTOPH VON DER MALSBURG**
Developing the visual system: experiments and early theories

Wednesday, August 23

- 9:15 am **CHRISTOF VON DER MALSBURG**
Neuronal development: unsupervised learning algorithms
- 11:00 am **CHRISTOF KOCH**
The gradient model of motion detection; psychophysics
and theory of motion detection in primates

Thursday, August 24

- 9:15 am **JOHN RINZEL**
Phase-space analysis of Hodgkin - Huxley; Theory of
dynamical systems

Friday, August 25

- 9:15 am **DAVID ZIPSER**
Using learning algorithms to model cortical computations
- 11:00 am **CHRISTOF KOCH**
The Computational approach to vision; Marr's legacy;
Edge detection; zero-crossings and cortical cells.

WEEK 4

Monday, August 28

- 1:30 pm **RICHARD ANDERSEN**
Eye movements in primates: experiments and models
- 3:15 pm **DAVID VAN ESSEN**
The primate visual system

Tuesday, August 29

- 1:30 pm **RICHARD ANDERSEN**
How does cortex derive 3-D structure from motion:
theories, psychophysics and electrophysiology
- 3:15 pm **TONY ZADOR**
Memory I: Biophysics

Wednesday, August 30

- 1:30 pm **TERRENCE SEJNOWSKI**
Memory II: Networks

3:15 pm **CHRISTOF KOCH**
Oscillations in cortical systems: experiments
and theories

Thursday, August 31

1:30 pm **TERRENCE SEJNOWSKI**
Back-propagation as applied to shape-from-shading
and the oculo-motor system

3:15 pm **EDWARD ADELSON**
Motor perception and motion energy: relationships between
motion models

Friday, September 1

9:15 am **KEVIN MARTIN**
Cortical neurons

11:00 am **DAVID VAN ESSEN**
Detailed models of the visual system: orientation
selectivity

MARINE BIOLOGICAL LABORATORY
Methods in Computational Neuroscience Course
August 6 - September 2, 1989
Student Project Descriptions

● Project title: Phase Locked Loop based model for tactile decoding

Name/Institution: Ehud Ahissar, The Hebrew University, Jerusalem, Israel.

Project description:

Tactile stimuli can be regarded as a carrier frequency signal (which carries the texture "pixel" density) modulated by a low frequency signal (which carries the texture shape) through a Frequency Modulation (FM).

The model tries to apply to the brain the decoding scheme which is used in electronic FM receivers, in which the basic circuit is a Phase Locked Loop (PLL) circuit. The idea in this circuit is to "look" a local oscillator to the incoming carrier signal by a feedback loop in which the error signal carries the modulation signal.

The model includes one local oscillator neuron, one input neuron and 20 phase comparator neurons. Every neuron includes the following items:

1. full H.-H. spikes in the soma
2. one dendrite compartment with several excitatory and inhibitory synapses
3. one bifurcating axon

● Project Title: Single Tone Processing in the Cochlea, Cochlear Nerve, and Cochlear Nucleus.

Name/Inst.: Jeff Arle, Univ. of Connecticut Health Center Neuroscience Program, MD-PhD program

Project Description: Questions Addressed:

1. What is the population response of the 3 major cell types in the cochlear nucleus to single tone stimuli? (globular bushy cells, stellate cells, and fusiform cells).
2. Do these response properties reflect what is already known about single cell responses to single tones? Related measures include phase-locking, post-stimulus time histograms, and place coding.
3. It is known that there are many inhibitory synapses on the tertiary dendrites of the bushy cells. Although it is not yet clear as to their origin, the question can be asked whether these play any role in the functional properties of the cell.

The simulation involves 3 stages of processing:

1. The basilar membrane (BM) movement in the cochlea for single tone stimuli can be modeled to a good first approximation by the following equation: $z(x,t) = (r(x) \cdot \sin(2\pi \cdot w \cdot t + p(x)))$ where $z(x,t)$ is the amplitude of a point x on the BM at any point in time t , $r(x)$ is the maximum spike rate for this particular stimulus intensity and frequency w and $p(x)$ is the phase in π radians for the particular intensity and frequency w .

The spike rate and phase data were taken directly from population recordings from the auditory nerve in cats (Kim and Molnar, 1973). This equation will reproduce the appropriate temporal traveling wave of BM movement as it tunes to the stimulus frequency.

2. Output of the cochlear nerve fibers is approximated by comparing the fraction $z(x,t)/r(x)$ with a random threshold varying between 0 and $r(x)$ max. If the fraction is larger than the threshold then the fiber "fires" an action potential and adds a dead time of 0.5 msec. This type of cochlear nerve model is a close approximation to the actual spike rate data found in the literature.

3. There are 3 cell types simulated in the cochlear nucleus section of the model: globular bushy cells, stellate cells, and fusiform cells.

Each cell is composed of a soma (a non-linear modified MacGregor type), an axon, and three dendritic compartments. Each dendritic compartment has passive excitatory and inhibitory synaptic channels. There are 100 of each cell type, 100 cochlear nerve fibers, and 100 points composing the BM.

The model contains a total of 1200 compartments, 1800 synapses, 400 axons (including the cochlear nerve fibers) and a script code link to simulate the temporal movement of the BM as described above. The simulation, including extensive memory and graphics demands, models 100msec in ~2200 cpu seconds.

Results:

The simulation suggests several hypotheses, although results from the rapid development of a simulation such as this need much further validation. With regard to the initial questions being addressed the modeling can make the following points: 1) the population responses have both regular and irregular components to them, for each of the cells types. It is clear that spatially there is a definitive frequency representation along the Bm and that those tonotopic areas of the cell populations respond in a regular pattern of firing to the stimulus. Interestingly, off-best frequency areas that do respond, (due to the temporal and amplitude information in the traveling wave), respond with a more irregular pattern in their firing. Often these areas are out of phase and frequency with the best-frequency areas within the populations; 2) distinct cell response types can indeed be differentiated from the graphs of population coding, and although peri- or post-stimulus time histograms were not calculated, (which are the most common form of comparison to the literature), one can see that the data can be easily transformed to match such types of analysis. It appears that if this were done there would be a very good fit to the actual PSTH data. Bushy cells clearly phase lock to the stimulus frequency up to at least 1200 Hz, which is the highest frequency that was tested. Stellate cells show their characteristic 'chopping' behavior which may increase in frequency as the stimulus frequency increases, yet is not phase locked to the stimulus. Fusiform cells would appear to exhibit the buildup behavior that seems to be often associated with them; 3) the issue of tertiary dendritic

function in the bushy cells, as well as lateral inhibition in the AVCN stellate cells yielded the results that feedback inhibition from both populations of stellate cells on the tertiary dendrites of bushy cells had little or no discernible effect on their firing properties and that lateral inhibition in one population of stellar cells gave a very different pattern of activity than non-inhibited circuitry (see graphs). It is not clear what kind of information could be carried in such a diffuse firing pattern, though such a simple nearest neighbor circuit of lateral inhibition may not be mimicking the actual circuitry.

Beyond these conclusions, the fusiform cells seem to have a relatively uniform firing pattern that seems dependent on their tonotopic organization. Moreover, they may be acting as a temporal low-pass filter of the BM envelope, allowing the 'shape' of the envelope to pass on to higher auditory centers perhaps for use in complex auditory pattern recognition. Much more work needs to be done in the DCN, especially regarding the intrinsic circuitry, to make any further generalizations.

• Project Title: Simulation of the Electrolateral Line Lobe (ELL):
Descending Control of Electrosensory Processing.

Name/Institution: **Bradford O. Bratton**, Department of Zoology, The University of Oklahoma, Norman, OK 73072

Project description:

This project involved modeling the gain control mechanisms or feedback connections in the electrosensory system of weakly electric fish. The electro-sensory lateral line lobe (ELL), the first-order electrosensory processing station, receives major descending inputs in addition to afferents from the electroreceptors. The nucleus praeeminalialis (nP) is the principle source of this descending input to the ELL.

The ELL was modeled from known anatomical and physiological data as a four neuron group with inhibitory connections between cells. Each of the four cell types receive afferent input from the receptors onto their basilar dendrites. The four neurons modeled in the ELL are:

E-cells (or basilar pyramidal cells) with ascending projections to the nP, the Polymorphic cells which make inhibitory connections onto the other three ELL cells, Granular-1 cells which inhibit E-cells and Polymorphic cells and the Granular-2 cells and the Polymorphic cells extend into the dorsal molecular layer and ventral molecular layer (not modeled in this simulation at this time). Finally, the ascending E-cells project to two cell types in the nP (stellate and multipolar cells) and feedback onto the ELL ventral and dorsal molecular layers (believed to be the gain control part of this circuit).

The simulation takes into consideration only one simple network containing the four ELL cells with afferent input and simple nP output and feedback without consideration to the three ELL maps, other ELL celltypes or later inhibition.

Progress/Results:

The four cell types in the ELL were modeled as each having a basilar dendrite with both a Na and K channel and one afferent axon synapse from the receptor cell. One receptor input cell was used for the entire ELL group but with four axons in which the weight could be controlled from the T-receptor graph window. Both the E-cell and Polymorphic cells had apical dendrites onto the dorsal and ventral molecular layers (layers not modeled yet) and received inhibitory synapses from Granular-1 and Granular-2 cells.

Progress was made in getting this section of the ELL network to function in an appropriate fashion although the physiological data for comparison is not able to confirm the simulation at the level of Granular cell and Polymorphic cell behavior. The second section of the model which included the nP projection and its feedback connections to the ELL were not completed in Woods Hole due to time. This section might have made the simulation behavior more predictable from expectations of the physiological responses.

● Project Title}: Electrical Geometry of CA3 Pyramidal Cells in the Hippocampus

Name/Institution: Gilles **Laurent**, Cambridge University, United Kingdom; Patrick **Lynn**, University of Colorado, Boulder, Colorado.

Project description:

The aim of the project was to simulate the electrical behavior of an isolated CA3 pyramidal cell. The goal was to understand the effects of the distribution through the dendritic structure of active channels upon the overall integrative properties at the cell body.

Data informing the modeling effort include the following:

1. membrane currents from voltage-clamp data on CA1 cells. Outward currents: transient A current half inactivation at -80 mV, slope of the Boltzmann fit of the inactivation curve 7.5 mV, time constant of decay 30 ms, activation from -50 mV; delayed rectifier current, very slow inactivation with a time constant of 3 s; Calcium dependent K currents, (high threshold) -30 mV, (low threshold) -60 mV. There are known to be inward Calcium and Sodium conductances, whose distribution on the dendritic trees and the soma were parameters of investigation.
2. Electronic parameters were taken from Traub 1982, Neuroscience 7, pp. 1223-1242.
3. Response properties of CA3 cells to current pulses were found in Wong et al. 1979, PNAS 76, pp. 986-990.

Progress/Results:

Simulation specifications:

A single cell was simulated with a variable number of compartments, the types of conductances and their densities specifiable for each individual compartment. This description was read in from a file, and was generally limited to a small number of compartments. One new object, my-vdep-gate, was created to accommodate a broad range of rate functions, including one that was a function of voltage and intracellular Calcium concentration.

● Project title: Orientation selectivity in visual cortex.

Name/Institution: Reinoud Maex, University of Leuven, Belgium.

The problem: given an isotropical organization of LGN-cell receptive fields, what pattern of neuronal connections can produce, one synapse further, receptive fields which are asymmetrical in space (orientation selectivity, end-stopping) and time (direction and speed selectivity).

There are two possible approaches: one can start with an anisotropic input from OGN-cells onto cortical cells (Hubel and Wiesel model) and add, step by step, other connections (short-range inhibition and long-range facilitation, cross-orientation inhibition) in order to mimic physiological data.

Another approach is: lateral inhibition is an ubiquitous phenomenon in the cortex (experimentally and computationally), so let's see what the receptive fields look like in a layer of cells where there are minor spatial variations in the strength of the lateral inhibitory synapses. This can then lead to a study of the response to dynamic stimuli of a two-dimensional recurrent inhibitory network.

● Project title: Central Pattern Generator in Tritonia

Name/Institution: Jisoon Ism/Seoul National University, Korea.

Project description:

The sea slug Tritonia uses 14 neurons to generate escape swimming upon external stimulus. Those neurons can be classified into 5 functional groups.

The swimming pattern is simulated with 5 model neurons representing all functional groups. Each model neuron is capable of generating, transmitting, and receiving electrical pulses.

Progress/Results:

I started with 3 model neurons which are known to be more directly related than others to the motion of Tritonia. I reproduced the results of Silvie and Matt at Caltech who performed practically the same simulation before. Then I introduced 2 more neurons which indirectly control the swimming pattern.

My simulation indicates that these 5 neurons produce activity patterns which are closer to actual experimental recordings (e.g., a more "graceful" termination of the swimming motion) than 3 neurons alone do. I am presently working on an even more realistic simulation using all the 14 neurons.

Simulation specifications:

(e.g. number of components, simulation time, new objects)

Number of components: 5 neurons and 30 synaptic connections.

Simulation time: 4 minutes per simulation.

● Project title: Simulation of associative plasticity of the CA3 recurrent collaterals in the mammalian hippocampus.

Name/Institution: Dean V. Buonomano, University Texas Medical School, Houston, Texas

Project description:

Long-term potentiation has been identified at three different loci in the hippocampus: perforant path to dentate gyrus, mossy fibers to CA3, and in CA1. The mossy fiber to CA3 path undergoes a nonassociative form of LTP which is not dependent on NMDA receptors. Recently, however it has been demonstrated that the CA3 recurrent collaterals exhibit associative neural network. Due to the longstanding evidence of the importance of the hippocampus in memory, my interest was to attempt to simulate the CA3 region. The circuit was based on a layer of both excitatory and inhibitory elements. The excitatory elements receive external input and feedback onto each other. The inhibitory elements received excitatory input from the excitatory elements and provided local feedback inhibition onto the excitatory elements. The synapses of the excitatory elements onto themselves were plastic, implemented by using a NMDA (Hebbian) type mechanism.

Progress/Results:

The NMDA type plasticity in its simplest form permitted strengthening of the synapse between cells that were activated during the same

stimuli presentation. However, with subsequent input patterns, often the same group of neurons would become active and inhibit each others. Thus, there was too much overlap in the synapses used to represent a particular input pattern. This is not a surprising result, emphasizes the importance of an anti-Hebbian type rule in this system.

Simulation specifications:

(e.g. number of components, simulation time, new objects)

100 excitatory cells/100 inhibitory cells/25 input cells. Each cell consisted of passive conductances, excitatory and inhibitory synapses and an axon.

● **Project title:** Synchronous oscillations in V1: A possible role in the organization of receptive fields in V4.

Name/Institution: Ehud Zohary, Hebrew University, Jerusalem, Israel.

Project description:

Recently, a number of papers have been published (Gray et al., 1989, Eckhorn et al., 1988) where it was shown that cells in cat primary visual cortex tend to fire synchronously to a visual stimulus (as seen from the cross-correlograms) if and only if they share the same tuning properties (such as preferred orientation) and the distance between the two cells is smaller than a few mm. It also seems likely that such an effect could only be accomplished if there was a common input to these cells since local coupling between nearest neighbors is not robust enough to maintain such a linkage over such distances. There have been some attempts to connect these oscillations to perceptual phenomena such as figure-ground separation or conjunctive binding.

The project was designed to check the usefulness of such a design, first from the point of view of efficiency of information transmission. The main point is that synchronous firing is a useful property if many cells converge on some target neuron. Thus, even if the cross-correlation is weak (typically the synapse strength is 0.05), due to the degree of convergence synchronous input could drive the target cell while this would not be so with asynchronous inputs. (see Abeles, 1982). Note that this would be so if and only if a

visual stimulus that would be of the preferred orientation would be presented; furthermore, the weak strength of the oscillator ensures that the input cells would have a typically small receptive field.

The model consisted of:

25 cells (at V1) each having a membrane potential fluctuating around V_{rest} with a Gaussian distribution.

An oscillating neuron firing at 40 Hz with a weak synapse on each of the V1 cells.

A V4 target cell with a receptive field typically 25 times the input (v1) cells.

Project/Results:

The V4 cell fired in a much more steady fashion than any of the cells driving it did. This is obvious from the autocorrelation function that had a peak at 40 Hz. However, the exact parameters of the strength of the driving oscillator to each of the input units could be assessed only crudely and varied from cell to cell. This, however, leads to the conclusion that one should expect the autocorrelation function of neurons at higher visual areas to be stronger than what is apparent in V1. This can be tested experimentally in our laboratory.

Furthermore, this only strengthens the developing notion that these oscillations might have some significant meaning in the coding of perceptual phenomena.

● Project title: Sound Localization in Barn Owl (*Tyto alba*)

Name/Institution: Petra Leuchtenberg, University of Bonn, West Germany

Project description:

The barn owl uses interaural time difference for sound localization in the horizontal plane. The neuronal sensitivity to this disparity originates in the brainstem nucleus laminaris. Afferents from the

ipsilateral and contralateral magnocellular cochlear nuclei (CN), the first synaptic station in the brain's auditory system, interdigitate in the nucleus laminaris.

Intracellular recordings from these afferents show orderly changes in conduction delay with depth in the nucleus. These changes are comparable to the range of interaural time differences available to the owl. Thus the afferent axons act as delay lines and provide anatomical and physiological bases for a neuronal map of interaural time differences in the nucleus laminaris. (C.E.Carr and M.Konishi, PNAS, USA, Nov. 1988).

The temporal resolution of the coincidence detecting cells seems to be much higher than expected by an ordinary Hodgkin-Huxley cell.

Project description:

As a first approach, I simulated a pair of afferents (originating from two CN-cells) connecting to a single coincidence detecting cell (NL cell) by chemical synapses. The axonal delay lines as well as the NL-cell contain multiple Hodgkin-Huxley-compartments, to find out the resolution of this simple model. Playing with parameters, as distribution of channels, motive, strength and spatial distribution of synapses, I plan to find out significant contributors to the high temporal resolution of these neurons.

● Project title: Membrane properties in the photoresponse of Limulus retinular cells

Name/Institution: Eduardo Solessio, Syracuse University

Project description:

Intracellular recordings from retinular cells, the photoreceptor cells in Limulus eye, show that these cells respond with a series of potential fluctuations---often referred to as "bumps"---when stimulated with flashes of low intensities. At higher flash intensities the response is characterized by a fast transient at stimulus onset followed by a sustained depolarization.

In the late 60s Knight, Dodge and Toyoda proposed that "bumps" be considered as discrete events resulting from localized changes in membrane conductance induced by light. The total response emerged from the summation of "bumps". In this model "bumps" adapted with light intensity, turning smaller and faster with increasing intensity, thus explaining the transition between the "bumpy" and the sustained responses. Later recordings done "in situ" by other investigators demonstrated the existence of two types of fluctuations induced at low stimulus intensities: small potential fluctuations that corresponded closely with the "bumps" described above, and large potential fluctuations, perhaps due to a regenerative process. At high stimulus intensities the large potential fluctuations are observed only at onset (figure 1). To my knowledge, the nature of the large potential fluctuations has not been determined yet.

The purpose of the project was twofold. First, implementation of the receptor model proposed by Knight, Dodge and Toyoda with the addition of voltage gated channels to account for the large potential fluctuations and a "controlled" potassium conductance to account for adaptation effects. Second, interconnect several of the modeled receptors with gap junctions and observe the response of the receptors for different gap junction resistances. It is presently believed that the changes in sensitivity induced by the circadian clock might be mediated by modulation of the magnitude of electrical coupling between the receptors.

Note that this model does not deal explicitly with the photo-transduction process. Light effects are introduced as short, transient changes in the sodium conductance of the membrane. The course of the conductance changes was represented by an alpha function and the time between the conductance changes was derived from a random generator. The mean rate of the random generator could be varied. It was assumed that mean rate was related to light intensity.

Simulation specifications:

The receptor was implemented with a single RC compartment and the following channels included:

1. a Na channel controlled by the random generator as described above,

2. a K channel with a slow time course,
3. a voltage gated Na channel,
4. a voltage gated K channel.

The conductance of the K channel was controlled by the random generator, for a rough approximation of a K conductance that is controlled by Ca---the assumption made that the intracellular Ca concentration was related to the "bump" rate. The two voltage gated channels were implemented with the parameters corresponding to squid axon. The validity of this assumptions is discussed below.

Project/Results:

The model of a single receptor was implemented first. Tests were run for two specific membrane resistance values and several mean rates. From the results shown in figure 2 it may be concluded that:

1. Large potential fluctuations were initiated by fast Na conductance changes and not necessarily by reaching a threshold potential. This fact is not always obvious from intracellular recordings.
2. Low membrane resistance values are not compatible with the generation of large potential fluctuations.
3. Increase of the bump rate resulted in summation of the bumps and a suppression of the large potential fluctuations during the sustained depolarization phase. A large potential fluctuation was elicited at stimulus onset, this does not contradict actual recordings.
4. At very high bump rates the response tended to saturate.
5. Adaptation effects can be noted.
6. The model is robust in that it does not require a very fine tuning of the parameters to work.

For the second part of the project four receptor units were interconnected with resistances resembling gap junctions. The value of the resistances was varied and the computed response of each receptor unit was displayed. From the results in figure 3 it may be concluded that:

1. High coupling resistance values virtually uncouple the receptors.
2. Low coupling resistance values result in a synchronized response of the receptors. This effect is very obvious at the lower rates. In addition, the lower resistance introduced substantial loading, decreasing the amplitude of the resulting bumps and also the probability of eliciting large potential fluctuations.

Conclusions:

This simple model shows that some properties of the response of receptors can be attributed to the membrane characteristics.

The difference in response characteristics between excised and "in situ" eyes might be due to a difference in receptor resistance. In particular, combining the results of parts 1 and 2, it might be hypothesized that without efferent activity the gap junction resistance decreases and mutual loading attenuates the response.

The results obtained for coupled and uncoupled receptors give rise to new questions. Namely, if the receptors are thought to be uncoupled during the day, coupling at night does not seem to increase sensitivity, unless coupling is accompanied by another change, for instance, closing of the K channels that are dependent on calcium. Unfortunately there is no data regarding this point.

The results obtained were presented to Dr. Kaplan. In his opinion the large potential fluctuations are slower than those obtained in the simulation. He suggested that probably a calcium voltage gated mechanism would be closer to reality.

● Project title}: Local Interactions in the Globus Pallidus: a reproduction of functional interactions observed with extracellular recordings in the

behaving monkey.

Name/Institution: Dieter **Jaeger**, University of Michigan, Michigan.

MOTIVATION: To increase the understanding of functional properties of Globus Pallidus (GP) neurons and local interactions. The specific aim was to reproduce cross-correlation data from extracellular recordings in behaving primates.

METHOD: Three GP neurons were simulated on a DECstation 3100 using Genesis, a general purpose neural network simulator. The geometry and physiological properties of GP neurons were taken from the literature as much as possible. The spike trains of the simulated neurons were used to calculate interval histograms and autocorrelations for each neuron as well as cross correlation histograms between pairs of neurons.

RESULTS: The simulated neurons were made to fire spontaneously by elevating the resting membrane potential above the threshold for Hodgkin Huxley type active Na channels. This activity was modulated by random inhibitory inputs to the dendrites which acted through GABA A receptor like chloride channels. Exponential interval histograms were observed as a consequence. The neurons were now coupled through low resistance dendrodendritic gap junctions. This led to functional interactions between the neurons. The types of cross correlations observed were indeed very similar as those seen in the behaving monkey.

CONCLUSION: The simulation described shows a good agreement between simulated spike trains using a realistic neural model and spike trains recorded from behaving primates. It seems therefore feasible to explore the mechanisms of neural interactions underlying observed cross correlations with computer models of realistic neurons.

● Project title: Simulation of the Central Pattern Generator of the escape swimming in Tritonia.

Name/Institution: Sherif **Botros**/Massachusetts Institute of Technology, Cambridge, MA.

Project description:

The purpose of the project was to implement the neural circuitry of the CPG of swimming in tritonia. A three neuron model developed by Peter Getting was used using the same parameters. The models used for the different neurons were simplified models of the integrate and fire type. This is important from a practical point of view for two main reasons. First, the parameters to be estimated for the model were largely reduced which made the parameter estimation part much easier. Second, since there are no very fast or voltage dependent channels, the equations are much less stiff, and are easier to integrate using larger time steps. At the same time this simplified model of the neuron was adequate to capture the important features exhibited by real neurons.

I was also interested in examining what is the effect of the different parameters on the behavior of the model, since biological neurons of the same type could be substantially different but the overall emergent properties of the network are similar and very robust. Also we could sometimes remove part of the circuit, without affecting much its behavior.

Progress/Results:

I was able to reproduce Peter Getting's simulations using the same parameters (see attached figures). The system seemed to be very redundant. I was able to remove many of the channels, and still obtain the same qualitative behavior of the network. For example removing the faster K channel in the 3 neurons at the same time will not affect much the behavior of the circuit as a whole, however, it will affect the behavior of each neuron if separated from the network (see attached figure). Also removing the self excitatory input to DSI and VSI neurons had minor effects on the behavior of the network. However, some of the connections were very crucial to the behavior of the network, such as the mutual inhibition between the VSI and DSI neurons, which is intuitive.

Simulation specifications:

The network consisted of three neurons. Each neuron comprised one compartment, two kinds of K channels, a threshold which rises and then decays exponentially after the firing of the neuron. The VSI neuron also had a third K channel of the "A" type. The total number of synapses between the three neurons is 14. The model was implemented using "genesis". The time it takes to simulate 20.00 sec is about 11.00 minutes on a DECstation 3100 with integration time step of .2 msec.

● Project title: Active Compartmental Model of Purkinje Cell.

Name/Institution: Tony Bell, Free University of Brussels (V.U.B.);
Paul Bush, University of California at San Diego

Project description:

We entered the lengths and diameters of 1089 dendritic compartments of a Guinea pig Purkinje cell into a computer model. The data was obtained from a paper by Shelton (Neuroscience 14: 111-131, 1989), describing the electrotonic properties of a passive membrane model using these dimensions. We decided to incorporate active conductances in the same compartments to study the non-linearities occurring in the dendritic tree of the Purkinje cell, an arborisation of unparalleled complexity in the nervous system.

The phenomenon of calcium spikes in the dendrites, and the possibility of "dendritic hotspots" are of particular interest to us as a possible basis of complex nonlinear, even "logical", spatio-temporal integration properties. An additional motivation was the investigation of plateau potentials in Purkinje cells and their possible role in a "figure/ground" theory of cerebellar function.

A graphical display of proportional cylinders was an important part in the visualization of calcium spiking, capturing the heterogeneous distribution of voltage in the tree. It also enabled us to view simulated calcium distribution in the light of calcium-sensitive dye-based studies of Purkinje cells, which have revealed exceptionally high calcium levels in distal dendrites during complex spiking caused by climbing fiber activation.

Results:

Initial estimates of the conductance distributions in the cell have produced some realistic phenomena. Somatic sodium spikes induced by depolarizing current injections invade the whole dendritic tree within 1 or 2 ms. Using 3 conductance schemas, with increasing density of high threshold calcium channels moving distally along the dendritic tree, bursts of calcium spikes were observed, causing massive depolarizations which disrupted spiking at the soma.

Simulation specifications: 1089 compartments, average of 3 conductances each.

Time:

With over 3000 active conductances, a timestep of 10 us (forward Euler, 1st order in space and time), and graphical update every 100 us, the model simulated 1 ms in 40s of real time on the DEC 3100. This was encouraging in view of the inefficiency of our numerical methods. We are not paying too much for the high interactivity on these machines.

● Project title: A simplified model of LGN-V1 feed-forward and feed-back interconnections

Name/Institution: Merav Galun, Department of Neurobiology, Hebrew-University, Israel

Project description:

The motivation of the project was to produce simple properties of LGN on-center off-surround units and V1 simple orientation selective cells incorporating basic but prominent anatomical and physiological data. The following constraints were met: a) LGN units had a center-surround receptive field "inherited" from the retinal input. b) Feed-forward projections from LGN to layer 4 in V1 and from layer 4 to layer 6 was incorporated in a Hubel-Wiesel manner which produced crude orientation preference, highly dependent on stimulus contrast. c) Each of the 3 layers (LGN, layer 4 and layer 6) had 80% excitatory neurons and 20%

inhibitory, both having the same characteristic receptive field. d) Output from each layer was only excitatory (both feed-forward and feed-back) and connected to both excitatory and inhibitory units. Feed-forward fibers connected to excitatory and inhibitory units in the same manner. e) Feed-back and feed-forward connections had the same strength.

Lateral inhibition was implemented in each layer:

LGN inhibitory units

inhibited their neighboring excitatory units in a radial manner.

Layer 4 and layer 6 had the same scheme of lateral inhibition which was composed of both inhibiting cross-orientation units of same spatial receptive field and inhibiting neighboring units with same orientation preference.

Feed-back connection scheme between layer 6 and LGN was aimed to achieve end-inhibition in the responses of LGN units (with layer 6 units summing over length). This was constrained by the fact that this input does not seem to enlarge the receptive field of LGN units or make them orientation selective.

Progress/results:

This general scheme was implemented. Background noise that was added to the system produced mean firing rate of 3-4 spikes/sec. LGN units showed the expected center-surround response with nonsymmetrical end-inhibition (when the bar is extended only in one direction no significant decrease in the firing rate is observed.) Only crude orientation selectivity was achieved in layer 4. Selectivity was sharpened in layer 6. However, the change of sign between summation in layer 6 and inhibition in layer 4 was very hard to get without a mediator (in a form of a "peri Geniculate Nucleus"). Currently, for some yet unknown reason (probably the lateral inhibition) layer 6 shows symmetrical end-inhibition. The next step involves trying to sharpen orientation selectivity with added excitatory lateral interactions.

Simulation specifications: The simulation is composed of 400 neurons. Each unit has one compartment and fires when it reaches threshold. One msec "real-time" takes one second simulation time.

● Project title: Computing target range in the auditory thalamus of the mustached bat.

Name/Institution: John **Butman**, Washington University, Dept. of Biology, Box 1137, St. Louis, MO 63110

Project description:

Biosonar ranging in the mustached bat is performed in the FM-FM area of auditory cortex where neurons are arranged systematically according to pulse-echo delay, the primary cue for encoding target range. These neurons serve as non-linear multipliers or AND gates which respond to the coincidence of neural inputs. By implementing a series of delay lines for the neural response to the pulse, these neurons are able to perform a cross-correlation of the pulse and echo in the time domain.

To implement this model, two isofrequency lamina (40 neurons each) in the inferior colliculus served as inputs to a sheet of 100 cells within the medial geniculate body where combination sensitivity is first observed. Inputs from the first lamina of the IC (corresponding to the pulse) activated neurons in the MGB with a subthreshold excitation with systematically increasing delays. Inputs from the second lamina (corresponding to the echo) also resulted in a subthreshold excitation.

Results:

The most important observation of this model was the sensitivity of delay tuning to the level of excitatory inputs from the colliculus. The failure of lateral and feedback inhibitory connections to ameliorate this problem suggests that combination sensitivity and gain control are somewhat antagonistic processes and that gain control must be performed at or below the level of the colliculus.

Additional modeling attempts to use more realistic parameters for thalamic neurons, such as Ca^{++} spikes and NMDA receptors, met with limited success.

● Project title: Two Cell Interactions---Coupled Oscillators

Name/Institution: Paul Frankel, Applied Mathematics, Brown University, R.I.

Project description:

D. Kleinfeld, and his associates at Bell Laboratories have recently formed excitatory (and inhibitory) connections between the L7 and L12 (L10 and Left Upper Quadrant) neurons of the Aplysia in vitro. In both cases the phenomenon of bistability was observed due to the slow coupling between the cells. I have recently developed by examining one simple graph. A shortfall of this result is that it suppresses the phase relationship between the cells when they are active. Kleinfeld states that fast excitatory coupling implies in phase, and fast inhibitory coupling implies in phase, and fast inhibitory coupling implies out of phase behavior. Dr. Rinzel, at the National Institutes of Health, has a model which predicts a much richer dependence of phase on the coupling. I am trying to use the method of averaging to get an analytical result which predicts phase dependence on the time scale of the coupling. In conjunction with this work I need a simulation of the two cell behavior whose details will be enhanced by the analytical work, and which will provide feedback to help guide and validate the analytical work.

Two cell, multi-state systems, Kleinfeld argues, can be considered building blocks for higher order networks which are based on multiple stable states, such as the Hopfield model and the work by Little. Such a simple network with bistability can also be thought of as fundamental units in the CPGs in Tritonia and other lower order animals.

In a broader sense, the phase behavior of two coupled oscillators can be applied to many other systems, such as cell reproductive cycles, and coupled chemical oscillators. A good understanding of two oscillators can also help in the understanding of a large population of coupled oscillators, as is now being argued can form the basis for the mysterious cortical oscillations.

Progress/Results:

Matt Wilson, as a tutorial for the course, produced a two cell model. In order to make it useful for my purposes I needed some modifications. This included writing an FFT routine, recompiling Genesis with a Cross Correlation routine written by Dieter Jaeger, and with the help of Matt Wilson, making an exponential averaging routine for continuous systems. With the phase plot option supplied by Matt, I have all the tools I need to run simulations

In the simulations run so far I have learned that the details of anode break response becomes crucial to approaching the switching from one bistable state to the other. This makes it easier to switch from steady state to stable oscillations than to switch from active to silent with a brief current pulse.

The phase of the two cells in the runs performed so far exhibit strictly out of phase behavior as displayed both in the phase plot and in the cross-correlation display.

MARINE BIOLOGICAL LABORATORY
1989 Course Faculty
Methods in Computational Neuroscience
August 6 - September 2, 1989

James M. Bower, Co-Director
California Institute of Technology

Christof Koch, Co-Director
California Institute of Technology

Faculty

Paul Adams, Lecturer
State University of New York, Stony Brook

Edward Adelson, Guest Lecturer
Media Laboratory, Massachusetts Institute of
Technology

Daniel Alkon, Lecturer
NIH/NINDS

Richard Andersen, Lecturer
Massachusetts Institute of Technology

Clay Armstrong, Lecturer
University of Pennsylvania

Upinder Bhalla, Lab Instructor
California Institute of Technology

Avis Cohen, Lecturer
Cornell University

Nancy Kopell, Lecturer
Boston University

Rudolfo Llinas, Lecturer
New York University Medical Center

Michael Mascagni, Lecturer
NIH

Mark Nelson, Lab Instructor
California Institute of Technology

John Rinzel, Lecturer
NIH

MARINE BIOLOGICAL LABORATORY
1989 Course Faculty
Methods in Computational Neuroscience

Faculty (continued)

Idan Segev, Lecturer
Hebrew University, ISRAEL

Terrence Sejnowski, Lecturer
Salk Institute

John Uhley, Lab Instructor
California Institute of Technology

David Van Essen, Lecturer
California Institute of Technology

Christof Von Der Marlsburg, Lecturer
University of Southern California

Matthew Wilson, Lab Instructor
California Institute of Technology

Anthony Zador, Guest Lecturer
Yale University

David Zipser, Lecturer
University of California, San Diego

MARINE BIOLOGICAL LABORATORY
1989 Accepted Student List

Methods in Computational Neuroscience

Ehud Ahissar, Graduate Student
Hebrew University/Hadassah Medical School,
ISRAEL

Jeffrey E. Arle, Graduate Student
University of Connecticut

Tony Bell, Graduate Student
Vrije Universiteit Brussel, BELGIUM

Sherif Maher Botros, Graduate Student
Massachusetts Institute of Technology

Bradford O. Bratton, Post-Doctoral
University of Oklahoma

Dean V. Buonomano, Graduate Student
University of Texas Medical School

Paul C. Bush, Graduate Student
University College, Oxford
U.K.

John Butman, Graduate Student
Washington University

Paul H. Frankel, Graduate Student
Brown University

Merav Galun, Graduate Student
Life Sciences Institute, ISRAEL

Jisoon Ihm, Post-Doctoral
Seoul National University, KOREA

Dieter Jaeger, Graduate Student
University of Michigan

Gilles Laurent, Post-Doctoral
University of Cambridge, UK

Petra Leuchtenberg, Graduate Student
University of Bonn, FRG

Patrick Lynn, Graduate Student
University of Colorado, Boulder

MARINE BIOLOGICAL LABORATORY
1989 Accepted Student List

Methods in Computational Neuroscience

Reinoud Maex, Post-Doctoral
Katholieke Universiteit-Leuven, BELGIUM

Paul A. Moore, Graduate Student
Boston University Marine Program/MBL

Valeriy Nenov, Post-Doctoral
University of California, Los Angeles

Eduardo Solession, Graduate Student
Syracuse University

Ehud Zoharry, Graduate Student
Life Sciences Institute, ISRAEL

**Marine Biological Laboratory
Summer Course**

Methods in Computational Neuroscience

August 5- September 1, 1990

This course is for advanced graduate students and postdoctoral fellows in neurobiology, physics, electrical engineering, computer science and psychology with an interest in "Computational Neuroscience". A background in programming (preferably in C or PASCAL) is highly desirable. Limited to 20 students.

This four-week course presents the basic techniques necessary to study single cells and neural networks from a computational point of view, emphasizing their possible function in information processing. The aim is to enable participants to simulate the functional properties of their particular system of study and to appreciate the advantages and pitfalls of this approach to understanding the nervous system.

The first section will focus on simulating the electrical properties of single neurons (compartmental models, active currents, interactions between synapses, calcium dynamics). The second part of the course will deal with the numerical and graphical techniques necessary for modeling neuronal networks. Examples of such simulations will be drawn from the invertebrate and vertebrate literature (visual system of the fly, learning in *Hermissenda*, mammalian olfactory and visual cortex). In the final section, connectionist neural networks relevant to perception and learning in the mammalian cortex, as well as learning algorithms (e.g. back-propagation) will be analyzed and discussed from a neurobiological point of view.

The course includes daily lectures and laboratories. The laboratory section is organized around GENESIS, the Neuronal Network simulator developed at the California Institute of Technology, running on 20 state-of-the-art, single-user, graphic color workstations. Students are expected to work on a simulation project of their own choosing.

Co-Directors: James M. Bower and Christof Koch, Computation and Neural System Program, California Institute of Technology.