Neuroscience: Theory meets Experiment
What is known about the brain and what can be treated theoretically?

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1) Short-term Memory Project
Analysis of task related activity in the monkey prefrontal cortex

2) Plasticity Project
Influence of Structured Input to modification of synaptic weights

3) Applied Statistics
Development of tools for analyzing neuronal data

4) Modeling of Neuronal Networks
Liquid Computing. Systems which are far from equilibrium. Columns as canonical computational devices.

5) Anesthesia Project
Searching for anesthesia related signal characteristics in human EEG, to understand the mechanisms of anesthesia at the system level.
Complexity of the brain

1m
Brain / Nervous system
~ 100 functionally different areas

1mm
Network of Neurons
~ $10^{12}$ neurons

1µm
Neuron
~ $10^6$ active elements, e.g. synapses, ion-pumps

1nm
Transmitter, Modulators, Channel, etc.
e.g. neurotransmitter, vesicles, receptors
The Brain: Topology

1m
1mm
1µm
1nm

Motor cortex
Somatosensory cortex

Frontal cortex

The central visual pathway

Retina
Optic nerve
Chiasm
Thalamus (LGN)
Visual cortex
Complexity of the brain

~ 10^{12} Neurons

Network of Neurons

Network: Cortex – White and gray matter
Network: Divergence and Convergence

Abeles, Gerstein, Aersten

Complexity of the brain

Neuron

\[ \sim 10^6 \text{ active elements, e.g. synapses, ion-pumps} \]
Neuron: Integration of signals in space and time

structure function signal

synapse dendrite pre-synaptic axons

synaptic signal transduction (excitatory, inhibitory)
signal integration

spike-generation

Neuron: Integration of signals in space and time

cf Alexander Borst
Neuron: Integration of signals in space and time
Neuron: Diversity

Neuron: Diversity

Ion-Pumps

K+ 

Na+ 

inside 

outside 

gordon-diplom
\[ CV = \sum_{k}[E_k - V] \cdot G_k + G'(V - V') + G''(V' - V) \]
Synaptic Modification (one type of learning)

- **Causality** = potentiation
- **Acausality** = depression

\[ \Delta t < 0 \]  
\[ \Delta t > 0 \]

- Pre-synaptic
- Post-synaptic

Neuron: topology II

- Pre-synaptic (Axon)
- Post-synaptic
- Synapse
- Soma
- Dendrite

Makram (1997)
Abbott (2001)

cf Alexander Borst
Theory meets Experiments: Two exemplary Projects

1. Synaptic changes of weights in neurons bombarded with structured input
   (Impact of different Renewal Processes – Poisson versus Gamma)
   
   Cooperation with:
   Larry Abbott – Brandeis University / Boston
   Klaus Obermayer - TU-Berlin

2. Short-term Memory:
   Data analysis for finding important characteristics of activity used for storing information.
   
   Cooperation with:
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   Emery Brown – Harvard / Boston

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Basic idea of the project

\[ p(t) = (\gamma \cdot \lambda)^t \cdot e^{-\gamma \cdot \lambda} \cdot \frac{e^{-\frac{\lambda}{1}}}{(\gamma - 1)} \]

Presynaptic spike trains:
- \( \gamma \) spike trains uncorrelated and correlated with varying shape-factor (\( \gamma \)) of the process
  (MIP: Multiple interaction process, e.g. Kuhn 2003)
- ISI probability density:

\[ CF = \sum [C_0 - C] G_j \quad \text{with} \quad G_j(t) = \frac{t}{C} \]
Poisson process

Gamma process ($\gamma=10$)
Renewal processes: Gamma versus Poisson

Properties of a Poisson process

- Spike-times and inter-spike-intervals are independent from previous ones
  - No serial correlation in spike-times

Properties of \( \gamma \)-Processes

- Inter-spike-intervals are independent from each other
- Spike-times are depending on previous ones
  - Serial correlations in spike-times
  - Cross-correlation depends on initial state for short periods of time

Compound Poisson and \( \gamma \)-processes

<table>
<thead>
<tr>
<th>( N )</th>
<th>( \gamma = 1 )</th>
<th>( \gamma = 10 )</th>
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Compound processes of \( N \) independent and statistically identical processes
Impact of serial correlations in short epochs in time

Serial correlations in spike times modulate the cross-correlation for short epochs.

→ Compound epochs of γ-process with synchronous initial spike times can be approximated by one modulated Poisson process.

Epochs of compound γ-process with random initial spike times (initial phase) can be approximated by compound Poisson.

Prediction (Influence of γ-spike trains on a IF or LIF neuron):
Postsynaptic spike-train is a compound process of:
1. Spike-train driven by noise (poissonian)
2. Spike-train reflecting structure of the γ-process
→ Modulation of spike triggered pre-synaptic event density
→ Compound ISI
→ Synchronized epochs are random in time → No periodicity

Postsynaptic spike-train statistics in case of Poisson and γ-presynaptic activity
Spike triggered pre-synaptic event density: Poisson process

Simulation parameter:
Population sizes
a) 40 inhibitory
b) 160 excitatory
Rates:
a) Inhibitory: 30/sec
b) Excitatory: 20/sec
\[ \lambda_{\text{inh}} = \alpha * \lambda_{\text{exi}} \]
\[ \alpha = 1.5 \]
Balance Exi./Inh.: 1

Spike triggered pre-synaptic event density: \( \gamma \)-process (\( \gamma = 10 \))

Simulation parameter:
Population sizes
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Spike triggered pre-synaptic event density: $\gamma$-process ($\gamma=10$)

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- **Population sizes**
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  \[ \alpha = 1.5 \]
- Balance Exi./Inh.: 1

**Predicted properties:**
- Modulation of spike triggered pre-synaptic event density
- Compound ISI
- No periodicity

**Synaptic Modification**

- Pre-synaptic (Axon)
- Post-synaptic
- Synapse
- Post-synaptic Dendrite
- Soma

**Causality**
- $\Delta t < 0$
  - Potentiation
- $\Delta t > 0$
  - Depression
STDP in case of presynaptic $\gamma$-spiketrains

Poisson: $\gamma = 7$

$\alpha = 1$

$\alpha = 1.5$

$\alpha = 2$

STDP in case of presynaptic $\gamma$-spiketrains
**Conclusions**

- **Influence of γ-spike trains on a conductance based model neuron and STDP**

- Serial Correlation of spike-times modulate the cross-correlation for short epochs in time.

- Resulting post spike-train is a compound process of a noise driven process (e.g. Poisson) and a γ-process

- STDP can be strongly facilitated by γ-processes

- Relation of presynaptic excitatory and inhibitory rate modulates the sensitivity for synchronized epochs and STDP
  - Most effective if e.g. $\lambda_{\text{inh}}=1.5\times\lambda_{\text{exi}}$
  - Less effective if e.g. $\lambda_{\text{inh}}=\lambda_{\text{exi}}$ or $\lambda_{\text{inh}}=2\times\lambda_{\text{exi}}$
Theory meets Experiments: Two exemplary Projects

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Perception - Gestalt-criteria 1920's

Gestalt law: closure

Perception - Gestalt-criteria 1920's

Gestalt law: similarity

gordon-diplom
Perception - Gestalt-criteria - 21. Century

Grossmann Blake 2001
Recording neural activity

**Multi channel Recordings**
- Channel: 8-16
- Sampling: 32 ks/sec
- Online waveform screening

**MUA- Filter**
- Filter
  - Width: 1-150Hz
  - Sampling: 1 ks/sec

Feature extraction in the visual pathway (Retina + LGN)
- **Medium response**
- **Strong response**
- **Weak response**

- On
- Off
- Light
Orientierungsselektivität im primären visuellen Kortex (V1)

Hubel und Wiesel cf. Roelfsema 2002

"Super smart Grand mother cell"

Idea: Hierarchical data processing - abstraction

„Super smart Grand mother cell“

receptive field

TEO
V4
V2
V1

cf Roelfsema 2002
Information = Relation
Dynamic relations between neurons

Hypothesis: Assembly Coding (binding of information)

Information is represented by groups of neurons
Relationships are formed by temporal coordination of cell activities

Assembly Hypothesis – expected signal properties

Properties of recorded spiketrains

1. Spike trains show significant autocorrelation
2. Properties are modulated (e.g. Spike rate, ISI)
3. Spike trains show large variability over trials
4. Different intrinsic timescales for different trials (different reaction and perception times, different internal states) may cause different timescales for the recorded data.
5. Instability of properties during the whole experiment (over hours)
6. Joint-Spike events are rare

If one is willing to use rate as a measurement often used terms for sources of variability are:
1. Rate modulation
2. Trial by trial variability
3. Latency variability

Data: monkey primary motor cortex
38 trials: delayed-pointing task

Using Surrogate Data to realize H0

Real Data

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<th>sliding window (500 ms)</th>
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Surrogate Jittering +/-2

Real Data

Nreal(Pat_ID) = 1
Nsurro(Pat_ID) = 0

Short Term Memory: Delayed discrimination task

Stimulus

500 ms 3 sec Reaction time

Delay Test

500 ms 3 sec Reaction time

Stimulus Delay Test

Match

Left Button

Non-Match

Right Button
Performance Analysis – correct versus incorrect discrimination

Frequency of interactions stronger for correct than incorrect

Frequency of interactions stronger for incorrect than correct

Assembly Hypothesis – expected signal properties

Spike trains

LFP

Coherence
Phase Locking

Precise spike synchronization
2) Assessing stability of phase differences

signal is filtered by using a Gabor wavelet

Percentage of sites with increased power / phase locking for correct performance

Power                      Phase-locking
Conclusions

1) Synchronized firing and coherent oscillations are performance-related.

Thank you for your attention
Variation of the relation between rates of excitatory and inhibitory presynaptic input ($\alpha$)

$$\lambda_{\text{inh}} = \alpha \times \lambda_{\text{exi}}$$
Spike triggered average: pre-synaptic $\gamma$-process ($\gamma = 10$)

Simulation parameter:
Population sizes
a) 40 inhibitory
b) 160 excitatory

Rates:
a) Inhibitory: 30/sec
b) Excitatory: 20/sec
$\Rightarrow \alpha = 1.5$

Balance Ext./Inh.: 1

Spike triggered average: pre-synaptic $\gamma$-process ($\gamma = 10$)

Simulation parameter:
Population sizes
a) 40 inhibitory
b) 160 excitatory

Rates:
a) Inhibitory: 34/sec
b) Excitatory: 20/sec
$\Rightarrow \alpha = 1.7$

Balance Ext./Inh.: 1
Spike triggered average: pre-synaptic $\gamma$-process ($\gamma=10$)

Simulation parameter:

Population sizes
a) 40 inhibitory
b) 160 excitatory

Rates:
- Inhibitory: 40/sec
- Excitatory: 20/sec

$\Rightarrow \alpha = 2$

Balance Exc./Inh.: 1

gordon-diplom