Overview over different methods

**Machine Learning**
- REINFORCEMENT LEARNING
  - example based
- Dynamic Prog. (Bellman Eq.)
- Monte Carlo Control
- SARSA
- Q-Learning

**Classical Conditioning**
- Classical Conditioning
  - Anticipatory Control of Actions and Prediction of Values
- δ-Rule
  - supervised L.
- Eligibility Trace
  - $TD(\lambda)$ often $\lambda=0$
- Neur.TD-formalism
- Neur.TD-Models
  - (“Critic”)
- ISO-Learning
  - (“Criticism”)
- ISO-Control
  - technical & Basal Gangl.
- Actor/Critic
  - “Critic”
- SARSA

**Synaptic Plasticity**
- Correlation of Signals
- Hebb-Rule
- Differential Hebb-Rule
  - (“slow”)
- Rescorla/Wagner
- LTP ($LTD=anti$)
- DIFFERENTIAL HEBB-RULE
  - (“fast”)
- STDP-Models
  - biophysical & network
- ISO-Model of STDP

**Biophys. of Syn. Plasticity**
- Dopamine
- Glutamate
- Biophys. of Syn. Plasticity
- LTP

**Evaluative Feedback (Rewards)**
- SARSA
- Correlation based Control
  - (non-evaluative)
- ISO-Control

**Non-Evaluative Feedback (Correlations)**
- Neuronal Reward Systems
  - (Basal Ganglia)
Hebbian learning

When an axon of cell A excites cell B and repeatedly or persistently takes part in firing it, some growth processes or metabolic change takes place in one or both cells so that A’s efficiency ... is increased.

Donald Hebb (1949)
Hebbian Learning

...correlates inputs with outputs by the...

...Basic Hebb-Rule: \( \frac{d\omega_1}{dt} = \mu \, v \, u_1 \) \( \mu \ll 1 \)

Vector Notation following Dayan and Abbott:

Cell Activity: \( v = w \cdot u \)

This is a dot product, where \( w \) is a weight vector and \( u \) the input vector. Strictly we need to assume that weight changes are slow, otherwise this turns into a differential eq.
Hebbian Learning

\[ \frac{d\omega_1}{dt} = \mu u_1 v \]
Single Input
\[
\frac{d\omega_1}{dt} = \mu \, v \, u_1 \quad \mu \ll 1
\]

Many Inputs
\[
\frac{dw}{dt} = \mu \, v \, u \quad \mu \ll 1
\]
As \( v \) is a single output, it is scalar.

Averaging Inputs
\[
\frac{dw}{dt} = \mu \, \langle v \, u \rangle \quad \mu \ll 1
\]
We can just average over all input patterns and approximate the weight change by this. Remember, this assumes that weight changes are slow.

If we replace \( v \) with \( w \cdot u \) we can write:
\[
\frac{dw}{dt} = \mu \, Q \cdot w \quad \text{where } Q = \langle uu \rangle \text{ is the input correlation matrix}
\]

Note: Hebb yields an instable (always growing) weight vector!
Synaptic plasticity evoked artificially

Examples of Long term potentiation (LTP) and long term depression (LTD).

LTP First demonstrated by Bliss and Lomo in 1973. Since then induced in many different ways, usually in slice.

LTD, robustly shown by Dudek and Bear in 1992, in Hippocampal slice.
Hippocampal pathways exhibiting LTP

- CA1 pyramidal cell
- Schaffer collaterals
- CA3 pyramidal cell
- Granule cell
- Dentate gyrus
- Mossy fibers
- Perforant path
Artificially induced synaptic plasticity.

Presynaptic rate-based induction

Bear et. al. 94
Depolarization based induction

Feldman, 2000
NMDA receptor activation is necessary and sufficient for LTP induction.
LTP will lead to new synaptic contacts
Long-term depression (LTD) is the opposite of LTP

It is elicited by low frequency stimulation

![Diagram showing CA3 and CA1 pyramidal cells with Schaffer collaterals, stimulus, and record.]
Synaptic Plasticity:
Dudek and Bear, 1993

A. Population EPSP slope (% of baseline) over time from onset of LFS (min.).

B. Change in EPSP slope (%) with frequency of conditioning stimulation (Hz).

LTP (Long-Term Potentiation)
LTD (Long-Term Depression)
Conventional LTP

Symmetrical Weight-change curve

The temporal order of input and output does not play any role
Spike timing dependent plasticity - STDP

Markram et. al. 1997

Pre before Post: LTP

Post before Pre: LTD

C

EPSP amplitude (% of control)

Time (min)

80 m

1.5 mV

50 mV

50 ms

+10 ms

-10 ms

Markram et. al. 1997
Spike Timing Dependent Plasticity: Temporal Hebbian Learning

Pre follows Post: Long-term Depression

Causal (possibly)

Pre precedes Post: Long-term Potentiation

Acausal

Synaptic change %

Weight-change curve
(Bi&Poo, 2001)
Different Learning Curves

(Note: X-axis is pre-post, We will use: post - pre, which seems more natural)
At this level we know much about the cellular and molecular basis of synaptic plasticity.

But how do we know that “synaptic plasticity” as observed on the cellular level has any connection to learning and memory?

What types of criterions can we use to answer this question?
'Activity-dependent synaptic plasticity is induced at appropriate synapses during memory formation, and is both necessary and sufficient for the information storage [...].'

(Martin et al., 2000; Martin and Morris, 2002)
Assessment criterions for the synaptic hypothesis: (From Martin and Morris 2002)

1. DETECTABILITY: If an animal displays memory of some previous experience (or has learnt a new task), a change in synaptic efficacy should be detectable somewhere in its nervous system.

2. MIMICRY: If it were possible to induce the appropriate pattern of synaptic weight changes artificially, the animal should display ‘apparent’ memory for some past experience which did not in practice occur.

Garner et al. 2012, Science, 335, 1513-1516
3. ANTEROGRADE ALTERATION: Interventions that prevent the induction of synaptic weight changes during a learning experience should impair the animal’s memory of that experience (or prevent the learning).

4. RETROGRADE ALTERATION: Interventions that alter the spatial distribution of synaptic weight changes induced by a prior learning experience (see detectability) should alter the animals memory of that experience (or alter the learning). Similarly: Stimulation of a set of learned neuron should elicit a memory.

Liu et al. 2012, Nature
Proof the SPM Hypothesis

**Detectability:** If an animal displays memory of some previous experience, a change in synaptic efficacy should be detectable somewhere in its nervous system.

**Mimicry:** Conversely, if it were possible to induce the same spatial pattern of synaptic weight changes artificially, the animal should display 'apparent' memory for some past experience which did not in practice occur.

**Anterograde Alteration:** Interventions that prevent the induction of synaptic weight changes during a learning experience should impair the animal's memory of that experience.

**Retrograde Alteration:** Interventions that alter the spatial distribution of synaptic weights induced by a prior learning experience (see detectability) should alter the animal's memory of that experience.

Martin et al., 2000; Martin and Morris, 2002
Rats were trained for three or five days in a skilled reaching task with one forelimb, after which slices of motor cortex were examined to determine the effect of training on the strength of horizontal intracortical connections in layer II/III.
Detectability

Example from Rioult-Pedotti - 1998

Fig. 1. Consequences of motor skill learning on field-potential responses evoked in layer II/III horizontal connections of M1. (a) Mirror-symmetric placement of stimulating (stim) and recording (rec) microelectrodes bilaterally in layers II/III of M1 in a coronal slice containing both hemispheres, wm, white matter. (b) Single-case examples of field potentials (averages of five sweeps), evoked at 60% maximum stimulation intensity from a single trained (top) and a single paired-control (bottom) animal. Dark lines represent the trained M1 or left M1, hatched lines, the untrained M1 or right M1. (c) Group average responses for trained (top, n = 7) and control (bottom, n = 20, paired and naive) rats at 60% maximal stimulation intensity, illustrating enhanced field potential in the horizontal pathway of M1 contralateral to the limb used in the reaching task. Same format as (b).

The amplitude of field potentials in the forelimb region contralateral to the trained limb was significantly increased relative to the opposite ‘untrained’ hemisphere.
Detectability
e.g. Reaching Task

Rioult-Pedotti, et al., 1998
Proof the SPM Hypothesis

Detectability: If an animal displays memory of some previous experience, a change in synaptic efficacy should be detectable somewhere in its nervous system.

Mimicry: Conversely, if it were possible to induce the same spatial pattern of synaptic weight changes artificially, the animal should display 'apparent' memory for some past experience which did not in practice occur.

Anterograde Alteration: Interventions that prevent the induction of synaptic weight changes during a learning experience should impair the animal's memory of that experience.

Retrograde Alteration: Interventions that alter the spatial distribution of synaptic weights induced by a prior learning experience (see detectability) should alter the animal's memory of that experience.

Martin et al., 2000; Martin and Morris, 2002
The memory of context “A” is first marked in a chemically genetic manner (pink neurons). This memory is then activated by using a drug (Garner et al., 2012) or by optogenetic stimulation (Liu et al., 2012) while the animal is in a different place - context “B” (blue neurons) - and subject to fear conditioning. Later memory retrieval is only successful if the animal remembers both contexts “A” + “B” (pink and blue neurons). If the drug or stimulation is not been present during retrieval, the pattern of neuronal firing is different and does not induce the related fear behavior.
Proof the SPM Hypothesis

**Detectability:** If an animal displays memory of some previous experience, a change in synaptic efficacy should be detectable somewhere in its nervous system.

**Mimicry:** Conversely, if it were possible to induce the same spatial pattern of synaptic weight changes artificially, the animal should display 'apparent' memory for some past experience which did not in practice occur.

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Martin et al., 2000; Martin and Morris, 2002
Anterograde Alteration
e.g. Morris Water Maze

Learn the position of the platform

Morris et al., 1986
Anterograde Alteration
e.g. Morris Water Maze

Learn the position of the platform

Morris et al., 1986
Proof the SPM Hypothesis

**Detectability:** If an animal displays memory of some previous experience, a change in synaptic efficacy should be detectable somewhere in its nervous system.

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Martin et al., 2000; Martin and Morris, 2002
Retrograde Alteration

e.g. Place Avoidance

Pastalkova et al., 2006
Proof the SPM Hypothesis

**Detectability:** If an animal displays memory of some previous experience, a change in synaptic efficacy should be detectable somewhere in its nervous system.

**Mimicry:** Conversely, if it were possible to induce the same spatial pattern of synaptic weight changes artificially, the animal should display 'apparent' memory for some past experience which did not in practice occur.

**Anterograde Alteration:** Interventions that prevent the induction of synaptic weight changes during a learning experience should impair the animal's memory of that experience.

**Retrograde Alteration:** Interventions that alter the spatial distribution of synaptic weights induced by a prior learning experience (see detectability) should alter the animal's memory of that experience.

only evidences!!!

Martin et al., 2000; Martin and Morris, 2002
Back to the Math. We had:

Single Input
\[ \frac{d\omega_1}{dt} = \mu \, v \, u_1 \quad \mu << 1 \]

Many Inputs
\[ \frac{dw}{dt} = \mu \, v \, u \quad \mu << 1 \]

As v is a single output, it is scalar.

Averaging Inputs
\[ \frac{dw}{dt} = \mu \, <v \, u> \quad \mu << 1 \]

We can just average over all input patterns and approximate the weight change by this. Remember, this assumes that weight changes are slow.

If we replace v with \( w \cdot u \) we can write:
\[ \frac{dw}{dt} = \mu \, Q \cdot w \quad \text{where } Q = <uu> \text{ is the input correlation matrix} \]

Note: Hebb yields an instable (always growing) weight vector!
Covariance Rule(s)

Normally firing rates are only positive and plain Hebb would yield only LTP. Hence we introduce a threshold to also get LTD

\[
\frac{d\mathbf{w}}{dt} = \mu (v - \Theta) \mathbf{u} \quad \mu \ll 1 \quad \text{Output threshold}
\]

\[
\frac{d\mathbf{w}}{dt} = \mu v (\mathbf{u} - \Theta) \quad \mu \ll 1 \quad \text{Input vector threshold}
\]

Many times one sets the threshold as the average activity of some reference time period (training period)

\[\Theta = \langle v \rangle \quad \text{or} \quad \Theta = \langle \mathbf{u} \rangle \quad \text{together with} \quad v = \mathbf{w} \cdot \mathbf{u} \quad \text{we get:}\]

\[
\frac{d\mathbf{w}}{dt} = \mu \mathbf{C} \cdot \mathbf{w}, \text{where } \mathbf{C} \text{ is the covariance matrix of the input}
\]

\[\mathbf{C} = \langle (\mathbf{u} - \langle \mathbf{u} \rangle)(\mathbf{u} - \langle \mathbf{u} \rangle) \rangle = \langle \mathbf{uu} \rangle - \langle \mathbf{u}^2 \rangle = \langle (\mathbf{u} - \langle \mathbf{u} \rangle)\mathbf{u} \rangle\]
The covariance rule can produce LTD without (!) post-synaptic input. This is biologically unrealistic and the BCM rule (Bienenstock, Cooper, Munro) takes care of this.

**BCM- Rule**

\[
\frac{dw}{dt} = \mu \nu u (v - \Theta) \quad \mu << 1
\]

*Dudek and Bear, 1992*
The covariance rule can produce LTD without (!) post-synaptic input. This is biologically unrealistic and the BCM rule (Bienenstock, Cooper, Munro) takes care of this.

**BCM- Rule**

\[
\frac{dw}{dt} = \mu v u (v - \Theta) \quad \mu \ll 1
\]

Experiment

Dudek and Bear, 1992
The covariance rule can produce LTD without (!) post-synaptic input. This is biologically unrealistic and the BCM rule (Bienenstock, Cooper, Munro) takes care of this.

**BCM- Rule**

\[
\frac{dw}{dt} = \mu v u (v - \Theta) \quad \mu << 1
\]

As such this rule is again unstable, but BCM introduces a sliding threshold

\[
\frac{d\Theta}{dt} = \nu (v^2 - \Theta) \quad \nu < 1
\]

Note the rate of threshold change \( \nu \) should be faster than then weight changes (\( \mu \)), but slower than the presentation of the individual input patterns. This way the weight growth will be over-dampened relative to the (weight – induced) activity increase.
Evidence for weight normalization:
Reduced weight increase as soon as weights are already big (Bi and Poo, 1998, J. Neurosci.)

BCM is just one type of (implicit) weight normalization.
Other Weight normalization mechanisms

Bad News: There are MANY ways to do this and results of learning may vastly differ with the used normalization method. This is one down-side of Hebbian learning.

In general one finds two often applied schemes: Subtractive and multiplicative weight normalization.

Example (subtractive):
\[
\frac{1}{\mu} \frac{d\mathbf{w}}{dt} = \nu \mathbf{u} - \frac{\nu (\mathbf{n} \cdot \mathbf{u}) \mathbf{n}}{N}
\]

With N, number of inputs and \(\mathbf{n}\) a unit vector (all “1”). This yields that \(\mathbf{n} \cdot \mathbf{u}\) is just the sum over all inputs.

Note: This normalization is *rigidly* applied at each learning step. It requires global information (info about ALL weights), which is biologically unrealistic.

One needs to make sure that weight do not fall below zero (lower bound). Also: Without upper bound you will often get all weight = 0 except one.

Subtractive normalization is highly competitive as the subtracted values are always the same for all weight and, hence, will affect small weight relatively more.
Weight normalization:

Example (multiplicative): \[
\frac{dw}{dt} = \mu \left( vu - \alpha v^2 w \right), \quad \alpha > 0
\]

(Oja’s rule, 1982)

Note: This normalization leads to an asymptotic convergence of \(|w|^2\) to \(1/\alpha\).

It requires only local information (pre-, post-syn. activity and the local synaptic weight).

It also introduces competition between the weights as growth of one weight will force the other into relative re-normalization (as the length of the weight vector \(|w|^2\) remains always limited.
Eigen Vector Decomposition - PCA

We had:

$$\frac{dw}{dt} = \mu \langle v \ u \rangle \quad \mu << 1$$

We can just average over all input patterns and approximate the weight change by this. Remember, this assumes that weight changes are slow.

If we replace $v$ with $w \cdot u$ we can write:

$$\frac{dw}{dt} = \mu \ Q \cdot w$$

where $Q = \langle uu \rangle$ is the input correlation matrix

And write: $Q \cdot e_\nu = \lambda_\nu e_\nu,$

where $e_\nu$ is an eigenvector of $Q$ and $\lambda_\nu$ is an eigenvalue, with $\nu = 1, \ldots, N.$ Note for correlation matrices all eigenvalues are real and non-negative.

As usual, we rank-order the eigenvalues: $\lambda_1 \geq \lambda_2 \geq \ldots \geq \lambda_N.$
Eigen Vector Decomposition - PCA

Every vector can be expressed as a linear combination of its eigenvectors:

$$\mathbf{w}(t) = \sum_{\nu=1}^{N} c_\nu(t) \mathbf{e}_\nu \quad \star \star$$

Where the coefficients are given by:

$$c_\nu(t) = \mathbf{w}(t) \cdot \mathbf{e}_\nu \quad \star$$

Entering in and solving for $c_\nu$ yields:

$$c_\nu(t) = c_\nu(0) \exp\left(\frac{\lambda_\nu t}{\mu}\right)$$

Using $\star$ with $t=0$ we can rewrite $\star \star$ to:

$$\mathbf{w}(t) = \sum \exp\left(\frac{\lambda_\nu t}{\mu}\right)(\mathbf{w}(0) \cdot \mathbf{e}_\nu)\mathbf{e}_\nu$$
Eigen Vector Decomposition - PCA

\[ w(t) = \sum \exp\left(\frac{\lambda_{\nu} t}{\mu}\right)(w(0) \cdot e_{\nu}) e_{\nu} \]

As the \( \lambda \)'s are rank-ordered and non-negative we find that for long \( t \) only the first term will dominate this sum. Hence:

\[ w \approx e_1 \quad \text{and, thus,} \quad v \approx e_1 \cdot u \]

As the dot product corresponds to a projection of one vector onto another, we find that hebbian plasticity produces an output \( v \) proportional to the projection of the input vector \( u \) onto the principal (first) eigenvector \( e_1 \) of the correlation matrix of the inputs used during training.

Note \( \exp\left(\frac{\lambda_1 t}{\mu}\right) \) will get quite big over time and, hence, we need normalization!

A good way to do this is to use Oja’s rule which yields:

\[ w = \frac{e_1}{\sqrt{\alpha}}, \quad t \rightarrow \infty \]
Panel A shows the input distribution (dots) for two inputs $\mathbf{u}$, which is Gaussian with mean zero and the alignment of the weight vector $\mathbf{w}$ using the basic Hebb rule. The vector aligns with the main axis of the distribution. Hence, here we have something like PCA.

Panel B shows the same when the mean is non zero. No alignment occurs.

Panel C shows the same when applying the covariance Hebb rule. Here we have the same as in A. (One has subtracted the average!)
Visual Pathway – Towards Cortical Selectivities

Receptive fields are:
- Binocular
- Orientation Selective

Receptive fields are:
- Monocular
- Radially Symmetric

Visual Cortex

Area 17

Retina

light ——> electrical signals
Monocular Deprivation

Normal

Ocular dom, group

Left
Right

% of cells

Response (spikes/sec)

angle

1 2 3 4 5 6 7

10

20

Left dom
Right dom

Ocular dom, group

Right

Left

1 2 3 4 5 6 7

15

30

10

20

30

15

Left dom
Right dom

group

group
Modelling Ocular Dominance – Networks

The ocular dominance map:

With gradual transitions
Magnification (monkey)

Larger Map after thresholding
(Monkey)
Modelling Ocular Dominance – Single Cell

Left \quad u_l \quad w_l \quad v

Eye input

Right \quad u_r \quad w_r

\[ v = w_r u_r + w_l u_l \]

We need to generate a situation where through Hebbian learning one synapse will grow while the other should drop to zero. Called: Synaptic competition (Remember “weight normalization”!)

We assume that right and left eye are statistically the same and, thus, get as correlation matrix \( Q \):

\[
Q = <uu> = \begin{pmatrix}
<u_r u_r> & <u_r u_l>\\
<u_l u_r> & <u_l u_l>
\end{pmatrix} = \begin{pmatrix}
q_S & q_D \\
q_D & q_S
\end{pmatrix}
\]
Modelling Ocular Dominance – Single Cell

Eigenvectors are: \( \mathbf{e}_1 = \frac{1}{\sqrt{2}} \begin{pmatrix} +1 \\ +1 \end{pmatrix} \) and \( \mathbf{e}_2 = \frac{1}{\sqrt{2}} \begin{pmatrix} +1 \\ -1 \end{pmatrix} \)

with eigenvalues \( \lambda_1 = q_S + q_D \) and \( \lambda_2 = q_S - q_D \)

Using the correlation based Hebb rule: \( \frac{d\mathbf{w}}{dt} = \mu \mathbf{Q} \cdot \mathbf{w} \)

And defining: \( \mathbf{w}_+ = \mathbf{w}_r + \mathbf{w}_l \) and \( \mathbf{w}_- = \mathbf{w}_r - \mathbf{w}_l \)

We get: \( \frac{d\mathbf{w}_+}{dt} = \mu(q_S + q_D)\mathbf{w}_+ \) and \( \frac{d\mathbf{w}_-}{dt} = \mu(q_S - q_D)\mathbf{w}_- \)

We can assume that after eye-opening positive activity correlations between the eyes exist. Hence: \( q_S + q_D > q_S - q_D \)

And it follows that \( \mathbf{e}_1 \) is the principal eigenvector leading to equal weight growth for both eyes, which is not the case in biology!
Modelling Ocular Dominance – Single Cell

Weight normalization will help, (but only subtractive normalization works as multiplicative normalization will not change the relative growth of $w^+$ as compared to $w^-$):

$$\frac{1}{\mu} \frac{dw}{dt} = \nu u - \frac{v(n \cdot u)n}{N}$$

As: $n = \begin{pmatrix} 1 \\ 1 \end{pmatrix}$ we have $e_1 \sim n$ which eliminates weight growth of $w^+$. (because entering $e_1$ into the above yields: $Q \cdot e_1 - (e_1 \cdot Q \cdot n)n/N = 0$)

While, on the other hand: $e_2 \cdot n = 0$, (vectors are orthogonal). Hence the weight vector will grow parallel to $e_2$, which requires the one to grow and the other to shrink.

What really happens is given by the initial conditions of $w(0)$.

If: $w(0) \cdot e_2 \sim w_r(0) - w_l(0) > 0$

$w_r$ will increase, otherwise $w_l$ will grow.
Modelling Ocular Dominance – Networks

The ocular dominance map:

With gradual transitions
Magnification (monkey)

Larger Map after thresholding
(Monkey)
Modelling Ocular Dominance – Networks

To receive an ocular dominance map we need a small network:

\[
\frac{dv}{dt} \sim -v + W \cdot u + M \cdot v
\]

Here the activity components \(v_i\) of each neuron collected into vector \(v\) are recursively defined by:

\[
\begin{align*}
\text{output } v \\
\text{input } u & \quad u_1 \quad u_2 \quad u_3 \\
M & \quad W
\end{align*}
\]

Where \(M\) is the recurrent weight matrix and \(W\) the feed-forward weight matrix. If the eigenvalues of \(M\) are smaller than 1 than this is stable and we get as the steady state output:

\[
v = W \cdot u + M \cdot v
\]
Modelling Ocular Dominance – Networks

Defining the inverse: \[ K = (I - M)^{-1} \]

Where \( I \) is the identity matrix. This way we can rewrite:

\[ v = K \cdot W \cdot u \]

An ocular dominance map can be achieved similar to the single cell model but by assuming constant intracortical connectivity \( M \).

We use this network:
Modelling Ocular Dominance – Networks

We get for the weights:

$$\frac{1}{\mu} \frac{dW}{dt} = \langle vu \rangle = K \cdot W \cdot Q$$

where $Q = \langle uu \rangle$ is the autocorrelation matrix.

For the simple network (last slide) we can write:

$$v = w_r u_r + w_l u_l + M \cdot v$$

afferent intra-cortical

Again we define $w_+$ and $w_-$ (this time as vectors!) and get:

$$\frac{dw_+}{dt} = \mu(q_S + q_D)K \cdot w_+ \quad \text{and} \quad \frac{dw_-}{dt} = \mu(q_S - q_D)K \cdot w_-$$

With subtractive normalization we can again neglect $w_+$

Hence the growth of $w_-$ is dominated by the principal eigenvector of $K$
Modelling Ocular Dominance – Networks

We assume that the intra-cortical connection structure is similar everywhere and, thus, given by \( K(|x-x'|) \). Note: \( K \) is NOT the connectivity matrix. Let us assume that \( K \) takes the shape of a difference of Gaussians.

If we assume periodic boundary conditions in our network we can calculate the eigenvectors \( e^\nu \) as:

\[
* \quad e_x^\nu = \cos \left( \frac{2\pi \nu x}{N} - \phi \right) \quad \nu = 0, 1, 2, \ldots, \frac{N}{2} \quad \phi \in [0, 2\pi]
\]

Note: In some way this is a „trick“: we assume that \( K \) takes this shape in order to get the right result in the end….

\( K \) can be thought of as factor that spatially modulates the input correlations given by \( q_s \) and \( q_d \).
The eigenvalues are given by the Fourier Transform $\tilde{K}$ of $K$

The principal eigenvector is given by * (last slide) with: $\nu = \arg \max \tilde{K}$

The diagram above plots another difference of Gaussian $K$ function (A, solid), its Fourier transform $\tilde{K}$ (B), and the principal eigenvector (A, dotted). The fact that the eigenvector’s sign alternates leads to alternating left/right eye dominance just like for the single cell example discussed earlier.
Tuning curves

Ocular Dom. Distr.

Orientation Selectivity

Response difference indicative of ocularity
Orientation Selectivity

Normal

Binocular Deprivation

Eye-opening

Adult

Eye-opening