A saturation model for impaired learning with enhanced plasticity

based on work in preparation by: T.D. Barbara Nguyen-Vu, Grace Q. Zhao, Han-Mi Lee, SL, Surya Ganguli, Carla J. Shatz, Jennifer L. Raymond

Subhaneil Lahiri

Stanford University, Applied Physics

July 10, 2013

A saturation model for impaired learning with enhanced plasticity
based on work in preparation by: T.D. Barbara Nguyen-Vu, Grace Q. Zhao, Han-Mi Lee, SL, Surya Ganguli, Carla J. Shatz, Jennifer L. Raymond

Subhaneil Lahiri

Stanford University, Applied Physics

July 10, 2013

Outline

Saturation by enh. plasticity impairs learning

2013-07-10

└Outline

Outline

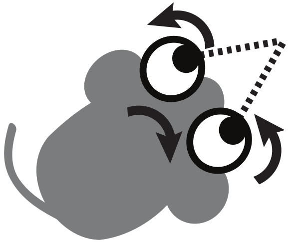
1. VOR learning and the cerebellum
2. The effects of enhanced plasticity and saturation
3. Modelling approach
4. Modelling results

Section 1

VOR learning and the cerebellum

Section 1
VOR learning and the cerebellum

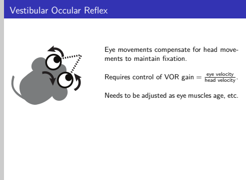
Vestibular Occular Reflex

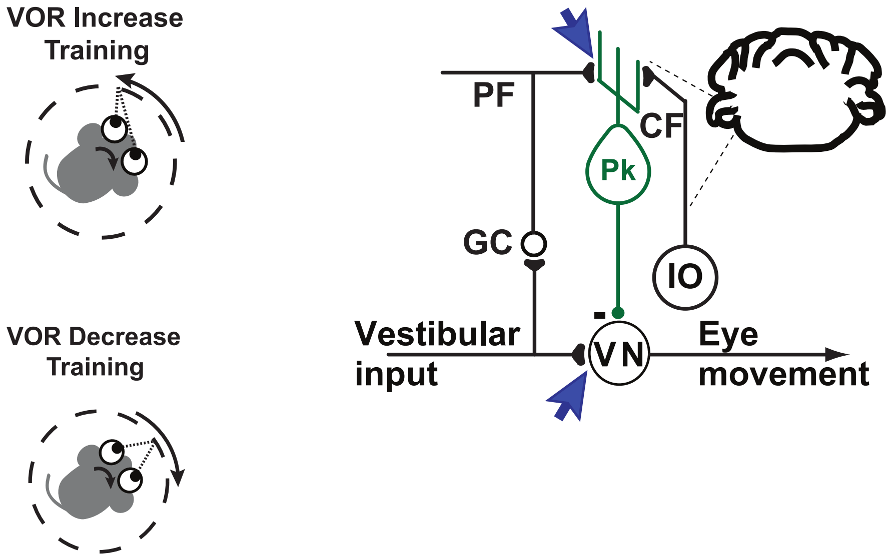


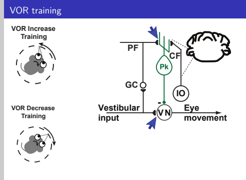
Eye movements compensate for head movements to maintain fixation.

Requires control of VOR gain $= \frac{\text{eye velocity}}{\text{head velocity}}$.

Needs to be adjusted as eye muscles age, etc.







1. trick brain into thinking VOR gain needs adjusting my moving visual stimulu
2. anti-phase →increase gain
3. in phase →decrease gain
4. Gain change involves cerebellum
5. Marr-Albus-Ito: Pf-Pk synapses
6. Lisberger-Miles: Vestibular input-VN synapses
7. Different mechs for different freq, head angle, gain up/down.
8. Different Pk cells have different tunings.
9. Gain up in case of interest: LTD in Pf-Pk in flucculus
10. Gain down: uses different mech for behaviour, but does reverse LTD in Pf-Pk in flucculus

Section 2

The effects of enhanced plasticity and saturation

Section 2
The effects of enhanced plasticity and saturation

Questions

- Can the saturation effect overcome the enhanced plasticity?
- How can a little reverse bias help, but too much hurt?
- Can we find a purely synaptic explanation of these results?

Questions
- Can the saturation effect overcome the enhanced plasticity?
- How can a little reverse bias help, but too much hurt?
- Can we find a purely synaptic explanation of these results?

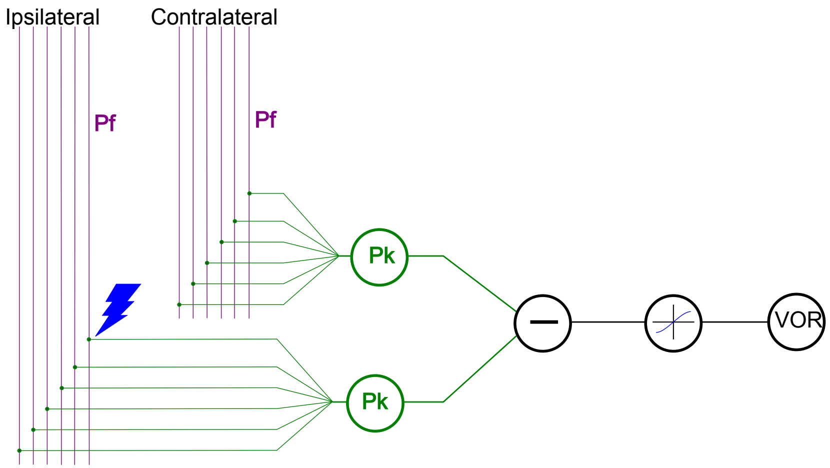
1. in competition
2. first makes sense, but second?
3. This is a question about synaptic populations after all.

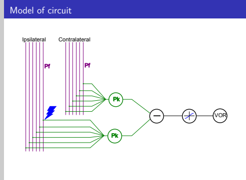
Section 3

Modelling approach

Section 3
Modelling approach

Model of circuit





1. Contralateral baseline shift compensates for Our baseline shift
2. Gain increase due to LTD at lightning
3. Gain decease due to plasticity elsewhere, but also reverses LTD at lightning
4. Nonlinearity here won't affect our questions, as long as it doesn't change
5. Nonlinearity before compensation could change things

Simplifying assumptions

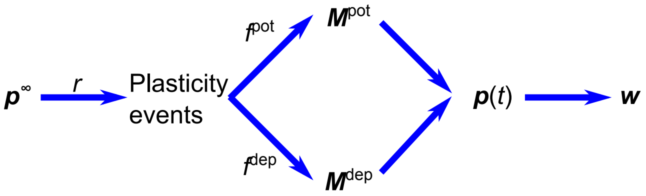
- No spatial/temporal correlations in plasticity events.
- Potentiating/depressing plasticity events $\sim$ Poisson processes.
- Potentiation and depression are described by Markov processes.
  [Fusi et al. (2005), Fusi and Abbott (2007), Barrett and van Rossum (2008)]

Simplifying assumptions
- No spatial/temporal correlations in plasticity events.
- Potentiating/depressing plasticity events $\sim$ Poisson processes.
- Potentiation and depression are described by Markov processes.
  [Fusi et al. (2005), Fusi and Abbott (2007), Barrett and van Rossum (2008)]

1. allows us to concentrate on synapse, not neuron/network
2. No filing system
3. don't care if STDP...
4. looks like binary synapse from outside. Inside...

Dynamics

There are N identical synapses with M internal functional states.



$$\frac{\mathrm{d}\mathbf{p}(t)}{\mathrm{d}t} = r\mathbf{p}(t)\mathbf{W}^{\mathrm{F}}, \qquad \mathbf{W}^{\mathrm{F}} = f^{\text{pot}}\mathbf{M}^{\text{pot}} + f^{\text{dep}}\mathbf{M}^{\text{dep}} - \mathbf{I},$$

$$\mathbf{p}^\infty\mathbf{W}^{\mathrm{F}} = 0.$$

1. stoch process has steady state.
2. Prior activity puts it in this state. row vec.
3. plasticity events at rate r
4. fraction pot/dep
5. probs changed by Markov matrices, prob $i \to j$
6. Readout: synaptic weight vec when in each state.
7. Memory at $t = 0$, keep track of pot/dep
8. subsequent: average over pot/dep

Modelling VOR learning

Mutation:	Changes mechanism of LTD	$\Longrightarrow$ change $\mathbf{M}^{\text{dep}}$.
Training:	Changes statistics of LTP/LTD	$\Longrightarrow$ change r, f^{pot}, f^{dep}.
Learning:	Change in VOR gain	$\Longrightarrow$ decrease in $\langle \mathbf{w} \rangle$.

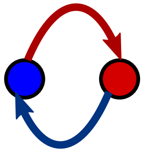
Modelling VOR learning
Mutation: Changes mechanism of LTD $\Longrightarrow$ change $\mathbf{M}^{\text{dep}}$.
Training: Changes statistics of LTP/LTD $\Longrightarrow$ change r, f^{pot}, f^{dep}.
Learning: Change in VOR gain $\Longrightarrow$ decrease in $\langle \mathbf{w} \rangle$.

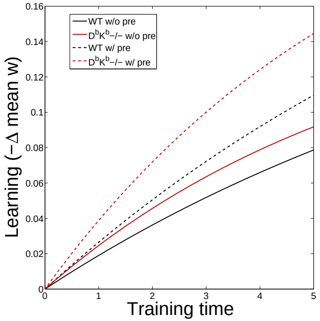
1. lower threshold $\rightarrow$increase off-diagonal elements.
2. Only parameters we have. Don't care about r.
3. Only output we have. Don't keep track of synaptic identity.

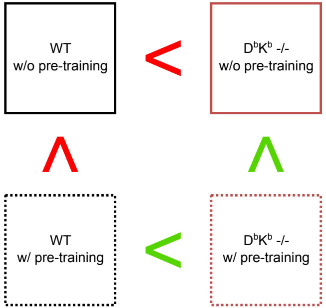
Section 4

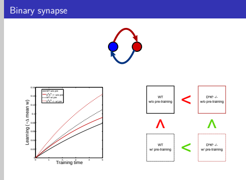
Modelling results

Section 4
Modelling results





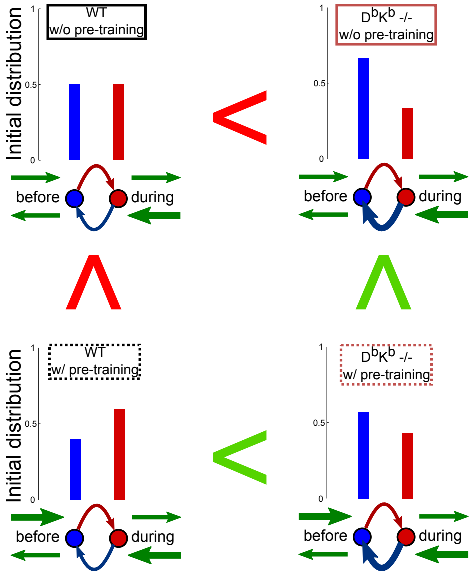


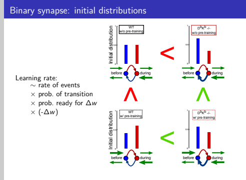


1. Compare solid curves
2. Compare black curves
3. understand why next slide

Binary synapse: initial distributions

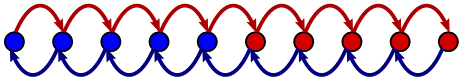
Learning rate:
- $\sim$ rate of events
- $\times$ prob. of transition
- $\times$ prob. ready for Δw
- $\times$ (-Δw)

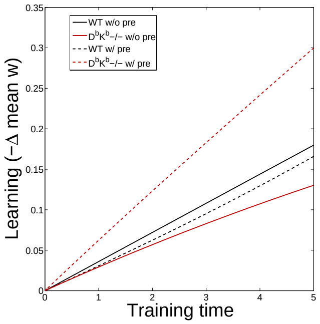


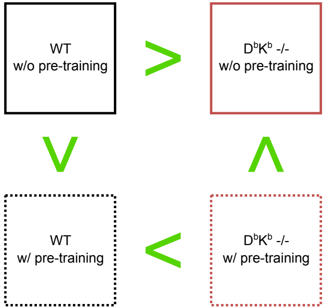


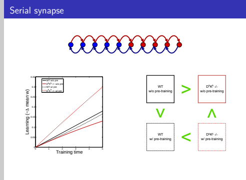
1. WT: start with everything equal – just for illustration, not essential
2. WT: during training, increase f^{dep} (green arrow) $\to$ weakening.
3. KO: inc q^{pot} $\to$ bias
4. KO: competition between inc prob trans & dec prob ready
5. KO: first one wins. see why after next model
6. pre: reduces/reverses bias. always helps.

Serial synapse



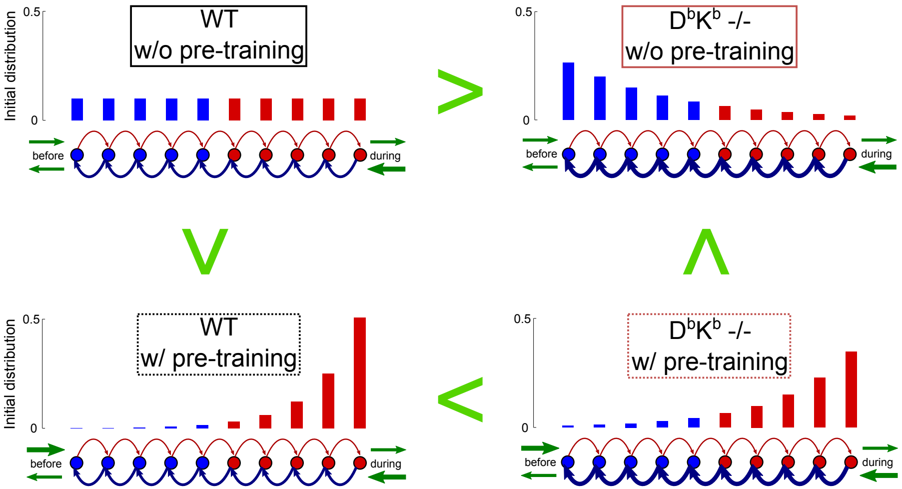


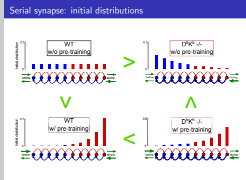




1. Still looks binary from outside. Hidden states (not essential).
2. Only see Δw at boundary.
3. understand why next slide

Serial synapse: initial distributions





1. WT: start with everything equal – just for illustration, not essential
2. WT: during training, increase f^{dep} (green arrow) $\rightarrow$ weakening.
3. KO: inc q^{pot} $\rightarrow$ bias, now exponential
4. KO: comp. between inc prob trans & dec prob ready, now only at bndry
5. KO: second one wins, now exponential
6. pre: reduces/reverses bias.
7. pre: little reverse bias repopulates bndry, helps.
8. pre: too much reverse bias moves away from bndry, hurts.
9. maths next slide

Mathematical explanation

Serial synapse: $\mathbf{p}_i^\infty \sim \mathcal{N}\left(\frac{q^{\text{pot}}}{q^{\text{dep}}}\right)^i$.

Learning rate $\sim \mathbf{p}_{M/2}^\infty \left(\frac{q^{\text{dep}}}{q^{\text{pot}}}\right) = \mathcal{N}\left(\frac{q^{\text{pot}}}{q^{\text{dep}}}\right)^{\frac{M}{2}-1}$.

For $M > 2$: larger $q^{\text{dep}} \implies$ slower learning.

For $M = 2$: larger $q^{\text{dep}} \implies$ larger $\mathcal{N} \implies$ faster learning.

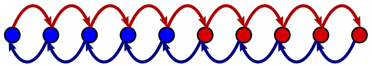
Mathematical explanation
Serial synapse: $\mathbf{p}_i^\infty \sim \mathcal{N}\left(\frac{q^{\text{pot}}}{q^{\text{dep}}}\right)^i$.
Learning rate $\sim \mathbf{p}_{M/2}^\infty \left(\frac{q^{\text{dep}}}{q^{\text{pot}}}\right) = \mathcal{N}\left(\frac{q^{\text{pot}}}{q^{\text{dep}}}\right)^{\frac{M}{2}-1}$.
For $M > 2$: larger $q^{\text{dep}} \implies$ slower learning.
For $M = 2$: larger $q^{\text{dep}} \implies$ larger $\mathcal{N} \implies$ faster learning.

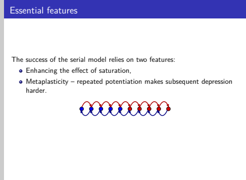
1. Detailed balance. Exponential decay.
2. for large enough M, q^{pot}, overcome $\mathcal{N}$
3. Other factor in $\mathbf{p}^\infty$ smaller $\implies \mathcal{N}$ larger.

Essential features

The success of the serial model relies on two features:

- Enhancing the effect of saturation,
- Metaplasticity – repeated potentiation makes subsequent depression harder.

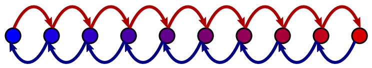


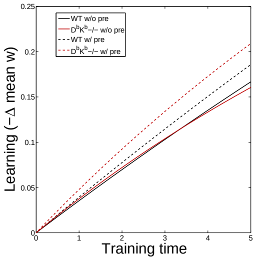


1. due to exponential decay
2. push away from boundary where signal generated
3. borne out by other models that fail/succeed

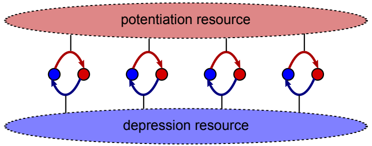
Other models that fail

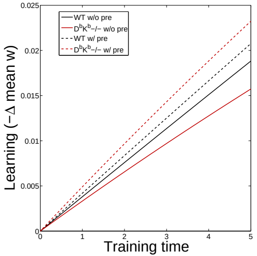
Multistate model



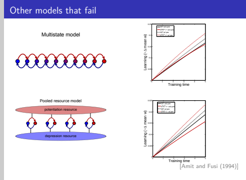


Pooled resource model





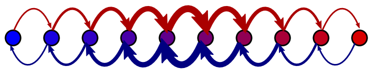
[Amit and Fusi (1994)]

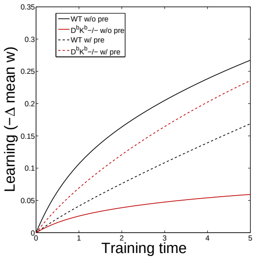


1. MS: linear weights, unlike serial.
2. like bunch of binary synapses in series.
3. solid curves: fails early on , but catches up quickly
4. black curves: fails badly
5. No real enhancement of saturation, no metaplasticity.
6. All transitions contribute: pushing to end has little effect.
7. Pooled: resource depleted by pot/dep. replenished by reverse.
8. solid curves succeed: enhanced saturation
9. black curves fail: opposite metaplasticity, pot makes dep easier

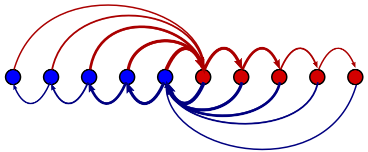
Other models that work

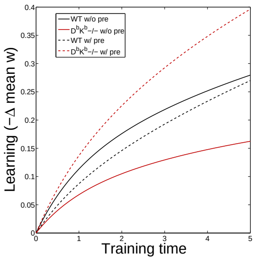
Non-uniform multistate model



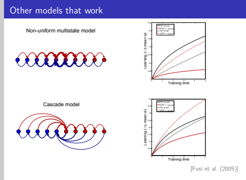


Cascade model





[Fusi et al. (2005)]



1. Both models, trans probs decay exponentially from centre.
2. Nonuni: linear weights. Cascade: binary weights.
3. Enhanced saturation and metaplasticity
4. Pushing to end makes pot and dep harder
5. Note: hidden states not necessary

Conclusions and further questions

- The saturation effect overcome the enhanced plasticity, if it is enhanced. Requires complexity
- A little reverse bias can help, but too much hurts, if repeated potentiation makes depression harder. Requires metaplasticity
- We can find a purely synaptic explanation of VOR behaviour, iff the synapses have these features.
- We used behaviour to constrain molecular structure of synapses!
- Can we constrain it further with more experiments?

Conclusions and further questions
- The saturation effect overcome the enhanced plasticity, if it is enhanced. Requires complexity
- A little reverse bias can help, but too much hurts, if repeated potentiation makes depression harder. Requires metaplasticity
- We can find a purely synaptic explanation of VOR behaviour, iff the synapses have these features.
- We used behaviour to constrain molecular structure of synapses!
- Can we constrain it further with more experiments?

1. e.g. exponential deacy, resource depletion,. . .
2. e.g. moving away from weight boundary, or weaker transitions.
3. Other explanations? Non-linearity in PK cell?

References I

- S. Fusi, P. J. Drew, and L. F. Abbott.
  "Cascade models of synaptically stored memories".
  Neuron, 45(4):599–611, Feb 2005.
  11 22
- S. Fusi and L. F. Abbott.
  "Limits on the memory storage capacity of bounded synapses".
  Nat. Neurosci., 10(4):485–493, Apr 2007.
  11
- A. B. Barrett and M. C. van Rossum.
  "Optimal learning rules for discrete synapses".
  PLoS Comput. Biol., 4(11):e1000230, Nov 2008.
  11

2013-07-10

References I

S. Fusi, P. J. Drew, and L. F. Abbott.
"Cascade models of synaptically stored memories".
Neuron, 45(4):599–611, Feb 2005.
S. Fusi and L. F. Abbott.
"Limits on the memory storage capacity of bounded synapses".
Nat. Neurosci., 10(4):485–493, Apr 2007.
A. B. Barrett and M. C. van Rossum.
"Optimal learning rules for discrete synapses".
PLoS Comput. Biol., 4(11):e1000230, Nov 2008.

References II

D. J. Amit and S. Fusi.
"Learning in neural networks with material synapses".
Neural Computation, 6(5):957–982, 1994.
21

References II
D. J. Amit and S. Fusi.
"Learning in neural networks with material synapses".
Neural Computation, 6(5):957–982, 1994.
21