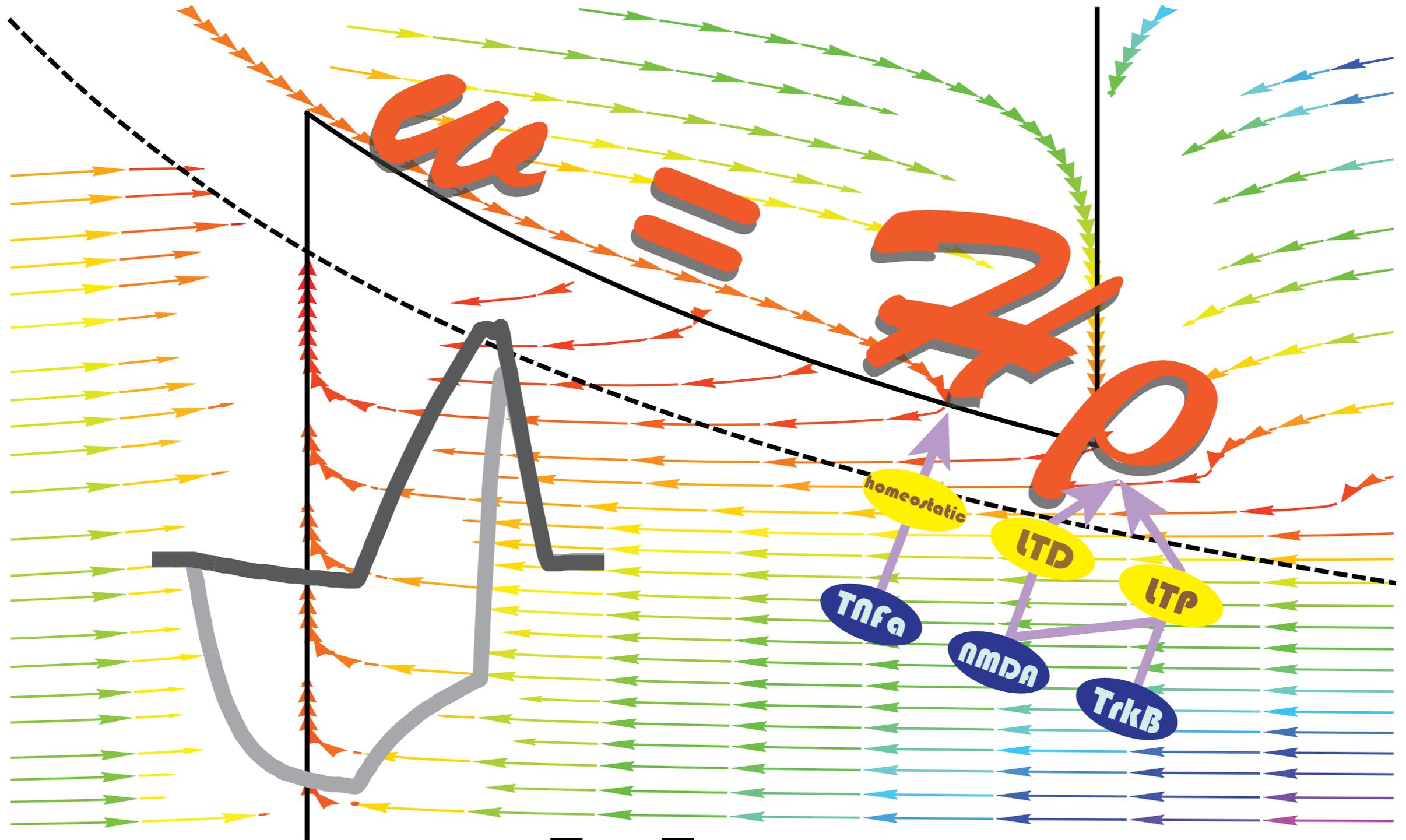


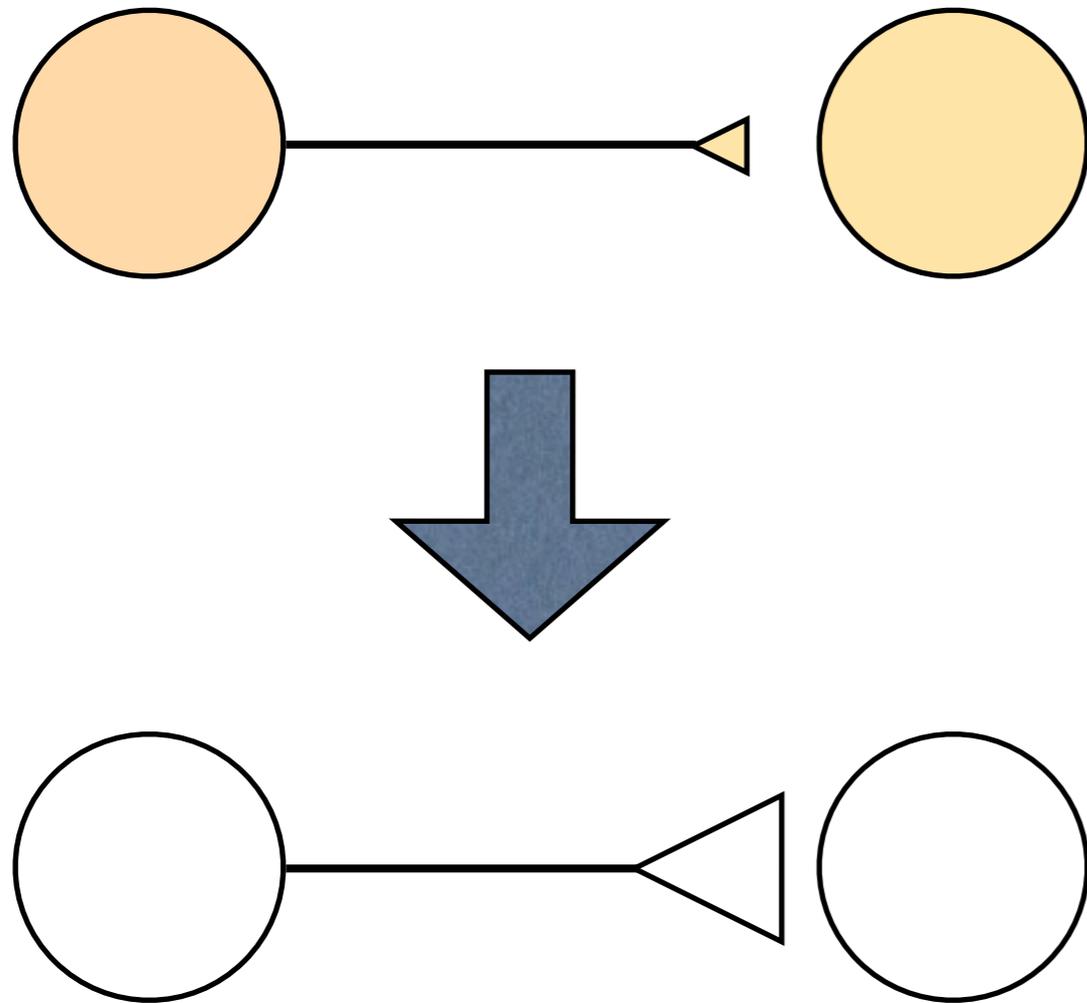
Modeling the dynamic interaction of Hebbian and homeostatic plasticity



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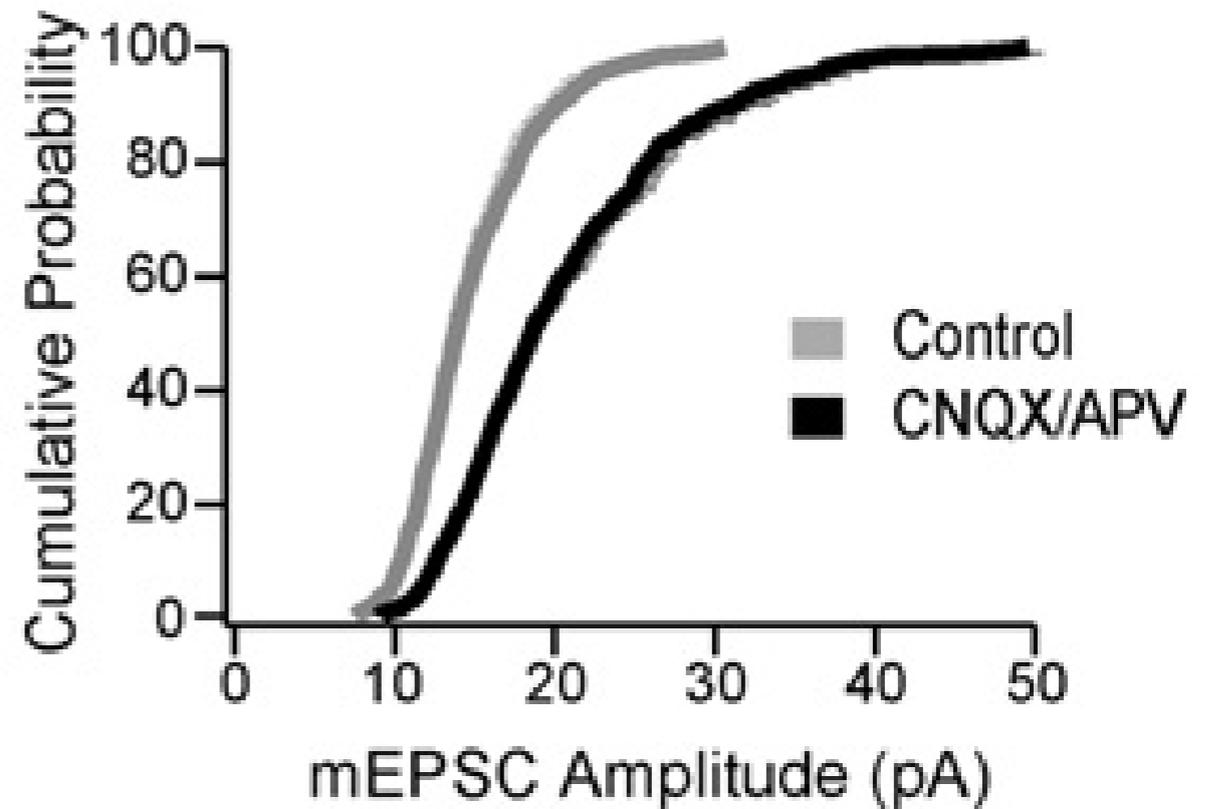
Hebbian plasticity

“neurons that fire together wire together”



homeostatic plasticity

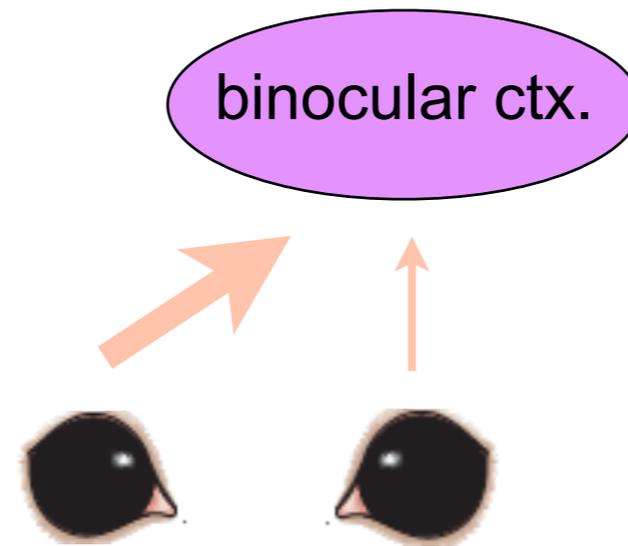
scale synaptic strength to maintain activity



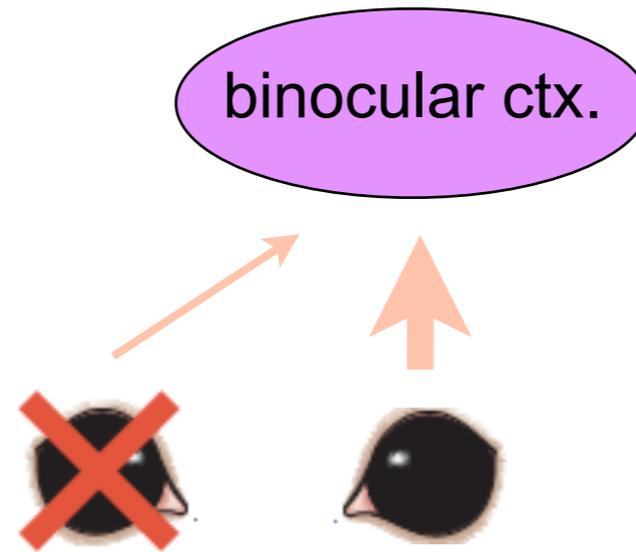
Kaneko et al. 2008b

Interaction?

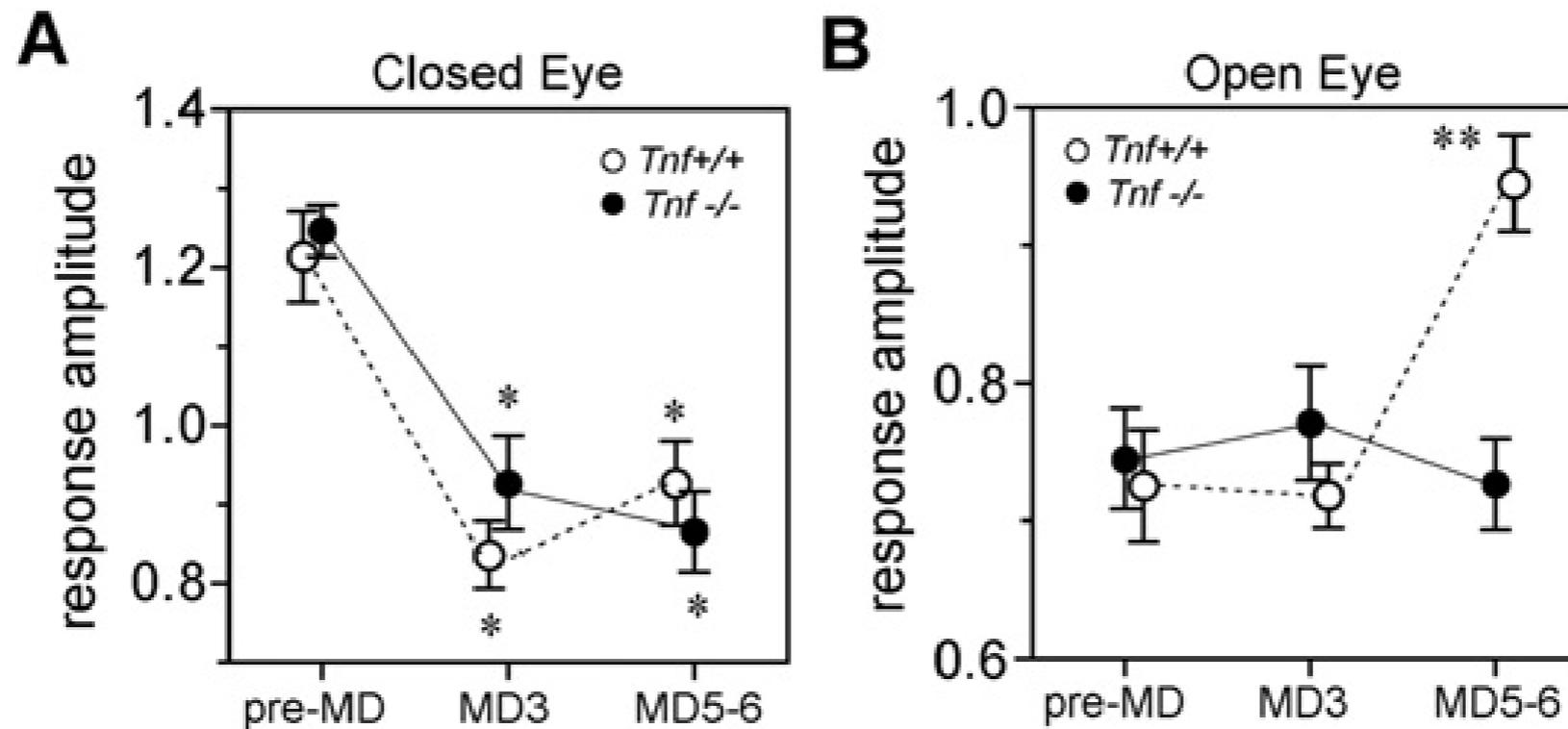
Ocular dominance plasticity in V1: biological mechanisms and dynamics



Ocular dominance plasticity in V1: biological mechanisms and dynamics



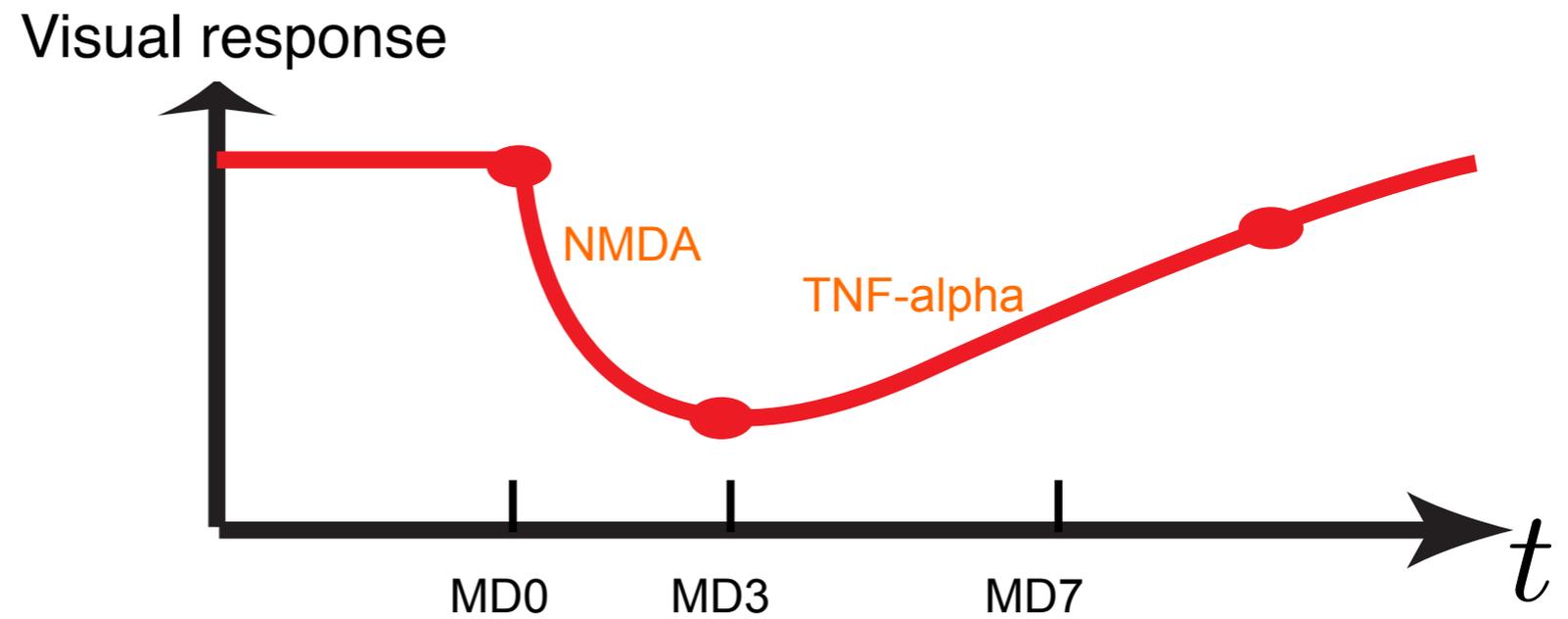
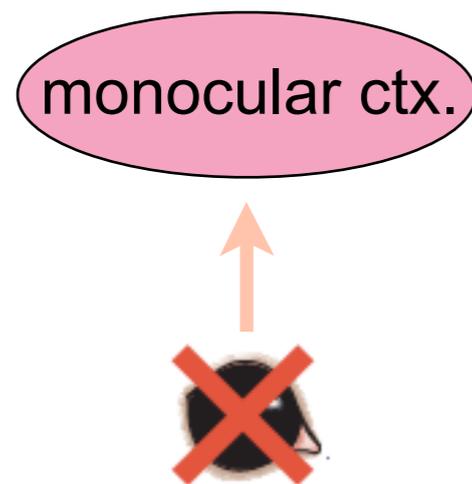
Ocular dominance plasticity in V1: biological mechanisms and dynamics



- *Fast NMDA dependent depression of the deprived-eye (MD0-MD3)*
- *Slow TNF-alpha mediated potentiation of the open-eye (MD3-MD6)*
- *BDNF-TrkB dependent recovery from MD (under binocular vision)*

MD result in the monocular cortex

Fast Hebbian depression and slow homeostatic potentiation



Kaneko et al. 2008b

Conventional models assume that the two kinds of plasticity cancel each other.

“fast”
Hebbian

“slow”
homeostatic

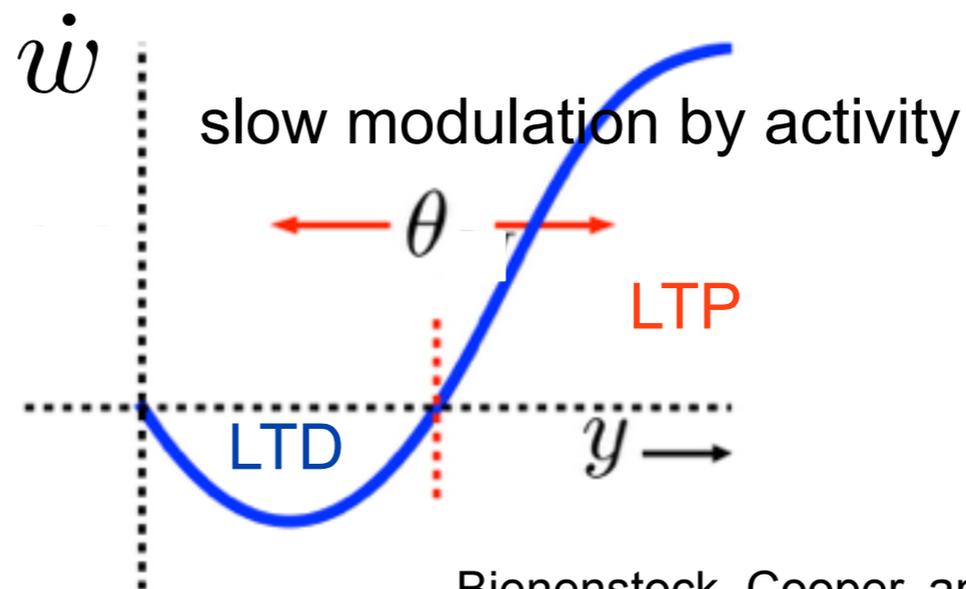


How can homeostasis be powerful enough and slow at the same time?

There are two ways to impose slow homeostasis.

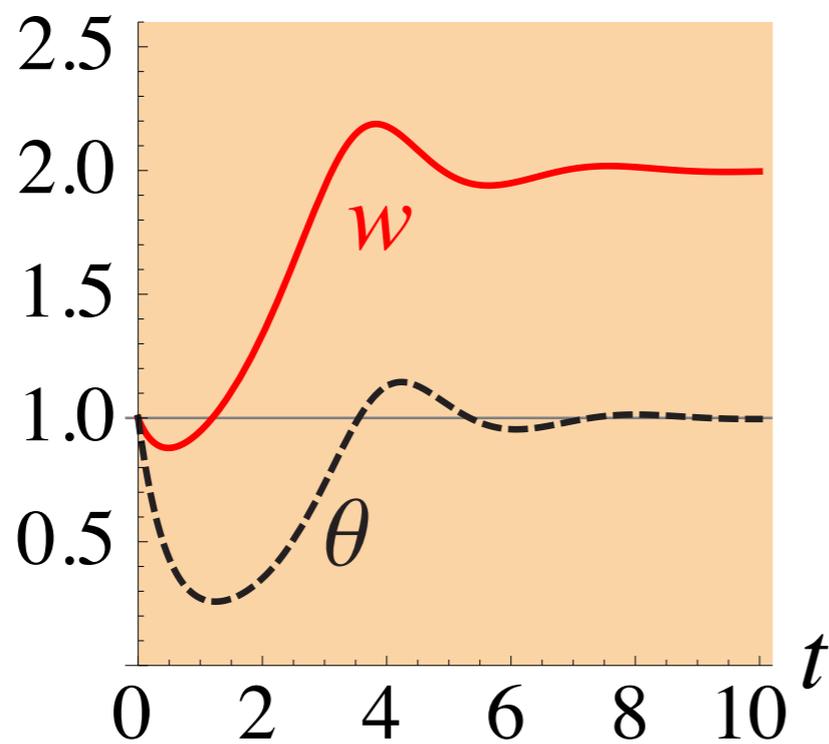
- Weak homeostasis (small magnitude) tends to be overwritten by powerful depression.
- Slow averaging or delay tends to cause oscillation of synaptic weights.

Model 1: BCM rule

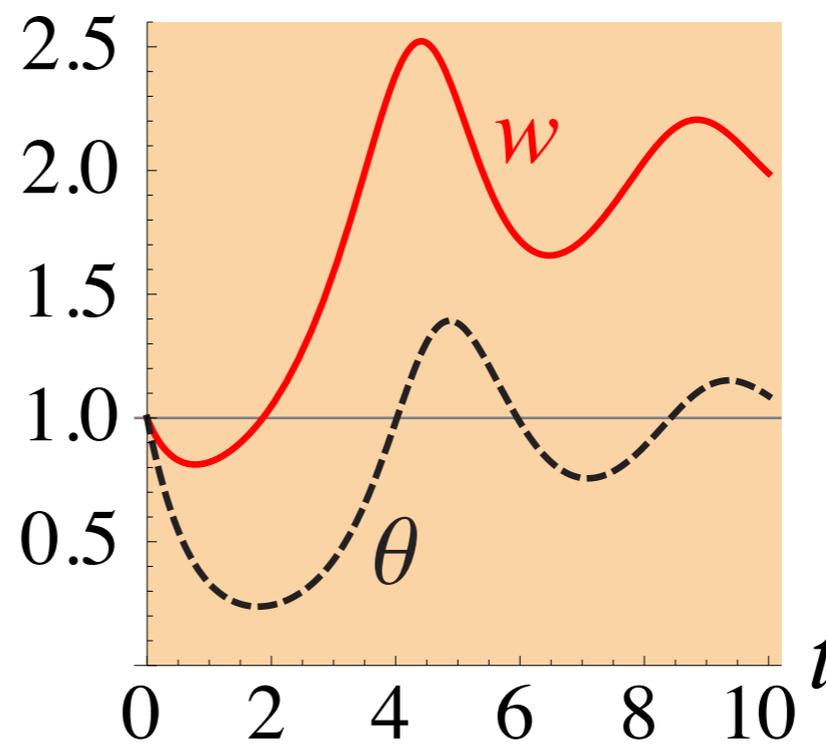


Bienenstock, Cooper, and Munro, *J. Neurosci.* 1982.

fast threshold change



slow threshold change



When the threshold change is too slow, strong oscillation happens.

Slow feedback control cannot catch up with the fast unstable Hebbian component, which generally causes oscillation.

Model 2: Stable Hebbian and multiplicative homeostatic plasticity

$$y = wx$$

Hebbian

homeostatic

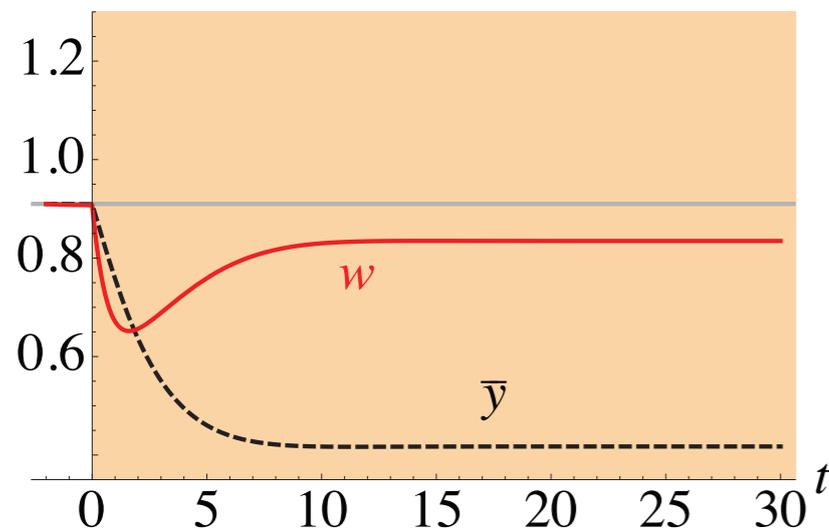
$$\tau \dot{w} = [1 - w]_+ [xy - \theta]_+ - [w - w_{\min}]_+ [\theta - xy]$$

$$\dot{\bar{y}} = -\bar{y} + y$$

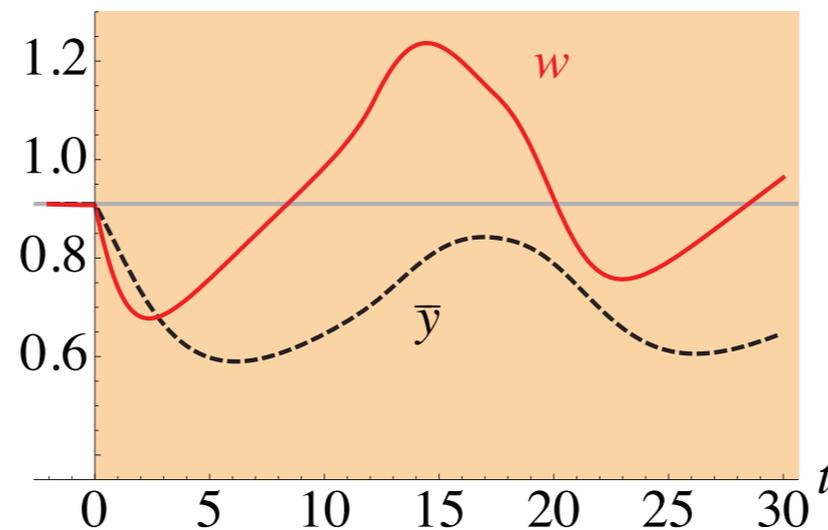
$$[x]_+ = \begin{cases} x & (\text{if } x > 0) \\ 0 & (\text{otherwise}) \end{cases}$$

A

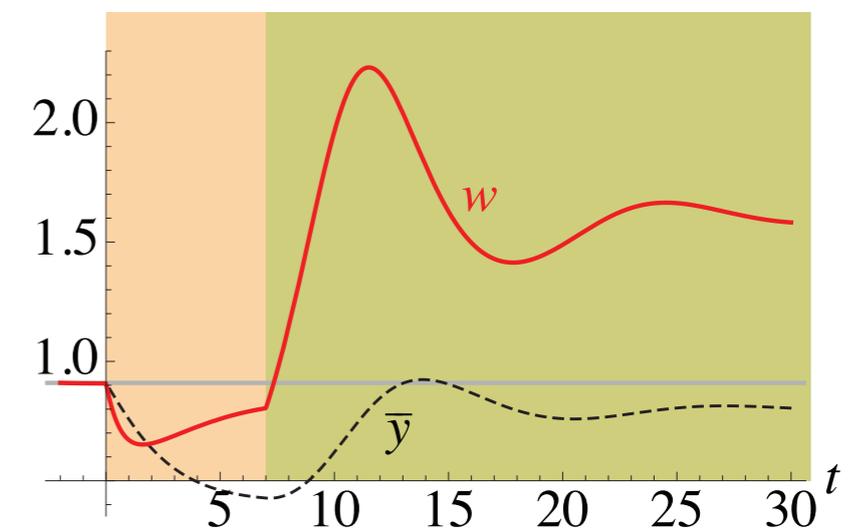
Strong MD



Weak MD



NMDA blockade

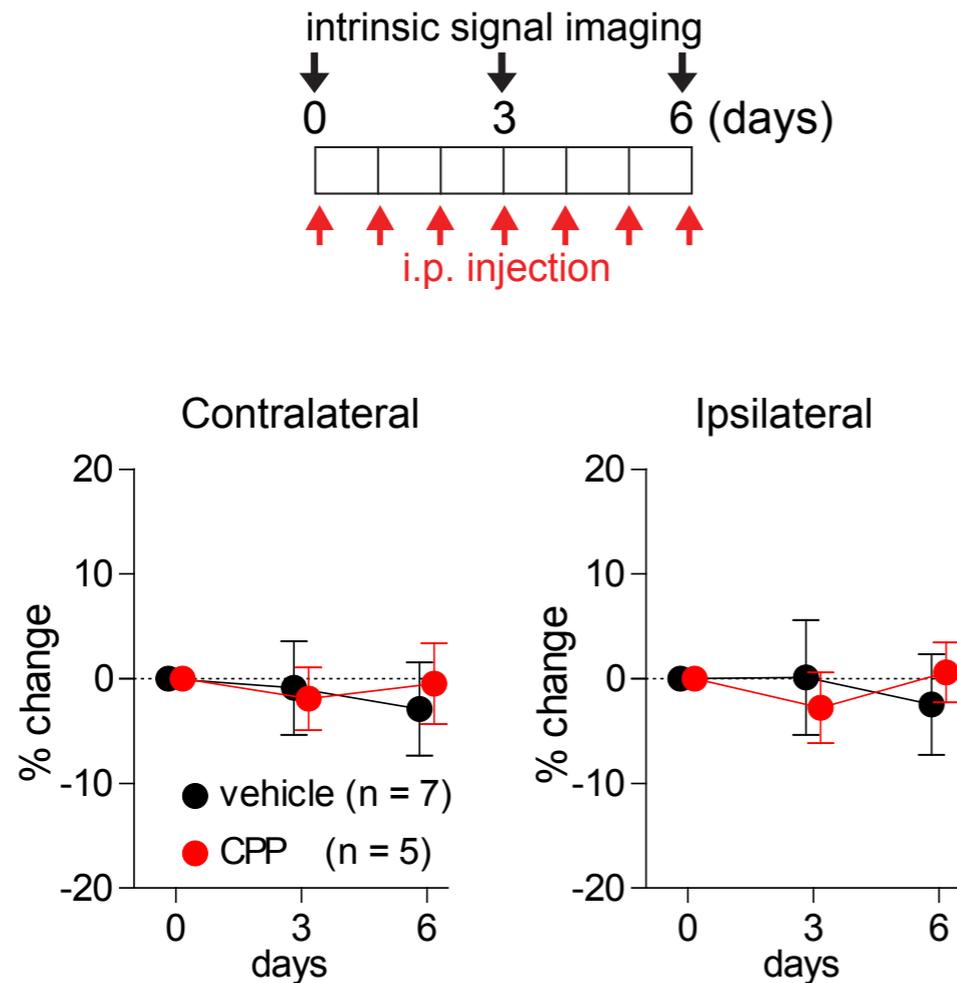


Fine tuning is required — otherwise oscillatory.

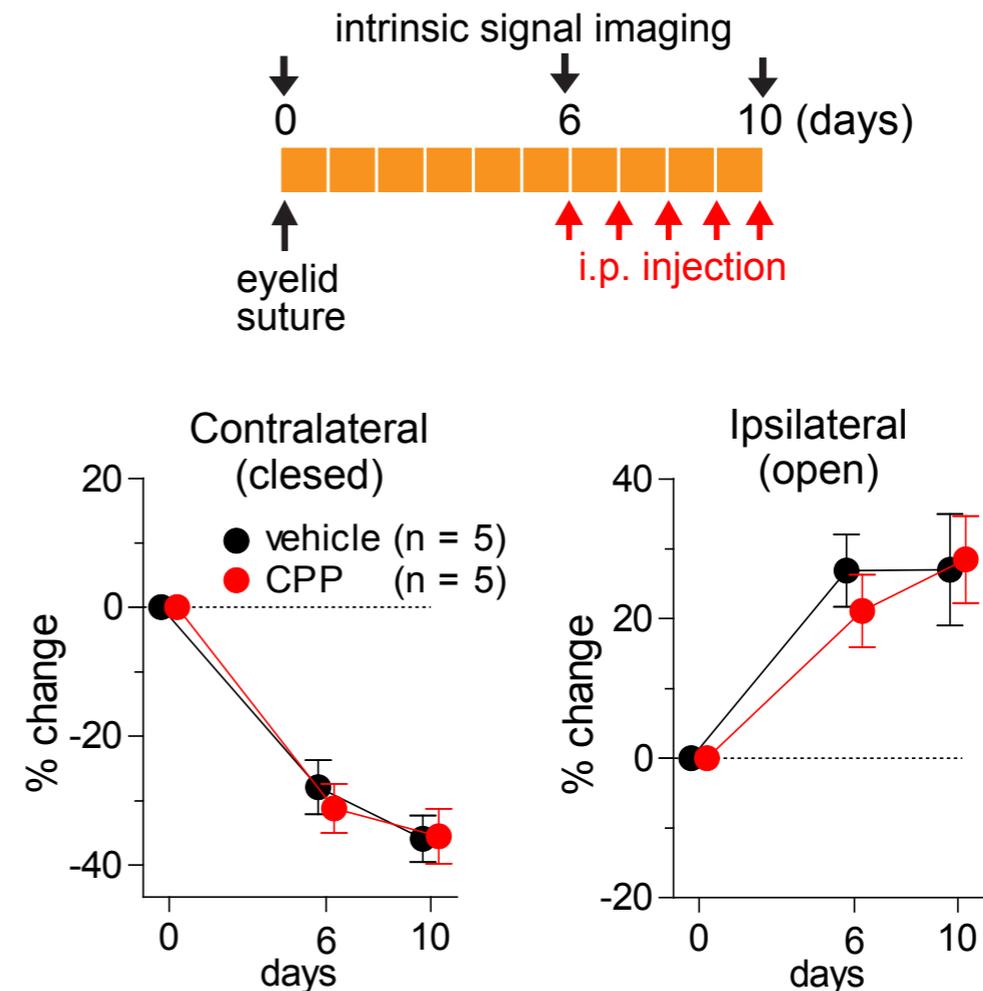
This interaction predicts an alteration of visual response if Hebbian plasticity is blocked.

Hebbian and homeostatic plasticity are not constitutively active and balanced

NMDA blockade under normal condition

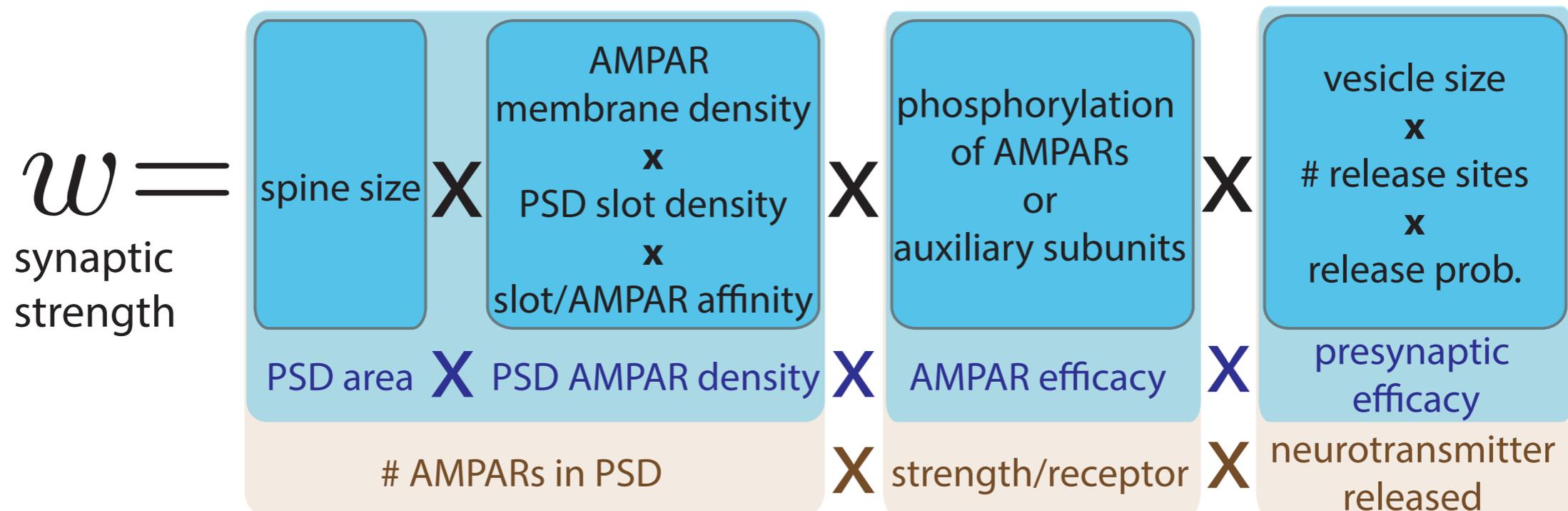
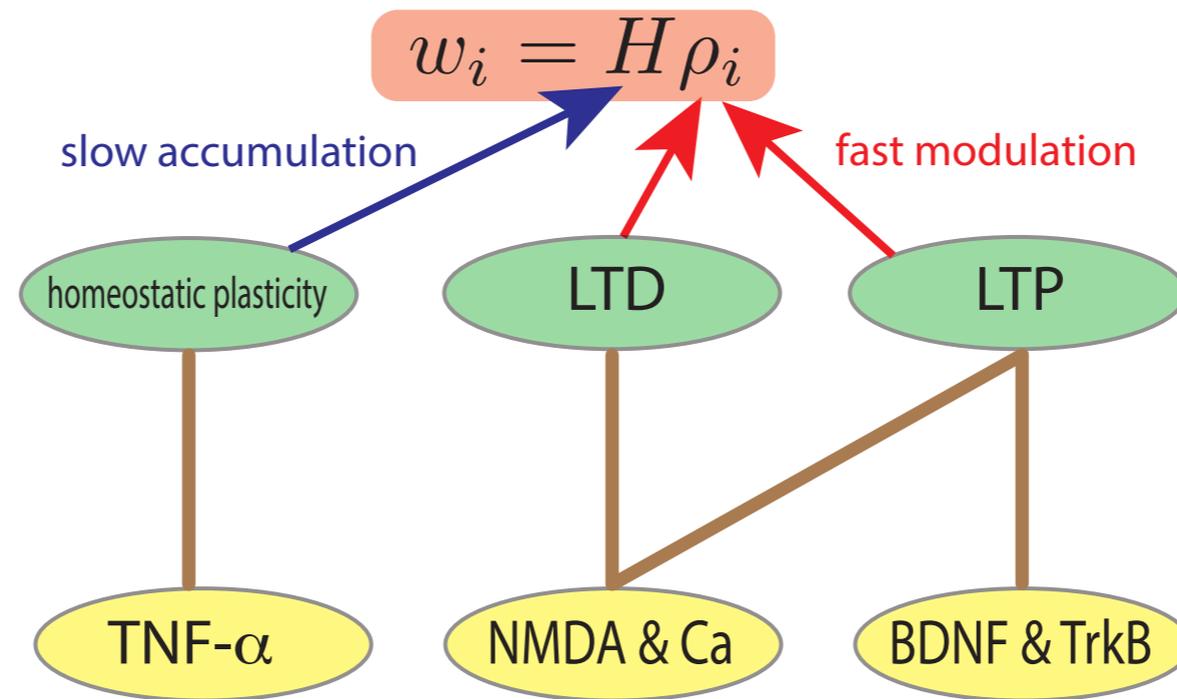


NMDA blockade after MD

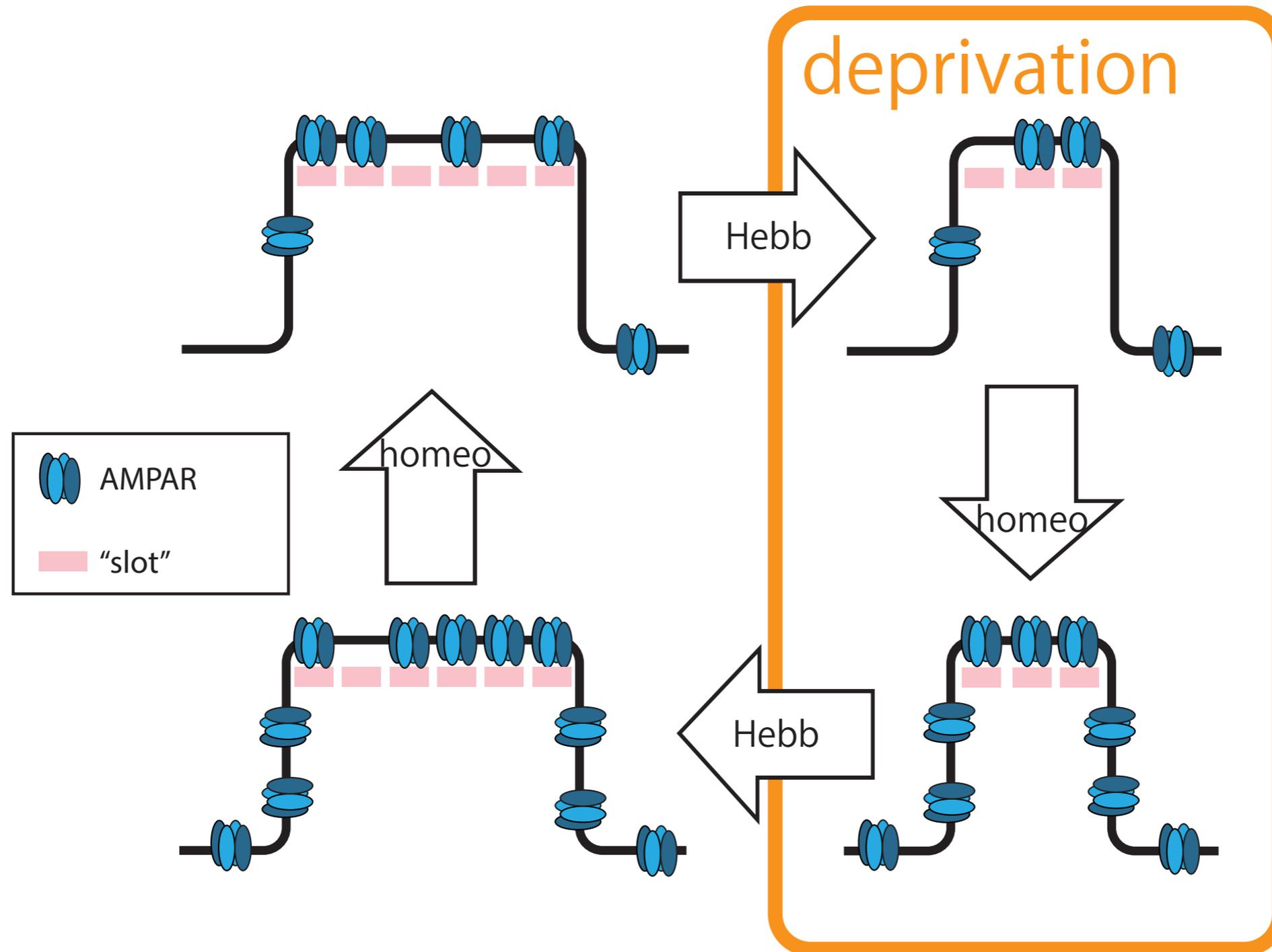


These results indicate that homeostatic plasticity is not active at steady states.

The two-factor model:
synaptic strength as a product of Hebbian and homeostatic variables



Schematic behavior: One possible implementation



The two-factor model

$$y = wx$$

$$w = H\rho$$

$$\tau \dot{\rho} = (1 - \rho)[xy - \theta]_+ - (\rho - \rho_{\min})[\theta - xy]_+$$

$$\dot{H} = H(1 - y)$$

The two-factor model

$$y = wx$$

$$w = H\rho$$

$$\tau\dot{\rho} = (1 - \rho)[xy - \theta]_+ - (\rho - \rho_{\min})[\theta - xy]_+$$

$$\dot{H} = H(1 - y)$$

$$\dot{w} = \dot{H}\rho + H\dot{\rho}$$

Equivalent

$$\tau\dot{w} = (H - w)[xy - \theta]_+ - (w - H\rho_{\min})[\theta - xy]_+ - \tau w(y - 1)$$

$$\dot{H} = H(1 - y)$$

The two-factor model

$$y = wx$$

$$w = H\rho$$

$$\tau\dot{\rho} = (1 - \rho)[xy - \theta]_+ - (\rho - \rho_{\min})[\theta - xy]_+$$

$$\dot{H} = H(1 - y)$$

$$\dot{w} = \dot{H}\rho + H\dot{\rho}$$

Equivalent

$$\tau\dot{w} = (H - w)[xy - \theta]_+ - (w - H\rho_{\min})[\theta - xy]_+ - \tau w(y - 1)$$

$$\dot{H} = H(1 - y)$$

Previous Model 3

$$\tau\dot{w} = [1 - w]_+[xy - \theta]_+ - [w - w_{\min}]_+[\theta - xy]_+ - \gamma w(\nu - 1)$$

$$\dot{\nu} = -\nu + y$$

The two-factor model

$$y = wx$$

$$w = H\rho$$

$$\tau\dot{\rho} = (1 - \rho)[xy - \theta]_+ - (\rho - \rho_{\min})[\theta - xy]_+$$

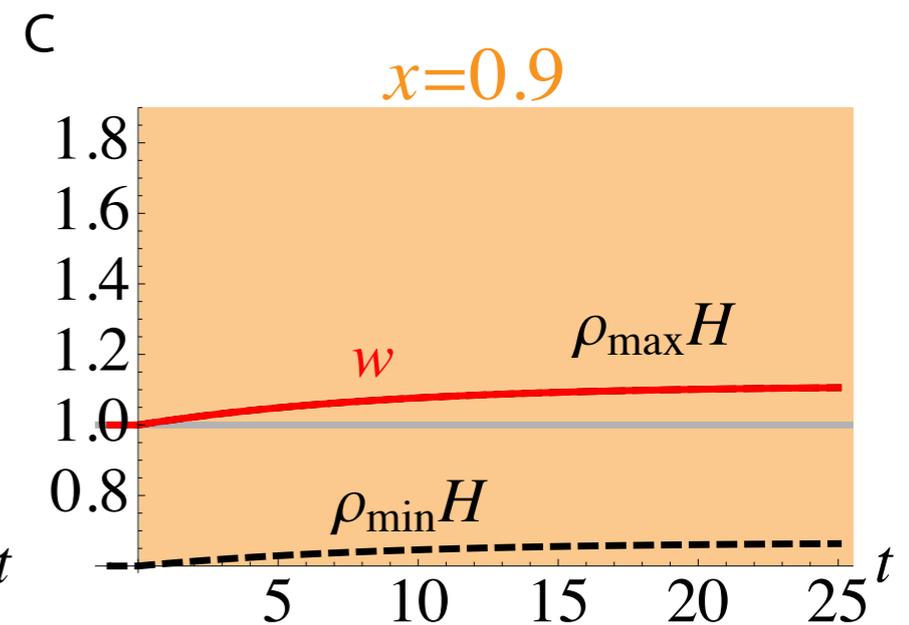
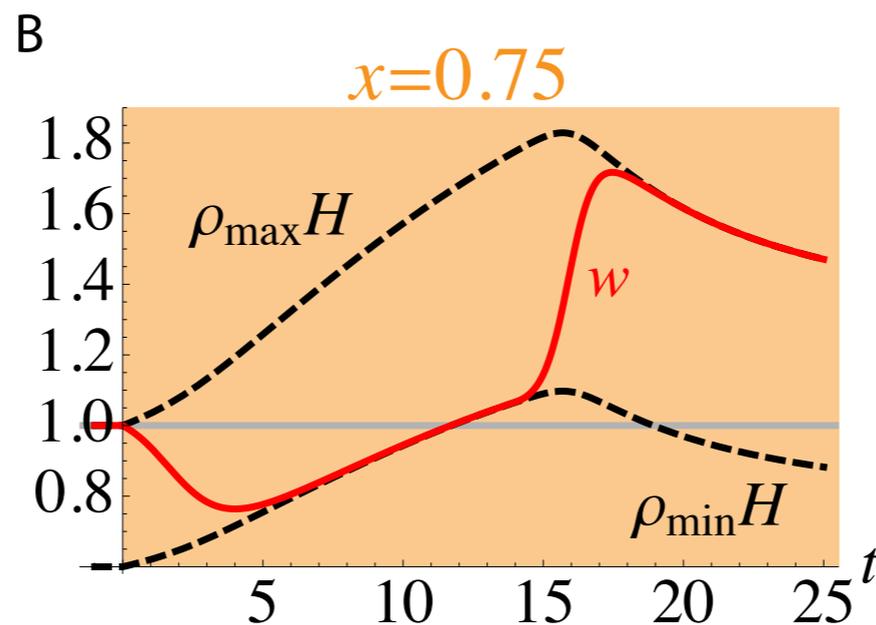
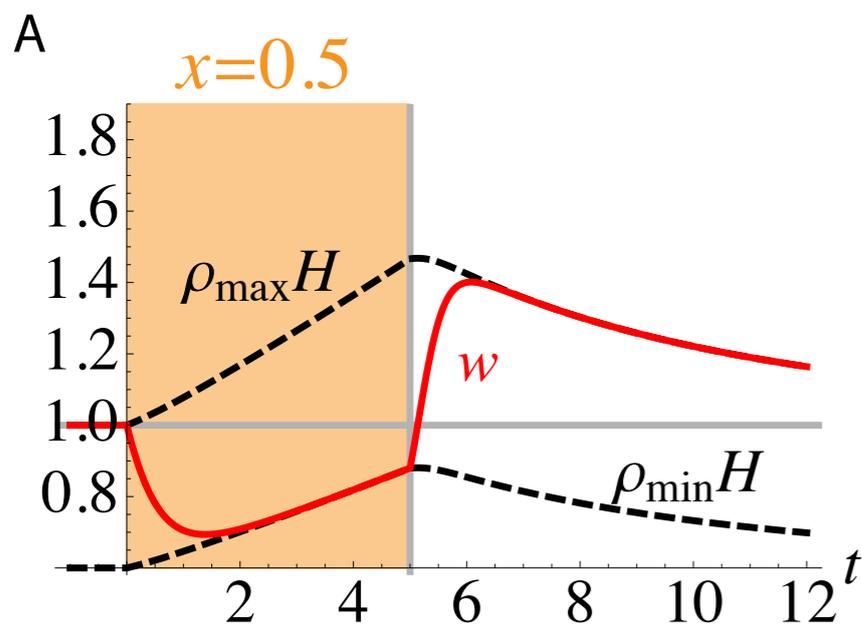
$$\dot{H} = H(1 - y)$$

$$\dot{w} = \dot{H}\rho + H\dot{\rho}$$

Equivalent

$$\tau\dot{w} = (H - w)[xy - \theta]_+ - (w - H\rho_{\min})[\theta - xy]_+ - \tau w(y - 1)$$

$$\dot{H} = H(1 - y)$$



Modeling multiple synapses

$$w_i = H \rho_i$$

BDNF and TrkB

$$\tau_\rho \dot{\rho}_i = (1 - \rho_i) [C_i]_+ - (\rho_i - \rho_{\min}) [-C_i]_+$$

NMDA and Ca

$$\tau_H \dot{H} = f(H, \langle y \rangle)$$

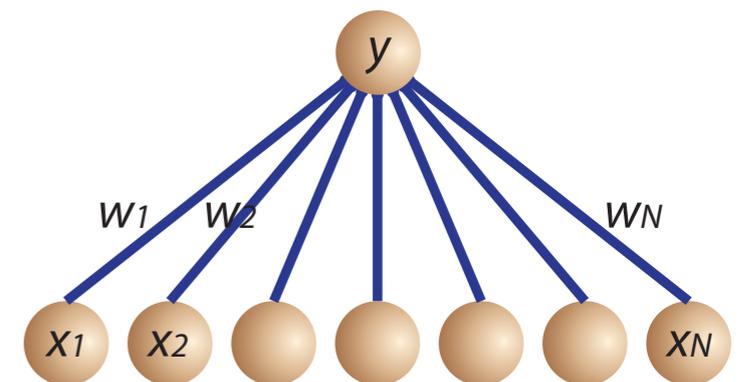
TNF-alpha

Postsynaptic firing rate:

$$y = \sum_{i=1}^N w_i x_i$$

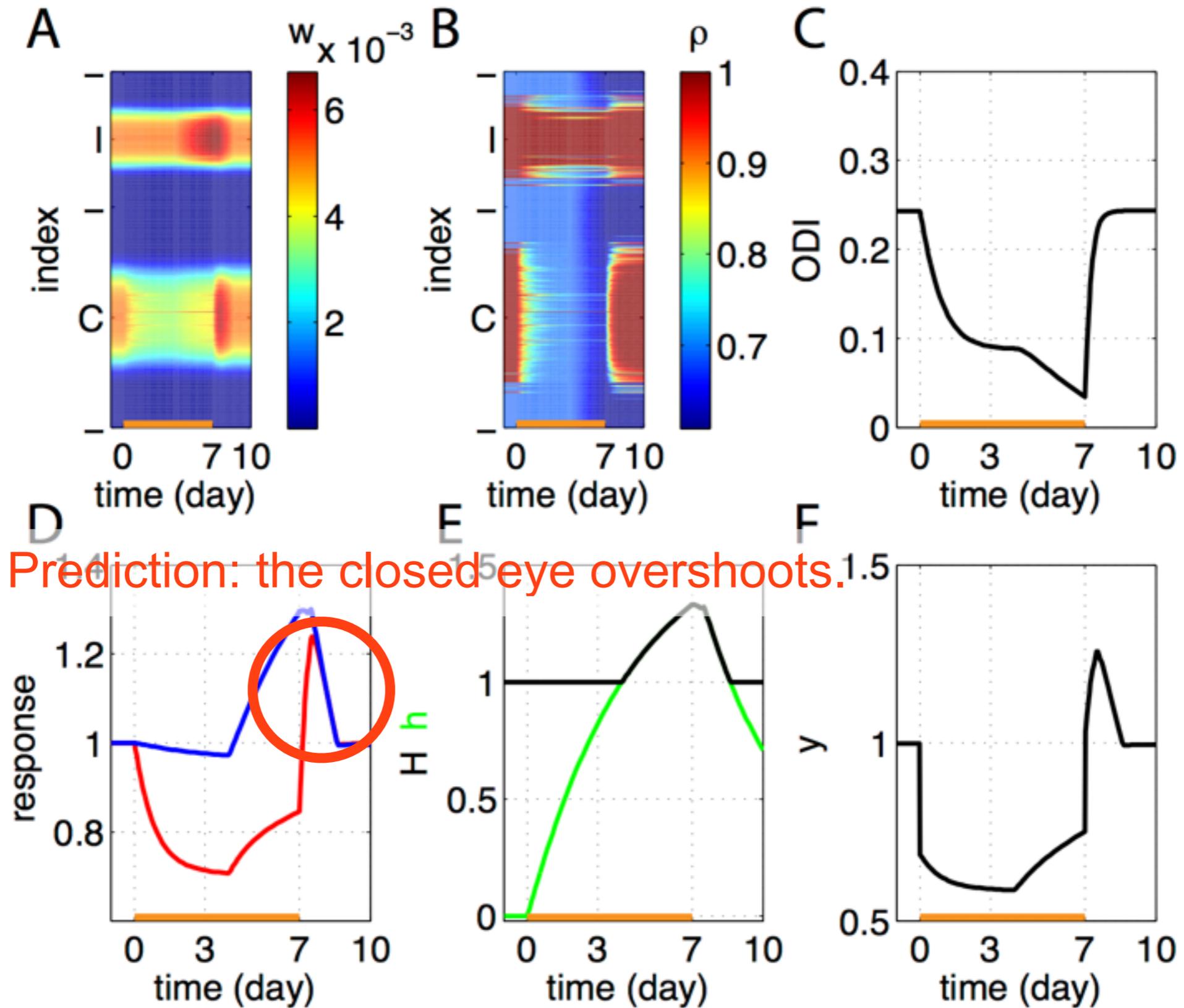
pre-post covariance:
(minus a threshold)

$$C_i = \text{Cov}[x_i, y] - \theta$$

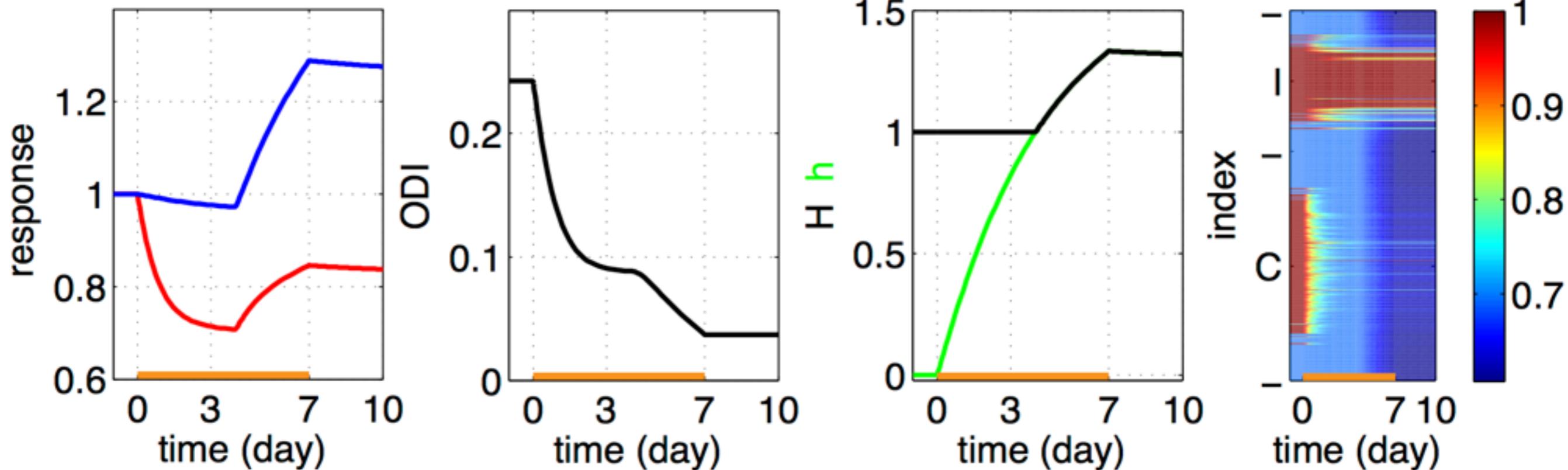


locally correlated Gaussian input

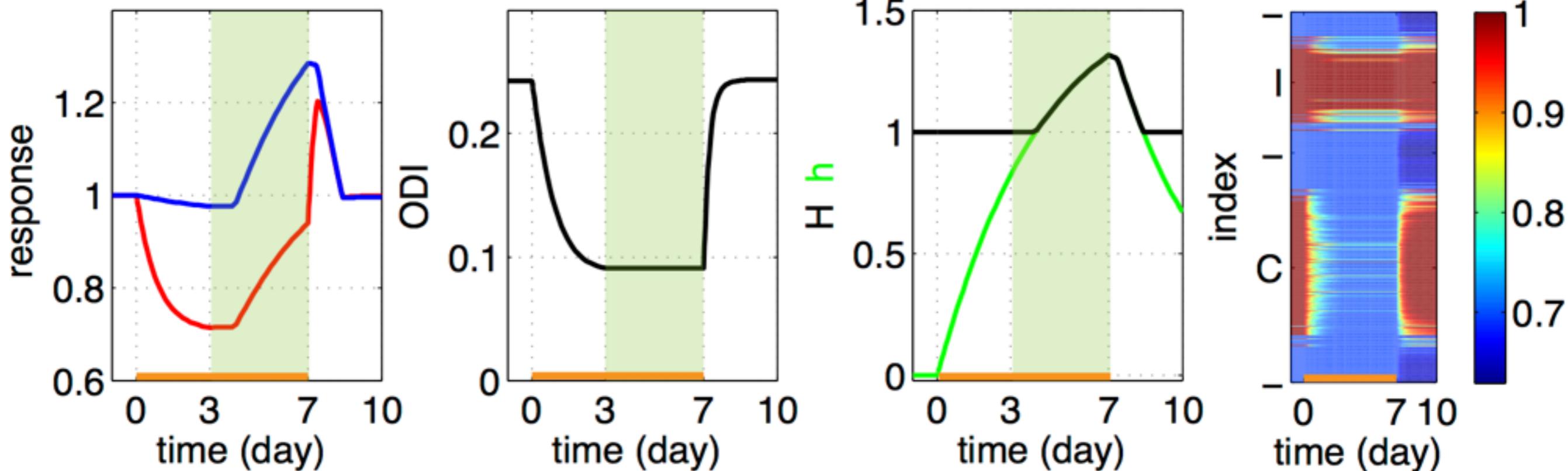
Binocular cortex, MD



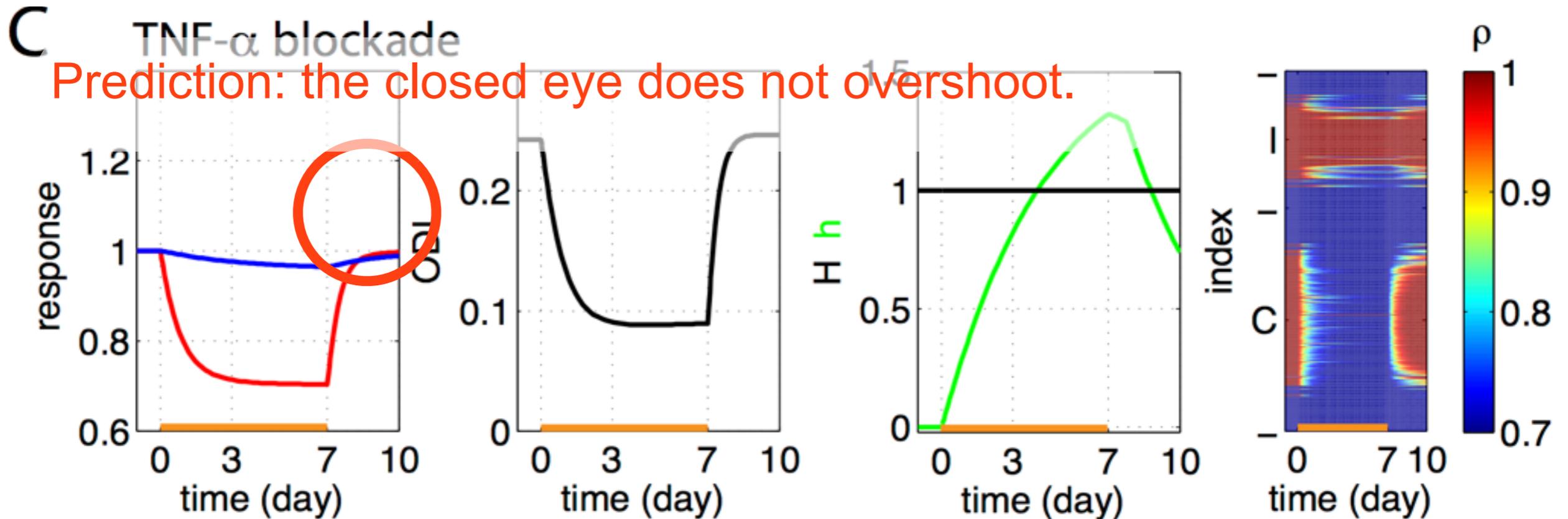
A TrkB inactivation



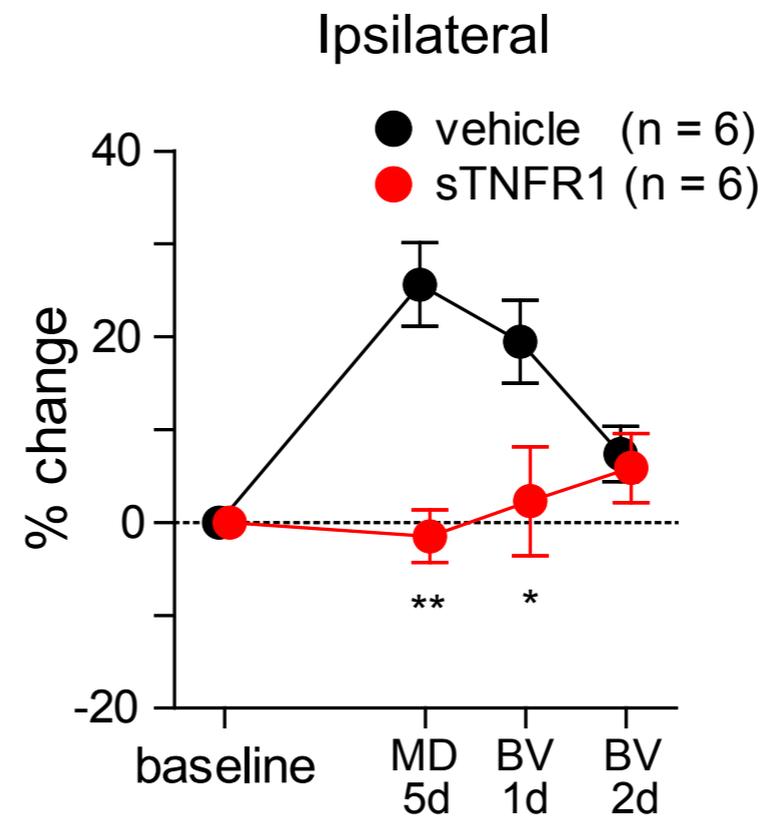
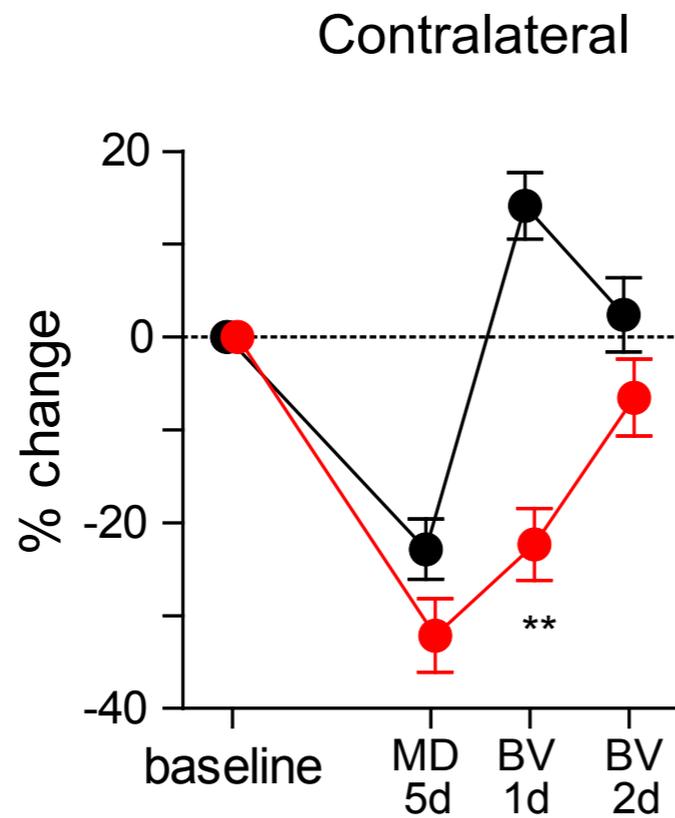
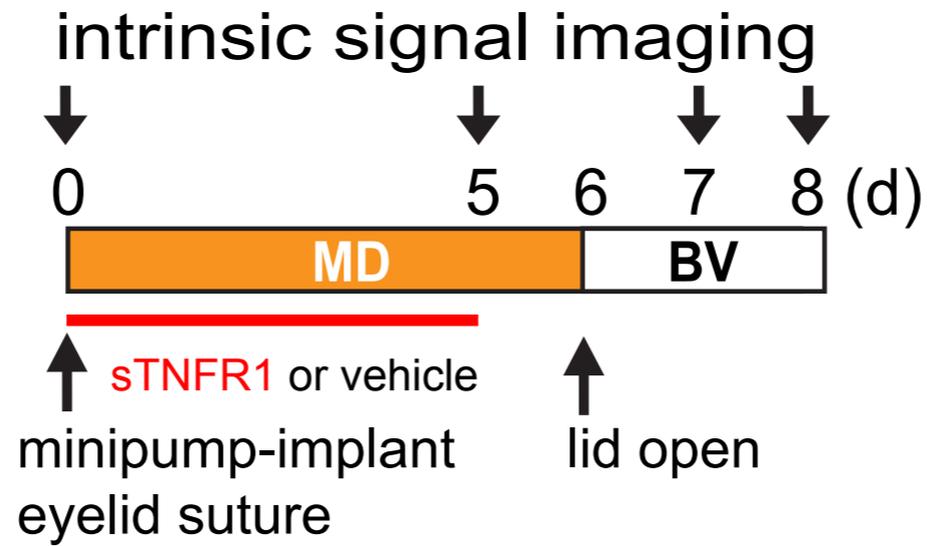
B NMDA blockade



The closed eye overshoot did not happen under the TNF-alpha blockade.



Prediction confirmed: The closed-eye overshoot is TNF-alpha dependent.



Summary

- Hebbian learning is intrinsically unstable except when LTP or LTD is saturated. In existing models, homeostatic plasticity typically stabilizes synapses at non-saturated values of LTP or LTD.

As homeostatic learning is made slow, oscillations of synaptic strengths may occur and ultimately the stabilization fails.

- In the proposed two-factor model, plasticity rule remains stable as homeostasis is made arbitrarily slow. The model has plausible biophysical substrates.
- The model captures the transient behaviors of OD plasticity under various experimental conditions.
- Model's predictions about constitutively inactive plasticity rules and TNF-alpha-dependent previously closed-eye overshoot were experimentally verified.
- Maintaining these two processes through separable factors allows dynamic range for coding to be maintained while allowing Hebbian mechanisms to freely learn synaptic patterns without interference.
- The dynamical interaction we propose here may describe a key biological principle underlying memory and learning in neuronal circuits.

Collaborators

Megumi Kaneko (UCSF)
Michael P. Stryker (UCSF)
Kenneth D. Miller (Columbia)

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