

**Adaptation-induced plasticity in V1:
adapting
and
anti-adapting neurons**

By

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CERTIFICATE

This is to certify that the thesis entitled “Adaptation-induced plasticity in V1: adapting and anti-adapting neurons” being submitted by Mr. Ketan Bajaj for the award of the degree of Doctor of Philosophy in the Department of Electrical Engineering, the Indian Institute of Technology, Delhi, is a record of bona fide work done by him under my supervision and guidance. The matter embodied in this thesis has not been submitted for the award of any other degree or diploma.



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*I bow before the Consciousness manifested through
my mother, my father & my teacher,
my wife & my children, my brother & my sister,
and my friends & my relatives
which has been with me
while I have been working for my PhD thesis.*

*I bow before the Consciousness
which has been a continuous source of
motivation, inspiration,
guidance, strength and support.*

*I bow before the Consciousness
which is within me
and within which I am.*

*I bow before the Consciousness
which I have been searching for
and
will continue to do so...*

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Abstract

We develop a model that predicts how long duration adaptation while causing a decrease in excitability of adapting neurons can cause an increase in excitability of 'anti-adapting' neurons, even without any synaptic interactions between these groups of neurons. Adapting V1 neurons have orientation preferences similar to or near adapting stimulus; whereas anti-adapting neurons have orientation preferences near orthogonal to the adapting. The model includes neural and glial intrinsic mechanisms and dynamics in the intra- and extracellular space. Adapting neurons accumulate calcium and sodium ions within their cytoplasm and release potassium to the extracellular space. This leads to an ionic imbalance in the local neural tissue causing calcium depletion and potassium accumulation in the narrow free extracellular space. Prolonged spiking activity of adapting neurons also leads to an increase in intracellular calcium in nearby glial cells. These extracellular disturbances spread towards anti-adapting neurons via diffusion in the free extracellular and via glial assisted mechanisms such as glial spatial buffering of potassium and glial calcium responses.

Intracellular ionic disturbances cause a decrease in excitability, whereas extracellular calcium depletion, potassium accumulation and glial calcium responses contribute to an increase in neural excitability. Decrease and increase in neural excitability due to these mechanisms oppose each other. Following adaptation when preferred test stimuli are presented, adapting neurons exhibit a net decrease in excitability; response suppression due to intracellular disturbances dominates response facilitation due to extracellular disturbances. On the other hand, anti-adapting neurons exhibit a net increase in excitability; as there is little suppression due to intracellular disturbances but there is substantial response facilitation due to extracellular disturbances. We discuss experimental evidence that supports our model conclusions. To our knowledge this is the first ever model for adaptation that includes elaborate mechanisms as discussed here.

We then apply our model to explain various experimental observations. Long-duration adaptation causes a repulsive shift in orientation tuning curves of primary visual

cortical cells. This adaptation-induced orientation plasticity is prominent at pinwheel centers, where following adaptation, orientation tuning curves exhibit suppression at adapting flank and facilitation at opposite flank. We predict that adaptation-induced decrease in excitability of adapting neurons and increase in excitability of anti-adapting neurons is able to explain multiple features of adaptation-induced orientation plasticity.

We use our model to reproduce tilt after-effect, a well known perceptual after-effect of adaptation and then predict how attention to stimulus orientation can reduce tilt after-effect and adaptation-induced orientation plasticity. We then apply the model to predict how adaptation-induced increase in excitability of anti-adapting neurons can facilitate discrimination of two stimuli having orientation near orthogonal-to-adapting. We further predict how long-duration adaptation followed by presentation of orthogonal-to-adapting test stimulus can facilitate potentiation of synapses from anti-adapting neurons. We suggest explanations for aggravated tilt aftereffect in migraine, motion aftereffect and spatial frequency aftereffect. We discuss how our model represents stress, adaptation and homeostasis in context of primary visual cortex.

We conclude by discussing future research directions that have emerged as an outcome of this thesis.

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