# Homeostatic plasticity

Homeostatic plasticity is a form of synaptic plasticity that allows neurons to maintain stable activity levels over time. It is crucial for the brain's ability to adapt and function optimally despite changes in internal and external conditions. Unlike other forms of plasticity that strengthen or weaken synapses based on experience or activity (e.g., Hebbian plasticity), homeostatic plasticity ensures that overall neural activity remains within a functional range, preventing excessive excitation or inhibition that could lead to dysfunction or disease.

## Key Features of Homeostatic Plasticity

Stabilization of Neural Activity: Homeostatic plasticity helps maintain the stability of neural circuits by adjusting the strength of synaptic connections in response to prolonged changes in activity. For example, if a neuron becomes too active, homeostatic mechanisms may reduce synaptic strength (synaptic scaling) to bring activity levels back to a stable range.

## Synaptic Scaling

One of the primary mechanisms of homeostatic plasticity is synaptic scaling. This process involves the uniform adjustment of the strength of all synapses on a neuron to ensure that overall activity remains stable. If a neuron's activity decreases for an extended period, synaptic strengths may be increased, and if activity increases, synaptic strengths may be reduced.

## Regulation of Excitability

Homeostatic plasticity also involves adjustments in intrinsic excitability, where the neuron's overall responsiveness to inputs can be altered by changing ion channel densities or properties.

## Temporal Dynamics

Homeostatic plasticity operates on a slower timescale compared to rapid forms of plasticity like long-term potentiation (LTP) or long-term depression (LTD). This slower adjustment allows the nervous system to balance flexibility and stability, adapting to long-term changes while maintaining consistent functionality.

## Importance in Health and Disease

Homeostatic plasticity is essential for preventing conditions like epilepsy, which can arise from excessive neuronal excitability, and for ensuring that learning and memory processes do not lead to runaway synaptic changes that could destabilize neural networks. Dysregulation of homeostatic

mechanisms is implicated in various neurological disorders, including autism, schizophrenia, and neurodegenerative diseases.

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Lakhani et al. identified the specific features of cellular or network activity that were maintained after the perturbation of GABAergic blockade in two different systems: mouse cortical neuronal cultures where GABA is inhibitory and motoneurons in the isolated embryonic chick spinal cord where GABA is excitatory (males and females combined in both systems). They conducted a comprehensive analysis of various spiking activity characteristics following GABAergic blockade. We observed significant variability in many features after blocking GABAA receptors (e.g. burst frequency, burst duration, overall spike frequency in culture). These results are consistent with the idea that neuronal networks achieve activity goals using different strategies (degeneracy). On the other hand, some features were consistently altered after receptor blockade in the spinal cord preparation (e.g. overall spike frequency). Regardless, these features did not express strong homeostatic recoveries when tracking individual preparations over time. One feature showed a consistent change and homeostatic recovery following GABAA receptor block. They found that spike rate within a burst (SRWB) increased after receptor block in both the spinal cord preparation and cortical cultures, and then returned to baseline within hours. These changes in SRWB occurred at both single cell and population levels. The findings indicate that the network prioritizes the spiking dynamics within a burst, which appear to be variable under tight homeostatic regulation. The result is consistent with the idea that networks can maintain an appropriate behavioral response in the face of challenges.Significance statement Homeostatic plasticity plays a critical role in maintaining optimal neural function, particularly during development when the system undergoes repeated functional challenges. In our current study, GABA receptor activity was blocked in two different systems, one in which GABA is inhibitory and another in which GABA is excitatory. In both, they observed that the spike rate within a burst (SRWB) consistently increased and homeostatically returned to control levels in the continued presence of the blocker, demonstrating the importance of SRWB maintenance. When a network is called into action or is functionally engaged during a synaptic barrage, a critical feature that is homeostatically maintained is the spike rate during this activity, which would be crucial for network behavioral performance [1].

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Lakhani A, Gonzalez-Islas C, Sabra Z, Au Young N, Wenner P. Homeostatic Regulation of Spike Rate within Bursts in Two Distinct Preparations. eNeuro. 2024 Aug 19:ENEURO.0259-24.2024. doi: 10.1523/ENEURO.0259-24.2024. Epub ahead of print. PMID: 39160070.