Journal of Alzheimers and Dementia

Open Access Opinion Article

Synaptic Plasticity Impairments in Neural Networks

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DESCRIPTION

Synaptic plasticity allows neurons to adjust their strength in response to activity, forming the basis of learning and memory. Disruption of plasticity impairs adaptive signaling and reduces the capacity of neural networks to respond to stimuli. Mechanisms contributing to impaired plasticity include altered calcium signaling, protein synthesis deficits and abnormal receptor trafficking. Long-Term Potentiation (LTP) and Long-Term Depression (LTD), central to adaptive plasticity, rely on precise molecular signaling that is sensitive to both intrinsic and extrinsic stressors. Calcium signaling is critical for activity-dependent synaptic changes. Calcium entry through N-Methyl-D-Aspartate (NMDA) receptors or voltage-gated channels triggers kinase cascades that regulate gene transcription, receptor insertion and cytoskeletal remodeling. Disruptions in calcium handling, whether due to mitochondrial dysfunction or channelopathies, impair LTP induction and synaptic strengthening. Similarly, excessive calcium can trigger excitotoxicity and dendritic spine loss. Maintaining calcium homeostasis is essential for synaptic adaptability and network stability.

Protein synthesis at dendritic spines supports the local strengthening or weakening of synapses. Dysregulation of translation machinery, ribosomal function or messenger Ribonucleic Acid (mRNA) transport reduces the availability of critical proteins for receptor anchoring, cytoskeletal remodeling and signal transduction. Impairments in these processes compromise the structural changes needed for plasticity, limiting learning capacity and memory retention. Receptor trafficking is a dynamic process influencing synaptic responsiveness. A-Amino-3-Hydroxy-5-Methyl-4-Isoxazole-propionic Acid (AMPA) receptor insertion at postsynaptic membranes is essential for potentiation, while receptor internalization contributes to depression. Disruption in receptor trafficking, caused by abnormal phosphorylation or cytoskeletal instability, reduces signal transmission efficiency and impairs adaptive responses. Both excitatory and inhibitory receptors must be properly regulated to maintain network balance.

Glial contributions also influence plasticity. Astrocytes provide metabolic support and modulate neurotransmitter clearance, while microglia regulate synaptic pruning and respond to injury. Dysregulated glial activity alters local neurotransmitter levels and spine density, impairing plasticity and destabilizing circuits. Inflammatory signaling from glia can further reduce synaptic adaptability, particularly in aging or neurodegenerative conditions. Oxidative stress and metabolic imbalance affect plasticity by impairing signaling cascades, reducing energy supply and damaging membranes. Mitochondrial dysfunction limits Adenosine Triphosphate (ATP) availability for vesicle cycling, receptor trafficking and kinase activity, reducing the ability of synapses to respond dynamically. Antioxidant support and metabolic optimization help maintain plasticity under these conditions.

Environmental stressors, including chronic psychological stress, toxins and poor nutrition, negatively impact synaptic plasticity. Stress hormones alter calcium signaling, receptor sensitivity and dendritic spine morphology, while toxins disrupt vesicle release and metabolic function. Nutritional deficits affect membrane composition and neurotransmitter synthesis, reducing responsiveness to activity-dependent cues. Therapeutic approaches to support plasticity include pharmacological modulation of calcium signaling, receptor function and protein translation, alongside interventions that improve metabolic and mitochondrial health. Lifestyle strategies such as cognitive training, exercise and diet enhance synaptic adaptability and promote network

Received: 17-Nov-2025; Manuscript No: IPAD-25-23843; **Editor assigned:** 20-Nov-2025; Pre QC No: IPAD-25-23843 (PQ); **Reviewed:** 04-Dec-2025; QC No: IPAD-25-23843; **Revised:** 11-Dec-2025; Manuscript No: IPAD-25-23843 (R); **Published:** 18-Dec-2025; DOI:10.36648/ipad.25.8.76

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Citation: Farrow L (2025) Synaptic Plasticity Impairments in Neural Networks. J Alz Dem. 08:76.

efficiency. Maintaining synaptic plasticity is essential for preserving cognitive function, learning and memory capacity across the lifespan.

CONCLUSION

In conclusion, synaptic plasticity impairments arise from disruptions in calcium signaling, protein synthesis, receptor trafficking, glial activity and metabolic support. Addressing these factors through integrated interventions helps maintain neural adaptability and preserves functional networks.