Stress, Structure, and Recall: A Spatial Axon-Growth Model Connecting Developmental Synaptogenesis and Attractor Memory

Rakesh Sengupta

Krea University, India

Lekhana Priya Gundapaneni

Krea University, India

Abstract

Early-life stress is a major risk factor for cognitive deficits, yet the circuit-level mechanisms remain unclear. We hypothesize that systemic stress hormones and localized neurotrophic deprivation during critical periods alter synaptic connectivity. To investigate, we developed a large-scale computational model (N = 100 paired seeds) simulating two developmental stages: an activity-independent growth phase where axons navigate a BDNF chemoattractant landscape, and a functional phase probed with a recurrent attractor model. The "trauma" condition modeled a "Double Hit": a transient cortisol pulse increasing stochastic axon diffusion, combined with localized BDNF deprivation that weakened synaptic efficacy. Trauma produced a significant rise in axon tortuosity ($p < 0.001$), yielding structurally inefficient networks with collapsed spectral radius. Functionally, recurrent gain loss impaired retention of clustered memory engrams (≈41% deficit), while random patterns remained robust. Parameter sweeps confirmed this pathology arises non-linearly from guidance noise and trophic weakness, linking developmental stress to memory impairments.

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