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Computational modeling of spatial cognition

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Cognitive functions brought into existence by the coherent, highly organized activity of large ensembles of cells. Yet, we have lacked the conceptual tools that would enable us to link the characteristics of the individual neurons and synapses to the properties of large-scale cognitive representations. For example, many observations point out a link between weakening synapses and weakening memory, and it is widely believed that the former causes the latter—but how exactly? If the synaptic strengths decrease, e.g., by 5%, will the time required to learn a particular task increase by 1%, by 5% or by 50%? Will the answer depend on the original cognitive state? Can an increase in spatial learning time, caused by a synaptic depletion, be compensated by increasing the population of active neurons or by elevating their spiking rates? Answering these questions is impossible without a theoretical framework that connects the individual cell outputs and the large-scale cognitive phenomena that emerge at the ensemble level. Using Topological tools for Big Data analysis, we develop a framework1-6 that allows us to address some of these questions computationally, i.e., produce in silico description of spatial learning mechanisms by generalizing over copious amounts of electrophysiological data. Here, we present a mechanism of spatial memory deterioration as a function of synaptic depletion in the hippocampal network and discuss a cross-scale scenario of why the decreasing numbers of active place cells, the altered place cell firing rates and the "brain wave" frequency spectra may correlate with spatial learning deficits observed in Alzheimer's disease.

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