Hotspots of dendritic spine turnover facilitate clustered spine addition and learning and memory. Frank AC, Huang S, Zhou M, Gdalyahu A, Kastellakis G, Silva TK, Lu E, Wen X, Poirazi P, Trachtenberg JT, Silva AJ. Nat Commun. 2018 Jan 29;9(1):422. PubMed

Re-interpretation of the findings in terms of the semblance hypothesis

See supplementary figure 8 in the above article. Learning leads to loss of spines & formation of new spines at those regions (spine turnover). Why should spines get lost? Based on the semblance hypothesis, learning leads to inter-neuronal inter-spine interaction leading to inter-postsynaptic functional LINKs (IPLs) (see **figure 8** in FAQ section of this website). Inter-spine fusion is at the extreme end of this spectrum of changes. The nature of IPLs depends on several factors. These include a) nature of fatty acids in the phospholipid molecules that form spine membranes, and b) intensity of stimuli that affect propagation of signals towards the IPLs. If IPL formation leads to an extreme change of inter-neuronal inter-spine fusion, then it will lead to mixing of the contents of cytoplasm of two neurons. Since even adjacent neurons of a similar type vary in their gene expression/protein content (Kamme et al., 2003; Cembrowski et al., 2016), it is reasonable to expect cellular mechanisms for closing the fusion pore. If it is not possible, then the neurons will trigger mechanisms to remove the spines. This can explain spine loss. As a homeostatic mechanism, the involved neurons can be expected to produce new spines using phospholipids that resist inter-spine fusion. Thus, the basic operational mechanism of semblance hypothesis can be extended to provide a mechanistic explanation for spine turnover during learning.

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