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Can calcium ion contribute to morphological plasticity of a spine?

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Introduction

Structural plasticity of a spine, which is a change in the spine morphology with synaptic stimulation, has been reported from several labs. Structural plasticity is thought to be a consequence of the induction of long-term potentiation. Some reports suggested the role of actin molecules in the structural plasticity, and the change in F-actin structure will play a pivotal role in the morphological change of a spine [1-4]. The structure of F-actin is controlled by complex mechanisms, and the molecular mechanisms which contribute to morphological plasticity of a spine are not understood yet. Here, we performed several simulations to see whether the intracellular calcium ion can trigger the structural plasticity of a spine. Simulation results have shown calcium could be a molecule triggering the morphological change of a spine. From these simulation results, we propose a hypothetical mechanism involved in the structural plasticity.

Methods

Morphological models including mushroom spines and filopodium with different size in head and neck diameter were constructed using A-Cell software [5,6]. The 3D morphology was compartmentalized, and Ca^{2+} entry through NMDA receptors and medium- and low-affinity Ca^{2+} buffers were embedded to corresponding compartments. Ca^{2+} diffusion within a spine or filopodum was calculated using Fick's equation. Figure 1 shows the overall reaction schemes and the model morphology.

Results

First we simulated the change in the concentration of intracellular calcium ion ($[Ca^{2+}]_i$) in filopodia. The peak $[Ca^{2+}]_i$ was increased as the length of filopodium was increased as was expected (Fig. 2 left). However, it was saturated at the filopodium length longer than 1 μm and kept almost the same level. Next, the diameter of a spine

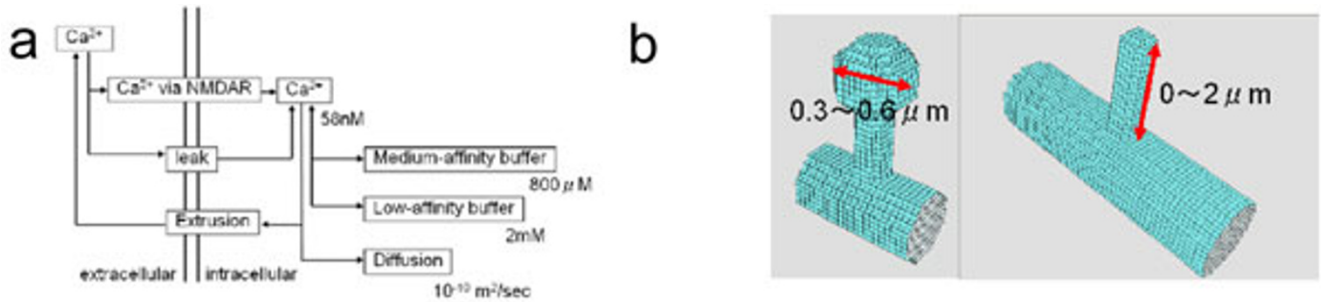

Figure 1
Overall reaction scheme (a) and morphologies used in simulations (b).

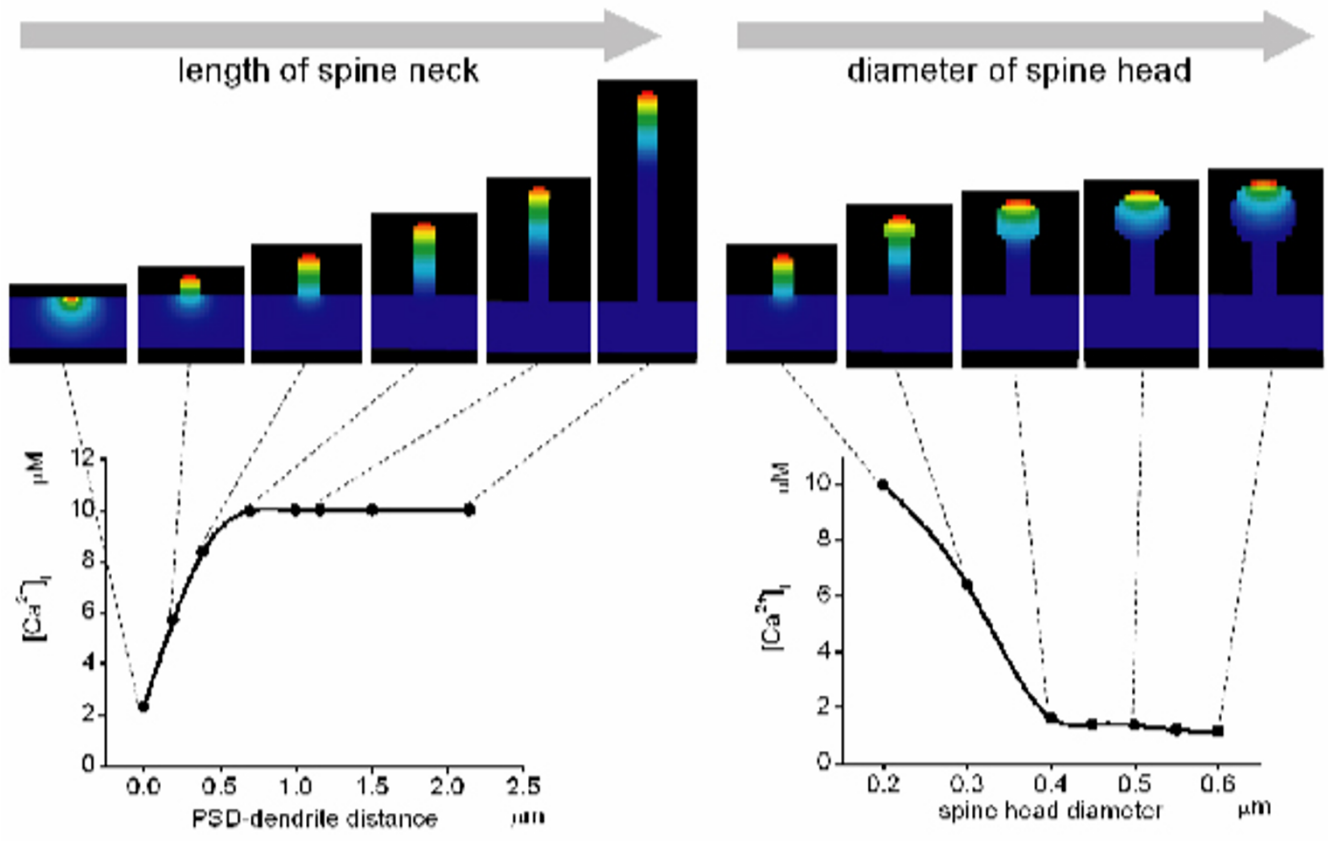

Figure 2
The change in $[Ca^{2+}]_i$ by the change in the size of filopodium (left) and a spine (right).

head was changed with fixed length of spine neck. With the increase in the spine head diameter, the peak $[Ca^{2+}]_i$ was decreased as was expected (Fig. 2 right). However, $[Ca^{2+}]_i$ reached a minimum and it kept almost the same level even if the diameter was increased further.

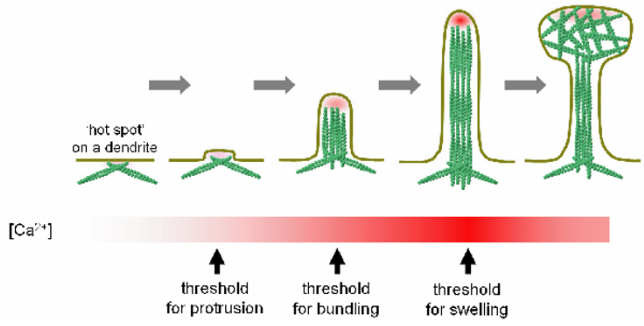

Figure 3
Hypothetical mechanism triggering morphological plasticity.

Discussion

The present simulation results have shown the change in $[Ca^{2+}]_{i\ with\ a}$ change in the size of a filopodium and a spine. This suggests that $[Ca^{2+}]_i$ can be a triggering molecule for the structural plasticity. The hypothetical mechanism is shown in Figure 3. First, calcium concentration in a localized region of a dendrite is increased forming a 'hot spot'. Second, actin polymerization begins at the 'hot spot' and the protrusion develops increasing the peak $[Ca^{2+}]_i$ at its tip. Third, this increase in $[Ca^{2+}]_i$ results in further actin polymerization and its bundling. Fourth, protrusion develops further and the peak $[Ca^{2+}]_i$ increased. At some level of $[Ca^{2+}]_i$ (threshold level), the actin structure at the tip of filopodium is changed from bundling to a meshwork forming a spine head.

References

1. Maletic-Savatic M, Malinow R, Svoboda K: **Rapid dendritic morphogenesis in CA1 hippocampal dendrites induced by synaptic activity.** *Science* 1999, **283:**1923-1927.
2. Matsuzaki M, Honkura N, Ellis-Davies GC, Kasai H: **Structural basis of long-term potentiation in single dendritic spines.** *Nature* 2004, **429:**761-766.
3. Fukazawa Y, Fukazawa Y, Saitoh Y, Ozawa F, Ohta Y, Mizuno K, Inokuchi K: **Hippocampal LTP is accompanied by enhanced F-**

actin content within the dendritic spine that is essential for late LTP maintenance in vivo. *Neuron* 2003, **38:**447-460.
4. Okamoto K, Nagai T, Miyawaki A, Hayashi Y: **Rapid and persistent modulation of actin dynamics regulates postsynaptic reorganization underlying bidirectional plasticity.** *Nat Neurosci* 2004, **7:**1104-1112.
5. Ichikawa K: **A Modeling Environment with Three-Dimensional Morphology, A-Cell-3D, and Ca^{2+} Dynamics in a Spine.** *Neuroinformatics* 2005, **3:**49-64.
6. Ichikawa K: **A-Cell: graphical user interface for the construction of biochemical reaction models.** *Bioinformatics* 2001, **17:**483-484.