A dendritic spine shrinkage in response to extracellular high K^+ is inhibited by Co^{2+} or Cd^{2+}

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In the brain, neurons communicate with each other at excitatory and inhibitory synapses. In each excitatory synapse, a dendritic spine receives the excitatory neurotransmitter released by an axon terminal. An extracellular high K^+ induces robust depolarization and mimics massive synaptic transmission. A dendritic spine transiently shrinks in high K^+ solution. In this study, we investigated the mechanisms of the spine shrinkage using cultured hippocampal neurons. The spine shrinkage observed in response to high K^+ was inhibited in the presence of extracellular Co^{2+} or Cd^{2+}. Then we investigated Ca^{2+}-signaling pathway because these metal ions are supposed to be wide spectrum Ca^{2+}-channel blockers. We preincubated neurons with ROCK inhibitor Y27632 or CaMKII inhibitor KN-93. However, neither of them inhibited the spine shrinkage. Finally, we tested whether myosin is involved in the spine shrinkage. A myosin ATPase inhibitor, blebbistatin, did not inhibit the spine shrinkage in high K^+. Taken together, extracellular high K+-triggered Ca^{2+}-influx seem to induce the spine shrinkage; however, the downstream signaling pathway or the motor machinery has not yet been determined.