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Mechanosensor TRPV2 Regulates Axonal Outgrowth during Development

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Summary

It is specific characteristics that neurons can grow to the length of more than 1 m in humans. This mechanism of elongation has been called ‘‘passive stretching’’. From embryonic stages, the passive stretching-dependent axonal outgrowth begins. As our body grows, the distances between neuronal cell bodies and growth cones gradually increase, thereby exerting tensile forces on the axons. It has never been identified for a long time which molecules are the mechanosensors for it. We previously reported that TRPV2 was a mechanosensor channel which contributed axonal outgrowth in membrane stretch dependent manner. These results indicate that TRPV2 might be an important component for passive stretching, if TRPV2 can detect very weak mechanical stimulus. In this study, we examined whether TRPV2 can detect such very weak mechanical stimulus by a Ca^{2+}-imaging method and a whole-cell patch clamp recording. We also examined whether the activation of TRPV2 by weak mechanical stimulus lead to the enhancement of axon outgrowth by a time-lapse imaging method. Finally, we identified that TRPV2 had a potential to detect very weak mechanical stimulus, and the activation of TRPV2 promoted axon outgrowth. Taken together, TRPV2 is a strong candidate molecule for passive stretch-dependent axonal outgrowth as an important cell-sensor, which highly permeates the Ca^{2+} ion.