University of Tsukuba:
Retrograde TGF-β signaling regulating peripheral axon regeneration

Hiroshi Hasegawa¹, Mika Arai², Atsushi Suzuki², Fan Wang³, Zhigang He⁴

¹ School of Integrative and Global Majors, University of Tsukuba.
² Graduate School of Integrative Human Sciences, University of Tsukuba
³ Department of Cell Biology, Duke University
⁴ F.M. Kirby Neurobiology Center, Boston Children's Hospital, and Department of Neurology, Harvard Medical School

In mammals, neurons in the central nervous system have low regenerative ability for axons. There is no medical treatment for central nervous system disorders with axonal damages, including spinal cord injury and cerebral infarction. Many recent studies have focused on transplantation of new neurons developed from induced pluripotent stem cells (iPS) cells. However, the treatment to induce axonal regeneration with suppression of neuronal death after injury would be milder and safer method to treat the patients.

In contrast to the central neurons, peripheral neurons have an ability to regenerate their axons after injury. Understanding of the molecular mechanisms governing peripheral nerve regeneration will contribute to the development of medicines treating nerve injury. However, molecular mechanisms controlling peripheral axon regeneration has not been well understood.

We have screened the molecules regulating neurite growth and found that the transforming growth factor-β (TGF-β) family cytokines as potential regulator of the regeneration of peripheral neurons after injury. I will introduce our recent results on the role of TGF-β signaling from the skin to dorsal root ganglia (DRG) to regulate axonal elongation. In addition, our study revealed the potential downstream genes of TGF-β signaling pathway in the DRG neurons, which will be also discussed.