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DBS: From the point of view of glial functions

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Glial dysfunction is a cause of brain diseases including neuropathic pain. We previously demonstrated that in the primary somatosensory cortex (S1) of neuropathic pain model mice, astrocytes become activated, release synaptogenic molecules such as thrombospondin, and cause uncontrolled synapse-formation, thereby leading to misconnection of innocuous and nocuous networks. This misconnection in the S1 cortical networks is a cause of neuropathic pain allodynia. Thus, an appropriate control of astrocytes can be a major therapeutic strategy. Deep brain stimulation (DBS) is frequently used for the treatment of neurodegenerative diseases such as PD, psychiatric diseases, and neuropathic pain. Although its therapeutic effects are recognized, the molecular mechanism is not well understood. Recently, DBS is shown to affect glial cell functions especially astrocytic Ca^{2+} signals and gliotransmission, which may be involved in its therapeutic effect. Thus, glial cells could be a potential therapeutic target for these diseases. When there is an effective treatment such as DBS, the etiology can be elucidated by thoroughly verifying its action. It is important to link such a clinical top-down research with basic bottom-up research, for which pharmacology plays a central role as an interface. In this symposium, I will also discuss how to promote young scientists by sharing such an attractive and dynamic collaboration of basic and clinical researches.