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発表の概要と成果（抄録を公開している URL がある場合、「概要・成果」を記載した上で、URL を末尾に記してください。また、抄録 PDF は別途ご提出ください。なお、抄録 PDF は Web 上には公開されません。）

SUMOylation is a reversible post translational modification. Covalent conjugation of small ubiquitin-like modifier (SUMO) regulates the stability and function of the target protein. SUMOs are then removed from the substrates by sentrin/SUMO-specific proteases (SENPs). There are six SENPs in mammals: SENP1 – 3 and 5 – 7. Of these SENPs, SENP1, 2, and 5 are reported to exhibit endopeptidase activity that is required for the generation of mature conjugatable form of SUMO besides its isopeptidase activity for SUMO deconjugation from the target protein, indicative of the importance of these SENPs' activity in both SUMOylation and deSUMOylation. In vertebrates, there are four SUMO proteins: SUMO1-4. SUMO2 and SUMO3 share 96% sequence identity and thus are often referred to as SUMO2/3. SUMO2/3 but not SUMO1 have a consensus SUMOylation sequence, enabling SUMO2/3 to form polySUMO chain. SUMO4 seems not to be mature form, indicating its disability to be attached to target proteins. SENPs exhibit SUMO isoform preference. For example, SENP1 processes both SUMO1 and SUMO2/3, whereas SENP3 and 5 prefers SUMO2/3, although SENP5 can process SUMO1 to a lesser extent. Numerous studies have implicated that the balance of those enzymes governing SUMOylation and de-SUMOylation are crucial for various physiological and pathological processes in neurons. Several recent studies reported SUMOylation is involved in Alzheimer's disease, Huntington's disease, and ataxia. However, localization and function of those enzymes such as SENP5 has not been described in detail in the central nervous system. We performed immunoelectron microscopy and demonstrated SENP5 localization to presynaptic terminals, postsynaptic spines, and mitochondria in the axon terminals in the adult brain. In addition, developmental immunostaining and western blot analysis showed the abundant expression of SENP5 in the embryonic brain. We investigated the physiological roles of SENP5 on the cerebral cortex development. Forced expression or knockdown of SENP5 in the primary cultured neurons resulted in the reduced number of neurites and perturbation of the axonal extension. Accordingly, the overexpression of SENP5 in E14.5 embryonic cortex using in utero electroporation repressed the proper migration of neurons, leading the accumulation of newborn neurons in the intermediate zone. These results suggest that SENP5 play a vital role in the differentiation and migration of neurons.