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申請者（ふりがな）	佐藤彩佳　（　さとうあやか　）
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発表者（※学会発表の場合のみ記載、共同発表者の氏名も記載すること）	○佐藤彩佳、山田晴也、秋山博紀、榊原伸一
発表題目（※学会発表の場合のみ記載）	脱 SUMO 化酵素 SENP5 によるミトコンドリア形態制御

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. Numerous studies have implicated that the balance of those enzymes governing SUMOylation and de-SUMOylation are crucial for various physiological and pathological processes in the central nervous system. A mitochondrion is a highly dynamic organelle that undergoes fission and fusion. Dynamin-related protein 1 (Drp1) has a crucial role in the mitochondrial fission machinery. SUMOylation/deSUMOylation controls mitochondrial dynamics through Drp1 modulation. However, localization and function of those enzymes such as SENP5 has not been described in detail in neural development. Here, we identified a novel SENP5 isoforms (SENP5S) and observed its competed with other SENPs and promotes SUMOylation. In addition, we found SENP5 forced expression changed mitochondrial morphology through regulating SUMOylation of Drp1. Moreover, SENP5 perturbations 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. Accordingly, 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. These results suggest that SENP5 play a vital role in mitochondria morphology during cortical development.