Calcium-dependent secretion activator 1 is a protein that in humans is encoded by the CADPS gene.

CADPS encodes a novel neural/endocrine-specific cytosolic and peripheral membrane protein required for the Ca2+-regulated exocytosis of secretory vesicles. CADPS acts at a stage in exocytosis that follows ATP-dependent priming, which involves the essential synthesis of phosphatidylinositol 4,5-bisphosphate (PtdIns(4,5)P2). Alternative splicing has been observed at this locus and three variants, encoding distinct isoforms, are described.

Ischemic stroke triggers a cascade of events that facilitates neuroprotection and spontaneous recovery, which accounts for a major part of functional recovery. Despite the cellular and molecular facilitations on neural protection, the molecular mechanisms of spontaneous recovery have not been fully understood. Ca2+ -dependent activator protein for secretion 1 (CAPS1), a member of the CAPS family, plays a major role in synaptic transmission and synaptic effectiveness by regulating vesicle exocytosis. The molecular mechanism of CAPS1 in spontaneous recovery after ischemic stroke was studied. In this study, transient left middle cerebral artery occlusion (MCAO) was used as the ischemic stroke model. The whole brain magnetic resonance imaging (MRI) and neurological score analysis showed decreased infarct volume and neurological scores at 7 days compared to 1 day after MCAO, suggesting spontaneous recovery. Western blot analysis showed elevated BDNF and CAPS1 expression levels in the bilateral hippocampus at both 1 day and 3 days after MCAO. Then, inhibition of CAPS1 by adeno-associated virus (AAV) microinjection in the hippocampus attenuated the spontaneous recovery of both motor and memory impairment induced by MCAO. In addition, elevated p-TrkB levels were detected after MCAO, which were reduced by CAPS1-AAV microinjection, indicating that CAPS1 could induce BDNF secretion after ischemic stroke. Moreover, they found the elevated combination of CAPS1 with dense core vesicles (DCV) in the hippocampus at both 1 day and 3 days after MCAO, which could also be inhibited by CAPS1-AAV microinjection, indicating the potential mechanism of CAPS1 in regulating BDNF release after MCAO. Finally, we found that CAPS1/BDNF signaling could influence neurogenesis in the hippocampus after MCAO. In conclusion, CAPS1 regulates neurogenesis by upregulating BDNF release in the hippocampus, which finally facilitates spontaneous recovery after ischemic stroke [1)]

1)
Liu D, Zheng Y, Chen Y, Jiang Y, Wang H, Li L, Ma L. Ca2+ -dependent activator protein for secretion 1 promotes spontaneous recovery in ischemic stroke by regulating BDNF secretion. J Neurochem. 2023 Mar 14. doi: 10.1111/jnc.15808. Epub ahead of print. PMID: 36916413.