SPARC

- SPARC promotes malignant progression and predicts poor prognosis in cervical squamous cell carcinoma
- Scaling of vagus nerve stimulation parameters does not achieve equivalent nerve responses across species
- AO Spine Clinical Practice Recommendations for Diagnosis and Management of Degenerative Cervical Myelopathy: Evidence Based Decision Making - A Review of Cutting Edge Recent Literature Related to Degenerative Cervical Myelopathy
- Modulating the blood-brain barrier in CNS disorders: A review of the therapeutic implications of secreted protein acidic and rich in cysteine (SPARC)
- Harmonizing neuropathic pain research: outcomes of the London consensus meeting on peripheral tissue studies
- Prediction of brain metastasis development with DNA methylation signatures
- Identification of high-performing antibodies for SPARC-related modular calcium-binding protein 1 (SMOC-1) for use in Western Blot and immunoprecipitation
- Spatial transcriptomics of gastric cancer brain metastasis reveals atypical vasculature strategies with supportive immune profiles

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Secreted Protein Acidic and Rich in Cysteine, is a type of extracellular matrix protein. SPARC is involved in various biological processes, including cell adhesion, proliferation, differentiation, and angiogenesis. It is expressed in a wide range of tissues and has been implicated in various diseases, including cancer, fibrosis, and cardiovascular disease. SPARC is also known by other names, such as osteonectin or BM-40, and is encoded by the SPARC gene.

Matricellular proteins have been implicated in pathologies after subarachnoid hemorrhage (SAH). To find a new therapeutic molecular target, a study aimed to clarify the relationships between serially measured plasma levels of a matricellular protein, secreted protein acidic and rich in cysteine (SPARC), and delayed cerebral ischemia (DCI) in 117 consecutive aneurysmal SAH patients with admission World Federation of Neurological Surgeons (WFNS) grades I-III. DCI developed in 25 patients with higher incidences of past history of hypertension and dyslipidemia, preoperative WFNS grade III, modified Fisher grade 4, spinal drainage, and angiographic vasospasm. Plasma SPARC levels were increased after SAH, and significantly higher in patients with than without DCI at days 7-9, and in patients with VASOGRADE-Yellow compared with VASOGRADE-Green at days 1-3 and 7-9. However, there were no relationships between plasma SPARC levels and angiographic vasospasm. Receiver-operating characteristic curves differentiating DCI from no DCI determined the cut-off value of plasma SPARC ≥ 82.1 ng/ml at days 7 - 9 (sensitivity, 0.800; specificity, 0.533; and area under the curve, 0.708), which was found to be an independent determinant of DCI development in multivariate analyses. This is the first study to show that SPARC is upregulated in peripheral blood after SAH, and that SPARC may be involved in the development of DCI without angiographic vasospasm in a clinical setting [1)].

Secreted protein acidic and rich in cysteine (SPARC) was widely expressed in Vascular Smooth Muscle Cells (VSMCs) of human intracranial aneurysms (IAs) and could reduce the capability of self-repair.

This indicates that SPARC may play a role in the promotion of IAs formation and progression, but the mechanism remains unclear. In a study, Tao et al. further investigated whether SPARC could induce phenotypic modulation of Human Brain Vascular Smooth Muscle Cells (HBVSMCs) and sought to elucidate the role of SPARC-mediated autophagy involved in it. The results demonstrated that SPARC inhibited the expression of contractile genes in HBVSMCs and induced a synthetic phenotype. More importantly, SPARC significantly up-regulated multiple proteins including autophagy marker microtubule-associated protein light chain 3-II (LC3-II), Beclin-1, and autophagy-related gene 5(ATG5). Furthermore, SPARC could promote p62 degradation. The autophagy inhibitor 3- methyladenine (3-MA) significantly blocked SPARC-induced phenotypic modulation of HBVSMCs. We further sought to elucidate the molecular mechanism involved in SPARC-induced autophagy, and found that SPARC could activate the AMPK/mTOR signaling pathway in HBVSMCs. AMPK could be pharmacologically inhibited by Compound C (CC), which significantly decreased the phosphorylation of AMPK into p-AMPK, increased the phosphorylation of mTOR into p-mTOR, and decreased LC3-II, Beclin-1 and ATG5 levels. This suggested that activated AMPK/ mTOR signaling is related to SPARC-mediated autophagy. These results indicated that SPARC plays a role in the phenotypic modulation of HBVSMCs through autophagy activation by AMPK/mTOR signaling pathway [2].

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Nakajima H, Kawakita F, Oinaka H, Suzuki Y, Nampei M, Kitano Y, Nishikawa H, Fujimoto M, Miura Y, Yasuda R, Toma N, Suzuki H; pSEED group. Plasma SPARC Elevation in Delayed Cerebral Ischemia After Aneurysmal Subarachnoid Hemorrhage. Neurotherapeutics. 2023 Feb 13. doi: 10.1007/s13311-023-01351-x. Epub ahead of print. PMID: 36781745.
2)
Tao L, Xianjun T, Shaowei Z, Weiying Z, Bin H, Jinhao S, Feng L, Yunyan W. SPARC Induces Phenotypic Modulation of Human Brain Vascular Smooth Muscle Cells via AMPK/mTOR-Mediated Autophagy. Neurosci Lett. 2019 Sep 6:134485. doi: 10.1016/j.neulet.2019.134485. [Epub ahead of print] PubMed PMID: 31499136.