NCAPG

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NCAPG, also known as non-SMC condensin I complex subunit G, is a protein that plays a crucial role in chromosome condensation during cell division. Here are some key points about NCAPG:

Function: NCAPG is a subunit of the condensin I complex, which is involved in the structural organization of chromosomes. It helps in compacting the DNA strands and ensuring their proper segregation during mitosis and meiosis.

Structure: NCAPG is a large protein consisting of multiple domains. It contains a HEAT (Huntingtin, elongation factor 3, protein phosphatase 2A, and TOR1) repeat domain, which is involved in protein-protein interactions.

Association with other subunits: NCAPG interacts with other subunits of the condensin I complex, including SMC2, SMC4, NCAPD2, and NCAPH. These interactions are essential for the assembly and function of the condensin complex.

Cellular localization: NCAPG is primarily localized to the nucleus, where it associates with chromatin during cell division. It is recruited to specific regions of the chromosomes to promote their condensation and segregation.

Role in chromosome dynamics: NCAPG, along with other condensin subunits, helps in resolving and compacting duplicated chromosomes. It facilitates the formation of higher-order chromatin structures, ensuring the faithful segregation of genetic material during cell division.

Implications in disease: Dysregulation or mutations in NCAPG can lead to chromosomal instability and contribute to the development of various genetic disorders and cancer. Abnormalities in the condensin complex have been observed in conditions such as Cornelia de Lange syndrome and cancer progression.

Understanding the functions and mechanisms of NCAPG and the condensin complex is an active area of research in cell biology and genetics, as it provides insights into chromosome dynamics and the maintenance of genome integrity.

NCAPG in glioma

- UBTD2 protein molecules emerges as a key prognostic protein marker in glioma: Insights from integrated omics and machine learning analysis of GRM7, NCAPG, CEP55, and other biomarkers
- Nonstructural maintenance of chromatin condensin I complex subunit G promotes the progression of glioblastoma by facilitating Poly (ADP-ribose) polymerase 1-mediated E2F1 transactivation
- The Recurrent-Specific Regulation Network of Prognostic Stemness-Related Signatures in Low-Grade Glioma
- The U2AF65/circNCAPG/RREB1 feedback loop promotes malignant phenotypes of glioma stem cells through activating the TGF-beta pathway

- The role of NCAPG in various of tumors
- A convolutional neural network model for survival prediction based on prognosis-related cascaded Wx feature selection
- The Higher Expression of CDCA2 Associated with Poor Prognosis in Glioma
- NCAPG Promotes Tumor Progression and Modulates Immune Cell Infiltration in Glioma

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Studies have shown that NCAPG is frequently overexpressed in glioma tissues compared to normal brain tissue. Increased expression of NCAPG has been associated with higher tumor grade and poor prognosis in glioma patients.

Prognostic marker: High expression of NCAPG has been identified as an independent prognostic marker for overall survival and progression-free survival in glioma patients. Its overexpression correlates with shorter survival times and increased risk of tumor recurrence.

Functional implications: NCAPG plays a role in promoting cell proliferation, migration, invasion, and angiogenesis in glioma cells. It affects multiple signaling pathways involved in these processes, including the Wnt/β-catenin pathway, PI3K/AKT pathway, and EGFR signaling.

Potential therapeutic target: Given its involvement in glioma progression, NCAPG has been explored as a potential therapeutic target. Inhibition of NCAPG expression or function has shown promise in reducing glioma cell growth and invasion in preclinical studies.

Association with other molecular alterations: NCAPG expression has been found to correlate with other molecular alterations commonly observed in glioma, such as mutations in IDH1, TP53, and EGFR amplification. These associations suggest that NCAPG may interact with other key molecular pathways involved in glioma development.

Overall, the overexpression of NCAPG in glioma and its association with poor prognosis highlights its potential as a biomarker and therapeutic target in the management of this aggressive brain tumor. Further research is needed to elucidate the exact mechanisms by which NCAPG contributes to glioma progression and to explore its therapeutic potential in clinical settings.

The function effects of NCAPG downregulation or overexpression were evaluated in GBM cell proliferation, migration, invasion, and self-renewal in vitro and tumor growth in vivo. The molecular mechanism of NCAPG was researched.

They identified that NCAPG was upregulated in GBM and associated with poor prognosis. Loss of NCAPG suppressed the progression of GBM cells in vitro and prolonged survival in mouse models of GBM in vivo. Mechanistically, Hou et al. revealed that NCAPG positively regulated E2F1 pathway activity. By directly interacting with PARP1, a co-activator of E2F1, and facilitating the PARP1-E2F1 interaction to activate E2F1 target gene expression. Intriguingly, they also discovered that NCAPG functioned as a downstream target of E2F1, which was proved by the ChIP and Dual-Luciferase results. Comprehensive data mining and immunocytochemistry analysis revealed that NCAPG expression was positively associated with the PARP1/E2F1 signaling axis.

The findings indicate that NCAPG promotes glioblastoma progression by facilitating PARP1-mediated E2F1 transactivation, suggesting that NCAPG is a potential target for anticancer therapy [1].

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Hou J, Huang P, Xu M, Wang H, Shao Y, Weng X, Liu Y, Chang H, Zhang L, Cui H. NCAPG promotes the progression of glioblastoma by facilitating PARP1-mediated E2F1 transactivation. Neuro Oncol. 2023 Jul 9:noad111. doi: 10.1093/neuonc/noad111. Epub ahead of print. PMID: 37422706.