Serine/threonine-protein kinase Nek2 is an enzyme that in humans is encoded by the NEK2 gene.

NEK2 has been shown to interact with MAPK1 and NDC80.

Protein kinase which is involved in the control of centrosome separation and bipolar spindle formation in mitotic cells and chromatin condensation in meiotic cells. Regulates centrosome separation (essential for the formation of bipolar spindles and high-fidelity chromosome separation) by phosphorylating centrosomal proteins such as CROCC, CEP250 and NINL, resulting in their displacement from the centrosomes. Regulates kinetochore microtubule attachment stability in mitosis via phosphorylation of NDC80. Involved in regulation of mitotic checkpoint protein complex via phosphorylation of CDC20 and MAD2L1. Plays an active role in chromatin condensation during the first meiotic division through phosphorylation of HMGA2. Phosphorylates: PPP1CC; SGOL1; NECAB3 and NPM1. Essential for localization of MAD2L1 to kinetochore and MAPK1 and NPM1 to the centrosome. Isoform 1 phosphorylates and activates NEK11 in G1/S-arrested cells. Isoform 2, which is not present in the nucleolus, does not [Uniprot].

NIMA-related kinase 2 (NEK2) as a functional binding protein of enhancer of zeste homolog 2 (EZH2) that plays a critical role in the posttranslational regulation of EZH2 protein in glioma stem cells (GSCs). NEK2 was among the most differentially expressed kinase-encoding genes in GSC-containing cultures (glioma spheres), and it was required for in vitro clonogenicity, in vivo tumor propagation, and radioresistance. Mechanistically, the formation of a protein complex comprising NEK2 and EZH2 in glioma spheres phosphorylated and then protected EZH2 from ubiquitination-dependent protein degradation in a NEK2 kinase activity-dependent manner. Clinically, NEK2 expression in patients with glioma was closely associated with EZH2 expression and correlated with a poor prognosis. NEK2 expression was also substantially elevated in recurrent tumors after therapeutic failure compared with primary untreated tumors in matched GBM patients. We designed a NEK2 kinase inhibitor, compound 3a (CMP3a), which efficiently attenuated GBM growth in a mouse model and exhibited a synergistic effect with radiotherapy. These data demonstrate a key role for NEK2 in maintaining GSCs in GBM by stabilizing the EZH2 protein and introduce the small-molecule inhibitor CMP3a as a potential therapeutic agent for GBM [1)].

1)
Wang J, Cheng P, Pavlyukov MS, Yu H, Zhang Z, Kim SH, Minata M, Mohyeldin A, Xie W, Chen D, Goidts V, Frett B, Hu W, Li H, Shin YJ, Lee Y, Nam DH, Kornblum HI, Wang M, Nakano I. Targeting NEK2 attenuates glioblastoma growth and radioresistance by destabilizing histone methyltransferase EZH2. J Clin Invest. 2017 Jul 24. pii: 89092. doi: 10.1172/JCI89092. [Epub ahead of print] PubMed PMID: 28737508.