FAM83D

Up-regulation of the family with sequence similarity 83 member D (FAM83D) has been acknowledged as a vital contributor for the carcinogenesis of numerous cancers. The relevance of FAM83D in glioblastoma (Glioblastoma), however, is not well understood. A work aimed to determine the possible roles and mechanisms of FAM83D in Glioblastoma. By analyzing The Cancer Genome Atlas (TCGA) data, Wang et al. found dramatic increases in FAM83D expression in Glioblastoma tissue. They also observed elevated levels of FAM83D in the clinical specimens of Glioblastoma. In vitro data showed that silencing FAM83D resulted in remarkable antitumor effects via inhibiting the proliferation, invasion, and epithelial-mesenchymal transition of Glioblastoma cells. Moreover, the knockdown of FAM83D improved sensitivity to the chemotherapy drug temozolomide. In-depth mechanism research revealed that the silencing of FAM83D strikingly decreased the phosphorylation levels of AKT and glycogen synthase kinase-3β and prohibited activation of the Wnt/β-catenin pathway. The suppression of AKT abolished FAM83D-mediated activation of the Wnt/β-catenin pathway. The re-expression of β-catenin reversed FAM83D-silencing-induced antitumor effects in Glioblastoma cells. In addition, Glioblastoma cells with FAM83D silencing exhibited reduced tumorigenic potential in vivo. Overall, the data from this work show that the inhibition of FAM83D displays antitumor effects in Glioblastoma via down-regulation of the AKT/Wnt/β-catenin pathway and propose FAM83D as a new therapeutic target for Glioblastoma [1)].

1)
Wang J, Quan Y, Lv J, Gong S, Ren P. Inhibition of FAM83D displays antitumor effects in glioblastoma via down-regulation of the AKT/Wnt/β-catenin pathway. Environ Toxicol. 2022 Feb 12. doi: 10.1002/tox.23488. Epub ahead of print. PMID: 35150198.