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"*Jun and its Connections with Wnt and Notch*."

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Desmoid-type fibromatosis (DTF) is a locally aggressive soft tissue tumor. Though not metastasizing, tumors tend to relapse locally causing severe morbidity. In this study we are investigating whether the transcription factor JUN drives DTF and how JUN interconnects with two important drivers of DTF, beta catenin and Notch. Analyzing patient samples through IHC, JUN is commonly expressed in demoid cells. Additionally, on the molecular level, 92 of 421 genes upregulated in DTF are also target genes of Jun. In vitro, desmoid cells express activated Jun and a co-immunoprecipation assay shows the connection between Jun and beta-Catenin. In co-cultures, neutrophils increase beta-catenin in desmoid cells. Next to spontaneous desmoid-like tumors, Jun inducible mice reliably develop desmoid-like tumors upon local doxycycline administration and JUN induction. In conclusion, our results indicate that Jun contributes to DTF and that it interacts with beta-catenin. Further experiments, both in vitro and in vivo, will indicate whether Jun is a promising target for the treatment of DTF.