

C/EBP β cooperates with MYB to maintain the oncogenic program of AML cells

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A new editorial perspective titled "[C/EBP \$\beta\$ cooperates with MYB to maintain the oncogenic program of AML cells](#)" has been published in *Oncotarget*.

In this new paper, researcher Karl-Heinz Klempnauer from Westfälische-Wilhelms-Universität discusses recent studies on the role of transcription

factor MYB in [acute myeloid leukemia](#) (AML). MYB has been identified as a key regulator of a transcriptional program for self-renewal of AML cells.

The MYB gene initially attracted attention as the progenitor of a retrovirally-transduced oncogene that induces a myeloid leukemia in chickens. MYB encodes a transcription factor with essential roles in the development of the hematopoietic system and the proliferation and differentiation of hematopoietic progenitor cells. Subsequent work identified MYB also as a crucial player in the development and maintenance of leukemia in humans.

"Recent work summarized here has now highlighted the CCAAT-box/enhancer binding protein beta (C/EBP β) as an essential factor and potential therapeutic target that cooperates with MYB and coactivator p300 in the maintenance of the leukemic cells," the paper states.

More information: Karl-Heinz Klempnauer, C/EBP β cooperates with MYB to maintain the oncogenic program of AML cells, *Oncotarget* (2023). [DOI: 10.18632/oncotarget.28377](https://doi.org/10.18632/oncotarget.28377)

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