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Beta-catenin mutations are not observed in chronic myeloid leukemia

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ABSTRACT

Aims and background. Studies reporting activated Wnt signaling in all stages of chronic myeloid leukemia (CML) have demonstrated that deregulation of the pathway plays a role in the pathogenesis of this disease. Several reports have suggested mechanisms for the deregulated Wnt signaling and beta-catenin stabilization observed in CML. One possible mechanism for beta-catenin stabilization could be the acquisition of mutations at its N-terminal domain, especially in the third exon where it is marked via phosphorylation for degradation. We sought to determine whether mutations in the third exon of the beta-catenin gene are responsible for the observed Wnt activation in CML

Material and methods. We screened bone marrow specimens from 33 patients with CML in the chronic phase and also examined the K562 cell line for beta-catenin mutations.

Results. None of the patients nor the K562 cell line were found to carry mutations.

Conclusion. Beta-catenin amino-terminal mutations are not observed or very rare and therefore are not the underlying mechanism of activated Wnt signaling in CML.

Key words: beta-catenin, chronic myeloid leukemia, Wnt signaling, BCR-ABL.

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