

# ХРОНИЧНА МІЕЛОЦІДНА ЛЕВКЕМІЯ

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ОБЗОРИ

REVIEWS

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## MOLECULAR BIOLOGY OF CHRONIC MYELOID LEUKEMIA

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**Summary.** Chronic myeloid leukemia (CML) is characterized by Philadelphia chromosome and fusion BCR-ABL gene, resulting in a synthesis of hybrid Bcr-Abl oncoproteins with an elevated tyrosine kinase activity. Depending on the BCR gene breakpoint location, three main forms of the BCR-ABL fusion gene might be generated. Each of them encodes distinctive Bcr-Abl oncoprotein, associated with a characteristic disease phenotype: p210<sup>Bcr-Abl</sup>(+) "classical" CML, p190<sup>Bcr-Abl</sup>(+) "CML with monocytosis" and p230<sup>Bcr-Abl</sup>(+) "neutrophilic" CML. Bcr-Abl interact with multiple signal transducers, structural proteins and other cellular effector molecules leading to altered cellular adhesion, activation of mitogenic signalling cascades and inhibition of apoptosis. The elucidation of the molecular biology of CML provides the opportunity to design new therapeutic approaches, targeting various steps in the pathogenesis of CML. The main strategies used for this purpose include blocking BCR-ABL gene expression at mRNA or protein level and/or disruption of the Bcr-Abl-associated pathways by inhibition of target proteins that are modulated by Bcr-Abl.

**Key words:** leukemia, myeloid, chronic/genetics; fusion proteins, bcr-abl/genetics; Philadelphia chromosome; translocation genetics (source: MeSH)

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