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Pathophysiology and Molecular Targets in Myeloid Neoplasia

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submissions:

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Message from the Guest Editor

Dear Colleagues,

Myeloid cancers developing from the aging hematopoietic system share common genetic and epigenetic aberrations, irrespective of the clinical phenotype. While current treatment options are clearly focused on the disease phenotype and the underlying driver mutations, cancers within the myeloid spectrum rather represent a continuum of diseases. The underlying genetic and epigenetic landscape, comparable metabolic requirements, common functional dependencies, and shared interface with the immune system may facilitate the definition of pan-myeloid disease mechanisms and therapeutic targets. Identification of common pathophysiologic mechanisms may facilitate development of therapies to prevent progression or induce regression of the underlying clonal landscape. In this Special Issue, we aim to focus on commonalities and differences between preleukemic conditions and various blood cancers of the myeloid spectrum that may serve as therapeutic targets in the future. Contributions may therefore include primary research articles, reviews, as well as perspectives (if solicited by the editorial board).



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Special Issue



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