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Multi-Faceted Epigenetic Dysregulation in Acute Myeloid Leukemia

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Message from the Guest Editors

AML is a complex heterogenous disease characterized by the presence of highly proliferative myeloid progenitor cells with reduced capacity to differentiate. Recent advanced understanding combining knowledge about recurring genetic mutations and epigenetic alterations suggests that AML is an epigenetic disease, mainly because the disease is driven by driving/recurring mutations in epigenetic modulators, DNA/chromatin modifiers, RNA splicing, 3D chromosomal interactions, and chromatin remodeling complexes SWI/SNF and NPM1. Many of these regulatory processes consist of multiple enzymes and are probably interconnected. Moreover, the role of IDR (intrinsically disordered region) in oncoproteins such as NUP98-HOXA9 or UTX to form liquid condensate that controls transcription and genome architecture has provided new insights for epigenetic understanding in AML.

In this Special Issue, original research articles and reviews are welcome. Research areas may include (but are not limited to) the following: chromatin regulation, nuclear topology and 3D chromatin architecture, liquid condensates and phase separation, epi-mechanisms, RNA modifications, epi-metabolism.













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Message from the Editor-in-Chief

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