Special Issue

Development of Small Molecules for Acute Myeloid Leukemia Therapy

Message from the Guest Editors

Acute myeloid leukemia (AML) is an aggressive form of hematologic malignancy. Despite improved treatment-adapted approaches, more than 70% of AML patients have a low 5-year survival rate due to refractory or drug resistance, which warrants the identification of promising novel therapeutic targets and development of specific small molecule inhibitors. The US Food and Drug Administration (FDA) has approved small compounds targeting FLT3 mutations (Midostaurin), IDH1 mutation (ivosidenib), and IDH2 mutation (enasidenib), which provides new hope for targeted therapy as well as for the cure of AML. In addition, extensive efforts have been devoted to identifying attractive and safe druggable targets, delineating the underlying molecular mechanisms during leukemogenesis, and developing (designing) selective and effective small inhibitors for AML therapy. For instance, dysregulation of enzymes involving in epigenetic modifications (DNA methylation, histone modification, and N6-methyladenosine RNA modification) leads to leukemogenesis, and a variety of compounds targeting those enzymes have been identified for translational medicine and clinical trials.

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