



Targeting FLT3 Mutations in AML (Acute Myeloid Leukemia)

Guest Editor:

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Message from the Guest Editor

Dear Colleagues,

Mutations in the FMS-like tyrosine kinase 3 (FLT3) gene occur in about 30% of all acute myeloid leukemia (AML) patients. Whilst *FLT3*-ITD mutations confer undoubtedly poor prognosis with higher risk of relapse and lower survival rate, the role of *FLT3*-TKD mutations still remains unclear. *FLT3* mutations are *gatekeeper* mutations in AML and, as demonstrated in different murine models, play a key role as co-operative mutation in AML development. This makes FLT3 mutation a valid target for therapy.

With this Special Issue, we kindly invite our colleagues to submit their latest research findings, reviews or perspectives covering either biological or clinical aspects of *FLT3* mutations in AML. The topics may include new knowledge on pathways involved in leukemogenesis, as well as description of *in vitro* and *in vivo* *FLT3*-mutated AML models. Clinical significance of *FLT3* mutations in AML and related debated issues are invited to be addressed.





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Message from the Editor-in-Chief

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