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Targeting FLT3 Mutations in AML (Acute Myeloid Leukemia)

Guest Editor:

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Deadline for manuscript submissions:
closed (30 September 2022)

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 o f *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

Cancers is an international online journal addressing both clinical and basic science issues related to cancer research. The journal is publishing in Open Access format, which will certainly evolve to ensure that the journal takes full advantage of the rapidly changing world of information and knowledge dissemination. It publishes high-quality clinical, translational, and basic science research on cancer prevention, initiation, progression, and treatment, as well as other related topics, particularly to capture the most seminal studies in the rapidly growing area of immunology, immunotherapy, and tumor microenvironment.

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