Special Issue

Protein Kinase in Leukemia

Message from the Guest Editors

Protein kinases are required to phosphorylate substrates on threonine, serine or tyrosine residues, allowing structural protein modification leading to enzyme activation or recruitments of signaling partners at docking sites, giving raise to signaling transduction pathways. About 500 human genes encode kinases. Kinase activity controls signaling pathways involved in many cellular processes, such as cell growth, proliferation, differentiation or metabolism. In hematopoiesis, many kinases play a key role at different steps of blood cell generation, and deregulation, through mutations, expression or location, often leads to malignancies. Among these, several chronic or acute leukemias are driven by deregulated kinases, for which recent decades have seen the development of targeted therapies. Although tyrosine phosphorylations represent only 0.1% of whole phosphorylation, deregulated tyrosine kinases are involved in a large number of oncogenic mechanisms. The paradigm of kinase targeting is remembered by the development of the first inhibitor FDA-approved in chronic myeloid leukemia (CML) treatment, imatinib.

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Message from the Editor-in-Chief

Cancers (ISSN 2072-6694) is an international, online journal addressing both clinical and basic science issues related to cancer research. The journal will continue its 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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