

Special Issue

Oncogenesis of Lymphoma

Message from the Guest Editors

Advances in the fields of molecular genetics, immunology and virology have clarified the mechanisms involved in lymphomagenesis. Human lymphomas have been found to be heterogeneous, not only pathologically but also in terms of pathogenetic pathways, cellular derivation and the tumor microenvironment.

Traditionally, some types of lymphomas are consistently correlated with genetic abnormalities involving *BCL2* and *BCL1*, respectively. It is well known that the molecular pathway in Burkitt lymphoma involves activation of *MYC*, inactivation of *p53* and infection by EBV. Furthermore, in diffuse large B-cell lymphomas (DLBCL), molecular studies have shown rearrangements in *BCL2*, *BCL6* and *MYC* genes. A new concept, viral cooperation, has been revealed in lymphomagenesis by molecular virologic studies on primary effusion lymphomas (PELs). In immune-deficient/dysregulated patients, PEL tumor cells, in addition to consistent infection by KSHV/HHV8, are also commonly infected by EBV. Finally, recent insights from genetics, epigenetics and knowledge in the cellular microenvironment have led to the refinement of diagnostic definition and, hopefully, appropriate therapy.

Guest Editors

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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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