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Oncogenesis of Lymphoma

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Message from the Guest Editors

Dear Colleagues,

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.

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Message from the Editor-in-Chief

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