Retrovirology

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Oral presentation

The Mechanism of Epstein-Barr Virus Persistence in Vivo and its Relationship to the Origins of EBV Associated Lymphoma

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Epstein-Barr virus persists latently within resting memory B lymphocytes. To gain access to this compartment the virus first infects and activates a naïve B cell and then drives it to differentiate into a resting memory B cell. To achieve this the virus uses four different viral latent gene transcription programs which are also expressed in EBV associated lymphomas e.g. the growth program in immunoblastic lymphoma, the default program in Hodgkin's disease and the EBNA1 only program in Burkitt's lymphoma. This suggests:

1. that the EBV associated lymphomas arise when the normal progress of a latently infected, activated, naïve B cell to a resting, memory B cells is blocked.

2. the viral transcription program used by the tumor reflects the normal cellular counterpart that it is derived from.

The mechanism of EBV persistence will be described and the origins of the EBV lymphomas, predicted from this model, will be discussed.