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Targeting TRAF3 downstream signaling pathways in B cell neoplasms

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B cell neoplasms comprise >50% of blood cancers. However, many types of B cell malignancies remain incurable. Identification and validation of novel genetic risk factors and oncogenic signaling pathways are imperative for the development of new therapeutic strategies. We and others recently identified TRAF3, a cytoplasmic adaptor protein, as a novel tumor suppressor gene in B lymphocytes. We found that TRAF3 inactivation results in prolonged survival of mature B cells, which eventually leads to spontaneous development of B lymphomas in mice. Corroborating our findings, TRAF3 deletions and mutations frequently occur in human B cell chronic lymphocytic leukemia, non-Hodgkin lymphoma (such as splenic marginal zone lymphoma and mantle cell lymphoma), multiple myeloma, and Waldenström's macroglobulinemia. In this context, we have been investigating TRAF3 signaling mechanisms in B cells, and are developing new therapeutic strategies to target TRAF3 downstream signaling pathways in B cell neoplasms. Here the author will present and discuss the new translational data that demonstrate the therapeutic potential of targeting TRAF3 downstream signaling pathways in B lymphoma and multiple myeloma.

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