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Molecular mechanisms of B lymphomagenesis induced by TRAF3 inactivation

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TRAF3, a member of the TRAF family of cytoplasmic adaptor proteins, is employed in signaling by a variety of immune receptors, including the tumor necrosis factor receptor superfamily, Toll-like receptors, NOD-like receptors, and RIG-I-like receptors. To explore the *in vivo* functions of TRAF3 in B lymphocytes, we recently generated a genetically modified mouse model that has the TRAF3 gene specifically deleted in B cells. We found that TRAF3 deletion 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 inactivating mutations were identified in human B cell neoplasms, including multiple myeloma, splenic marginal zone lymphoma, B cell chronic lymphocytic leukemia, and mantle cell lymphoma. We are currently investigating the molecular mechanisms of TRAF3 inactivation-initiated B lymphomagenesis using complementary human and mouse model systems. To approach this, we employ a number of cutting-edge strategies in our study, including microarray analyses, proteomics, bioinformatics, and deep sequencing. Here the author presents new data of this project, which provide useful information for rational design of novel therapeutics and treatment strategies to combat human B cell malignancies.

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