Nfkb 1 is dispensable for Myc-induced lymphomagenesis.

Rel/NF-kappaB transcription factors are critical arbiters of immune responses, cell survival, and transformation, and are frequently deregulated in cancer. The p50 NF-kappaB 1 component of Rel/NF-kappaB DNA-binding dimers regulates genes involved in both cell cycle traverse and apoptosis. Nfkb 1 loss accelerates B cell growth and leads to increased B cell turnover in vivo, phenotypes akin to those manifested in B cells of Emu-Myc transgenic mice, a model of human Burkitt lymphoma. Interestingly, Emu-Myc B cells express reduced levels of cytoplasmic and nuclear NF-kappaB 1 and have reduced Rel/NF-kappaB DNA-binding activity, suggesting that Myc-mediated repression of NF-kappaB 1 might mediate its proliferative and apoptotic effects on B cells. Furthermore, Nfkb 1 expression was reduced in the majority of Emu-Myc lymphomas and was also suppressed in human Burkitt lymphoma. Nonetheless, loss of Nfkb 1 did not appreciably affect Myc’s proliferative or apoptotic responses in B cells and had no effect on lymphoma development in Emu-Myc mice. Therefore, Nfkb 1 is dispensable for Myc-induced lymphomagenesis.