Since the initial cloning of RelA and its close relationship to c-Rel, the cellular homolog of the viral oncoprotein v-Rel, the nuclear factor ?B (NF-?B) signaling pathway and its upstream activating kinase complex (I?B-kinase) have been suspected to play a major role in tumorigenesis. This was further corroborated by the discovery of oncogenic mutations in NF-?B proteins in certain lymphoid malignancies and the notion that NF-?B is persistently activated in a large variety of solid tumors. With the advent of conditional knockout mice allowing tissue-specific targeting of the various components of the NF-?B signaling pathway, it was possible to genetically test the cell autonomous and non-autonomous functions of NF-?B in inflammation-associated cancer as well as sporadic cancers. Here, we review molecular evidence that demonstrates the various functions of NF-?B during different tumor stages and that supports the rationale to target NF-?B in cancer prevention and therapy.

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