A precise balance between cellular apoptosis and cellular survival is essential for the proper functioning of the immune system. Whereas apoptosis eliminates self-reactive or supernumerary lymphocytes, survival signaling that counteracts apoptotic programs is needed to allow B and T lymphocytes that recognize pathogens to become activated and expand in response to infection. A major regulator of lymphocyte survival and activation is the transcription factor NF-kappaB. Controlled activation of NF-kappaB is essential for normal innate and adaptive immune responses, and dysregulated NF-kappaB signaling in lymphocytes contributes to diseases ranging from chronic inflammation and autoimmunity to lymphoma. The core NF-kappaB activating machinery composed of the NF-kappaB, IkappaB and IKK proteins is relatively well-characterized, but it is less clear how distinct upstream stimuli activate NF-kappaB in a tissue-, time- and signal-specific manner. In this review, we discuss recent insights into the specific signal transduction pathways leading to NF-kappaB activation that are triggered by engagement of the antigen receptors of T and B cells. We focus mainly on T cell receptor (TCR)-mediated NF-kappaB activation and draw parallels to B cell receptor (BCR)-mediated NF-kappaB activation where appropriate.