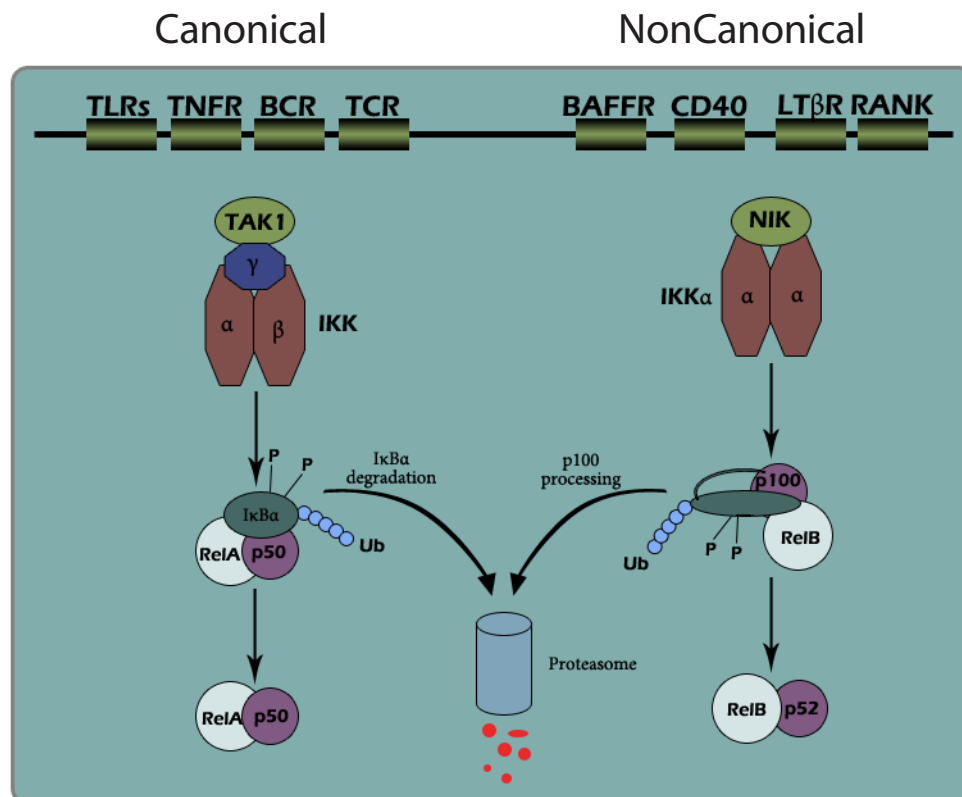


NF-κB Signaling

Nuclear factor-kappa B (NF-κB) is a very important transcription factor that regulates key genes involved in inflammation, tumorigenesis, apoptosis, autoimmune disorders, and inflammation. NF-κB is activated in response to proinflammatory cytokines, stress, UV rays, lymphokines, viral antigens etc. This transcription factor in its inactive form is generally sequestered in the cytoplasm, bound to inhibitor of kappa B (IκB). NF-κB is a fast-acting transcription factor because it does not have to be transcribed and translated after the harmful cellular stimuli, as it is already present in the cytoplasm. Appropriate stimuli for e.g. binding of the bacterial antigen LPS (lipopolysaccharide) to its Toll-like-receptors (TLR), or tumor necrosis factor (TNF) binding to its receptor TNFR etc. initiates the signaling pathway. The nuclear localization signal (NLS) of NF-κB is masked by its binding to IκB in the cytoplasm. In the presence of appropriate stimuli, the IκB kinase (IKK) phosphorylates serine residues on IκB's, which are then degraded by ubiquitination. With the exposed NLS, the NF-κB now translocates into the nucleus, where it binds to specific cis-acting elements on specific genes, thereby resulting in their transcription. These genes are responsible for key physiological responses like cell survival, proliferation, inflammatory or immune response. NF-κB also regulates the expression of IκB which is transcribed and translated. This newly formed IκB now sequesters NF-κB again into the cytoplasm, thereby resulting in a feed-back loop.

Since NF-κB is so actively involved in a number of key events like cell proliferation and cell survival, many cancer cells have constitutively active NF-κB with mutations. Specifically blocking its expression in tumor cells may cause these active proliferating cells to die. Hence, currently, several pharmaceutical companies have active programs to develop anti-cancer and anti-inflammatory drugs that function via blockade of the NF-κB signaling pathway.

Canonical and Non-Canonical NF-κB Signaling Pathways



Antibodies

Name	Cat. #
CIKS/Act1 Antibody	3314-100
IkB α Antibody	3252-100
IkB α Antibody	3315-100
IKK α /IKK-1 Antibody	3185-100
IRAK-4 Antibody	3580-100
MYD88 Antibody	3244R-100
NFkB Blocking Peptide	3038BP-50
NFkB p105 Antibody	3897-200
NFkB p105 Blocking Peptide	3897BP-50
NFkB p50 Antibody	3354R-100
NFkB p50 Blocking Peptide	3354RBP-50
NFkB p65 Antibody	3012-100
NFkB p65 Antibody	3038-100
NIK Antibody	3193R-100
NIK Blocking Peptide	3193RBP-50
TANK Polyclonal Antibody	3348-100
TLR1 Antibody	3446-100
TLR10 Antibody	3932-100
TLR10 Blocking Peptide	3932BP-50
TLR11 Antibody	3931-100
TLR11 Blocking Peptide	3931BP-50
TLR2 Antibody	3552R-100
TLR2 Antibody	3569-100
TLR2 Blocking Peptide	3552RBP-50
TLR3 Antibody	3445R-100
TLR3 Blocking Peptide	3445BP-50
TLR4 Antibody	3251-100
TLR4 Antibody	3253-100
TLR4 Blocking Peptide	3253BP-50
TLR5 Antibody	3555R-100
TLR5 Blocking Peptide	3555RBP-50
TLR7 Antibody	3557-100
TLR8 Antibody	3558-100
TLR9 Antibody	3559R-100
TLR9 Blocking Peptide	3559RBP-50
TNF-alpha Antibody	3052R-100
TNF-alpha Antibody	3053R-100
TNF-alpha Antibody	3054-100
TRAF3 Antibody	3563-100
TRAF6 Antibody	3566R-100
TreakR Antibody	3564-100
TROY/TAJ Antibody	3562R-100

Proteins/Enzymes

Name	Cat. #
RIPK2, Active	7747-5

Inhibitors

Name	Cat. #
PDTC	1676-100
A77 1726	1973-5, 25
BAY 11-7082	1867-10, 50
Celastrol	1940-5, 25
Honokiol	1762-10, 50
IKK- ϵ Kinase Inhibitor I	1810-1,5
IKK- ϵ Kinase Inhibitor II	1811-1,5
ML130	1974-5, 25
Parthenolide	1868-10, 50
Rocaglamide	1863-100
Triptolide	1761-1, 5