

DB036: NFkB p52 (C20)

Background:

Members of the rel/NFkappaB family of transcription factors are involved in the regulation of cellular responses, such as growth, development, and the inflammatory response. They share a structural motif known as the rel homology region (RHR), the C-terminal one third of which mediates protein dimerization (2, 6, 8). Complexes of p50 (NF-kB1) or p52 (NF-kB2) are generated through the processing of p105 and p100 precursors, respectively. These are usually associated with members of the Rel family (p65, c-Rel, Rel B). The homo- and heterodimer formed through combinations of NF-kB/Rel proteins bind distinct kB sites to regulate the transcription of different genes (7, 9). In resting cells, NFkappaB is retained in the cytoplasm bound to inhibitory proteins of the IkappaB family. Degradation of IkappaB proteins occurs with cell activation, via of variety of signals, including inflammatory cytokines and bacterial lipopolysaccharides (LPS) as well as oxidative and fluid mechanical stress. This results in nuclear translocation of NfkappaB and the transcriptional gene activation of proinflammatory genes (1, 9). It has been suggested that NFkappaB plays a role in the development of numerous pathological states. Activation of NFkappaB induces gene programs leading to transcription of factors that promote inflammation, such as leukocyte adhesion molecules, cytokines, and chemokines. It is also thought that there are some substances with possible anti-inflammatory effects that are also NFkappaB regulated. There is some evidence indicating NFkappaB as a key factor in the pathophysiology of cardiac ischemia-reperfusion injury as well as the development of insulin dependent Diabetes Mellitus (4, 3).

Origin:

NFkB p52 (C20) is provided as an affinity purified rabbit polyclonal antibody, raised against a peptide mapping to the carboxy terminus of mouse NFkB p52.

Product Details:

Each vial contains 200 μg/ml of affinity-purified rabbit IgG, NFkB p52 (C20) *DB036*, in 1 ml PBS containing 0.1 % sodium azide and 0.2% gelatin.

Competition Studies:

A blocking peptide is also available, DB036P, for use in competition studies. Each vial contains 100 µg of peptide in 0.5 ml PBS with 0.1% sodium azide and 100 µg BSA.

Specificity:

NFkB p52 (C20) *DB036* reacts with NFkB p52 and p100 of mouse, rat, and human origin by western blotting, immunoprecipitation and immunohistochemistry.

Storage:

Store this product at 4° C, do not freeze. The product is stable for one year from the date of shipment.

References:

- 1. Lentsch AB, Ward PA. The NFkappaBb/IkappaB system in acute inflammation. Arch Immunol Ther Exp (Warsz). 48(2): 59-63.
- 2. Hatada EN, Krappmann D, Scheidereit C. NF-kappaB and the innate immune response. Curr Opin Immunol. 2000 Feb; 12(1): 52-8
- 3. Ho E, Bray TM. Antioxidants, NFkappaB activation, and diabetogenesis. Proc Soc Exp Biol Med. 1999 Dec; 222(3): 205-13.
- 4. Valen G, Yan ZQ, Hansson GK. Nuclear factor kappa-B and the heart. J Am Coll Cardiol. 2001 Aug; 38(2): 307-14.
- 5. Chen FE, Kempiak S, Huang DB, Phelps C, Ghosh G. Construction, expression, purification and functional analysis of recombinant NFkappaB p50/p65 heterodimer. Protein Eng. 1999 May; 12(5): 423-8.
- Sengchanthalangsy LL, Datta S, Huang DB, Anderson E, Braswell EH, Ghosh G. Characterization of the dimer interface of transcription factor NFkappaB p50 homodimer. J Mol Biol. 1999 Jun 18; 289(4): 1029-40.
- 7. Huang DB, Huxford T, Chen YQ, Ghosh G. The role of DNA in the mechanism of NFkappaB dimer formation: crystal structures of the dimerization domains of the p50 and p65 subunits. Structure. 1997 Nov 15; 5(11): 1427-36.
- 8. Magnani M, Crinelli R, Bianchi M, Antonelli A. The ubiquitin-dependent proteolytic system and other potential targets for the modulation of nuclear factor-kB (NF-kB). Curr Drug Targets. 2000 Dec; 1(4): 387-99.