

DESCRIPTION

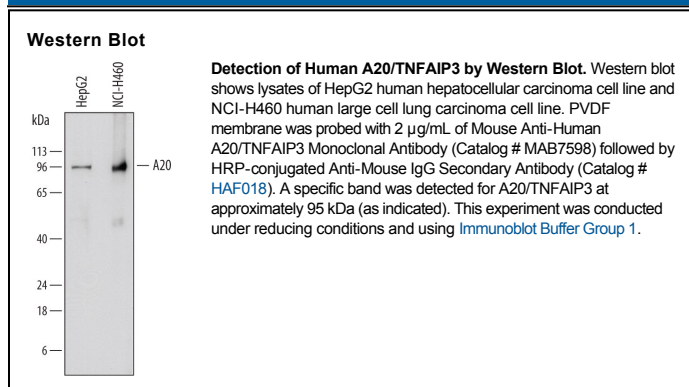
Species Reactivity	Human
Specificity	Detects human A20 in direct ELISAs. In direct ELISAs, no cross-reactivity with recombinant human A20/TNFAIP3 (aa 440-790) is observed.
Source	Monoclonal Mouse IgG _{2B} Clone # 775912
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	<i>E. coli</i> -derived recombinant human A20 Lys91-Leu263 Accession # P21580
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 µm filtered solution in PBS.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Western Blot	2 µg/mL	See Below

DATA



PREPARATION AND STORAGE

Reconstitution	Sterile PBS to a final concentration of 0.5 mg/mL.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

A20 (TNFα-induced protein 3) is a cytoplasmic zinc finger protein that inhibits NFκB activity and tumor necrosis factor-mediated programmed cell death. The protein interacts with NAF1 and inhibits TNF-induced NFκB-dependent gene expression by interfering with RIP- or TRAF2-mediated transactivation signaling. A20 contains an N-terminal domain which has deubiquitinating enzyme activity and removes ubiquitin chains from receptor-interacting protein (RIP), thus mediating distinct regulatory effects in the down-regulation of NFκB signaling.