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SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

mail@szabo-scandic.com

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Datasheet for KCB002

HRP Western Blot Anti-Mouse IgG Antibody

Overview

Description:	HRP Western Blot Anti-Mouse IgG Antibody - KCB002
Item No.:	KCB002
Size:	100 µg
Applications:	WB, IP
Host Species:	Goat

Product Details

Background:	HRP is used in applications primarily for its ability to amplify a weak signal and increase detectability of a target molecule. HRP-conjugated secondary antibodies are utilized in conjunction with specific chemiluminescent substrates to generate the light signal and HRP conjugates have a very high turnover rate, yielding good sensitivity with short reaction times. Chemiluminescence is the best all-around method for western blot detection as these systems eliminate the hazards associated with radioactive materials and toxic chromogenic substrates. These methods are also unmatched in the speed and sensitivity compared to traditional alternatives, utilizing film to record and store data permanently. Blots detected with chemiluminescent methods are easily stripped for subsequent reprobing with additional antibodies. This item is a component of kit (KCA002). The Kit contains all the reagents necessary to perform chemiluminescent Western Blots to detect mouse primary antibodies.
Synonyms:	Anti-mouse IgG HRP conjugated, HRP-linked Antibody, Anti-Mouse HRP Secondary Antibody, Horseradish Peroxidase-Conjugated Antibody
Host Species:	Goat
Conjugate:	Peroxidase (HRP)
Clonality:	Polyclonal
Detection Kit Type:	Chemiluminescent Western Blot Kit

Application Details

Tested Applications:	WB
Suggested Applications:	IP (Based on references)

Application Note:	Western Blot Analysis
Assay Dilutions:	All assays should be optimized by the user. Recommended dilutions (if any) may be listed below.
ELISA:	1:10,000-1:100,000
IHC:	1:500-1:5,000
WB:	1:5,000-1:40,000

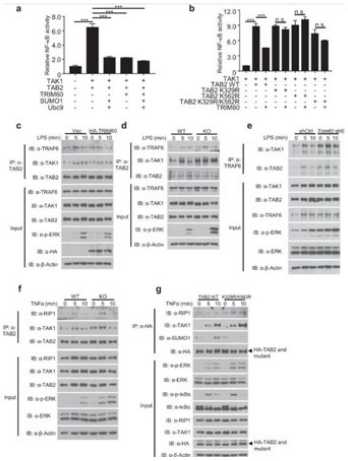
Formulation

Concentration:	1.0 mg/ml by UV absorbance at 280 nm
Buffer:	0.02 M Potassium Phosphate, 0.15 M Sodium Chloride, pH 7.2
Preservative:	0.01% (w/v) Gentamicin Sulfate. Do NOT add Sodium Azide!
Reconstitution Volume:	100 µL
Reconstitution Buffer:	Restore with deionized water (or equivalent)

Shipping & Handling

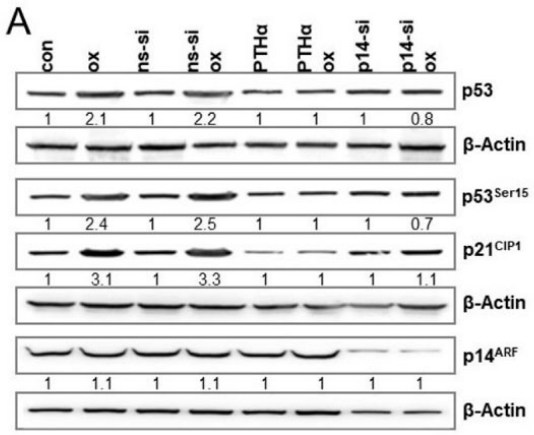
Shipping Condition:	Wet Ice
Storage Condition:	See kit insert for complete instructions.
Expiration:	See kit insert for complete instructions.

Images

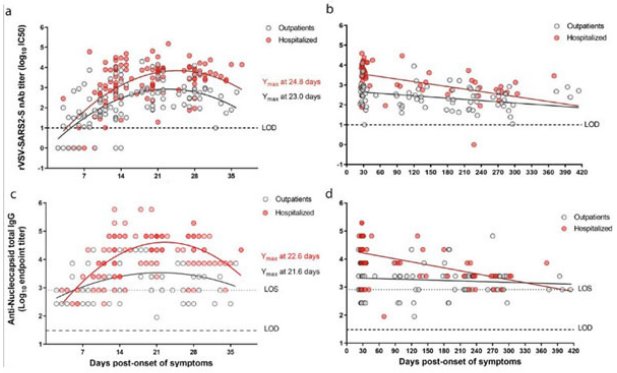


Western Blot

SUMOylation of TAB2 inhibits NF-κB activation by suppressing the TRAF6/TAB2/TAK1 complex. a Dual luciferase assay analysis of the effects of TRIM60-mediated SUMOylation on TAK1/TAB2-induced NF-κB activity. HEK293T cells were transiently transfected with the indicated plasmids, and the dual luciferase assay was performed. b Dual luciferase assay analysis of the effects of TAB2 mutants on the TRIM60-mediated suppression of NF-κB activity. c IP and WB analyses of the TRAF6/TAB2/TAK1 complex in control and HA-TRIM60-overexpressing RAW cells stimulated by LPS as indicated. Formation of the TRAF6/TAB2/TAK1 complex was examined in BMDMs (d) and RAW cells (e). Cells were stimulated with LPS for the indicated amounts of time, and IP and WB analyses were performed. f TRIM60 suppresses RIP1/TAB2/TAK1 signalosome formation in MEFs. IP and WB analyses of the RIP1/TAB2/TAK1 complex in MEFs. WT and TRIM60 KO MEFs were stimulated with TNFα as indicated, followed by IP and WB analyses. g IP and WB analyses of MAPK/NF-κB signaling activation and RIP1/TAB2/TAK1 complex formation. TAB2-deficient MEFs rescued with WT or TAB2-K329R/K562R were stimulated with TNFα as indicated, followed by IP and WB analyses to detect RIP1/TAB2/TAK1 complex formation, TAB2 SUMOylation, and phosphorylation of ERK and IκBα. The formation of the TRAF6/TAB2/TAK1 complex in c and d are quantified by ImageJ and shown as Supplementary Fig. 8a and b, respectively. The firefly luciferase activity levels in a and b were normalized to the Renilla luciferase activity levels and are presented as the mean ± SEM. ***P < 0.001; n.s. no significance (one-way ANOVA followed by Tukey's multiple comparisons). The data are representative of three independent experiments (a–g). HRP-conjugated Goat anti-mouse secondary antibody (p/n KCB002) was used. Fig 5. PMID: 33184450.



Western Blot
(A) LoVo cells were either exposed to PTHα (30 µM) or transfected with non-specific siRNA (ns-siRNA) or p14ARF specific siRNA. Cells were treated with 2.5 µM oxaliplatin 8 h after siRNA or 1 h after PTHα treatment. 120 h upon oxaliplatin exposure, the expression of p14ARF, p21CIP1, and p53, as well as the phosphorylation of p53 at Ser15 was measured by immunodetection. HRP conjugated goat anti-mouse (p/n KCB002) and HRP conjugated goat anti-rabbit (p/n KCB003) were used. Fig 7. PMID: 33922007.



Neutralization
Longitudinal dynamics of neutralizing and anti-N antibody responses to SARS-CoV-2 infection from outpatient and hospitalized individuals. a,b. The half-maximum inhibitory concentration (IC50) of sera was determined by microneutralization assay of recombinant vesicular stomatitis virus carrying SARS-CoV-2 spike protein (rVSV-SARS2-S). a. Neutralizing antibody (nAb) titres (log10 IC50) from n = 30 outpatients (116 samples; grey circles) and n = 35 hospitalized (112 samples; red circles) at 2 to 37 days post-symptom onset. c. Longitudinal nAb titres (log10 IC50) from n = 36 outpatients (85 samples) and n = 31 hospitalized (58 samples) taken from day 23 (outpatients) or day 25 (hospitalized) until day 414 post-symptom onset. c,d. The end-point titres of anti-N IgG were determined by ELISA using a recombinant SARS-CoV-2 nucleocapsid protein. Samples and time points are the same as those in A and B. a-c. The second order polynomial (quadratic) curve fitting was used to establish the days at which peak titres occurred (Ymax). b–d. Continuous decay fit is shown with the red and gray line for the corresponding patient group. Every data point represents results from two technical replicates. HRP conjugated goat anti-mouse (p/n KCB002) was used at 1:3000. Fig 1. PMID: 35366624.

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