

Datasheet for ABIN965415

Goat anti-Mouse IgG Antibody (HRP)





Overview

Quantity:	100 μg
Target:	IgG
Reactivity:	Mouse
Host:	Goat
Clonality:	Polyclonal
Conjugate:	HRP
Application:	Western Blotting (WB)

Product Details

Target Details

Target:	IgG
Abstract:	IgG Products
Target Type:	Antibody
Background:	Synonyms: Anti-mouse IgG conjugated, -linked Antibody, Anti-Mouse Secondary Antibody, Horseradish Peroxidase-Conjugated Antibody

Application Details

Application Notes:	Western Blot Analysis
Comment:	Detection Kit Type: Chemiluminescent Western Blot Kit

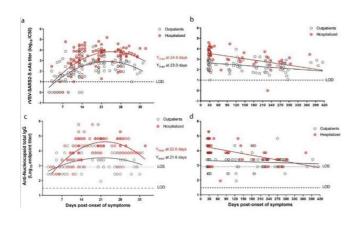
Application Details

Restrictions: For Research Use only

Handling

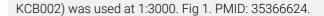
Buffer:	0.02 M Potassium Phosphate, 0.15 M Sodium Chloride, pH 7.2
Preservative:	Gentamicin sulfate
Handling Advice:	Do NOT add Sodium Azide! Use of Sodium Azide will inhibit enzyme activity of horseradish peroxidase.
Storage:	4 °C

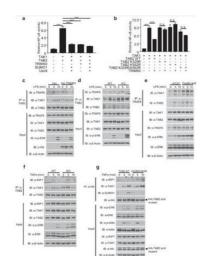
Images



Neutralization

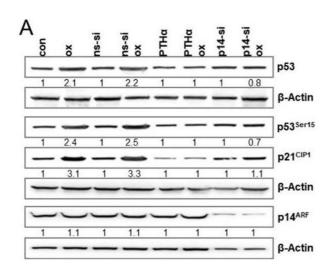
Image 1. Longitudinal dynamics of neutralizing and anti-N responses to SARS-CoV-2 infection from antibody outpatient and hospitalized individuals. a,b. The halfmaximum inhibitory concentration (IC50) of sera was determined by microneutralization assay of recombinant vesicular stomatitis virus carrying 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 = 31hospitalized (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





Western Blotting

Image 2. SUMOylation of TAB2 inhibits NF-kB 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-kB 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 NFκB 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 RIP1/TAB2/TAK1 suppresses 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-кВ signaling activation and RIP1/TAB2/TAK1 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 formation, TAB2 SUMOylation, complex and phosphorylation of ERK and IkBa. 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 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 antimouse secondary antibody (p/n KCB002) was used. Fig 5. PMID: 33184450.

Western Blotting

Image 3. (A) LoVo cells were either exposed to PTHa (30 μ M) or transfected with non-specific siRNA (ns-siRNA) or p14ARF specific siRNA. Cells were treated with 2.5 μ M oxaliplatin 8 h after siRNA or 1 h after PTHa 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.