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Anti-Lysozyme LYZ Rabbit Monoclonal Antibody

Catalog Number: M01811

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Overview

Product Name	Anti-Lysozyme LYZ Rabbit Monoclonal Antibody
Reactive Species	Human, Mouse, Rat
Description	Boster Bio Anti-Lysozyme LYZ Rabbit Monoclonal Antibody catalog # M01811. Tested in WB, IHC, ICC/IF, IP applications. This antibody reacts with Human, Mouse, Rat.
Application	IP, IF, IHC, ICC, WB
Clonality	Monoclonal EBO-12
Formulation	Rabbit IgG in stabilizing components, phosphate buffered saline, pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol. *This antibody is supplied in a stabilized formulation. Compatibility with conjugation reactions depends on the chemistry of the conjugation method used. For conjugation methods that are not compatible with the stabilizing components present in this formulation, a carrier-free antibody format is required.
Storage Instructions	Store at -20°C for one year. For short term storage and frequent use, store at 4°C for up to one month. Avoid repeated freeze-thaw cycles.
Host	Rabbit
Uniprot ID	P61626

Technical Details

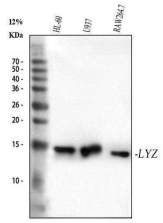
Immunogen	A synthesized peptide derived from human Lysozyme
Isotype	Rabbit IgG
Form	Liquid
Concentration	0.5mg/ml
Purification	Affinity-chromatography
Suggested Dilutions	WB, 1:1000-5000

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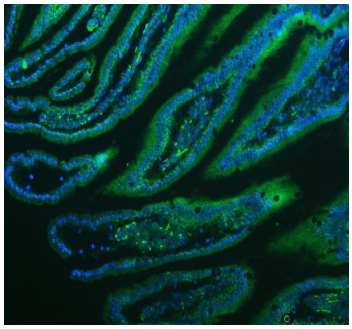
	IHC, 1:50-200 ICC/IF, 1:50-200 IP, 1:20

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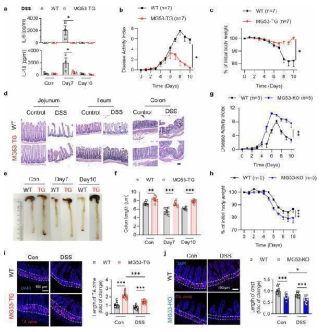
Anti-Lysozyme LYZ Rabbit Monoclonal Antibody (M01811) Images



Western blot analysis of Lysozyme using anti-Lysozyme antibody (M01811). Electrophoresis was performed on a 5-20% SDS-PAGE gel at 70V (Stacking gel) / 90V (Resolving gel) for 2-3 hours. The sample well of each lane was loaded with 30 ug of sample under reducing conditions. Lane 1: human HL-60 whole cell lysates, Lane 2: human U937 whole cell lysates, Lane 3: mouse RAW264.7 whole cell lysates. After electrophoresis, proteins were transferred to a nitrocellulose membrane at 150 mA for 50-90 minutes. Blocked the membrane with 5% non-fat milk/TBS for 1.5 hour at RT. The membrane was incubated with rabbit anti-Lysozyme antigen affinity purified monoclonal antibody (Catalog # M01811) at 1:5000 overnight at 4°C, then washed with TBS-0.1%Tween 3 times with 5 minutes each and probed with a goat anti-rabbit IgG-HRP secondary antibody at a dilution of 1:5000 for 1.5 hour at RT. The signal is developed using an Enhanced Chemiluminescent detection (ECL) kit (Catalog # EK1002) with Tanon 5200 system. A specific band was detected for Lysozyme at approximately 15 kDa. The expected band size for Lysozyme is at 17 kDa.



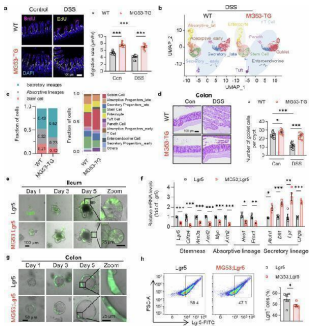
IF analysis of Lysozyme using anti-Lysozyme antibody (M01811). Lysozyme was detected in a paraffin-embedded section of mouse colon tissue. Heat mediated antigen retrieval was performed in EDTA buffer (pH 8.0, epitope retrieval solution). The tissue section was blocked with 10% goat serum. The tissue section was then incubated at 1:50 rabbit anti-Lysozyme Antibody (M01811) overnight at 4°C. DyLight®488 Conjugated Goat Anti-Rabbit IgG (BA1127) was used as secondary antibody at 1:500 dilution and incubated for 30 minutes at 37°C. The section was counterstained with DAPI. Visualize using a fluorescence microscope and filter sets appropriate for the label used.



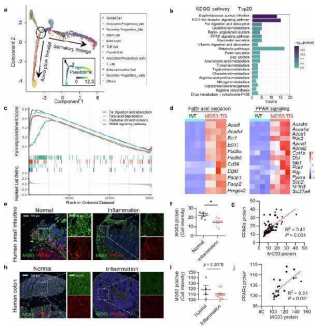
MG53 attenuates intestinal injury in mouse models. a Serum IL-6 and IL-18 levels of MG53-TG and their WT littermates at the indicated time points following DSS treatment. n = 3 for each group. Overexpression of MG53 attenuated DSS-induced IBD symptoms, including disease activity index (DAI; b), body weight change (c), disruption of intestinal structure as demonstrated by H&E staining of the jejunum, ileum, and colon (d ; scale bar, 100 um), and shortening of the colon as evidenced by the representative images (e) and statistic results of colon length (f) in MG53-TG and WT mice. n = 7 for each group. g , h Depletion of MG53 exacerbated DSS-induced IBD symptoms. The DAI (g) and body weight change (h) of MG53-KO and the WT littermates at the indicated time points after DSS challenge. n = 5 for each group. Immunofluorescence staining of Ki-67 (red) to indicate the TA zone and the statistic results of its length in MG53-TG (i , n = 20 for each group) or MG53-KO (j , n = 13 for each group) as compared with their corresponding WT

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littermates. The nuclei were indicated by DAPI staining (blue); scale bar, 100 um. Normal distribution was confirmed by Shapiro–Wilk test. Data were analyzed using two tailed paired t test (a – c , f – j). All data were presented as mean ± s.e.m. * $p < 0.05$, ** $p < 0.01$, and *** $p < 0.001$ as compared with the corresponding controls Full size imageIndex in PubMed under a CC BY license. PMID: 40494851



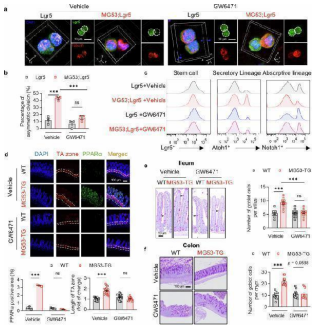
MG53 overexpression promotes secretory lineage commitment. a Immunofluorescence staining of BrdU (red) and EdU (yellow) in the ileum of MG53-TG and their WT littermates subjected to DSS treatment and administered BrdU 48 h and EdU 24 h before euthanasia. n = 10 for each group. Scale bar, 100 um. b UMAP of scRNA-seq of Epcam + CD45 − intestinal epithelial cells collected from MG53-TG and their WT littermates on Day 7 of DSS treatment, n = 5 for each group. c Proportions of stem, secretory, and absorptive cells in general and each particular cell clusters. d Representative images and statistic results of PAS staining of goblet cells of MG53-TG and WT mice in the colon. n = 10 for each group. Scale bar, 100 um. e Representative images of intestinal organoids derived from MG53;Lgr5 mice and their Lgr5 littermates at the indicated time points. f Relative mRNA levels of the marker genes in the transcriptome analysis of the crypt organoids. n = 4 for each group. g Representative images of colonic organoids derived from MG53;Lgr5 mice and their Lgr5 littermates at the indicated time points. h Representative FACS profiles and statistic results of Lgr5 + cells in the colonic organoids derived from MG53;Lgr5 and the control Lgr5 mice. n = 6 for each group. Normal distribution was confirmed by Shapiro–Wilk test. Data were analyzed using two tailed paired t test (a , d , f , and h), and were presented as mean ± s.e.m. * $p < 0.05$, ** $p < 0.01$, and *** $p < 0.001$ as compared with the corresponding controls Full size imageIndex in PubMed under a CC BY license. PMID: 40494851



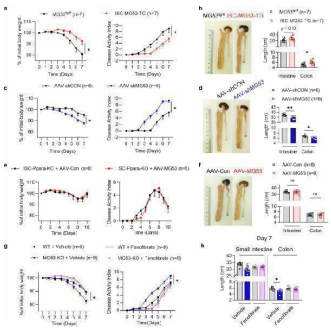
PPAR signaling is enhanced by MG53 overexpression. a Trajectory analysis of Epcam + CD45 − intestinal epithelial cells Using Monocle 2. b KEGG pathway enrichment analysis of the differentially expressed genes between the secretory and absorptive branches. GSEA results (c) and the heatmap of differentially expressed genes (d) related to fatty acid oxidation and PPAR signaling derived from the bulk RNAseq data of the intestinal organoids from MG53-TG and WT mice. n = 4 for each group. Representative images (e) and statistic results of signal intensity of the immunofluorescence staining of MG53 (green) and PPARalpha (red) determined by Aperio Scanscope scanner and Halo software (f), as well as the correlation of the levels of these two proteins (g) in the human small intestine of normal subjects (n = 5) and patients with intestinal inflammation (n = 8). Nuclei were stained with DAPI (blue); scale bars as indicated. Representative images (h) and statistic results of signal intensity of the immunofluorescence staining of MG53 (green) and PPARalpha (red) determined by Aperio Scanscope scanner

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and Halo software (i), as well as the correlation of the levels of these two proteins (j) in the human colon of normal subjects (n = 4) and patients with intestinal inflammation (n = 12). Normal distribution was confirmed by Shapiro–Wilk test. Data were analyzed using Mann–Whitney U test (f , i) and Pearson correlation analysis (g , j). Data were presented as mean ± s.e.m. * p < 0.05 as compared with the corresponding controls Full size imageIndex in PubMed under a CC BY license. PMID: 40494851



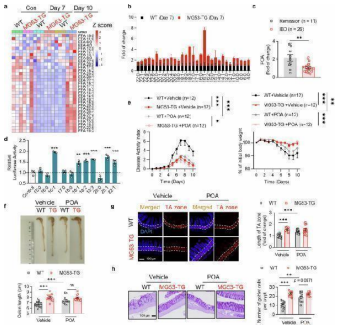
PPARalpha mediates the effect of MG53 in promoting asymmetric division of Lgr5 + cells. Representative images (a) and statistic results (b , n = 3 for each group) of asymmetric division of Lgr5 + cells with or without GW6471 (10 uM, 24 h) treatment. Lgr5 + cells were in green; all the cells were visualized by tubulin (red). Scale bar, 10 um. c FACS analysis of Lgr5 + , Atoh1 + , and Notch1 + cells in the Lgr5 and MG53;Lgr5 organoids treated with GW6471 or vehicle. d Immunofluorescence staining of Ki-67 (red) and PPARalpha (green) and statistic results of PPARalpha positive cells (n = 3 for each group) and the length of TA zone (n = 18 for each group) in mice treated GW6471 or vehicle. Nuclei were stained with DAPI (blue); scale bar, 100 um. Representative images and statistic results of PAS staining of goblet cells in the ileum (e ; n = 10 for each group; TG, MG53-TG; scale bar, 50 um) and colon (f ; n = 10 for each group; scale bar, 100 um). Normal distribution was confirmed by Shapiro–Wilk test. Data were analyzed using one-way ANOVA with Tukey post hoc test (b , d , e , and f) and were presented as mean ± s.e.m. ns not significant and *** p < 0.001 as compared with the corresponding controls Full size imageIndex in PubMed under a CC BY license. PMID: 40494851



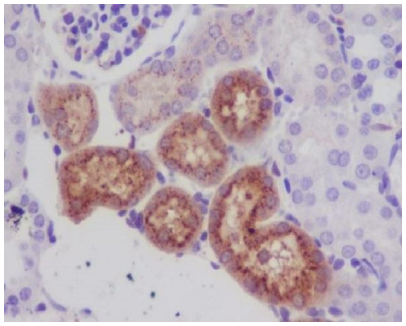
Activation of PPARalpha signaling is required for MG53-induced alleviation of intestinal injury. Body weight change and DAI (a) as well as the representative images and statistic results of the intestinal length (b) of ISC-MG53-TG and the control MG53 tg-fl mice. n = 7 for each group. Body weight change and DAI (c) as well as the representative images and statistic results of the intestinal length (d) of the Lgr5 mice with ISC-specific inhibition of MG53 expression via infection with adeno-associated virus with inducible expression of shRNA targeting MG53 (AAV-shMG53) or the control mice infected with control shRNA (AAV-shCON). n = 6 for each group. Body weight change and DAI (e) as well as the intestinal length (f) of ISC-Ppara-KO mice with or without ISC-specific MG53 overexpression via injecting AAV-MG53 or AAV-CON. n = 8 for each group. Body weight change and DAI (g) as well as the intestinal length (h) of MG53-KO and control WT mice treated with PPARalpha agonist fenofibrate or vehicle following DSS treatment. n = 8 for each group. Normal distribution was confirmed by Shapiro–Wilk test. Data were analyzed using two-tailed paired t test (a – e , g), Mann–Whitney U test (f , h), and were presented as mean ± s.e.m. ns not significant, * p < 0.05 and ** p < 0.01 as compared with the corresponding

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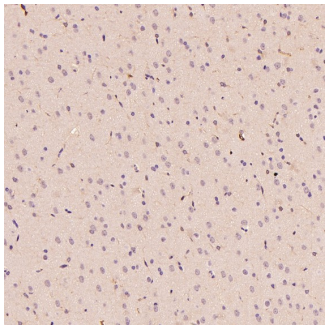
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Enhanced PPARalpha activation by palmitoleic acid contributes to MG53-mediated amelioration of intestinal injury. a Heatmap of free fatty acids (FFA) in MG53-TG and WT intestinal tissues at the indicated time points during DSS treatment. n = 3 for each group. b Changes in the intestinal content of FFA in the MG53-TG and their WT littermates at day 7 after DSS treatment. n = 3 for each group. c Relative change in POA concentrations in the serum of IBD patients in remission (n = 11) or flare (n = 26). d Luciferase reporter activity driven by the PPARalpha-responsive element in the presence of different FFA in HEK293 cells. n = 3 for each group. Body weight change and DAI (e) as well as the colon length (f) of MG53-TG (TG) and their WT littermates challenged with DSS with or without POA treatment. n = 12 for each group. g Immunofluorescence staining of Ki-67 (red) and statistic results of the length of TA zone of MG53-TG and their WT littermates with or without POA treatment. n = 15 for each group. The nuclei were indicated by DAPI staining (blue); scale bar, 100 um. h Representative images and statistic results of PAS staining of goblet cells in the colon after POA treatment. n = 10 for each group. Scale bar, 100 um. Normal distribution was confirmed by Shapiro–Wilk test. Data were analyzed using one-way ANOVA with Tukey post hoc test (c , e), two tailed t test (d , f – h) and were presented as mean ± s.e.m. ns not significant, * $p < 0.05$, ** $p < 0.01$, and *** $p < 0.001$ as compared with the corresponding controls Full size imageIndex in PubMed under a CC BY license. PMID: 40494851



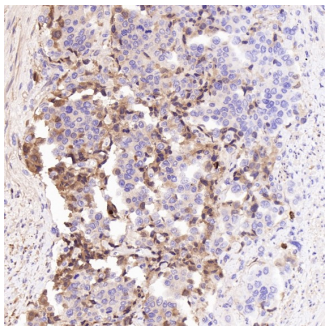
Immunohistochemical analysis of paraffin-embedded mouse kidney, using Lysozyme Antibody.

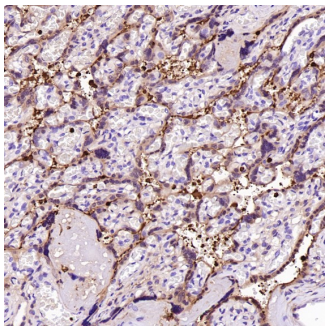


Immunohistochemical analysis of paraffin-embedded Rat cerebral cortex, using the Antibody at 1:2000 dilution.

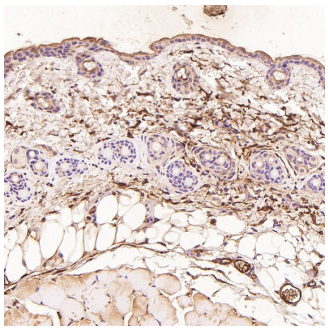
Immunohistochemical analysis of paraffin-embedded Human thyroid cancer, using the Antibody at 1:4000 dilution.

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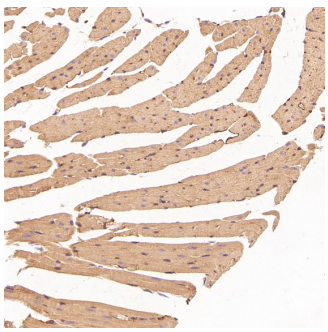




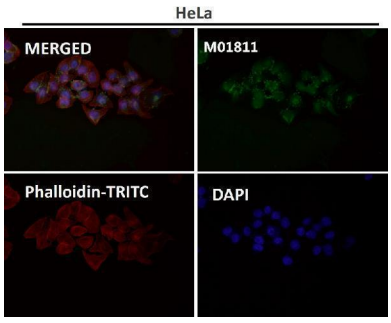
Immunohistochemical analysis of paraffin-embedded Human placenta, using the Antibody at 1:4000 dilution.



Immunohistochemical analysis of paraffin-embedded Mouse skin, using the Antibody at 1:2000 dilution.



Immunohistochemical analysis of paraffin-embedded Mouse heart, using the Antibody at 1:2000 dilution.



Immunofluorescent analysis using the Antibody at 1:50 dilution.

3 Publications Citing This Product

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1. PubMed ID: -, Cuiling Guo,Dandan Guo,Liu Fang,Tingting Sang,Jianjun Wu,Chengjie Guo,Yujie Wang,Ying Wang,Chaojie Chen,Jiajun Chen,Rong Chen,Xingya Wang,Ganoderma lucidum polysaccharide modulates gut microbiota and immune cell function to inhibit inflammation and tumorigenesis in colon,Carbohydrate Polymers,Volume 267,2021,118231,ISSN 0144-8617, https:// doi.org/10.1016/j.carbpol.2021.118231.

2. PubMed ID: 33483079, Sang T,Guo C,Guo D,Wu J,Wang Y,Wang Y,Chen J,Chen C,Wu K,Na K,Li K,Fang L,Guo C,Wang X. Suppression of obesity and inflammation by polysaccharide from sporoderm-broken spore of Ganoderma lucidum via gut microbiota regulation. Carbohydr Polym.2021 Mar 15;2

3. PubMed ID: 33483079, Sang T,Guo C,Guo D,Wu J,Wang Y,Wang Y,Chen J,Chen C,Wu K,Na K,Li K,Fang L,Guo C,Wang X. Suppression of obesity and inflammation by polysaccharide from sporoderm-broken spore of Ganoderma lucidum via gut microbiota regulation. Carbohydr Polym.2021 Mar 15;2

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