

# Product Datasheet

## MGST2 Antibody - BSA Free

### NBP1-82653

Unit Size: 0.1 ml

Store at 4C short term. Aliquot and store at -20C long term. Avoid freeze-thaw cycles.

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Updated 12/2/2025 v.20.1

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**NBP1-82653**

MGST2 Antibody - BSA Free

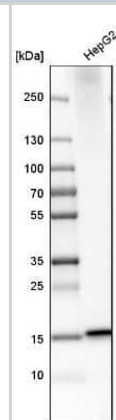
Product Information	
Unit Size	0.1 ml
Concentration	Concentrations vary lot to lot. See vial label for concentration. If unlisted please contact technical services.
Storage	Store at 4C short term. Aliquot and store at -20C long term. Avoid freeze-thaw cycles.
Clonality	Polyclonal
Preservative	0.02% Sodium Azide
Isotype	IgG
Purity	Affinity purified
Buffer	PBS (pH 7.2) and 40% Glycerol

Product Description	
Description	Novus Biologicals Rabbit MGST2 Antibody - BSA Free (NBP1-82653) is a polyclonal antibody validated for use in IHC, WB and ICC/IF. Anti-MGST2 Antibody: Cited in 1 publication. All Novus Biologicals antibodies are covered by our 100% guarantee.
Host	Rabbit
Gene ID	4258
Gene Symbol	MGST2
Species	Human
Immunogen	This antibody was developed against Recombinant Protein corresponding to amino acids: LKYKVTPPAVTGSPEFERVFRAQQNCVEFYPIF

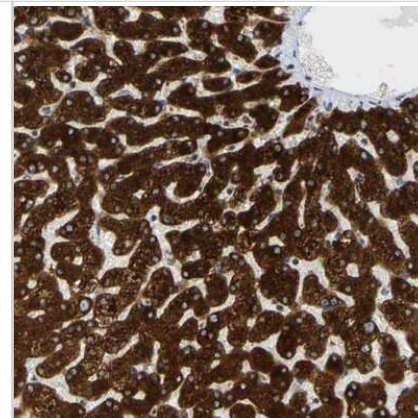
Product Application Details	
Applications	Western Blot, Immunohistochemistry-Paraffin, Immunohistochemistry
Recommended Dilutions	Western Blot 0.04-0.4 ug/ml, Immunohistochemistry 1:200 - 1:500, Immunohistochemistry-Paraffin 1:200 - 1:500
Application Notes	For IHC-Paraffin, HIER pH 6 retrieval is recommended.

**Images**

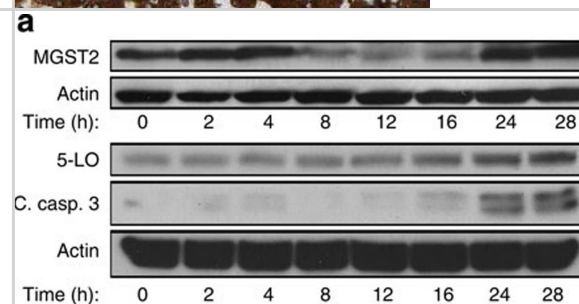
Western Blot: MGST2 Antibody [NBP1-82653] - Analysis in human cell line HepG2.



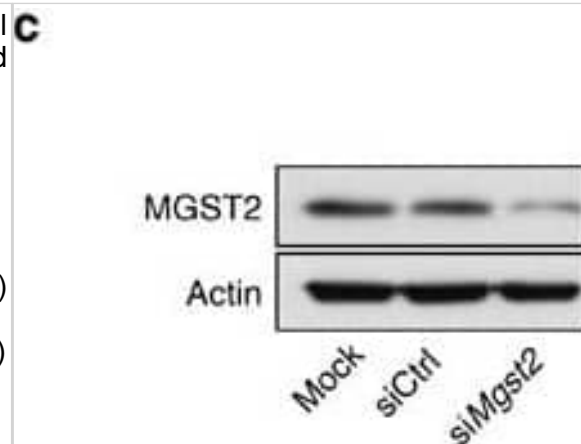
Immunohistochemistry-Paraffin: MGST2 Antibody [NBP1-82653] - Staining of human liver shows strong cytoplasmic positivity in hepatocytes.



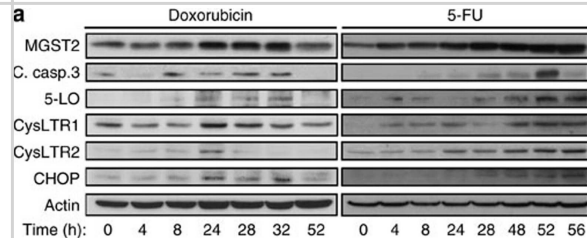
ER stress triggers expression and nuclear localization of proteins involved in LTC<sub>4</sub> biosynthesis. (a) Immunoblot of proteins expressed in WISH cells at different times after induction of ER stress with BfA. Blots are representatives of three replicates. (b–e) Immunostain of the indicated proteins following treatment of WISH cells with vehicle or BfA. Trans. is transmission light microscopy. Nuclei were counterstained with Hoechst 33258 (Hoechst). Shown are merges of 5-LO and MGST2; 5-LO, Hoechst and cPLA2; Hoechst, 5-LO and FLAP; and MGST2 and the ER marker protein disulfide isomerase (PDI). Bars, 5  $\mu$ m. (f) Quantification of per cent co-localization of FLAP and MGST2 with 5-LO, as determined by analysis of confocal microscopy images.  $n=6$ ,  $P<0.0001$  for both pairs. (g) Quantification of per cent nuclear localization of the indicated proteins as determined by analysis of confocal microscopy of the images shown in panels b–e.  $n\geq 6$ ,  $P<0.0001$  for all samples. Values in f and g represent the mean $\pm$ s.d. Statistical significance was determined using one-way analysis of variance (ANOVA). Image collected and cropped by CiteAb from the following open publication (<https://pubmed.ncbi.nlm.nih.gov/26656251>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



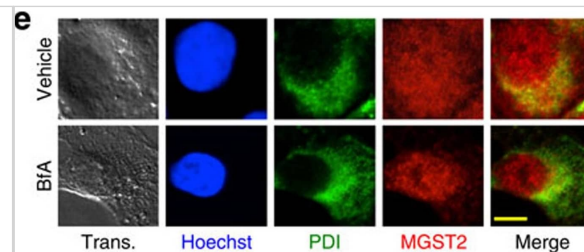
The MGST2-LTC4 pathway elicits ER stress-triggered cell death. Survival was determined by crystal violet staining and is relative to vehicle-treated cells. (a,b) Survival of WISH cells transfected with the indicated siRNA, treated with BfA (0.5  $\mu\text{g ml}^{-1}$ , 24 h). Bar, 100  $\mu\text{m}$ .  $n=3$ ,  $^{**}P<0.02$ . (c) Immunoblot of MGST2 in extracts of WISH cells treated as in a. (d,e) Survival of WISH cells treated with BfA and pranlukast. Bar, 200  $\mu\text{m}$ .  $n=3$ ,  $^{***}P<0.001$ . (f,g) Survival of human HaCaT pre-keratinocytes treated with BfA (1.3  $\mu\text{g ml}^{-1}$ , 48 h) and BAY u9773 (80 nM). Bar, 100  $\mu\text{m}$ .  $n=4$ ,  $^{***}P<0.0001$ . (h,i) Survival of WISH cells treated with BfA (48 h) and BAY cysLT2. Bar, 500  $\mu\text{m}$ .  $n=4$ ,  $^{***}P<0.001$ . (j,k) Survival of HaCaT pre-keratinocytes treated with the proteasome inhibitor MG262 (0.05  $\mu\text{M}$ ) and zileuton. Bar, 50  $\mu\text{m}$ .  $n=4$ ,  $^{***}P<0.001$ . (l,m) Survival of B16 cells treated with Tm, thapsigargin (Tg) or BfA (1.3  $\mu\text{g ml}^{-1}$ ) and the CysLTR1 antagonist MK571. Bar, 200  $\mu\text{m}$ .  $n=4$ ,  $^{***}P<0.001$ . (n) Immunoblot of the necrosis marker HMGB1 (top panel) in media of B16 cells treated with BfA (1.3  $\mu\text{g ml}^{-1}$ ) and MK571 (MK). Ponceau S staining served as loading control. (o) Immunoblot of cleaved caspase-3 in extracts of WISH cells treated with Tm (2  $\mu\text{g ml}^{-1}$ , 48 h) and the indicated inhibitors. (p,q) Survival of B16 cells treated with LTC4. Bar, 200  $\mu\text{m}$ .  $n=3$ ,  $^{***}P<0.001$ . (r) Survival of B16 cells treated with LTC4 or LTD4. Bar, 200  $\mu\text{m}$ . (s,t) Survival of HEK 293T cells stably transfected with Tet-inducible Mgst2 expression vector, treated with doxycycline (2  $\mu\text{g ml}^{-1}$ , 48 h). Bar, 200  $\mu\text{m}$ .  $n=3$ ,  $^{***}P<0.001$ . Immunoblots c,n and o are representatives of three replicates. Values in b,e,g,i,k,m,q and t represent the mean $\pm$ s.d. Statistical significance was determined using one-way ANOVA. Image collected and cropped by CiteAb from the following open publication (<https://pubmed.ncbi.nlm.nih.gov/26656251>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



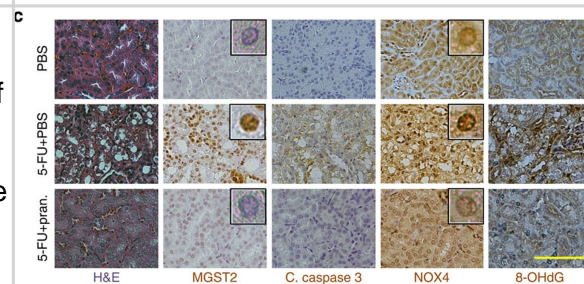
Chemotherapy-activated MGST2-LTC4 pathway triggers NOX4-mediated oxidative DNA damage and cell death. (a) Immunoblot of the indicated proteins in extracts of WISH cells treated with doxorubicin (5  $\mu\text{M}$ ) or 5-FU (20  $\mu\text{g ml}^{-1}$ ) for the indicated times. (b) Immunostain of 5-LO and MGST2 in WISH cells treated with vehicle or doxorubicin (Doxo, 5  $\mu\text{M}$ ). Nuclei were counterstained with Hoechst. Trans. is transmission light microscopy. All image channels except the transmission light microscopy were merged. Bar, 5  $\mu\text{m}$ . (c,d) Immunostain of LTC4 in WISH cells pre-treated with vehicle, siControl or siMgst2, followed by vehicle or doxorubicin (1  $\mu\text{M}$ , 36 h). Nuclei were counterstained with Hoechst. Bar, 20  $\mu\text{m}$ .  $n=3$ ,  $^{****}P<0.0001$ ,  $^{***}P<0.001$ . (e,f) Immunostain of NOX4 in cells treated with vehicle or doxorubicin. Bar, 20  $\mu\text{m}$ .  $n=3$ ,  $^{***}P<0.001$ . (g,h) ROS detection with DCFH-DA in cells treated with vehicle or doxorubicin (2.5  $\mu\text{M}$ , 48 h) in the absence or presence of the indicated inhibitors. Bar, 50  $\mu\text{m}$ .  $n=6$ ,  $^{***}P<0.0001$ . (i,j) Immunostain of the dsDNA break marker  $\gamma$ -H2AX in cells treated with doxorubicin (5  $\mu\text{M}$ , 48 h) in the absence or presence of the indicated LTC4 receptor antagonists. Bar, 20  $\mu\text{m}$ .  $n=3-4$ ,  $^{***}P<0.0001$ . (k,l) Survival of primary WT and Mgst2-deficient MEFs, treated with vehicle or doxorubicin (10  $\mu\text{M}$ ) and then stained with crystal violet. Bar, 50  $\mu\text{m}$ . Viabilities of vehicle-treated WT and KO MEFs were taken as 100% survival, respectively.  $n=3$ ,  $^{***}P<0.005$ . Images a and b are representatives of three replicates. Values in d,f,h,j and l represent the mean $\pm$ s.d. Statistical significance was determined using one-way ANOVA. Image collected and cropped by CiteAb from the following open publication (<https://pubmed.ncbi.nlm.nih.gov/26656251>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



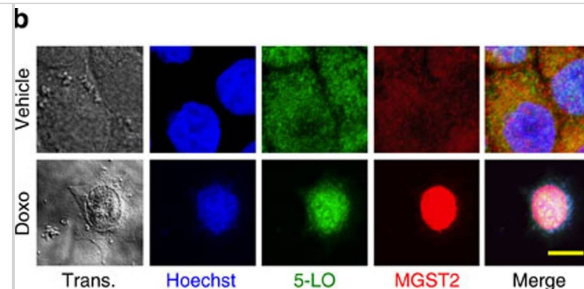
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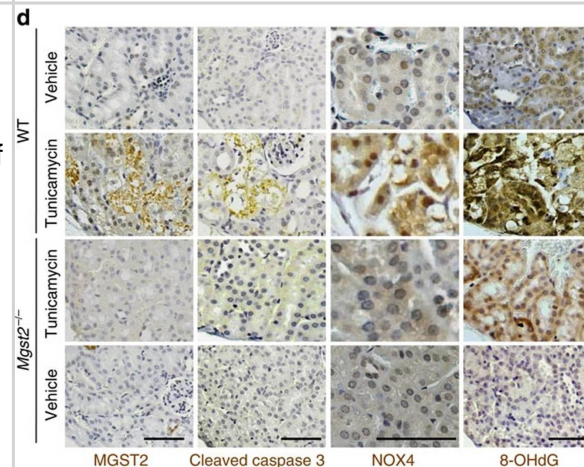
Mgst2 deficiency and LTC<sub>4</sub> inhibition attenuate 5-FU-triggered DNA damage and toxicity. (a) Survival of WT and Mgst2-deficient mice (10 per group) given 5-FU (300 mg kg<sup>-1</sup>, ip) at time=0.  $P=0.0085$ . (b) Survival of WT 129/Sv mice (18 per group) treated with 5-FU as in a. Vehicle or pranlukast (1 mg kg<sup>-1</sup>) were administered at days 0, 1, 2, 5, 6 and 7.  $P=0.032$ . The statistical significance was determined in a and b using the Gehan–Breslow–Wilcoxon test. (c) Haematoxylin–eosin (H&E) stain and immunostain of the indicated proteins and 8-OHdG in kidney slices of WT mice treated with 5-FU (300 mg kg<sup>-1</sup>, ip at time=0) followed by five administrations of PBS or pranlukast (Pran., 3 mg kg<sup>-1</sup>, ip) as in b. Kidneys were processed at day 13. Bar, 50  $\mu$ m. Insets: enlarged images showing immunostained nuclei. The images of kidney slices are representatives of slices from three mice per group. (d) Survival of U266 myeloma cells treated with bortezomib alone or together with the indicated LTC<sub>4</sub> inhibitors (10  $\mu$ M each).  $n=3$ . (e) Survival of CCRF-CEM T-cell leukaemia lymphoblasts treated with doxorubicin together with the indicated LTC<sub>4</sub> inhibitors (10  $\mu$ M each).  $n=3$ . No significant differences were found between vehicle and any of the inhibitors in d and e. Statistical significance was determined using one-way ANOVA. Image collected and cropped by CiteAb from the following open publication (<https://pubmed.ncbi.nlm.nih.gov/26656251>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



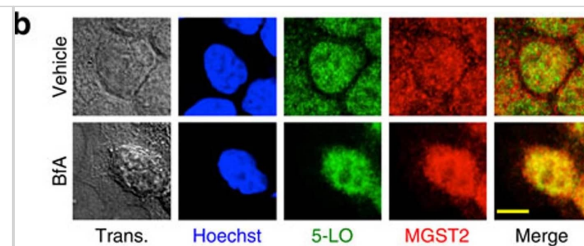
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Mgst2 deficiency and LTC4 inhibition attenuate ER stress-triggered oxidative damage to mouse kidneys and mouse morbidity. (a) Haematoxylin–eosin stained kidney slices from WT and Mgst2-deficient mice given a single dose of Tm (1.5 mg kg<sup>-1</sup>, ip) at time=0. Kidneys were removed and processed on day 4. The image is a representative of 10 kidneys obtained from 5 mice per group. Bar, 200  $\mu$ m. (b) Quantification of per cent areas of vacuoles representing damage to kidney proximal tubules shown in a. n=5, \*\*\*P<0.001. Values represent the mean $\pm$ s.d. Statistical significance was determined using one-way ANOVA. No vacuoles were observed in kidneys of untreated mice. (c) Immunohistochemical stains of proximal tubules (brown) using anti-aminopeptidase A in kidney sections from WT and Mgst2-deficient mice treated with Tm as in a. Nuclei were counterstained with haematoxylin (grey-blue). Bar, 50  $\mu$ m. (d) Immunohistochemical stains of the indicated markers in kidney sections as in c. Figures are representatives of kidneys from three mice. Bars, 50  $\mu$ m. (e) Survival of WT and Mgst2-deficient 129/Sv mice (20 per group) given Tm (2.5 mg kg<sup>-1</sup>, ip) at time=0. P=0.0393. (f) Survival of WT 129/Sv mice (10 per group) given Tm (1.5 mg kg<sup>-1</sup>, ip) at time=0 and daily administrations of either vehicle or pranlukast (ip, 1 mg kg<sup>-1</sup>, vertical arrows). P=0.0009. The statistical significance was determined in e and f using the Gehan–Breslow–Wilcoxon test. Image collected and cropped by CiteAb from the following open publication (<https://pubmed.ncbi.nlm.nih.gov/26656251>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



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## Publications

Efrat Dvash, Michal Har-Tal, Sara Barak et al. Leukotriene C<sub>4</sub> is the major trigger of stress-induced oxidative DNA damage. *Nature Communications* 2015-01-01 [PMID: 26656251] (WB, IF/IHC, ICC/IF, Mouse)



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General: novus@novusbio.com

### **Products Related to NBP1-82653**

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NBP1-82653PEP	MGST2 Recombinant Protein Antigen
NBP2-33376H	Blue Marker Antibody (6F4-F6) [HRP]
HAF008	Goat anti-Rabbit IgG Secondary Antibody [HRP]
NB7160	Goat anti-Rabbit IgG (H+L) Secondary Antibody [HRP]
NBP2-24891	Rabbit IgG Isotype Control

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### **Limitations**

This product is for research use only and is not approved for use in humans or in clinical diagnosis. Primary Antibodies are guaranteed for 1 year from date of receipt.

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