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DATA SHEET

Mouse Anti-DDIT3/CHOP/GADD153 [H5]: MC0462, MC0462RTU7

Intended Use: For Research Use Only

Description: DDIT3 (DNA-damage-inducible transcript 3, CHOP, GADD153) is a member of the CCAAT/enhancer-binding protein (C/EBP) family of transcription factors. It is a small nuclear protein that is capable of forming heterodimers with other C/EBP members such as C/EBP and LAP (liver activator protein), and preventing their DNA binding and function of C/EBP to classical binding sites. Inversely, the C/EBP GADD153 heterodimer gains binding activity to other non classical C/EBP stress related targets. Under normal cellular conditions this protein is not expressed in detectable levels, but is highly unregulated during times of cellular/ER stress. Examples of DDIT3 inducing stress include: treatment with tunicamycin, nutrient starvation and reducing agents that interfere with the calcium flux across the ER membrane. The protein is implicated in adipogenesis and erythropoiesis, is activated by endoplasmic reticulum stress, and promotes apoptosis.

Specifications

Clone:	H5
Source:	Mouse
Isotype:	IgG3k
Reactivity:	Human, mouse, rat
Immunogen:	Peptide mapping at the C-terminus of human DDIT3
Localization:	Nucleus or cytoplasm
Formulation:	Antibody in PBS pH7.4, containing BSA and ≤ 0.09% sodium azide (NaN3)
Storage:	Store at 2°- 8°C
Applications:	IHC, ELISA, IF, IP, WB

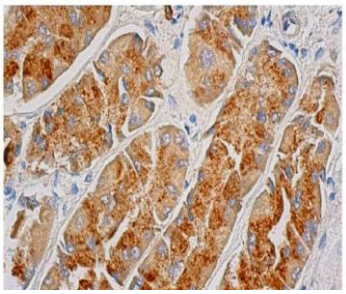
Package:

Description	Catalog No.	Size
DDIT3/CHOP/GADD153 Concentrated	MC0462	1 ml
DDIT3/CHOP/GADD153 Prediluted	MC0462RTU7	7 ml

IHC Procedure*

Positive Control Tissue:	Thyroid gland, testis, kidney, upper stomach
Concentrated Dilution:	50-200
Pretreatment:	Tris EDTA pH9.0, 15 minutes Pressure Cooker or 30-60 minutes water bath at 95°-99°C
Incubation Time and Temp:	30-60 minutes @ RT
Detection:	Refer to the detection system manual

* Result should be confirmed by an established diagnostic procedure.



FFPE human upper stomach stained with anti-DDIT3 using DAB showing cytoplasmic staining of glandular cells

References

1. Valdecoxib improves lipid-induced skeletal muscle insulin resistance via simultaneous suppression of inflammation and endoplasmic reticulum stress. Tae Jin Kim, et al. Biochem Pharmacol. Jun;188:114557, 2021.
2. New Cardiomyokine Reduces Myocardial Ischemia/Reperfusion Injury by PI3K-AKT Pathway Via a Putative KDEL-Receptor Binding. Leonardo Maciel, et al. J Am Heart Assoc. Jan 5;10(1):e019685, 2021.
3. A conserved N-terminal motif is required for complex formation between FUS, EWSR1, TAF15 and their oncogenic fusion proteins. Christer Thomsen, et al. FASEB J. Dec;27(12):4965-74, 2013. doi: 10.1096/fj.13-234435.

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