funakoshi
FRONTIERS IN LIFE SCIENCE

Research Use Only

JAPAN MADE QUALITY

For Amyotrophic Lateral Sclerosis (ALS) research: SOD1 mutant specific antibody

Anti-SOD1 (ALS-related mutants) Clone : MS785, MS27

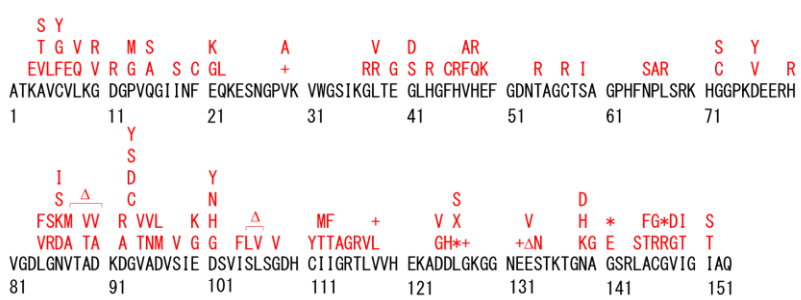
For more information : http://www.funakoshi.co.jp/exports_contents/81146

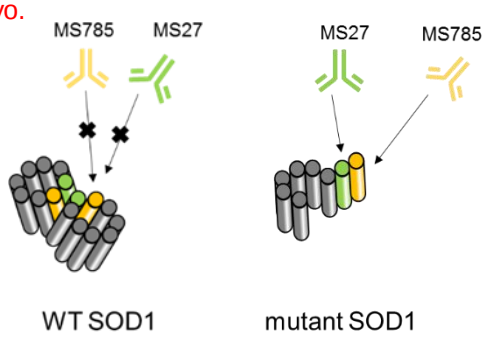
Our Anti-SOD1 Rat monoclonal antibodies (Clone : MS785 and MS27) specifically detects SOD1 mutant.

SOD1 mutant and ALS

Amyotrophic Lateral Sclerosis (ALS) is a fatal adult-onset neurodegenerative disease. According to many research results, SOD1 (Cu/Zn superoxide dismutase) is one of the major causative genes of ALS and SOD1 mutant proteins cause (evoke, lead) ALS through a gain of toxic function.
Wild type SOD1 forms homo dimer. However, it was reported that mutant SOD1 forms the different conformation, having toxic function by binding to Derlin-1, an ER-associated degradation (ERAD) machinery protein. Prof. Hidenori Ichijo and his colleagues developed two novel rat monoclonal anti-SOD1 antibodies, clone MS785 and MS27, which specifically bind to conformationally altered SOD1, not detecting wild type SOD1 homo-dimer.
Both clones are succeeded in detecting over 100 SOD1 mutants (Ref.2), please visit our website for further information about the list of detectable SOD1 mutants.

This product has been commercialized under the license of the University of Tokyo.





Features and Antibody Information

・Specific to SOD1 mutant (monomer type)

Clone	MS785	MS27
Host	Rat (Monoclonal)	
Subclass	IgG2b/κ	IgG2a/κ
Reactivity	Human	
Purification	Protein G	
Format	50% Glycerol/PBS	

Application

IP, IC and ELISA
Note : MS785 and MS27 detect both wild and mutant type of SOD1 in denatured condition.

Reference

1. Fujisawa *et al., Ann. Neurol.*, **72**, 739 ～749 (2012)
2. Fujisawa *et al., Neurobiol. Dis.*, **82**, 478 ～ 486 (2015)

Product Information

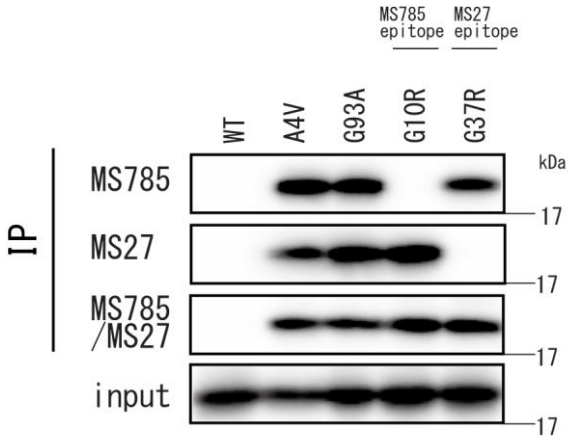
[Manufacturer : FNA]

Product Name	Catalog #	Size	Storage
Anti-SOD1(ALS-related mutants) Cocktail, Human, Rat-Mono(MS785/MS27)	FDV-0021A	100 µg	-20 ℃
Anti-SOD1(ALS-related mutants), Human, Rat-Mono(MS785)	FDV-0021B		
Anti-SOD1(ALS-related mutants), Human, Rat-Mono(MS27)	FDV-0021C		

Example Data

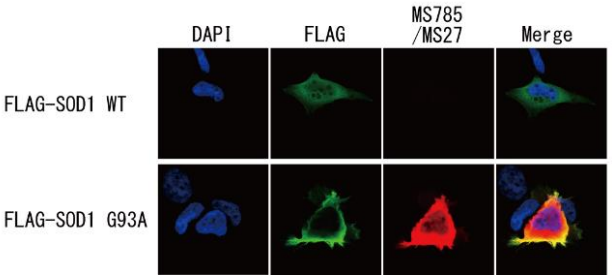
Detection of SOD1 mutants by immunoprecipitation assay

SOD1 wild type or mutants-expressing HEK293 cells were lysed by 1% Triton X100/TBS buffer. After lysis, add MS785 (5 µg), MS27 (2 µg) or cocktail (1 µg) and incubate for 12 hours. Subsequently, SOD1-antibody complexes were captured by Protein G-beads. Neither MS785 nor MS27 single detected some specific mutants which have the mutation on the antibody's epitope. MS785/MS27 cocktail overcame this problem.



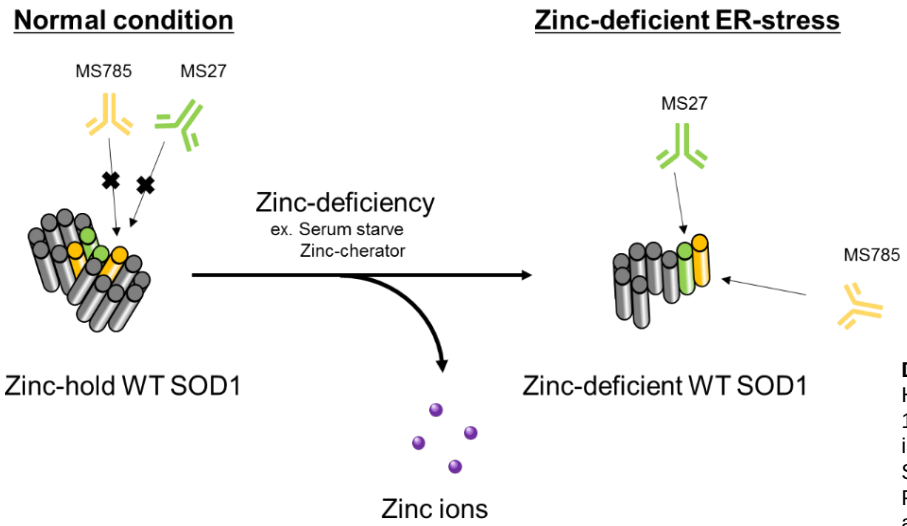
Detection of SOD1 mutants by immunocytochemistry

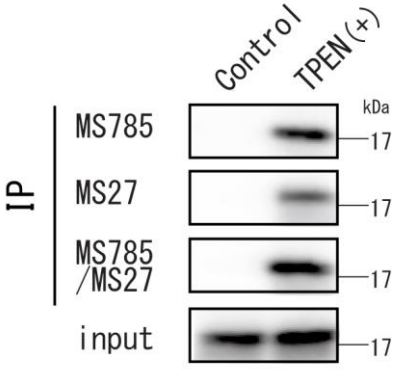
Flag-tagged SOD1 wild type (WT) or G93A mutant-expressing HEK293 cells were fixed and incubated with primary antibodies (1 µg/ml MS785/MS27).
Although anti-FLAG antibody clearly visualized either WT and G93A mutant, MS785/MS27 cocktail only detected G93A mutant.



SOD1 and ER stress

ALS is also considered as an ER stress disease. Zinc-deficiency is one of the causes of ER stress and depletion of zinc from SOD1 induces drastic conformational change.
Under the zinc-deficient condition, endogenous wild-type SOD1 has a conformation similar to ALS-related mutants. In fact, both MS785 and MS27 could specifically recognize wild-type SOD1 under the zinc-deficiency.
Clone MS785 and MS27 are also powerful tools to investigate zinc-related ER-stress research as well.





Detection of endogenous SOD1 WT under ER-stress
HEK293 cells were cultured in the presence and absence of 10 µM TPEN After treatment, cells were lysed and immunoprecipitated with 1-5 ug of MS785, MS27 or cocktail. Subsequently, SOD1-antibody complexes were captured by Protein G-beads. Under the zinc-deficient ER-stress, these antibodies could recognize endogenous SOD1 wild type with mutant like conformation.

NOTE

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