BOSTER
Antibody and ELISA Experts

BOSTER BIOLOGICAL TECHNOLOGY Co.,Ltd.
3942 B Valley Ave, Pleasanton, CA, 94566

Phone: 888-466-3604 Fax: 925-215-2184 Email: boster@bosterbio.com Web: www.bosterbio.com

Monoclonal Anti-Smad4 Antibody

Catalog Number: MA1089

Description

Lot No.	08A12
Clone	DCS-46
Size	100μg/vial
Form	lyophilized
Ig type	mouse IgG1
Specificity	No cross reactivity with other proteins.
Species	Human
Immunogen	Recombinant human Smad4 (DPC4).
Contents	Mouse ascites fluid, 1.2% sodium acetate, 2mg BSA, with 0.01mg NaN_3 as preservative.

Application

	Concentration	Tested Species	Antigen Retrieval
Western blot	2-4μg/ml	Human	-
Immunocytochemistry	1μg/ml	Human	-

Other applications have not been tested.

Optimal dilutions should be determined by end users.

Preparation and storage

Reconstitution: 1.2% sodium acetate or neutral PBS. If 1ml of PBS is used, the antibody concentration will be 100μg/ml.

Storage: At -20˚C for one year. After reconstitution, at 4˚C for one month. It can also be aliquotted and stored frozen at -20˚C for a longer time.
Avoid repeated freezing and thawing.

Relevant detection systems

Boster provides a series of assays reacted with primary antibodies. Antibody can be supported by chemiluminescence kit EK1001 in WB, supported by SA1021 in ICC.

Background

SMAD4 plays a pivotal role in signal transduction of the transforming growth factor beta superfamily cytokines by mediating transcriptional activation of target genes. Smad4 signalling in T cells is required for suppression of gastrointestinal cancer. Mutational inactivation of SMAD4 causes TGF-beta unresponsiveness and gave a basis for understanding the physiologic role of this gene in tumorigenesis. Mutations in DPC4 (SMAD4) cause juvenile polyposis syndrome, but only account for a minority of cases.

Reference

1. Kim, B.-G.; Li, C.; Qiao, W.; Mamura, M.; Kasprzak, B.; Anver, M.; Wolfraim, L.; Hong, S.; Mushinski, E.; Potter, M.; Kim, S.-J.; Fu, X.-Y.; Deng, C.; Letterio, J. J. : Smad4 signalling in T cells is required for suppression of gastrointestinal cancer. *Nature* 441: 1015-1019, 2006. Note: Erratum: Nature 444: 780 only, 2006.
2. Houlston, R.; Bevan, S.; Williams, A.; Young, J.; Dunlop, M.; Rozen, P.; Eng, C.; Markie, D.; Woodford-Richens, K.; Rodriguez-Bigas, M. A.; Leggett, B.; Neale, K.; Phillips, R.; Sheridan, E.; Hodgson, S.; Iwama, T.; Eccles, D.; Bodmer, W.; Tomlinson, I. : Mutations in DPC4 (SMAD4) cause juvenile polyposis syndrome, but only account for a minority of cases. *Hum. Molec. Genet.* 7: 1907-1912, 1998.
3. Howe, J. R.; Roth, S.; Ringold, J. C.; Summers, R. W.; Jarvinen, H. J.; Sistonen, P.; Tomlinson, I. P. M.; Houlston, R. S.; Bevan, S.; Mitros, F. A.; Stone, E. M.; Aaltonen, L. A. : Mutations in the SMAD4/DPC4 gene in juvenile polyposis. *Science* 280: 1086-1088, 1998.