

FAAH (27-Y): sc-100739

BACKGROUND

FAAH (fatty acid amide hydrolase) is a membrane-bound enzyme fatty acid amide hydrolase, responsible for the hydrolysis of multiple primary and secondary fatty acid amides, including the neuromodulatory compounds anandamide and oleamide. The degradation of anandamide to arachidonic acid and oleamide to oleic acid, terminates the signaling function of these molecules. FAAH degrades amides and esters with equivalent catalytic efficiency, enabling FAAH to function effectively as both an amidase and esterase. FAAH contributes to anandamide uptake by creating and maintaining an inward concentration gradient for anandamide. A natural single nucleotide polymorphism mutation in human FAAH in its homozygous form is strongly associated with problem drug use. This results in a missense mutation (385C→A) that converts a conserved proline residue to threonine (Pro129→Thr), producing an FAAH variant that displays normal catalytic properties but enhanced sensitivity to proteolytic degradation. Genetic mutations in FAAH constitute an important risk factor for problem drug use. The human FAAH gene maps to chromosome 1p33.

REFERENCES

- Cravatt, B.F., et al. 1996. Molecular characterization of an enzyme that degrades neuromodulatory fatty acid amides. *Nature* 384: 83-87.
- Giang, D.K. and Cravatt, B.F. 1997. Molecular characterization of human and mouse fatty acid amide hydrolases. *Proc. Natl. Acad. Sci. USA* 94: 2238-2242.

CHROMOSOMAL LOCATION

Genetic locus: FAAH (human) mapping to 1p33; Faah (mouse) mapping to 4 D1.

SOURCE

FAAH (27-Y) is a mouse monoclonal antibody raised against a C-terminus region of FAAH of human origin.

PRODUCT

Each vial contains 100 µg IgG_{2a} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

FAAH (27-Y) is recommended for detection of FAAH of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)] and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for FAAH siRNA (h): sc-106807, FAAH siRNA (m): sc-145000, FAAH shRNA Plasmid (h): sc-106807-SH, FAAH shRNA Plasmid (m): sc-145000-SH, FAAH shRNA (h) Lentiviral Particles: sc-106807-V and FAAH shRNA (m) Lentiviral Particles: sc-145000-V.

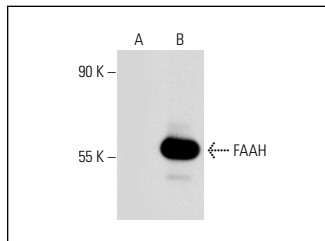
Molecular Weight of FAAH: 67 kDa.

Positive Controls: FAAH (h): 293T Lysate: sc-112472, A-431 whole cell lysate: sc-2201 or c4 whole cell lysate: sc-364186.

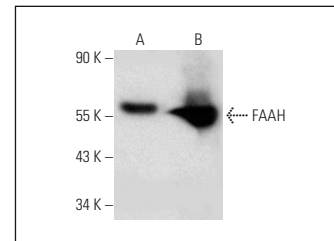
STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

DATA



FAAH (27-Y): sc-100739. Western blot analysis of FAAH expression in non-transfected: sc-117752 (A) and human FAAH transfected: sc-112472 (B) 293T whole cell lysates.



FAAH (27-Y): sc-100739. Western blot analysis of FAAH expression in A-431 (A) and c4 (B) whole cell lysates.

SELECT PRODUCT CITATIONS

- Smaga, I., et al. 2017. Changes in the brain endocannabinoid system in rat models of depression. *Neurotox. Res.* 31: 421-435.
- Martínez-León, E., et al. 2018. Fibronectin modulates the endocannabinoid system through the cAMP/PKA pathway during human sperm capacitation. *Mol. Reprod. Dev.* 86: 224-238.
- Maia, J., et al. 2019. Effects of cannabis tetrahydrocannabinol on endocannabinoid homeostasis in human placenta. *Arch. Toxicol.* 93: 649-658.
- Amini, M., et al. 2020. Involvement of endocannabinoid system, inflammation and apoptosis in diabetes induced liver injury: role of 5-HT3 receptor antagonist. *Int. Immunopharmacol.* 79: 106158.
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- Tiwari, S., et al. 2020. Gender-specific changes in energy metabolism and protein degradation as major pathways affected in livers of mice treated with ibuprofen. *Sci. Rep.* 10: 3386.
- Wang, D.C., et al. 2020. Recovery of BDNF and CB1R in the prefrontal cortex underlying improvement of working memory in prenatal DEHP-exposed male rats after aerobic exercise. *Int. J. Mol. Sci.* 21: 3867.
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- Metz, V.G., et al. 2022. Cannabidiol treatment prevents drug reinstatement and the molecular alterations evoked by amphetamine on receptors and enzymes from dopaminergic and endocannabinoid systems in rats. *Pharmacol. Biochem. Behav.* 218: 173427.

RESEARCH USE

For research use only, not for use in diagnostic procedures.