β-Amyloid (1-40), FAM-labeled TFA

Cat. No.:	HY-P2550A
Molecular Formula:	$C_{217}H_{306}N_{53}F_3O_{66}S$
Molecular Weight:	4802.22
Sequence:	FAM-Asp-Ala-Glu-Phe-Arg-His-Asp-Ser-Gly-Tyr-Glu-Val-His-His-Gln-Lys-Leu-Val-Phe-Phe-Ala-Glu-Asp-Val-Gly-Ser-Asn-Lys-Gly-Ala-Ile-Ile-Gly-Leu-Met-Val-Gly-Gly-Val-Val
Sequence Shortening:	FAM-DAEFRHDSGYEVHHQKLVFFAEDVGSNKGAIIGLMVGGVV
Target:	Amyloid-β
Pathway:	Neuronal Signaling
Storage:	Sealed storage, away from moisture and light, under nitrogen Powder -80°C 2 years -20°C 1 year * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light, under nitrogen)

BIOLOGICAL ACTIVITY

Description	β-Amyloid (1-40), FAM-labeled TFA is a FAM fluorescently-labelled β-Amyloid (1-40) peptide (λex= 492 nm and λem= 518 nm).
In Vitro	Apical-to-basolateral exchange across endothelial monolayer of fluorescent Aβ42 (FAM-labeled Human β-Amyloid (1-42) and Fluor 488-labeled β-Amyloid (1-42)) or scramble Aβ42 (FAM-labeled scrambled β-Amyloid (1-42)) (1-100 μM) is monitored in transendothelial electrical resistance (TEER) during cell monolayer's formation over time (over 120 min in presence or absence of Aβ24). Human β-Amyloid (1-24) (1 μM) results in H-Aβ42 retention, thus reducing its efflux through the BBB and therefore preventing an efficient mechanism of Aβ42 clearance[1]. The in vitro BBB model is used to investigate the passage of FAM-labeled or 488-conjugated H-Aβ42 across the endothelial cell monolayer. The addition of fluorescently labeled H-Aβ42 (FAM-labeled Human β-Amyloid (1-42) or Fluor 488-labeled β-Amyloid (1-42)) to the apical side of cell inserts results in effective BBB crossing, FAM-labeled scrambled β-Amyloid (1-42) is not efficiently transcytosed. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. L M Jungbauer, et al. Preparation of fluorescently-labeled amyloid-beta peptide assemblies: the effect of fluorophore conjugation on structure and function. J Mol Recognit. Sep-Oct 2009;22(5):403-13.

[2]. Sonia Mazzitelli, et al. Amyloid-β 1-24 C-terminal truncated fragment promotes amyloid-β 1-42 aggregate formation in the healthy brain. Acta Neuropathol Commun. 2016 Oct 10;4(1):110.

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