

Product Information



BACE (human recombinant)

Item No. 10227 • Batch No. XXXX

Synonyms: ASP-2, BACE-1, Memapsin-2, Membrane-bound Aspartic Protease, β -Secretase
Source: Recombinant C-terminal His-tag protein purified from *E. coli*
M_r: ~48 kDa
Purity: $\geq 95\%$
Stability: ≥ 6 months at -80°C
Supplied in: 100 mM sodium borate, pH 8.5, containing 200 mM sodium chloride
Protein Concentration: *batch specific* mg/ml
Activity: *batch specific* U/ml
Specific Activity: *batch specific* nmoles/min/mg. One unit is defined as the amount of enzyme required to produce 1 nmole of EDANS per minute at 25°C in 50 mM sodium acetate, pH 4.5, containing 10 μM fluorescent peptide H-RE(EDANS)EVNLDAEFK (Dabcyl)R-OH

Accumulation of the β -amyloid peptide ($\text{A}\beta$) in the brain is implicated as the primary cause of neurodegeneration and progression of Alzheimer's disease (AD).¹ The β -amyloid peptide is derived from sequential proteolytic cleavage of the amyloid precursor protein (APP) by β - and γ -secretases. Initial cleavage by β -secretase (BACE), a membrane-anchored aspartic protease, generates a soluble N-terminal fragment and a membrane-associated C-terminal fragment.² The C-terminal fragment then undergoes proteolysis by γ -secretase to give the $\text{A}\beta$ peptide.

BACE has been shown to be the major β -secretase and a promising target as it initiates the first step in $\text{A}\beta$ production.¹ Inhibition of BACE activity could potentially block the entire cascade of AD pathogenesis. In addition, BACE deficient mice do not generate $\text{A}\beta$ peptide. In transgenic murine models of AD driven by $\text{A}\beta$ overproduction, BACE deficiency rescued memory deficits and cholinergic dysfunction.³ Additionally, the fact that β -secretase is an aspartic protease has also raised the hope that its therapeutic inhibitor can be as successful as that against HIV protease.

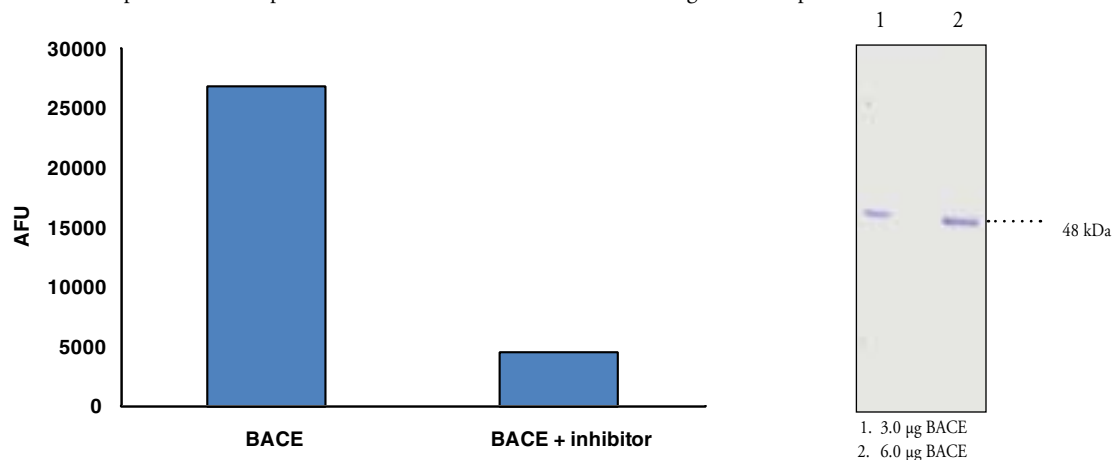


Figure 1: Inhibition of BACE activity

Figure 2: Coomassie stained SDS-PAGE of purified BACE

References

1. Hong, L., Turner, R.T.I., Koelsch, G., *et al.* *Biochem. Soc. Trans.* **30**(4), 530-534 (2002).
2. Grüninger-Leitch, E., Schlatter, D., Küng, E., *et al.* *J. Biol. Chem.* **277**(7), 4687-4693 (2002).
3. Pietrak, B.L., Crouthamel, M.-C., Tugusheva, K., *et al.* *Anal. Biochem.* **342**, 144-151 (2005).

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