APA485Mu61 100μg
Active N-Terminal Pro-Brain Natriuretic Peptide (NT-ProBNP)
Organism Species: *Mus musculus (Mouse)*
Instruction manual

FOR RESEARCH USE ONLY
NOT FOR USE IN CLINICAL DIAGNOSTIC PROCEDURES

13th Edition (Revised in Aug, 2023)

[PROPERTIES]

Source: Eukaryotic expression.
Host: 293F cell
Residues: Tyr27~Arg76
Tags: N-terminal His-tag
Purity: >90%
Endotoxin Level: <1.0EU per 1μg (determined by the LAL method).
Buffer Formulation: PBS, pH7.4, containing 5% Trehalose .
Original Concentration: 50μg/mL
Applications: Activity Assays.
(May be suitable for use in other assays to be determined by the end user.)
Predicted isoelectric point: 9.6
Predicted Molecular Mass: 7.5kDa
Accurate Molecular Mass: 8kDa as determined by SDS-PAGE reducing conditions.

[USAGE]

Reconstitute in ddH_2O to a concentration of 0.1-0.2 mg/mL. Do not vortex.

[STORAGE AND STABILITY]

Storage: Avoid repeated freeze/thaw cycles.
Store at 2-8°C for one month.
Aliquot and store at -80°C for 12 months.
Stability Test: The thermal stability is described by the loss rate. The loss rate was determined by accelerated thermal degradation test, that is, incubate the protein at 37°C for 48h, and no obvious degradation and precipitation were

observed. The loss rate is less than 5% within the expiration date under appropriate storage condition.

[SEQUENCE]

YPLGSPSQSPEQFKMQKLLELIREKSEEMAQRQLLKDQGLTKEHPKRVLR

[ACTIVITY]

N-Terminal Pro Brain Natriuretic Peptide (NT-ProBNP) is a cleavage fragment of proBNP predominantly secreted by ventricular cardiomyocytes upon myocardial stretch, ischemia, or volume overload. Synthesized as a 108-amino acid precursor, proBNP splits into bioactive BNP and stable NT-ProBNP during myocardial secretion. Circulating NT-ProBNP clearance relies on renal filtration without receptor-mediated degradation, rendering it a superior cardiac biomarker with longer half-life and stable plasma concentration. It sensitively reflects ventricular wall stress, widely applied for diagnosing, stratifying risk and monitoring heart failure, myocardial infarction, and pulmonary hypertension. Elevated levels correlate strongly with cardiac dysfunction, adverse cardiovascular outcomes, and all-cause mortality, serving as a core clinical indicator for cardiac load assessment. NT-ProBNP interacts with ANGPT2 (ANG2) to jointly exacerbate myocardial microvascular injury and ventricular remodeling under cardiac stress.Briefly, ANG2 was diluted serially in PBS with 0.01% BSA (pH 7.4). Duplicate samples of 100 μL were then transferred to NT-ProBNP-coated microtiter wells and incubated for 1h at 37℃. Wells were washed with PBST and incubated for 1h with anti-ANG2 pAb, then aspirated and washed 3 times. After incubation with HRP labelled secondary antibody for 1h at 37℃, wells were aspirated and washed 5 times. With the addition of substrate solution, wells were incubated 15-25 minutes at 37℃. Finally, add 50 μL stop solution to the wells and read at 450/630nm immediately. Measured by its binding ability in a functional ELISA. When recombinant mouse NT-ProBNP is immobilized at 2 μg/mL(100 μL/well), the concentration of ANG2

that produces 50% optimal binding-response is found to be approximately 0.256 μg/mL.

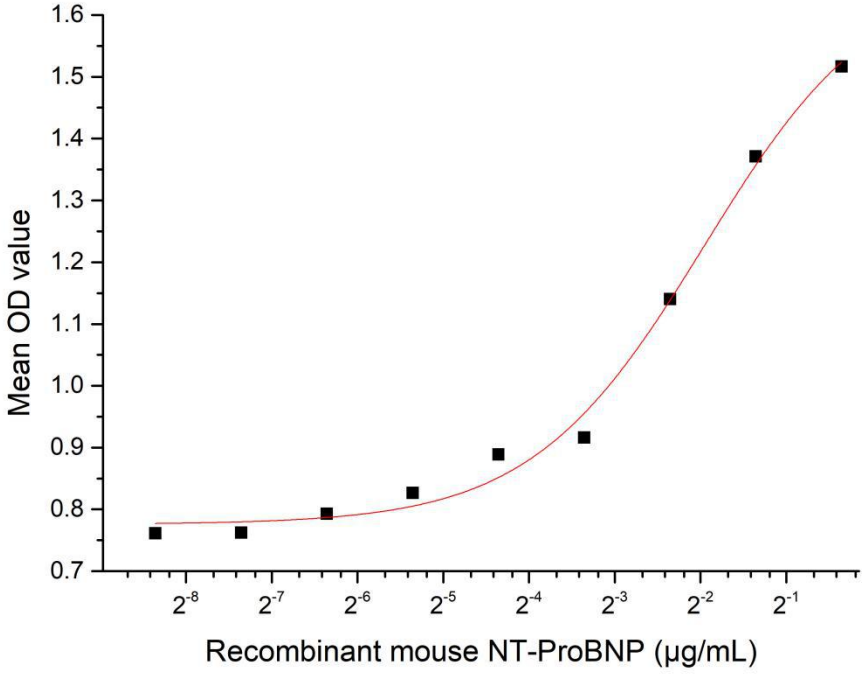

Figure 1. The binding activity of recombinant mouse NT-ProBNP and human ANG2

[IDENTIFICATION]

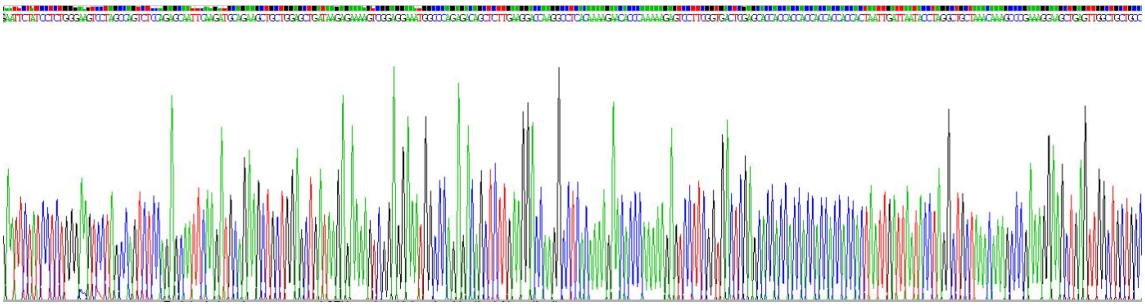

Figure 2. Gene Sequencing (extract)

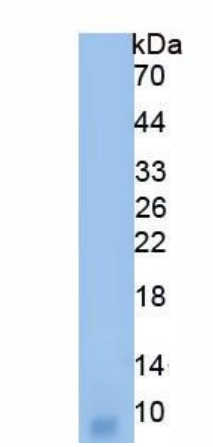

Figure 3. SDS-PAGE

Sample: Active recombinant NT-ProBNP, Mouse

[IMPORTANT NOTE]

The kit is designed for research use only, we will not be responsible for any issue if the kit was used in clinical diagnostic or any other procedures.