SANTA CRUZ BIOTECHNOLOGY, INC.

BNP (Q-16): sc-18817

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Natriuretic peptides comprise a family of three structurally related molecules: atrial natriuretic peptide (ANP), brain natriuretic peptide (BNP) and C-type natriuretic peptide (CNP). ANP and BNP act mainly as cardiac hormones, produced primarily by the atrium and ventricle, respectively, while the gene encoding C-type natriuretic peptide is expressed mainly in the brain. These peptides possess potent natriuretic, diuretic and vasodilating activities and are implicated in body fluid homeostasis and blood pressure control. ANP, BNP and CNP are highly homologous within the 17-residue ring structure formed by an intramolecular disulfide linkage. The genes which encode for ANP and BNP map to human chromosome 1p36.22. The gene which encodes for CNP maps to human chromosome 2q37.1.

REFERENCES

1. Saito, T., et al. 1975. Proceedings: Systemic-pulmonary arteriovenous fistula—a report of a case. Jpn. Circ. J. 39: 723.
2. Mair, J., et al. 2001. The impact of cardiac natriuretic peptide determination on the diagnosis and management of heart failure. Clin. Chem. Lab. Med. 39: 571-588.
3. Cowie, M.R., et al. 2002. BNP and congestive heart failure. Prog. Cardiovasc. Dis. 44: 293-321.
4. Hobbs, F.D., et al. 2002. Reliability of N-terminal pro-brain natriuretic peptide assay in diagnosis of heart failure: cohort study in representative and high risk community populations. BMJ 324: 1498.
5. Hall, C. 2004. Essential biochemistry and physiology of NT-proBNP. Eur. J. Heart Fail. 6: 257-260.
6. Pfister, R., et al. 2004. Use of NT-proBNP in routine testing and comparison to BNP. Eur. J. Heart Fail. 6: 289-293.
7. Lellouche, N., et al. 2007. Usefulness of preimplantation B-type natriuretic peptide level for predicting response to cardiac resynchronization therapy. Am. J. Cardiol. 99: 242-246.
8. Sheen, V., et al. 2007. The use of B-type natriuretic peptide to assess volume status in patients with end-stage renal disease. Am. Heart J. 153: 244.e1-244.e5.

SOURCE

BNP (Q-16) is an affinity purified goat polyclonal antibody raised against a peptide mapping within an internal region of BNP of rat origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-18817 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

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

APPLICATIONS

BNP (Q-16) is recommended for detection of precursor and mature γ-BNP and BNP 45 of rat 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)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000); non cross-reactive with ANP and CNP.

Molecular Weight of glycosylated BNP precursor: 25-36 kDa.

Molecular Weight of deglycosylated mature BNP: 12 kDa.

Positive Controls: C6 whole cell lysate: sc-364373.

RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use donkey anti-goat IgG-HRP: sc-2020 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible donkey anti-goat IgG-HRP: sc-2033 (dilution range: 1:2000-1:5000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto A Blocking Reagent: sc-2333 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml). 3) Immunofluorescence: use donkey anti-goat IgG-FITC: sc-2024 (dilution range: 1:100-1:400) or donkey anti-goat IgG-TR: sc-2783 (dilution range: 1:100-1:400) with UltraCruz™ Mounting Medium: sc-24941.

SELECT PRODUCT CITATIONS

1. Chu, C.H., et al. 2011. Histone acetylation is essential for ANG-II-induced IGF-IIR gene expression in H9c2 cardiomyoblast cells and pathologically hypertensive rat heart. J. Cell. Physiol. 227: 259-268.
2. Bian, Z.Y., et al. 2012. Disruption of mindin exacerbates cardiac hypertrophy and fibrosis. J. Mol. Med. 90: 895-910.
3. Lu, J., et al. 2013. Interferon regulatory factor 3 is a negative regulator of pathological cardiac hypertrophy. Basic Res. Cardiol. 108: 326.

RESEARCH USE

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

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.