



Human BNP Polyclonal Antibody

Antigen Affinity-Purified Anti-Human BNP Rabbit Antibody

Catalog Number: GR126057

Background

Brain natriuretic peptide is a 32-amino acid polypeptide secreted by the ventricles of the heart in response to excessive stretching of cardiomyocytes. BNP is synthesized as a 134-amino acid preprohormone (preproBNP), encoded by the human gene NPPB.[1] Removal of the 25-residue N-terminal signal peptide generates the prohormone, proBNP, which is stored intracellularly as an O-linked glycoprotein; proBNP is subsequently cleaved between arginine-102 and serine-103 by a specific convertase into NT-proBNP and the biologically active 32-amino acid polypeptide BNP-32, which are secreted into the blood in equimolar amounts.[2] The release of BNP is modulated by calcium ions. BNP is secreted attached to a 76-amino acid N-terminal fragment in the prohormone called NT-proBNP (BNPT), which is biologically inactive. Once released, BNP binds to and activates the atrial natriuretic factor receptors NPRA, and to a lesser extent NPRB, in a fashion similar to atrial natriuretic peptide (ANP) but with 10-fold lower affinity. The biological half-life of BNP, however, is twice as long as that of ANP, and that of NT-proBNP is even longer, making these peptides better targets than ANP for diagnostic blood testing. The physiologic actions of BNP are similar to those of ANP and include decrease in systemic vascular resistance and central venous pressure as well as an increase in natriuresis. The net effect of these peptides is a decrease in blood pressure due to the decrease in systemic vascular resistance and, thus, afterload. Additionally, the actions of both BNP and ANP result in a decrease in cardiac output due to an overall decrease in central venous pressure and preload as a result of the reduction in blood volume that follows natriuresis and diuresis. A normal level BNP or NT-proBNP can rule out acute heart failure in the emergency setting. However, an elevated BNP or NT-proBNP should never be used to "rule in" acute or chronic heart failure in the emergency setting due to lack of specificity.[3] BNP and NT-proBNP are also typically increased in patients with left ventricular dysfunction, with or without symptoms (BNP accurately reflects current ventricular status, as its half-life is 20 minutes, as opposed to 1–2 hours for NT-proBNP).[4] Low BNP was found to be a predictor of survival to age 90 in men,[5] and BNP may be a reliable predictor of cardiovascular mortality in diabetics.[18]

References

1. Sudoh T, et al. (1989) *Biochem. Biophys. Res. Commun.* 159 (3), 1427-1434.
2. Schellenberger U, et al. (2006). *Arch. Biochem. Biophys.* 451 (2): 160–6.
3. Maisel A, et al. (2002) *N Engl J Med.* 347 (3): 161–7.
4. Atisha D, et al. (2004) *Am. Heart J.* 148 (3): 518–23.
5. Nilsson, G; e tal. (2014) *Healthy Aging Research.* 3 (5): 1–10.
6. Bhalla MA, et al. (2004). *J. Am. Coll. Cardiol.* 44 (5): 1047–52.



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Description

Species reactivity: Human

Specificity: Detects human BNP in direct or indirect ELISAs and Western blots.

Source: Polyclonal rabbit IgG

Purification: Antigen Affinity purified

Immunogen: *E. coli* derived recombinant human BNP, Ser103 –His134

Accession # NP_002512.1.

Endotoxin Level: <0.10 EU per 1 µg of the antibody by the LAL method.

Formulation: lyophilized from a solution containing PBS and trehalose (100 µg/ml).

Application

Reconstitution: Reconstitute at 0.2 mg/ml in sterile PBS

Recommended concentration:

Western blot: >0.1 µg/ml

Immunocytochemistry: 5-15 µg/ml

ELISA: 0.2-0.6 µg/ml

Stability & Storage

Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 12 months at -20°C.
- 1 month after reconstitution at 4 °C, from date of receipt.
- 6 months after reconstitution at -20°C to -70°C from date of receipt.

DECLARATION

THIS REAGENT IS FOR IN VITRO LABORATORY TESTING AND RESEARCH USE ONLY. DO NOT USE IT FOR CLINICAL DIAGNOSTICS. DO NOT USE OR INJECT IT IN HUMANS AND ANIMALS.

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