Genorise® Recombinant Human BNP

Catalog #: GR119163

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 a. (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

Size: 10 µg
Source: *E coli* derived
Component: Ser103-His134
Accession # NP_002512.1
Predicted Molecular Mass: 4 kDa (monomer)

Specifications

SDS-PAGE: 4 kDa, reducing conditions
Purity: >95%, by SDSPAGE under reducing conditions and visualized by silver stain.
Formulation: Lyophilized from a 0.2 µm filtered PBS with BSA as carrier protein at 5 µg/ µg.

Preparation and Storage

Reconstitution: Reconstitute at 100 µg/mL in sterile PBS.
Shipping: The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage: Use a manual defrost freezer and avoid repeated freeze thaw cycles.

- 6 months from date of receipt, -20 to -70°C as supplied.
- 3 months, -20 to -70°C under sterile conditions after reconstitution.

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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