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Editorial

A Short Note on Atrial Natriuretic Peptide (ANP)

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DESCRIPTION

The NPPA gene encodes Atrial Natriuretic Peptide (ANP) or Atrial Natriuretic Factor (ANF), a natriuretic peptide hormone produced by the cardiac atria. The natriuretic peptides like ANP, BNP, and CNP are a structurally related family of hormone or paracrine factors. ANP's principal purpose is to reduce the amount of enlarged Extra Cellular Fluid (ECF) by boosting salt excretion in the kidneys. ANP is produced and released by cardiac muscle cells in the walls of the heart's atria. Volume receptors in these cells respond to greater atrial wall stretching caused by increased atrial blood volume. Secondary effects of ANP's blood volume reduction include decreased Extra Cellular Fluid (ECF) volume, better cardiac ejection fraction with enhanced organ perfusion, lower blood pressure, and higher serum potassium. Various counter-regulatory mechanisms acting simultaneously on each of these secondary effects may attenuate or cancel these effects. ANP is a 28-amino-acid peptide that has a 17-amino-acid ring in the center. A disulfide link between two cysteine residues at positions 7 and 23, forming a ring. BNP (brain natriuretic peptide) and CNP (C-type natriuretic peptide) are closely associated to ANP since they all have the same amino acid ring structure. ANP is one of nine structurally identical natriuretic hormones, seven of which are produced in the atrium.

The human NPPA gene, which is found on the short arm of chromosome 1, encodes ANP as an inactive preprohormone. The NPPA gene, which has two introns and three exons, is expressed largely in atrial myocytes and results in the production of preproANP, a 151-amino-acid polypeptide with a high molecular mass. The preprohormone is triggered by cleavage of the 25 amino acid signal sequence, which results in proANP, a 126 amino acid peptide that is the main form of ANP stored in the atria's intracellular granules. ProANP is produced and transformed to the 28-amino-acid C-terminal mature ANP on the cell surface by the cardiac trans-membrane serine protease corin after atrial cells are stimulated. It was recently revealed that ANP may also be O-glycosylated.

There are three types of atrial natriuretic peptide receptors that natriuretic peptides bind to. They're all cell surface receptors with the following names:

1. Guanylyl Cyclase-A (GC-A) also known as natriuretic peptide receptor-A (NPRA/ANPA) or NPR1
2. Guanylyl Cyclase-B (GC-B) also known as natriuretic peptide receptor-B (NPRB/ANPB) or NPR2
3. Natriuretic Peptide Clearance Receptor (NPRC/ANPC) or NPR3

NPR-A and NPR-B both feature a single membrane-spanning segment with an extracellular ligand-binding domain. By binding and segregating ANP from circulation, NPR-C primarily serves as a clearing receptor. The NPR-C binds to all natriuretic peptides.

The use of ANP for the treatment of acute heart failure and renal dysfunction is controversial. While this molecule has been proven to successfully restore several hemodynamic parameters after heart failure and to provide clinical relief for renal damage, it is unknown if it will lower mortality or have long-term consequences. As a result, further research is needed to better understand ANP's therapeutic benefits. ANP homologs that have recently been produced are being tested for the treatment of acute heart failure. However dietary salt limitation had no impact on the hypertensive state. In any case, they would be reinforced by additional examinations of the manner by which ANP-free homeostatic systems (like renal reactions to hypertension and volume extension) persistently adjust to supported volume development. In addition, dependable estimations of flowing ANP, renin, and aldosterone levels are required. In outline, despite the fact that ANP-subordinate balance of vascular porousness was perceived not long after the revelation of ANP, the significance of changes in vascular penetrability in controlling the appropriation of water between the plasma space and the interstitial space has not been generally perceived.

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proved to be safe, well-tolerated, and effective in the treatment of acute heart failure in preliminary studies. Further investigators would do well to be directed by the cautious assessments of the renal and cardiovascular capacities in these animals. No critical changes in food, water, and sodium were found in the endothelial cell-explicit GC-A knockout animals.