Brief Note

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Study of Heart, Cancer and Cardio-Oncology

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Book Review

The heart is now regarded as an important endocrine organ, as it produces many vasoactive peptides that have natriuresis, vasodilation, anti-inflammatory, and anti-cardiac remodelling properties. In the case of heart failure, natriuretic peptides become resistant or less effective, whilst inhibitors of their catabolism have been proven to reduce hospitalisation and mortality. Many human tumours can be reduced or eliminated with current chemotherapy treatments, but this comes at the cost of cardiovascular damage, which can occur during or after the treatment.

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These peptides were firstly implicated in the regulation of circulatory homeostasis showing pressure lowering properties, natriuretic, diuretic and kaliuretic effects because of inhibition of Renin-Angiotensin System (RAAS) and sympathetic outflow. They are produced by cardiovascular, brain and renal tissues. Dendroaspis Natriuretic Peptide (DNP), isolated from the venom of the green mamba and in patients with heart failure but still with unclear functions, is another peptide in the family.

The atrial granule produces and stores ANP, but the ventricle does not in normal circumstances. Mechanical stretching of cardiomyocytes, hypoxia, cold, or hormonal stimuli (angiotensin, catecholamine, endothelin, vasopressin, and glucocorticoid) cause transcription of proteins with GATA binding promoters and the production of Pre-ProANP (151 amino acids [aa]). Pro-ANP (126 aa) is generated and stored in the atria after a signal peptide of 25 aa is removed. Stimulation produced by a trial and ventricular stretch due to pressure or volume overload, cleavage of Pro-ANP by corin, resulting in the active C-terminal -ANP (28 aa) spilling into the coronary sinus and being dispersed in the systemic circulation, and the inactive N-terminal Pro-ANP (98 aa).

Urodilatin is a similar natriuretic ANP peptide that is generated in the kidneys and has local salt and water excretion properties. Later fragmentation of NT-ProANP by multiple peptides: Long Acting Natriuretic Peptide (LAMP), Vascular Dilator (VSDL), and Kaliuretic Peptide (KP) have been shown in some studies. -ANP has a half-life of 2 minutes and activates NP receptor A, which is highly expressed in the kidney, adrenal, lung, terminal ileum, aorta, and adipose tissue. It catalyses the conversion of GTP to GMPc, which regulates ion channels, nuclear translocation, gene expression, and protein phosphorylation, resulting in blood pressure reduction and sympathectomy.

Pre-ProBNP is the form of BNP that is stored and secreted by the normal atrial (132 aa) Ventricles only secrete when left ventricular function is insufficient. Following the removal of a 26aa peptide, 108 ProBNP is generated, which is then cleaved by furin (or corin) into the active BNP32 with a half-life of 2-28 minutes and the inactive N-Terminal ProBNP. Hypoxia, pressure or volume overload, interleukine-1, interleukine-6, and tumour necrosis factor- have all been linked to the generation of ProBNP. In addition to natriuresis, diuresis, and vasodilation, BNP protects the heart by decreasing cardiomyocyte necrosis and apoptosis, as well as lowering fibrosis and cardiac hypertrophy.

Current immunoassays do not distinguish between the many circulating BNP peptides, detecting ProBNP, O-glycosilated BNP, BNP, and NT-ProBNP, and do not represent bioactivity. In the case of heart failure, BNP-32 is almost non-existent in plasma. In a similar fashion to ANP, BNP works by binding to the NP receptor A.

CNP was discovered in various tissues (atrium, ventricle, kidney, endothelium, blood cells, and chondrocyte) after being discovered in the brain (as BNP). ProCNP is a 103-aa protein that is cleaved by furin into two peptides, one with 53 amino acids

(CNP- 53) and the other with 22 amino acids (CNP-22), both of which have identical activity and roles. CNP-53 is found mostly in the endothelium of the heart and the brain, whereas CNP-22 is found primarily in plasma and cerebral spinal fluid. CNP has no natriuretic properties and acts as a vascular tone regulator when it binds to NP's receptor B.