Journal of Autacoids

Open access Short Communication

Specific Endogenous Autacoids Demonstrating Cardio Protective Effects for Cardiac Diseases

Luke Richardson*

Department of Pharmacology, Marshall University, United States

INTRODUCTION

Chronic heart disease shares an inflammatory condition of unknown origin. In atherosclerosis, myocarditis, myocardial infarction, or atrial fibrillation, increasing evidence suggests that unresolved inflammation contributes to disease chronicity, exacerbation, and morbidity. After injury or infection of the heart, acute inflammation is a normal process necessary to repair damaged tissue, eliminate pathogens, and promote restoration of normal function and structure. Although conventional anti-inflammatory strategies to inhibit the production of inflammatory promoters, including the use of inhibitors, did not promote homeostasis, increasing evidence suggests that activating specific endogenous autacoids may provide a solution. Suggest that it can promote and maintain cardio-protective effects. Recent discoveries of active resolution mechanisms suggest that pro-resolution signalling and cellular processes may help shut down inflammation and combat the development of a chronic profile in heart disease.

DESCRIPTION

Inflammation is a complex program of active processes characterized by a well-coordinated sequence of initiation and resolution stages aimed at promoting homeostasis. When inflammation resolves unsuccessfully, tissues experience an unresolved inflammatory state that, if left uncontrolled, can lead to chronic inflammatory disease through exacerbation of structural damage, development of fibrotic areas, and loss of function. A variety of human diseases exhibit typical unresolved inflammatory profiles. Inflammatory diseases include cancer, neurodegenerative disease, asthma, right heart disease, atherosclerosis, myocardial infarction or atrial fibrillation. New evidence for a role has emerged, including the involvement of solubilizing chemical mediators in the acute phase of inflammation. Although there is extensive knowledge of the role of specialized pro-resolving mediators in neurodegenerative diseases, atherosclerosis, obesity, or liver fibrosis, little is known about their efficacy in controlling inflammation-associated arrhythmic heart disease. Resolvins have been shown to be bioactive lytic mediators. Resolvin stops neutrophil activation and infiltration, stimulates the polarization of monocytes into anti-inflammatory macrophages, activates inflammatory residues and phagocytosis of neutrophils by macrophages, leading to efferocytosis and infiltration. Promote clearance. The purpose of this review is to discuss failed resolution mechanism paradigms that may promote arrhythmia in inflammatory conditions induced by right heart disease. Inflammation is involved in atrial fibrillation, but classical anti-inflammatory molecules are ineffective. Recent evidence suggests that failure to resolve inflammation results in persistent inflammatory signals, and that a new family of drugs called resolvins promote inflammation resolution. Right heart disease is associated with atrial fibrillation. Experimental right heart disease shows evidence of activation of the atrial inflammatory pathway. Here, we investigated the effects of resolvin therapy on atrial arrhythmia remodelling in experimental right heart disease. Timely resolution of inflammation is essential to restore tissue homeostasis and prevent chronic inflammatory diseases. Resolution of inflammation is an active process regulated by a variety of pro-resolution mediators, including annexins and specialized pro-resolution lipid mediators that counteract excessive inflammatory responses and stimulate pro-resolution mechanisms [1-4].

CONCLUSION

Protective effects have been extensively studied in preclinical animal models. However, research into the function of these molecules in human disease is just beginning. This review article focuses on recent advances in the role of lytic mediators

Received:	31-January-2023	**Manuscript No:**	JAC-23-15822
Editor assigned:	02-February-2023	**PreQC No:**	JAC-23-15822 (PQ)
Reviewed:	16-February-2023	**QC No:**	JAC-23-15822
Revised:	21-February-2023	**Manuscript No:**	JAC-23-15822 (R)
Published:	28-February-2023	**DOI:**	10.35841/jac.4.1.05

Corresponding author Luke Richardson, Department of Pharmacology, Marshall University, United States, E-mail: Richardson-luke53@mum.edu

Citation Richardson L (2023) Specific Endogenous Autacoids Demonstrating Cardio Protective Effects for Cardiac Diseases. Autacoids J. 4:05.

and pharmacological opportunities to promote lytic pathways in preclinical models and patients with the various human diseases.

ACKNOWLEDGEMENT

None.

CONFLICT OF INTEREST

The author's declared that they have no conflict of interest.

REFERENCES

1. Tokumura A (1995) A family of phospholipid autacoids: Occurrence, metabolism and bioactions. Pergamon Prog LipidRes 34(2):151-184.
2. Narumiya S, FitzGerald GA (2001) Genetic and pharmacological analysis of prostanoid receptor function. J Clin Invest 108(1):25-30.
3. Chow L, Radio S, Sears T, Mcmanus B (1989) Insensitivity of right ventricular endomyocardial biopsy in the diagnosis of myocarditis. J Am Coll Cardiol 14(4): 915-920.
4. Wijeysundera D, Beattie W, Austin P, Hux J, Laupacis A (2010) Non-invasive cardiac stress testing before elective major non-cardiac surgery: Population based cohort study. BMJ 340:5526.