

Intracrine signaling by G-protein coupled receptors in the heart

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Bio:

After attending K-12 in Port Alberni, British Columbia, Bruce Allen completed his BSc in Biochemistry and both MSc and PhD degrees in the Faculty of Pharmaceutical Sciences at the University of British Columbia. Postdoctoral studies were in the Smooth muscle research group at the University of Calgary. Upon completing his postdoctoral training, he was recruited to the Montreal Heart Institute and Université de Montréal where he works on the role of stress-activated protein kinases and on G protein-coupled receptors in cardiac cells.

Abstract:

The response of the heart to increased blood pressure is to get enlarge, or 'hypertrophy'. Cardiac hypertrophy can lead to serious heart diseases such as myocardial infarction and cardiac arrhythmias: either of these may progress to heart failure over time. Certain factors, including the peptide endothelin, can stimulate the individual muscle cells of the heart to hypertrophy. Although this enlargement of the heart can initially be beneficial in maintaining adequate cardiac output, if the process of enlargement persists, a point is reached where the heart can no longer function properly. In 2003, the Allen lab demonstrated that endothelin receptors are also present on the membrane that surrounds the nucleus in the muscle cells of the heart. We now know that these receptors can alter gene expression. The pattern of genes expressed suggests that activation of endothelin receptors on the nucleus may actually be used to prevent or reverse enlargement of the heart. Subsequent studies have also shown that angiotensin II receptors and beta-adrenergic receptors are also located on the nuclear membrane in heart cells. Work is ongoing to determine the role of these receptors and evaluate their potential as targets for future drug development.