Khadija Rafiq
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Biography

Khadija Rafiq has her expertise in immunology and cellular biology. Over the past several years she has been investigating how the immune system affects cardiac myocyte growth and cardiac function with a focus on signaling molecules that are activated by inflammatory proteases. Her research interest focuses on elucidating the role of inflammatory serine proteases in the development of diabetic cardiomyopathy. It is well known that inflammation plays a role in the development of diabetic cardiomyopathy. The goals of her research are to identify novel signaling mechanisms that control cardiac cell growth and apoptosis.

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IMMUNE SYSTEM AND HEART FAILURE

Heart failure (HF) is the final clinical entity of many diverse disease causes and mechanisms. HF refers to a state of inadequate cardiac function to maintain systemic perfusion at a rate commensurate with the requirements of the body at rest or during states of increased demand. Mortality is comparable to that of the most common cancers, with a 50% 5-year survival. Despite advances in our understanding of the pathophysiology and treatment of HF, this malady continues to be a major public health burden with an enormous impact on the cost of healthcare. Current research efforts are focused on understanding novel mechanisms and signaling pathways. Immune activation and inflammation have been postulated as important pathophysiological events in this process. Cardiac inflammation is major pathophysiological mechanism operating in the failing heart, regardless of HF etiology. Experimental and clinical studies have suggested that inflammation in the development of heart failure is related to an imbalance between pro-inflammatory and anti-inflammatory cytokines. Furthermore, disturbances of the cellular and humoral immune system are frequently observed in heart failure. Therefore, it is essential to understand the immunological mechanisms involved in HF in order to develop useful therapies against the life threatening disorder.

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