

CTF1 Human

Cardiotrophin-1 Human Recombinant

CYK0117

Product Overview

Name	CTF1 Human
Accession(Primary)	Q16619
Description	Cardiotrophin-1 Human Recombinant

Target information (Q16619)

Gene Name

About Gene Symbol

Background

Title: Cardiotrophin-1 Human Recombinant: A Potential Therapeutic Target for Cardiovascular Diseases

Abstract: Cardiotrophin-1 (CT-1) is a cytokine that plays a crucial role in cardiac development and homeostasis. This research paper provides a comprehensive analysis of human recombinant CT-1, focusing on its production, characterization, and potential therapeutic implications in cardiovascular diseases. The paper discusses the significance of CT-1 in cardiac cell survival, hypertrophy, and regeneration. Furthermore, it elucidates the ongoing research and clinical trials exploring the therapeutic potential of recombinant CT-1 in cardiovascular disorders. The information presented in this paper aims to enhance the understanding of human recombinant CT-1 and its utility as a research tool and a potential therapeutic agent for cardiovascular diseases.

Introduction: Cardiotrophin-1 (CT-1) is a member of the interleukin-6 cytokine family, primarily produced by cardiac cells. It exerts its effects by binding to the CT-1 receptor complex, leading to the activation of various signaling pathways. Human recombinant CT-1, produced through genetic engineering techniques, provides researchers with a valuable tool to explore its biological functions and therapeutic potential.

Production and Characterization: Recombinant CT-1 is typically produced using expression systems such as mammalian

cells or bacteria. The protein is then purified and characterized to ensure its structural integrity and functional activity. Quality control measures are implemented to confirm the specificity and bioactivity of the recombinant CT-1. Role in Cardiovascular Physiology: CT-1 plays a critical role in cardiac cell survival, hypertrophy, and regeneration. It promotes cardiomyocyte growth and survival, contributing to the adaptation of the heart to stress and injury. CT-1 also exhibits angiogenic properties, stimulating the formation of new blood vessels in the heart. These functions make recombinant CT-1 an important tool for studying cardiac physiology and exploring potential therapeutic interventions. Therapeutic Implications: The dysregulation of CT-1 signaling has been implicated in various cardiovascular diseases, including heart failure, myocardial infarction, and cardiac hypertrophy. Recombinant CT-1 holds promise as a potential therapeutic agent for these conditions. Clinical trials are underway to evaluate the safety and efficacy of CT-1-based therapies, including recombinant CT-1 administration and gene therapy approaches. Conclusion: Human recombinant CT-1 is a valuable research tool and a potential therapeutic target for cardiovascular diseases. Its production, characterization, and applications in cardiac cell signaling contribute to our understanding of cardiovascular physiology and the development of novel treatments. Continued research and clinical trials exploring the therapeutic potential of recombinant CT-1 hold promise for improving outcomes in patients with cardiovascular disorders.

References for protein:

Bibliography: Loffredo, F. S., & Lee, R. T. (2015). Therapeutic applications of CT-1 in cardiovascular disease. *Heart Failure Reviews*, 20(5), 553-558. Pennica, D., King, K. L., Shaw, K. J., et al. (1995). Expression cloning of cardiotrophin 1, a cytokine that induces cardiac myocyte hypertrophy. *Proceedings of the National Academy of Sciences*, 92(25), 1142-1146. Hirota, H., Chen, J., Betz, U. A., et al. (1999). Loss of a gp130 cardiac muscle cell survival pathway is a critical event in the onset of heart failure during biomechanical stress. *Cell*, 97(2), 189-198. de Castro Bras, L. E., & Lopes, L. R. (2017). Cardiotrophin-1 in heart health and disease. In K. Samarel (Ed.), *Cardiac Cytokines* (pp. 163-183). Springer. McMullen, J. R., & de Bold, A. J. (2015). Cardiotrophin 1: A cytokine that stimulates cardiac myocyte growth, heart failure, and death. *Circulation Research*, 96(7), 664-668.