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ABSTRACT

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Abstract 14396: Cardioprotective Effects Of Glycyrrhizin On Hyperglycemic Cardiac Tissues

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Originally published 8 Nov 2021 | Circulation. 2021;144:A14396

Abstract

Introduction: Myocardial fibrosis and dysfunction is one of the major cardiac complications of long-term diabetes. Prolonged hyperglycemia is known to induce myocardial dysfunction often leading up to heart failure.

Hypothesis: The objective of this study was to investigate the cardioprotective effect of glycyrrhizin (GLC) on myocardial damage in engineered in-vitro human cardiac tissues. Engineered 3D tissue chips present an ideal microenvironment via therapeutically relevant interfaces to study molecular- and cellular-level events and mimic human-specific disease states, and identify new therapeutic targets in vitro.

Methods: AC16 human cardiomyocyte cells were used to 3D bioprint cardiac tissue chips based on prior published work. In our study, the 3D bioprinted cardiac tissue chips (CTC) were cultured using normo- (5mM) and hyper-glycemic (25mM) conditions for up to 48 hrs. For the GLC treatment group, a subset of CTC cultured using hyperglycemic conditions were treated with 50 mM of GLC for 24 hours.

Results: CTC cultured under hyperglycemic conditions demonstrated altered levels of connexin-43 (CX43) and Troponin-I implying cardiomyocyte injury. Exposure to hyperglycemia revealed changes in epigenetic markers: histone methylation marker (H3K9me)-1, Sirtuin-1, and Histone Deacetylase (HDAC)-2 as well as in inflammatory and stress related mediators such as heat shock protein (HSP)-60, receptor for advanced glycation end products (RAGE), toll like receptor (TLR)-4, high mobility group box (HMGB)-1 and CXC chemokine receptor (CXCR)-4. CTC exposed to 25mM glucose for 24 hours resulted in the downregulation of HSP60 and Sirtuin-1. Prolonged exposure to hyperglycemia led to decrease in the expression of CX43 and CXCR4; thereby adversely affecting cardiomyocyte function. Upregulated expression of DNA-binding nuclear protein HMGB1 along with changes in H3K9me1 indicated long-term hyperglycemia-induced damage to cardiomyocytes. GLC treated CTC exhibited a decrease in the expression of RAGE, TLR4 and also demonstrated altered expression of CX43, CXCR4, and troponin I.

Conclusions: This study suggests that GLC possesses cardioprotective effects in human cardiomyocytes exposed to prolonged hyperglycemia.

Footnotes

Author Disclosures: For author disclosure information, please visit the AHA Scientific Sessions 2021 [Online Program Planner](#) and search for the abstract title.

Circulation

November 16, 2021
Vol 144, Issue
Suppl_1

Article Information

Article Metrics [View all metrics](#)

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Originally published November 8, 2021

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Inflammation and inflammatory markers Epigenetics

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