Indian Journal of Experimental Biology
Vol. 63, April 2025, pp. 291-300
DOI: 10.56042/ijeb.v63i04.11870

Subsarcolemmal mitochondrial dysfunction aggravates ischemia-reperfusion injury in diabetic rat hearts on fructose and cholesterol diets

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Received 31 July 2024; revised 04 February 2025

The rising prevalence of diabetes mellitus and metabolic syndrome, driven by diets high in fat and fructose, has significantly increased the risk of cardiovascular complications, including ischemia-reperfusion (IR) injury. Understanding the mechanisms linking dietary stressors to mitochondrial dysfunction in diabetes is crucial to addressing this health burden. Mitochondrial dysfunction, particularly in the subsarcolemmal fraction, is a hallmark feature of diabetic cardiomyopathy and IR injury. This study assessed the impact of a high-fat and fructose diet on IR injury in diabetic rat hearts. Forty-eight Wistar rats were assigned to four groups: normal, diabetes mellitus (DM), DM with a cholesterol diet (CD), and DM with a fructose diet (FD). Isolated hearts subjected to IR injury via Langendorff perfusion demonstrated cardiac hypertrophy, increased oxidative stress, and mitochondrial dysfunction in CD and FD groups compared to normal rats. FD rats exhibited significantly higher serum glucose, insulin resistance, brain natriuretic peptide expression, and subsarcolemmal mitochondrial damage, resulting in worse cardiac recovery and greater myocardial injury post-IR than CD rats. The findings underscore the detrimental effect of a fructose-rich diet on cardiac and mitochondrial health in diabetic conditions. This study highlights the need for dietary interventions targeting mitochondrial function to reduce cardiovascular risk in diabetic populations.

Keywords: High-fructose diet, Cholesterol diet, Insulin resistance, Langendorff perfusion, Oxidative stress, Diabetic cardiomyopathy