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Research Article

Aspalathin, a dihydrochalcone *C*-glucoside, protects H9c2 cardiomyocytes against high glucose induced shifts in substrate preference and apoptosis

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Abstract

Scope

Energy deprivation in the myocardium is associated with impaired heart function. This study aims to investigate if aspalathin (ASP) can ameliorate hyperglycemic-induced shift in substrate preference and protect the myocardium against cell apoptosis.

Methods and results

H9c2 cells were exposed to, either normal (5.5 mM) or high (33 mM) glucose concentrations for 48 h. Thereafter, cells exposed to 33 mM glucose were treated with metformin (1 μM) or ASP (1 μM), as well as a combination of metformin and ASP for 6 h. In vitro studies revealed that ASP improved glucose metabolism by decreasing fatty acid uptake and subsequent β-oxidation through the decreased expression of adenosine monophosphate-activated protein kinase threonine 172 (pAMPK (Thr172)) and carnitine palmitoyltransferase 1 (CPT1), while increasing acetyl-CoA carboxylase *(ACC)* and glucose transporter 4 (*GLUT4)* expression. ASP inhibited high glucose induced loss of membrane potential in H9c2 cells as observed by an increase in 5′ ,6,6′-tetrachloro-1,1′,3,3′ -tetraethylbenzimidazolyl-carbocyanine iodide (JC-1) ratio (orange\red fluorescence) and decreased apoptosis by reducing intracellular reactive oxygen species and DNA nick formation, while increasing glutathione, superoxide dismutase, uncoupling protein 2 (*UCP2)*, and Bcl-2\Bax ratio.

Conclusion

Our study provides evidence that ASP increases glucose oxidation and modulates fatty acid utilization producing a favorable substrate shift in H9c2 cardiomyocytes exposed to high glucose. Such a favorable shift will be of importance in the protection of cardiomyocytes in the diabetic heart.

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