| Session: | Molecular Mechanisms in Cellular Processes |
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| Submitting user: | Ms. Tamsheel Fatima Roohi |
| Email: | tamsheelfatimaroohi22@gmail.com |
| Author(s): | Presenter: Ms. Tamsheel Fatima Roohi Ph.D. Research Scholar Department of Pharmacology, JSS College of Pharmacy, JSS Academy of Higher Education & Research, Mysore-570015 Karnataka, India. E-mail: tamsheelfatimaroohi22@gmail.com Mobile: (+91) 7376288410 |
| Туре: | Poster |
| Title: | β-Amyrin and metformin, alone and in combination, protect NRK-52E cell lines from high glucose-induced nephrotoxicity by attenuating oxidative stress, apoptosis, endoplasmic reticulum stress, and inflammation |
| Keywords: | β -Amyrin; Combination; Diabetic Nephropathy; NRK-52E; Nephrotoxicity; Endoplasmic Reticulum Stress; Apoptosis |
| Abstract: | Introduction: Diabetes, a global health issue, can cause diabetic nephropathy in one-third of its sufferers. Recently, β -amyrin, a natural pentacyclic triterpene, has garnered interest for its potential to combat diabetes and treat chronic kid-ney disease resulting from the condition. In this study, we investigate the anti-hyperglycemic and renoprotective effects of β -amyrin, both individually and in combination with metformin, through the use of NRK-52E cell lines. Methods: We conducted an investigation into the impact of β -amyrin and metformin, both separately and in combination, on NRK-52E cell lines under normo- |
| | glycemic and hyperglycemic conditions. High glucose-cotreated NRK-52E cells were exposed to these compounds, and their effects were evaluated through several parameters, including cell viability assessed by the cell counting kit-8 assay, apoptosis via flow cytometry, reactive oxygen species (ROS) levels using H2DCFDA expression, and gene expression studies via RT-qPCR to examine endoplasmic reticulum (ER) stress, apoptotic markers, and inflammatory markers. Results: Under normoglycemic conditions, the test compounds did not exhibit a significant impact on the cell lines. However, in the presence of high glucose, β - |

amyrin, metformin, and their combination demonstrated a noteworthy improvement in cell viability, with the combined treatment yielding superior results compared to individual treatments. Flow cytometry analysis revealed a substantial reduction in apoptotic cells, particularly with the combination treatment (p<0.05). Moreover, the test compounds effectively inhibited high glucoseinduced ROS production, with the combination treatment displaying the most significant reduction. Gene expression studies demonstrated downregulation of ER stress, apoptotic markers, and inflammatory markers, with the combination treatment consistently yielding the greatest reductions.

Conclusions: β -amyrin and metformin exhibit renoprotective effects in high glucose-induced renal cells, suggesting a promising therapeutic approach for mitigating hyperglycemia's adverse effects on renal function. Further research in diabetic patients is needed to validate these findings.