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IN VITRO EVALUATION OF THE INFLAMMATORY RESPONSE INDUCED BY EXCESS FRUCTOSE

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INTRODUCTION

Fructose is a monosaccharide naturally found in fruits, honey, and some vegetables, but its modern consumption has increased significantly due to its addition to processed foods and soft drinks, especially as high-fructose corn syrup. Excessive intake has been linked to metabolic disorders such as obesity, type 2 diabetes, and cardiovascular diseases. Recent studies suggest that fructose can act as an inflammatory trigger, promoting oxidative stress and mitochondrial dysfunction. This study aims to investigate the effects of fructose on macrophage polarization and its role in inflammatory response.

MATERIAL AND METHODS

RAW 264.7 macrophages were obtained from BCRJ and cultured in DMEM High Glucose with 10% FBS at 37°C. Fructose solutions were prepared in DMEM (High and Low Glucose) to reach final concentrations of 1000, 3000, 10000, and 30000 mg/L. Cell viability was assessed by MTT assay after 21 h of treatment, measuring absorbance at 570 nm. Nitric oxide (NO_2^-) levels were quantified in culture supernatants using the Griess reaction, with absorbance read at 550 nm.

RESULTS

Cell viability assessed by the MTT assay showed a concentration-dependent cytotoxic effect of fructose. In DMEM High glucose, fructose, at 30000 mg/L, reduced cell viability to a rate of 10%. In DMEM Low glucose, fructose reduced cell viability to a rate of 14%, at the same concentration. Intermediate concentrations (1000 and 3000 mg/L) maintained viability above 80% in both conditions. In the NO_2^- assay, LPS stimulation significantly increased nitrite levels, confirming the induction of a pro-inflammatory phenotype. Fructose at 1000, 3000, and 10000 mg/L led to lower NO_2^- levels, while 30000 mg/L caused a significant increase, suggesting a pro-inflammatory response at the highest dose.

CONCLUSIONS

Fructose exposure reduced cell viability in a dose-dependent manner and modulated the inflammatory response of macrophages. While lower concentrations showed minimal effects, high concentrations (30000 mg/L) significantly decreased viability and increased NO production, indicating potential pro-inflammatory and cytotoxic effects at excessive levels.

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