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ABSTRACT BOOK

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CELLULAR STRESS

AUTOPHAGY AND APOPTOSIS MODULATION BY AQUEOUS EXTRACTS FROM LEAVES AND RHIZOMES OF *Posidonia oceanica* ON HEPG2 HEPATOCARCINOMA CELLS

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Bioactive compounds produced by aquatic species exhibit a wide range of therapeutic effects in humans, representing encouraging prevention and/or treatment agents and beneficial supplements for the development of functional food and food-packaging material.[1-2] Within this context, aqueous extracts, obtained from green leaves (GLE) and rhizomes (RE) of the seagrass *P. oceanica*, were tested on HepG2 hepatocarcinoma (HC) cells to study cell viability/proliferation, cell cycle, apoptosis and autophagy modulation.[3,4] Both GLE and RE affected cell viability in a dose-response manner and the IC_{50} at 24h was calculated and used in the subsequent assays. Analyses of cell cycle and Annexin-V FITC binding indicated the apoptosis-promoting effect of both extracts, as also proven by the detection of the activation of caspase-1, -2 and -6 after exposure to both extracts and the additional and prominent activation of caspase-3 in the presence of the sole GLE. The intracellular accumulation of acidic vesicular organelles, hallmarks of autophagy, decreased after both treatments, more drastically after exposure to RE. Viability inhibition was not reverted by co-treatment of RE with the autophagy-stimulator rapamycin, suggesting a more extensive cell damage. The intracellular accumulation of the protein markers LC3, Beclin-1, p62/SQSTM1 and hsp60, related to the autophagic process and cytoprotection, at 4, 14 and 24 h of exposure was further studied through Western blot. Protein levels were downregulated in treated cells *vs.* controls with the exception of LC3 II/I ratio at 4 h and p62/SQST1 at all experimental times. The expression levels of *BCL2*, *BAX*, *BAD*, *FOS*, *JUN* and *DAPK* genes, involved in the apoptotic and autophagic processes, were analyzed through Real time-PCR. The results showed the downregulation of *DAPK*, *BAX* and *FOS* at 4 and 14 h of exposure, the upregulation of *JUN* at both times and the variation of *BCL2*/*BAX* and *BCL2*/*BAD* expression ratios in treated cells *vs.* controls. The results obtained suggest the prominent involvement of apoptotic promotion and autophagy downregulation in the anti-HC ability of both extracts from *P.oceanica*, and prompt further investigation aimed to identify the substance(s) responsible for the cytotoxic effect, thus opening the way to promising biomedical and nutraceutical applications.

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THE POST-TRANSCRIPTIONAL RNA EDITING LANDSCAPE OF STRESSED RETINAL EPITHELIAL CELLS: AN EPITRANSCRIPTOMICS APPROACH

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To date it is known that oxidative stress plays a fundamental role in retinal degenerative diseases, especially in inherited retinal dystrophies, but the molecular genetic causative mechanisms are not yet fully understood. In the present work, we carried out a comprehensive profiling of RPE cells treated with the oxidant agent N-retinylidene-N-retinyl ethanolamine (A2E). We considered a follow-up of four time points (1 h, 2 h, 3 h and 6 h) after exposure and compared them to untreated controls (time zero). We detected a total of about 63,000 between annotated and de novo RNA editing sites throughout all time-related samples. Approximately 19% of these RNA editing sites were found within 3′ UTR, including sites common to all time points that were predicted to change the binding capacity of 359 miRNAs towards 9654 target genes. After GO and KEGG enrichment analyses, different clusters of RNA editing sites associated with ECM and vascularization alterations, possibly related to RPE cell apoptosis, were identified. In addition, gene expression differences in deaminase family ADAR, APOBEC and ADAT members, already known to be involved in canonical and tRNA editing events, were detected. Collectively, the transcriptomics approach used in this work showed dynamic RNA editome profiles in RPE cells for the first time, elucidating new molecular mechanisms of retinal degeneration.

THE CHAPERONE SYSTEM IN SALIVARY GLAND DEVELOPMENT

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The chaperone system (CS) canonical function is to maintain protein homeostasis. Proper folding of nascent peptides is crucial in developing tissue. The chief components of the CS are

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