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MELATONIN TRIGGERS ER STRESS-ASSOCIATED APOPTOSIS IN HEAD AND NECK CANCER CELLS

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Melatonin Triggers ER Stress-Associated Apoptosis in Head and Neck Cancer Cells

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ABSTRACT

Melatonin is a methoxyindole synthesized and released principally by the pineal gland. Besides the functions of controlling sleep-wake, circadian, and seasonings cycles, melatonin also exhibits oncostatic features mitigating cancer at all stages through receptor-dependent and/or -independent mechanisms. Due to its low toxicity, melatonin has been used in the clinic as an endogenously generated agent helpful in cancer prevention and treatment. However, its role in head and neck cancer remains largely unknown. Here we report that melatonin displays strong effects on cell viability inhibition and apoptosis induction in a wide variety of head and neck cancer cells. Endoplasmic reticulum (ER) stress has shown to be highly associated with many forms of cell death. Intriguingly, increased protein levels of ATF6 and IRE-1, coupled with enhanced phosphorylation levels of PERK were seen in cells treated with melatonin. All these molecules are ER-resident transmembrane proteins responding to ER Stress, therefore, these observations strongly suggest that melatonin induces head and neck cancer cell apoptosis via triggering ER stress. Considering that little progress has been made in the treatment of head and neck cancer in the past decade, our findings lay the scientific foundation for promoting the use of melatonin for constraining this deadly disease.

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