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Pineal melatonin and cancer management: A review

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The neuro endocrine and immune systems were once considered as separate entities but recently both have been reported to be linked bidirectional. Melatonin, a neuro-hormone produced mainly by the pineal gland till date has revealed itself to be a pleiotropic and multitasking molecule although the neurohormone has been considered as a component for entraining the circadian system. It is a biogenic indoleamine and has attained a significant status as immune regulator. Any neurotransmitter, neuroendocrine factor and hormone can drastically change many physiological processes including immune function. The environmental stimulus and seasonal fluctuations affects the nervous system, endocrine functions and the incidence of opportunistic diseases. Report agrees that drug administration may lead to depression or stimulation of immune and other physiological responses depending on the time and dose of hormone and timing of their administration. Melatonin also stimulates the immune system and increases the cancer killing activity of macrophage, monocytes, NK cells, T helper cells and eosinophils. All of which are involved in cancer cell destruction. Additionally melatonin inhibits angiogenesis from existing blood vessels. Tumors get their nutrition through blood vessels and as they grow, they require an increasingly greater supply of blood vessels to feed themselves. Preventing new blood vessel growth limits their food intake and causes them to shrink or stop growing. Melatonin has properties which enable it to block the effects of estrogen upon cancer cells; this is important because certain forms of estrogen stimulate the growth of hormonally influenced concerns, such as breast cancers. The anti-proliferative activity of melatonin has focused on breast cancer possibly because melatonin has shown to modulate the activity of several aspects of endocrine physiology. According to the "melatonin hypothesis" decrease in melatonin is believed to lead an increase in estrogen level and an increase in the turnover of breast epithelial stem cells ultimately increases the risk for malignant transformation. Further, a very well known fact that melatonin acts through its receptors ML1 (ML1a & ML1b) which are high affinity melatonin receptors. The low affinity receptors are designated as ML2 & ML3. The ML1&ML2 are coupled to the adenylyl - cyclase and cyclic AMP inhibition where as mass spectrometry and enzymatic data confirms ML3 as quinone reductase (QR2) which is a detoxifying enzyme. The induction of this enzyme is actually associated with the decrease in susceptibility to cancer initiation and progression. Further melatonin function destroys cancer in multiple ways. Firstly because it is toxic to cancer cells, induces apoptosis and/or cancer cell auto destruction as well as directly kills cancer cells. It slows down the tumor growth via inhibiting epidermal growth factor receptor (EGFR) on cancer cells. The EGFRs play an important role in cancer growth and proliferation therefore blocking their receptors in cancer cells prevents them from carry out their role. Finally as an antioxidant, melatonin reduces inflammation, a condition that enables cancer's survival and it scavenges free radicals so that they do not damage normal cells and make them vulnerable to further genetic mutations. Physiologic and pharmacologic concentrations of the pineal hormone melatonin have shown chemo preventive, oncostatic and tumor inhibitory effects in a variety of *in vitro* and *in vivo* experimental models. Molecular mechanisms and signaling pathways involved in oncostatic function have now been partially identified, but surely deserve further investigation for better understanding of the physiological and therapeutic implications of melatonin as a cell protection molecule.

Biography

Seema Rai has completed her PhD from Banaras Hindu University. She has been the recipient of Fast Tract Young Scientist and Pool Scientist Award from Department of Science & Technology (DST) and Council of Scientific and Industrial Research (CSIR), New Delhi. She has also received the prestigious Invitation European fellowship as Post-doctoral studies from University of Insubria, Department of Clinical and Experimental Pharmacology, Varese, Italy in collaboration with the Institute of Pathology, Locarno Switzerland. She is Head of the Department of Zoology, Guru Ghasidas Vishwavidyalaya and a Central University. She has published more than 25 papers in reputed journals and has been serving as an Editorial Board Member of many reputed national and international Journals.

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