

## عنوان مقاله:

Melatonin prevent immortalization through reduction of hTERT mRNA gene expression in breast cancer cells

## محل انتشار:

یازدهمین کنگره بین المللی سرطان پستان (سال: 1394)

تعداد صفحات اصل مقاله: 2

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## خلاصه مقاله:

Breast cancer with the high prevalence in the world is yet one of the most challenging subjects in women's health. Increase knowledge of the biology and the molecular alterations in breast cancer, with heterogeneous nature, has facilitated the design of targeted therapies. It is not surprising that big efforts have been dedicated up to now to identify the new agents with antineoplastic efficacy that improve the conventional therapy. Melatonin a derivative of amino acid tryptophan, essentially produce by pineal gland and other organs. The studies highlight the potential role of melatonin in the prevention of mammary cancer and urge further investigation to dissect the possible underlying molecular mechanisms of this hormone in breast cancer. hTERT is the key component of the human telomerase complex that controls telomerase activity. hTERT expression is repressed in most normal human somatic cells, but high levels of its expression and activity are found in the majority of human tumors. In this study we evaluated the effect of melatonin on hTERT expression in breast cancer MCF7 cells. The cells affected with different doses of melatonin, viability and metabolic activity determined in compare with control, also we showed the hTERT mRNA gene expression after treatment with Melatonin in compare with control cells without affecting any drugs. Our result indicated that Melatonin could decrease the viability of the MCF7 cells. Metabolic activity of the cells with different doses of melatonin decreased. Also the hTERT mRNA gene expression by physiologic doses of melatonin, showed significantly reduction. Conclusion: It seems that, the oncostatic property of melatonin in reduction of viability and .metabolic activity in MCF7 cancer cells is at least in part, through the inhibition of telomerase

## کلمات کلیدی:

Breast cancer, Melatonin, mRNA gene Expression, Telomerase

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