Melatonia e câncer de mama

As células cancerosas são células em sofrimento por algum motivo sério o qual deve ser procurado e sanado. Sempre existe um ou mais motivos, que se forem desprezados serão os responsáveis pela recidiva do problema ou das metastases. Estas células necessitam de cuidados e não de aniquilação. JFJ

Melatonin and mammary cancer: a short review
Sánchez-Barceló EJ, Cos S, Fernández B, Mediavilla MD. Department of Physiology and Pharmacology, School of Medicine, University of Cantabria, 39011 Santander, Spain. barcelo@unican.es

Melatonin is an indolic hormone produced mainly by the pineal gland. The former hypothesis of its possible role in mammary cancer development was based on the evidence that melatonin down-regulates some of the pituitary and gonadal hormones that control mammary gland development and which are also responsible for the growth of hormone-dependent mammary tumors. Furthermore, melatonin could act directly on tumoral cells, as a naturally occurring antiestrogen, thereby influencing their proliferative rate. The first reports revealed a low plasmatic melatonin concentration in women with estrogen receptor (ER)-positive breast tumors. However, later studies on the possible role of melatonin on human breast cancer have been scarce and mostly of an epidemiological type. These studies described a low incidence of breast tumors in blind women as well as an inverse relationship between breast cancer incidence and the degree of visual impairment. Since light inhibits melatonin secretion, the relative increase in the melatonin circulating levels in women with a decreased light input could be interpreted as proof of the protective role of melatonin on mammary carcinogenesis. From in vivo studies on animal models of chemically induced mammary tumorigenesis, the general conclusion is that experimental manipulations activating the pineal gland or the administration of melatonin lengthens the latency and reduces the incidence and growth rate of mammary tumors, while pinealectomy usually has the opposite effects. Melatonin also reduces the incidence of spontaneous mammary tumors in different kinds of transgenic mice (c-neu and N-ras) and mice from strains with a high tumoral incidence. In vitro experiments, carried out with the ER-positive MCF-7 human breast cancer cells, demonstrated that melatonin, at a physiological concentration (1 nM) and in the presence of serum or estradiol: (a) inhibits, in a reversible way, cell proliferation, (b) increases the expression of p53 and p21WAF1 proteins and modulates the length of the cell cycle, and (c) reduces the metastatic capacity of these cells and counteracts the stimulatory effect of estradiol on cell invasiveness; this effect is mediated, at least in part, by a melatonin-induced increase in the expression of the cell surface adhesion proteins E-cadherin and beta(1)-integrin. The direct oncostatic effects of melatonin depend on its interaction with the tumor cell estrogen-responsive pathway. In this sense it has been demonstrated that melatonin down-regulates the expression of ERAlpha and inhibits the binding of the estradiol-ER complex to the estrogen response element (ERE) in the DNA. The characteristics of melatonin's oncostatic actions, comprising different aspects of tumor biology as well as the physiological doses at which the effect is accomplished, give special value to these findings and encourage clinical studies on the possible therapeutic value of melatonin on breast cancer.

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