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Melatonin decreases estrogen receptor binding to estrogen response element sites on the *OCT4* gene in human breast cancer stem cells

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Cancer stem cells (CSCs) pose a challenge in cancer treatment, as these cells can drive tumor growth and are resistant to chemotherapy. Melatonin exerts its oncostatic effects through the estrogen receptor (ER) pathway in cancer cells; however, its action in CSCs is unclear. Here, we evaluated the effect of melatonin on the regulation of the transcription factor *OCT4* (Octamer Binding 4) by estrogen receptor alpha (*ERα*) in breast cancer stem cells (BCSCs). The cells were grown as a cell suspension or as anchorage independent growth, for the mammospheres growth, representing the CSCs population and treated with 10 nM estrogen (E2) or 10 µM of the environmental estrogen Bisphenol A (BPA) and 1 mM of melatonin. At the end, the cell growth as well as *OCT4* and *ERα* expression and the binding activity of *ERα* to the *OCT4* was assessed. The increase in number and size of mammospheres induced by E2 or BPA was reduced by melatonin treatment. Furthermore, binding of the *ERα* to *OCT4* was reduced, accompanied by a reduction of *OCT4* and *ERα* expression. Thus, melatonin treatment is effective against proliferation of BCSCs *in vitro* and impacts the ER pathway, demonstrating its potential therapeutic use in breast cancer.

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