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Regular Article

Signal transduction of the melatonin receptor MT1 is disrupted in breast cancer cells by electromagnetic fields

Rainer Girgert [1 2 *], Volker Hanf [3], Günter Emons [1], Carsten Gründker [1]

[1]Department of Gynecology and Obstetrics, University of Göttingen, Göttingen, Germany

[2]Department of Nephrology, University of Göttingen, Göttingen, Germany

[3]Department of Gynecology and Obstetrics, Klinikum Fürth, Fürth, Germany

email: Rainer Girgert (rainer.girgert@med.uni-goettingen.de)

[*]Correspondence to Rainer Girgert, Department of Nephrology, University of Göttingen, Robert-Koch-Strasse 40, D-37075 Göttingen, Germany.

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breast cancer • electromagnetic fields • gene expression • melatonin

ABSTRACT

The growth of estrogen-receptor positive breast cancer cells is inhibited by the pineal gland hormone, melatonin. Concern has been raised that power-line frequency and microwave electromagnetic fields (EMFs) could reduce the efficiency of melatonin on breast cancer cells. In this study we investigated the impact of EMFs on the signal transduction of the high-affinity receptor MT1 in parental MCF-7 cells and MCF-7 cells transfected with the MT1 gene. The binding of the cAMP-responsive element binding (CREB) protein to a promoter sequence of BRCA-1 after stimulation with melatonin was analyzed by a gel-shift assay and the expression of four estrogen-responsive genes was measured in sham-exposed breast cancer cells and cells exposed to a sinusoidal 50 Hz EMF of 1.2 μT for 48 h. In sham-exposed cells, binding of CREB to the promoter of BRCA-1 was increased by estradiol and subsequently diminished by treatment with melatonin. In cells exposed to 1.2 μT, 50 Hz EMF, binding of CREB was almost completely omitted. Expression of BRCA-1, p53, $p21^{WAF}$, and c-myc was increased by estradiol stimulation and subsequently decreased by melatonin treatment in both cell lines, except for p53 expression in the transfected cell line, thereby proving the antiestrogenic effect of melatonin at molecular level. In contrast, in breast cancer cells transfected with MT1 exposed to 1.2 μT of the 50 Hz EMF, the expression of p53 and c-myc increased significantly after melatonin treatment but for $p21^{WAF}$ the increase was not significant. These results convincingly prove the negative effect of EMF on the antiestrogenic effect of melatonin in breast cancer cells. Bioelectromagnetics, 2009. © 2009 Wiley-Liss, Inc.

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