Title:	Knockdown of receptor for advanced glycation end products attenuate 17α-ethinyl-estradiol dependent proliferation and survival of MCF-7 breast cancer cells Author links open overlay panel
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Abstract:	Background 17α-ethinyl-estradiol (17α-EE), a synthetic estrogen is the world's most widely and commonly used orally bioactive estrogen. Currently, 17α-EE is in use in all formulations of contraceptive pills and is implicated in the complication of breast cancer. Receptor for advanced glycation end products (RAGE) is a cell surface immunoglobulin class of molecule. RAGE is involved in the complication of various cancers. Methods and results This study indicates that treatment of MCF-7 breast cancer cells with 17α-EE enhances the expression of estrogen receptor related receptor gamma (ERRγ), followed by enhanced level of oxidative stress and subsequent activation of the transcription factor, nuclear factor kappa-B (NF-kB), leading to increase in RAGE expression. RAGE thus expressed by 17α-EE treatment causes further enhancement of the oxidative stress which, in turn, activates expression of cell cycle protein cyclin D1 and subsequent induction of MCF-7 breast cancer cell proliferation. RAGE also enhanced phosphorylation of prosurvival protein AKT and increased expression of Bcl2, an antiapoptotic protein. Conclusion In MCF-7 breast cancer cells, 17α-EE-ERRγ interaction induces the expression of RAGE, which in turn, enhances the number of MCF-7 breast cancer cells through a multiprong action on the divergent molecules like cyclin D1, AKT and Bcl2. General significance This is the first report which explains the intermediate role of ERRγ in the 17α-EE dependent RAGE expression in MCF-7 breast cancer cells. This report for the first time explains that RAGE is important not only for MCF-7 breast cancer cell proliferation but also for its survival and anti-apoptotic activities.
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