The uterine and vascular actions of estetrol delineate a distinctive profile of estrogen receptor alpha modulation, uncoupling nuclear and membrane activation

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Résumé en anglais	Estetrol (E4) is a natural estrogen with a long half-life produced only by the human fetal liver during pregnancy. The crystal structures of the estrogen receptor alpha (ERalpha) ligand-binding domain bound to 17beta-estradiol (E2) and E4 are very similar, as well as their capacity to activate the two activation functions AF-1 and AF-2 and to recruit the coactivator SRC3. In vivo administration of high doses of E4 stimulated uterine gene expression, epithelial proliferation, and prevented atheroma, three recognized nuclear ERalpha actions. However, E4 failed to promote endothelial NO synthase activation and acceleration of endothelial healing, two processes clearly dependent on membrane-initiated steroid signaling (MISS). Furthermore, E4 antagonized E2 MISS-dependent effects in endothelium but also in MCF-7 breast cancer cell line. This profile of ERalpha activation by E4, uncoupling nuclear and membrane activation, characterizes E4 as a selective ER modulator which could have medical applications that should now be considered further.
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