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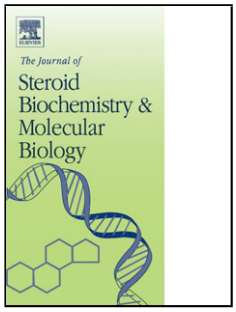

Title: Role of G-protein-coupled estrogen receptor (GPER/GPR30) in maintenance of meiotic arrest in fish oocytes

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ACCEPTED MANUSCRIPT

Role of G-protein-coupled estrogen receptor (GPER/GPR30) in maintenance of meiotic arrest in fish oocytes

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Highlights

- Estrogens maintain meiotic arrest of fish oocytes
- Estrogens act through GPER/EGFR pathway to maintain meiotic arrest
- PGRMC1 is required for expression of EGFR on oocyte membrane and meiotic arrest
- Bisphenol A prevents oocyte maturation through GPER/EGFR pathway
- 2-hydroxy-estradiol-17β acts as a GPER antagonist to promote oocyte maturation

Abstract

An essential role for GPER (formerly known as GPR30) in regulating mammalian reproduction has not been identified to date, although it has shown to be involved in the regulation a broad range of other estrogen-dependent functions. In contrast, an important reproductive role for GPER in the maintenance of oocyte meiotic arrest has been identified in teleost fishes, which is briefly reviewed here. Recent studies have clearly shown that ovarian follicle production of estradiol-17β (E_2) maintains meiotic arrest in several teleost species through activation of GPER coupled to a stimulatory G protein (G_s) on oocyte plasma membranes resulting in stimulation of cAMP production and maintenance of elevated cAMP levels. Studies with denuded zebrafish oocytes and with microinjection of GPER antisense oligonucleotides into oocytes have demonstrated the requirement for both ovarian follicle production of estrogens and expression of GPER on the oocyte surface for maintenance of meiotic arrest. This inhibitory action of E_2 on the resumption of meiosis is mimicked by the GPER-selective agonist G-1, by the GPER agonists and nuclear ER antagonists, ICI 182,780 and tamoxifen, and also by the xenoestrogen bisphenol-A (BPA) and related alkylphenols. GPER also maintains meiotic arrest of zebrafish oocytes through estrogen- and BPA-dependent GPER activation of epidermal growth factor receptor (EGFR) and mitogen-activated protein kinase (MAPK) signaling. Interestingly, progesterone receptor component 1 (PGRMC1) is also involved in estrogen maintenance of meiotic arrest through regulation of EGFR expression on the oocyte plasma membrane. The preovulatory surge in LH secretion induces the ovarian synthesis of progestin hormones that activate a membrane progestin receptor alpha (mPRα)/inhibitory G protein (Gi) pathway. It also

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