

Impact of Progesterone in Breast Cancer

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ABSTRACT

The critical role of ovarian sex steroid hormones (estrogens and progesterone) in the promotion and maintenance of breast cancer growth was suggested more than a century ago that demonstrated the bilateral oophorectomy resulted in remission of breast cancer in a premenopausal woman. Following the discovery that many Oestrogen Receptor-Positive (ER+) breast cancers are oestrogen dependent, extremely efficient adjuvant and chemopreventive medicines for these tumours were developed. Epidemiologic research also suggests that some exogenous synthetic progestogens (progestins) used with oestrogen as menopausal hormone treatment or contraception raise the risk of breast cancer. The discovery that estrogen+progestin menopausal hormone treatment increases the risk of breast cancer resulted in a significant decrease in the long-term use of these medicines to ameliorate postmenopausal symptoms.

Keywords: Breast Cancer; Progesterone; Epidemiologic; Prostate Cancer

DESCRIPTION

In contrast to the firmly related pharmaceutical effects of progestins to breast cancer risk, the etiologic significance of endogenous progesterone in the development of breast cancer is unknown. Progesterone has been implicated in the development of breast cancer in mechanistic investigations, although limited epidemiologic data have not offered significant support for a risk connection with circulating levels [1]. The previous studies suggested that progesterone metabolites have both pro- and anti-carcinogenic effects, and that the balance of these factors may contribute to breast cancer risk, but this hypothesis has received little attention in population-based research to date, owing to a lack of available assays [2].

Finally, studies show that progesterone signalling is involved in the aetiology of breast tumours in BRCA1 mutation carriers, implying that chemoprevention to disrupt downstream signalling may be beneficial [3].

Complex factors such as: dependence on and interaction with estrogens and other hormones (eg, androgens, prolactin, etc.), variation in exposure levels, eg: during the menstrual cycle or pregnancy, availability of sensitive assays, limited data on the roles of progesterone metabolites, and difficulties in assessing Progesterone Receptor (PR) isotypes. It is difficult to determine the specific effects of estrogens and progesterone since

transcription of PR is influenced partly, but not entirely, by Oestrogen Receptor (ER)-mediated transcriptional processes [4].

Furthermore, many of the activities of estrogens and progesterone, particularly in "normal" breast tissues, are mediated via paracrine pathways in which ligand binding to receptor-positive cells induces the production of substances that regulate the cell division of nearby receptor-negative cells. As a result, levels in tissues and blood may change, and biological effects may be influenced locally.

Given the inconsistent results and the interest in determining whether inhibiting progesterone/PR signalling is useful in breast cancer prevention and therapy, we critically examine the evidence linking progesterone to breast cancer risk and offer future possibilities for addressing existing knowledge gaps [5].

CONCLUSION

Progesterone is a 21-carbon steroid that performs its primary physiological functions by binding to the progesterone receptors A and B (PR-A and PR-B), which initiate transcription of targeted genes, resulting in the transformation of proliferative endometrium to secretory endometrium in an estrogen-primed uterus. Progesterone's physiological significance is generally limited to the peri- and post-ovulatory periods of the menstrual cycle, as well as pregnancy. Progesterone is generated by the

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corpus luteum during the menstrual cycle, commencing in the early postovulatory phase. Its rate of production rises from around 1 mg/day during the follicular phase to about 25 mg/day during the luteal phase.

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