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Estradiol/progesterone interaction in normal and pathologic breast cells.

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Abstract

In most target cells of the female genital tract, adequate cell differentiation is obtained via the successive and synergistic actions of estradiol (E2) and progesterone (P). This mainly due to the fact that progesterone receptor (PR) synthesis involves the prior action of estradiol through its receptor (ER). In normal breast, E2 stimulates the growth of the ductal system whereas lobular development depends on progesterone secretion. In other words E2 + P, when secreted in an adequate balance, permit the complete and proper development of the mammary gland. On the other hand progesterone may also have an antagonistic action against E2. The antiestrogen activity of progesterone is mediated through a decrease in the replenishment of E2 receptor and the synthesis of 17 beta-hydroxysteroid dehydrogenase, which leads to an accelerated metabolism of E2 to E1 in the target organ itself. These biochemical events, which have been well documented in the endometrium, have also been shown in cultures of normal breast epithelial cells as well as in differentiated fibroadenomas with high cellular density. In addition, data from the literature show that E2 added to human breast cells increases cell multiplication by means, eventually, of the synthesis of growth factors. Progesterone and progestins have a reverse effect. Data from our laboratory indicate that in normal cultured cells E2 and progestins are also antagonists with regard to cell multiplication. From these different data, it is postulated that in human beings, long periods of a luteal-phase defect leading to an unopposed estrogen effect might be a promoter of carcinogenesis in the breast.

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