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Regulation of CRBP1 In Mammary Epithelial Cells

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Abstract
Breast cancer is the second leading cause of death of women in the United States, warranting further investigation into preventative therapies. It has been well documented that early pregnancy results in a lifetime decreased risk of breast cancer in humans and mounting evidence suggests that the retinoic acid pathway may play an important role in this protective effect. Cellular retinol binding protein-1 (CRBP1) is an essential component of the retinoic acid pathway and we propose that it plays an important role in pregnancy-induced protection against breast cancer. In order to investigate the role of CRBP1 in parity-induced protection against breast cancer, we utilized both mouse and human mammary epithelial cells. We examined the effect that pregnancy has on CRBP1 expression, how CRBP1 is regulated by growth promoting and inhibiting agents, if loss of CRBP1 is essential for the induction of the apoptotic pathway, and how CpG methylation of key breast cancer genes relates to known risk factors for the disease. Based on our study, CRBP1 is persistently upregulated in

response to pregnancy in the mouse mammary gland at both the RNA and protein levels. Using a cell culture model, we established that CRBP1 is regulated by chemical agents that both promote and inhibit cellular growth. Utilizing CRBP1 knockout mice, we demonstrated that CRBP1 is not essential for induction of radiation induced apoptosis in parous mice. Finally, through methylation analysis, we examined how known breast cancer risk factors correlate to CpG methylation of three important genes for breast cancer and noted interesting trends that warrant future study.

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