The Informed Woman:
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Estrogen, Progesterone And Breast Cancer

What Effect do Hormones Have?

Molecular biologist, Dr. Ben Formby of Copenhagen, Denmark and Dr. T.S. Wiley at the University of California in Santa Barbara have researched two genes, BCL2 and P53, and their effect on female-specific cancers and prostate cancer.

Cells of breast, endometrium, ovary and prostate, were grown in the laboratory. Estrogen (estradiol) was added to the cells. This hormone turned on the BCL2 gene, causing the cells to grow rapidly and not die. Then, progesterone was added to the cell cultures. Cell reproduction stopped and the cells died on time (apoptosis).

This methodology was applied to all the above types of cancer. The BCL2 gene, therefore, stimulates the growth of these cells and the risk of cancer. On the other hand, the P53 gene promotes apoptosis or programmed cell death and thereby, reduces the risk of cancer. Estradiol upregulates or stimulates the production of the BCL2 gene, while progesterone upregulates or stimulates the production of the P53 gene.

Transdermal estradiol increased the cell proliferation rate by 230%, while transdermal progesterone decreased the cell proliferation rate by >400%. A combination estradiol/progesterone cream maintained the normal proliferation rate. This is direct evidence that estradiol (a potent estrogen) stimulates hyper-proliferation of breast tissue cells and progesterone mediates hyper-proliferation.

A second study by noted researcher Bent Formby, Ph.D. was just published with more insightful results. To determine the biologic mechanism of why progesterone inhibits the proliferation of breast cancer cells, a variety of cancer cell lines with different receptors and different expression of genes were exposed to progesterone. Exposure to progesterone induced a maximal 90% inhibition of cell proliferation in T47-D breast cancer cells and no measurable response to MDA-231 progesterone-receptor negative breast cancer cells. An impressive 43% of the T47-D cancer cells had undergone apoptosis (programmed cell death) within 24 hours after exposure to progesterone. Further analysis showed that the genetic expression by T47-D cancer cells of the bcl-2 gene was down regulated, and that of the p53 gene (tumor suppressor gene) was up regulated. Since the p53 gene expression induces cell apoptosis and the bcl-2 gene when expressed inhibits apoptosis, if one's cancer cells are progesterone-receptor positive, then progesterone as part of one's therapy appears to be very important. However, 50% of breast cancer cell lines have mutant or no p53 oncogene expression, so in this


Premature Birth


Osteoporosis


Infertility

instance, genistein therapy might be helpful.

Therefore unopposed estradiol causes these same types of cancer. Since Breast cancer is considered to be a hormone dependent cancer it is critically important to avoid the factors that would promote too much estradiol.

Are Birth Control Pills Safe?

In order for natural progesterone to stimulate the production of the P53 gene it must attach itself to progesterone receptors found in abundance in breast, ovarian, and endometrial cells. If a woman is taking birth control pills or any other form of synthetic progesterones (progestins, progesterone acetate, medroxy-progesterone acetate) these synthetic progesterones will occupy progesterone receptors and prevent natural progesterone from occupying the receptor site. Synthetic progesterones not only fail to produce the P53 gene but prevent it's production by blocking natural progesterone from occupying the progesterone receptor and in the presence of excess estradiol, dramatically increase a woman's risk for female-specific cancers.

There are **12 references** to tests on BCL2 and P53, and how they are affected by progesterone & estrogen. This information has been published, in part, in the following journals:

- The American Cancer Society Journal
- The Journal of Clinical Endocrinology
- The American Journal of Pathology
- International Journal of Cancer
- The Journal of the American Medical Association (JAMA)
- Fertility and Sterility - Journal of the American Society For Reproductive Medicine

Clearly some of the underlying causes of breast cancer are too much estrogen relative to too little of the helpful benefits of the P53 gene. This is especially so in the presence of trans fatty acids (hydrogenated fats).