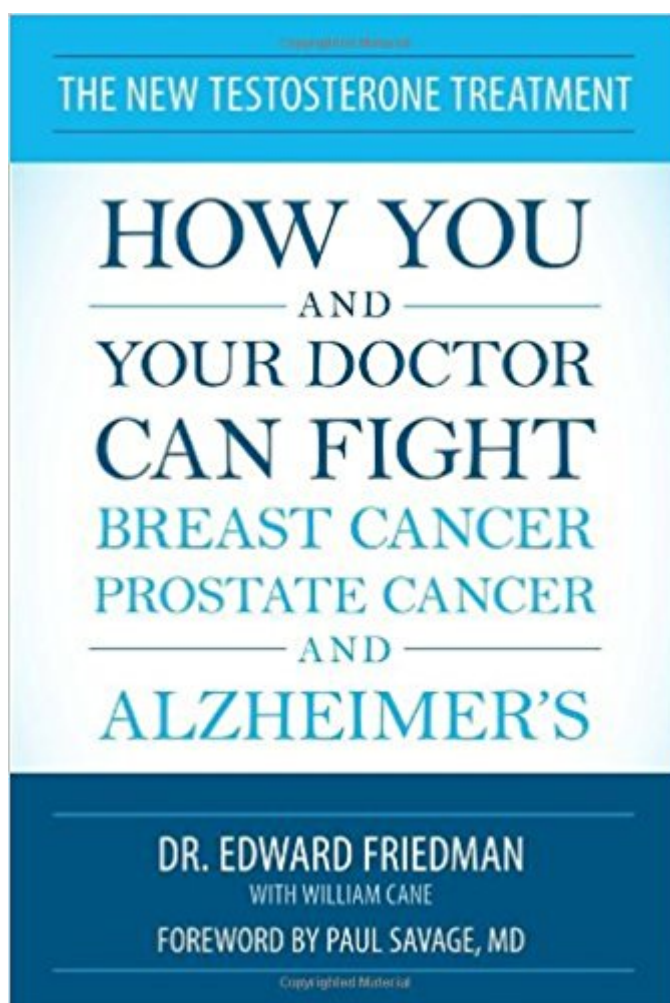


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The New Testosterone Treatment: How You And Your Doctor Can Fight Breast Cancer, Prostate Cancer, And Alzheimer's



Synopsis

Written by the leading authority on hormone receptors and prostate cancer, this book reveals the surprising truth about how you can prevent and treat breast cancer, prostate cancer, and Alzheimer's with testosterone and other FDA-approved drugs. For decades, doctors have sought to combat prostate cancer under the mistaken assumption that testosterone fueled its growth. But the latest research into the nature of hormone receptors and therapies using bioidentical instead of synthetic hormones have caused a shift in thinking and new hope for treating this cancer with testosterone. Today the medical profession equates a diagnosis of Alzheimer's with a death sentence. In fact, the only thing doctors do is throw ineffective drugs at it and resign themselves to failure. For the first time, this book explains how testosterone can halt the disease and cure early-stage Alzheimer's. Similar breakthroughs for fighting breast cancer follow close on the heels of these revelations, outlining how the avoidance of synthetic progestins and the use of aromatase inhibitors are crucial tools in prevention and treatment. At the core of this book is the remarkable observation that we experience our highest hormone levels during our teen years--a time of life when there is no breast cancer, prostate cancer, or Alzheimer's. Could bringing hormones back to teen levels be the key to vibrant good health? The answer is a resounding yes. This thoroughly researched guide to the latest biomedical research is must-reading for medical professionals and anyone concerned about their health.

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Customer Reviews

Edward Friedman, PhD is regarded as the world's foremost authority on models explaining how hormone receptors affect prostate cancer. Educated at the University of Chicago, where he works in the department of mathematics, he holds a doctorate in biophysics and theoretical biology. A frequent speaker at medical conferences, his work has appeared in numerous medical and biological journals, including Theoretical Biology and Medical Modelling, the FASEB Journal, the Journal of Urology, British Journal of Urology, and European Urology. William Cane taught English at Boston College and CUNY for two decades and is the author of eight books, including Clubhouse Confidential (with Luis Castillo), Write Like the Masters, and The Birth Order Book of Love.

Article Written by Carol Petersen, RPh, CNP [f&f](#) [-f](#) [œ Women's](#) [f&f](#) [-f](#) [,cs](#)
International Pharmacy

There is no shortage of information and opinions concerning hormone treatments or the "best" way to test for hormone deficiencies, not to mention how to use hormones or confirm if a hormone intervention is working. However, upon reading Dr. Edward Friedman's new book, *The New Testosterone Treatment: How You and Your Doctor Can Fight Breast Cancer, Prostate Cancer, and Alzheimer's*, it occurred to me that hormone receptors are really THE thing we should examine. Regardless of the testing method, the specific hormone, or its intended result, all hormone action occurs at the receptor sites.

What are Receptors? Receptors are protein structures designed to snag passing hormones. Receptors for hormones that poke through the cell membrane are called membrane receptors. Other receptors are inside the cell (intracellular receptors) in the cytosol, and more receptors are in the cell nucleus. The number of receptors is not stagnant, and varies according to nutrients and the environment. Once a receptor captures a hormone, that cell receives instructions for an action, such as cell replication, manufacturing other proteins, moderating cell activity, and programming abnormal cell death. A single hormone can produce action within minutes of binding. Receptors manipulate the cell's action by upregulating or down-regulating the production of proteins.

Hormones Have Affinities Conventional practitioners insist that as long as a hormone receptor receives a hormone [f&f](#) [-f](#) [•](#) whether it is identical to the human hormone or not [f&f](#) [-f](#) [•](#) all hormones and hormone-like substances should be considered equal. This thinking completely ignores the research identifying different affinities for different hormone receptors. As an example, the hormone estriol is generally considered a weak estrogen. This is because the binding of estriol on a receptor, in comparison to estradiol binding on the same receptor, produces less response. In sharp contrast, the receptors for estrogen in the urinary tract, bladder, and vaginal tissue have a much greater affinity for estriol. A study published in the *New England Journal of Medicine* demonstrated dramatic differences in effectiveness in treating urinary

tracts in elderly women with recurrent infection. Clinically, estriol also shines when treating vaginal dryness, outperforming estradiol and other estrogens.

Receptors Are Promiscuous

Even though receptors have affinities, they are not very discriminating about binding and can be affected by synthetic hormones as well as "the real thing." Receptor activity can be blocked or accentuated. For example, medroxyprogesterone acetate, a progestin rather than real progesterone, not only interferes with progesterone receptors but can block testosterone and cortisol receptors too. Because testosterone has such a positive effect on potential breast and prostate cancer (see below), this could help explain why this synthetic hormone is so frequently associated with increases in breast cancer, as reported in the Women's Health Initiative study.

A Hormone Receptor Model

Dr. Friedman, a theoretical biologist, describes the hormone "big picture" and also offers a theory on what he calls the Hormone Receptor Model. He believes that his model answers questions about how breast and prostate cancer initiate, and how this information can be used to target very specific treatment based on bioidentical hormones (particularly testosterone) to change the course of these diseases. Dr. Friedman states that breast and prostate cancer are fundamentally identical in their causes, presentation, and progression.

Introducing Bcl-2

Bcl-2 is a protein produced by hormone stimulation in the cell nucleus of cancer cells. This protein is of high importance in the discussion of breast and prostate cancer. Cancer cells are immortal; they escape the normal program for cell death called apoptosis. The Bcl-2 protein shields cancer cells from their normal cell destruction.

Estrogen Receptors

Estrogen Receptor Beta (ER-Beta) stimulation has a positive result, which is that the production of the Bcl-2 protein is down-regulated, thus depriving cancer cells of their immortality. Moreover, it also has an anti-inflammatory effect. Estrogen Receptor Alpha (ER-Alpha) increases inflammation and the production of the Bcl-2 protein. When breast cancer tissue is examined and reported as estrogen receptor positive, that information is incomplete. We need to know the concentrations of the different estrogen receptors. A dominance of ERBeta receptors is good. One feature of cancer cells is that the further the cancer progresses, the more ERAAlpha receptors are available.

Types of Estrogen and Their Binding Properties

Estradiol binds to both alpha and beta receptors with equal strength. Estrone binds to alpha receptors five times more tightly than to beta receptors, and estriol binds to ER-beta 3.2 times more tightly than it will bind to ER-alpha. So, the amount of Bcl-2 being produced is dependent upon which estrogen is binding, how strongly it is binding, and the concentration of each type of receptor. Hence, estrone is considered to be potentially more pro-cancer, while estriol is considered to be potentially more anti-cancer. (Please see our newsletter *Estrogen: Friend or Foe?* for more information.)

Progesterone Receptors

Progesterone Receptor B diminishes the production of Bcl-2

when activated, thereby also depriving cancer cells of their immortality. Fortunately, Receptor Bs tend to predominate, making the presence of progesterone typically more anti-cancer than pro-cancer. Progesterone Receptor A increases Bcl-2 and stimulation of this type of receptor is associated with BRCA1 and BRCA2 mutations. According to Dr. Friedman, the few women with these mutations also have increased numbers of Progesterone Receptor A. In turn, this leads to an increased Bcl-2 production protecting cancer cells. He outlines a different strategy to use in this situation (please refer to Dr. Friedman's book for more detailed information).

Androgen Receptors

The membrane androgen receptor behaves differently in men than it does in women. In women, stimulation of this receptor causes a decrease in Bcl-2; in men, it causes an increase in Bcl-2. In both men and women, stimulation of the intracellular androgen receptors decrease Bcl-2 and also causes the production of other anti-cancer proteins. However, if there is a shortage of testosterone to stimulate the intracellular receptors, the shortage favors more cancer cell growth.

Dr. Friedman's Synopsis

The above summary is a very simplistic synopsis of Dr. Friedman's views on hormone receptors and their role in diseases. A synopsis of Dr. Friedman's treatment program includes the following: He suggests that Vitamin D (which is a hormone) should always be considered first and foremost with a diagnosis of breast or prostate cancer. There is no downside to ensuring that vitamin D levels are optimized, and activation of the vitamin D receptor helps destroy cancer cells. (Please see our newsletter Vitamin D: The Sunshine Hormone for more information.) He states that ample amounts of testosterone are very protective against both breast and prostate cancer. He advises on the use of aromatase inhibitors to hamper the conversion of testosterone to estrogens, which can lead to more activation of ER-alpha receptors. He believes that estriol is underutilized, and that it could be supplemented generously to shift stimulation to the ER-Beta receptors. Premarin, with its predominance of estrone, clearly is a therapy that shifts the stimulation to the ER-Alpha receptors.

Dr. Friedman offers some very thought provoking ideas about using bioidentical hormones in the treatment of breast and prostate cancer. Although his theory is not yet tested, some practitioners have already begun incorporating elements of it.

A study recently published by Dr. Rebecca Glaser illustrates strong evidence for the idea that testosterone can be protective, and perhaps even effective as a treatment for breast cancer. She recently presented the results of 1,268 women who were receiving testosterone treatment along with an aromatase inhibitor. Although her study is designed for 10 years, she is already observing a dramatic decrease in breast cancer incidence in her study group, as compared to other studies and population statistics, at the 5 year mark.

Dr. Friedman feels that his methods are not intended to be a "cure" but a means to control cancer. He claims that the only side-effect is that, instead of suffering from the

disfigurement and secondary effects of cancer surgery, radiation, and the debilitation of hormone deprivation and chemotherapy drugs, restoring hormones to more youthful levels will yield a zest for life while living with cancer.

References

The New Testosterone Treatment: How You and Your Doctor Can Fight Breast Cancer, Prostate Cancer, and Alzheimer's by Edward Friedman, PhD, and Michael Cane; Prometheus Books; New York, NY; 2013.

"A controlled trial of intravaginal estriol in postmenopausal women with recurrent urinary tract infections" by R. Raz and W.E. Stamm; New England Journal of Medicine; September 1993.

"Reduced breast cancer incidence in women treated with subcutaneous testosterone, or testosterone with anastrozole: A prospective, observational study" by R.L. Glaser and C. Dimitrakakis; Maturitas; December 2013.

Several years ago, Dr. Friedman wrote a very technical article that was extremely well researched and clearly cited. I found that article because my sister was diagnosed with Stage 4 breast cancer in 1998 and we were looking for alternatives to standard protocol which ultimately fail if diagnosed with Stage 4 metastatic breast cancer. Because his journal article was so clearly written, I could understand much of it (even given my very limited technical background). His book takes the same very complex topics and simplifies them for people with no technical background, yet he has maintained incredible integrity by extensively citing the research on which he bases his conclusions. Every cancer researcher should read this book if only to take advantage of the research Dr. Friedman has conducted to determine which old research should be fully or partially discarded because of newer more credible findings. The footnotes and bibliography of this book are reason enough to buy it as a reference. My only minor criticism of the book is the repetitiveness of certain key concepts. However, others might see that as an effective way of reinforcing important concepts. Buy this book. If you have breast or prostate cancer, it will help you understand what you are dealing with and how you might help yourself.

The author of this book, a biologist of theory, bemoans the fact that the discoveries in breast and prostate cancer, which he has made are ignored by the medical profession. Not surprising, for it prompts the question "What constitutes a discovery?" In science, a good idea or hypothesis on its own is insufficient evidence. It must be subjected to the experimental method and then published, before general acceptance is accorded. The author's hypothesis (and that is all that it is) is that the two diseases result from an imbalance in their hormonal environments and treatment should take cognisance of this and be planned accordingly. In the case of prostate cancer, testosterone should constitute an essential addition to therapy. Now

this suggestion may be correct, for recent trials although small, are encouraging, with one probable cure reported. He intimates that the originator of such a good idea should be duly rewarded. Unfortunately this is unlikely to happen. There would be numerous claimants and honour will, without doubt, be bestowed on those who have established proof. Most of the book is devoted to an in depth description of the hormonal control of normal and abnormal breast and prostate function with an emphasis on hormone cell receptors. There is really no need to elaborate further for Nature has an annoying habit of disregarding logical deductions. So much so, that most discoveries in science arise from mistakes, chance findings or intuitions. The latter might apply to the author's hypothesis for he does state "One night as I drifted off to sleep, I began thinking, 'What did I learn from this book?' Editorial intervention is essential, for repetition abounds. The figures, drawn by Michael Christian, are a real treat and easily understood. Synthetic hormones are bad, bio-identical ones are good and studies that include the one variety cannot be compared with those that incorporate the other. Hormone replacement therapy using the synthetic type increases the risk of breast cancer. Theories of the causation of cancer are discussed and dismissed and the author presents his own in relation to breast and prostate cancer. Are they correct? I don't know. In essence it all begins with an excessive accumulation of estradiol in the relevant organs. Just when you have come to grips with the cancer story, Alzheimer disease is thrown into the pot. It's as though the author jumped on his horse and galloped in all directions. Nevertheless his theories are novel and ought to be assessed by those who profess to be cancer researchers. This book is not for the faint-hearted. Would I recommend it? Those with exceptional powers of concentration could give it a go. Mere mortals should watch the press for progress in the field.

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