



# Compassionate Oncology Medical Group

*Bob Leibowitz, M.D.*

DIPLOMATE  
AMERICAN BOARDS OF INTERNAL MEDICINE  
AND SUBSPECIALTIES OF  
MEDICAL ONCOLOGY AND HEMATOLOGY

## PROSCAR GREETINGS ON FATHER'S DAY **2008 Update**

Dr. Bob has been using Proscar since it first became FDA approved in 1992 to treat **all** of his prostate cancer patients. Dr. Bob acknowledges that he is probably the only prostate cancer expert who continues to favor using Proscar rather than Avodart. I acknowledge that Avodart is a more potent inhibitor of Type 1 and Type 2 five-alpha-reductase receptors than Proscar; that it lowers DHT (dihydrotestosterone) levels more than Proscar, and that it inhibits Type 1 5-alpha reductase, while Proscar does not, and that Avodart should be more effective. However, when Avodart first became available, we prescribed it to more than 50 Compassionate Oncology Medical Group prostate cancer patients who were being treated with Proscar, 5 mg once each day, so-called Finasteride Maintenance® Therapy. Patients were told to discontinue their Proscar and switch to Avodart, 0.5 mg (two per day for 14 days, then one each day). About one month later, we rechecked their PSA levels. If their PSA was lower on Avodart, they stayed on it; if higher, they stopped Avodart and restarted taking one Proscar per day. We checked monthly PSA's for all men either on Proscar or Avodart to see if we could identify any clinical trends. For all but a handful of patients, PSA levels were lower on Proscar compared to Avodart. Within three or four months, almost all of our patients were back on Proscar.

Although Avodart is a more potent inhibitor of 5-alpha reductase, the anti-prostate cancer benefits of Proscar may be due to other mechanisms. It is known, for example, that Proscar has antiangiogenic properties.

I wrote my first Proscar paper in celebration of Father's Day June 1997. June 15, 2008 brought us another Father's Day and since there is some new pertinent information regarding Proscar and prostate cancer, I decided to update "Proscar Greetings." Posted May 2008 in Reuters Health was an article titled, "Finasteride cuts prostate cancer risk without increase in aggressive disease." The initial conclusion reached by the Prostate Cancer Prevention Trial (PCPT) that finasteride reduces the risk of prostate cancer but increases the occurrence of aggressive tumors was only half true, according to findings presented at the American Urological Association meeting in Orlando, Florida in May 2008.

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A re-analysis of the data from the PCPT which involved over 18,000 men reveals that while the drug does cut the risk of prostate cancer, it does **not** promote the development of higher grade disease in men who develop prostate cancer.

In examining whether finasteride actually promoted the development of more aggressive tumors, Dr. Steven A. Kaplan, from Weill Cornell Medical College in New York, and colleagues decided it was important to consider the prostate-shrinking effect of the drug.

The team hypothesized that by shrinking the gland, finasteride simply made aggressive tumors easier to spot. Thus, they theorized if the analysis controlled for the decrease in prostate volume resulting from finasteride, highly aggressive tumors would be no more common than in the placebo group.

In fact, this is just what the group found.

Dr. Kaplan told Reuters Health that **finasteride cut the overall risk of prostate cancer by 34%**. The risk of tumors with Gleason scores 5, 6, and 7, "the most commonly diagnosed and treated cancers, was **markedly reduced**." Importantly, **the drug did not increase the risk of aggressive tumors, those with Gleason scores of 8, 9, and 10.**

The message for clinicians, Dr. Kaplan emphasized, is that "finasteride should be part of a discussion for cancer prevention, and certainly when used for benign prostatic hyperplasia, finasteride is safe."

The May 27, 2008 edition of the National Cancer Institute Cancer Bulletin, Volume 5, Number 11, pages 1-3, reports on the largest completed prostate cancer prevention trial. These authors have also determined that the initial results from this trial underestimated the benefits of Proscar and overestimated the potential risks of Proscar; all of this according to three new analyses of the data from that trial. I discussed one analysis above.

Two of the analyses were presented on May 18<sup>th</sup> at the annual American Urological Association Meeting. All three appeared on-line on May 18, 2008 in Cancer Prevention Research. The study involved over 18,000 participants. The initial conclusions from that study were incorrect. The authors of these three analyses have now shown that there was a 28%

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reduction in the risk of developing high-grade prostate cancer, as well as a 34% reduction in developing any grade prostate cancer. Two of the analyses were conducted independently using 500 prostatectomy samples from the more than 2,000 patients diagnosed with cancer in the PCPT to estimate the "true rate of high-grade disease" in the two study arms. One study arm was Proscar; the other study arm was placebo. Proscar was not associated with an increase in high-grade disease; instead, it had a modest protective effect.

The third analysis determined that Proscar prevented the development of **clinically significant** prostate cancer rather than only preventing prostate cancers that would probably never have caused a problem in the patient.

I believe that this new information reinforces the proven benefit of Proscar in helping to prevent prostate cancer in a 19,000 patient study, and describes additional benefits from Proscar for both preventing as well as helping to treat prostate cancer.

**PROSCAR GREETINGS ON FATHER'S DAY, 1997**

I have been asked so often and by so many different doctors and patients -- "Why Proscar?"

I actually have addressed this issue several times in the past -- in one or two prior (early) publications or informationals; and more recently in a community lecture given in February 1997 at Brotman Memorial Hospital in Culver City.

I was impressed with an article that appeared in the *Journal of Urology*, Vol. 148, pp. 1201 - 1204, October **1992**. The article's lead author is Joseph Presti, Jr. All patients had previously undergone a radical prostatectomy and then had rising PSA's.

The abstract of this article states that:

"A total of 28 untreated patients with asymptomatic, stage D<sub>2</sub> (this means that the patient has known metastatic disease to bones plus/minus lymph nodes) prostate cancer were randomized in a prospective double-blinded fashion to receive finasteride (10 mg per day), a 5-alpha reductase inhibitor or to receive

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placebo. Proscar is finasteride. Neither the patient nor the doctor knew who was on the medicine or on placebo. Patients were evaluated every three weeks by rectal examination, serum PSA, and prostatic acid phosphatase (PAP) levels, and at six-week intervals by bone scan and transrectal ultrasound determinations of prostatic volume. Patients could have their medicine stopped at week 6 at the discretion of the investigator if the PSA levels increased from baseline. After 12 weeks, all patients were reevaluated.

Of the patients, 13 received finasteride and 15 received placebo. The two groups did not differ statistically with respect to patient age, initial PSA and PAP level, or the extent of metastases on initial bone scan. A statistically significant decrease from their baseline PSA at weeks 3 and 6 occurred in the finasteride group" (-22% decrease in PSA) versus the placebo group; and at week 6, the results were -15.1% for the finasteride group compared to a rise of +11.7% for the placebo-treated group. What this tells me is that in patients with stage D prostate cancer, the men treated with finasteride saw their PSA's decrease 15.1% at week 6 versus a rise in PSA of 11.7% in men treated with placebo.

The abstract goes on:

"A decrease in serum PSA in the finasteride group suggests that finasteride exerts a minor effect in patients with prostate cancer."

Well, that is something positive." A minor effect; a decrease in PSA of 15% for Proscar patients versus an increase of 11.7% for placebo treatment. I know which group I would prefer to be in if I had stage D prostate cancer and was part of that study. Placebo effect is an interesting, fascinating side story. "Placebo effect" is a well known phenomenon. In a placebo controlled study, you give one-half of the study patients a medicine and one-half of the study patients a dummy or inert pill that looks exactly like the first medicine and neither the doctor nor the patient knows whether they are receiving the true medicine or the placebo pill. You then ask each patient to record any benefit or side effects.

In such studies, on average, about 5-15% of patients who receive the placebo will report diarrhea or some stomach trouble; 5-10% may report constipation; 7% or so may report that their (placebo) pill gave them headaches; 7% could report

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dizziness, pain, etc., etc. And depending on what you are studying, as many as 38-40% of patients will report significant improvement in the symptoms for which they were being treated, even though they received the inert placebo pill. That reminds me of something I often tell patients:

God heals the body (the patient);  
the doctor takes the credit;  
and then gets to bill the patient.

Finasteride, a 5-alpha reductase inhibitor, blocks the conversion of testosterone (T) to dihydrotestosterone (DHT). DHT is approximately 5-10 times more potent than T in stimulating prostate cancer to grow.

Men who are born with 5-alpha reductase deficiency do not ever develop prostate cancer. They do not have any DHT, although they have very high levels of testosterone. This tells us that DHT is necessary for prostate cancer to develop.

The Presti study later extended their original observations. After the first 12 weeks, those patients on finasteride who demonstrated more than a 25% decrease in PSA (at week 12) and those receiving placebo in the initial study entered an extension study.

"No serious adverse reactions occurred in the finasteride group and no patients were discontinued from the study due to clinical or laboratory adverse experiences," even though the Proscar patients were treated daily for 24 weeks with twice as high a dose of Proscar as I recommend.

"Those patients receiving continuous Proscar therapy for 12 and 24 weeks continued to have suppression of PSA."

That information helped lay the foundation for my recommending Proscar as the third drug in my Triple Hormone Blockade® recipe (or Triple Androgen Blockade®, if you prefer). Additionally, I reasoned that Finasteride (Proscar) Maintenance® Therapy should also be beneficial. The Presti study suggested that Proscar worked best in patients with small amounts of prostate cancer. Proscar as a single agent in men with advanced disease is not nearly as effective as LHRH agonists (Lupron or Zoladex) or antiandrogens (like flutamide or Casodex). Never use Proscar as a single agent for men with advanced disease.

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The Presti article also mentions their opinion that Proscar is more effective for prostate cancer cells in the prostate. They cite some experimental data suggesting Proscar may not be as effective for any prostate cancer cells that have already spread (metastasized) to lymph nodes, bones, or elsewhere. This is consistent with my use of Proscar (Finasteride Maintenance® Therapy) since men who had completed their 13 months of Triple Hormone Blockade® usually have had an unmeasurable PSA for an average of nine months; and hence would have a low total body tumor burden. This low volume disease is the best setting for (Proscar) Finasteride Maintenance® Therapy.

If the interested reader looks back on some of my earlier publications, I emphasized that combined androgen blockade (consisting of an LHRH agonist plus an antiandrogen, e.g., flutamide, etc.) works best on prostate cancer cells in the prostate. Labrie had pointed out that men who presented with both locally advanced disease and who also had metastatic disease to the bones, when treated with combined androgen blockade, almost never failed in the prostate. Only 1-1/2% of men progressed in the prostate area. The other 98-1/2% of men progressed in distant sites, primarily bones.

You can look at this in a simple way and explain that the hormone blocking medicines are "super concentrated" in the prostate (compared to metastatic areas) and hence more effective in the prostate.

What is probably more accurate is the following:

The cancer cells that are confined to the prostate consist primarily of less aggressive cells that try to behave themselves. However, a few of the cells mutate and/or acquire biologic aggressiveness. They figure out a way to escape from the prostate; usually by entering a blood vessel in the prostate area. They then have to find a way to survive while they circulate in the blood. They travel in veins and enter the right side of the heart. They are pumped into the lungs; they are then returned to the left side of the heart. They are pumped through the aorta and then must find their way to bones. Then they must figure out a way to leave the blood vessel and settle into the bones (like grass seeds). Then they have to figure out a way to attract blood vessels to them (angiogenesis) to bring them oxygen and nourishment.

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These cells are obviously the most aggressive of the cancer cells and have probably undergone a series of molecular biology "events;" changes that enhance their growth capabilities; and/or other changes that lower or eliminate tumor suppressor proteins (like p53). These tumor suppressor proteins inhibit cancer cells from growing. If one of your chromosomes takes a hit (molecular biological type hit), it might not produce any more suppressor protein and hence the cancer cells are free to grow.

This helps to explain why you would expect hormone blockade to be most effective for prostate cancer cells in the prostate compared to the bones. The cells in the prostate consist primarily of less aggressive cells. The cells in metastatic sites almost always contain much higher percentages of more aggressive cells. Men who are destined to die from prostate cancer don't die from prostate cancer cells in the prostate; they die from the cells that left the prostate and spread to distant sites, especially bones. When cancer cells spread, we say that the cells have metastasized. If prostate cancer cells spread to bones, for example, the patient does not have bone cancer; he has prostate cancer cells in his bones. And medicines that kill prostate cancer cells in the prostate are also very effective at killing prostate cancer cells wherever they are located in the body. When we are able to confirm that a patient has metastatic prostate cancer, we also describe this situation by stating the disease is systemic, meaning that the prostate cancer cells are not only found locally in the prostate, but also can be found systemically (in other areas of the body). When cancer cells are found in sites distant from the prostate, we say that the patient has systemic involvement rather than just local involvement. If prostate cancer is already systemic at the time it is found, than no form of local therapy can cure the patient since local treatment only kills prostate cancer cells in the prostate. This patient needs a form of treatment that can kill prostate cancer cells locally as well as the cells that are systemic. Systemic treatments are medicines for prostate cancer and include HB, chemotherapy and/or antiangiogenic agents. Since the mid-1990's, I have stated that in my opinion, "**The Best Form of Local Therapy is Systemic Therapy.**"

And, remember, if there are prostate cancer cells away from the area of the prostate, in my opinion, no form of local treatment can cure you. In fact, in my opinion, debulking even 99% of the volume of the local prostate cancer cells is more likely to

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cause harm rather than benefit. In my opinion, debulking is essentially never indicated. Only systemic treatments can kill prostate cancer cells away from the prostate.

In the past, I usually reserved chemotherapy for men who had hormone resistant or refractory prostate cancer. Even as of Mother's Day 2008, most prostate cancer experts are opposed to using chemotherapy for their patients with earlier disease. But by the late 1990's, I began to recommend using chemotherapy in patients with less advanced disease. By 2001, I recommended up-front chemotherapy for patients who presented with high-risk clinically localized disease, along with 13 months of Triple Hormone Blockade®, rather than Triple Hormone Blockade® alone.

Over the subsequent years additional information supporting my inclusion of Proscar has been published. Today I am certain that it has been a major factor in the success of my work.

In January 1996, the *Journal of Urology*, Vol. 155, pp. 3-9, published an article by Harry A. Guess (and others) titled:

"The Effect of Finasteride on PSA,  
Review of Available Data."

This article reviewed the available data on the effect of finasteride on serum levels of PSA.....in men with benign prostatic hyperplasia (BPH) and prostate cancer.

One of their conclusions:

"To interpret serum PSA levels in men with BPH treated with finasteride for six months or longer, the serum PSA level should be multiplied by two....."

I hear fairly often from some patients that their urologist does not want them to take Proscar because it interferes with PSA measurement and complicates the picture. I have a problem believing that criticism is valid. But if that is the only reason your doctor won't prescribe Proscar for you, tell him you will handle the difficult cloudy, complicated picture and multiply your PSA by two to compensate for the Proscar effect. Calculators are also available, if needed, to help in the multiplication process.

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The article goes on:

"Little further suppression of PSA occurs with finasteride treatment beyond 12 months. Therefore, the 'multiply by two' rule continues to apply beyond one year of treatment." (Dr. Bob adds that further follow-up shows that you have to multiply the PSA by 2.2 to correct for the Proscar or Avodart effect in patients who have been on Proscar or Avodart for more than about one year.)

But beginning on page 6 of the article comes the blockbuster news:

**"The Effect of Finasteride on PSA in Men with  
Prostate Cancer"**

A study enrolled 120 men, 48-89 years old. All of these men had previously undergone a radical prostatectomy (see my other publications to understand why I essentially almost never recommend radical prostatectomy or any form of radical local treatment).

Each of these 120 men were found to have rising PSA's following their radical prostatectomy. This meant that the radical surgery did not cure them; they failed surgery and had serial rising PSA's. None of them had abnormal bone scans and none had received any prior hormone blockade.

Therefore by definition, all of these 120 men had asymptomatic, occult, low volume (low total body tumor burden) metastatic prostate cancer.

Where was the PSA coming from, you ask? Where, indeed. Following a radical prostatectomy you are supposed to have an unmeasurable PSA within 30 days or less. The half-life of PSA is two to three days. PSA comes from: 1.) Benign prostate tissue – but by definition a radical prostatectomy removes all normal prostate tissue. If, following a radical prostatectomy you have serially rising PSA's, then the source of the person's PSA must be from the second source: 2.) Prostate cancer cells.

These prostate cancer cells could be located in the bed of the prostate (a so-called local recurrence) or in the distant sites (metastatic disease) or in both.

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In my opinion, isolated local recurrences almost never happen. Radiation therapy given for a PSA rising in the first year after radical prostatectomy does not result in an unmeasurable PSA five years later for even 10% of men. This is a major reason why I don't treat so-called local recurrence with more local treatment. I believe that more than 90% of these men have systemic disease and should be treated systemically. I advise 13 months of Triple Hormone Blockade® followed by Proscar maintenance. In 2001, this recommendation changed since prior local treatment shortens the duration of PSA control following 13 months of Triple Hormone Blockade®. We usually, but with significant exceptions, also add 15 doses of low-dose weekly Taxotere, Emcyt, and carboplatinum chemotherapy. This treatment is extraordinarily well-tolerated and does not cause nausea or vomiting.

Back to our study:

These 120 men were prospectively randomized to receive finasteride or placebo. They were followed for two years.

One group of men had an initial PSA at the start of the trial of less than one (remember they all had serially rising PSA's after radical prostatectomy so their PSA's could have been 0.2, then 0.5, then 0.7, then 0.9, but would still be less than 1 at the time the study started). For the men given placebo, their PSA's rose. For all of the men on finasteride with a baseline PSA less than 1, their PSA levels remained less than 1 for the next two years!!

For the men whose baseline PSA was between 1 and 10, the finasteride treated group had their PSA's remain suppressed for 14 months longer than placebo treated patients. Proscar delayed the rises in PSA for 14 months, and PSA's did not rise as high as placebo-treated patients. The study ended at 24 months. To me, this means that Proscar exerted a positive effect on prostate cancer cells, since their PSA had to be coming from prostate cancer cells. Since Proscar kept low PSA's under 1 for 2+ years, and delayed and decreased the PSA rise for men whose baseline PSA was between 1 and 9.9, it means that Proscar controlled their PSA. I submit this means that Proscar was controlling prostate cancer; not curing it, but controlling it.

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Most importantly to me: What happened to the finasteride treated group who subsequently underwent treatment with antiandrogens plus either LHRH agonists or surgical castration? (Remember I feel surgical castration should be outlawed for all men.)

Did the prior treatment with finasteride cause hormone resistance? No! Did the Proscar treated group respond the same to subsequent "strong" hormone blockade as did the placebo group? Yes!!! The article states:

"Men withdrawn from finasteride therapy due to recurrence who subsequently underwent medical or surgical castration had PSA responses similar to those not treated with finasteride, indicating that the recurrent disease was still hormonally responsive."

This article addresses the men with minimal low volume disease. This shows us that Proscar can control the PSA in this setting. I submit that since the only logical source of PSA in these men is from prostate cancer cells, then I must conclude that Proscar is controlling and (or hopefully) killing prostate cancer cells. These men don't have benign prostate cells; their PSA must be coming from cancer cells; Proscar kept their PSA controlled; Proscar must either be helping or certainly at worst not hurting. I bet helping. There is no way it can be hurting unless it caused hormone resistance and this study shows it did not (as of the close of the study).

This above information should help the reader understand why I use Finasteride (Proscar) Maintenance Therapy®. I have also used the following story to explain this:

Imagine if I told you that at the end of your 13 months of Triple Hormone Blockade®, I want you to eat one chocolate chip cookie a day. This will make your PSA go up more quickly, and make it rise higher; but don't worry.

You would probably tell me I was crazy and you would not take the chocolate chip cookie.

Now imagine that you are completing your 13 months of Triple Hormone Blockade® and I advise you to take Proscar, 5 mg once a day, so-called Finasteride Maintenance® Therapy. I inform you that with Proscar, your PSA will stay suppressed longer, and when it starts to rise, it will rise slower and not get as

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high as it would without the Proscar. Do you want to tell me that somehow this could be bad?? It prolongs disease-free survival (the length of time before a PSA rises); gives you a longer period of peace of mind; and might favorably impact long-term control of prostate cancer. And my results?? Please see my paper, "THB Update: The Demise of the (Fool's) Gold Standard; The Rise of the Platinum and Diamond Standard."

I have received a copy of an article authored by Nicholas Bruchovsky, Goldenberg and Gleave which will be appearing in *Advances of Urology*, Vol. 10, 1997. For the first time ever, I have seen other investigators write favorably about some of my up until now "unique" approaches to patients with prostate cancer. On page 311, this article mentions that there is the "potential for augmenting intermittent androgen suppression with other modalities such as 5-alpha reductase inhibitors." I am so pleased to see these pioneers in the treatment of prostate cancer, especially intermittent androgen blockade (IAB) pioneers, positively mention 5-alpha reductase inhibitors, such as Proscar, as a potential treatment to favorably influence IAB results. On page 315 of their article, they go on to state that "the possibility of using 5-alpha reductase inhibitor finasteride as a form of maintenance therapy to reduce the rate of increase in serum PSA during the next off-treatment period should be considered." Up until this article, I felt rather alone in my belief that Proscar could do this. Although there were probably many reasons for the Vancouver group to consider these statements, I hope that my own prior work and publications may have favorably influenced this world-renowned and respected group with regards to their considering the use of Proscar in these settings. I have recently spoken on the phone to Dr. Bruchovsky and in this personal communication he mentioned to me that he believes that Proscar maintenance therapy would probably prolong disease-free survival by at least 25% in men on IAB.

Hopefully, you now have the answer to: "Why, Proscar?"

When someone asks you for one of the reasons you are doing so well, answer: "Why Proscar, of course!!"

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**THE TRILOGY HAS BEEN COMPLETED**

This paper completed my 1997 "Trilogy" series. I wrote this trilogy to enable the reader to get inside my brain and, hopefully, understand my logical overview of prostate cancer. I began writing papers on prostate cancer in 1993. To date (as of June 1997), I had written about 125 total pages on these subjects. At first, I was astounded to discover that my review of existing prostate cancer medical literature found cure rates from radical prostatectomy and radical radiation therapy to be far less than believed; and side effects to be far more common and more severe than most authors' would admit.

I tried to approach this exciting, evolving frontier (?last major frontier) of clinical oncology. Prostate cancer treatment practices, and its medical literature, was a field dominated almost exclusively by surgeons (urologists) and radiation therapists. As a Harvard-trained board certified medical oncologist, I brought, perhaps, an entirely different perspective to this field.

In actuality, I believe that my analysis of prostate cancer has been pure logic. I identify most closely with the purely logical Mr. Spock from Star Trek. The more I studied this area, the much clearer the logic became. It was as if I could focus my energy and mind, and a clear path showed itself to me. I began to wonder why it was that others could not see, what to me, was so clear and logical. It was not as if I felt I "found" some deeply hidden near impossible puzzle that only I could translate. In fact, it was almost the opposite. I kept asking and wondering, "Why can't everyone else see what to me is so simple, obvious and logical?"

And in the past three months, it seems that a very small minority of prostate cancer specialists are seeing some of Mr. Spock's logic.

I am certain that IAB is superior (probably far superior) to continuous blockade. This concept should be able to be proven and will be widely practiced within the foreseeable future. (Dr. Bob adds that in the summer of 2007, for the first time ever, some prostate "experts" stated that IAB was at least as good as, and probably better than continuous hormone blockade. It took ten years for "them" to stop telling "us" that we should not use IAB because "they" felt it was experimental. My response to them is a quote I originated and often use:

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**"Everyone is entitled to their own (wrong) opinion."**

I am certain that radical local treatment, as initial therapy for prostate cancer will be much harder to eliminate, or at least radically restrict. Although 1997 may not be the beginning of the end for radical local treatments, it should be the end of the beginning.

I believe this trilogy of papers has expressed my opinions and has allowed me to explain my logic to you.

I hope that these works will help me be remembered as a former fruste of "Darth Vader" to radical local treatments, and as one of the earlier pioneers to confidently state that continuous hormone blockade is (will be proven to be) inferior to IAB.

My trilogy concludes:

When new topics again motivate me; or evolving literature supports or refutes me, I shall again become prolific.

Until then --

Be happy,  
Be well,  
Live long and prosper,

**BOB LEIBOWITZ, M.D. AKA DR. BOB**

P.S. As of July 4, 1997, for almost all men with prostate cancer, remember that:

**THE BEST LOCAL TREATMENT IS SYSTEMIC TREATMENT AND THE  
BEST SYSTEMIC THERAPY FOR MEN WITH LOW OR INTERMEDIATE-RISK  
PROSTATE CANCER IS TRIPLE HORMONE BLOCKADE®**

June 1997

Revised April 2004

Revised June 15, 2008 - **Happy Father's Day!**

**\*\*** None of the above should be construed as medical advice or consultation, and anything discussed in this paper is meant for information only. All medical treatments, consultations, decisions and recommendations can only be made by the patient and his/her treating physician.

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