PCNG

PROSTATE CANCER NETWORKING GROUP
of
Greater Cincinnati

Founder: Bob Kanter - Convener: Robert Young - Conveners Emeriti: Adrian Boie, Lou Stadler
- Newsletter: Kees DeJong & Fran Stanton-
Treasurer: Jerry Smith, 1621 Raglan Av., Cincinnati OH 45230 – Please, make checks payable to the Wellness Community

779-0144 Adrian Boie: 1989, PSA 13, GS 9; RP, EBRT, IHT
751-6888 Kees DeJong: 1996, PSA 24, GS 9; IHT
221-6736 John Hoffmann: 1997, PSA 5, GS 6, RP, EBRT

TELEPHONE CONTACTS:

528-2769 Gordon Huntley: 1999, PSA 4, GS 9, RP and Orchiectomy
733-5745 Bill Riggs: 1995, PSA 33, GS 6, RP, EBRT, HT

761-9645 Lou Stadler: 1987, PSA NA, GS 7; EBRT, HT
542-4908 Fran Stanton: 1999, PSA 157, GS 8; HT, EBRT+Brachy
321-1693 Robert Young: 1999, PSA >1,000, GS 7; HT

19xx: year of diagnosis - PSA: Prostate Specific Antigen - GS: Gleason Score - RP: Radical Prostatectomy - EBRT: External Beam Radiation Therapy - Brachy: Brachytherapy ('seeds') - HT: Hormonal Therapy - IHT: Intermittent Hormonal Therapy

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Our next Large Group meeting will be held on July 25, 2001
Women are Welcome!

6:30 pm: hospitality and networking
7:00 pm: new members - sharing
7:45 pm: networking

8:00 pm Robert Young on "How to use the Internet without having a computer"

Our next Small Discussion Group meeting will be held on August 8, from 7:00-9:00 pm

One discussion group for men
A separate discussion group for their spouses, partners, or family members

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This Month's Feature Article

"The Auditorium of Sarasota Memorial Hospital was the scene of the first annual Distinguished Lecturer Series sponsored by The James F Mullen Memorial Fund, Inc., successor organization to Man To Man, Incorporated. This gathering represented the culmination of the dreams and hopes of Jim Mullen, who in 1990 founded the program known as Man To Man, now the property of the American Cancer Society."

*Dr. Andrew C. von Eschenbach of the MD Anderson Cancer Center in Houston (TX) presented in January "**Recent Research In Treating Prostate Cancer**". In our Newsletter we present the last part of his lecture.*

…… we know that the progression or behavior of the cancer cell, and specifically a prostate cancer cell, is dependent upon two things. It is dependent upon changes that are occurring within that cancer cell itself, and it is also dependent upon that cancer cell's interaction with it's environment. Both of these factors are important.

Oncogenes and tumor-suppressor genes

Some of the things that are occurring within the cancer cells themselves have to do with things

that promote the growth of the tumor. This growth is under the control of a number of genetic alterations that influence how these cancer cells behave. Some of these genes are called oncogenes; they are like the accelerator in a car. When there is a problem with an oncogene, it is as if the accelerator in the car is stuck in the open position; the cells are driven to increase. Some of the other genes that affect cancer cell growth are called tumor-suppressor genes. These act like the brakes on a car. If there is a defect in a tumor-suppressor gene, it is like having no brakes and the car cannot stop. The cell is again, continuing to grow out of control. Some of the genes that we have recognized in the prostate that have to do with it's growth and proliferation are oncogenes like BCL2 and tumor-suppressor genes like P53.

When there is a problem with P53, it is a problem where there are not brakes in this cancer cell, and no way of stopping it once it is growing. One strategy we think about in being able to effect the cancer cell is, can we restore the defective brake within that cell? One way of doing that is by taking a normal P53 gene, and attaching it to an adenovirus. When the virus infects the cell, carrying along with it this normal P53 gene, that gene may be able to restore the defective brake. We do this by injecting the viral P53 directly into the cancerous prostate, much the way we do brachytherapy, by using ultrasound control. We have learned that by doing this procedure before a radical prostatectomy, we could study the effectiveness under the microscope after the prostate had been removed. The P53 genes were there, putting on the brakes. That, in itself, did not completely destroy, or control, all the cancer in the prostate, but by restoring the brakes, we could potentiate some of the other treatments that also put brakes on the cancer cell, like androgen ablation and radiation therapy. One of our current protocols today, is to use P53 combined with radiation therapy as a way of biologically destroying the cancer cell.

Introducing Anti Angiogenesis

The other thing that we have learned is that in addition to cancer cells being able to proliferate, that in order for them to grow and develop into tumors larger than just the size of a pinhead, they have to be able to get oxygen, they have to be able to get a food supply. The way they do that is by tricking the body into bringing new blood vessels into the tumorous area so that these cells can get more oxygen, more food, and grow into a larger lump. This process of new blood supply is called angiogenesis. One of the strategies we have of blocking a tumor is to prevent this development of new blood vessels: anti angiogenesis.

We now have drugs that can block the effect of that tumor cell and prevent it from creating its new blood supply. Some of those drugs are ones that were used in the past, like thalidomide. That same thalidomide that had horrible complications when given to pregnant women precisely because the thalidomide blocked the development of limb buds in the fetus. Babies were born without arms and legs and other parts. We use that drug now, enabling us to stop the growth of tumor cells. We use thalidomide in combination with other drugs. We are also using it after radical prostatectomy and radiation therapy when patients show a rise in PSA but no clinical evidence of disease, in an effort to see if we can prevent the progression of small tumor cells that are obviously growing and were not completely destroyed by local treatment.

Thwarting The Metastatic Process

Finally, one other important biologic strategy is to be able to interfere with the process of metastasis. We now know that cancer cells just don't haphazardly, randomly, spread to other body parts. They go there in a very precise and a very specific way. The precision has to do with the fact that these cancer cells are interacting with stromal cells, or the environment that they find themselves in, and that this environment then presents us an opportunity for being able to prevent metastasis. The behavior of the cell is now being determined, not by its own properties of genetic alterations, but by the environment that it finds itself in. One of the questions that was being posed to us was 'why do prostate cancer cells have this very specific propensity to metastasize to lymph nodes and bones? What was it that was so special about this environment that makes it so likely to be a place for prostate cancer?'

When prostate cancer cells metastasize to bone, they cause the bone to form more bone and so in an x-ray it becomes white and chalky because of the calcium. Unlike most tumors when they metastasize, which eat away the bone; prostate cancer does just the opposite. We have known for a long time that this was a characteristic pattern of prostate cancer. What it was telling us was that the cancer cells were talking to the bone cells. What we know now is that the bone cells were talking to the cancer cells. The prostate cancer cells were sending a message to the bone to cause the bone cells to develop, but the

bone cells were also sending a message to the prostate cancer cells causing them to develop. We know now that bone stromal cells make a bone protein, which is normally supposed to be a bone growth factor, which stimulates the growth of prostate cancer cells. The reason why we have prostate cancer going to bone in a favorable environment; there are growth factors and there is a food supply present that the prostate cancer cells like.

We now can exploit that by realizing that maybe we can change the bone environment and, instead of being hospitable to prostate cancer cells, make it just the opposite, and make it hostile. This is a trial that we just finished and has been published in Lancet <see abstract below>. Men who had hormone-independent progression of prostate cancer involving bone were treated with adriamycin, a chemotherapy drug. But some of them also got a drug called strontium 89, which directly affects the bone cells. The patients were randomized as to who got adriamycin plus a placebo and who got adriamycin and strontium 89.

In the men who had strontium, which affected the bone environment, there was a much better outcome than the patients who got only the adriamycin alone, affecting just the cancer cells. There was an 80% reduction in PSA in the former group versus 36% in the latter. There was also significant improvement in their time to progression, almost double, and some improvement in their overall survival; showing and demonstrating that altering the environment could significantly alter the outcome of the tumor. We now need to look at what we could do with altering the macro environment - not only the immediate environment of the cancer cell, but also the environment of the patient himself. What can we learn about nutrition, the immune status, and other hormones that can significantly influence the macro environment and alter the outcome of the tumor?

A Paradigm For The Future

The new paradigm for the future, whether we are talking about latent or clinically significant or advanced cancer, we are now learning the biologic steps that are controlling or regulating this process. With each of these biologic steps that we are learning about, we are opening up a whole new portfolio of treatment that we can use to be able to influence or prevent that progression. When we are able to do that, we then open up a whole net set of strategies that can help us be able to prevent a prostate cancer cell from progressing and growing to the point that it results in a painful and difficult death for patients. Some of those things will have to do with diet; some of those things will have to do with creating or causing these cancer cells to die. Some of these will have to do with the ability to prevent their growth or cause them to not become angry and aggressive in their appearance. But, the point I leave you with, is that the future is much more hopeful than the past because our approach to prostate cancer in these next few years will be dramatically different than it was in the past.

Next month the Large Group meeting will be held on August 29, 2001

The speaker, Dr. B. Aron, will discuss:

"Diagnosis and Prognosis in Prostate Cancer"

In every struggle the only ones who can truly grasp your fear, your pain, your grief, and your stamina that may sometimes fail are those who share the battlefield with you. It is no different when the enemy is prostate cancer, and the fight is for your integrity as a man as well as your life.

www.phoenix5.org/battle.html

BOOKS: Click on **www.prostatepointers.org/prostate/lay/apilgrim** for an excellent book on PC. Interested in prostate cancer and nutrition? Dr. Bob Arnot's "The Prostate Cancer Protection Plan" is a fine book on this topic. See also Dr. Myers et al.'s "Eating Your Way to Better Health. The Prostate Forum Nutrition Guide."

MAGAZINES:
Cancer Communication -PAACT (Lloyd Ney +) - (616) 453-1477 **www.paactusa.org/**
Insights -PCRI (Stephen Strum, M.D.) - (310) 743-2110 **www.prostate-cancer.org**
PC-REF Reporter -PC-REF (Israel Barken, M.D.) - (619) 287-6682 **www.prostatecancer.com**
Prostate Bulletin -(Dr. Ballentine Carter, The Johns Hopkins University) ($195/year - 4 issues) **prostate.urol.jhu.edu/**
Prostate Forum -(Charles Myers, MD, (804) 974-1303 **www.prostateforum.com** ($36/year for 12 issues)
The Prostate Cancer Exchange -ECPCP (James Lewis, Ph.D.) - (516) 942-5000

WEB SITES : **www.phoenix5.org** (Robert Young's web site!), **www.prostatepointers.org/prostate**, **www.psa-rising.com/**, **www.prostatelab.com**, **www.cooleyville.com/cancer, www.yananow.net** (an Australian site), **www.capcure.org/** (with many videos of the September 2000 Conference)
www.asco.org/prof/me/html/01abstracts/toc.htm (Abstracts of the ASCO 2001 Annual Conference)

PC STUDENT: as of 7/18: **www.ncbi.nlm.nih.gov**, search in PubMed 24,003 abstracts of medical papers on Prostate Cancer; **clinicaltrials.gov/ct/gui/c/r**, search 125 clinical trials for PC patients; visit the UC Health Sci. Library, tel. 558-5628.

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