Editor’s note: At the time Dr. Moyad wrote this column, FDA approval of Provenge was still undecided. On May 9th, Dendreon announced that it received a “Complete Response Letter,” commonly known as an “approvable” letter, from FDA regarding its Biologics License Application (BLA). However, the FDA requested additional clinical data to support the efficacy claim.

Sorry, no jokes, and no promotions in this column, but just a moment of serious reflection and my personal human or emotional opinion to guide me.

I use to love going to the hospital with my dad when I was a kid. I just wanted to hang out with him and figure out what he exactly did for his job so we could share that interest together. He was and is still is a urologist (best doctor I ever met), and I remember discussing prostate cancer with him and watching other doctors talk about it as if the magic bullet or “hope” was right around the corner. I really believe they thought that within 5-10 years there was going to be newer and more effective treatments offered, and some doctors really believed that in 10-15 years the disease itself would be wiped out. Keep in mind that this was over 30 years ago and now we have 1 drug (yes, 1 drug) approved for the extension of life for hormone-refractory prostate cancer (HRPC) patients, and patients are aware that it is an interesting and potentially effective medication (Taxotere®), but it is not an easy drug to take or stay on for a long period of time.

The bottom line is that it seems many of us, including myself, misjudged how difficult an enemy this HRPC
A SURVIVOR REVIEWS THE 2007 ASCO PROSTATE CANCER SYMPOSIUM

Commentary by Jerry Kattke, prostate cancer survivor and member of the Us TOO Palmieri Chapter in Lombard, IL

ASCO (the American Society of Clinical Oncology) has, for the third year, sponsored a symposium on prostate cancer, attended by oncologists, urologists, and radiologists. This year’s symposium was held in Orlando, FL from February 22-24.

The symposium consisted of 11 General Sessions, many Special Lectures, a series of Oral Abstracts, and a variety of poster presentations. I left the meeting with several strong impressions:

Several speakers mentioned the link between hormone therapy, diabetes, and heart disease.

Dr. Matthew R. Smith (Massachusetts General Hospital) said that men on hormone therapy typically lose 2-3 kilograms of muscle mass and gain the same weight in fat. He also said that men on hormone therapy frequently develop insulin resistance (i.e., needing more insulin than normal to burn sugar) and they develop elevated levels of triglycerides and cholesterol.

The message: we need to watch our diets, weight, blood sugar, triglycerides, and cholesterol, and keep our exercise levels high. This message was reinforced by Dr. Henry Tsai (Harvard Medical School) who did a study on hormone therapy and cardiac mortality using the CaPSURE database, and Dr. Derek Raghaavan (Cleveland Clinic).

The robotic-assisted laparoscopic prostatectomy is rapidly becoming the “standard of care” for those who choose surgery.

IMRT (Intensity Modulated Radiation Therapy) is the “standard of care” for external beam radiation, but there remains an important place for brachytherapy (radioactive seed implants). Docoetaxel is the “standard of care” for those who need chemotherapy.

For older men deemed unsuitable for definitive local therapy, there is only a small difference in survival between those who start hormone therapy early and those who wait. To balance quality of life against survival, Dr. Studer (University Hospital of Bern, Switzerland) recommends starting hormone therapy when the PSA reaches 50 or when the PSA doubling time drops below 12 months.

There were other points of interest from the symposium. Some of the new therapies being investigated are abiraterone, BMS641988, HSP90, and AZ2171. Dr. Tomasz Beer (Oregon Health and Science University), who is running a clinical trial on vitamin D, provided some interesting data from Norway correlating the diagnosis of prostate cancer with sunlight. Dr. Raghaavan (mentioned earlier) made a plea for more US government funding for clinical trials; he said that we now finally have younger physicians who are trained to conduct trials properly and new drugs on the horizon, but funding has been diverted to “other uses.”

Finally, I found the talk by Dr. Hamdy (University of Sheffield, UK) quite interesting. He reported that a group was commissioned on the UK 20 years ago with the task of predicting the future of cancer treatment. They were absolutely correct in predicting that significant progress would be made in treating breast, prostate, and ovarian cancers, but that there would be little progress with lung, liver, stomach, pancreas, or brain cancers.

They were incorrect in predicting that progress in prostate cancer would come from improved therapies; progress has, in fact, been because of earlier detection due to the PSA test. But Dr. Hamdy said that PSA testing has caused its own problems; we are significantly overtreating prostate cancer; he called it “harvesting prostates.” He said that if we biopsy every man with a PSA of 2.5 or more we will do 15 times as many prostatectomies as we do today, and the majority of these will be what he called “indolent” disease, not requiring treatment; the pa-

(Continued on page 4)
UCSD Cancer Researchers Report Ability to Detect Cancer at Earliest, Curable Stage

Researchers at the Moores Cancer Center at the University of California, San Diego report that they have developed a new method for detecting cancer very early in its development, when it consists of just a few cells. The best existing detection methods are not able to detect a tumor until it consists of about one million cells.

The paper, published in the April 18 issue of the online journal PLoS ONE, describes a series of proof-of-concept experiments in which the researchers, working with a prostate cancer cell line, were able to select out and amplify tiny amounts of cancer-causing DNA in the presence of more than 99.9 percent of normal DNA. Current methods for identifying mutant DNA would not work in clinical settings because they require isolation of relatively pure cancer cells. This is not feasible for clinical samples, which typically contain large amounts of the person’s normal cells.

“We have developed a new technology for very early detection of virtually any type of solid-tumor cancer based upon damaged DNA, which is where all cancers begin,” said co-author Dennis A. Carson, M.D., professor of medicine and director of the Moores Cancer Center. “We are now working with engineers toward the fabrication of the clinical devices that will enable this to be widely used in patients.”

Carson said they are several years away from clinical testing, but ultimately individuals will be able to be screened for DNA markers of cancer cells using simple clinical samples such as blood or urine. Using this same technology, physicians will be able to easily and inexpensively monitor the status of patients by looking for the DNA markers. If the treatment worked, there would be no mutated DNA and the patient would be cured. Such monitoring would also shorten the time needed to determine if the treatment is not working so another approach could be instituted.

The technology, called Primer Approximation Multiplex PCR (PAMP), is based upon an enzyme reaction that only works when a piece of DNA has been deleted or is abnormally joined to another piece of DNA, according to co-author Yu-Tsung Liu, MD, PhD, assistant project scientist at the Moores Cancer Center.

Liu explains: “When a cancer cell mutates, it often brings together two pieces of DNA that are normally apart. We have developed an enzyme reaction that works well only when two DNA pieces that are normally separated are close together. This technology amplifies the mutant DNA and then uses a microarray to identify the specific mutation. Our experiments were conducted on a specific gene mutation that is well-known for its role in cancer, called CDKN2A, but this technology would work on any DNA abnormality.”

For more information, please call Nancy Stringer at Moores Cancer Center at (619) 543-6163 or by e-mail at <nstringer@ucsd.edu>.

BUSINESS WIRE, 18 April 2007
POSSIBLE NEW CANCER TREATMENT?

Antifungal Drug Stops Blood Vessel Growth

Researchers at Johns Hopkins have discovered to their surprise that a drug commonly used to treat toenail fungus can also block angiogenesis, the growth of new blood vessels commonly seen in cancers. The drug, itraconazole, already is FDA approved which may fast-track its use as an antiangiogenesis drug.

In mice induced to have excess blood vessel growth, treatment with itraconazole reduced blood vessel growth by 67 percent compared to placebo. “We were surprised, to say the least, that itraconazole popped up as a potential blocker of angiogenesis,” says Jun O. Liu, Ph.D., professor of pharmacology. “We couldn't have predicted that an antifungal drug would have such a role.”

The researchers worked with cells from human umbilical cords, a rich source of blood vessels, and exposed them to 2,400 existing drugs - including FDA- and foreign-approved drugs, as well as nonapproved drugs that had passed safety trials - to see which ones could stop the cells from dividing.

“The best outcome was to find an already approved drug that worked, and the fact that we did was very satisfying,” says Liu, whose study appears online in ACS Chemical Biology.

As an antifungal drug, itraconazole blocks a key enzyme for making fungal cholesterol, causing these primitive life forms to become fragile and break apart. It turns out that itraconazole can block the same enzyme in blood vessels, but researchers said that this can’t be the only mechanism of action because related antifungal drugs had a much lower inhibitory effect.

“Our screening test did show that cholesterol-lowering statins also appear to stop blood vessel growth,” Liu says, “so there is likely some important connection between cholesterol and angiogenesis.” While the researchers still must tease out exactly how itraconazole works to stop vessel growth, and test it in animals with cancer, they have high hopes for its use.

Science Daily, 27 April 2007

RESULTS WITH GVAX

(Continued from page 1)

identified, and many of these proteins had not been identified previously as prostate cancer-associated antigens.

GVAX is currently being studied both as a single agent and in combination with docetaxel in two Phase III clinical trials for patients with metastatic hormone-refractory prostate cancer. This Phase III program is supported by results from two, independent, multicenter, Phase II clinical trials.

GVAX is composed of two prostate cancer cell lines that have been modified to secrete granulocyte-macrophage colony stimulating factor, an immune stimulatory hormone, and irradiated for safety.

FDAnews Drug Pipeline Alert 18 April 2007

PROSTATE SYMPOSIUM

(Continued from page 2)

Patient would live out his life never significantly bothered by the cancer. His prediction: we will soon find new biological markers that will enable us to identify the cases of “indolent disease” (which will not be treated) and we will treat the remainder much as we do today, except that robotics will continue to take a greater role, and that treatment will continue to migrate toward a few comprehensive cancer centers and surgeons who will do a large number of prostatectomies and do them very well.

If you have high speed internet access, you may wish to consider looking at the video of these presentations: they can all be found on the ASCO website under “Virtual Meeting.”

INCREASE PROSTATE CANCER AWARENESS IN THE WORKPLACE!

Celebrate

SNEAKERS@WORK

Day on June 15th

Bob Bogan (right) and Norm Thoresen (left) lead Sneakers@Work Day activities for the Zablocki VA Medical Center of Milwaukee, WI Us TOO Chapter.
EPCA-2 BLOOD TEST
(Continued from page 3)

- Men with prostate cancer confined to the prostate.
- Men with prostate cancer spreading outside the gland at surgery.
- A diverse group of patients with benign conditions of other organs as well as those with other cancer types.

In patients with an EPCA-2 cutoff level ≥ 30 ng/mL considered at risk for prostate cancer, EPCA-2 had 92% specificity (95% CI, 85% – 96%) for healthy men and those with benign prostatic hyperplasia and 94% sensitivity (95% CI, 93% – 99%) for overall prostate cancer. The specificity for PSA in these groups of patients was only 65% (95% CI, 55% – 75%).

The investigators report, “The results of our study have shown that EPCA-2 is a novel biomarker associated with prostate cancer that has high sensitivity and specificity and accurately differentiates between men with organ-confined and non–organ-confined disease.”

Medscape Medical News, 27 April 2007

COPPER DRUG PROMISING IN FIGHTING CANCER

Researchers from the Barbara Ann Karmanos Cancer Institute in Detroit, MI presented strong evidence today that an antibiotic typically prescribed for Alzheimer’s patients could be effective in eventually treating certain types of cancers.

Q. Ping Dou, PhD, leader, Prevention Program at Karmanos, and members of his lab announced the findings of their study showing that the drug Cloquinol (CQ) appears to have an anti-tumor effect in mice bearing human prostate cancer cells. The announcement was made at the American Association of Cancer Research (AACR) annual meeting in Los Angeles, CA.

Dr. Dou and his team turned to the copper-binding compound CQ after discovering that prostate, breast, brain, colon and lung tumor tissue often have a higher level of copper than normal tissue. According to Dr. Dou, this led his team to investigate whether cancers with high levels of copper can

(Continued on page 6)

DOCTOR MOYAD: WHAT’S UP WITH PROVENGE®
(continued from page 1)

really is when you consider all the compounds that have been thrown at it over the past 3 decades. Think about it for a second, hundreds or perhaps thousands of clinical trials and only one drug approved. Hundreds and really thousands upon thousands of lives and only one drug approved.

Well, one could argue that it was a funding issue and I am sure part of this is true because I still think cancer funding is so pathetically funded in this country that sometimes I am even surprised that one drug got approved. However, we also need to be honest and admit that one cannot argue that thousands of men have volunteered to be part of clinical trials in the past because it represented the right thing to do and it represented “hope” to us, and them. The hope that they could live longer to do more things they wanted to do, the hope of spending just a little more time with their families, and the hope that future generations would die of other diseases at older ages and not of prostate cancer. Finally, I believe they trusted and hoped that their doctor would do the right thing for them and future patients and this is how and why many of them were recruited to be in these trials.

Provenge was overwhelmingly recommended to be approved by the FDA advisory panel in March and simply put it was a democracy and the FDA almost always follows this democratic recommendation. Obviously the approval caught some by surprise and evoked an almost visceral and at times unusual scientific opposition to approval with most comments revolving around the fact that more data was needed. Yes, the science of medicine has been so well represented over the past several months with p-values, statistical significance and words like “time to progression” being thrown out as reasons as to why it should not be approved. In all the discussions I never really heard much about the word “hope,” “patients,” and the “past track record” in treating this disease. In other words, the science of medicine was well represented but who represented the “human” side of medicine in this controversy?

Let’s say that in 3-5 years the vaccine is completely ineffective what will have been lost except a tremendous gain of even more immunologic knowledge? Patients are not children that need to be completely protected by doctors at every stage of life or at every stage of treatment. HRPC patients understand at this point of their disease the general outcome and what is and is not available to them. It is time to respect the fact that patients want some new options or new directions because this at least represents progress. To deny patients the “option” to try this vaccine is to be almost completely ignorant of the past and current options that patients have today for HRPC.

Urologists and oncologists prescribe hundreds if not thousands of second-line hormonal therapies for advanced patients daily but NONE of these drugs have ever been FDA approved for extending life. Why? One reason is because of the lack of options at this stage of the disease and doctors want to at least attempt to extend the quality or quantity of life despite not having FDA approval with these medications, but this is exactly what makes doctors so wonderfully “human” and not just 100% science oriented.

Patients deserve better and instead of opening up the floodgates even more than they are opened to vitamin clinics in Mexico City, the Dominican Republic, and a host of other countries, I sincerely hope that by the time you read this column the vaccine will now be available to all patients with HRPC. If Provenge is delayed or rejected for several years than not only will I be disappointed but I have to believe some of the opposition to this vaccine will have allowed the science of medicine to completely cloud the human side of medicine. Patients have rights, they deserve better, and they should be allowed a voice especially when so many have sacrificed up to this point with the hope that we would deliver

(Continued on page 8)
lead to targeted treatment. Karmanos researchers found that after binding 
CQ to the copper in prostate tumor tissue, the drug induces cell death in human prostate cancer cells.

According to Dr. Dou, his lab will take this information and work with 
Karmanos clinicians to create a treatment selectively targeting the copper found in tumors. “We want to find out if we can target cancers that express high levels of copper,” says Dr. Dou. “We hope to discover a compound that promotes tumor-killing activity.”

Based in midtown Detroit, MI, the 
Barbara Ann Karmanos Cancer Institute is one of 39 National Cancer Institute-designated comprehensive cancer centers in the United States. It cares for more than 6,000 new patients annually on a budget of $216 million, 
conducting more than 700 cancer-specific scientific investigation programs and clinical trials. Through the commitment of 1,000 staff, including nearly 300 faculty members, and supported by thousands of volunteer and financial donors, the Institute strives to prevent, detect and eradicate all forms of cancer. John C. Ruckdeschel, M.D. is the Institute's president and CEO.

PRNewswire, 17 April 2007

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**COPPER TARGETS CANCER**

(Continued from page 5)

**ZOLEDRONIC ACID FOR PROSTATE CANCER BONE METASTASIS CARRIES RISK OF RENAL TOXICITY**

Renal impairment occurs in onequarter of patients with hormone-refractory prostate cancer (HRPC) who are treated with zoledronic acid for bone metastasis, Boston researchers report. They say these phase IV findings indicate that the risk of nephrotoxicity with the bisphosphonate is higher in an outpatient clinical setting than was seen in earlier trials.

A team at the Dana Farber Cancer Institute in Boston, Massachusetts, conducted a review of the medical records of 122 patients with HRPC and bone metastases who were treated with zoledronic acid at their institution between December 1999 and April 2005. Renal impairment was found in 23.8% of patients.

Risk varied according to length of 
treatment or previous treatment with pamidronate, Dr. William K. Oh and colleagues report in the March 15th issue of the journal Cancer (vol. 109, pp. 1090-6, 2007).

Specifically, renal impairment 
ocurred in 11.1% of patients on zoledronic acid for less than 6 months but in 26.3% of patients on the agent for 24 months or longer. Signs of nephro-
toxicity were seen in 45.5% of patients previously treated with pamidronate but in only 19.0% of patients with no history of pamidronate therapy.

Increasing age, a history of hypertension, smoking and renal impairment were other risk factors for nephrotoxicity. Impairment was grade 1 in about half of cases. Renal impairment accounted for drug discontinuation in 20.8% of patients.

“Given the evidence about the nephrotoxicity of zoledronic acid, the outcomes of ongoing clinical trials may be important,” the Dana Farber researchers conclude.

“Because newer anticancer therapies may improve patient survival, it will be important to evaluate whether increased exposure to zoledronic acid with other potentially nephrotoxic medications increases the likelihood of renal toxicity” in this patient population.

Reuters Health, 9 April 2007

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**STRATIFICATION OF PATIENT RISK BASED ON PROSTATE-SPECIFIC ANTIGEN DOUBLING TIME AFTER RADICAL RETROPUBIC PROSTATECTOMY**

Tollefson MK, Slezak JM, Leibovich BC, Zincke H, Blute ML


**OBJECTIVE:**

To assess the risk of local recurrence, systemic progression, and death from cancer among patients who experience biochemical relapse (BCR) after radical retropubic prostatectomy and to stratify those patients by prostate-specific antigen (PSA) doubling time (DT).

**PATIENTS AND METHODS:**

We identified patients who experienced 
BCR (defined as a PSA level <0.4 ng/ 
ml), after radical prostatectomy from 
January 1, 1990, to December 31, 1999, for prostate adenocarcinoma. The PSA-DT was calculated by log linear regression using all PSA values within 2 years of BCR. Local recurrence- and systemic progression- free survival and cancer-specific survival were estimated using the Kaplan-Meier method and analyzed by the log-rank test and Cox models.

**RESULTS:**

BCR was noted in 1,521 (27%) of 
5,533 men during the follow-up period. 
Of the 1,064 patients with a calculable 
PSA-DT, 322 (30%) had a PSA-DT of 
less than 1 year, 357 (34%) had a PSA- 
DT of 1 to 9.9 years, and 385 (36%) 
had a PSA-DT of 10 years or more. 
Patients with a PSA-DT of 10 years or 
more were less likely to have a higher 
prostate-specific antigen level, Gleason score, advanced pathologic stage, and seminal vesicle invasion. Patients with a PSA-DT of 10 years or more were at low risk of local recurrence (hazard ratio [HR], 0.09; 95% confidence interval [CI], 0.06-0.14; compared with patients with a PSA-DT of <1 year), systemic progression (HR, 0.05; 95% CI, 0.02-0.13), or death from cancer (HR, 0.15; 95% CI, 0.05-0.43).

**CONCLUSIONS:**

PSA-DT is an independent predictor of clinical disease recurrence and mortality after surgical BCR. Risk stratification into high-, intermediate-, and low-risk categories based on the PSA-DT provides helpful clinical information and assists in the development of salvage therapy trials.
FROM THE DOCTOR: PHYSICIAN COMMENTARY ON SELECTED ARTICLES IN THIS MONTH’S HotSheet
By Gerald W. Chodak, MD
This month’s HotSheet has many new and interesting articles starting with the potential for a new test that may function better than PSA. The test measures EPCA-2 and in preliminary studies may allow more men to avoid unnecessary biopsies while also identifying those men whose cancer has spread. It is too early to establish, however, if it will indeed replace the PSA test but a number of questions must be answered before we make the substitution. First, the test is not perfect meaning that by avoiding more unnecessary biopsies, more cancers will be missed and by incorrectly telling some men they have incurable cancers, some will miss out from potentially curative therapy.

Developing new screening tests is a most complex process. We will have to wait longer to see the true impact of the EPCA-2 blood test.

Dr. Moyad’s column addresses some of the frustrations associated with the slow pace of progress in treating advanced prostate cancer. As previously commented on, the Provenge vaccine may be an important new development for some men, but the final answer is not in. One of the questions to answer is which patients should be treated? Should it be limited to those men with disease similar to those in the study or will the attitude be give to all men even those not studied because “maybe it will help.” The danger in opening the floodgate is that the company already has many new articles starting with the potential for a new test that may function better than PSA. The test measures EPCA-2 and in preliminary studies may allow more men to avoid unnecessary biopsies while also identifying those men whose cancer has spread. It is too early to establish, however, if it will indeed replace the PSA test but a number of questions must be answered before we make the substitution. First, the test is not perfect meaning that by avoiding more unnecessary biopsies, more cancers will be missed and by incorrectly telling some men they have incurable cancers, some will miss out from potentially curative therapy.

Another test even further away from prime-time use is the DNA methodology described by Dr. Liu called PAMP. This sounds somewhat similar to a previous methodology that failed to meet up to expectations. Little can be said at this time about its eventual effectiveness and here too, more data will be anxiously awaited. A number of the articles address new approaches to treatment. Two are most relevant to patient care today. Investigators at the Mayo clinic found that how fast the PSA increased after local therapy (PSA doubling time) was administered determined which patients were at risk from their prostate cancer.

This information is very important because it coincides with other studies that found a slowly rising PSA does not convey much risk to a patient and therefore immediate therapy is not necessary. This potentially could enable many men to avoid unnecessary treatment even when their PSA is rising.

Another study with potential relevance to men being treated for advanced disease is the one from Boston showing that nearly 25% of men on Zoledronic acid (Zometa®) developed kidney impairment during treatment. The importance of this finding is that patients on this treatment need to be carefully monitored and the drug must be administered slowly to avoid potential kidney injury.

A summary of studies from this year’s ASCO meeting addresses the latest on robotic prostatectomy. The best analysis at this time is that it is not the technique that matters, it is the surgeon. Excellent surgeons get excellent results regardless of the technique.

A number of the studies involved very preliminary findings from laboratory studies. One study found that an FDA approved anti-fungal drug appears to block new blood vessel growth, a critical requirement needed for tumor growth. The drug is called Itraconazole and it has similar properties to zole and it has similar properties to growth. The drug is called Itraconazole and it has similar properties to growth. The drug is called Itraconazole and it has similar properties to growth. The drug is called Itraconazole and it has similar properties to growth. The drug is called Itraconazole and it has similar properties to growth. The drug is called Itraconazole and it has similar properties to growth.

A new protein-targeted drug inhibited growth of human lung and colon cancer cells. Integrating the use of drugs targeted to specific cancer proteins into current chemotherapy regimens to improve the efficacy of systemic treatment is an important clinical goal at Fox Chase Cancer Center.

This synergistic effect—in which the effect of two agents is greater than the sum of their individual effects—appeared when using the combination of MCP110 and Taxol on laboratory cell cultures of human Kaposi’s sarcoma and mouse models carrying human lung and colon cancer cells.

Vladimir Khazak, PhD, director of biology at NexusPharma, Inc., in Langhorne, PA and molecular biologist Erica A. Golemis, PhD presented the research in an AACR poster session. The work also appears in the March 1 issue of the AACR journal Molecular Cancer Therapeutics.

MCP compounds were demonstrated to have the ability to inhibit growth of cultured cancer cells that depend on interactions of the Ras and Raf oncoproteins—growth-promoting genes that can transform cells to cancerous ones if the oncogene is activated inappropriately. The growth signals sent by these oncogenes use an enzyme pathway called MAPK (mitogen-activated protein kinase). Several widely used drugs, including paclitaxel (Taxol®), docetaxel (Taxotere®) and long-time standby vincristine work through this mechanism and inhibit cell division by blocking microtubule formation.

“Combination chemotherapies may use two drugs that either have the same target or two different targets said Golemis. Another approach—the one we’ve taken here—is to combine a pathway-targeted drug with conventional chemotherapy.

“Together, these findings indicate that MCP compounds have potential to be effective in combination with other anticancer agents,” they concluded.

Science Daily, 16 April 2007

CHEMOTHERAPY EFFICACY ENHANCEMENT: NEW AGENT HAS SYNERGISTIC EFFECT WITH STANDARD DRUGS

(Continued on page 8)
on the promise of allowing treatments with less side effects for patients that have so few options.

It is hypocritical to aggressively advertise and offer mechanical devices or other “less invasive therapies” at almost every medical institution in this country with no randomized trials but not to allow a potentially life extending therapy for many dying patients because not enough men have gone through enough randomized trials. Well, some may say that two wrongs do not make a right, but what is so wrong in my opinion was that a democracy clearly recommended approval but a handful do not want to believe that this opinion of the majority was correct. Perhaps if this same energy or passion in the opposition for Provenge was redirected to another area of prostate cancer it may play a small part in my ability to tell my son the next time he comes to the hospital with me that daddy thinks there will be no prostate cancer by the time he needs a PSA test, but this time it may turn out to be true. No offense to my father of course who I know in his heart really believed we were going to cure or control this thing already in his lifetime, but I guess what my dad never figured on was that the science of the disease might replace the art or human side of medicine.

Please do not mention the dollars, or the p-values, or the time to progression or whether or not this side effect was found at 5% versus 3%, or the degrees or number of publications because we have already endlessly debated those issues and that is what an FDA recommendation panel has already debated quite well. Instead, please now mention the thousands of patients that we have recruited in clinical trials because we sold them and recruited them on “hope” in my opinion, and for what, only to see one drug approved in my 42 years of life; and for God (yes, I meant to use this word) sake bring back the human side of medicine because it is desperately needed in this debate (otherwise you might as well allow a machine take care of HRPC patients).

Reference:
The thousands of “hopeful” patients that I have met over the past 30 years.

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**US TOO INTERNATIONAL: OUR MISSION**

Communicate timely, personalized and reliable information enabling informed choices regarding detection and treatment of prostate cancer.

**DOC MOYAD’S COLUMN (continued from page 5)**

An additional FDA approved drug that may have a role in prostate cancer therapy is Clioquinol. This drug binds copper which is found in high concentration in some tumors. As a first step, Dr. Dhou and his co-workers in Detroit found that this drug caused prostate cancer cells growing in a laboratory dish to die. Considerable time will be needed to further explore if it has a potential role in prostate cancer patients.

Lastly, MCP 110 is yet another new agent with an ability to kill cancer cells growing in a dish. The drug appears to work in conjunction with traditional chemotherapy but at this time, its effect on prostate cancer cells is unknown.

**FROM THE DOCTOR**

(Continued from page 7)

another antifungal agent, ketoconazole, which for years has been used as a second line treatment for prostate cancer after castration is no longer effective. Clinical studies will be needed to determine if it has a role in prostate cancer patients.

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