Adding Prednisone to Zytiga – More Side Effects?

Zytiga (abiraterone acetate)

I have heard a few prostate cancer patients comment that they are worried about taking Zytiga (abiraterone) because they are concerned about the side effects of the 5 mg prednisone (2 x day) which is prescribed with the Zytiga. People who have been on prednisone for other reasons (gout, arthritis, etc) may have had prednisone side effects which might make them hesitate to try Zytiga – just for this reason.

However, when prednisone is given with Zytiga, it's given for a different reason. One of the purposes is to actually LESSEN side effects from the Zytiga treatment. Simply put, Zytiga (abiraterone) can actually lower blood levels of cortisol in many patients, so the prednisone is considered cortisol "replacement", which can help reduce side effects from Zytiga treatment. This is a much different situation than giving prednisone alone for other reasons such as gout or arthritis.

An article in December 2014 of The Oncologist says it this way, "…glucocorticoid compensates for abiraterone-induced reductions in serum cortisol and blocks the compensatory increase in adrenocorticotropic hormone seen with abiraterone.

Consequently, **5 mg prednisone twice daily serves as a glucocorticoid replacement therapy when coadministered with abiraterone acetate…"**

Dr Leonard Gomella also discusses the issue in **this online video**, *"I think you can safely say that low dose of prednisone does not cause any specific corticosteroid toxocity…"*

Since prednisone administration is clearly different than prednisone prescribed for other reasons, talk to your pharmacist if you have any concerns. Zytiga is usually administered through a Specialty Pharmacy, not a retail or neighborhood pharmacy. Talk to a pharmacist at one of these locations if you have any questions. Make sure the information you are working from is correct as you make your ongoing treatment decisions for your prostate cancer.

Also, if you would like to receive a **copy of one of our new brochures called "Ask Your Pharmacist (too…)",** please email PAACT at **paact@paact.help with your request and mailing address.**

(The brochure is completely free,
but donations are always appreciated if possible.)

AR-V7 Blood Test now Available – for mCRPC Patients

The AR-V7 blood test may help patients and doctors make decisions on treatment choices when prostate cancer patients are **metastatic, and castrate resistant**, or **mCRPC.** The term AR-V7 stands for androgen-receptor splice variant 7. This blood test recently became available to the public through Johns Hopkins' Molecular Diagnostics Lab.

No blood test is perfect, but **having a negative AR-V7 test** has shown the following in a study of 62 men, published in New England Journal of Medicine Sept 2014:

- **Better PSA response to Zytiga (abiraterone) and/or Xtandi (enzalutamide)**
- **Better progression-free survival and overall survival when taking Zytiga (abiraterone) and/or Xtandi**

In another **small study (37 men) published in JAMA Oncology June 2015**, having a **positive AR-V7 test did not hinder men from responding to chemotherapy**, specifically Taxotere (docetexel) and/or Jevtana (cabazitaxel). In addition, it was noted that *"certain patients with detectable AR-V7 at baseline converted to AR-V7 negative status during the course of taxane*

therapy [chemo].” There are no promises that a man’s AR-V7 test can be improved (from positive to negative) after doing Taxotere or Jevtana, but at a dinner presentation at ASCO 2016, Dr Antonarakis stated that so far, they have seen this in *“about 50% of patients.”*

Dr Dan Petrylak also discusses AR-V7 in this short (2016) video.

IMPORTANT – The AR-V7 blood test **may or may not be covered by your insurance. If it is NOT covered by insurance, the cost is **approximately $1,000** right now. It is advised that you call your insurance company and ask about insurance coverage. It is essential to give your doctor the right CPT code to write on the order – See Step 7 below.*

As a patient or caregiver, you will need to do the following to obtain an AR-V7 test:

1. Verify that your prostate cancer is **“metastatic, castrate-resistant.”**
2. **Print this requisition form** from Johns Hopkins lab, and show to your doctor. **Ask your doctor if he will write the order/script for the test. (The doctor who writes the script for the AR-V7 test *CANNOT be in FL or NY*.)**
3. **Find a local lab who is willing to draw the blood for you**, according to detailed instructions on AR-V7 requisition form. (tip – Medical oncologists usually have their own lab for blood draw, in their clinic.)
4. There are **special FedEx refrigerated shipping instructions you have to follow.** The FedEx package must also **arrive at Johns Hopkins lab before 10:00 am the next day** (not in the afternoon). *If the lab who draws your blood cannot do this shipping procedure, you may*

have to do it and pay for it yourself. For further questions on this shipping to JH lab, you can contact them at molecularpathresults@jhmi.edu or (410) 955-1438. Hours are 8:30 am – 5:30 pm EST, M-F.

5. Since the blood needs to be received by Johns Hopkins the next morning, you cannot draw your blood at your local lab on a Friday. **Make sure your AR-V7 blood test is drawn Mon – Thu.**
6. The turnaround time can vary, but test results are usually released to your doctor in 1 – 2 weeks. **Ask for your own copy.**
7. For information on the ordering code (CPT code), call the JH lab at (410) 955-1438.

*For more information on this article, call Jan Manarite of PAACT at her home office – (239) 208-4400, or email her at JManarite@paact.help

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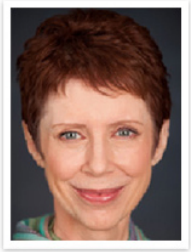
How Fear Affects Cancer Survivorship – Jesse Gruman PhD, 2013

by JESSIE GRUMAN, PHD | PATIENT



A recent Wall Street Journal article about how post-traumatic stress syndrome can be caused by cancer and stroke brought to mind the variety of responses many people experience in response to cancer diagnosis and treatment. Th e lingering intensity of those responses – physical, psychological, social and behavioral – can affect whether and how we attend to the tasks of survivorship; that is, monitoring and addressing the unique health challenges that follow treatment for cancer.

Sam, a friend of mine, told me that his anxiety is starting to rev up about his annual scan to check for a recurrence of his esophageal cancer. It's early July. His appointment is in mid-September. He doesn't want to go. He will force himself to go. He will worry more each day as the test date approaches.

The sound technician for a recent talk I gave recounted how, 17 years after his radical prostatectomy, he insists on having his PSA tested every six months, despite the one year interval recommended by the guideline. "From the time the blood is drawn to when I get the results I'm still a wreck. And in between tests, my worry is like a pebble in my shoe. It's small, but it's always there."

Some of us are able to approach our survivorship care as just the next few necessary chores. Others have had enough of the cancer experience by the time we have finished treatment: we refuse to participate in any monitoring or testing at all. Some of us – like Sam – muscle through: constantly surfing the waves of worry.

And some of us take matters into our own hands. Like the sound technician above, we insist on surgery or medication now or we demand more frequent testing than is recommended. We devise our own dietary, physical and mental regimens and employ a range of alternative medicine approaches – sometimes substituting them for standard medical approaches – in an

effort to reduce our apprehension and to reclaim some sense that we can control our future.

I wish I had known earlier that a strong emotional response to cancer treatment is fairly common.

I recall becoming nauseous at the prospect of walking into a hospital (any hospital!) and the build-up of crushing fear in the days before getting a simple PAP test. At first these responses kept me far away from any follow-up care. Then when my fear of a recurrence exceeded my fear of testing for a recurrence, I found myself panicking prior to every checkup, every test. I believed these were rational responses to the highly toxic, aggressive treatment and callous care of an adolescent surprised by a diagnosis of Hodgkin's lymphoma and the threat of impending death at age 19.

I wish I had known earlier that there was no need to suffer so much or so long from these lingering fears.

Talking with others who experience similar anxieties might have made it seem more normal. A behavioral intervention by a mental health professional could have drained some of the anxiety.

As control of pain and nausea become more effective, perhaps fewer of us will experience such responses. But the effect of a cancer treatment affects each of us differently. Increased recognition by our clinicians of its potential impact and help finding effective approaches to accommodating our new reality can help to calm the waves of emotion that get in the way of returning to the lives we love.

Cancer treatment can affect physical, emotional, cognitive, social, behavioral and occupational aspects of our lives. Survivorship care by definition is care of the whole person. It sometimes takes my breath away that my own fear

could easily have stood in the way of the discovery and treatment
of my four subsequent cancers. I wish I had known earlier how easy it would be to undermine
the possibility of benefitting fully both from the treatment I received and the recommended monitoring and testing because I couldn't see that I needed help with my fear.

Jessie Gruman is the founder and president, Center for Advancing Health. She is the author of Aftershock: What to Do When You or Someone you Love is Diagnosed with a Devastating Diagnosis. She blogs regularly on the Prepared Patient Blog.

Men Who Speak Up: An Awareness Campaign from Survey Results – Jan Manarite and Rick Profit, 2015

Thank you to all the men and caregivers who took the time to participate in the recent IPCC/Bayer survey about advanced prostate cancer & its symptoms. Your involvement allowed us to hear you, and gather statistics to create awareness about some of the problems when prostate cancer patients don't discuss their pain, fatigue and other symptoms. The results of the survey also illustrated how caregivers

perceive the patient's pain and symptoms differently, and communicate differently. A website has been launched where you can view illustrated results of the survey, a doctor's discussion guide for advanced prostate cancer patients, and more.

One statistic that struck me was that 71% of men said that sometimes they didn't know what was causing their symptoms. But that was also about the same percentage of men (70%) who were reported "ignoring" their symptoms **(the most common symptom is pain)** What this reminded me of, was that men often experience joint pain at the onset of hormone therapy, but this pain is usually from arthritis exacerbated by the hormone therapy – not a cancer related pain. So there is a difference between symptoms from the cancer, and side effects from the cancer treatment. Discussing your pain in more depth with your nurses or physicians should empower them to help you understand the difference between the two. It is often a relief to know that your pain is not a symptom of your cancer.

On the other hand, if it is a symptom of your cancer, you may have other cancer treatments available to you (besides simple pain medications). Th is is where the conversation is important
between patient and physician.

So, thank you to all the caregivers and patients who took time to answer these survey questions, either online or by phone. We hope this Men Who Speak Up campaign brings more awareness, conversation and results to men who are battling advanced prostate cancer. We also hope you'll find

something useful on the Men Who Speak Up website. PAACT is grateful to have been a part of this project along with members of the IPCC (International Prostate Cancer Coalition).

Watch this **short video of Jan Manarite's interview** – *"We now have some treatments that can be given to you BECAUSE you have pain…"*

A Prostate Cancer Treatment that Worked for Me, by Doug F, 2013

Doug F lives in Michigan, USA. He was 67 when he was diagnosed in November, 2004. His initial PSA was 6.30 ng/ml, his Gleason Score was 9, and he was staged T3a. His choice of treatment was Surgery (Robotic Laparoscopic Prostatectomy).

Here is his story.

I started yearly PSA testing in 1995. My PSA stayed about the same for the first few years, then it started to climb. In 2004 after a biopsy (at the age of 67), I was diagnosed with a Gleason 4+4 and 4+5 prostate cancer. I then had a Radical Prostatectomy, followed by salvage radiation. Neither worked. It was already systemic prior to the removal of the prostate. I had advanced prostate cancer that was very aggressive. My PSA can double every 10 to 12 days if I'm not on some kind of treatment.

I went on hormone therapy after I found out it was systemic. This worked for only a few years. Eight months continuous, and

the rest of the time on intermittent therapy. I did Lupron by itself, and also Combination Androgen Blockade (CAB) with Casodex and Proscar. I then became refractory to Lupron. My PSA would go up even though I was at a castrate level. So, I started Estradiol patches. First on one patch .1mg, then followed by 3 patches, and ended up using 4 patches .4 mg. With 4 patches I was able to get my testosterone to lower than castrate levels. Sometimes better than when I was on Lupron. I was on the patches for 15 months and maintained an average PSA of 1.2 to 1.6. My highest Estradiol level during this time was over 500. This is a much higher Estradiol level than most women over the age of 45 would have. I knew if I wanted my PSA below 0.1ng/ml, I would have to use between 6 and 8 patches per week. This was a total pain… because they are very hard to keep on your body. I also would have to keep my Estradiol level somewhere over 700.

Then I decided to make an Estradiol compound that would replace the patches. Just rubbed the gel on my arm daily. It worked for me, but not as good as I wanted. I found that my math was horribly incorrect. I put the compound to rest for a while and will get back to making a new compound at a later date. I'm confident that the next time it will work much better than my first try did. Remember that the pharmaceutical companies would have run this trial for years with many patients. I ran it for 3 months with one patient, myself. Luckily, I was able to keep my PSA very low through the years….. Even though I had a very aggressive cancer, my highest PSA was 2.33 ng/ml for about a week. The rest of my PSA readings were below 2.0 ng/ml. This requires constant attention to keep it under control.

Going back to 2004: Prior to finding that I had prostate cancer, I met Harry Pinchot from PCRI (Prostate Cancer Research Institute) and Brad Guess (who at the time worked as a PA for Dr. Mark Scholz) for lunch. Most of the discussion at lunch was on prostate cancer. One of many things that I

learned from Harry that day was…

• You have to know more about treating your prostate cancer, than the doctor treating you.

• The other thing that I learned from him was you had to do a lot of studying in order to stay ahead of the game.

He felt that many new drugs and treatments would be coming available in the near future. “And he was sure right on that comment.”

Harry and I had many interesting discussions over a four year period. He was the most knowledgeable lay person I had ever met anywhere in the country on prostate cancer. It was a major loss to prostate cancer patients when he passed away in January of 2008.

As Harry said, do a lot of studying. So I started studying advanced prostate cancer for the next 8 years. I still average 30 to 40 hours per week looking at a computer screen. I am also very involved with two support groups in Michigan. I either give a talk or show talks given by doctors from around the country. I also talk to many PCa patients around the country on procedures and available drugs for treating their prostate cancer. I’ve been blessed by having a doctor that has allowed me to make my own decisions on how to treat myself since March of 2007. My first decision was when I decided to go on intermittent hormone therapy (we usually discuss the new protocol to make sure I don’t kill myself).

Dexamethasone is a drug that I have wanted to try for the last 3 to 4 years. I tried it for a month about a year ago. At that time, my T [testosterone] level was between 60 and 80. I found it will not work unless your T level is below 20, preferably as low as you can get it, like below 10. I wanted to prove that it wouldn’t work having a high Testosterone level.

For the time being I went back on Lupron, knowing that it

would not work for me. But that was OK……. all I wanted it to do was get my T level below 15. I added an Estradiol patch to assist the Lupron in lowering the T level… it worked.

The patch will usually reduce your T and PSA level. The patch will also reduce your hot flashes that are a pain for almost all of us on hormone therapy. Or you can use them alone to treat your cancer without any other drugs. They will also reduce bone loss. I have seen patients recover over 60% of their bone loss by using about 8 patches, but always check with your doctor first. So, then I started using Dexamethasone 1 mg per day along with Lupron to get a synergistic response, and it worked. In 30 days my PSA went from 1.63 down to 0.06. I also put on one patch .1 mg for two weeks to help keep my T level as low as I could get it. The next 30 days it dropped from 0.06 TO 0.007 (undetectable).

Knowing that Dexamethasone and Prednisone are in the same family of drugs, I decided to try Prednisone 7 mg for 30 days to see if it would work for me the same way that Dexamethasone did. But Dexamethasone has a couple pathways that Prednisone does not have. Just as I thought, it didn't work at all, even though my Testosterone level was below 10 ng/dL as a result.

On Jan 29, 2013 my PSA went from 0.007 up to 0.13 – quite a jump for 30 days. *Just a little information: (1.0 mg of Dexamethasone equals 6.6 mg of Prednisone).* I then went back on Dexamethasone, but this time I dropped the dose from 1.0 to 0.75 mg to see if a lower dose would work.

March 4-2013, the PSA dropped from 0.13 to 0.05 with a T level below 10. I would have to see how much energy I still had left with a dexamethasone dose of 0.75 mg. One Estradiol patch was also used with this test. I used the same protocol until APRIL 8-2013, which was the last month from my 3-month Lupron shot.

On April 8, 2013 my PSA dropped from 0.05 to 0.01 PSA <0.01, T

level below 10. My next update and posting will take place after May 10, 2013. Stay tuned.

There are a couple of more ways that I'm going to treat myself over the next few months. They too will be posted once I finalize the course of action of exactly what I'm going to do. I ran a DHT [dihydrotestosterone] blood test to see what my DHT level is while using Dexamethasone. I knew it would drop it but wasn't expecting it to drop as much as it did. Normal levels are 119 to 719, mine went down to <2.50 or less. This proves to me that Dexamethasone will take care of DHT levels. Remember DHT is anywhere between 5 to 8 times more potent than normal Testosterone levels. This is something that all advanced patients should monitor periodically, in my view.

A WORD OF CAUTION

– Remember that Dexamethasone is a steroid. You will have to drop the dosage slowly when you decide to get off the drug. There are many people that have used both Dexamethasone and Prednisone that have had problems when they are trying to wean off the drug. Most have been on very high doses, and some have just failed to reduce the dosage slowly enough. My goal is to start at 1.0 mg, then drop to 0.75 mg, and then maybe to 0.50 mg. (From 1 mg and below is considered a low dosage).

Check with your doctor and pharmacist before you try using this drug because of the possibility of drug-drug interactions…….and also how they recommend weaning off of it.

UPDATED April 2013

1) April 8-2013 PSA my dropped from 0.05 to <0.01, T level below 10.

2) My next treatment will be two drugs only, 4 Estradiol patches (.4 mg to keep my T level low) and Dexamethasone .75 mg.

3) I will see how this new protocol works for me in about 30 days.

4) I have a couple more ways I'm going to treat myself over the next few months. I will post the details as soon as I know what I'm going to do.

UPDATED May 2013

Blood test results from May 8th were PSA 0.004 (T level below 10). My next treatment I will be using the same two drugs, but increasing the Dexamethasone from 075 mg to 1 mg. The reason I'm increasing the Dexamethasone is because I also have a disease called PMR that is normally treated with prednisone. I'm doing the two birds with one stone treatment. I will see if this protocol will continue to work for me over the next month or two. My thoughts as of now are to run this until it fails. As we all know, this treatment is just a temporary patch until the cancer outsmarts what I'm doing. If anyone tries treating themselves this way, let me know what kind of results you obtain. There have been 5 of us (that I know of) that have tried it. It worked on 3 and failed on 2.

*Doug's e-mail address is: wynz@comcast.net

Clarifying Concepts – Every Drug has 2 Names, Jan Manarite, 2015

Every Drug has 2 names. From antibiotics to chemotherapy, every drug has a brand name and a generic name. For the

patient, **this can be confusing**, especially when trying to research a treatment, which is essential in developing better questions, and essential in Shared-Decision Making with physicians. If we are going to 'share the decision,' we must share the language and share the information. Sometimes this is harder than it should be for the patient or caregiver. The table to the right is a tool you can use to help recognize both names of a treatment or drug as you are doing research, whether it is on the internet, or at a medical library. This list is not all-inclusive, but hopefully illustrates an important issue in your cancer research, which is that every drug has 2 names, and even a third name in its earlier clinical trial days. So don't let this confuse you – look to recognize both names, the generic name and the brand name. Don't be discouraged if you 'Google' one name, and find the other. As always, continue to research your ideas and questions before you ask your physician(s). A researched question is a better question. And a better question always gets you a better answer.

FOR SOME EXAMPLES OF TREATMENTS & DRUGS OFTEN USED IN BPH & PROSTATE CANCER PLEASE SEE THE CHART BELOW.

BRAND NAME	GENERIC NAME
Proscar	finasteride
Avodart	dutasteride
Jalyn	dutasteride + tamsulosin
Flomax	tamsulosin
Rapaflo	silodosin
Hytrin	terazosin
Casodex	bicalutamide
Eulexin	flutamide
Nilandron	nilutamide
Nizoral	ketoconazole
Firmagon	degarelix
Lupron	leuprolide acetate (intramuscular injection)
Eligard	leuprolide acetate (subcutaneous injection)
Trelstar	triptorelin pamoate
Zoladex	goserelin acetate

Zytiga	abiraterone acetate
Xtandi	enzalutamide (MDV3100 in trials)
PROVENGE	sipuleucel-T
Xofigo	radium 223 (alpharadin in trials)
Taxotere	docetaxel
Jevtana	cabazitaxel
Zometa	zoledronic Acid
XGEVA	denosumab (larger dose)
Prolia	denosumab (smaller dose)
Cipro	ciprofloxacin
Levaquin	levofloxacin
Tylenol	acetaminophen
Aleve	naproxen sodium
Advil, Motrin	ibuprofen
No brand name in U.S.	aspirin

Look for other upcoming articles on **Clarifying Concepts** to help patients and caregivers in their research, formulation of questions and Shared Decision Making with their physicians.

Which Doctor When?…. Jan Manarite, 2015

Which Doctor When?…

When I walked this prostate cancer journey with my husband for over 13 years, we were often confused by the different titles

that physicians had. Some sounded similar. Some doctors gave certain treatments – some didn't. And, of course, the instinct to change my husband's lead physician, plus the right to do so was sometimes overwhelming. We changed lead physicians 3 times during that 13 years, but landed with one who we could develop a good relationship with, and worked with him for over 9 years. But it was always easier for me to suggest a change than it was for my husband. It was his cancer at stake, his patient-doctor relationship. Changing doctors was scary, but it always paid off when we thoughtfully followed our instincts.

These **3 short articles** I've written come from those experiences, plus my 14 years as a prostate cancer advocate, writer and speaker**. You can find these articles and more online at PAACT's Blog – www.PAACT.help/Blog**

Changing Physicians?
Changing doctors was scary, but it always paid off when we *thoughtfully* followed our instincts.

(1) What's the Difference Between an Urologist and a Medical Oncologist? *And Which One Should I be Seeing?*

If you are a prostate cancer patient, the **urologist is** most likely the physician who did your biopsy, gave you your diagnosis, your Gleason Score and discussed your treatment options with you. He may have discussed prostate surgery (prostatectomy), especially since he/she is a surgeon. The urologist is the first doctor you see, deal with and probably develop a bond with since he delivered the message that you had cancer.

A **medical oncologist** is a cancer doctor who treats all types

of cancer. In some other cancers, he is the first doctor you see, not the surgeon. In prostate cancer, he is often the third doctor you see, after the urologist and the radiation oncologist. This can be confusing for patients. They may not know which one to see or if they even have a choice. I would argue that you always have a choice and ultimately it is always up to you, the patient. Period.

I walked the prostate cancer journey with my husband for 13 years. He was metastatic at diagnosis, so we saw a medical oncologist in the first week. In fact, he was so advanced that he never had a prostate biopsy. His diagnosis came from a metastatic tumor removed from his spine. So his case is completely different than most men. But I did learn to work through the system, trying to keep him and his cancer as the most important things.

Here are some **differences between most urologists and medical oncologists** that should help you decide who you would like to see for your prostate cancer care.

Differences Between an Urologist and a Medical Oncologist	**Urologist**	**Medical Oncologist**
Trained in *surgery*?	**Yes**	**No**
Trained in *internal medicine*?	**No**	**Yes**
Trained in *hematology*? (issues of the blood)	**No**	**Yes**
Trained in *pain management*?	**Usually No**	**Usually Yes**
Can administer hormone therapy shots?	**Yes**	**Yes**

Can administer chemotherapy?	**No**	**Yes**
Can administer Xgeva or Prolia?	**Yes**	**Yes**
Can write prescriptions?	**Yes**	**Yes**
Can check CBC bloodwork same day/in house?	**Usually no**	**Usually yes**
Has dedicated, clinical Nurse Practitioner or Physician Assistant?	**Usually no**	**Usually yes**

For further reference –

1. The Best Medical Team for Advanced Prostate Cancer Treatment. April 2014, Everyday Health; http://www.everydayhealth.com/health-report/conditions/how-pick-advanced-prostate-cancer-medical-team.aspx
2. Health Professionals Associated with Cancer Care. August 2014, American Cancer Society; http://www.cancer.org/treatment/findingandpayingfortreatment/choosingyourtreatmentteam/health-professionals-associated-with-cancer-care
3. I am not an Urologist. February 2014, Mark Scholz, MD; http://prostatesnatchers.blogspot.com/2014/02/i-am-not-urologist.html
4. Finding the prostate cancer specialist who is right for YOU. March 2011, The New Prostate Cancer Infolink; http://prostatecancerinfolink.net/tips-tools/pick-prostate-cancer-specialist

(2) What's the difference between a Radiologist and a Radiation Oncologist?

A **Radiologist** is a physician you probably never meet, yet

still impacts the understanding and treatment course of your cancer. He **reads and interprets your imaging** or your radiology exams. In prostate cancer, radiology exams include CT Scan, MRI, and X-ray (most commonly). Ultrasound would also be considered radiology, but in the case of prostate cancer, most urologists do their own ultrasounds and don't use radiologists. There are other imaging techniques which are called "nuclear medicine" because they require an injection that is radioactive or a "radio-pharmaceutical." Nuclear medicine imaging in prostate cancer includes Bone Scans (both the T99 and the F18) and all PET Scans (C11 Choline, C11 Acetate and F18 or Sodium Fluoride).

Now, to complicate issues just a little more….there are Radiologists who DO see patients, therefore they treat prostate cancer, but they are the exception to the rule. Sometimes they are referred to as interventional radiologists. In our world of prostate cancer, some familiar names would include Dr. Duke Bahn in CA who is known for cryotherapy and focal cryo, Dr. Fred Lee in MI (retired) who did the same, and Dr. Gary Onik in FL who also does cryo and focal cryo. There are also radiologists like Dr. Aytekin Oto in Chicago who are doing work in focal laser treatment for prostate cancer. (Note – focal treatments treat part of the prostate as opposed to all of the prostate.)

Now – on to the **Radiation Oncologist.** This is the physician who **administers your radiation treatments for cancer,** so this is much different than a radiologist. There are so many types of radiation treatments in prostate cancer, I will not attempt to name them all. But at the very least, think of daily radiation treatments to the prostate and short term radiation to metastatic disease as common treatments given by radiation oncologists. There is also radioactive seed implantation to

the prostate (brachytherapy) which usually involves both the radiation oncologist and the surgeon (urologist).

One last type of radiation that a radiation oncologist may administer is an injectable radiation for bone metastases called Xofigo (radium 223). This is for men who are metastatic and on hormone therapy. As you may know, this is called mCRPC (metastatic castrate-resistant prostate cancer). The other type of physician who might also administer Xofigo is a Nuclear Medicine Physician.

Simply understanding the differences between physicians can help you in your research and help you decide who to make an appointment with. This is all part of patient empowerment – we hope this explanation is helpful to you.

(3) What's the difference between a Radiologist and a Nuclear Medicine Physician? ***In fact – What the heck is Nuclear Medicine?***

Again a r**adiologist** is a physician you probably never meet, yet still impacts the understanding and treatment course of your cancer. He **reads and interprets your imaging** or your radiology exams, such as CT, MRI and X-ray.

A Nuclear Medicine physician also reads and interprets imaging, but some types of imaging are not called radiology – they are called nuclear medicine or nuclear imaging. In prostate cancer the most common nuclear imaging exams are Bone Scans, and all types of PET Scans. These are considered nuclear medicine because the patient receives an injection that is radioactive, called a radio-pharmaceutical as part of

the imaging. (An MRI or CT Scan can sometimes require an injection, but it is not a radiopharmaceutical, so they are not nuclear medicine.)

Radiologists and Nuclear Medicine physicians read and interpret your imaging.

Another way to state the difference between imaging with radiology vs nuclear medicine, is that **radiology is designed to see anatomy (shapes & sizes),** while **nuclear medicine is designed to see physiology (cells, molecules, chemical interactions, etc.).** If you've ever had a PET scan (nuclear imaging) for your prostate cancer, you may have had it "fused" with a CT scan (radiology). That's because the PET is better at visualizing cancer cells, but the CT scan is better at visualizing anatomy such as organs, bones, etc. Since no medical imaging is 100% perfect, using 2 different techniques together often improves the accuracy of the exam.

Something else unusual about Nuclear Medicine, is that it includes both imaging and treatment – because a radiopharmaceutical can be used for either, and the radiopharmaceutical (injection) is what makes something "nuclear medicine." So, nuclear medicine physicians not only deal with imaging, but they also administer a few select treatments. In prostate cancer, this is mainly one treatment right now, which is Xofigo (radium 223). Xofigo was FDA approved in 2013 for men on hormone therapy, with rising PSA and bone metastases. **See full Xofigo information online at www.xofigo-us.com/patient.** So if you are scheduled to start Xofigo, you may have a clinic visit scheduled to see a nuclear medicine physician, who you may have never met before in your cancer care. The only other type of doctor who administers Xofigo is a Radiation Oncologist – See article (2) above.

In the world of prostate cancer there are many twists and turns along the journey. The medical system is often complicated and sometimes overwhelming. Understanding the medical system can help you navigate your journey. Knowing a little about the different physicians will be helpful. So, if you hear the word nuclear medicine, know that it includes both imaging and treatment in prostate cancer, and know that it's slightly different than radiology when it comes to your imaging. Use this information to formulate better questions for your nurses or medical oncologist. Better questions always bring better answers. **Stay empowered**.

The Importance of Belonging to a Support Group, Gene Van Vleet, COO, IPCSC, San Diego CA, 2011

Following the recent consternation provoked by the recommendation of the U.S. Preventive Services Task Force (USPSTF) panel to discontinue PSA testing, it is fitting that we address the benefits provided by support groups in dealing with Prostate Cancer.

Commonly a man visits his general practitioner on an annual basis for a general physical. This can include **bloodwork that includes a PSA test and a digital rectal exam (DRE). Make sure you keep a log of your results.** Too often the patient is not informed of the results, but rather is informed "everything is OK." If the PSA test and/or the DRE is of concern to the physician the patient is referred to an urologist for further

diagnosis. All too often the next step is a biopsy. Once a biopsy is performed, the roller coaster of treatment options begins. WAIT A MINUTE!!! What is missing here? Patient knowledge and understanding!

As principal of a highly active support group it is my experience that I am most commonly contacted by patients either after a biopsy or after a relapse following treatment. I can help fill the knowledge gap and **lead men to information with which they can learn to be their own case managers rather than be reliant on physicians' recommendations.** I am by no means a medical professional but I have the knowledge of collective experience to aid men through the confusion of dealing with our troublesome disease. Further, I bring specialists in dealing with our disease to our group meetings to keep us informed of the latest developments without the restrictions of learning and protocol too often suffered within the medical community.

Let us begin with the myth of the PSA test. It was never intended to be utilized as an indicator of the seriousness of the cancer. Its best value is as a marker to monitor elevation over time. Concerns should develop if the score doubles within a year. Simple logic must be used if the score begins rising. It could be because of an ancillary infection. It could be because the test was performed by a different laboratory. It could be because of strenuous exercise prior to the test. One should first verify the test before proceeding. If the PSA is proved to be validly escalating, be real careful about the usual next step—Biopsy.

There has been significant progress lately in prostate imaging that can analyze the condition of the prostate BEFORE an invasive biopsy is performed. Should such imaging indicate the need, a biopsy can then be performed aided by that imaging. Please, no more random biopsies that may miss the troublesome area! Where do you get these tests? Your support group is a good place to find where such imaging is available in your

area.

If you have reached the stage of having a biopsy performed, a Gleason score results, which has been the landmark for determining the seriousness of the cancer. Other tests are arising and being validated that can assist in this determination as well. Your support group will likely know their status or can lead you to sources that will know.

We hear too often of cases where a patient is given treatment without a complete medical check-up. Pity the poor man who saw his urologist because his PSA was rising rapidly. He was given a hormone injection and consequently suffered atrial fibrillation and, later, a mild stroke. His physician failed to check his overall physical condition. Incomplete medical training associated with proper health investigation before treatment can be a problem.

Once a Gleason score is rendered, too often treatment is implemented before a thorough understanding of the possible effects on the patient's life are achieved. Surgery? Radiation? Cryoablation? HIFU? Hormone Therapy? et al. What a maze of possibilities exist. Get involved with your support group and benefit from their knowledge and experience.

We think one of the major oversights in treatment possibilities is no treatment at all or Active Surveillance (often called Watchful Waiting). Dr. Duke Bahn has quantified those tests that can be monitored by a patient over time in concert with his doctor without undergoing invasive treatment (see PAACT article in March, 2011 issue). An important element of this choice is the mental capacity to overcome a man's natural urge to do something to "cure" the disease. Be careful of that word. When someone uses it, make them define to you what they mean. It might be that they consider you "cured" if you don't experience signs over a much shorter time span than you expect.

If you are faced with making a treatment choice, be sure you develop an understanding of the possible side effects of the chosen treatment. Another unfortunate issue in dealing with prostate cancer is that it is difficult to predict how a patient will react to the treatment. Your physician may cite percentages of success, but there is yet no way to ensure what your experience will be. Be sure to check the experience of the doctor treating you. The most experienced doctor will achieve the best results. And, for sure, seek second opinions from unassociated doctors. This can be difficult because of insurance coverage limitations, but it will be in your best interest. Remember, you are your own case manager. Have confidence that unless you are diagnosed at a late stage the disease is generally slow moving. You have time to assess your treatment possibilities before committing.

Stay involved with your support group. You will find comfort in networking with others to help them as you are being helped. The natural tendency is to be involved through treatment and then disengage. There is value in continuing to stay involved to learn of advances in diagnosis and treatment. It keeps you aware of monitoring your own condition. Too many of the newcomers to our group are experiencing recurrence. Staying abreast of your condition and what is developing in the treatment of the disease will surely give you the opportunity to deal with it successfully. Remember, YOU CAN LIVE WITH PROSTATE CANCER!

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C11 Acetate PET/CT Imaging for Prostate Cancer, Fabio Almeida MD, 2012

PET imaging of cancer metabolism is commonly performed with F18 fluorodeoxyglucose (FDG), and has become one of the primary tools in the evaluation of cancer patients [1]. This is based on the well-established understanding that many cancers are highly glycolytic or have increased metabolism of glucose [2]. Although FDG may accumulate in aggressive and undifferentiated tumors, most prostate cancers demonstrate poor uptake of FDG, probably because most of these are well differentiated tumors. Additionally, FDG is secreted into the urinary system, often interfering with pelvic pathologic findings and therefore significantly limiting its usefulness. PET imaging of other metabolic pathways, such as amino acid or lipid metabolism, has now been explored in cancer. Fatty Acid Synthase (FAS) participates in controlling the lipid composition of cell membranes, and is over-expressed in many human cancers, particularly prostate cancer [5,7]. The degree of its over-expression appears to be correlated with tumor aggressiveness [6]. Among the different PET tracers that have been specifically evaluated for lipid metabolism imaging, Carbon-11-Acetate (C11-Acetate) demonstrates utility for detecting recurrent prostate cancer.

Recurrent & Metastatic Prostate Cancer

Prostate carcinoma is the second most leading cancer cause of death in American men. The presence of an elevated prostate specific antigen (PSA) level after definitive treatment, such as prostatectomy (RP) or radiation therapy (RT) is suggestive of recurrence. Unfortunately, recurrence of prostate cancer after treatment is frequent, occurring within 10 years in about 30%-40% of patients. After RP, a PSA level of greater

than 0.2 ng/mL, confirmed by two consecutive measures, can be associated with either residual or recurrent disease. After radiation therapy (RT), a mPSA value of 2.0 ng/mL above the nadir represents persistent or recurrent disease. The management of recurrent prostate cancer depends strongly on whether recurrence is confined to the prostatic bed (local failure), the regional lymph nodes in the pelvis or if distant spread has occurred. Although a trend of increasing PSA has been proposed as a way of predicting local recurrence versus distant recurrence, only imaging procedures are capable of discriminating between these scenarios [3,4].

Therapeutic options in recurrent and advanced prostate cancer are rapidly expanding. Thus, there is a need to develop imaging approaches that will a) allow for detection of and discrimination between local recurrence and distant metastatic disease, and b) permit the monitoring of tumor responses to these new therapeutic approaches. Typically restaging for a recurrence is performed with a combination of ultrasound guided trans-rectal (TRUS) biopsy, computed tomography (CT), magnetic resonance imaging (MRI), and bone scans (BS). None of these however are very effective at detecting recurrences early enough to help select patients for salvage therapy with a curative intent. Additionally, these may limit the potential use of novel therapies by their inability to detect recurrences.

Carbon-11 Acetate Positron Emission Tomography (C11-Acetate PET)

Several small studies have evaluated the relationship between serum PSA levels and detection of prostate cancer recurrence with C11-Acetate PET. In a study of 25 patients by Fricke et al., the degree of C11-acetate uptake was correlated with serum PSA levels [8]. Kotzerke, et al., evaluated a series of patients with suspected recurrence based on serum PSA measurements [9]. Transrectal ultrasound followed by biopsy served as the gold standard for C11-acetate PET imaging

findings. C11-Acetate was true positive for disease recurrence in 15/18 patients with biopsy proven recurrence and was true negative in all 13 patients without recurrent disease by biopsy. Sensitivity was 83% and specificity 100%. Additionally, 4 of 5 patients with biopsy proven cancer and positive C11-Acetate PET imaging findings had serum PSA levels of less than 2.0 ng/mL. Sandblom, et al., evaluated 20 patients with elevated PSA levels ranging from 0.5 to 8.1 ng/mL after radical prostatectomy [10]. C11-Acetate PET identified disease sites in 75% of the patients. In this study, all PET-positive patients had serum PSA levels of greater than 2.0 ng/mL. “False positive” findings were reported in 3 patients. One patient exhibited tracer uptake in the chest, which was subsequently confirmed to represent non-small cell lung cancer, while two other patients had inflammatory changes, one in the esophagus and the other in the mediastinum. As expected, this study suggested that C11-Acetate uptake is not cancer specific, but rather, a probe of lipid metabolism which may also be altered in inflammatory disease.

Comparative studies

A few groups have compared the diagnostic performance of C11-Acetate with that of other metabolic PET imaging probes in patients with prostate cancer. In a small study, Kotzerke, et al., evaluated 12 patients with prostate cancer [11]. C11-Acetate and C11-Choline, a substrate of choline kinase that is also incorporated into membrane lipids, were compared in patients after initial diagnosis, at the time of biochemical recurrence or after radical prostatectomy. The study found that C11-Acetate was not excreted into the bladder while urinary excretion was variable for C11-Choline. In terms of overall biodistribution and tumor uptake, the diagnostic performance of both imaging probes was found to be comparable. In our own institution, we compared lesion detectability using FDG and C11-Acetate imaging in a small

group of prostate cancer patients with recurrent or metastatic disease [12]. Eighteen patients were imaged with both FDG and C11-Acetate PET with a PSA ranging from 0.32 – 13 ng/ mL (mean 5.0ng/mL). C11-Acetate PET detected tumors in 14 (78%) patients, whereas FDG PET detected lesions in only 2 (14%) of the imaged patients. In the two FDG PET positive patients, the PSA was relatively higher than in the other patients, with values of 7.8 and 11.15 ng/mL, respectively. C11-acetate PET was also positive in these two patients, detecting more disease with a significantly higher tumor to background uptake ratio. C11-acetate PET detected recurrence in the intact prostate or prostate bed in 5 patients, lymph node involvement in 6, bone in 4 and liver in 1. In 3 of 5 patients with lesions detected on C11-acetate, the PSA was < 1.0 ng/mL. These studies suggest that C11-choline and C11-acetate appear to have a comparable accuracy for detecting local recurrence and metastatic disease in early PSA recurrence, while FDG PET does not seem to provide significant diagnostic value in this context.

Preliminary Results from the Arizona Molecular Imaging Center

As part of an ongoing FDA-approved clinical investigation, the Arizona Molecular Imaging Center has thus far performed over 150 C11-Acetate PET/CT imaging studies, significantly more than have been previously published from a single institution in the U.S. Preliminary results from our studies have been very encouraging, and demonstrate a direct benefit to many patients that would not be achievable with any other standard imaging technique. See Cases 1-4 below for examples of positive imaging studies. In our experience thus far, the overall detection rate of C11 Acetate PET/CT imaging for recurrent or metastatic disease has been 85%. When we separate the positive findings into various PSA levels, the detection rate has been 73% for PSA values of 0.4 – 1.0 ng/mL, 89% for 1.0 – 2.0 ng/mL and 93% for > 2.0 ng/mL. Our results to date have shown a higher detection rate than data from previously

published studies, likely in part due to our use of more modern, state-of-theart PET/CT imaging technology which allows for better detectability and localization of smaller lesions, and due to establishing a standardized imaging protocol based on tracer kinetics which had been lacking in prior studies.

Most of our study patients are still in early follow-up. However, in several patients with initial follow-up after additional therapy, such as radiation therapy directed toward the recurrence or metastasis, or after surgical removal of the lesion identified on the C11-Acetate images, there has been a significant decrease in PSA, confirming the accuracy of the C11-Acetate imaging. Our imaging studies are further revealing the inadequacy of using PSA as a predictor of local versus distant recurrence. An initial evaluation of our positive studies show that recurrence was identified outside of the prostate gland or prostate bed (extraprostatic involvement) in 73% of patients over a wide range of PSA values, and as low as 0.5ng/mL. Positive nodes only in the pelvis where seen in 17% of patients and 25% showed lesions only in the bone.

Patients with prior prostatectomy and a rising PSA represent one of the most challenging groups, as many are offered salvage radiation therapy to the prostate bed without any imaging evidence of where the recurrence actually is. The success of prostate bed salvage radiation therapy is said to depend on the margin status and PSA, with a rate of success of only about 40% when the PSA is <0.5, and far less when the PSA is >0.5 [13]. When we review this subset of patients in our study (59 patients), we find that with a PSA of 0.5 – 2.0ng/mL, one third of patients are found to have recurrence limited to the prostate bed, a third to have only pelvic node involvement and another third of these patients to have bone involvement. The ability of the C11-Acetate to differentiate where the disease is located, then allows for appropriate selection of patients for which radiation to the prostate bed

is more likely to succeed, and also identify which patients may benefit from radiation also given to the pelvic lymph nodes. The PSA values were clearly not able to provide this differentiation and prostate bed radiation therapy based on the PSA alone would have failed in the majority of these patients.

Case example 1.

Gentleman with prostatectomy 10 years previously. External beam radiation 1 year previously for a rising PSA. The PSA continued to increase up to 6.9 ng/ mL. The 3 dimensional Carbon-11 Acetate PET/CT images show a small metabolic lymph node in the left pelvis (yellow arrows). This would not have been diagnosed on CT alone based on its small size. Other areas of 'red' seen on the images are of normal Carbon Acetate in the intestines, kidney's, liver and spleen. No other lesions were seen. The left pelvis node was treated with IMRT and the PSA then decreased to 0.9 ng/mL, confirming involvement of the identified node.

<ADD PHOTO Here, C11Acetate_Almeida 2012>

Case example 2.

Gentleman with Gleason 7 prostate cancer and external beam radiation (EBRT) to the prostate 4 years previously. PSA nadir was 0.43ng/mL. Rising PSA to 3.9 ng/mL. The 3 dimensional Carbon-11 Acetate PET/ CT images show a metabolic focus in the right side of the prostate gland (yellow arrows). No other lesions were seen. The prostate recurrence was confirmed by biopsy with subsequent Brachytherapy performed. The PSA decreased to 0.6 ng/mL after treatment.
<ADD PHOTO Here, C11Acetate_Case 2 2012>

Case example 3.

Gentleman with Gleason 6 prostate cancer. Brachytherapy and external beam radiotherapy 12 years previously. PSA nadir was

0.16 ng/mL. Rising PSA to 2.17 ng/mL. The 3 dimensional Carbon-11 Acetate PET/ CT images show a single small metabolic lymph node in the left upper pelvis (yellow arrows). As in Case example #1, this would not have been diagnosed on CT alone based on its small size. Bilateral pelvic lymph node dissection was performed with 13 nodes removed. The node identified on the C11-Acetate imaging study was confirmed to be involved with prostate cancer (Gleason 4+4=8) and all other removed nodes were negative/benign, confirming the solitary finding on the imaging study. The PSA decreased to 0.19 ng/mL after the lymph node surgery.

<ADD PHOTO Here, C11Acetate_Case 4 2012>

Case example 4.

Gentleman with prostatectomy 9 years previously. Rising PSA to 4.8 ng/mL. The 3 dimensional Carbon-11 Acetate PET/CT images showed a single bone metastasis involving the second cervical vertebral bone (yellow arrows). No other lesions were found in the bone, lymph nodes or in the prostate bed. The cervical lesion was confirmed on MRI. Initially this patient was to have salvage radiation treatment to the prostate bed, but with the C11-Acetate results the treatment plan was changed to radiation treatment just to the bone lesion. The PSA decreased to 0.2 ng/mL after the treatment.

<ADD PHOTO Here, C11Acetate_Case 4_2 2012>

Due to recent changes in FDA regulations regarding new radiopharmaceuticals such as C11 agents, access to C11-Acetate now requires participation in an approved clinical study. The Arizona Molecular Imaging Center has worked with the FDA to open an approved Phase II clinical investigation, and is pleased to offer Carbon-11-Acetate PET/CT imaging studies for localizing recurrent prostate cancer. Because this type of scan requires an on-site cyclotron, we are one of the few sites in the country capable of doing these studies, and

currently the only FDA-approved private site for C11-Acetate imaging for recurrent prostate cancer.

Our center is equipped with state-of-the-art PET/CT imaging, which provides an extra advantage in the detection of small lesions. The C11-Acetate study requires only a single intravenous injection of the tracer and the imaging procedure can be completed in about 20 minutes.

Future Directions

Although both C11-Acetate and C11-Choline are proving to be excellent imaging tools for the detection of recurrent prostate cancer, the very short half-life of C11 (20 minutes) and the need for an on-site cyclotron are limiting factors for wide spread adoption. Other PET imaging agents are being investigated, such as Fluorine-18 labeled amino acids, which may provide similar results as C11-Acetate. The Arizona Molecular Imaging Center is working toward participating in further investigation of such agents as these may provide better patient access to quality prostate cancer imaging tools.

For information about participating in our C11-Acetate PET imaging clinical trial, please visit the ClinicalTrials.gov website: http://clinicaltrials.gov/ct2/show/record/NCT01304485 or call Dr. Fabio Almeida directly at 602.331.1771.

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Degarelix [Firmagon] – No More Agonizing Jeffrey Turner MD, 2010

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Since Huggins and Hodges established the link between prostate cancer and testosterone in 1941, androgen deprivation therapy has remained a cornerstone for systemic treatment of prostate

cancer. In 1979, the first prostate cancer patient was treated with a GnRH agonist at the Laval University Medical Center in Quebec City, Canada with credit of this discovery by Dr. Fernand Labrie. In 1985, gonadotropin-releasing hormone (GnRH) agonists were first approved by the FDA for the treatment of prostate cancer. Common trade names of GnRH agonists include leuprolide (Lupron), Trelstar, and Zoladex. These GnRH agonists take the place of the physiologic, pulsatile, intrinsic GnRH which is secreted from the hypothalamus gland. As a result, this desensitizes the pituitary gland and ultimately causes down-regulation of the receptors which prevents further pituitary secretion of leutinizing hormone, in turn arrests testicular secretion of testosterone. **Unfortunately, GnRH agonists can cause an initial flare or surge of testosterone which may last from 2-4 weeks** before medical castration is achieved. What exactly does this mean? **These surges can be associated with potentially devastating effects with patients having widespread disease including the potential for spinal cord compression in patients with spinal disease or significant worsening in bladder outlet obstructive symptoms in patients with locally advanced disease.**

On the contrary, GnRH antagonists [such as degarelix] can produce immediate suppression of testosterone *without any surge* and usually achieve adequate testosterone suppression within 48 hours. As a result, this prompted the discovery of GnRH antagonists which directly block the GnRH receptor and prevent signaling downstream. The prototype GnRH antagonist was Abarelix which was approved by the FDA in 2003. Unfortunately, a minority of patients sustained hypersensitivity reactions including anaphylaxis. As a result it was withdrawn from the U.S. market. More recently, **degarelix [brand name Firmagon]**, a new-generation GnRH receptor antagonist with low histamine-release was approved by the FDA in December 2008. Degarelix, fortunately, has not been associated with a significant risk of allergic reaction or anaphylaxis. The common side effects of degarelix are similar

to those of GnRH agonists including transient injection-site reactions. The pivotal trial which proved degarelix to be equivalent, or non-inferior, to leuprolide was published in December 2008 in BJU International by Klotz et al. This was a phase 3 trial which demonstrated that degarelix was clearly not inferior to leuprolide at maintaining low testosterone levels over a 1 year period. In fact, degarelix induced testosterone and PSA suppression better than leuprolide. Incidentally, an additional difference between the two was a lower incidence of urinary tract infections while using degarelix compared to leuprolide. In November 2009, Tombal et al in European Urology, provided results from a Phase 3, multicentre, randomized trial comparing degarelix to leuprolide in 610 patients. The objective of the trial was to compare activity in respect to PSA recurrence-free survival. Patients receiving degarelix showed a significantly lower risk of PSA progression or death compared to those on leuprolide. PSA recurrence occurred mainly in patients with advanced disease and in those with baseline PSA >20. PSA recurrences occurred almost exclusively in patients with metastatic prostate cancer or high baseline PSA during this study.

Before the discovery of PSA, urologistis relied on serum prostatic acid phosphatase (PAP) and serum alkaline phasphatase (S-ALP) to assess the status of men at risk for or under treatment for metastatic prostate cancer. Alkaline phosphatase is a blood test which is often elevated in patients with metastatic disease as a marker of bone turnover. Hence, escalation of alkaline phosphatase can be a concern for progressive metastatic disease. In November 2009, Shröder et al in BJU International, published the results of a randomized, phase 3 trial comparing changes in alkaline phosphatase levels in patients on degarelix and leuprolide. This trial demonstrated that in patients with metastatic disease or those with PSA ≥50 at baseline achieved greater reductions in alkaline phosphatase levels while on degarelix compared to leuprolide. This implied the possibility that

degarelix offers a longer time to progression as well as a survival benefit compared to GnRH agonists. Over the next few years, prostate cancer therapy will become increasingly tailored to meet the needs of individual patients with specific disease characteristics with continued development of novel, targeted therapies. Nonetheless, endocrine therapy will remain a critical pharmacologic cornerstone and it would be anticipated that new treatments in relation to hormone blockade will continue to evolve. Analysis of the current data suggests that degarelix may provide an advantage over traditional GnRH agonists in relation to faster and more efficient testosterone and PSA suppression, absence of a testosterone surge (risking potential complications such as spinal cord compression and bladder outlet obstruction), and better control over of the disease with respect to progression of bone metastases.

Dr. Jeffrey Turner is a native of Southern California, having grown up in Orange County and attended the University of Southern California. He was the first student to graduate from USC’s 3-year accelerated science program with a bachelor’s degree in Biological Sciences. Subsequently, he was employed as a research associate at UCLA in infectious diseases and molecular biology. He completed his medical training in Vancouver, British Columbia. He has also trained clinically at MD Anderson Cancer Center with a focus on genitourinary malignancies. He is board certified in medical oncology and internal medicine. After two years of training, he graduated from fellowship in June of 2009 with a focus on both prostate and renal cell carcinomas. His most recent publication was an expert opinion on Finasteride in July 2010. He has been working with Dr. Bob Leibowitz at Compassionate Oncology Medical Group since July 2009.