



Informed Prostate Cancer Support Group Inc.

"A 501 C 3 CORPORATION ID # 54-2141691"



May 2018 NEWSLETTER

P.O. Box 420142 San Diego, CA 92142

Phone: 619-890-8447 Web: <http://ipcs.org>

We Meet Every Third Saturday (except December)



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Next Meeting

May 19, 2018

10:00AM to Noon

Meeting at

Sanford-Burnham-
Prebys Auditorium

10905 Road to the
Cure, San Diego CA
92121

**SEE MAP ON THE
LAST PAGE**

Monday, May 14, 2018

Volume 11 Issue 5

What We Are About

Our Group offers the complete spectrum of information on prevention and treatment. We provide a forum where you can get all your questions answered in one place by men that have lived through the experience. Prostate cancer is very personal. Our goal is to make you more aware of your options before you begin a treatment that has serious side effects that were not properly explained. Impotence, incontinence, and a high rate of recurrence are very common side effects and may be for life. Men who are newly diagnosed with PCa are often overwhelmed by the frightening magnitude of their condition. Networking with our members will help identify what options are best suited for your life style.

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Editor: Stephen Pendergast

PROSTATE CANCER IT'S ONLY 2 WORDS NOT A SENTENCE



Paul E. Dato, MD – Decision Factors in Treatment Selection and Urology Update

April 2018 IPCSG
Meeting Summary
by Bill Lewis.

Options, esp. in low or intermediate risk disease
can be overwhelming. Options in high risk or met-

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Video DVD's

DVD's of our meetings are available in our library for \$10ea. Refer to the index available in the library. They can also be purchased through our website: <http://ipcs.org> Click on the 'Purchase DVD's' tab.

The DVD of each meeting is available by the next meeting date.

astatic disease are more limited but still daunting. Options are influenced by many factors, including whether the disease is hormone-sensitive or “castration-resistant,” non-metastatic or metastatic, asymptomatic or symptomatic. Early on, after any active surveillance period, there are many choices of local therapies. Often, on recurrence/metastasis, this may be followed by ADT, secondary hormonal therapies, immunotherapies, androgen biosynthesis inhibitors, radionucleotide therapy, chemotherapy, antiandrogens, and/or palliative chemotherapy, depending on the course of the disease. Treatment considerations also include the patient’s age and expected life span (which is affected by genetics, education, income / social status, and support networks), co-morbidities (i.e., other diseases present, which may decrease life expectancy, increase the severity of side effects, and bring possible drug-drug interactions), the stage and grade of the cancer, the potential benefits of each treatment option (i.e., “cure” vs. palliation), and the patient’s concerns about side effects. The physician’s bias is also a significant factor: urologists (who are surgeons), and radiation oncologists often favor the treatment modality they are personally most familiar with.

The stage and grade of the cancer are very important, and are assigned by established guidelines. The location(s) of the cancer are ultra-important, as is the aggressiveness. Genomic assessment, especially for cancers found early, can be helpful in determining likely aggressiveness, in addition to the Gleason score.

Key patient concerns include the quantity of expected lifespan with the disease vs. one’s quality of life, and “costs” that include side effects, money and time. Note, for example, that side effects may be more tolerable (more “worth the pain”) for a younger patient than a very old one.

A Memorial Sloan-Kettering study showed in a small group of twenty patients, that they were able to achieve an apparent cure of bone-metastatic disease in 20% of the men, by a combination of ADT, surgery, and radiation. No cures were achieved in the lymph-node-metastatic men, but nearly all the men in both groups had a period of time of nearly-undetectable PSA. See [https://www.goldjournal.net/article/S0090-4295\(16\)30850-0/fulltext](https://www.goldjournal.net/article/S0090-4295(16)30850-0/fulltext)

Results from a Belgian study published last year showed that men with oligometastatic disease as recurrence after some kind of primary therapy (not specified), who were then treated with surgery or cyber-knife, were able to postpone the need to go on ADT for a median time of 8 months, thus avoiding the side effects of ADT for that period of time. See <https://doi.org/10.1200/JCO.2017.75.4853>

A question: Options other than ADT for metastatic disease? Proscar and Avodart are not considered ADT, and may help some, but not likely a lot. Chemo has always been studied with ADT, not alone. Some follow wholistic regimes, but Dr. Dato hasn’t seen them be successful.

Definition of metastatic disease? Any prostate cancer outside the pelvis area. Cancer in local lymph nodes is not considered “metastatic,” but would be considered “locally advanced disease.”

Discussion of Erleada (apalutamide): It is an oral androgen receptor inhibitor, in the same class of inhibitors as Casodex (bicalutamide) or Xtandi (enzalutamide). It is approved for non-metastatic (not outside the pelvic area) castrate-resistant prostate cancer, based on the SPARTAN phase 3 trial. The approval was based upon “Metastasis Free Survival;” the time until metastases began to be found. This new standard, which is a “surrogate” for overall survival, is intended to allow demonstration of a benefit in cases where it may take many years to determine the overall survival benefit. This can allow a drug to obtain FDA approval earlier, so that patients can begin receiving the drug. MFS improvements have been shown in other studies to correlate with subsequently observed overall survival. Erleada is the first drug approved specifically on the basis of demonstrated MFS improvement.

MFS was quite significantly improved – by 2 years for Erleada+ADT over placebo+ADT. There was a 72% reduction in the risk of metastasis or death, and the drug was generally well tolerated. The most

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common side effects include fatigue as well as falls (16%), fractures (12%), rash (24%), low thyroid (8%), high K+, and/or seizure (0.2%).

Regarding oligo- and micro-metastatic disease, it was shown that patients with pelvic lymph node tumors responded as well to Erleada as the others, so the drug seems effective beyond the prostate area, and Dr. Dato believes it will soon be approved for treating fully metastatic disease. It's also expected that Xtandi will soon be approved for the same use as Erleada now is approved, so there will be two agents available to treat "non-metastatic" castrate-resistant disease.

Cost of Erleada is similar to Xtandi and Zytiga; about \$10,000 per month. It typically takes about a year after FDA approval, for Medicare and other insurance plans to cover a new drug. There are foundations that can assist, especially for those without their own means.

A similar issue of government approval relates to the frequency or timing of PSA testing. Now it's considered an issue for doctors and patients to jointly decide. But clearly, during the period when PSA testing was officially discouraged, there were more men who did not find their disease until it was at a more-advanced, less treatable state.

Candidates for Erleada: those on hormone therapy, or who were surgically castrated, with a rising PSA, especially if it is rising relatively quickly (say, a doubling time of 15 months or less).

A question about biopsy vs. MRI: In Europe, an international 35-center study of 500 men was done of using MRI for screening, and reported in the past month. Using magnetic resonance imaging (MRI) to guide targeted biopsies increased the detection of clinically significant cancer by 12% when compared with standard transrectal ultrasonography guided biopsy. MRI revealed no sign of prostate cancer in more than one in four men, avoiding the need for unnecessary biopsy or treatment, according to the authors. See <https://www.birmingham.ac.uk/news/latest/2018/03/prostate-cancer-birmingham-university-precision.aspx>

Dr. Dato noted that about 20% of men with prostate cancer are missed by MRI, but caught by a biopsy. As the article noted, MRI can identify high Gleason disease, but not usually at the lower level of Gleason = 6. However, genomic testing shows that some Gleason = 6 tumors are more aggressive than average, so there is some risk in skipping a biopsy.

Regarding biopsies, there are the issues of the number of cores that are positive, the % of each core that has disease, and the issue of non-biopsied areas – which MRI can help with. There remain many unknowns and grey areas for making decisions about treatments – not just one definitive test. It's like a set of puzzle pieces without the boxtop showing what the final picture will look like. You consider many different pieces, and try to get the aggregate picture.

What about transperineal vs rectal biopsies? They're more painful, and difficult in practice. Most urologists aren't trained to do them. They do offer a lower infection rate, and access to areas of the prostate not reachable via the rectum. But infections in rectal biopsies have been reduced by pre-screening for drug sensitivity, or by using two antibiotics together (as Dr. Dato does). MRI-guided biopsies can reach farther than standard (rectal) biopsies, but still can't reach all areas.

Usefulness of high-dose Casodex? It's frequently used in Europe, but has not been approved in the US. It does have higher side effects, particularly gynecomastia (breast growth) – which is a side effect not seen with Erleada. High dose Casodex as monotherapy has been used in Europe, but is not approved in the US. Dr. Dato tried it, but ran into insurance companies unwilling to pay for it, due to the lack of FDA approval. Erleada is not approved as monotherapy – it is used with ADT such as Lupron or Firmagon.

NY Times article about genomic testing? Such tests are used as guidance for low- and intermediate-risk patients by Dr. Dato. For "very low risk" patients, usually there is not enough cancerous tissue in

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the biopsy cores to run the tests. In such cases, he does an MRI, but after the biopsy has healed – often waiting 6 to 18 months until just before a confirmatory biopsy. Genomic tests costs are in the range of \$2-3000, before insurance.

An update on immunotherapy: It has been found that the microenvironment of castration resistant prostate cancer is highly immunosuppressive! A bi-directional approach [stimulation, with modulation to avoid inflammation in other tissues] is likely to produce the best results. Combination therapies will likely be the next phase of immunotherapy. Medications are being developed for DNA mismatch repair.

Useful websites: Medscape; Society for Immunotherapy of Cancer.

Intermittent hormone therapy? Three months on, three months off has worked for a member for 5 years. His PSA rises quickly, say to 6, when he is off the Lupron for only 2 months. Dr. Dato says that this approach goes on the theory that it will avoid castration resistance occurrence happening as fast, and allows the man to enjoy periods without the side effects of testosterone suppression. He has some patients on this therapy. In a small study, some castrate-resistant men were given supplemental testosterone, and some, but not all, then became “hormone sensitive” again. We don’t yet know how to predict who will respond this way.

CAR-T therapy? No data yet on efficacy.

Roles of urologists and oncologists? Typically, biopsies are done by urologists, and may continue to care for the patient if surgery is chosen. On recurrence, an oncologist may be consulted. In general, oncologists are involved when the disease is more advanced. It’s appropriate to consult with a radiation oncologist in many cases. Informed choice!

Is prostate cancer curable? He has patients with essentially zero PSA even 15-20 years after surgery or radiation. But for many, there is some persistence of cancer cells, which may begin to multiply.

Use of supplements? None have placebo-controlled, double-blind studies. But some may be helpful. It will be a clinical trial of one patient – yourself.

Possible stimulation of the immune system, by irradiating a metastasis? That does seem to happen in some cases. But there’s not solid data yet. Hopefully, liquid biopsies being developed may help track success in such patients, to determine whether there are higher or lower levels of circulating tumor cells, to determine success.

Is there any data on whether nanoknife, HIFU, or brachytherapy is best for stimulation of the immune system? Not yet. There is animal data, but not human data so far.

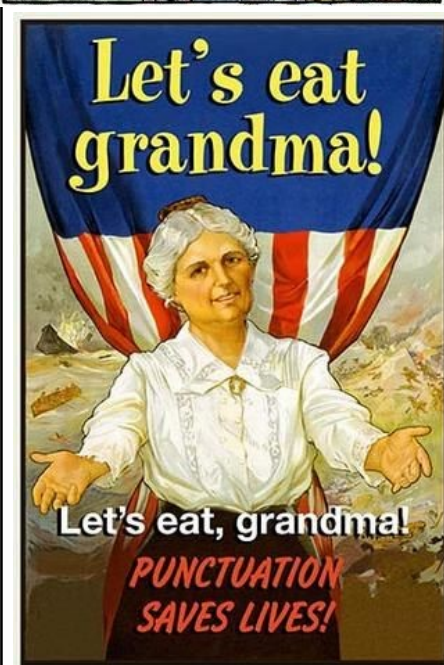
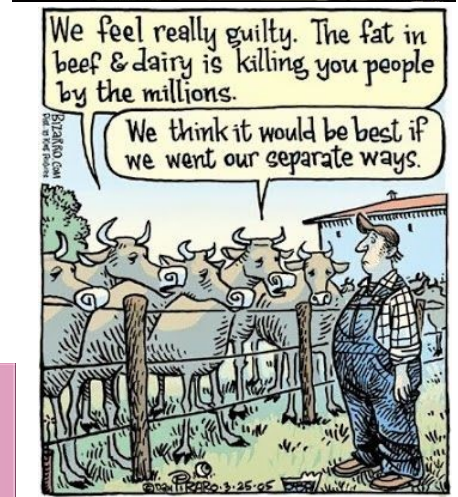
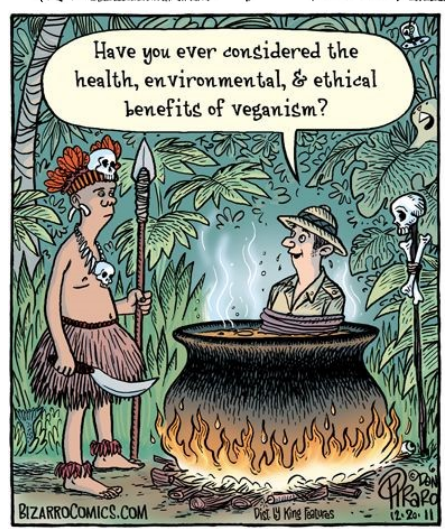
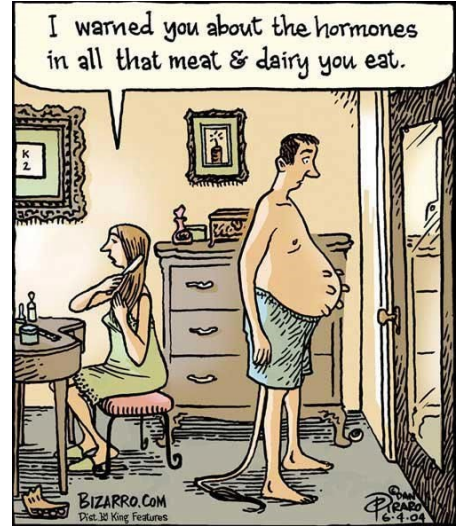
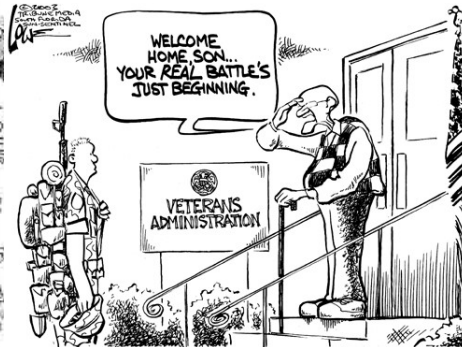
There is an annual Genesis patient conference that has traditionally been held in June, that will be open this year to the general public. However, it has been moved to early October to more coincide with Prostate Cancer Awareness Month in September.

A video of the April IPCSG meeting, including the slides, will be available via the website shortly before the next meeting, or at the May meeting on the 19th.

USPSTF Nixes PSA Screening— Notable announcement:

[Screening for Prostate Cancer: US Preventive Services Task Force Recommendation Statement | Cancer Screening, Prevention, Control | JAMA | JAMA Network](#): This 2018 updated Recommendation Statement from the US Preventive Services Task Force concludes that clinicians should not screen for prostate cancer in men aged 55 to 69 years who do not express a preference for screening (C recommendation) and recommends against PSA-based screening in men > 70 years

ON THE LIGHTER SIDE



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FUTURE MEETINGS

- Meeting Date SPEAKERS
- May 19 Dr Schwartzberg - Nanoknife

- **For further reading:**

<http://spendergast.blogspot.com/2018/03/prostatecancernews-2018-03.html>

- **For Comments, Ideas and Questions,**
email to Newsletter@ipcs.org

INTERESTING ARTICLES

EXCERPT FROM TESTOSTERONE+ PROSTATE CANCER

BY JOHN MULHALL, MD • ORIGINALLY APPEARED IN PROSTATEPEDIA SEPTEMBER 2017, VOLUME 3 NO 1 •
WWW.PROSTATEPEDIA.NET

Dr. John Mulhall is the Director of the Male Sexual & Reproductive Medicine Program at Memorial Sloan Kettering Cancer Center in New York City.

Prostatepedia spoke with him about erectile dysfunction and testosterone replacement therapy after prostate cancer. Find more information at www.prostatepedia.net

MANY MEN ASSUME THAT THIS ERECTILE DYSFUNCTION IS CAUSED BY LACK OF TESTOSTERONE, BUT WHAT IS TESTOSTERONE'S ROLE IN NORMAL ERECTILE FUNCTION?

Dr. Mulhall: Testosterone is not really a general erectogenic hormone, which is a common misconception. Even some urologists put patients on testosterone to help erections. You do not need a lot of testosterone for erections, so unless somebody's testosterone level is incredibly low, it's unusual for testosterone to help with erections.

Testosterone is involved in many other nonsexual processes, though. It's a motivation hormone. It's a mood- stabilizing hormone. The normal testosterone range is between about 300 and 800. Testosterone levels below 200 put you at risk for osteoporosis, diabetes, and premature cardiovascular events. The problem in the United States is that a lot of men on testosterone shouldn't be on it because their levels are normal. Twenty percent of men will go on testosterone and they have never had their testosterone levels checked. This is staggering.

ARE TESTOSTERONE LEVEL CHECKS PART OF A NORMAL CHECKUP?

Dr. Mulhall: It's not part of a normal checkup. The signs or symptoms that a patient has a testosterone level that should be checked include diabetes and testicular atrophy. If you've been exposed to chemotherapy, had testicular radiation, stem cell transplants, or bone marrow treatment, you should have your testosterone level checked.

Then there are a bunch of people who need testosterone and don't get it because family doctors think testosterone supplementation causes prostate cancer. It does not. The literature is black and white. Testosterone therapy does not cause prostate cancer. And low testosterone is a bad thing.

WHAT IS THE ROLE OF TESTOSTERONE IN PROSTATE CANCER?

Dr. Mulhall: Prostate cancer has difficulty growing without testosterone present. On the surface of a malignant prostate cell, there is an androgen receptor that is maximally stimulated at testosterone levels at or above 150 ng/ml. It doesn't grow more beyond that. That's called the saturation model. There's good scientific evidence to support this concept.

If your level's 250, and we put you on testosterone, we know that prostate volume and PSA barely change for most men. If five years after radiation you have hormone therapy with a testosterone level that's still castrate, say around 50, and then go on testosterone, your PSA will change because your androgen receptor is not yet maximally stimulated. That's the clinical evidence.

There is no link between your testosterone level, whether it's 290 or 1400, and the development of prostate cancer. Testosterone is necessary to make PSA. You need testosterone to bind to the androgen receptor on the cell to generate PSA. If your testosterone is below 200, you probably don't have an optimal testosterone level to make PSA.

A man with low testosterone may have higher-grade, higher-stage prostate cancer because there is a delay in diagnosis. His PSA is fine, but his testosterone level is really low. There is good evidence that if you give testosterone to men in randomized, controlled trials, there is

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no higher instance of prostate cancer in the group who get testosterone than in those who get placebo. Again, this supports the saturation model concept.

DOES THIS THEN MEAN THAT PSA MAY NOT BE THE BEST MARKER FOR PROSTATE CANCER?

Dr. Mulhall: It means you shouldn't look at PSA in isolation without looking at testosterone levels. Some men have Gleason 7 (high-volume) cancer, but their PSA is 1.2 and their testosterone level is low. The biopsy confirms prostate cancer. PSA should not be looked at in isolation. If it's elevated, that's one thing.

The question really is: Is it safe to give men who already have prostate cancer testosterone supplementation? Some physicians have been giving testosterone to these men for 12 years. There is no consensus.

We always write in the medical record that the patient has been made aware of the absence of long-term safety for testosterone therapy in the prostate cancer population. But there is no long-term data. It's been estimated that we need to follow 75,000 men for 15 years to answer that question.

Six to 12 weeks after radical prostatectomy, we give testosterone to men with an undetectable PSA, favorable pathology, and Gleason 6 or 7 organ-confined cancer. We have data on 360 men and haven't seen a single PSA elevation—not one. But that's 360 men, not 75,000.

...

<https://www.onclive.com/web-exclusives/frontline-advances-continue-in-castrationsensitive-prostate-cancer>

Frontline Advances Continue in Castration-Sensitive Prostate Cancer

Angelica Welch

The first-line treatment of patients with metastatic castration-sensitive prostate cancer has undergone a significant number of changes in the last few years. Most recently, the FDA approved abiraterone acetate (Zytiga) in combination with prednisone for patients with metastatic high-risk castration-sensitive disease.

This approval of abiraterone was based on findings from the phase III LATITUDE trial. In the study, there was a 38% reduction in the risk of death with the addition of abiraterone and prednisone to androgen deprivation therapy (ADT) compared with ADT alone.¹

Another pivotal trial of abiraterone in prostate cancer was STAMPEDE. This trial showed that the addition of abiraterone to standard initial therapy lowered the relative risk of death by 37% and improved progression-free survival by 71% in both nonmetastatic and metastatic patients with high-risk hormone-naïve disease.²

Although there is much excitement with androgen receptor (AR)-directed agents, docetaxel remains a staple of treatment. James Luke Godwin, MD, says that clinicians have now reached an interesting decision point on how to treat their patients in the frontline setting moving forward.

In an interview during the 2018 *OncLive*® State of the Science Summit™ on Prostate Cancer, Godwin, assistant professor, Kimmel Cancer Center Network, Thomas Jefferson University Hospital, recapped recent advancements in frontline metastatic castration-sensitive prostate cancer and highlighted emerging agents with potential in this space.

OncLive: How has first-line treatment for patients with castration-sensitive prostate cancer evolved?

Godwin: This is a space that has been evolving over the past couple of years. Since 2014 or 2015, we have learned that adding docetaxel upfront provides a survival benefit when added to ADT as opposed to ADT alone. That became a standard of care in 2017, with data from 2 large studies—LATITUDE and STAMPEDE—showing impressive data with the addition of abiraterone to ADT upfront in this setting. Currently, we have good data for 2 separate agents that have different toxicity profiles.

For clinicians, when you meet a patient with metastatic castration-sensitive prostate cancer, it is an interesting decision point. What therapy should clinicians add to standard ADT in the first-line setting? That is an open question.

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What are the differences in toxicity profiles between the 2 agents?

Docetaxel is a traditional chemotherapy; it is an old drug that has been used for a very long time. Common side effects include neutropenia, decreased cell counts, fatigue, and neuropathy. The benefit of docetaxel is that you give it for a set time period—only 6 cycles—and then that therapy is complete. You would then continue with ADT alone. Although there is a period where a patient may experience more toxicity, they are only on the therapy for a definitive amount of time. The majority of men in the CHAARTED study, which showed the survival benefit for docetaxel upfront, completed all 6 cycles as planned without dose reduction.

Abiraterone is generally well tolerated. The side effect profile does include some cardiovascular risk and hypertension, which can be managed. However, when considering first-line treatment with abiraterone, you are considering committing a patient to a longer treatment course than with docetaxel. When making that decision, the clinician should work with the patient and consider toxicity, length of treatment, and cost to figure out what works best for that particular patient.

Is there any rationale to look at other agents, such as enzalutamide?

Absolutely, there are trials ongoing looking at enzalutamide in the frontline space. There are the ENZAMET and ARCHES studies. There is also the PEACE1 study, which is looking at a variety of combinations. That trial will have some head-to-head data looking at docetaxel plus ADT versus abiraterone plus ADT, also with or without local radiation therapy dependent on the patient population. There are many subgroups in that study, so that will hopefully add some extra data for us to consider when choosing therapy.

There are also trials looking at combinations of all of the above, such as chemotherapy plus a next-generation AR-targeting agent. There is a trial looking at the combination of abiraterone and enzalutamide upfront. There are also multiple large phase III clinical studies that are ongoing, which will probably read out over the next few years and give us more data in which to decide the best possible therapies for our patients.

What is the potential for immunotherapy in this setting?

Immunotherapy in prostate cancer is a complex topic. Of the genitourinary malignancies that we treat, prostate cancer has been the most resistant to experience those responses to immunotherapy that we see in our patients with renal cell carcinoma or urothelial carcinoma. However, there are some strategies that have been looked at to sensitize the local tumor microenvironment in prostate cancer, to try to figure out how to infiltrate that space better or to make the prostate cancer cells seem more visible to the immune system.

Combination therapies using vaccines plus immunotherapy are being looked at. There are multiple National Institutes of Health studies looking at that sort of approach, and there are large studies looking at the addition of immunotherapy to AR-targeted agents. For instance, the KEYNOTE-199 trial is looking at the addition of pembrolizumab (Keytruda) to enzalutamide in patients with metastatic castration-resistant prostate cancer.

These are questions that we are continuing to ask, and we hope that we can find a way to select patients who may have prostate cancer and could respond to immunotherapy.

What are the remaining questions in this population?

It is pretty clear that men with high-volume metastatic disease significantly benefit from the addition of either docetaxel or abiraterone upfront. For even earlier disease states, or disease states where there is not as much disease burden, the question is a little more open. As we get longer follow-up from those trials such as STAMPEDE and LATITUDE, we will get more information about abiraterone in particular.

The other question is sequencing. Does it make sense to do AR-targeted therapy first and then chemotherapy, or vice versa? Where do combinations fit in? There is also the potential for immunotherapy. Those are all open questions asking how to best combine agents and sequence them. That is the purpose of a lot of these trials, and hopefully we will get some of these answers in the next few years.

NETWORKING

The original and most valuable activity of the INFORMED PROSTATE CANCER SUPPORT GROUP is “networking”. We share our experiences and information about prevention and treatment. We offer our support to men recently diagnosed as well as survivors at any stage. Networking with others for the good of all. Many aspects of prostate cancer are complex and confusing. But by sharing our knowledge and experiences we learn the best means of prevention as well as the latest treatments for survival of this disease. So bring your concerns and join us.

Please help us in our outreach efforts. Our speakers bureau consisting of Lyle LaRosh, Gene Van Vleet and George Johnson are available to speak to organizations of which you might be a member. Contact Gene 619-890-8447 or gene@ipcs.org to coordinate.

Member and Director, John Tassi is the webmaster of our website and welcomes any suggestions to make our website simple and easy to navigate. Check out the Personal Experiences page and send us your story. Go to: <http://ipcs.org>

Our brochure provides the group philosophy and explains our goals. Copies may be obtained at our meetings. Please pass them along to friends and contacts.

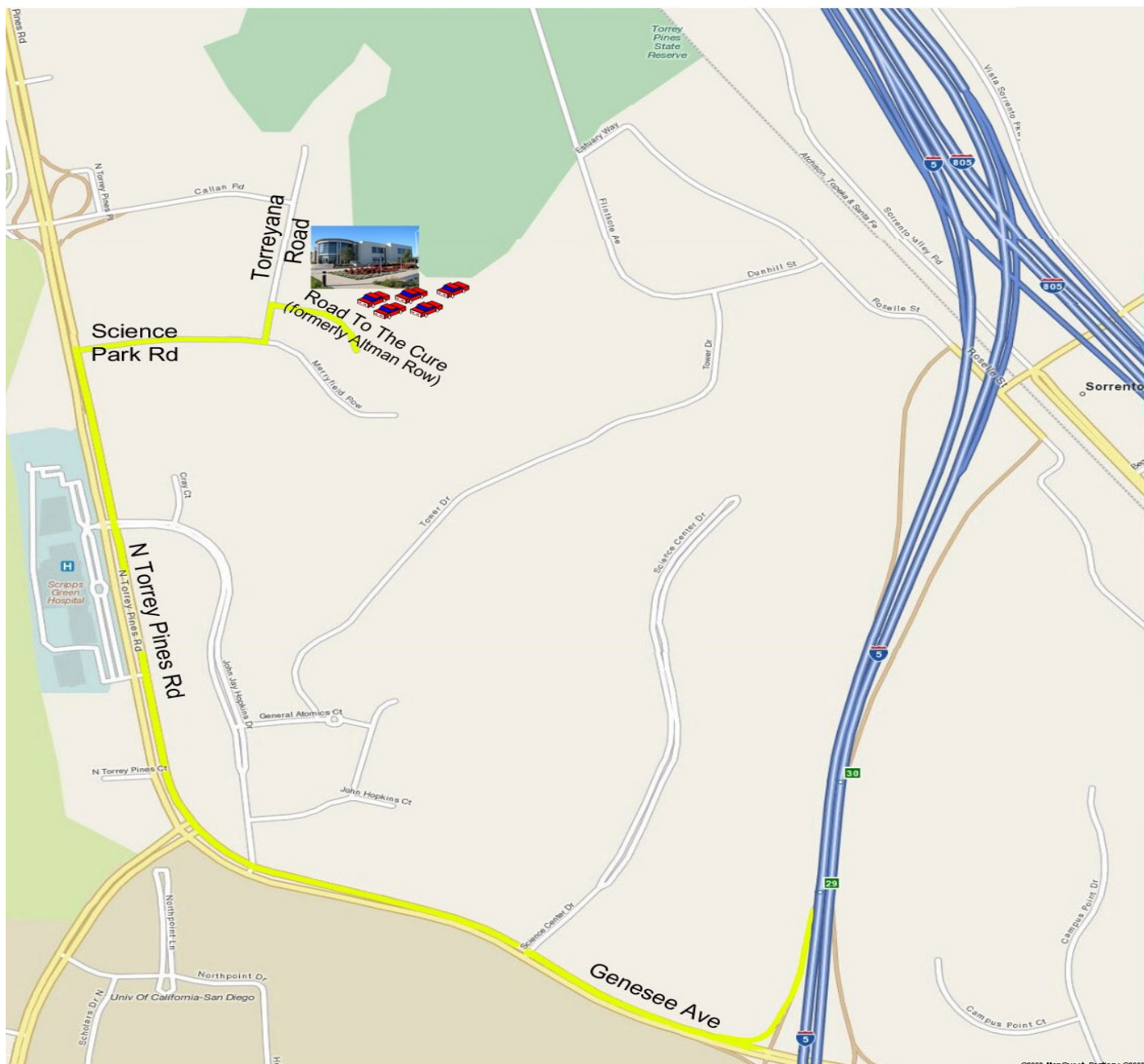
Ads about our Group are in the Union Tribune 2 times prior to a meeting. Watch for them.

FINANCES

We want to thank those of you who have made special donations to IPCSG. Remember that your gifts are tax deductible because we are a 501(c)(3) non-profit organization.

We again are reminding our members and friends to consider giving a large financial contribution to the IPCSG. This can include estate giving as well as giving in memory of a loved one. You can also have a distribution from your IRA made to our account. We need your support. We will, in turn, make contributions from our group to Prostate Cancer researchers and other groups as appropriate for a non-profit organization. Our group ID number is 54-2141691. Corporate donors are welcome!

If you have the internet you can contribute easily by going to our website, <http://ipcs.org> and clicking on “Donate” Follow the instructions on that page. OR just mail a check to: IPCSG, P. O. Box 4201042, San Diego CA_92142



Directions to Sanford-Burnham-Prebys Auditorium **10905 Road to the Cure, San Diego, CA 92121**

Take I-5 (north or south) to the Genesee exit (west).

Follow Genesee up the hill, staying right.

Genesee rounds right onto North Torrey Pines Road.

Do not turn into the Sanford-Burnham-Prebys Medical Discovery Institute or Fishman Auditorium

Turn right on Science Park Road. Watch for our sign here.

Turn Left on Torreyana Road. Watch for our sign here.

Turn Right on Road to the Cure (formerly Altman Row). Watch for our sign here.