

Informed Prostate Cancer Support Group Inc.

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November 2019 NEWSLETTER

P.O. Box 420142 San Diego, CA 92142 Phone: 619-890-8447 Web: http://ipcsg.org We Meet The Third Saturday of Each Month (except December)



Wednesday, November

Volume 12 Issue 11

Next Meeting: November 16, 2019 - Dr. Richard Lam - Hormone therapy for Prostate Cancer: Past, Present, and Future 10:00am—12:00 @ Sanford Burnham Prebys Medical Discovery Institute Auditorium



A double board-certified internist and oncologist, Richard Lam, MD, has been specializing full time at Prostate Oncology Specialists in the treatment of prostate cancer since 2001. Dr. Lam will present the most up to date Prostate Cancer information in an informative and easily understood format. He also brings a great wit with a touch of humor that makes this a presentation not to be missed!.

- For further Reading: https://spendergast.blogspot.com/2019/03/prostate-cancer-news-of-interest-for.html
- For Comments, Ideas and Questions, email to Newsletter@ipcsg.org

October 2019 Informed Prostate Cancer Support Group Meeting:
Prostate Cancer Management: When to Pull the Ripcord
By Phranq D. Tamburri, NMD
Summary by Bill Lewis

Dr. Tamburri is Medical Director of Prostate Second Opinions, in Phoenix, Scottsdale and Seattle. He is an expert in the field of prostate cancer assessment, diagnosis, and treatment from a balanced natural and allopathic perspective. He has been teaching this subject to doctors for over 20 years.

The presentation was directed toward helping surgery- and radiation-averse men decide when conventional treatment options become necessary. A doctor-focused article on this issue is available online at https://ndnr.com/mens-health/cap-update-2018-when-to-pull-the-ripcord/

Questions that a new prostate cancer patient (often only armed with a high-PSA report) may have include the following: Do I have cancer? (Yes, all men have some cancer cells in their body.) Do I have "real prostate cancer"? (What's an unreal prostate cancer?) Is it aggressive? (Aggression does not mean what one typically thinks it means. It means Gleason Score! Urologists need the Gleason Score to protect themselves legally in moving forward with treatment. That's why they push for biopsies.) See below for the answer to "Why is my PSA high?" Men also want to know "Is it going to kill me"? (It only kills you if it gets out of the prostate — so that may be a better question.) How long do I have until it gets out? Why do I — or don't I — have symptoms?

A tangential issue is that men ask about erections. The #I cause of erectile dysfunction (ED) is diabetes. Anoth-

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Organization

a 501c3 non-profit organization - all positions are performed gratis



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PROSTATE CANCER—2 WORDS, NOT A SENTENCE 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.

Be your own health manager!!

Meeting 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://ipcsg.org Click on the 'Purchase DVDs' tab.

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

From the Editor

In the Newsletter this Month

We end this year of meetings with what is certain to be an informative talk by Dr. Lam. Last month Dr. Tamburri gave those in the initial treatment decision stage food for thought, and his talk is summarized by Bill Lewis.

Another busy month in the Prostate Cancer field yielded several articles of interest:

- Dr. Phillip J. Koo reports how Next-Generation Imaging is providing information for advanced PCa Management
- Institute of Cancer Research (ICR) reported an annual blood test for the BRCA2 gene could help spot PCa more precisely than PSA.
- Eric C. Kauffman reports on a new robot-assisted radical prostatectomy technique, extended prostatic urethral preservation (EPUP), with enhanced continence outcomes and penile length.
- UCLA receives grant for prostate cancer diagnosis, treatment.
- Higher 'free' testosterone and a growth hormone blood levels are leading indicators for prostate cancer.

Join the IPCSG TEAM

If you consider the IPCSG to be valuable in your cancer journey, realize that we need people to step up and HELP. Call **President** Lyle LaRosh @ 619-892-3888; **Vice President** Gene Van Vleet @ 619-890-8447; or **Meeting facilitator** George Johnson @ 858-456-2492.

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er significant issue is "polypharmacy" – they are taking too many drugs. The #2 cause is cardiovascular problems. Another common cause is psychosomatic – depression or anxiety. Or low testosterone. None of these is the domain of a urologist. ED is essentially always a side effect of a non-prostate medical problem. Ironically, it can also be a side effect of surgery on the prostate – by a urologist.

Some men are anxious to get treatment. Do they want to treat the cancer, or the PSA? As an example, a man started motorcycle riding with a cross-country trip that abused his prostate (too much sitting without moving around) so that his PSA went to 16 by the time he got to Chicago. A biopsy gave 1% of Gleason 6 in a single core of twelve taken. He was told he needed a prostatectomy! Dr. Tamburri asked about a prior PSA test. Two weeks before the trip, for an insurance policy his wife insisted he get, his PSA was measured at 1.3. So, although the biopsy showed he had cancer, that cancer was obviously present when his PSA was 1.3. Should he be treated? That's the kind of question this presentation answers below.

Dr. Tamburri gets into the details with his patients, including the history (as above) and the patient's perspective and goals, such as quality of life vs. longevity. It makes a huge difference. Does he want to kill the cancer – possibly requiring chemotherapy – or live with it – perhaps using natural methods - until dying of something else? Another aspect is the perspective of the spouse (or significant other, including family caretakers) - which may be quite different from the man's. Besides these personalities, it is appropriate to consider how the "personality" of the cancer (mainly the Gleason Score, but because it is a mutation and there are over 1000 variants that have been characterized genetically) and the personality of the physician (and how he is affected by the medical system he works in) factor into treatment decisions.

Dr. Tamburri's perspective on biopsy safety – "Does it spread the cancer?" – is that the lack of a significant uptick in deaths due to prostate cancer when doing biopsies first became common, is strong evidence that the procedure is not harmful in this sense.

Regarding MRI's for patients early in their prostate cancer journey, Dr. Tamburri sees them, along with Color Doppler ultrasound imaging, as only "setting the stage" for a biopsy, because the latter is still needed in order to move forward with treatment. Men have increasingly demanded imaging, but often don't understand

that it doesn't give a definitive answer about the cancer. Hospitals have also promoted MRI's for revenue.

Prostate cancer only becomes fatal if it escapes the prostate – with one exception. If the ureter is blocked, urine can back up into the kidneys, resulting in a heart attack. What happens if the prostate cancer gets out of the prostate "today"? In Dr. Tamburri's experience, if untreated, men begin to have arthritic issues, low energy, and anemia in about 5 years, then would have five years of treatments including drugs like Zytiga or Xtandi, then a number of additional years of advanced treatments.

One advantage of prostate cancer vs. many other cancers is the ability we have to detect it relatively early, so that treatment can begin as soon as it is warranted. Most other cancers are not detected until they are more advanced and serious.

When should treatment stop? When is the cancer gone? What is the patient's goal? Is it to eradicate the cancer, or to survive with maximum quality of life until death comes by some other cause?

A PSA increase may be due to any of three causes: prostatitis (the #1 cause of elevated PSA in the range up to 15), BPH (benign prostatic hyperplasia, which means an increased number of cells in the prostate, and which usually means prostate enlargement), or prostate cancer. Dr. Tamburri created a Venn diagram to show how these causes interrelate. Note that prostatitis means inflammation, and is only due to an infection 10% of the time. Most of the time, physical activity or inactivity (sitting for long periods without moving around) compressing the prostate is the cause. Autoimmune diseases or diabetes (sugar in the blood) can cause general inflammation, which also affects the prostate. Prostate cancer also causes inflammation, so antioxidants and antiinflammatories are recommended as natural methods. Best choices may be fish oils (or high-grade EFA's [essential fatty acids]), and curcumin (especially liposomal curcumin for absorbability), which are supported by scientific evidence.

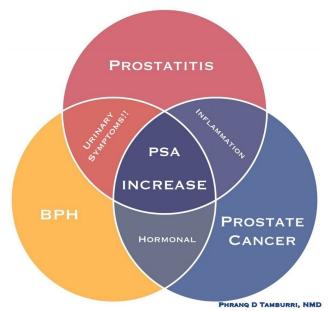
BPH and prostate cancer are related through the effect of testosterone (and dihydrotestosterone), where drugs such as finasteride or dutasteride that reduce or block the hormone(s) can reduce or prevent a PSA rise. Saw palmetto is known to reduce testosterone, but would be ineffective if the cause of the PSA rise is inflammation.

BPH and prostatitis each can cause urinary symptoms, even if no cancer is present. If the PSA is only mildly elevated, then the presence of urinary symptoms

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(frequency, weak stream, etc.) points away from prostate cancer, and toward either or both of these other causes.



In answer to the question of "when to pull the ripcord," Dr. Tamburri presented three levels of risk tolerance. The lowest risk tolerance would be to begin treatment when the PSA is >4, a lesion is suspected based on imaging (MRI or Color Doppler ultrasound), and a needle biopsy confirms the presence of any prostate cancer. Level 2 risk would be a PSA > 10; % free PSA < 10%; PSA increase of >1 unit for 3 consecutive years; a strong history of family prostate cancer or use of carcinogens; a biopsy with >3 positive cores, any core >30%, or any core with Gleason >/= 4+3; any HGPIN (high-grade prostatic intraepithelial neoplasia - considered a precursor lesion to development of invasive prostate cancer); or any perineural invasion. Level 3 risk was presented as a three-canary model – how far into the coal mine a man might go, as the canaries progressively showed poor health (green - yellow - red indicators).

First, PSA density (PSA divided by the prostate volume): 0.0-0.15 (= green), 0.15-0.30 (= yellow),,0.30+ (= red).

Second, Prostatic Acid Phosphatase (PAP; normal is typically around 3; it's an indicator that the cancer has escaped or is about to escape the prostate): lower 2/3 normal (=green), upper I/3 normal (=yellow), abnormal (=red).

Third, T-Status determined by imaging (see full definitions on the internet): T1 /clear margins (=green), T2 /positive margin withOUT extension (=yellow), T3 /positive margin WITH extension (=red).

If all the level 3 risk indicators are red, it's definitely time to pull the ripcord and get active treatment!

Questions:

Is Gleason 6 really cancer? Doctors are wondering about this, since it is very different biologically and in imaging. Can prostate cancer change from 6 to 7, or from 7 to 8, etc.? We don't know. But recent research suggests that such changes may be possible. It's also possible that a small amount of a higher Gleason may have been present early on, but was not detected in a biopsy.

After a prostatectomy, if the PSA goes up, but no cancer tumor can be found, is that really prostate cancer? The simple answer is yes. If the prostate has been removed, any PSA that is generated has to be from prostate cells that have begun to grow somewhere else in the body – whether near or far from the prostate bed.

How does finasteride affect PSA? On average, over several months, it drops the PSA by half. It, and dutasteride, are 5-reductase inhibitors, and are in one class of drugs (expensive, months to full effectiveness, and with significant side effects). Another class of drugs used for urinary symptoms comprises the alpha inhibitors, such as FloMax (much less expensive, effective in days, and few side effects, with no effect on PSA). Note that urinary symptoms are more often caused by a relatively small prostate with a "dense" form of BPH that squeezes the urethra. In contrast, there is a less-dense type of BPH that makes for a large prostate, but rarely causes urinary problems.

Do you recommend turmeric and curcumin for BPH? He recommends curcumin for "everything else," but notes that it can help with inflammation caused by BPH, so in those cases may be useful.

Recommendations for sweating and hot flashes caused by ADT? I. Exercise. 2. Accupuncture. 3. Contrast hydrotherapy – which he finds to be the most helpful. That's saunas and (cold) baths. Saunas were a daily thing among the Japanese when he was in Japan, and provided profound overall health benefits. Unfortunately, they seem hard to fit in with the American way of life. A Jacuzzi? Absolutely.

What about diet? The ketogenic diet is popular. Caloric restriction seems to help. Look at anti-aging literature/organizations, eg., lef.org (Life Extension Foundation). A key issue is to reduce the ratio of sugar to insulin, which can be done with metformin, berberine, and/or cinnamon. Also, avoid foods which cause the blood sugar level to spike upward, because the insulin response is slower, and the sugar in the bloodstream meanwhile

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causes inflammation. This sugar in the blood is why diabetics get erection problems, blindness and increased cholesterol (as the body tries to heal the inflammation caused by the sugar). The anti-aging folks recommend watching out for the glycemic index of foods, and not eating a large meal at the end of the day. Also, increasing fiber intake. Exercising a little after eating, to burn off some of that free sugar. And metformin, which the FDA is considering approving as an anti-cancer drug. Or exercise, berberine & cinnamon if you prefer a more "natural" route.

More details are given in the video of this presentation, including the PowerPoint slides, which will be available for purchase via the website shortly before the next meeting, or at the November meeting on the 16th.

Member Suggested Items:

From Joel Pointon:

GoodRX.com—My Primary Care Doctor at Sharp told me about it when I found a 30 day supply of a medication to be \$400 with insurance at CVS....but less than \$60 for the same drug and a 90 day supply via a mailorder pharmacy. I have also found Pricing at 25% at a Walgreen's across the street from my CVS.

Prostate Cancer Foundation - Wellness Guide https://res.cloudinary.com/pcf/image/upload/ v1567177703/PCF-WellnessGuide-Single-Med f4q1m0.pdf

On the Lighter Side

YOUR MEDICAL DIAGNOSIS OPTIONS









Oh, that. Things got a little slow during your surgery, that's all."







"I HOPE THIS DOESN'T TURN INTO A TRADITION.

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Articles of Interest

Researchers Refining Role of Next-Generation Imaging in Prostate Cancer

https://www.onclive.com/web-exclusives/researchers-refining-role-of-nextgeneration-imaging-in-prostate-cancer

Phillip J. Koo, MD

18F-fluciclovine (Axumin)- and 68Ga-prostate-specific membrane antigen (68Ga-PSMA)—based PET imaging modalities have better sensitivity, specificity, and accuracy in detecting recurrent prostate cancer compared with conventional imaging tools, said Phillip J. Koo, MD.

"It's pretty clear that 18F-fluciclovine and 68Ga-PSMA –based imaging tools perform better than conventional imaging in terms of identifying osseous metastatic disease and soft tissue disease," said Koo. "There's no debate there."

The challenge, he explained, is how next-generation imaging modalities will impact current management strategies. Although there is a growing body of evidence to suggest that using metastasis-directed therapy in conjunction with new-generation imaging could lead to improved outcomes for patients with smaller lesions, more data are needed before that approach replaces conventional imaging.

"It's very easy to fall in love with these great imaging tools, but we need to remember that 99% of all these clinical trials were done with conventional imaging modalities," added Koo. "That's where we have the data."

In an interview during the 2019 OncLive® State of the Science Summit™ on Genitourinary Cancers, Koo, division chief of Diagnostic Imaging, Banner MD Anderson Cancer Center, discussed the use of conventional imaging as well as the next-generation imaging tools that have the potential to change the management of patients with recurrent prostate cancer.

OncLive: Could you discuss the imaging modalities that are being used in prostate cancer?

Koo: [In my presentation], I talked about the use of next-generation imaging. I also discussed the appropriate use of these imaging tools and the impact they have had on the management of patients with prostate cancer.

For decades, next-generation imaging is something that the community has asked for. Our conventional imaging tools have been limited to bone scan and computed tomography (CT). However, we know that we're probably missing a lot of lesions [with these conventional approaches]. It's important [to be able to identify these lesions] because you want to know whether or not the

disease is metastatic and where it might be confined to when you're considering therapy or potentially curative interventions.

Could you expand on the next-generation modalities that have emerged in recent years?

The one next-generation imaging tool that is most widely available and best performing is 18F-fluciclovine. It has been shown to be as good, if not a little better than choline C-11, which was the first novel radiopharmaceutical PET agent that was approved for use in the United States.

Are these modalities widely available?

18F-fluciclovine is widely available in the United States. I believe [it's available] at over 300 sites in the United States. The future of next-generation imaging is going to be focused on PSMA-based imaging agents. The use of these agents varies across the globe. In the United States, we do not have any FDA-approved PSMA PET agent. That being said, several sites in the United States are offering these agents under new drug applications or clinical trials. Once the generic 68Ga-PSMA-11 PSMA, or the commercialized versions of PSMA are FDA approved, we'll see greater availability across the United States.

Is there still a role for conventional imaging in practice?

At this point, it's a little premature to say that we will no longer need conventional imaging. That being said, it's safe to assume that better and more accurate imaging tools will lead to improved outcomes in the future. Therefore, conventional imaging may play a smaller role in the future.

Could you highlight some of the data that we have seen with next-generation imaging tools?

The biggest debate right now is what to do with that information. Many studies have shown that your management decisions will change if you use one of these imaging agents. The biggest question that we need to answer is whether those new management strategies are going to lead to improved outcomes for our patients. Some signals suggest improved outcomes, but there is still a lot more that we need to learn before we alter our practice.

Could you shed light on some of these new management strategies?

The biggest disease state that comes to mind is oligometastatic disease. If a patient presents with biochemical recurrence and a low prostate-specific antigen (PSA) of 0.5 or I, and you order conventional imaging, most likely the bone scan or CT will be negative. If you order one of these next-

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generation imaging tools, there's a high likelihood you might detect locally recurrent disease or local metastatic disease, potentially even oligometastatic disease outside the pelvis.

The question is, "How are we going to manage that patient?" According to surveys and consensus meetings, such as the Advanced Prostate Cancer Consensus Conference, it seems like many providers are treating patients with oligometastatic disease with metastasis-directed therapy. The data regarding the impact of this approach is growing. There's a study from The University of Ghent showing that the time to initiation of androgen deprivation therapy was longer in patients who had a CII-choline PET/CT and then received radiation or surgery on those identified lesions.

Where should research focus in the coming years? Many clinical trials in the United States and globally are ongoing—more than I could list off during this interview. At The University of Texas MD Anderson Cancer Center, and here, [in Arizona], we have a basket trial looking at patients with oligometastatic disease who have difference disease sites. It's very encouraging to see more people trying to tackle this question [of how best to manage these patients]. In the next couple years, we'll have clearer answers. It's also encouraging to see more industry-sponsored efforts looking at ways to incorporate these imaging tools into their trials. These prospective studies will help us answer many remaining questions.

What are the biggest challenge in this space? What steps are being taken to address it?

The biggest challenge right now is getting these PSMA agents approved and into practice. Once PSMA-based therapies are approved, we'll need some sort of companion diagnostic so that we can identify eligible patients. The second unmet need is that we need solid data to tell us that the changes we're making in how we manage our patients is making a significant difference. If it's not, then all we're doing is increasing the cost of care and potentially harming our patients. That information is vital to how we incorporate these tools into our practice in the future.

Is there anything else that is important to emphasize for these patients?

Whenever you order one of these tests, you want to know what you're going to do with the information before you order the test. An actionable result is needed in order for that test to be valuable. Based on a trial that enrolled a SPARTAN-like population, we know that 55%

of patients with nonmetastatic CRPC had NI disease when they were scanned with PSMA. The truth is those patients were actually included in the SPARTAN trial based on their bone scan and CT. However, they still had that 2-year improvement in metastasis-free survival. We need to make sure we stay true to the data.

The other message is that patients with CRPC have aggressive disease. PSA is a very important biomarker, but we know that patients will progress without a rise in PSA. We know this from the PREVAIL data that Alan H. Bryce, MD, of Mayo Clinic, authored that 25% of patients have radiographic progression without a rise in PSA. It's important for us to continue to closely monitor those patients as well.

High-risk men 'should get prostate cancer checks'

Men born at high risk of developing prostate cancer should have extra checks every year from the age of 40, experts say.

Men with certain mutations in their DNA, their genetic code, are more likely to develop prostate cancer.

Scientists at the Institute of Cancer Research (ICR) said an annual blood test could help spot tumours early, when they were easier to treat.

Prostate Cancer UK said any decisions needed to be made carefully.

The ICR researchers said about one in 300 men in the UK had mutations in Brca2, which increases their risk.

However, most will not know whether they carry the mutation in their DNA as it is not routinely tested for.

Brca2 mutations are the same genetic errors that increase the risk of breast and ovarian cancer in women - and men will have been tested for it only if such cancers run in the family.

Screening

Prostate-specific antigen (PSA) is a protein made only by the prostate gland.

PSA levels go up with prostate cancer but it is not a sufficiently reliable measure to justify screening all men for the disease.

Prof Ros Eeles said: "Our research shows very clearly that men with the Brca2 gene fault are at increased risk of aggressive prostate cancer and that regular PSA testing could go some way to improving early diagnosis and treatment.

She has been making the case at the National Cancer Research Institute Cancer Conference, in Glasgow.

She said men or anyone with a prostate and Brca2

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mutations were nearly twice as likely to have a severe cancer that needed treatment rather than simple monitoring.

Dr Matthew Hobbs, from the charity Prostate Cancer UK, said: "[We are] funding a project to model the long-term effectiveness of a range of potential screening strategies, including defining whether there are certain high-risk groups for whom the benefits of regular screening greatly outweighs the potential for overtreatment.

"It may be that screening all men with a Brca2 mutation could be one of the answers, so we will look carefully at the results of this study."

Feasibility and continence outcomes of extended prostatic urethral preservation during robot-assisted radical prostatectomy

Eric C. Kauffman

https://www.nature.com/articles/s41391-019-0173-y

Abstract Background

The prostatic urethra is conventionally resected during robot-assisted radical prostatectomy (RARP). We describe the technical feasibility and urinary continence outcomes of extended prostatic urethral preservation (EPUP) during RARP.

Methods

A single surgeon at a National Comprehensive Cancer Network institute performed 48 consecutive RARP operations using EPUP from March 2014 to March 2016, during which time 177 conventional non-EPUP RARP operations were performed by other surgeons. Prior to this period, the EPUP surgeon had performed 17 non-EPUP RARP operations over 15 months. Total intracorporeal urethral length (IUL) preserved during EPUP was measured intraoperatively. Associations of EPUP and IUL with continence recovery rates and/or times were tested in Fisher's exact and log rank univariate analyses and Cox logistic regression multivariable analyses.

Results

Median IUL preserved during EPUP was 4.0 cm (range 2.5–6.0 cm), and urethral dissections typically spanned the prostatic apex to mid-gland or base. Seven-week continence rates were significantly higher with versus without EPUP. EPUP patient rates of using 0 or 0–1 pads per day immediately after catheter removal were 19% and 35%, respectively. These rates increased significantly (53% and 76%, respectively), as did the IUL preserved

(median 5.0 cm), among more recent EPUP patients (n = 17), which suggested a learning curve. In multivariable analyses including all patients, an EPUP approach was an independent predictor of faster continence recovery. In multivariable analyses of the EPUP subset, a longer IUL preserved was independently associated with faster continence recovery. No EPUP patient had a urethral fossa positive margin, and apical positive margins were similarly infrequent among EPUP and non-EPUP patients.

UCLA receives grant for prostate cancer diagnosis, treatment

https://fox5sandiego.com/2019/10/31/ucla-receives-grant-for-prostate-cancer-diagnosis-treatment/

LOS ANGELES — The prostate cancer program at the UCLA Jonsson Comprehensive Cancer Center and UCLA Health has been awarded an \$8.7 million grant from the National Cancer Institute.

The Specialized Program of Research Excellence grant will support the development of new and innovative approaches for improving the diagnosis, prognosis and treatment of prostate cancer.

The 2019 designation recognizes UCLA's prostate cancer program as one of the best in the country and marks the fourth time it will receive the five- year cycle of funding, according to the university. The program is one of only eight such current programs and the only one to be awarded the designation in the state of California.

"For the past 15 years, the SPORE grant has played a pivotal role in bringing a sense of cohesiveness to our program," said the principal investigator of the grant, Dr. Robert Reiter, a professor of urology and director of the UCLA Prostate Cancer Program. "It funds projects that include researchers and scientists from diverse disciplines and backgrounds all around campus, such as chemistry, nanotechnology, radiology, pathology and stem cell biology, to help accelerate our goal of combating prostate cancer."

Under Reiter's leadership, the grant has led to significant discoveries having a major impact on how men with prostate cancer are treated, according to the university. Most notably, the grant helped support the work of Dr. Michael Jung, a UCLA distinguished professor of chemistry and biochemistry, and Dr. Charles Sawyers, a former professor of medicine and molecular pharmacology at UCLA, according to the university.

Both doctors helped develop enzalutamide and apalutamide, testosterone- blocker treatments that can pro-

long life for men who have failed hormone and chemotherapies, according to the university. The drugs have been used by thousands of men with castration-resistant prostate cancer, according to the university.

Developments in imaging for detecting prostate cancer have also been supported through the grant. UCLA was among the first places in the country to employ MRI for detection, diagnosis and management of prostate cancer. Now, MRIs are used regularly to detect and assess the aggressiveness of malignant prostate tumors.

Among American men, prostate cancer is the most common cancer diagnoses and the second-leading cause of cancer-related death. In 2018, there were an estimated 164,690 new cases of prostate cancer and 29,430 deaths from the disease reported in the United States.

Over the next five years, the grant will fund three translational research projects to find better ways to treat men with advanced stages of the disease that involve developing drug inhibitors for men with metastatic castration-resistant prostate cancer, using CAR T cell therapy to treat men with advanced prostate cancer and targeting a protein to help inhibit lethal prostate cancer.

"This technology being funded has the potential to transform the treatment of prostate cancer," said Dr. Michael Teitell, director of the Jonsson Cancer Center. "The support from the SPORE grant makes it possible for our researchers and physicians to bring observations from the clinic into the lab, to better understand why some patients respond, why some don't and to really understand it at the scientific level, so they can develop new drugs and tools to overcome the obstacles that currently exist."

High levels of two hormones in the blood raise prostate cancer risk

https://www.sciencedaily.com/ releases/2019/10/191031204628.htm

Men with higher levels of 'free' testosterone and a growth hormone in their blood are more likely to be diagnosed with prostate cancer, according to research presented at the 2019 NCRI Cancer Conference.

Other factors such as older age, ethnicity and a family history of the disease are already known to increase a man's risk of developing prostate cancer.

However, the new study of more than 200,000 men is one of the first to show strong evidence of two factors that could possibly be modified to reduce prostate cancer risk.

The research was led by Dr Ruth Travis, an Associate Professor, and Ellie Watts, a Research Fellow, both based at the

Nuffield Department of Population Health, University of Oxford, UK. Dr Travis said: "Prostate cancer is the second most commonly diagnosed cancer in men worldwide after lung cancer and a leading cause of cancer death. But there is no evidence-based advice that we can give to men to reduce their risk.

"We were interested in studying the levels of two hormones circulating in the blood because previous research suggests they could be linked with prostate cancer and because these are factors that could potentially be altered in an attempt to reduce prostate cancer risk."

The researchers studied 200,452 men who are part of the UK Biobank project. All were free of cancer when they joined the study and were not taking any hormone therapy.

The men gave blood samples that were tested for their levels of testosterone and a growth hormone called insulinlike growth factor-I (IGF-I). The researchers calculated levels of free testosterone -- testosterone that is circulating in the blood and not bound to any other molecule and can therefore have an effect in the body. A subset of 9,000 of men gave a second blood sample at a later date, to help the researchers account for natural fluctuations in hormone levels.

The men were followed for an average of six to seven years to see if they went on to develop prostate cancer. Within the group, there were 5,412 cases and 296 deaths from the disease.

The researchers found that men with higher concentrations of the two hormones in their blood were more likely to be diagnosed with prostate cancer. For every increase of five nanomoles in the concentration of IGF-I per litre of blood (5 nmol/L), men were 9% more likely to develop prostate cancer. For every increase of 50 picomoles of 'free' testosterone per litre of blood (50 pmol/L), there was a 10% increase in prostate cancer risk.

Looking at the population as a whole, the researchers say their findings correspond to a 25% greater risk in men who have the highest levels of IGF-I, compared to those with the lowest. Men with the highest 'free' testosterone levels face a 18% greater risk of prostate cancer, compared to those with the lowest levels.

The researchers say that because the blood tests were taken some years before the prostate cancer developed, it is likely that the hormone levels are leading to the increased risk of prostate cancer, as opposed to the cancers leading to higher levels of the hormones. Thanks to the large size of the study, the researchers were also able to take account of other factors that can influence cancer risk, including body size, socioeconomic status and diabetes.

Dr Travis said: "This type of study can't tell us why these factors are linked, but we know that testosterone plays a role in the normal growth and function of the prostate and that IGF-I has a role in stimulating the growth of cells in our bodies."

"What this research does tell us is that these two hormones could be a mechanism that links things like diet, lifestyle and body size with the risk of prostate cancer.

NETWORKING

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@ipcsg.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: https://ipcsg.org/personal-experience

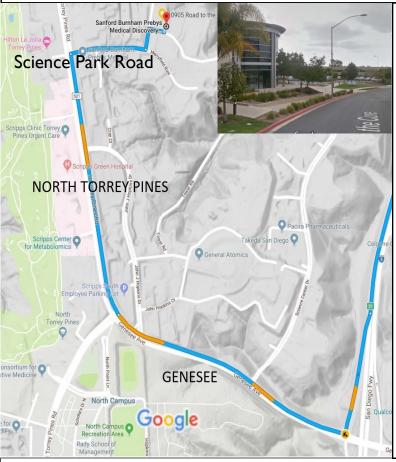
Our brochure provides the group philosophy and explains our goals. Copies may be obtained at our meetings. Please pass them along to friends and contacts.

FINANCES

We want to thank those of you who have made <u>special donations</u> to IPCSG. Remember that your gifts are <u>tax deductible</u> 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://ipcsg.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



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.

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