Informed Prostate Cancer Support Group Inc.
"A 501 C 3 CORPORATION ID # 54-2141691"

December 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)

Thursday, December

Volume 12 Issue 12

Next Meeting: ,

DECEMBER No Meeting. Merry Christmas and a very Happy New Year to you and your loved ones.

JANUARY 18, 2020 10:00am—12:00 @ Sanford Burnham Prebys Medical Discovery Institute Auditorium

Dr. A. J. Mundt is Professor & Chair of Radiation Medicine and Applied Sciences at UCSC, and now also Senior Deputy Director of Moores Comprehensive Cancer Center. .

An internationally-recognized academic radiation oncologist whose career has focused on the development and implementation of novel radiation technologies in a wide number of malignancies, he is an author of over 200 journal articles and book chapters, predominantly focused on advanced radiation technologies, Dr. Mundt has edited 3 academic textbooks, two devoted to intensity-modulated radiotherapy (IMRT) and image-guided radiotherapy (IGRT) with over 100 contributors from the United States, Canada, Europe and Asia

- **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

November 2019 Informed Prostate Cancer Support Group Meeting:

Androgen Deprivation Therapy

By Richard Lam, MD Prostate Oncology Specialists, Marina del Rey, CA

Summary by Bill Lewis

Testosterone is a hormone that stimulates development of male reproductive tissues, and is made throughout life, but gradually less with age. It is produced mainly in the testicles, but also (10%) in the adrenal glands. It stimulates secondary sexual characteristics such as increased muscle, bone integrity, and body hair. It can stimulate growth of both benign and cancerous prostate cells. **Androgen deprivation therapy (ADT)** deprives tumor (and normal) cells of testosterone.

ADT results in programmed cell-death. It is the cornerstone of systemic treatment of advanced prostate cancer. It can be introduced by **surgical castration**, which permanently eliminates 90% of the body's production of testosterone. More commonly, it is accomplished by "medical castration" using injections of an **LHRH** (Luteinizing Hormone Releasing Hormone) **agonist** such as Lupron (leuprolide), Eligard (also leuprolide), Trelstar (triptorelin) or Zoladex (goserelin). Normally, when androgen levels in

(Continued on page 3)

Organization

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

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NEWSLETTER
Table of Contents

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 ended this year of meetings with an informative talk by Dr. Lam on androgent deprivation therapy (ADT) summarized by Bill in this issue. .Also in this issue, we have some articles of interest including:
  - Showing how a high fat diet (such as a keto diet) can fuel prostate cancer progression by imitating a key cancer alteration.
  - An article discussing how testerone replacement therapy can be used after a radical prostatectomy.
  - An article discussing the increased risk of dementia following prolonged ADT.
  - Some positive news about immunotherapy extending life for select advanced PCa patients.
  - News of a home urine test for PCa monitoring and screening which could enhance initial diagnosis.

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.

(Continued from page 1)

the body are low, LHRH stimulates the pituitary gland to produce luteinizing hormone, which in turn stimulates the testicles to produce androgens, including testosterone. LHRH agonists, like the body's own LHRH, initially stimulate the production of luteinizing hormone (which temporarily increases the testosterone level, resulting in the so-called testosterone flare -- which can be prevented by taking Casodex (bicalutamide)). However, the continued presence of high levels of LHRH agonists causes the pituitary gland to stop producing/releasing luteinizing hormone, and as a result the testicles are not stimulated to produce androgens.

A third approach is anti-androgen monotherapy. **LHRH antagonists** such as Firmagon (degarelix; injected) prevent LHRH from binding to its receptors in the pituitary gland. This prevents the secretion of luteinizing hormone, which stops the testicles from producing androgens. This lowers testosterone within 48 hours, and is especially useful for men concerned about flare, or who have bone or spinal cord pain due to a tumor(s).

Androgen receptor blockers/antagonists (also called anti-androgens) are pills such as Casodex (bicalutamide), Xtandi (enzalutamide), Erleada (apalutamide), or Nubeqa (darolutamide) that compete with androgens for binding to the androgen receptor. By competing for binding to the androgen receptor, and in some cases by additional mechanisms, these treatments reduce the ability of androgens to promote prostate cancer cell growth. Since androgen receptor blockers do not block androgen production, they are rarely used on their own to treat prostate cancer. Instead, they are used in combination with either orchiectomy (surgical castration) or an LHRH agonist/antagonist.

Zytiga (abiraterone) pills **inhibit an enzyme called CYP-17**, which results in greatly decreased testosterone production from both testes and adrenals, and even prevents testosterone production within cancer cells. It also reduces cortisol, and prednisone is given as a pseudo-replacement to reduce side effects.

Indications for ADT: Newly diagnosed with intermediate or high risk disease; PSA relapse after initial treatment; metastatic disease; or castrate resistant disease. Not indicated for low risk disease or with surgery.

Intermediate risk prostate cancer is characterized by Gleason score 7, DRE (digital rectal exam): no or small nodule, PSA >10 and <20, >50% of cores involved, and imaging shows organ confined cancer (no extracapsular extension nor seminal vesicle involvement). Adding 4-6 months ADT to radiation improves cure rates, delays relapse and improves survival (average survival over 15 years).

High risk prostate cancer is characterized by Gleason score 8-10, DRE: large nodule, PSA >20, >50% of cores involved, and imaging shows extracapsular extension or seminal vesicle involvement. Adding 18-24 months ADT to radiation improves cure rates, delays relapse and improves survival (average survival >10 yrs). Adding Zytiga to ADT/radiation improves cure rates in super high risk patients.

Metastatic disease is typically controlled by ADT for 2 to 5 years. Several clinical trials have shown how traditional ADT (Lupron or the like) can be improved with Taxotere (docetaxel; Chaarted, 2015; Stampede 2016), Zytiga (Latitude & Stampede, 2017), Xtandi (Enzamet, 2019), or Erleada (Titan, 2019)

When traditional ADT (e.g., Lupron) fails, the cancer is designated as castrate resistant (CRPC) because it is growing despite very low testosterone. Then more advanced ADT is used. For metastatic disease, Zytiga or Xtandi is added. For non-metastatic disease, Erleada (Spartan trial, 2017), Xtandi (Prosper trial, 2019) and Nubeqa (Ararens trial, 2019) have been shown to delay the appearance of metastases by about two years. Nubeqa had fewer side effects (fatigue or seizures) than the other drugs.

Side effects of ADT can include any (typically, 2 – 5) of the following: Hot flashes (treat with medications or acupuncture), Fatigue (typical onset after 2 – 4 months; counteracted with exercise and weight lifting), Breast enlargement (prevented with medication or radiation), Osteoporosis (4-6% density loss per year of ADT; take calcium, vitamin D and/or medication), Muscle wasting (weight lifting),

(Continued on page 4)

(Continued from page 3)

Erectile dysfunction (low libido and erections; low dose Cialis used to prevent penile shrinking), Depression (medication), Weight gain (due to slower metabolism; eat healthy), Diabetes (ditto), High cholesterol (ditto), Anemia (typically only while on ADT), and/or Memory impairment (particularly with long-term ADT. A definite decrease in concentration when on ADT).

Some benefits are seen with **intermittent hormone blockade**: There is less toxicity (less side effects), it is less expensive, and it is less intimidating to patients, but it does not seem to delay hormone resistance. After a period of ADT, when the PSA reaches a targeted low level (see Q&A below) the drug(s) is/are stopped until the PSA rises to a preselected level. Then the cycle is repeated – in some cases, for many years. A typical cycle would be a year on ADT, and then 2 - 4 years of "holiday," with ten years of cancer control (with or without constant ADT). Pioneered by Dr. Lam's associates 20 years ago, intermittent therapy is now widely accepted. Treatment holidays can even be considered with metastatic disease being treated with the new anti-androgens, though the holidays are likely to be shorter.

Questions:

How to minimize lymphedema with ADT? The "utamides" are good. Zytiga, which comes with prednisone, may increase swelling. Radiation may also cause some swelling, but may extend life. It's a balance.

After radiation, when PSA rises, what to do? If the tumor(s) is/are thought to be in the prostate, then it might be treated again. If metastasized, then a decision needs to be made about intermittent ADT, or ADT plus a new anti-androgen, depending on the rate of PSA rise, the original Gleason score, etc.

Should a testosterone level of 800 shortly after surgery be treated to lower it? No.

Recently diagnosed at age 75 with Gleason 7 – what to do? Likely to survive another 15 years no matter what you do (apart from other health problems), so evaluate possible treatments' effects on quality of life. Active surveillance may bring anxiety and require frequent doctor visits. Treatment (radiation), especially if with a short course of ADT, may provide a cure. Intermittent ADT is another possibility. Depends on the individual's priorities.

Where to find trials? It's not particularly easy, but clinicaltrials.gov is a source. Sometimes your doctor will do (or has done) the legwork.

How to lengthen ADT holidays? If oligometastic disease, perhaps irradiation of one or more spots will help. Dutasteride and finasteride help. It's not yet clear if metformin helps.

ADT after radiation: Not needed for low risk disease, needed for high risk disease, and needs to be decided for intermediate risk disease, based on the patient's specific details.

What about focal cryotherapy? Useful when the goal is "less treatment" and reduced side effects. Typically not combined with ADT.

Deciding to add ADT after radiation? It's like insurance. Some want the additional chance of success; others consider the possible benefit not worth the side effects.

Targets for intermittent therapy? The nadir should be PSA below 0.1. Since cancer grows relatively more slowly at first, and then accelerates at some point, you want to stop it before the acceleration. That is typically in the range of PSA = 4 to 8.

Preference for finasteride or dutasteride? Dr. Lam feels they are both equally good.

Switching between Zytiga and Xtandi, when the first one begins to fail – is it effective? For CRPC patients, switching to Zytiga only works for about 1 out of 15 patients. If you reduce the Xtandi (instead of eliminating it), and add Zytiga, about 1 of 8 is benefitted. If Zytiga is used first, and then the switch is made to Xtandi, about 1 out of 4 men "respond." So Dr. Lam prefers to use Zytiga first. A test called ARV-7, developed by a company in San Diego, may predict if switching will help at all (i.e., better than 1 out of 20). Then would it be time for chemo? Yes.

After treatment (of metastatic disease with the PSA originally at about 400) with Casodex, Provenge and Firmagon, staying on the Casodex and Firmagon for some years, with an undetectable PSA and no detectable tumors, should a treatment holi-

(Continued on page 5)

day be considered? Dr. Lam recommended staying on treatment. The patient may get a 1 – 2 year holiday, but then the cancer may come back "angry."

What about Lutetium? In CRPC patients who have had multiple prior treatments, Lutetium works about half the time, and gives a benefit for 4 to 10 months. It's like liquid radiation, that goes to wherever the cancer is, and releases its radiation. Dr. Lam believes that if it were used earlier, at the start of castrate resistance, there would be a benefit of 18 to 36 months. But now it's only being studied for end-stage patients.

How long after starting Zytiga, would you expect to see the benefit? Three months. Sometimes it shows a benefit in a month. How long to stay on Zytiga? Usually "forever," but if you started with very little disease, Dr. Lam would consider a treatment holiday.

Does use of finasteride put you at risk of aggressive prostate cancer? Additional studies have shown that the early idea of such risk is false.

PSA after radiation or surgery? It often goes up for a few months after radiation, and for a month after surgery. So don't focus on that. Sometimes (in about 1 out of 4 men) it also bumps up 18 -24 months after radiation, to 3 – 5, then goes back down in a couple of months -- that is not a danger sign.

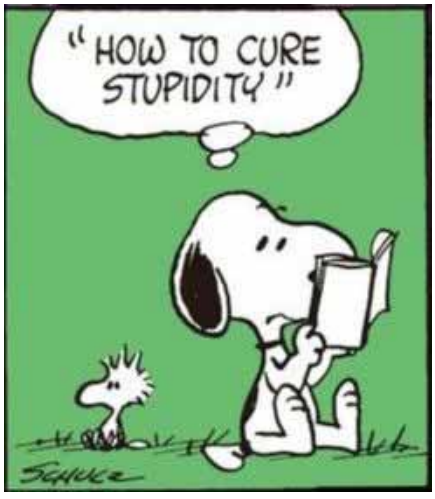
More about physical castration? It's permanent, and less expensive than ongoing ADT. You can take testosterone to create a treatment holiday. Side effects are not worse vs. ADT.

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 January 2020 meeting on the 18th.

On the Lighter Side

"Not a drop of clothes so far. This is our best Christmas ever!"

"Whew! Five surgeries in one day! Well, let's try to make this last one end on a happy note!"



Articles of Interest

High-fat diet proven to fuel prostate cancer progression by imitating a key cancer alteration

by McGill University
medicalxpress.com

What molecular event happens for prostate cancer to progress faster and to be deadlier when patients eat a high-fat diet? This is the question Dr. David P. Labbé, a scientist at the Research Institute of the McGill University Health Centre (RI-MUHC), and his colleagues recently elucidated. In a study published in *Nature Communications*, they showed that saturated fat intake induces a cellular reprogramming that is associated with prostate cancer progression and lethality. These findings could have a clinical utility in identifying patients at higher risk of a more aggressive, lethal disease. In addition, they suggest that dietary intervention involving the reduction of animal fat, and specifically saturated fat consumption in men with early-stage prostate cancer, could possibly diminish or delay the risk of disease progression.

Some genes—called oncogenes—play a role in cancer initiation and progression. MYC is one of those.

"In this paper, we showed that by mimicking a MYC overexpression, saturated fat intake makes prostate cancer worse," says Dr. Labbé, who started this study at the Dana-Farber Cancer Institute in the United States, under the supervision of Dr. Myles A. Brown, Director of the Center for Functional Cancer Epigenetics and Emil Frei III Professor of Medicine at Harvard Medical School.

"MYC overexpression profoundly rewires cellular programs and bolsters a distinctive transcriptional signature. MYC is a key factor in tumorigenesis, i.e. it induces malignant properties in normal cells and fuels the growth of cancer cells," adds Dr. Labbé, who is also assistant professor in the Department of Surgery, Division of Urology at McGill University.

Based on answers to validated food frequency questionnaires obtained from the Health Professionals Follow-Up Study and the Physician Health Study cohorts, researchers were able to stratify prostate cancer patients based on their fat intake—high-fat diet vs. low-fat diet—and the type of fat they were eating—either saturated, monounsaturated or polyunsaturated fat. By integrating dietary and gene expression data from 319 patients, researchers discovered that animal fat and specifically saturated fat consumption mimicked a MYC overexpression. They validated their findings in vivo using a murine prostate cancer model.

Strikingly, patients who had the highest level of the saturated fat intake (SFI) MYC signature were four times more likely to die from prostate cancer, compared to patients with the lowest level, independently of the patient's age or year at diagnosis. Even after adjusting the results for cancer Gleason grade—an indicator of the aggressiveness of the disease—this association remained significative.

Since fat consumption could be linked to an increase in body fat and obesity, and since obesity is also a risk factor associated with prostate cancer, Dr. Labbé used the Body Mass Index (BMI) to make sure that it was only saturated fat intake—and not obesity—that promoted the progression to a metastatic and lethal disease.

"Even after removing obesity from the equation, patients with high levels of the SFI-MYC signature are still three times more likely to die of prostate cancer," says Dr. Labbé. "Epidemiological studies have previously reported that saturated fat intake is associated with prostate cancer progression. Our study provides a mechanistic underpinning to this link and a basis to develop clinical tools aimed at reducing the consumption of saturated fat and increasing the odds of surviving."

The study also showed that for saturated fat to induce MYC reprogramming, the tissue needs to be transformed.

"In a prostate cancer patient, the prostate contains both tumour and normal tissue," explains Dr. Labbé. "We showed that saturated fat intake only affects the transcriptional program in the tumour tissue."

"Altogether, our findings suggest that a substantial subset of prostate cancer patients, including some without MYC amplification, may benefit from epigenetic therapies targeting MYC transcriptional activity or from dietary interventions targeting metabolic addictions regulated by MYC."

Knowing the dietary pattern of a patient or his level of physical activity, clinicians could eventually suggest some specific intervention to decrease the likelihood of progression to a lethal disease. But in order to do that, more research is needed.

"The impact of diet on cancer development has been first established more than 100 years ago. However, lifestyle-related data is only sparsely collected among patients, thereby limiting our capacity to define the mo-

(Continued on page 7)

(Continued from page 6)

lecular link between lifestyle factors and cancer initiation, progression and lethality," says Dr. Labbé. "We will start soon here at the RI-MUHC to gather dietary and physical activity and assess body fatness information from patients undergoing screening tests for different cancers. And with that data, combined with research in the laboratory, we hope to be able to build personalized interventions for patients who are more at risk of having their cancer progress rapidly, and to ultimately improve outcomes."

More information: David P. Labbé et al. High-fat diet fuels prostate cancer progression by rewiring the metabolome and amplifying the MYC program, *Nature Communications* (2019). DOI: 10.1038/s41467-019-12298-z

Citation: High-fat diet proven to fuel prostate cancer progression by imitating a key cancer alteration (2019, November 29) retrieved 29 November 2019 from https://medicalxpress.com/news/2019-11-high-fat-diet-proven-fuel-prostate.html

ascopubs.org

Impact of testosterone replacement therapy after radical prostatectomy on prostate cancer outcomes.

Brent Shane Rose

https://ascopubs.org/doi/abs/10.1200/JCO.2019.37.7_suppl.100

Background: Currently there is little data to guide the use of post-radical prostatectomy (RP) testosterone replacement therapy in prostate cancer. We sought to evaluate the impact of post-RP testosterone replacement on prostate cancer outcomes in a large national cohort.

Methods: We conducted a population-based cohort study using the Veterans Affairs Informatics and Computing Infrastructure. We identified node-negative and non-metastatic prostate cancer patients diagnosed between 2001-2015 treated with RP. We excluded patients for missing covariate and follow-up data. We then coded receipt of testosterone replacement after RP as a time-dependent covariate. Other covariates included: age, Charlson Comorbidity index, diagnosis year, body mass index, race, PSA, clinical T/N/M stage, Gleason score, and receipt of hormone therapy. Biochemical recurrence was defined as a post-RP PSA≥0.2. We evaluated prostate cancer-specific survival, overall survival, and biochemical recurrence free survival using multivariable Cox regression.

Results: Our cohort included 28,651 patients, of whom 469 (1.6%) received testosterone replacement after RP. Median follow up was 7.4 years. There were no differences in clinical T stage, median post-RP PSA (testosterone: 0 non-testosterone: 0; p = 0.18), or hormone therapy use between treatment groups. Testosterone patients were more likely to be of younger age, have higher comorbidity, non-black, have a lower median pre-treatment PSA (5.0 vs 5.8; p < 0.001), and have higher BMI. The median time from RP to TRT was 3.0 years. After controlling for potential confounders, we found no difference in prostate cancer specific mortality (HR 0.73; 95% CI 0.32-1.62; p = 0.43), overall survival (HR 1.11; 95% CI 0.86-1.44; p = 0.43), non-cancer mortality (HR 1.17; 95% CI 0.89-1.55; p = 0.26) biochemical recurrence free survival (HR 1.07; 95% CI 0.84-1.36; p = 0.59) between testosterone users and non-users.

Conclusions: Our results suggest that testosterone replacement is safe in prostate cancer patients who have undergone RP, though prospective data is necessary to confirm this finding.

Risk of dementia following androgen deprivation therapy for treatment of prostate cancer

Quoc-Dien Trinh

nature.com

Abstract

Background

Evidence for androgen deprivation therapy (ADT) and risk of dementia is both limited and mixed. We aimed to assess the association between ADT and risk of dementia among men with localized and locally advanced prostate cancer (PCa).

Methods

We conducted a retrospective cohort study using SEER-Medicare-linked data among 100,414 men aged ≥ 66 years and diagnosed with localized and locally advanced PCa (cT1–cT4) between 1992 and 2009. We excluded men with a history of stroke, dementia, or use of

(Continued from page 7)

psychiatric services. Men were followed until death or administrative end of follow-up at 36 months. Inverse-probability weighted Fine-Gray models were used to estimate hazard ratios (HR) and 95% confidence intervals (CI) for Alzheimer's, all-cause dementia, and use of psychiatric services by duration of pharmacologic ADT (0, 1–6, and ≥ 7 months).

Results

Among 100,414 men with PCa (median age 73 [IQR: 69–77] years; 84% white, 10% black), 38% (n = 37,911) received ADT within 6 months of diagnosis. Receipt of any pharmacologic ADT was associated with a 17% higher risk of all-cause dementia (HR 1.17, 95% CI 1.07–1.27), 23% higher risk of Alzheimer's (HR 1.23, 95% CI 1.11–1.37), and 10% higher risk of psychiatric services use, though the confidence interval included the null (HR 1.10, 95% CI 1.00–1.22). Longer duration of ADT (≥7 months) was associated with **a 25% higher risk of all-cause dementia, 34% higher risk** of Alzheimer's, and 9% higher risk of psychiatric services, compared with no ADT.

Conclusions

Our study supports an association between pharmacologic ADT and higher risk of all-cause dementia, Alzheimer's, and use of psychiatric services among men with localized and locally advanced PCa.

Download references

Cite this article

Krasnova, A., Epstein, M., Marchese, M. *et al.* Risk of dementia following androgen deprivation therapy for treatment of prostate cancer. *Prostate Cancer Prostatic Dis* (2019) doi:10.1038/s41391-019-0189-3

Download citation

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Prostate cancer 'super responders' live for 2 years on immunotherapy

Charles Bankhead,

sciencedaily.com

Some men with advanced prostate cancer who have exhausted all other treatment options could live for two years or more on immunotherapy, a major clinical trial has shown.

Researchers found that a small proportion of men were 'super responders' and were alive and well even after the trial had ended despite having had a very poor prognosis before treatment.

The study found that one in 20 men with end-stage prostate cancer responded to the immunotherapy pembrolizumab -- but although the number who benefited was small, these patients sometimes gained years of extra life.

The most dramatic responses came in patients whose tumours had mutations in genes involved in repairing DNA, and the researchers are investigating whether this group might especially benefit from immunotherapy.

The phase II clinical trial was led globally by a team at The Institute of Cancer Research, London, and The Royal Marsden Foundation Trust, and involved 258 men with advanced prostate cancer who had previously been treated and become resistant to androgen deprivation therapy and docetaxel chemotherapy.

The study is published today (Wednesday) in the *Journal of Clinical Oncology* and was funded by the drug's manufacturer Merck, Sharpe & Dohme.

Overall, 5 per cent of men treated with pembrolizumab saw their tumours actually shrink or disappear, while a larger group of 19 per cent had some evidence of tumour response with a decrease in prostate-specific antigen (PSA) level.

Among a group of 166 patients with particularly advanced disease and high levels of PSA, the average length of survival was 8.1 months with pembrolizumab.

Nine of these patients saw their disease disappear or partly disappear on scans. And of these, four were super-responders who remained on treatment at the end of study follow-up, with responses lasting for at least 22 months.

A second group of patients whose PSA levels were lower but whose disease had spread to the bone lived for an average of 14.1 months on pembrolizumab.

New larger trials are now under way to test whether men with DNA repair gene mutations in their tumours, or those whose cancer has spread to the bone, should receive pembrolizumab as part of their care.

The study also compared the effectiveness of pembrolizumab in men whose tumours had a protein called PD-L1 on the surface of their cancer cells and those whose tumours did not. Targeting PD-L1 activity with pembrolizumab takes the 'brakes' off the immune system, setting it free to attack cancer cells.

But the study found that testing for PD-L1 was not sufficient to tell which patients would respond to treatment. Men with PD-L1 in their tumours survived 9.5 months compared with 7.9 months for patients without PD-L1 in their tumours.

Identifying better tests to pick out who will respond best will be critical if pembrolizumab is to become a standard part of prostate cancer treatment.

Pembrolizumab was well tolerated, with 60 per cent of patients reporting any side effects and only 15 per cent of patients experiencing grade 3-5 side effects.

Professor Johann de Bono, Regius Professor of Cancer Research at The Institute of Cancer Research, London, and Consultant Medical Oncologist at The Royal Marsden NHS Foundation Trust, said:

"Our study has shown that a small proportion of men with very advanced prostate cancer are super responders to immunotherapy and could live for at least two years and possibly

"We don't see much activity from the immune system in prostate tumours, so many oncologists thought immunotherapy wouldn't work for this cancer type. But our study shows that a small proportion of men with end-stage cancer do respond, and crucially that some of these men do very well indeed.

"We found that men with mutations in DNA repair genes respond especially well to immunotherapy, including two of my own patients who have now been on the drug for more than two years. I am now leading a larger-scale trial specifically for this group of patients and am excited to see the results."

Professor Paul Workman, Chief Executive of The Institute of Cancer Research, London, said:

(Continued from page 8)

"Immunotherapy has had tremendous benefits for some cancer patients and it's fantastic news that even in prostate cancer, where we don't see much immune activity, a proportion of men are responding well to treatment.

"A limitation with immunotherapy is that there's no good test to pick out those who are most likely to respond. It's encouraging to see testing for DNA repair mutations may identify some patients who are more likely to respond, and I'm keen to see how the new, larger trial in this group of patients plays out."

Home urine test for prostate cancer could revolutionize diagnosis

Shaun Mehr, Associate Publisher,
sciencedaily.com

A simple urine test under development for prostate cancer detection can now use urine samples collected at home -- according to new research from University of East Anglia and the Norfolk and Norwich University Hospital.

Scientists pioneered the test which diagnoses aggressive prostate cancer and predicts whether patients will require treatment up to five years earlier than standard clinical methods.

Their latest study shows how the 'PUR' test (Prostate Urine Risk) could be performed on samples collected at home, so men don't have to come into the clinic to provide a urine sample -- or have to undergo an uncomfortable rectal examination.

This is an important step forward, because the first urination of the day provides biomarker levels from the prostate that are much higher and more consistent. And the research team hope that the introduction of the 'At-Home Collection Kit' could revolutionise diagnosis of the disease.

Lead researcher Dr Jeremy Clark, from UEA's Norwich Medical School, said: "Prostate cancer is the most common cancer in men in the UK. It usually develops slowly and the majority of cancers will not require treatment in a man's lifetime. However, doctors struggle to predict which tumours will become aggressive, making it hard to decide on treatment for many men.

"The most commonly used tests for prostate cancer include blood tests, a physical examination known as a digital rectal examination (DRE), an MRI scan or a biopsy.

"We developed the PUR test, which looks at gene expression in urine samples and provides vital information about whether a cancer is aggressive or 'low risk'.

"Because the prostate is constantly secreting, the collection of urine from men's first urination of the day means that the biomarker levels from the prostate are much higher and more consistent, so this is a great improvement.

"Being able to simply provide a urine sample at home and post a sample off for analysis could really revolutionise diagnosis.

"It means that men would not have to undergo a digital rectal examination, so it would be much less stressful and should result in a lot more patients being tested."

The research team provided 14 participants with an At Home Collection Kit, and instructions. They then compared the results of their home urine samples, taken first thing in the morning, with samples collected after a digital rectal examination.

"We found that the urine samples taken at home showed the biomarkers for prostate cancer much more clearly than after a rectal examination. And feedback from the participants showed that the at home test was preferable.

"Using our At Home test could in future revolutionise how those on 'active surveillance' are monitored for disease progression, with men only having to visit the clinic for a positive urine result. This is in contrast to the current situation where men are recalled to the clinic every six to 12 months for painful and expensive biopsies.

"Because the PUR test accurately predicts aggressive prostate cancer, and predicts whether patients will require treatment up to five years earlier than standard clinical methods -- it means that a negative test could enable men to only be retested every two to three years, relieving stress to the patient and reducing hospital workload."

The Norfolk and Norwich University Hospital receives more than 800 referrals a year to investigate and treat potential prostate cancers. Prostate cancer usually develops slowly and the majority of cancers will not require treatment in a man's lifetime.

Robert Mills, Consultant Surgeon in Urology at the Norfolk and Norwich University Hospital, said: "This is a very exciting development as this test gives us the possibility of differentiating those who do from those who do not have prostate cancer so avoiding putting a lot of men through unnecessary investigations.

"When we do diagnose prostate cancer, the urine test has the potential to differentiate those who need to have treatment from those who do not need treatment, which would be invaluable. These patients go on to an active surveillance programme following the diagnosis which may involve repeat biopsies and MRI scans which is quite intrusive. This urine test has the potential to tell us whether we needed to intervene with these patients."

The research team say that their findings could also help pioneer the development of home-collection tests for bladder or kidney 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 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://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

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.