BREAKING NEWS ADT May Offer Some Protection From COVID-19 in Men with Prostate Cancer

A PCF-funded study of men with prostate cancer in Italy suggests that men with prostate cancer who were taking ADT were 4 times less likely to be infected with the coronavirus than men who were not on ADT (androgen deprivation therapy), and 5 times less likely to die. These new findings are a game-changer and may lead to potential treatments for COVID-19, even in men without prostate cancer.

https://www.pcf.org/blog/breaking-news-adt-may-offer-some-protection-from-covid-19-in-men-with-prostate-cancer/

Prostate Heidelberg

May 2020 Issue 194

For Education, Information and Support

Meeting Hall: Ivanhoe Uniting Church 19 Seddon Street, Ivanhoe
POB 241 Ivanhoe Victoria 3079
Email: prostateheidelberg@gmail.com
Website: www.prostateheidelberg.info

NEXT MEETING: Tuesday 19 May 2020 (Via ZOOM)
10am – 12:30pm

PHCSG's first Zoom meeting

PHCSG's first Zoom enabled meeting on 6 May went pretty smoothly, with fourteen attendees. As one participant commented, it was the first totally face to face meeting we've had, courtesy of the gallery screen format!

Jim Lloyd and Debra Garroun from PCFA joined the meeting. They reported that the PCFA is facing significant short term cashflow problems, courtesy of the financial pressures on corporate donors and the suspension of community fundraisers caused by social distancing. Most support groups are managing to stay in touch via the internet.

We then had a typically open and broad ranging discussion of individual progress and issues. Nobody reported COVID-19 related treatment problems, though many routine consultations had shifted to telehealth. Debra indicated that the relaxation of the constraints on elective surgery would likely facilitate the resumption of non-urgent prostatectomies.

In terms of future meetings, any shift back to our normal arrangements will first depend on Victorian Government changes to social distancing rules. But we will also need to consider the generally high risk profile of our membership in terms of gender, age and co-morbidity and what this might mean for the timing and organisation of future meetings. For at least the next couple of months, therefore, we would propose to continue with Zoom meetings, moving back to the third Tuesday in the month at 10:00am (including invited speakers), as follows:

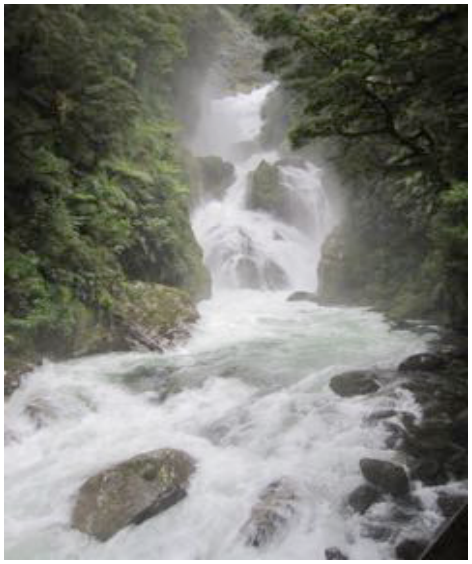
Tuesday 19 May (Zoom meeting)
Tuesday 16 June (Zoom meeting) Professor Grant McArthur
Executive Director of the Victorian Comprehensive Cancer Centre
Mike 0438 616 240

Prostate Heidelberg Cancer Support Group

PHCSG provides information, education and support for those affected by Prostate Cancer. At our meetings we are committed to:

- showing respect to members, speakers and guests
- allowing members to speak without interruption
- respecting confidentiality

TULSA – Novel MRI-guided ultrasound treatment destroys prostate cancer



Source: http://oncologynews.com.au/novel-mri-guided-ultrasound-treatment-destroys-prostate-cancer/

Transurethral Ultrasound ablation

The Tulsa method is designed to deliver precise doses of sound waves to diseased prostate tissue while sparing the healthy nerve tissue that surrounds the prostate. The method rivals current techniques that lack sophisticated imaging guidance and temperature monitoring, according to a news release from the Radiological Society of North America (RSNA).

Tulsa (TULSA –PRO available from Siemens Healthineers in Australia) uses a rod-shaped device inserted into the urethra with 10 ultrasound-generating elements that can cover the entire prostate gland. The elements are controlled automatically by a software algorithm that can adjust the shape, direction and strength of the ultrasound beam.

The procedure takes place in an MRI scanner, offering doctors the ability to monitor treatment. Researchers at the annual meeting of the RSNA added that the incision-free technique could also be applied in the treatment of benign enlargement of the prostate gland.

Data from a 115-man study of the Tulsa-Pro ablation clinical trial revealed that clinicians delivered Tulsa treatment to the entire gland in treatments that lasted an average of 51 minutes. Clinically significant cancer was eliminated in 80% of participants at 12 months.

Of 111 men, 72 (65%) showed no evidence of any cancer at biopsy after one year, while blood levels of prostate-specific antigen (a prostate cancer marker) fell by a median of 95%. Tulsa showed low rates of severe toxicity and no bowel complications as well.

"Unlike with other ultrasound systems on the market, you can monitor the ultrasound ablation process in real-time and get immediate MRI feedback of the thermal dose and efficacy," study co-author and director of prostate MR imaging and interventions and prostate MR imaging research at the University of California at Los Angeles Dr. Steven Raman said in the release. "It's an outpatient procedure with minimal recovery time."

"There are two very unique things about this system," added Raman. "First, you can control with much more finesse where you're going to treat, preserving continence and sexual function. Second, you can do this for both diffuse and localised prostate cancer and benign diseases, and including benign hyperplasia."

Update from the ORIOLE Study

https://www.pcf.org/c/treatment-of-oligometastasis-update-from-the-oriole-study/

Whack-a-Mole
A Treatment of Oligometastasis

By Janet Farrar Worthington

To the growing list of strategies for attacking prostate cancer, let us add this approach: **Whack-a-Mole**.

That's how Johns Hopkins radiation oncologist Phuoc Tran, M.D., Ph.D., describes it to his patients. The actual scientific name for this highly sophisticated strategy is stereotactic ablative radiotherapy (SABR, highly focused, intense doses of radiation), for men with *oligometastasis* – up to three small bits of cancer that have broken away from the main prostate tumor and started to grow elsewhere.

Previously, PCF reported on the Baltimore ORIOLE study, led by Tran. Tran was enrolling patients in a small study to see if men with oligometastasis would benefit from SABR in addition to treatment of their primary tumor.

His strategy was a new one – part of a general rethinking of what represents curable prostate cancer. The boundary used to be very clear: prostate cancer was either confined to the prostate or prostate bed, or it wasn't. **Like a light switch with no dimmer, there was no in-between:** a man with only one metastasis was believed to face the same fate, eventually, as a man with widespread metastases. It was just a matter of time.

But Tran believed that the lines of prostate cancer were **not so clear-cut** as scientists had assumed; that instead of two circles – localized and metastatic cancer – that didn't connect, we might be dealing with **a Venn diagram, with oligometastasis as the critical area where the two circles overlap**. "It may be that the window of curability is wider than we thought," he said, and we all hoped that he was right.

Tran and colleagues at Johns Hopkins, Stanford, and Thomas Jefferson University recently published results of the ORIOLE Phase 2 clinical trial in *JAMA Oncology*. The results are promising: 54 men with oligometastasis were randomly assigned either to treatment with SABR or to observation. To detect and keep track of the oligometastases, the study used PSMA-PET scanning, which uses a small molecule linked to PSMA (prostate-specific membrane antigen, found on the surface of prostate cancer cells) as a radioactive tracer. This PSMA-targeting tracer can highlight areas of cancer as small as a BB – much smaller than can be seen on regular PET or CT imaging. "PSMA-PET allows us to treat lesions we otherwise couldn't see," Tran explains. "A CT or bone scan would miss those lesions, and patients would presumably not do as well."

At six months, 61 percent of the men in the observation group progressed – compared to **only 19 percent of the men who received SABR.**

(continued)

"We also saw a **significantly decreased risk of new metastatic lesions** using PSMA PET-CT," says Tran. "The men in the SABR group did considerably better. This is a definite signal that we can perhaps modify metastatic disease."

This was a Phase 2 study, and "we need larger Phase 3 trials," he says. **"But this is very positive, and we hope that in the future, we will be able to change the course of metastatic disease in some men."**

Some interesting points here: First, Tran and colleagues hope that "complete metastatic ablation of oligometastatic prostate cancer may provide an alternative to early initiation of androgen deprivation therapy (ADT)." The key question, Tran says, is, "can we alter the natural history of metastatic prostate cancer with metastasis-directed therapy (MDT)?" They don't know the answer yet. Most men with oligometastatic disease who get these spots of cancer zapped don't experience a complete drop in PSA. This, Tran says, suggests that "residual micrometastases are present but undetectable." Does SABR simply reset the clock – pushing the snooze button? Or, as the scientists hope, does it make the cancer less likely to form new metastases?

"This [study outcome] has the potential to be practice-changing…"

"It's like Whack-a-Mole": In the ORIOLE trial, Tran and colleagues looked for circulating tumor DNA (ctDNA), and identified certain gene signatures that can tell if a man is more likely to respond to SABR. "Patients who don't have these mutations responded very well," he says. They also have learned from this and other research that men with oligometastasis fall into three basic groups. "Some men do really well after one course of SABR," with no recurrence of cancer. A second group of men have a small recurrence. "Another site pops up; a microscopic metastasis that we couldn't see before establishes itself into a macrocsopic metastasis. It's a limited return of cancer and it responds to another round of SABR." Then some men, after a few months, have multiple new areas of cancer. "For these men, the SABR doesn't control the disease at all."

Imagine a green lawn, with one or two dandelions, Tran tells his patients: "You can pluck those two or three weeds, and wait and see. Sometimes you get lucky; sometimes another weed or two pops up, and you pluck them. It's like Whack-a-Mole. You can do that for a while," with repeated SABR treatments. As the scientists reported: "If a single round of MDT arrests the progression of some, but not all, lesions, subsequent rounds of MDT might salvage the remaining disease, until what remains is inadequate to support a metastatic phenotype." Basically, for some men, a treatment strategy. might be to keep knocking the cancer down until, like a prizefighter who's taken one too many blows to the head, it can't get back up. Imagine: **punch-drunk prostate cancer that may still be staggering around, but can't raise a fist. Wouldn't that be nice!**

That probably won't work in every man, Tran says. "Unfortunately, sometimes there will be a whole bunch of seeds all at once, and at that point, you need weed killer all over the lawn," or systemic therapy. However, SABR plus ADT, androgen-blocking drugs, or chemo might one day provide "the multipronged attack required to cure this disease."

Looking ahead: In a follow-up trial, called RAVENS, men with oligometastatic prostate cancer are randomly given either SABR alone, or SABR plus radium-223 (Xofigo®). "What we have seen in the men in that second group – the ones who have more isolated spots of cancer popping up – is, they're not recurring where they received the SABR, but in areas that were microscopic, and commonly in the bone." Radium-223 targets cancer in bone. "It releases a radioactive particle that is very toxic but is so focused that it only kills in a radius of two-three cell depths. It's ideal for microscopic disease."

More and larger studies are needed, but in the future, Tran envisions, men with oligometastasis will require more vigilant monitoring, and ideally, regular follow-up PSMA-PET scanning. "This has the potential to be practice-changing. We are very excited by our results, and by the potential to modulate the course of metastatic prostate cancer."

BE YOUR OWN ADVOCATE

Remember that you need to be your own advocate and take the time to learn as much as you can about treatment options available to you. Understand as much as you can so that you are in a position to ask your doctors all the relevant questions. With study and determination you can find the best treatment for your situation.

Long-term adjuvant ADT improves results of brachy boost therapy in unfavorable-risk prostate cancer patients

TROG 01.03 RADAR, begun in 2003, was a (partly) randomized clinical trial to help optimize therapy of unfavorable-risk patients. It explored such topics as use of Zometa, radiation dose escalation, and optimal duration of androgen deprivation therapy (ADT) when given along with ("adjuvant" to) radiation therapy (RT).

Zometa did not delay progression, which is similar to the STAMPEDE trial finding among men with metastatic hormone-sensitive prostate cancer when it was used without Celebrex.

The external-beam radiation (EBRT) doses they explored (66 Gy, 70 Gy and 74 Gy) were below today's standard of care (78 Gy-82 Gy), so have become irrelevant to current practice.

The assignment to various radiation doses was not randomized.

The benefit of long-term ADT (28 months vs 4 months) with dose-escalated EBRT in unfavorable-risk patients was proved by the DART 01/05 GICOR trial.

Based on the Kishan et al. study, brachy boost therapy may be the preferred treatment option for high-risk patients So we will turn our focus to the only outstanding question that this major trial can still shed light on - what duration of adjuvant ADT is necessary when unfavorable-risk patients are treated with high dose rate brachy-boost therapy (HDRBBT)?

Joseph et al. reported the 10-year outcomes of the TROG 01.03 RADAR trial conducted at 24 sites in Australia and New Zealand. From 2003 to 2007, patients were randomized on 6 vs 18 months of adjuvant ADT . There were 1,051 evaluable unfavorable-risk patients defined as:

Stage T2b-4 or

Stage T2a and Gleason score≥7 and PSA≥10 ng/ml

NCCN risk groups: 31% unfavorable intermediate-risk, 66% high-risk

Patients with positive pelvic lymph nodes (stage N1) or distant metastases (stage M1) on a bone scan/CT were excluded

The HDRBBT treatment given to 237 patients consisted of:

46 Gy in 23 treatments of EBRT followed by 19.5 Gy in 3 HDR brachy treatments (biologically equivalent to 88 Gy if given as EBRT-only)

All patients received 6 months of adjuvant ADT (Lupron) starting 5 months before EBRT

Half were randomized to get 12 extra months of ADT (total =18 months)

Pelvic lymph nodes were not treated

Distant progression (radiographic progression on a bone scan/CT) was the primary endpoint. The 10-year outcomes for those receiving HDRBBT were:

20% distant progression (25% less than 74 Gy EBRT)

2% local progression (71% less than 74 Gy EBRT)

15% bone progression (31% less than 74 Gy EBRT)

9% prostate cancer-specific mortality (25% less than 74 Gy EBRT)

23% all cause mortality (31% less than 74 Gy EBRT)

Distant progression was reduced by 39% by the longer ADT treatment. It was statistically significant even after adjustment for potentially confounding risk factors.

Longer ADT was beneficial independent of RT dose, whether EBRT or HDRBBT

13% of men receiving HDRBBT suffered urethral strictures vs 4% of men receiving 74 Gy EBRT (for full toxicity data, see report)

The cumulative incidence of transition to castration resistance was significantly lower in men receiving 18 months of adjuvant ADT with RT (in an earlier report). This establishes the importance of adding long-term (18 months) ADT for all unfavorable risk patients receiving radiation as a primary treatment. The adjuvant ADT gave better outcomes independent of the radiation dose. The Nabid et al trialproved that 18 months is as useful as 36 months in high-risk patients. But rather than slavish adherence to a single number, NCCN recommends 18 months to 3 years of adjuvant, at the discretion of the doctor.
The patient may wish to get more if:

there are multiple high-risk features (e.g., GS9-10, PSA>20, T3/4, PNI, rare histology, genomic risk)

there is suspicion of lymph node metastases, especially from advanced PET scans

side effects are very tolerable
The patient may wish to get less if:

there are lower risk features (e.g., GS 6-8, PSA<10, T2, no genomic risk)

advanced PET scans (Axumin or PSMA) are negative

side effects are onerous

treatment is entirely extremely hypofractionated (HDR brachy or SBRT monotherapy)

an additional systemic treatment (e.g., docetaxel, Zytiga, Xtandi, Erleada, or Nubiqa) is used experimentally
In an earlier observational meta-analysis, adjuvant ADT did not seem to add benefit to brachy boost therapy. This once again shows the limitation of observational studies. Only randomized clinical trials can provide the definitive proof we desire for decision-making.

Some patients think they can delay the transition to castration-resistance by eliminating or reducing the amount of ADT used with their RT. This shows that does not happen. Castration resistance is a consequence of genomic breakdown that always occurs as the cancer evolves. It may be facilitated by eliminating the hormone-sensitive cells and leaving only castration-resistant cells (this is called "competitive release"). By eliminating cancer cells as early as possible (before metastases have been detected) using HDRBBT and long-term adjuvant ADT, there is an opportunity to cure the disease. We are learning that cancer cells signal other cancer cells via extracellular vesicles to become like them. Even if it does not cure the patient, the profound reduction of the cancer load has a bigger effect on castration resistance than drug resistance does. This phenomenon was also noted in the TOAD randomized clinical trial. After there are metastases, an "evolutionary" personalized strategy (like this one) may be preferable.

Source: https://pcnrv.blogspot.com/2020/04/long-term-adjuvant-adt-improves-results.html

Source:
Dr Jacqueline Schmitt - Manager, Research Programs for PCFA

https://www.prostate.org.au/news-media/news/harnessing-the-immune-system-to-control-prostate-cancer-spread-to-bone/

Harnessing the immune system to control prostate cancer spread to the bone

In a ground-breaking discovery for men with aggressive prostate cancer, Australian scientists have found a new way to make prostate cancer cells that have spread to bone more visible, so that the immune system can more easily recognise and kill them.

The scientists found that the use of an existing drug could reprogram cancer cells to produce proteins that make the cells identifiable for targeting by immune cells. Impressively, the therapy not only blocked the growth of cancers in the bone, it also established immune memory, serving as greater protection against cancer recurrence. While further research is still needed, this approach may one day be used to make immunotherapy more effective for the treatment of aggressive prostate cancers.

Growth of prostate cancers cells in bone, known as bone metastases, occurs in up to 90% of men whose prostate cancer has become resistant to treatment. Bone metastases can occur many years after a man is first diagnosed and treated and scientists believe that these tumour growths are due to dormant prostate cancer cells or sleeping cancer cells. We have written about dormant cancer cells previously in an article called “Why cancer cells go to sleep”.

It is very difficult to detect dormant cancer cells. These tumours are often small and don’t produce symptoms, so patients are often unaware of them and conventional diagnostic tools are unable to “see” them. Dormant cancer cells also commonly operate in slow-metabolism mode, so even sophisticated diagnostic techniques, such as PET scans, may not pick up these tumours.

Interestingly, the real challenge lies not just in detecting these dormant cancer cells, but in stopping them from “waking up” and growing into large destructive tumours.

The project was led by scientists at the Peter MacCallum Cancer Centre, in collaboration with LaTrobe University, Garvan Institute of Medical Research and the University of Melbourne.

The scientists used a mouse model of prostate cancer to study dormant cancer cells in bone. In this model prostate cancer cells called RM1 cells were injected into the mice. These cells move through the blood and settle on the bones where over time they form bone metastases. Animal models like this are very important for scientists to study diseases like cancer. Before being injected into the mice, the RM1 cells were modified with a special dye system that allowed the scientists to “see” the cancer cells in bone using PET scans and differentiate dormant cancer cells from those that were growing.

The scientists were able to locate areas of bone harbouring prostate cancer cells and remove the cells to study the differences between dormant and growing cells. To do this they used a sophisticated state-of-the-art single cell sequencing technique that allowed the characteristics of individual cells to be examined. They found important differences between the cells.

Prostate cancer cells that had remained dormant in the mouse bones were found to express genes that made the cells susceptible to being attacked by the mouse's immune system, but in the growing cancer cells, these genes had been switched off. Many of these genes make proteins that are part of the important type I interferon (IFN) pathway. If IFN is inactive, the immune system does not see the cell as a threat so it will ignore it allowing the tumour to continue to grow.

The scientists went on to study prostate cancer bone metastasis samples removed from men being treated for castrate resistant prostate cancer. These men generously agreed to donate samples of their cancer for medical research. The scientists compared the primary tumours removed from the prostate with the bone metastasis tumours and found that genes associated with immune response including those associated with IFN signalling were reduced in the metastasis samples compared to the primary tumour.

'The confirmation that IFN was important for outgrowth of bone metastases in men with prostate cancer prompted us to look for ways to reactivate IFN production in the actively dividing cancers,' says Dr Katie Owen from Peter Mac, who led the study.

From their observations in the mouse and human studies, the scientists hypothesised that restoring IFN function would stop the cancer cells that were growing in bone. They decided to test drugs from a family of drugs called HDAC inhibitors. HDAC inhibitors have many effects on cancer cells and make them more susceptible to being attacked and killed by the immune system.

Through a series experiments, the research team found that the HDAC inhibitor Entinostat effectively restored IFN function and prevented the growth of prostate cancer cells in bone by increasing immune signals in the cells. In the mice studies, Entinostat was found to reactivate IFN in prostate cancer bone metastases slowing their growth and increasing the survival time of the mice when compared to mice not treated with the drug.

Importantly, Entinostat treatment was seen to trigger the activation of specific T cells that can kill tumours. When the drug was combined with an immune activator bone metastasis was completely eliminated.

'We have now identified how prostate cancer cells persist in bone, by specifically blocking immune proteins that reduce their visibility to the immune system. This opens the doors for new therapeutic approaches aimed at releasing the brakes on these immune pathways to target the cancer cells for immune destruction' says Associate Professor Belinda Parker.

'Future research will confirm whether use of agents that turn on IFN signalling in prostate cancer cells, including HDAC inhibitors, can be harnessed to offer new therapeutic opportunities in men with no current treatment options to combat bone metastasis through immune signalling restoration at the tumour cell level.'

The results from this study provide important information that can be built on to develop new treatments for men with advanced metastatic castrate resistant prostate cancer.

"This is a call to action," says PCFA CEO Professor Jeff Dunn. "Deaths from prostate cancer are avoidable, and we must not slow down the pace of work until Australia's fathers and sons are saved from this disease.

"The breakthrough demonstrates the tremendous value of Australian-based prostate cancer research towards a future free of prostate cancer."

This ground-breaking study was funded through a partnership with PCFA and the Movember foundation as a Movember Revolutionary Team Award. PCFA's research program funds many projects like this that are crucial to understanding prostate cancer and how it can be better detected and treated. Their work is dependent on the generous support of the Australian public.

Consider donating to prostate cancer research today and help the PCFA to support men with prostate cancer, their partners and families.

Prostate Heidelberg Cancer Support Group Meetings

While we are having to distance ourselves and unable to hold face to face group meetings we hope to engage speakers via video conferencing

Guest speakers scheduled for the first half of the 2020:

21 April CANCELLED
Dr. Cleola Anderiesz General Manager, Service Development and Clinical Practice at Cancer Australia "National data to improve cancer outcomes"

16 June
Professor Grant McArthur
Executive Director of the Victorian Comprehensive Cancer Centre
"Research at VCCC from a patient perspective"
Prof Grant McArthur's research interests include discovery of novel drug targets in cancer, targeting oncogenes, immunological effect of targeted therapies, clinical trials of targeted therapeutics, personalised medicine, melanoma, cell cycle control, metabolism and protein synthesis in cancer.

Prostate Cancer Trials

A study to see whether PET scans using a chemical called Exendin can detect metastatic prostate cancer

Status: Recruiting
Trial ID:ACTRN12619000791134
Recruitment date: 28/05/2019

Exendin PET scans are special nuclear medicine imaging scans. These scans can detect cancers whose cells have a chemical, called the GLP1 receptor, on their surfaces. However, we are unaware of any reports of the use of Exendin PET scanning in prostate cancer. The purpose of this study is to determine if prostate cancer can be detected by Exendin PET scans.

You may only be eligible for this study if you have metastatic castrate-resistant prostate cancer and have already been identified as potentially suitable for the study by our research team.

Study details

The study will assess whether PET scans using the chemical called Exendin can detect metastatic prostate cancer. Participants will have had a PET scan using the chemical PSMA (which is the usual type of PET scan used to detect prostate cancer) within the preceding 2 weeks. They will then have a single Exendin PET/CT scan at a major hospital. The procedure will involve:

a) Injecting the study chemical (called [68Ga]-DOTA-Exendin) into an arm vein.
b) Finger prick glucose testing to confirm participant blood sugar levels.
c) An Exendin PET scan 60 minutes after the chemical injection.

We hope this will demonstrate that prostate cancers can be detected by Exendin PET scans. If we can demonstrate that, we will have confirmed that prostate cancers bear GLP1 receptor.

Evaluation of a mainstream model of genetic testing for men with prostate cancer

Status: Recruiting
Trial ID:ACTRN12619000502134
Recruitment date: 28/03/2019

The National Comprehensive Cancer Network guidelines now suggest testing all men with metastatic prostate cancer for germline mutations predisposing to cancer, the purpose of this study is to determine whether this kind of testing is acceptable to patients with prostate cancer.

Who is it for?

You may be eligible for this study if you are an adult who has been diagnosed with prostate cancer.

Study details:

The study involves having a discussion with a member of your treating oncology team about genetic testing for an inherited gene that may have predisposed to developing prostate cancer.

If you agree to the testing, we will collect a saliva sample and send it for testing. The results will be available in about 4 weeks. If your result shows a change in one of the genes that we are testing for, that may have contributed to your development of prostate cancer, then we will refer you for a consultation with the Familial Cancer Service. After your oncologist provides you with the test result, we will provide you with a questionnaire, asking you about your opinion on the testing process.

It is hoped that this study will help determine if the testing process is acceptable to patients with prostate cancer.

Clinical trials may enable you to receive leading edge treatment, but you must also be fully informed of the risks, costs and safety issues of participating.

Internet Resources

Members have found the following websites useful

Prostate Cancer Foundation of Australia for guides & help
www.PCFA.org.au

Australian Cancer Trials
Information on clinical trials
www.australiancancertrials.gov.au

USA Prostate Cancer Foundation (Guide) PDF guide for men newly diagnosed with PC
www.PCF.org/guid

Us TOO International PCa Education (USA) USA PC support groups' information & newsletter
www.UsToo.org

Cancer Council Victoria for general support services
www.CancerVic.org.au

ExMed Cancer Program
Melbourne based 'best practice' exercise medicine program
www.EXMedCancer.org.au

ProstMate (PCFA) A companion to record PC results

Beyond Blue for help with depression and anxiety
HELPLINE 1300 22 4636

Continence Foundation of Australia for assistance with incontinence aids
HELPLINE 1800 33 0066

Australian Advanced Prostate Cancer Support Group for men diagnosed with advanced metastatic PC
www.JimJimJimJim.com

PCRI Prostate Digest (USA) Prostate Cancer Research Institute supporting research and disseminating information to educate and empower patients, families and the medical community www.pcri.org/insights

PAACT Newsletter (USA) Patient Advocates for Advanced Cancer Treatments
www.paact.help/newsletter/

The internet is a good source for research but it should not be trusted to give you answers for your personal care. Always speak to your doctor to clarify any medical advice.

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Max Shub	Facilitator
Mike Waller	Treasurer
Spiros Haldas	Library
David Bellair	Web Site
Michael Meszaros	Welfare Officer
Sue Lawes	Secretary/ Newsletter

PHCSG Meetings 2020 10am – 12:30pm

Tues 18 Feb (guest speaker)
Tues 17 March
Tues 21 April (guest speaker)
Tues 19 May
Tues 16 June (guest speaker)
Tues 21 July
Tues 18 August
Tues 15 September
Tues 20 October
Tues 17 November
Tues 15 December (including Xmas lunch)

Please note that all face to face meetings have been cancelled until further notice. Please check your email regularly for updates from the PHSCG Committee.

Disclaimer: *Information in this newsletter is not intended to take the place of medical advice. Please ask your doctor to clarify any details that may be related to your treatment. PHCSG have no liability whatsoever to you in connection with this newsletter.*