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.

Prostate Heidelberg

August 2020 Issue 197

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 PHCSG Meeting – Tues 18 Aug (via Zoom)

10am – 12:30pm

Join Zoom Meeting
https://us02web.zoom.us/j/89035075254

Our July Zoom meeting was well attended with two new members joining. However we are missing many familiar faces from our previous monthly meetings in Ivanhoe. If you are not familiar with 'Zoom' virtual meetings Max and Mike will be happy to talk you through the process.

In this month's newsletter:

- Key terminology from the AUA that we hope will help members to better understand their diagnoses and treatment options
- The UK, US and Australia have all have been developing liquid biopsies to help doctors come up with personalised treatment plans to enable them to stay one step ahead of PC
- The Perils and Pitfalls of "Treating PSA" in Advanced Prostate Cancer – the first part of an interesting article and why we shouldn't always be fixated on PSA numbers
- Reprogramming immune cells to attack prostate cancer
- Maintenance of sexual activity following ADT

We are also pleased to advise that our guest speaker at our August meeting is urologist Mr David Dangerfield with a special interest in restoring potency in men with ED after prostatectomy.

We want to ensure that everyone stays physically and mentally as well as possible at this difficult time. So please don't hesitate to contact us if there is anything you want to talk through in relation to your treatment or wellbeing.

Max Shub	0413 777 342
Mike Waller	0438 616 240
Michael Meszaros	0407 837 538

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

Source: https://www.auanet.org/guidelines/advanced-prostate-cancer?fbclid=IwAR2awTQPGYL_WpKLFgXnNABzXTHwxp78O24xZFtc-rxBN3y-Gq3kalVxVeA#x14882

Advanced Prostate Cancer Algorithm

KEY TERMINOLOGY (American Urological Association)

Term	Definition
DISEASE STATES	
Biochemical recurrence without metastatic disease	a rise in PSA in prostate cancer patients after treatment with surgery or radiation (PSA of 0.2ng/mL and a confirmatory value of 0.2ng/mL or greater following radical prostatectomy and nadir + 2.0ng/ mL following radiation); this may occur in patients who do not have symptoms
Hormone-sensitive prostate	prostate cancer that has either not yet been treated with ADT or is still responsive to ADT
Castration-resistant prostate cancer	disease progression despite ADT and a castrate level of testosterone (<50 ng/ dL); progression may present as either a continuous rise in serum PSA levels, the progression of pre-existing or new radiographic
High volume metastatic disease	presence of visceral metastases and/ or greater than or equal to four bone metastases with at least one outside of the vertebral column and pelvis
High-risk metastatic disease	disease that has a poorer prognosis in the presence of two of the three following high-risk features: Gleason >8, >3 bone lesions, or measurable visceral metastases
De novo metastatic disease	metastatic disease that is present at the time of initial prostate cancer diagnosis rather than recurring after previous treatment of localized cancer
DISEASE MANAGEMENT	
PSA doubling time	the number of months required for the PSA value to increase two-fold
Conventional imaging	CT, MRI, and 99mTc-methylene diphosphonate bone scan

ADT: androgen deprivation therapy; CT: computed tomography; HRR: homologous recombination repair; LHRH: luteinizing hormone-releasing hormone; mCRPC: met- astatic castration-resistant prostate cancer; MRI: magnetic resonance imaging; PET: positron emission tomography; PSA: prostate specific antigen

Early Evaluation

Clinicians SHOULD

- Obtain tissue diagnosis from primary tumor or site of metastases when clinically feasible in patients without prior histologic confirmation
- Discuss treatment options based on patient life expectancy, comorbidities, preferences, and tumor characteristics
- Treat patients incorporating a multidisciplinary approach
- Optimize pain control or other symptom support and encourage engagement with professional or community- based resources, including patient advocacy groups

Bone Health

Clinicians SHOULD

- Discuss the risk of osteoporosis associated with ADT and assess the risk of fragility fracture
- Recommend preventative treatment for fractures and skeletal-related events, including supplemental calcium, vitamin D, smoking cessation, and weight-bearing exercise, to patients on ADT
- Recommend preventative treatments with bisphosphonates or denosumab to patients at high fracture risk due to bone loss and recommend referral to physicians who have familiarity with the management of osteoporosis
- Prescribe a bone-protective agent (denosumab or zoledronic acid) for mCRPC patients with bony metastases to prevent skeletal-related events

(continued)

BIOCHEMICAL RECURRENCE WITHOUT METASTATIC DISEASE

Prognosis

Clinicians SHOULD

Inform patients regarding the risk of developing metastatic disease and follow patients with serial PSA measurements and clinical evaluation

Perform periodic staging evaluations consisting of cross sectional imaging (CT,MRI) and technetium bone scan in patients who are at higher risk for development of metastases

Clinicians MAY

Utilize novel PET-CT scans as an alternative to or in the setting of negative conventional imaging

Consider radiographic assessments based on overall PSA and PSA kinetics

Treatment

Clinicians SHOULD

Offer observation or clinical trial enrollment

Clinicians SHOULD NOT

Routinely initiate ADT

Clinicians MAY

Offer intermittent ADT in lieu of continuous ADT if ADT is initiated in the absence of metastatic disease

METASTATIC HORMONE SENSITIVE PROSTATE CANCER

Prognosis

Clinicians SHOULD

Assess the extent of metastatic disease (bone, lymph node and visceral metastasis) using conventional imaging

Assess the extent of metastatic disease (high versus low volume)

Assess if the patient is experiencing symptoms from metastatic disease

Obtain a baseline PSA and serial PSAs at a minimum of three to six month intervals after initiation of ADT and consider periodic conventional imaging

Offer genetic counseling and germline testing regardless of age and family history

Treatment

Clinicians SHOULD

Offer ADT with either LHRH agonists or antagonists or surgical castration

Offer continued ADT in combination with either androgen pathway directed therapy (abiraterone acetate plus prednisone, apalutamide, enzalutamide) or chemotherapy (docetaxel)

Clinicians MAY

Offer primary radiotherapy to the prostate in combination with ADT in selected patients with low-volume metastatic disease

Clinicians SHOULD NOT

Offer first generation antiandrogens in combination with LHRH agonists, except to block testosterone flare

Offer oral androgen pathway directed therapy without ADT

(continued)

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.

NON-METASTATIC CASTRATION RESISTANT PROSTATE CANCER

Prognosis

Clinicians SHOULD

Obtain serial PSA measurements at three to six month intervals and calculate PSA doubling time starting at time of development of castration- resistance

Assess for development of metastatic disease using conventional imaging at intervals of six to twelve months

Treatment

Clinicians SHOULD

Offer apalutamide, darolutamide, or enzalutamide with continued ADT to patients at high risk for developing metastatic disease

Clinicians MAY

Recommend observation with continued ADT, particularly for those at lower risk for developing metastatic disease

Clinicians SHOULD NOT

Offer systemic chemotherapy or immunotherapy outside the context of a clinical trial

METASTATIC CASTRATION RESISTANT PROSTATE CANCER

Prognosis

Clinicians SHOULD

Obtain baseline labs and review location of metastatic disease, disease-related symptoms, and performance status

Assess the extent of metastatic disease using conventional imaging at least annually or at intervals determined by lack of response to therapy

Offer germline and somatic tumor genetic testing

Treatment

Clinicians SHOULD

Offer continued ADT with abiraterone acetate plus prednisone, docetaxel, or enzalutamide

Consider prior treatment in sequencing agents and recommend therapy with an alternative mechanism of action

Offer radium-223 to patients with symptoms from bony metastases from mCRPC and without known visceral disease or lymphadenopathy >3cm

Recommend cabazitaxel rather than an alternative androgen pathway directed therapy in patients who received prior docetaxel and abiraterone acetate plus prednisone or enzalutamide

Offer a PARP inhibitor to patients with deleterious or suspected deleterious germline or somatic HRR gene-mutated mCRPC following prior treatment with enzalutamide or abiraterone, and/or a taxane-based chemotherapy

Offer pembrolizumab to patients with mismatch repair deficient or microsatellite instability high CRPC

Clinicians MAY

Offer sipuleucel-T to asymptomatic/minimally symptomatic patients

Offer cabazitaxel to patients who received prior docetaxel with or without prior abiraterone acetate plus prednisone or enzalutamide

Offer platinum-based chemotherapy to patients with deleterious or suspected deleterious germline or somatic HRR gene-mutated mCRPC following prior treatment with enzalutamide or abiraterone acetate, and/or a taxane-based chemotherapy who cannot use/obtain a PARP inhibitor

PLEASE NOTE: The prognoses and treatments in this article are standard of care [SOC] in the US. Treatments may vary in Australia. Please ensure you discuss your diagnosis and treatment options with your consulting specialist.

ASCO 2020: Blood Test Predicts Response to Prostate Cancer Treatment

Source: https://www.pcf.org/news/asco-2020-blood-test-predicts-response-to-prostate-cancer-treatment/ 30 May 2020

A new blood test can predict how well men with advanced prostate cancer will respond to treatment and could replace some of the existing methods used to characterise and track the disease.

The non-invasive test is less painful and cheaper than tissue biopsies and can help pick out men who are less likely to respond at the start of treatment, or those more likely to relapse later on.

This type of blood test, known as a liquid biopsy, could drive more precise patient care – allowing clinicians to tailor treatment for men with advanced prostate cancer, and to stop drugs that are unlikely to work as quickly as possible.

Researchers at The Institute of Cancer Research, London, and The Royal Marsden NHS Foundation Trust analysed traces of cancer DNA that had entered the bloodstream to assess the ability of liquid biopsies to inform and guide treatment of advanced prostate cancer.

The research, presented today at the American Society of Clinical Oncology (ASCO) virtual annual meeting, received funding from Roche and Genentech – the manufacturers of the technology – and from the charities Cancer Research UK, Prostate Cancer UK, the Movember Foundation and the Prostate Cancer Foundation.

The team looked at more than a thousand blood samples from 216 men with advanced prostate cancer who were taking part in a clinical trial looking at the benefit of the targeted drug abiraterone with or without an experimental drug, ipatasertib.

The researchers found that men with high levels of tumour DNA at the start of treatment had a significantly worse outcome – those patients with traces of cancer DNA in the blood saw their disease progress and get worse two and a half months earlier than those negative for ctDNA at the start of treatment.

Next, the team monitored patients with repeat blood tests while they were on treatment, to assess whether liquid biopsies could enable them to predict response to treatment.

Men who responded to treatment had the greatest fall in the amount of cancer DNA in the bloodstream – a 23 per cent reduction – while those who partially responded to treatment had a reduction of 16 per cent.

Conversely, men whose prostate cancer continued to get worse or stayed the same only saw a reduction of 1 per cent or 4 per cent, respectively.

Analysing the DNA from the blood tests, the researchers also found specific genetic changes associated with drug resistance – which indicate that men are at risk of early relapse. They detected mutations in the well-known cancer genes, p53, PTEN and PI3K/AKT.

The ICR – a charity and research institute – is focused on understanding and combating cancer evolution and drug resistance. It has £2 million still to raise for a new Centre for Cancer Drug Discovery, which will open later this year and will house an ambitious 'Darwinian' drug discovery programme.

Next, the team at The Institute of Cancer Research (ICR) and The Royal Marsden is planning to incorporate liquid biopsies into other clinical trials to assess their benefit in predicting men's response to treatment.

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

"Our study shows that a simple blood test could help us track how cancer evolves and responds to treatment – initially as part of clinical trials and eventually in routine care.

"These so-called liquid biopsy tests are minimally invasive, cost-effective and can be performed often and with ease. Tracking prostate cancer with a blood test instead of a painful surgical biopsy could significantly improve patients' quality of life.

"Our study offers further evidence for the huge potential of liquid biopsies to help guide treatment, design personal treatment plans and improve patient outcomes."

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

"Our exciting work to develop liquid biopsies is starting to have a real impact on how doctors can track the way cancers evolve and respond to treatment. These simple blood tests detect traces of cancer circulating in the bloodstream and help us anticipate cancer's next move. They can help doctors come up with personalised treatment plans and to stay one step ahead of the disease.

"This study showcases the value of liquid biopsies for guiding therapy. They are a faster, kinder, more flexible alternative to traditional tissue biopsies and are set to become a gold standard for cancer treatment."

Similar outcomes have been published in Australia and the US

The Perils and Pitfalls of "Treating PSA" in Advanced Prostate Cancer Part 1

https://www.prostatecancer.news/2020/07/the-perils-and-pitfalls-of-treating-psa.html

Friday, July 10, 2020

Prostate Specific Antigen (PSA) is a protein on the surface of all benign prostate cells and most malignant prostate cancer cells. In prostate cancer, expression of PSA is correlated with the size of the tumor [https://europepmc.org/article/med/20846711]. When prostate cancer first metastasizes, the tumor is limited in size by its blood supply. As it grows, the cancer creates its own blood supply by secreting growth factors called VEGF. The PSA from the cancer activates VEGF to form blood vessels that bring oxygen and nutrients to the cancer and lymph vessels to drain fluids from the growing tumor [https://elifesciences.org/articles/44478] Tumor blood supplies are not as patent as those of benign tissues. Healthy prostate tissues with patent blood supply, and micrometastases that have little or no blood supply put out very little detectable PSA into the serum (although the cells express high levels of PSA). But the leaky blood supply of tumors allows PSA to enter the serum where it is detected by a PSA test. So, the larger, more established tumors of a given patient create almost all of his detectable PSA.

"Treating PSA"

I. Selecting for low PSA subtypes

For most men with advanced prostate cancer, PSA is their best biomarker of progression - more detected PSA means more progression. This may change as the cancer evolves. A highly mutated tumor may put out less PSA. Highly undifferentiated kinds of prostate cancer, and other relatively rare sub-types (e.g., ductal, neuroendocrine, basal cell, "double negative," etc.) may evince little or no serum PSA.

So it is possible, when such phenotypes are present and they are mixed with "normal" prostate cancer, to provide treatments that kill off the "normal" prostate cancer cells, leaving the low-PSA subtypes behind. Such a situation has been identified in patients heavily treated with chemo and enzalutamide. It is called "treatment-emergent neuroendocrine prostate cancer" [https://ascopubs.org/doi/full/10.1200/JCO.2017.77.6880] and has been identified in 17% of heavily-treated patients.

Another example of a treatment that *may* select for low-PSA subtypes is Lu-177-PSMA. If the patient has two types of prostate cancer, one that expresses PSMA and PSA, while his other cancer expresses neither, PSMA-targeted therapy may eliminate the source of most of the PSA, leaving more virulent subtypes behind [https://www.prostatecancer.news/2019/12/why-lutetium-177-psma-treatment.html]. This type of situation is dangerous if one relies on PSA as the principal biomarker of progression

It is worth noting that two FDA-approved therapies for prostate cancer, Provenge and Xofigo, have been proven to increase survival, but have little or no effect on PSA.

This is the first part of an article giving an explanation of PCa progression. Look out for part 2 in the September PHCSG newsletter detailing:

PSA-based Endpoints

Danger of Withholding Early ADT

(continued)

II. Supplements that interfere with PSA tests

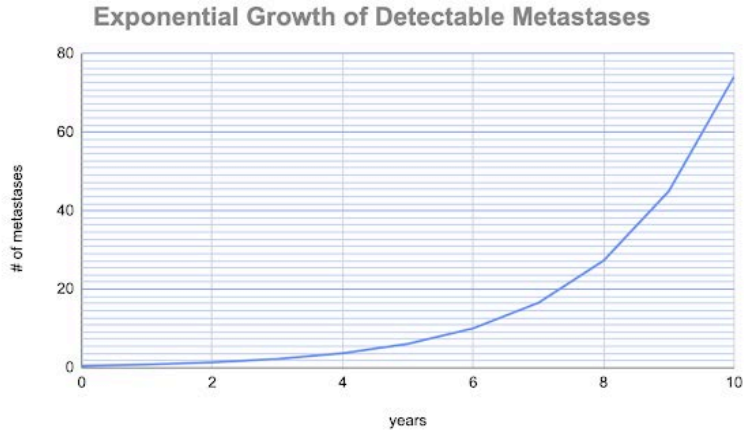
Patients often self-medicate in the hope of wresting some control over their cancer. The internet is full of "evidence" that this or that natural supplement may slow progression or even cure the cancer. Serum PSA is detected by an antibody that can detect amounts as low as a nanogram of PSA per ml of serum. This kind of sensitivity has a cost - the antibodies are subject to interference by other substances that may be present in the serum. So far, the list of substances that *may* interfere with PSA tests, creating false negatives, includes biotin, curcumin, genistein, EGCG, resveratrol, capsaicin, saw palmetto, pygeum, beta-sitosterol, and statins [https://www.prostatecancer.news/2019/04/if-you-are-using-psa-to-monitor-your.html]. The false negative PSA readings may fool the patient and his physician (who may not be aware of the patient's supplement use) into believing that the cancer is under more control than it really is. Patients who use any complementary therapies are twice as likely to die of their cancer [https://www.prostatecancer.news/2018/07/the-danger-of-complementary-and.html].

III. SBRT of oligometastases

1. Exponential growth

Because of Covid-19, many of us are now used to seeing exponential growth curves. Deaths from Covid-19 started very slowly in December through February. But then in March, the number of deaths climbed markedly. This illustrates the two striking features of exponential growth - the "flat" part with a very slow increase, followed by a "steep" part with a very rapid increase.

Among the biological systems that also follow an exponential growth curve are bacteria, viruses, and cancers. Here is a prototypical graph of the number of metastases in a patient.



In men who are PSA-recurrent after prostatectomy, it takes a median of 8 years for the first metastasis to become detectable [https://jamanetwork.com/journals/jama/fullarticle/189741]. After that, I've seen that more than a year can go by between the detection of the first metastasis and the next one. Some researchers, who should know better, observed that in their patients who had early metastases treated with radiation, new metastases did not occur for a long time. They attributed the delay to the treatment rather than the natural history of metastatic progression [https://www.prostatecancer.news/2017/05/unwarranted-conclusions-about.html]. It is impossible to know if there was a delay in progression without a randomized clinical trial.

What is really happening during this extended time period? The accepted theory is called "seed and soil." There are millions of cancer "seeds" in the serum, the lymph, around nerves, and hiding in various tissue reservoirs (mainly in bone tissue). While they appear to be quiescent, they are in fact changing the "microenvironment" of the tissue they are in. They are transforming the tissue to make it more conducive to prostate cancer growth, building networks of collagen, fat, blood vessels and nerves, influencing healthy cells to become cancerous, and preventing the immune system from destroying the new nests [https://www.prostatecancer.news/2019/12/beyond-psma-fapi-theranostics.html].

Because it takes such a long time to build up the metastases to the point that they are detectable by even our most sensitive PET/CT scan (the tumor detection limit is about 4 mm - hundreds of thousands of cells), it *seems* that there is little there and even less going on. This is called "oligometastatic" cancer. It *seems* like all the cancer can be picked off by playing whack-a-mole - zapping the few detected metastases with intense radiation (called SBRT) as they are detected. In fact, it is well-established that SBRT provides excellent "local control." "Local control" means that the metastases are usually completely annihilated by just one or two "zaps" [https://www.prostate.new/2019/08/one-large-zap-for-painful-bine.html]. Because the detected metastases are the source of almost all the PSA, PSA can fall to undetectable levels after such treatment of oligometastases. But the cancer is far from cured - the PSA has been treated, but the cancer is still micrometastatic and systemic.

Those who believe that such treatment can result in a durable remission believe that the immune system can clean up the rest of the cancer. The ORIOLE trial [https://www.prostatecancer.news/2019/09/sabr-to-oligometastases-slowshtml] showed that SBRT created a T-cell response. If that T-cell response is sustained, they argue, the activated immune system can "clean up" the rest of the cancer. The skeptics argue that T-cell responses are usually not sustained. Trials of numerous immunotherapies (e.g., Prostvac, GVAX, GM-CSF, etc.) have failed to show a benefit because the early T-cell responses are countered by adaptive responses. Prostate cancer is notoriously "cold" to immunotherapies.

(continued next month)

‘Reprogramming’ immune cells could switch defence into attack to treat prostate cancer

https://oncologynews.com.au/reprogramming-immune-cells-could-switch-defence-into-attack-to-treat-prostate-cancer/

Drugs that can ‘reprogramme’ immune cells from protecting cancer to attacking it could be an effective new treatment for some men with advanced prostate cancer, a new study shows.

Researchers found that immune cells called macrophages, already drawn into prostate cancer tumours to help promote growth, can be targeted and rewired to attack the disease.

In studies of cancer cells and mice with prostate cancer a so-called CXCR2 inhibitor, which blocks a protein receptor on macrophages, inhibited tumour growth by switching them from helping the tumour to triggering anti-tumour immune responses instead.

The drug, called AZD5069, is now being studied in a clinical trial led by researchers at The Institute of Cancer Research and our partner hospital The Royal Marsden NHS Foundation Trust, to test its effectiveness in men with advanced prostate cancer.

The new research, published in the journal published in the journal *Cell Reports,* was carried out by a European group of researchers led by the Istituto Clinico Humanitas in Milan, Italy, the Institute of Oncology Research in Switzerland, and in the UK by the ICR and The Royal Marsden.

Reprogramming immune cells to treat prostate cancer

Cancer cells are able to hide from the body’s immune system by manipulating their environments to help their growth, and one way they can do this is by recruiting macrophages.

Macrophages are white blood cells that play an important role in the immune system helping to protect the body, removing unwanted microbes and other ‘foreign’ bodies including cancer cells. But in prostate and some other cancers, they can be induced to protect the tumour instead of attacking it.

The new study showed that in some prostate cancers, these tumour-associated macrophages express a protein called CXCR2, which triggers immune responses that can protect tumours and help them grow.

In studies of cancer cells, researchers saw that CXCR2-expressing macrophages infiltrate prostate cancer and sustain tumour growth.

Higher levels of infiltration were associated with more aggressive cancer and worse outcomes for patients with the disease.

Reversing a survival mechanism for prostate cancer

The researchers showed that the CXCR2 antagonist AZD5069 reduced tumour growth in mice with prostate tumours, compared with untreated mice.

The drug reduced anti-inflammatory signals that benefit cancer cells and switched the immune cells to promote inflammation and trigger cell death – reversing an important survival mechanism for prostate cancer.

The researchers were looking in prostate cancer tumours missing a key tumour suppressing gene called PTEN, which is missing or mutated in about half of men with the disease. CXCR2-inhibiting drugs could be a future option for some of these men.

The research was largely funded by the European Research Council, with additional support from funders including the Josef Steiner Foundation.

Professor Johann de Bono, who is leading the clinical trial at the ICR and The Royal Marsden, said:

“Prostate cancer tumours can suck in white blood cells that actually protect them from our immune system, and to help them grow. Re-training these tumour-associated macrophages to target the disease could help treat aggressive prostate cancer.

“This study shows firstly that a receptor on white blood cells called CXCR2 gives tumour-associated macrophages pro-cancer characteristics in prostate cancer – and secondly, that by blocking this receptor can reverse this process in mice and actually turn the macrophages against the disease.

“We are now running a clinical trial to study the effects of CXCR2 inhibition in patients with

Maintenance of sexual activity following androgen deprivation in males

Cassian J.Duthie[a]Hannah J.Calich[b]Charlene M.Rapsey[c]ErikWibowo[a]

https://doi.org/10.1016/j.critrevonc.2020.103064

The following article on "sex after ADT" was the product of Dr. Erik Wibowo and his colleagues in New Zealand and Australia.

It is intended that the article will soon be freely available on LIFEonADT.com.

The core message is that complete loss of sexual interest and sexual performance is very common with ADT, but not inevitable. Various factors can help sustain some level of sexual activity for patients on ADT. These factors include the overall health of the patient, his age, his relationship with his partner before ADT, and the couple's willingness to explore novel sexual practices and aids.

Abstract

Androgen deprivation therapy (ADT) is a common treatment for men with systemic prostate cancer. However, ADT leads to sexual dysfunction, causing >80 % of couples to cease sexual activity completely.

Here, we use a biopsychosocial framework to review factors that may influence the ability of patients on ADT to remain sexually active. We address sexual factors prior to ADT, neurobiological factors, intermittent ADT, sex aids, exercise, sleep, partner factors, masculinity, non-penetrative intimacy, depressive symptoms, and access to counselling or patient education programs. We make suggestions for future research in order to extend our understanding in this field with the goal of improving evidence-based treatment protocols and practice. Importantly, we suggest that clinicians should discuss options for sexual intimacy after ADT with both patients and their partners, as sexual inactivity is not inevitable for most, and strategies are available for helping maintain sexual intimacy.

Please also see the bio for Mr David Dangerfield (page 10), our guest speaker in August, who is establishing a new microsurgery procedure to restore potency to PC men with ED after a prostatectomy.

Prostate Cancer Connections

is a new series of educational and interactive webinars that bring people together virtually and safely to access empowering, decision-making information and personal connections in a time of social distancing. In the first of the three webinars, there is discussion around *how and if men with prostate cancer should talk to their kids about how it could impact their lives*.
Video of this webinar is now available at:

"The Talk" Video:
https://youtu.be/9AQxublTsdA

Topics

- How and If Men with Prostate Cancer Should Talk to Their Kids About How it Could Impact Their Lives
- Passing Cancer to Sons and Daughters
- And Much More!

Prostate Heidelberg Cancer Support Group Meetings

While we are having to distance ourselves and are unable to hold face–to-face group meetings we are engaging speakers via video conferencing

18 August 2020

Mr David Dangerfield Urologist

MBBS, FRACS (Urol)

We are also pleased to advise that our guest speaker at our August meeting is urologist Mr David Dangerfield. Mr Dangerfield is at the forefront of establishing a new microsurgery procedure to restore potency to men who have had the erectile nerve connections to their penis damaged after having their prostate removed as a result of cancer.

Mr David Dangerfield completed his Medical Degree at University of Queensland and completed Urology Specialist Training in Queensland before returning to his home city of Melbourne, where he undertook fellowship training in Advanced Laparoscopy at Monash Health. He was appointed at Monash where he has provided Specialist Urological Services over the last 10 years. David acted as Director of Urology Training at Monash for 3 years, representing Monash Health at the Royal Australasian College of Surgeons. He has also provided private Urological Services to Melbourne's Southeast Suburbs over the last 10 years. He is a member of the Australian Medical Association, Urological Society of Australia and New Zealand and Royal Australasian College of Surgeons.

David has special interest in prostate cancer, kidney stone management, uro-oncology (kidney cancer, bladder cancer, testicular cancer), advanced laporoscopic surgery, voiding dysfunction, vasectomy and erectile dysfunction.

Mr David Dangerfield
Urologist
MBBS, FRACS (Urol)

Prostate Cancer Foundation of Australia

the LONG run

The Long Run is an awareness and fundraising event for Australian men and families impacted by prostate cancer.

All you need to do is run, walk or wheel 72km during September, when and where is up to you.

Every kilometer you cover will raise funds to bring us closer to a world where prostate cancer is no longer a burden and where all men and their families with a diagnosis are supported.

STEP 1 SIGN UP – Register for the challenge

STEP 2 SHARE THE NEWS – Tell everyone you know that you're in it for the LONG RUN. Ask them to help you support men impacted by prostate cancer

STEP 3 GET GOING – put on your sneakers, track your kilometres and help love go the distance today.

Website:
https://www.thelongrun.org.au

Enquiries:
thelongrun@pcfa.org.au

Phone:
1800 22 00 99

Internet Resources

Members have found the following websites useful

PCFA ONLINE COMMUNITY

The new PCFA Online Community is now live.

Log in to contact the online community with any questions –

onlinecommunity@pcfa.org.au

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.

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/

PHCSG Correspondance

Prostate Heidelberg
POB 241 Ivanhoe Vic 3079
prostateheidelberg@gmail.com
prostateheidelberg.info

PHCSG Correspondance

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
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.

2020 PHCSG Articles

If you have any feedback or wish to include articles on specific aspects of Prostate Cancer please contact Sue at:

prostateheidelberg@gmail.com

March 2020

- •PCFA Consumer Advisory- Coronavirus and Cancer

April 2020

- •Telehealth & Delayed Hospital Treatments due to COVID-19
- •Fexapotide Triflutate (FT) injection – a new kind of focal treatment to extend time on active surveillance

Prostate Cancer Trials

- •DASL-HiCaP Trial
- •Evaluation of a mainstream model of genetic testing for men with prostate cancer

May 2020

- •ADT May Offer Some Protection From COVID-19 in Men with Prostate Cancer
- •TULSA – Novel MRI-guided ultrasound treatment destroys prostate cancer
- •Whack-a-Mole A Treatment of Oligometastasis
- •Long-term adjuvant ADT improves results of brachy boost therapy in unfavorable-risk prostate cancer patients
- •Harnessing the immune system to control prostate cancer spread to the bone

Prostate Cancer Trials

- •A study to see whether PET scans using a chemical called Exendin can detect metastatic prostate cancer
- •Evaluation of a mainstream model of genetic testing for men with prostate cancer

June 2020

- •Evaluating the Outcomes of AS in Gleason Grade 2 Prostate Cancer
- •Advancing precision medicine for metastatic prostate cancer
- •Impact of Primary Prostate Cancer Treatment with Subsequent Metastatic Disease
- •Comparative Analysis & Survival Outcomes in a Real-World Practice Setting
- •Fexapotide Triflutate (FT) injection – a new kind of focal treatment to extend time on AS

Prostate Cancer Trials

- •Impact of 18F-DCFPyL PET scanning in patients undergoing post-prostatectomy Radiotherapy

July 2020

- •Testosterone Therapy does not Increase the Risks of PCR or Death after Definitive Treatment for Localised Disease
- •Association of Pre-Salvage Radiotherapy PSA Levels after Prostatectomy with Outcomes of Long-term Antiandrogen Therapy in Men with Prostate Cancer
- •Testosterone Replacement in the treatment of Advanced Prostate Cancer
- •Memorial Sloan Kettering Cancer Center PCa nomograms Prediction Tools

August 2020

- •Advanced Prostate Cancer Algorithm
- •Blood Test Predicts Response to PC Treatment (liquid biopsy)
- •The Perils and Pitfalls of Treating PSA in PCa
- •Reprogramming Immune Cells could Switch Defence into Attack in PCa
- •Maintenance of Sexual Activity Following ADT