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Join our Monthly meetings on the third Tuesday (Feb – Dec) 10am – 12:30pm

EFT Payments to:
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Prostate Heidelberg

February 2022 Issue 215

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 (Zoom) – Tuesday 15 February

10am – 12:30pm

To join via Zoom: Copy link and paste into your browser
https://us02web.zoom.us/j/84450281030?pwd=cnVJZzl5a3cweEljUG5GWjBJSUNBZz09

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

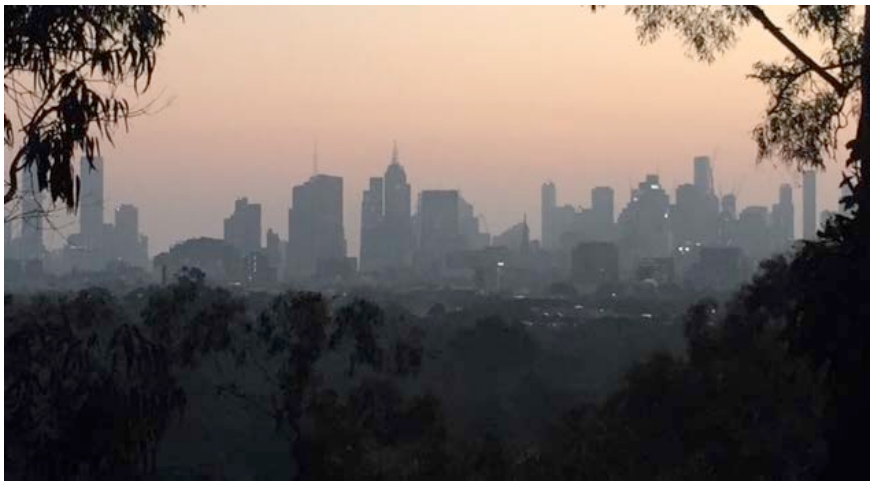
Back to ZOOM Meetings until further notice!

We look forward to welcoming and supporting existing and new members to our first meeting of the New Year on Tuesday 15 February. Please be advised, that as a precautionary measure, it will once again be via Zoom.

In this month's newsletter we highlight:

If there is anything you want to talk through in relation to your treatment or wellbeing please don't hesitate to ring:

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



Why aren't more men with advanced prostate cancer electing to have an orchiectomy rather than repeated ADT depot injections?

Most patients treated for advanced metastatic prostate cancer can expect to go on and stay on ADT for the remainder of their lives. One way to achieve sustained androgen suppression is with the standard LHRH drugs, like Lupron, Eligard and Zoladex. But another way is with a surgical castration (i.e., removal of the testicles), more formally known as an orchiectomy.

Research from more than a decade ago suggests that patients who elect a surgical castration are less anxious compared to those who must repeatedly receive depot injections of an LHRH drug. It is also known that after two years of androgen deprivation, the surgical procedure is less costly than repeated ADT depot injections.

So why aren't more men with advanced metastatic prostate cancer electing to have a surgical castration? A group of urologists in Iowa were curious to know if advanced prostate cancer patients on ADT drugs had ever been offered an orchiectomy as an alternative to repeated depot injections. They surveyed 68 patients on ADT about whether they had been told about the surgical option. They also asked the men whether they would consider surgical castration now that they knew of the option.

Only a third of the men recalled any discussion about an orchiectomy with their treating physicians. However, 40% said that they were interested in the procedure as an alternative to repeated ADT depot injections, once they knew about it.

The authors acknowledged that they "described orchiectomy in a mostly favourable light, specifically as a 30-minute outpatient procedure with minimal side effects…" and one that resulted in a comparable outcome in terms of appearance of the genitals (similar to genital appearance after long-term use of ADT).

It isn't clear whether the men were told that an orchiectomy is the same as "surgical castration." It is already known that the language used to discuss androgen deprivation is emotionally charged (Cushman at al. 2010). We don't know, for instance, if the men knew of Sigmund Freud's concept of "castration anxiety," or if this may have contributed to their view of orchiectomy.

This paper raises many questions about what patients understand 'androgen deprivation therapy' to be. Some men define castration specifically as a surgical procedure; as such, they don't consider themselves castrated if androgen suppression is achieved with ADT medications. A standard sales pitch for the ADT drugs is that the effects are reversible. However, this is irrelevant for metastatic patients, who need to have their testosterone suppressed for the remainder of their lives.

It would be intriguing to know what the physicians themselves think about having an orchiectomy. If they personally envision it as an irreversible body modification and find it unappealing for themselves (ala Freud), they may assume that their patients have the same view.

While this study showed that patients are not well informed about surgical castration, it doesn't answer an important question: Why don't all physicians inform patients with advanced prostate cancer about this ADT treatment option?

Source:
24 January 2022
http://www.lifeonadt.com/life-on-adt-blog/2022/1/24/why-arent-more-men-with-advanced-prostate-cancer-electing-to-have-an-orchiectomy-rather-than-repeated-

Learn to be your own researcher to make the best treatment decisions, by being proactive and an advocate for your own health

Could More Testosterone be the Key to Fighting PCa?

Source:
24 Jan 2022
Janet Farrar Worthington
https://www.pcf.org/c/could-more-testosterone-be-the-hidden-key-to-fighting-prostate-cancer-part1/?utm_source=NewsPulse&utm_

Androgen deprivation therapy (ADT) has been the bedrock of treatment for advanced prostate cancer for more than half a century. But investigators at Johns Hopkins are rethinking it – in a way that sounds counterintuitive – and driving new approaches to tackle treatment resistance. They're discovering that shaking up prostate cancer with high-dose testosterone makes it more vulnerable to other treatments.

Researchers Drive PCa "BATty" – Part One: The Concept of BAT

ADT slows prostate cancer's progress by shutting off testosterone. Eventually, however, cancer adapts to this new environment and PSA levels start to rise; this stage is called castrate-resistant prostate cancer (CRPC). ADT is not a curative treatment, and long-term ADT causes significant side effects, including fatigue, hot flashes, weight gain, and loss of sexual function.

Several years ago, medical oncologist Samuel Denmeade, M.D., Co-Director of the Johns Hopkins Prostate Cancer Program, and colleagues came up with a remarkable concept for attacking prostate cancer: alternating ADT with high-dose testosterone. "It had been known for a long time that something weird happened when you gave testosterone to prostate cancer cells," says Denmeade. "Yes, with low doses you could get the cancer cells to grow – but plenty of reports said that paradoxically, at high doses the cancer cells don't grow as well, or they die. Even Charles Huggins, who won the Nobel Prize for discovering hormonal therapy, said in his Nobel acceptance speech that another way to kill cancer would be to give too much hormone. I was always interested in that idea."

About 10 years ago, Denmeade conducted a small study to test the concept of using testosterone *against* prostate cancer. "At the time, it seemed like all the data and literature suggested that the dose was really important; it had to be a high dose." The hypothesis: Prostate cancer cells adjust to a very *low*-testosterone environment (created by ADT) by making *very high levels of the androgen receptor (AR)*. And here, as he says, "too much of a bad thing can be a good thing." These high levels of the AR now make cancer vulnerable to very *high* levels of testosterone. Cancer cells that survive this respond to high-dose testosterone by turning the AR back down – and making the cancer once again susceptible to very low testosterone.

"The idea is to screw up the cancer cell's ability to adapt." Denmeade and colleagues coined the term, Bipolar Androgen Therapy (BAT), "to capture these polar extremes of very high and very low. Not just making the testosterone high, but cycling between high and low." It's this cycling that seems to be the key to keeping the cancer off-balance, slowing its ability to flourish. In BAT, men experience high testosterone levels that decrease over a 28-day period, then bounce back up with the next testosterone injection.

In that early study, of just a handful of patients, "we were very cautious, because we didn't want to make the disease worse. We built in all these safety parameters. But we were surprised: it didn't seem we made anybody worse. It seemed very safe. The patients did very well, and some of them stayed on the testosterone for a year or more. Most of them felt really good. A number of them did not want to come off of it when it seemed they were progressing: they were just so happy to have more energy, and some of them could have sex again."

Armed with this initial clinical data to show that BAT was safe and to show some response, Denmeade received funding for additional proof-of-concept studies from PCF, among other sources. Larger studies at Johns Hopkins have followed, including RESTORE, TRANSFORMER, and COMBAT. Other trials testing this concept have been completed or are under way at the University of Washington, University of Colorado, and in Australia, Brazil, and the Netherlands.

Prostate Cancer Foundation
Curing Together.

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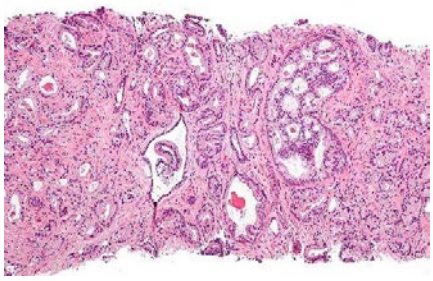
Treatments may vary in Australia. Please ensure you discuss your diagnosis and treatment options with your consulting specialist



Source:
4 January 2022
by H. Lee Moffitt Cancer Center & Research Institute

https://medicalxpress.com/news/2021-01-inflammation-adt-fatigue-prostate-cancer.html

Inflammation from ADT may cause fatigue in prostate cancer patients

Prostate cancer is one of the most common cancers among men in the U.S. For many patients, hormone therapy is a treatment option. This type of therapy, also called androgen deprivation therapy (ADT), reduces the level of testosterone and other androgens in the body. Lowering androgen levels can make prostate cancer cells grow more slowly or shrink tumors over time. However, patients receiving ADT often experience higher levels of fatigue, depression and cognitive impairment.

Moffitt Cancer Center researchers are investigating whether inflammation in the body, a side effect of ADT, contributes to these symptoms in prostate cancer patients. In a new study published in the journal Cancer, they pinpoint a specific inflammation marker that is associated with increased fatigue in this group of patients.

"This is the first study that we know of that examines the association between inflammation and symptoms of fatigue, depression or cognitive impairment in prostate cancer patients receiving ADT," said Heather Jim, Ph.D., corresponding author and co-leader of the Health Outcomes & Behavior Program at Moffitt. "Because the blocking of testosterone can increase inflammation in the body, we believe that inflammation may also be contributing to these symptoms."

For the study, the research team evaluated two groups of men: prostate cancer patients beginning ADT and a control group of healthy men the same age. The men were assessed at the start of the study and again at six and 12 months. Assessments included fatigue, depression and other neuropsychological tests and a blood draw. The bloodwork was to check for circulating markers of inflammation, specifically interleukin-1 receptor antagonist (IL-1RA), interleukin-6 (IL-6), soluble tumor necrosis factor receptor-2 (sTNF-R2) and C-reactive protein (CRP).

While the groups did not differ at baseline, researchers noticed a significant increase in fatigue and depressive symptoms in the ADT patients over the 12-month period. They also saw an increase in one inflammation marker, IL-6, in this group of patients.

"Interleukin-6 is a pro-inflammatory cytokine that is often associated with disruption of sleep and therefore fatigue," said Aasha Hoogland, Ph.D., lead study author and an applied research scientist in the Health Outcomes & Behavior Program at Moffitt. "Studies have shown testosterone can suppress the effects of IL-6, but ADT limits testosterone production in the body, which is why we may be seeing increased levels in this patient group."

The researchers say additional studies are needed to see if interventions, such as anti-inflammatory medications and exercise, can help alleviate fatigue and depressive symptoms in ADT patients.

Optimal duration of adjuvant ADT depends on the type of radiation used for high-risk patients

Source:
22 January 2022-02-06
Alan Edel

https://www.prostatecancer.news/2022/01/optimal-duration-of-adjuvant-adt.html#comment-form

No one *wants* to have androgen deprivation therapy (ADT), even if it is for a limited time. It has been known for a long time that it improves oncological outcomes when given with ("adjuvant to") radiation therapy in patients with high-risk prostate cancer. Several randomized clinical trials (RCTs) have tried to find the best duration to use it, but it is difficult to arrive at reliable optimization points- it would involve varying the duration for a large number of high risk patients. Kishan et al. have taken an innovative approach to solving this problem by combining several RCTs and a multi-institutional observational study. Unlike typical "meta-analyses," they compared similar patients across three studies.

The three studies they used in their analysis were:

1. The high-risk patients in the DART 01.05 GICOR RCT, which randomized patients to 28 months or 4 months of adjuvant ADT in patients getting high dose external beam radiation (EBRT-only). They found that 28 months is better than 4 months, but is there a duration that is less than 28 months for EBRT-only?

2. The patients in the TROG 03.04 RADAR RCT which randomized patients to 18 months or 6 months of adjuvant ADT in patients getting varying doses of EBRT or high dose rate brachy boost therapy (BBT). They found that 18 months is better than 6 months for BBT, but is there a duration that is less than 18 months for BBT?

3. The patients in a multi-institutional (retrospective, non-randomized) study who received varying durations of adjuvant ADT and EBRT-only or brachy boost therapy for their high risk PCa

They used distant metastasis-free survival (DMFS) as the endpoint of interest because it has been found to correlate well with eventual overall survival.

They went back to the original patient-level data to extract comparable patients when comparing them across studies.

This retained many of the advantages of each of the three studies.

While this innovative approach does not constitute the highest level of evidence (Level 1), it offers a degree of reliability that goes beyond simple observational studies.

They used two statistical methods to look at the data. In one analysis, they divided the durations into three parts:

≤6mo.

>6 - 18 mos

>18 mos

In another analysis (called "cubic splines") they found the best fit for the continuous data. Both analyses led to similar conclusions.

The best estimates for the best minimum adjuvant ADT duration are:

at least 26.3 months for EBRT-only

at least 12 months for BBT

But, one might object, didn't Nabid's PCS IV trial show that 18 months is as good as 36 months? Kishan points out that only about half of the cohort in that trial who were supposed to get 36 months of ADT actually got that much. And nearly a quarter of the 36-month cohort actually received *less* than 21 months.

The only data we've seen so far has been analyzed by the dose

they were intended to get, not by what they actually got. Also, why were the drop-out rates so high? The DART RCT had 95% compliance with the full 28 months, even though the radiation doses given were much higher.

There is a trade-off: BBT can come with severe late-term urinary side effects (among 19% in the ASCENDE-RT RCT), while the late-term urinary side effects are milder for EBRT-only (only 2.5% in DART).

Only the patient can decide if he is willing to take on 12 months of ADT with BBT vs over twice as long for EBRT-only, given the higher expected radiation toxicity with BBT.

There are several unanswered questions:

As we have seen, brief intense use of abiraterone or other advanced hormone therapy may obviate the need for longer ADT.

Decipher genomic analysis may indicate which patients may be able to get away with less hormone therapy, and which need more.

The PREDICT-RT RCT will eventually answer this question.

Does SBRT monotherapy or HDR brachy monotherapy still require adjuvant ADT? Those therapies can have almost as high a biologically effective dose as BBT but with fewer side effects. This study suggests that 12 months of ADT is beneficial with even the highest dose radiation, but future clinical trials will give a more reliable answer.

Standard-of-care dictates 2-3 years of adjuvant ADT when enlarged pelvic lymph nodes are found by CT or MRI. What is the optimum

duration when cancerous pelvic lymph nodes are only detected with a PSMA PET scan and not by CT? What about when they are t

oo small to be detected by any kind of imaging, and their presence is only *suggested* by risk characteristics?

What duration of adjuvant ADT minimizes biochemical recurrence-free survival and the need for any salvage treatment?

Will these estimates hold up if tested in an RCT?

PLEASE NOTE:
Treatments may vary in Australia. Please ensure you discuss your diagnosis and treatment options with your consulting specialist

How does ADT affect the brain?

Source:
24 December 2021

http://www.lifeonadt.com/life-on-adt-blog/2021/12/24/how-does-adt-affect-the-brain

Using some rather new neuro-imaging techniques, it is possible to map hormone-related changes in the functioning of various brain regions. This approach has now been used to understand how ADT affects the brain.

In a new study out of Spain, researchers used a type of neuro-imaging called *resting-state functional magnetic resonance imaging* (rs-fMRI) to investigate the potential impact of ADT on brain functioning. Rs-fMRI is used to visualize spontaneous brain activity when a person is 'at rest,' or not performing a particular task. In the current study, researchers looked at differences in rs-fMRI between men with prostate cancer, who were both on ADT (n = 49) and not on ADT (n = 15, controls). The men on ADT had been in treatment for at least 6 months, with an average of 3.5 years. Rs-fMRI was used to explore potential differences across the whole brain and in specific regions where there are more androgen receptors.

Compared to the controls, men on ADT displayed different patterns of resting-state neural activity particularly in certain frontal regions of the brain related to planning, initiating and coordinating movements. The results also suggest that ADT may alter brain function in the occipital cortex, which is involved in processing visual information. The findings appear to be consistent with previous research showing differences in spatial information processing related to ADT. Although not mentioned by the authors, it may also account in part for differences in attention to sexual images for men on ADT versus those not on ADT (Palmer-Hague et al. 2021).

When looking at connections and coordination between different brain regions (i.e., 'functional connectivity'), the researchers found that ADT may have differential effects depending on the region of interest. As the study authors describe, there may be changes in certain regions in terms of "how well individual brain regions activate in a concerted manner".

So, what does all of this mean for men on ADT? This study provides a small, preliminary glimpse into potential ADT-related alterations in the functioning of various brain regions. We don't yet know how these changes correlate with men's lived experience or with impacts on cognitive or psychological functioning. We need additional, longitudinal studies to better understand how the brain may adapt over time to ADT.

If you are worried about your brain's health, there are things one can do to protect cognitive function while on ADT. For example, physical activity can help preserve cognitive function as we age, whether or not we are on ADT.

To read the full article, see: https://www.nature.com/articles/s41598-021-02611-6

One man's mission to make prostate cancer fix open to all

Prominent business man's, Laurie Cox, positive experience with PSMA lutetium 177 treatment of his advanced, hormone resistant metastatic prostate cancer is covered in an article by Jill Margo, Health Editor of the Australian Financial Review (4 February 2022). (Article reproduced here: https://pcfa.org.au/media/791289/4-financial-review.pdf?utm_medium=email&utm_campaign=FEB%20%20Blue%20Sky%20Connect&utm_content=FEB%20%20Blue%20Sky%20Connect+CID_480f51f25becde8717fa014c44b4ef81&utm_source=Email%20marketing%20software&utm_term=Access%20the%20article)

Cox, whose financial position has allowed him to take full advantage of this experimental treatment, is keen for Australia to seize a unique opportunity and make this treatment accessible at an affordable price. The article explains that the treatment molecule comes in two variants, both developed in Germany: one patented by Novartis (from which the company expects to make a lot of money); and one generic , currently being trialled by Professor Michael Hoffman's team at the Peter MacCallum Cancer Centre. The article makes clear that the key to affordability and widespread access to the treatment is a reliable and permanent source of funding for the generic-based approach, which is only likely to be via federal government listing on the Medical Benefits Schedule.

Like all treatment decisions, you have to weigh how you feel about the potential benefits against the potential risks. No one can do that for you.

Pomegranate May Help Reduce the Risk of Certain Cancers – Study

Source:
6 November 2021
Joven Gray
https://cancersolutions.news/2021-06-11-pomegranate-reduces-risk-of-cancer.html

A review conducted to explore the anti-cancer properties of pomegranate has found sufficient evidence that supports its use for the prevention of certain cancers.

The review looked at cell culture and animal studies, as well as human clinical trials on the use of pomegranate against certain types of cancers. After compiling and analyzing all the data, the team confirmed that pomegranate could help reduce the risk of breast, prostate, lung, colon and skin cancers, as well as hepatocellular carcinoma.

According to the review, pomegranates are good sources of antioxidants, phenolic acids and flavonoids. Just the peel of the fruit is rich in ellagitannins, ellagic acid, gallic acid, hydroxybenzoic acids and punicalagin.

The researchers said these compounds contribute to the plant's anti-inflammatory, anti-proliferative, antioxidant and anti-tumorigenic properties. The team added that in the studies they've analyzed, pomegranates were able to modulate multiple signaling pathways used by cancer cells.

The team also discovered that the polyphenols present in pomegranates exhibited anti-carcinogenic effects in in vitro and in vivo experiments. Moreover, in animal studies, pomegranate extract showed the ability to inhibit the growth of lung, skin, colon and prostate cancer tumors. Meanwhile, in studies that involved human patients, punicalagin was discovered to have chemopreventive and chemotherapeutic properties against cancer cells.

"The therapeutic potential of pomegranate appears to be wide variety. So, leading to enhanced popularity as a natural compound and functional food for centuries. The pomegranate polyphenol; punicalagin, is known to have potent anticancer activity in breast, lung, and cervical cells," the researchers wrote.

Different parts of pomegranate are beneficial

In addition, in the studies they've reviewed, some parts of the pomegranate plant (i.e., peel, juice and oil) have shown the ability to inhibit tumor cell proliferation, cell cycle progression and angiogenesis — the formation of new blood vessels — in different types of cancers.

Pomegranate peels, as mentioned, are rich in flavonoids and antioxidants, which were found to help delay the proliferation of different cancer cells. Aside from suppressing cancer growth, the peel can also be used to get rid of acne, pimples, rashes and wrinkles, as it is a natural skin moisturizer. Moreover, it can be used as a sunscreen and as a natural remedy for dandruff and hair loss. The peel may also help relieve sore throat, improve dental hygiene, boost bone health and improve gut health.

Meanwhile, pomegranate juice is rich in antioxidants, vitamin C, vitamin K, folate and potassium. It also has antiviral and anti-inflammatory properties. The juice may help improve digestion, relieve inflammation caused by arthritis, promote heart health, lower blood pressure, improve learning and memory, enhance sexual performance and fertility, support physical endurance and lower blood sugar levels. According to the researchers, the juice also contains ellagic acid, which may be the reason behind the anti-carcinogenic effects of the juice.

On the other hand, pomegranate oil is rich in vitamins C and K, as well as the antioxidant punicic acid, giving it the ability to reverse the aging, fight inflammation, fade scars, brighten the skin and protect against damage caused by too much exposure to sunlight. (Related: The potential therapeutic uses of pomegranate seed oil.)

"In view of the different activities of pomegranate extracts, we believe that they are suitable further investigations as potential multiple target-oriented therapy and prevention and suppression for the wide variety of cancer and its pathological outcomes. It is hoped that the present review will provide some worthwhile clues for continues explorations of this most attractive botanical species," wrote the researchers.

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

Source:
10 July 2020
Allen Edel
https://www.prostatecancer.news/2020/07/the-perils-and-pitfalls-of-treating-psa.html

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

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. 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" (see this link) 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. One may be lulled into complacency by deceptively low PSA.

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.

II. Supplements that interfere with PSA tests

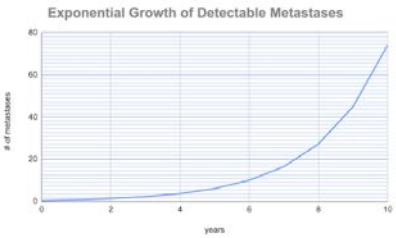
Patients often self-medicate in the hope of wresting some control over their cancer. The internet is full of "evidence" that this or that natural supplement may slow progression or even cure the cancer. Serum PSA is detected by an antibody that can detect amounts as low as a nanogram of PSA per ml of 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. 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.

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. 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. 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.

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 - millions 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". 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 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.

2. PSA-based Endpoints

What we really want to know is this: will the treatment enable patients to live longer? Overall survival is the gold standard of success of randomized clinical trials. The "problem" for clinical trials is that prostate cancer is such a slow killer, that it may take 15 years or more to discern a difference if patients have localized or recurrent prostate cancer at the start. (For most other types of cancer, 5-year overall survival is more than adequate.) Clinical trials are often ended when half of the control group die (median survival). But, depending on patient characteristics at the start, median survival may never be reached within the duration of the clinical trial.

Prostate cancer-specific survival (how long before patients succumbed to their prostate cancer) is little better. It is also hampered by the fact that patients with prostate cancer may die of something else sooner, possibly because their cancer was debilitating. It is often unclear to the doctor who signs the death certificate whether the cancer was the end cause, a contributing cause, or a non-contributing factor. To get clinical trial results before new medical science and technology renders the results irrelevant, we want to use surrogate endpoints that are highly correlated with and predict overall survival.

The earliest endpoints that can be used to measure the success of a prostate cancer therapy are PSA based. All of the following surrogate/secondary endpoints are PSA based:

- PSA50 - the percent who had a reduction in PSA by 50% or more
- Nadir PSA - the lowest PSA reached after therapy
- PSA doubling time (PSADT) - whether the therapy slowed PSA growth
- Biochemical recurrence (BCR) - depending on initial treatment, and there may be multiple salvage therapies, each with a PSA failure defined for it
- Biochemical Recurrence-Free Survival (bRFS)
- Biochemical failure (BF)- rise in PSA by a pre-specified amount post-therapy
- Biochemical No Evidence of Disease (bNED)
- Time to BCR/ BF
- Time to start of lifelong ADT (based primarily on a pre-defined PSA failure benchmark)
- Failure-free survival (FFS) or Progression-free survival (PFS) or Event-free survival (EFS) - defined as BF or radiological progression or clinical progression or death.

The following surrogate endpoints are *not* PSA-based:

- Clinical Progression-Free Survival (cPFS) - worsening of symptoms or performance status (see this link)
- Radiographic Progression-free Survival (rPFS) or Disease-free survival (DFS)- progression on scans or death
- Objective Response Rate (ORR) - tumor size or number reduction using RECIST criteria
- Change in Bone Scan Index
- Time to radiographic progression or failure
- Metastasis-free survival
- Clinical progression - pain, bone fracture, spinal compression

As an example of circular reasoning, we can see in the ORIOLE trial that 6-month Progression Free Survival (PFS) was chosen as the primary endpoint. PFS was defined as PSA progression (by >25% over nadir and by > 2 ng/ml) *or* radiographic progression *or* death. As we can readily see in the exponential growth curve, the odds of a new metastasis on a bone scan/CT are very low and there are not likely to be any deaths. Therefore, PFS was almost entirely PSA progression. But the protocol "treated PSA." It is therefore illogical to conclude, even for a Phase II trial, that oligometastatic treatment slowed progression.

It is worth noting that radiation of the prostate ("debulking") has no survival or progression advantage when there are multiple metastases, only when the metastatic burden is low. The prostate is, of course, the source of all metastases, and an ideal environment for metastases to develop and grow. Metastasis-to-prostate spread has been observed. In a meta-analysis of the two debulking trials called STOPCAP M1, researchers found that there was a statistically significant reduction in PSA progression (by 26%), even when there was no benefit in terms of metastatic progression or survival. Treating PSA even by debulking the entire prostate is not in and of itself of any oncological benefit (there may be a palliative benefit, however).

3. Danger of Withholding Early ADT

While ORIOLE, STOMP, and SABR-COMET were Phase 2 clinical trials whose results were not meant to change practice, many patients and their doctors (often under pressure from patients) would like to believe they do. If the metastases are in places that are safe to irradiate (e.g., away from the mediastinum), there is little risk in doing so. However, if they do not understand the circular reasoning evident in the ORIOLE trial, they may put off therapies that are known to increase survival. There is also a risk of unreasonable expectations.

Some patients (and doctors) believe that by delaying ADT, they can increase their quality of life, and delay castration resistance. Neither is true. Contrary to popular belief, *decreasing* the intensity of hormone therapy and *delaying* its use brings *earlier* castration resistance and death. The strongest evidence for this comes from the STAMPEDE (on Zytiga and Xtandi), LATITUDE, and SPARTAN trials. Among men who were *newly diagnosed* with metastatic prostate cancer:

- Overall survival was longer if men used Zytiga + ADT.

Like all treatment decisions, you have to weigh how you feel about the potential benefits against the potential risks. No one can do that for you.

- No difference based on the number of metastases
- Failure-free survival was longer if they used Zytiga + ADT

- Overall survival was longer if men used Xtandi+ADT
  - Survival was especially lengthened if there were fewer metastases
  - PSA progression-free survival was longer if they used Xtandi+ADT
- Overall survival was longer if men used Erleada+ADT
  - PSA progression-free survival was longer if they used Erleada+ADT

A clear pattern emerges: early use of intensive hormone therapy prolongs survival and prolongs the time to castration resistance. Men who were oligometastatic benefited from early, intense hormone therapy.

The TROG 03.04 RADAR trial examined the duration of hormone therapy in high-risk men treated with radiation. They found that, after 10 years of follow-up, men treated with 18 months of ADT survived *longer*, and reached castration resistance *later* compared to men treated with 6 months of ADT.

The TOAD trial looked at starting ADT at the first sign of recurrence vs. waiting for metastases to be detected. Men treated *earlier* reached castration resistance *later*. It also showed there was no major detriment to global health-related quality of life by starting ADT earlier.

Maha Hussain reported the results of a randomized clinical trial comparing intermittent vs continuous ADT in recurrent men with metastases. She found that:

- Time to castration resistance was not different for the two protocols
- For men with minimal disease, overall survival was 6.9 years for those on continuous therapy vs 5.4 years for those on intermittent therapy. The trial was underpowered for this difference to reach statistical significance.
- It took 4-5 years for the survival curves to start separating - long follow-up is needed to detect survival differences.

Taken together, all these major randomized clinical trials show that the best way to use ADT in the oligometastatic setting is to use it early and heavily. Reducing the number of cancer cells as quickly and effectively as possible, even reducing those cells that haven't begun to measurably contribute to PSA, extends survival. The effect of evolutionary selection pressure allowing castration-resistant cells to survive is dwarfed by the reduction in sheer numbers. Circular reasoning may harm patients.

4. Future Clinical trials

We have learned some lessons about clinical trials for oligometastatic treatment:

- It has to have long enough follow-up, depending on the setting: at least 5 years for newly diagnosed or recurrent men to allow time to get to the steep part of the exponential curve. It will take longer if more sensitive imaging is used.
- It must use radiographic progression-free survival, or similar, as its primary endpoint
- It must *not* use a PSA-related endpoint
- ADT must be used in at least the control group. It would be unethical to withhold the standard of care
- It should preferably use a PSMA PET/CT to locate metastases. The ORIOLE trial only found an advantage if patients were oligometastatic on both a PSMA PET/CT and a bone scan/CT. The use of more sensitive imaging will move the starting point to the left on the exponential curve, so it will take that much longer to detect a benefit.

These randomized clinical trials (RCTs) are currently active:

- The CORE RCT (active, no longer recruiting) at Royal Marsden Hospital in London will have 5 years of follow-up (completion in 2024) and will include freedom from widespread metastatic disease and overall survival among the outcomes looked at.
- The PCX IX RCT (among castration-resistant patients) at Jewish General Hospital in Montreal will have 5 years of follow-up (primary outcome in 2025) and has radiographic progression-free survival as its primary outcome.
- The PLATON RCT (among hormone-sensitive patients) in Canada will have 6 years of follow-up (primary outcome in 2025) and has radiographic progression-free survival as its secondary outcome. Oligometastatic men who have never had their prostates treated with RT will have prostate radiation too in both arms. ADT is given in both arms, advanced hormonals and chemo at the physician's discretion.
- The STEREO-OS RCT (recruiting, study completion in 2022) in France will look at radiographic progression-free survival with follow-up of up to 3 years.
- The FORCE RCT at the University of Michigan (primary completion in 2022) will compare systemic treatment with ADT and any of Taxotere, Zytiga or Xtandi (at the discretion of the treating physician) to similar systemic treatment plus metastasis-directed SBRT for men with mCRPC who have not yet had any of those advanced systemic therapies. They will evaluate progression-free survival after 18 months. "Progression" is defined as alive and at least a 20% increase (and at least 5 mm net increase) in the size of tumors or any new metastases. They will detect metastases via bone scan/CT, However, they will also test whether PSMA-based PET indicators are as useful among men with mCRPC as it is in men with newly recurrent disease.
- The VA STARPORT RCT (primary completion in 2025) in many VA hospitals in the US will randomize patients to systemic therapy + PET-directed radiation to 1-5 oligorecurrences or to systemic therapy alone. Unfortunately, they are using castration-resistance as their primary endpoint, which is problematic.

The START-MET RCT (primary completion in 2025) in Spain will randomize recurrent and newly diagnosed oligometastatic (≤3 on bone scan/CT and ≤5 on PSMA PET) men to standard-of-care (ADT+2nd line HT+prostate RT) or standard-of-care + SBRT to all metastases. 2-year radiographic progression is the primary outcome.

PLEASE NOTE:
Treatments may vary in Australia. Please ensure you discuss your diagnosis and treatment options with your consulting specialist

Physical exercise can improve the quality of life of just about any patient on ADT, right?

Source:

01 February 2022
http://www.lifeonadt.com/life-on-adt-blog/2022/2/1/physical-exercise-can-improve-the-quality-of-life-of-just-about-any-patient-on-adt-right

Well, not exactly. Let's start with some basic facts. Prostate cancer patients in general are not getting enough physical exercise and this is particularly true of men on ADT.

There is now a report out of Toronto that looks at the relationship between quality-of-life (QoL), physical activity, and the amount of time men on ADT spend being sedentary. The study specifically focused on men who were not getting the recommended 150 minutes per week of moderate to intense exercise. One hundred and six men, with a mean age of 72.2 years, were included in the study.

What is particularly interesting about this study is that the researchers divided the men into groups based on whether they reported a high or low quality of life to begin with. In simple terms, for some men, QoL improved more if they were less sedentary whereas for others being more active (less sedentary) didn't necessarily improve their QoL.

One can understand these results in common sense terms, reflecting on the aches and pains that come with aging. Men who were sufficiently uncomfortable with activities of daily living (to an extent that could not be accounted for statistically) were excluded from the study. But that didn't mean that all men in the study were vigorous and pain free. One can imagine that during the course of the study there were men who felt better by getting up and moving around, whereas for others just getting out of the chair might have been enough of a challenge to reduce their QoL.

This study is great for looking at a population of men on ADT who are often older, largely inactive, and spend a lot of time sitting. It suggests, however, that health physiologists, who work with this population, need to consider variables other than simply the absolute time spent sitting or moving around. The researchers point out that there are now wearable activity meters that record not just whether a person is sedentary or moving around, but how many times a person gets up from sitting. Being realistic, we cannot expect all patients on ADT to get to the gym for 150 minutes or more a week. So, we need some quality recommendations on whether those men should be spending more time doing activities of daily living and whether they should be doing them in longer or shorter bouts. If we are going to help this patient population, we need to give them good, evidence-based advice on simple things like how often they should get up from sitting.

In a similar vein, exercise physiologists need to consider other factors that influence QoL for men on ADT, recognizing that the ADT agents often lead to depression (which, in and of itself, can make it difficult to get up off the couch).

We look forward to more research from this group and the recommendations they come up with to help more patients on ADT become both more active and have a better QoL overall.

To read the study abstract, see:
https://link.springer.com/article/10.1007/s10865-022-00285-7

Gather My Crew was founded in 2017 by Dr Susan Palmer, a psychologist with many years' experience supporting families going through crisis. But it was not until her friend needed help that the idea for Gather My Crew was first born.

Her friend Rachel required back surgery and her recovery would involve six weeks of bed rest. Susan wanted to help keep life as normal as possible for Rachel and her family – and had a group of 30 wonderful people able to lend a hand. But it wasn't long before the text messages, ring-arounds, spreadsheets and late-night phone calls became too time consuming and difficult to manage.

It was a 'lightbulb' moment and the beginning of Gather My Crew...

After talking with colleagues looking for an easy, 'online help roster' that would make coordinating all of this help a breeze, Susan was shocked to discover that nothing like it existed.

Twelve months later, Gather My Crew was launched.

Created based on the expertise of people who had 'been there, done that', as well as the clinicians who supported them, Gather My Crew exists to make sure people going through a crisis get the right help, at the right time from their friends and family – without all of the stress that usually comes with coordinating help via traditional means.

Source:
https://www.gathermycrew.org.au

New mobile apps

We have launched a new app with increased functionality to help your patients or clients get the right help at the right time.

https://www.gathermycrew.org.au/getting-started/

New support service

We have a new website and referral partner resource pack for you to use as part of your service. Contact us to receive yours - it's all FREE!

https://www.gathermycrew.org.au/support-us/

Does One Recover Testosterone Faster when One Stops ADT from an LHRH Antagonist Versus an LHRH Agonist?

Source:
20 January 2022
http://www.lifeonadt.com/life-on-adt-blog/2022/1/20/does-one-recover-testosterone-faster-when-one-stops-adt-from-an-lhrh-antagonist-versus-an-lhrh-agonist

Both LHRH agonists (like Lupron, Eligard, and Zoladex) and LHRH antagonists (like Degarelix) suppress testosterone (T) equally well. But the antagonist, Degarelix, drives T down faster and does not cause an initial surge in T before T descends into the castrate range. So, what happens when one stops taking those drugs? Does T climb back into the normal range faster after ADT with an antagonist than an agonist?

Consider the situation where one is on ADT short term to improve the effectiveness of radiotherapy. Patients in that situation often hope to get their T levels back quickly when coming off ADT. A group of physicians in Okayama, Japan, explored the topic of T recovery with 112 patients going for brachytherapy to treat prostate cancer. Approximately 70% of the men were on an agonist and the rest were on an antagonist for short-term ADT.

Surprisingly, the T level climbed significantly faster after ceasing LHRH agonist ADT than LHRH antagonist ADT in the first three months post ADT. However, at the one-year mark there was no difference in the recovery between the patients on either class of drugs. In both treatment arms, there was 72 to 75% recovery of T after one year (which is comparable to what has been reported in previous studies).

It's not clear that these results can be generalized to other populations. For instance, in North America patients going on an LHRH agonist usually start on an anti-androgen, like bicalutamide (Casodex), before starting on an LHRH agonist. However, only a few patients in this Japanese study received Casodex. Thus, we don't know if early administration of the anti-androgen somehow influenced the rate of T recovery. What the data do suggest is that there's not a symmetry between how fast one can drive T down and how quickly it can recover.

To read the study abstract, see:
https://pubmed.ncbi.nlm.nih.gov/34955538/#article-details

PCa Clinical Trials & Studies

For Further information on current and recruiting trials visit:

https://www.anzup.org.au/content.aspx?page=prostatecancertrialdetails

Psma Intensity Can be Altered by Androgen and Phospho-SrC Obstruction

Trial ID NCT04925648
Phase 2
Anticipated Start Date 18/10/21

Location
Kinghorn Cancer Centre, St Vincent's Sydney
Peter Mac Melbourne

The study's purpose is to understand the appearance of your prostate-specific membrane
antigen (PSMA) PET scan after you take 14 days of treatment with a drug called dasatinib
alone or in combination with anti-testosterone drug call darolutamide.

Who is it for? You may be eligible to join this study if you have metastatic prostate cancer
and had a recent PSMA scan showing low PSMA uptake

Study Details:

Participants will receive dasatinib 100 mg daily or dasatinib 100 mg daily and darolutamide
600 mg twice daily for 14 days. They will undergo another PSMA PET scan after 14 days.
Participants will be followed up on day 7 of treatment and 30 days after treatment.

It is hoped that this research will provide insight into the mechanism of PSMA expression in
advanced prostate cancer.

Like all treatment decisions, you have to weigh how you feel about the potential benefits against the potential risks. No one

UPFRONTPSMA

A Randomised Phase 2 Study of Sequential 177Lu-PSMA617 and Docetaxel Versus Docetaxel in Metastatic Hormone-Naive Prostate Cancer

Protocol No 18/047

Patients with a diagnosis of de novo high-volume mHNPC who meet all the inclusion and exclusion criteria will be eligible for participation in this study.

Associate Professor Arun Azad (Principal Investigator) & Professor Michael Hofman (Nuclear Medicine Investigator)

140 participants

Further Information: ClinicalTrials.gov:

Austin Hospital
Peter MacCallum Cancer Centre
Alfred Health – The Alfred Hospital

Prostate Heidelberg Cancer Support Group Meetings

PHCSG organizes specialists and consultants to speak to members on a regular basis.

Details to follow

Did you Know?

FDG (fluoro-deoxy-glucose) is a different radioindicator for a PET scan that is used for almost all kinds of cancers. It is sometimes called a 'glucose' PET scan – it shows cancer that metabolizes glucose. In later stages, prostate cancer metabolizes glucose and expresses less PSMA.

Internet Resources

Members have found the following websites useful

Prostate Cancer Foundation of Australia for guides & help
https://www.pcfa.org.au
https://onlinecommunity.pcfa.org.au/

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

USA Prostate Cancer Foundation (Guide) PDF guide for men newly diagnosed with PC
https://www.pcf.org/guide/

Us TOO International PCa Education (USA) USA PC support groups' information & newsletter
https://www.ustoo.org

Cancer Council Victoria for general support services
https://www.cancervic.org.au

ExMed Cancer Program Melbourne based 'best practice' exercise medicine program
https://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

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

PAACT Newsletter (USA) Patient Advocates for Advanced Cancer Treatments
http://paact.help/newsletter-signup/

A Touchy Subject
https://www.youtube.com/channel/UCdyuxGuAuCWJbe-kZvwVSzQ

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.

PHCSG Correspondence

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

PHCSG Correspondence

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

PHCSG Meetings 2022 10am – 12:30pm

Tues 15 Feb
Tues 15 March
Tues 19 April
Tues 16 May
Tues 21 June
Tues 19 July
Tues 16 August
Tues 20 September
Tues 18 October
Tues 15 November
Tues 13 December (the second Tues to avoid the week prior to Xmas. Includes Xmas lunch – subject to COVID restrictions)

2022 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

January 2022

- Links between Gut Microbiome & Aggressive PCa
- Radpid PCa Screening Kits
- How Much Should You Eat?
- Abiraterone/DT Combo Associated with High Metastasis-Free Survival Rate
- Terbiom-161 Clinical Study Collaboration
- Electrical Pudendal Nerve Stimulation vs Pelvic Floor Muscle Training
- Identifying PSA Patterns in mHSPC Treated with Abiraterone & Prednisone
- Viagara Linked to Lower Risk of Alzheimer's
- Ductal Adencarcinoma
- BAT vs Enzalutamide in MCRPC
- Systemic Therapy Patterns in MCRPC
- Exercise May Stop Disease in its Tracks
- AI Accurately diagnoses PCa
- New Insights into Molecular Drivers of Treatment Resistance in PCa
- Decreased Fracture Rate by Manadating Bone Protecting Agents

February 2022

- Why Aren't More Men Electing to Have an Orchiectomy?
- Could More Testosterone be the Key to Fighting PCa? Part one
- Inflammation from ADT may Cause Fatigue
- Optimal Duration of ADT Depends on the Type of Radiation
- How does ADT Affect the Brain?
- Pomegranate may Help Reduce Certain Cancers – Study
- The Perils & Pitfalls of PSA in Advanced PCa
- One Man's Mission to Make PCa Fix Open for All
- Physical exercise can Improve Quality of Life
- Gather My Crew
- Does One Recover Testosterone Faster when Stopping LHRH Antagonist or Agonist?
- Clinical Trials & Studies

2021 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

January 2021
- Exercise Infographic
- Sexual Dysfunction & Shared Decision Making
- FDA Approves first Oral Hormone Therapy
- Prolonged ADT Reduces Cardio Fitness
- Reducing the Burden of Out-of-Pocket Expenses
- BAT Sensitizes CRPCa to Subsequent Therapy
- Targeting Bone Mets with Radiation in Oligorecurrent Men

Prostate Cancer Trials
- PEACE V:STORM
- UpFront PSMA Phase II
- NINJA

February 2021
- Advantages of Coffee
- Our Biological Clock
- Statins tied to Better Outcomes
- What's New in Inflammation
- New PC Management Techniques
- About the Patch Trial
- Eating a Colourful Diet
- Dose Painting
- Advancement in Focal Therapy

Prostate Cancer Trials
- Enza-P
- DASL-HiCaP Trial
- Lu-177-PSMA-617
- Adding Apalutamide to Radiotherapy & LHRH Agonist

March 2021
- Challenging Your Private Health Provider
- How Research is Prioritised – Norman Swan podcast
- Metastatic PCa – Don't Accept Complacency
- An mRNA Vaccine for Cancer
- Life After Treatment – Wellness Program
- Focal Therapy – If It Sounds Too Good to be True
- Immune Checkpoints on CTCs

April 2021
- Study finds cancer cells evade chemo by going dormant
- High Risk Localised PCa: Changing the rules
- Automated Pathological Assessment of PCa Biopsy Slides
- Final Results from TITAN Study
- SBRT for High Risk Patients
- Benefit of taking 1year of ADT after radiation for high risk PCa
- Novel Radiopharmaceutical beats Cabazitaxel in MCRPC
- Novatis announces phase III positive results
- Estrogen – Our Sister Hormone

Prostate Cancer Trials
- Enzalutamide With Lu PSMA-617 Versus Enzalutamide Alone
- Darolutamide Augments Standard Therapy for Localised Very High-Risk Cancer

May 2021
- Full on Kitchen Sink for High Risk Localized PCa
- Calcium & Vitamin D Supplements
- Favourable prognosis with adjuvant ADT after RT
- Healthy Lifestyle may offset Genetic Risk
- Additional Treatment Option
- New Type of Treatment could reawaken Immune Response
- Penile Rehabilitation
- Prostate Cancer Trial Results

June 2021
- Dry July
- Breakthrough in Disease resistance to drugs
- PyL PSMA Pet Imaging
- Does thel level of your Testosterone matter when on ADT?
- Stay Bone-Healthy
- ADT and the risk of Cariovascular Disease
- The Pros & Cons of Orchiectomy
- Risk of Serial Biopsies
- Reflections on 10 years on AS
- Improvements on Oligo-recurrent Therapies
- Time Pressure Decisions
- Research making Chemo Friendlier
- Trial Results on Exercise

July 2021
- Ground Breaking Early Cancer Detection
- What Should You Eat
- ADT What You Really Need to Know
- Anti Androgen Therapy
- Overall Survival with Metachronous MHSPC
- New Guidelines for Salvage Radiation
- Help for ED after RP
- Germline Testing

Prostate Cancer Trials
- Enz-P; DASL HiCaP; NINJA; Upfront PSMA
- 45 & Up Study Results

August 2021
- Targeting PSMA
- What is the Role of Modern Imaging
- Observation Vs SBRT for Oligometastatic PC
- Combined High-dose Salvage RT & HT in Oligorecurrent Pelvic Nodes
- Long Term Urinary & Erectile Function following RP
- Bone Resportion Inhibitors
- RT After RP
- Take Responsibility

Prostate Cancer Trials
- UpFront PSMA & MOSES Study

September 2021
- Targeting PSMA
- PEEK Study
- Skeletal Events & Bone Modifying Agents in Castration Resistant PC
- Abiraterone +docetaxel+ADT for Newly Diagnoses Metastatic PC
- Brief, Intense Radiation & Hormone Therapy for Very High Risk PCa
- Progression-directed Therapy for Oligoprogression
- Insights into PC metabolism
- Diagnostic Accuracy of PSMA 18F-DCFPyL PET/CT
- Risk of PC in relatives of PC
- Relugolix – Expected to Alter Treatment
- Whole-pelvic radiation Therapy for High-Risk Patients
- It's time to Retire a Common Biopsy
- Cognitive Function / Marital Status & PC Incidence
- Covid Passports
- Medical Bills: Out of Pocket Costs
- Prostate Cancer Trials
- UpFront PSMA & ENZAp

October 2021
- Continuous vs Intermittant ADT
- Predict Risk Tool
- Doubling Time Tool
- High Discontinuation Rate in AS
- AI Program Helps Detect PCa
- Plant Based Diet
- Obesity Ups MCRPCa Survival
- Impact of Hypofractionated RT on Patient Outcomes
- Controversy Around Testosterone Therapy
- Medications for ADT Hot Flashes
- Best Way to recover Urinary Continence after PR
- Diabetic Risk & ADT
- Abiraterone for NMPC
- When to Use Chemo

November 2021
- New PCa drug helping men live lomger
- What predicts who goes on continuous vs intermittent ADT
- Gut Bugs can drive PCA growth & resistance
- Exception to early salvage radiation
- PCa Urine Test
- New Strategy against Treatment resistant PCa
- Blood Test may help treat PCa
- Prostate Cancer Studies
- Caregiver Health Literacy/Supportive Care Program/access to Nutrition Info
- Optimal Dietary & Exercise

December 2021
- PCa Thwarted by Gut Microbiota
- Exercise is Medicine
- Giving Cancer a "Brown-Out"
- Wake Up! It's Time to Address Sleep Issues
- The Complex Natural Biochemistry of a Healthy Diet
- ADT: What You Really Need to Know
- Andropause and the Treatment Nobody Talks About
- Unlocking the Secrets of Sleeping Cancer Cells
- Treatment-Related Regret
- New PCa Treatment Coul d Improve Outcomes for Advanced Patients
- PCa Trials – Recruiting
- Promising Treatments & New Methods