MEMBERSHIP

FULL CALENDAR YEAR PHCSG MEMBERSHIP $20

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

EFT Payments to:
Prostate Heidelberg CSG
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Prostate Heidelberg

December 2021 Issue 213

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 14 December
10am – 12:30pm

To join via Zoom: Copy link and paste into your browser
https://us02web.zoom.us/j/83796990584?pwd=c3hVZ0JyVEVGcXVHYXZKRTFnQ1ZPUT09

PHCSG November – Face-to-face is Back!

We had an excellent turn out for our November meeting – one half ventured back, fully masked, to our venue in Ivanhoe, while everyone else joined via Zoom.

A reminder that our final meeting for the year is on the second Tuesday of the month - to simplify the run-up to Christmas for everyone.

Our speaker this month is Dr. Dixon Woon, Urological Surgeon, Uro-Oncologist "Prostate Cancer Treatment Progress over the last 10 years". See more about Dr Woon on page 12.

In the last few weeks we have had several requests asking for members to take part in surveys for health studies. We urge you to participate and share your views as all data collected will help improve services to members and the wider Prostate Cancer community.

To wind up 2021, we have included all local clinical PCa trials still recruiting. In this case you should be consulting your doctors to see if any of them are suitable.

Finally, we wish you a relaxing time over the holiday period and a safe and healthy 2022.

In this month's newsletter we highlight:

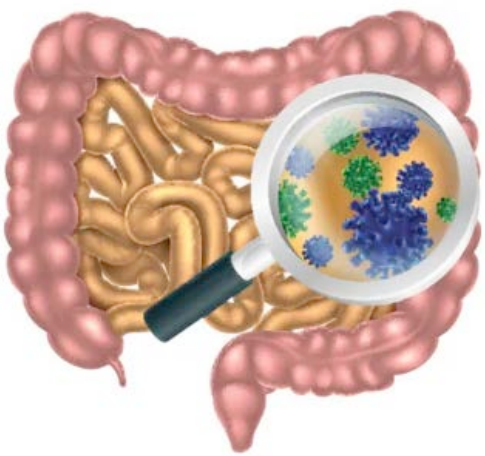
If there is anything you want to talk through in relation to your treatment or wellbeing please don't hesitate to ring:

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

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



A Widely Used Treatment for Advanced Prostate Cancer can be Thwarted by Gut Microbiota.

Source:
12 Oct 2021
https://www.inverse.com/mind-body/gut-health-prostate-cancer-

IN 1981, Barry Marshall had a hunch.

The Australian gastroenterologist believed a spiral-shaped bacteria called *H. pylori* was responsible for stomach lining inflammation, one of the first signs of stomach cancer. When his initial research on mice garnered a mixed response from his peers, he tried his hypothesis on himself.

Marshall and his mentor Robin Warren eventually won the Nobel Prize for discovering a link between gut bacteria and disease, a topic that's now explored in every facet of medicine from inflammatory bowel disease to depression.

New research is pushing Marshall's ideas forward, suggesting that a better understanding of gut bacteria could make prostate cancer therapies more effective.

In a study published [in October) in the journal *Science*, researchers found one of the most widely used treatments for advanced prostate cancer — androgen deprivation therapy (ADT) — can be thwarted by gut microbiota. In turn, methods designed to target and hinder certain gut bacteria could help this type of cancer therapy become more successful.

WHAT YOU NEED TO KNOW FIRST — Prostate development is influenced by hormones — mostly growth and reproductive hormones called androgens — mostly produced by the testicles. Testosterone, for example, is an androgen.

Ruminococcus gnavus was found in patients who were less receptive to androgen deprivation therapy, Michael Henke/Harvard Medical School

"When the cells in the prostate age and become abnormal or cancerous, their growth is still controlled by the androgens that were responsible for the formation of the prostate in the first place. "Dr. Giorgio Trinchieri, chief of the National Cancer Institute's Laboratory of Integrative Cancer Immunology, tells *Inverse*. Trinchieri is the co-author of a related perspective published alongside the new study.

Scientists have understood for decades that this control allows androgens to speed cancer growth. For this reason, ADT is one of the most widely used treatments for advanced prostate cancer.

Blocking androgen production usually slows cancer growth for a long time. However, cancer cells can eventually evolve to be able to grow without androgens and stop responding to ADT.

Ruminococcus gnavus is also found in higher proportions among people with Crohn's disease.

"This cancer treatment works better when the androgens are blocked as completely as possible, but in some cases, some androgens are still present in the prostate and the patients' disease worsens faster," Trinchieri explains.

In the new *Science* study, the research team finds this ADT resistance may be coming from an unsuspecting place — gut bacteria.

WHAT'S NEW — The researchers discovered certain gut bacteria — *Ruminococcus gnavus* and *Bacteroides acidifaciens* — in higher proportions among people who resist ADT and have an advanced stage of prostate cancer, called castration-resistant prostate cancer. These gut bacteria produce the very hormones ADT works to suppress.

By comparing the gut microbiomes of both mouse and human models, they were able to take the first step in creating a microbial blueprint of bacteria that influence prostate cancer outcomes.

Pinpointing how specific gut bacteria contribute to ADT resistance could help prevent the cancer treatment from failing. The team has already tested two courses of action in mice models.

For example, they found that using antibiotics to kill gut bacteria in mice delayed ADT resistance, though at the expense of all gut bacteria, good or bad. In some cases, fecal transplants from patients and mice who were susceptible to ADT — meaning the therapy was still working for them — were able to control tumor growth in resistant mice.

WHAT'S NEXT — Research around redesigning the gut microbiome to prevent cancer treatments from failing is still relatively new, and there are still questions future research will have to answer. For starters, the new study didn't uncover why androgen-producing bacteria increase in some people during ADT.

However, the new research also unexpectedly uncovered new information about how current therapies work.

The gut, or gastrointestinal tract, stretches from the mouth to the anus and includes the stomach.

There are two drugs currently used in ADT. One, androgen receptor blockers, prevent the effect of androgen from reaching prostate cells regardless of how or where the hormone is produced.

But drugs that block androgen production are theoretically only effective in blocking androgens produced through the normal pathways — not by bacteria.

"The authors somewhat unexpectedly show that at least one of these drugs, Abiraterone, also blocks the production of androgens from bacteria," Trinchieri says. "The clinical application of these data may not necessarily or uniquely be the targeting of the microbiome."

Doctors also have the bonus of better understanding which current drugs may work best for patients based on the bacteria living in their gut, and which drugs are able to target bacteria-produced androgens.

THE FUTURE OF CANCER TREATMENT

According to Trinchieri, research conducted in mice can't always be applied to humans, since the two species differ in many ways.

But that doesn't mean the new research isn't promising. The fact that androgen-producing bacteria were found at higher levels in both human patients and in mice undergoing ADT means there's a strong possibility that the mouse treatments may be effective in humans too, he says.

"ANTIBIOTICS OR A FECAL TRANSPLANT MAY REDUCE ANDROGEN-PRODUCING BACTERIA."

The results are particularly promising since the gut microbiome has been implicated in other cancers as well.

For example, a small study published in July in *Science* targeted specific gut microbiota known to interfere with immunotherapy melanoma treatments. The researchers, including Trinchieri, conducted the first human trial that used fecal transplants to attempt to redesign the gut microbiome to be more conducive to cancer therapies. In six of the 15 patients, it worked.

According to Trinchieri, targeted treatments are key when working with an ecosystem.

"The relative number of bacteria in the gut depends on maintaining equilibrium with all the other bacteria. We really are not yet able to kill off one or two bacterial species while preserving the others," says Trinchieri. "Antibiotics or a fecal transplant may reduce androgen-producing bacteria at the expense of killing other species and disrupting the ecosystem."

Because ADT is a long-term therapy used to keep tumors from growing rapidly, rather than curing prostate cancer, future treatments for ADT resistance will need to be effective long-term.

"Using a species that ecologically competes with the androgen-producing bacteria may be a smart approach," says Trinchieri.

Abstract: The microbiota comprises the microorganisms that live in close contact with the host, with mutual benefit for both counterparts. The contribution of the gut microbiota to the emergence of castration-resistant prostate cancer (CRPC) has not yet been addressed. We found that androgen deprivation in mice and humans promotes the expansion of defined commensal microbiota that contributes to the onset of castration resistance in mice. Specifically, the intestinal microbial community in mice and patients with CRPC was enriched for species capable of converting androgen precursors into active androgens. Ablation of the gut microbiota by antibiotic therapy delayed the emergence of castration resistance even in immunodeficient mice. Fecal microbiota transplantation (FMT) from CRPC mice and patients rendered mice harboring prostate cancer resistant to castration. In contrast, tumor growth was controlled by FMT from hormone-sensitive prostate cancer patients and *Prevotella stercorea* administration. These results reveal that the commensal gut microbiota contributes to endocrine resistance in CRPC by providing an alternative source of androgens.

Christina Dieli-Conwright, PhD, MPH
Harvard: Dana-Faber Cancer Institute

Exercise is Medicine

What this means for patients: Dr Dieli-Conwright has shown that exercise significantly benefits patients with prostate cancer, including improving fitness and quality of life, reducing obesity and other metabolic problems, and reducing muscle wasting. Exercise is a key "prescription" for better outcomes.

The use of exercise to enhance the lives of people diagnosed with cancer dates back 100 years, when doctors noticed an inverse relationship between cancer mortality and "muscular work." The field of exercise oncology has gained ground, especially in the last 10 years, as studies verified the many health benefits linked to consistent exercise. Much like diet, exercise is known to improve physical and mental quality of life for everyone, with very probable additional benefits to patients with prostate cancer. Today, exercise guidelines have been established for cancer survivorship, and include both aerobic and resistance exercise.

Dr Dieli-Conwright reported on several clinical trials of exercise inpatients with prostate cancer, especially among those undergoing ADT. Exercise interventions had multiple health benefits, including reduced waist circumference, greater lean mass, and improved fitness. Patients on active surveillance participating in high-intensity interval training had lower PSA levels and slower rise in PSA. Obese men saw improvements, such as a lower chance of developing type 2 diabetes. Overall, exercise should be considered paramount for patients seeking to optimize their health and quality of life during and after treatment. Future studies will help identify the most effective exercise "prescriptions" for prostate cancer survivors.

Giving Cancer a "Brown-Out"

Limiting Prostate Cancer's Fuel by Restricting Calories and Changing the Diet

Source:
22 Feb 2021
Janet Farrar Worthington
https://www.pcf.org/c/giving-cancer-a-brown-out/

Just when it seems like the picture of diet and prostate cancer is finally coming into focus, PCF-funded scientist Nicole Simone, M.D., a radiation oncologist at Thomas Jefferson University, has added a new dimension. It may not be just a question of the good foods you *do* eat and the bad foods you *don't* eat: It also appears to matter, very strongly, *how much you eat at all.*

Simone's research in prostate cancer and also in breast cancer suggests that restricting calories has many anti-cancer effects in the body – including, in mice, decreasing the likelihood of metastasis. It lowers inflammation, changes the gut microbiome, may decrease the side effects of systemic therapy and generally seems to slow down cancer. In effect, caloric restriction gives cancer a "brown-out," limiting its energy. "We're just beginning to understand the promise and the power of caloric restriction," says medical oncologist and molecular biologist Jonathan Simons, M.D. "If there were a drug that could do all these things, we'd prescribe it in a heartbeat."

Wait… aren't people with cancer supposed to keep their calories *up*? If you're thinking that *limiting* calories when someone's fighting cancer seems like the opposite of the common wisdom – well, you're right! "This is not what we were all taught in medical school," says Simone. And she's not entirely sure why this approach produces as many good effects as it does – but here's a clue: One way to look for various forms of cancer is with a PET scan, which involves injecting a radioactive dye. "That dye is actually a radio-labeled *glucose*," which is eagerly taken up by tumor cells because "cancer loves to eat. Cancer is metabolically active, and sugar is one of its favourite foods!"

Simone's laboratory has been investigating caloric restriction for several years. "Initially, we were looking for a way to increase the effectiveness of radiation and chemotherapy in tumors that have a poor response to standard therapies." In mouse models of hormone-sensitive breast cancer, Simone found that simply restricting the mice's daily caloric intake made a big difference: it not only altered cell metabolism and made cancer cells more vulnerable to radiation and chemotherapy. It also "decreased melastasis and increased overall survival."

If this worked in breast cancer, would it work in prostate cancer? Yes! "In several models of hormone-sensitive prostate cancer, we found the same," she says. "We were able to decrease tumor growth, decrease metastasis, and increase survival." Then Simone's lab tested caloric restriction in mice with castrate-resistant prostate cancer (CRPC), cancer that is no longer controlled by androgen deprivation therapy (ADT). Again, caloric restriction affected how tumors responded to radiation. "We saw some really interesting systemic, molecular changes," Simone says. "We wanted to take it a step further, and use that preliminary data as a launching pad to see what would happen in patients with prostate cancer if we put them on a caloric restriction diet."

Eating 25 percent less: In a pilot study, 20 patients – men diagnosed with localized prostate cancer who were scheduled to have prostatectomy – underwent caloric restriction for 21 days. Simone individually tailored each man's daily calorie total, based on what he had reported eating for several days ahead of time. "We figured out their average caloric intake and then decreased that by 25 percent." Simone's team also gave the men some dietary guidelines, encouraging (but not requiring) an anti-inflammatory diet with less refined sugar and processed food, more fruits, vegetables and complex carbohydrates. "The men were able to stick to the diets really nicely," she says. "We went over their diet logs and calculated their dietary inflammatory index. They did increase their anti-inflammatory foods! They also lost an average of 12 pounds each."

Could just three weeks of restricted-calorie, pretty much anti-inflammatory diet make a difference? Yes, in several ways:

A decrease in systemic inflammation. Men had changes in inflammatory markers in the blood, including a lower sedimentation rate (a blood test that measures inflammation).

Changes in the gut microbiome. Rectal swabs, taken before the men started the diet and three weeks later, were sent to PCF-funded investigator Karen Sfanos, Ph.D., at Johns Hopkins, who performed in-depth analysis. In the swabs taken at three weeks, Sfanos found a significant change in the gut microbes known to produce more butyrate! Butyrate is an important fatty acid that helps control inflammation and is made by beneficial bacteria. The fact that these microbes that make butyrate increased suggests that the population of bacteria in the gut changed for the better, simply with caloric restriction and an anti-inflammatory diet.

(continued page 5)

Less inflammation in the gut wall, as measured by lipopolysaccharides (LPS) in the blood. "When there is inflammation in the gut, it creates spaces between the epithelial cells in the gut wall." Inflammatory cells can "leak" out of the gut into the blood, and increase inflammation elsewhere.

Less inflammation in the tumor. "We saw a decrease in inflammatory markers such as NF-κB (an inflammatory pathway) in the tumor itself, and in MIR21." MIR21 is a microRNA gene (which makes RNA instead of proteins) that is believed to drive cancer development, growth, metastasis, and resistance to treatments. Simone is discussing this aspect with another scientist she met at PCF's Scientific Retreat, Shawn Lupold, Ph.D., of Johns Hopkins, who is a pioneer in the study of MIR21.

Ultimately, Simone believes, caloric restriction can play an important role for men with all stages of prostate cancer – but to make it even more effective will also require precision nutrition, based on precision oncology. In this case, that means figuring out whether someone's cancer prefers a diet that is sweet or savory. "Prostate cancer can metabolize through the glucose pathway, or through lipid pathways," says Simone. Understanding which pathway really appeals to a particular cancer – some prefer sugar, some really go for fat – "can tell us how your cancer is driving its own energy."

Thus, "if the tumor's feeding on lipids, we change the dial on fat content in the diet." And if the tumor prefers sugar, then a diet aimed at keeping sweets and simple carbohydrates to a minimum will foil the cancer's gustatory pleasure.

One of the biggest challenges with chemotherapy, ADT, or even radiation therapy, is resistance to treatment: the cancer evolves to minimize the damage of attempts to kill it. "Diet can almost be a more powerful tool," says Simone. "Cancers get smarter; a drug will work well for a while, then all of a sudden, cancer will figure out a way around it. The power of restricting food is that it provides less energy for the cancer to use up."

Note: Caloric restriction is done under careful supervision by medical professionals. It is strongly recommended that you talk with your doctor before making changes to your diet.

Prostate Cancer Foundation
Curing Together.

Wake Up! It's Time to Address Sleep Issues in Prostate Cancer

Stacy Loeb, MD, MSc, PhD (Hon)

New York University; Manhattan Veterans Affairs Hospital

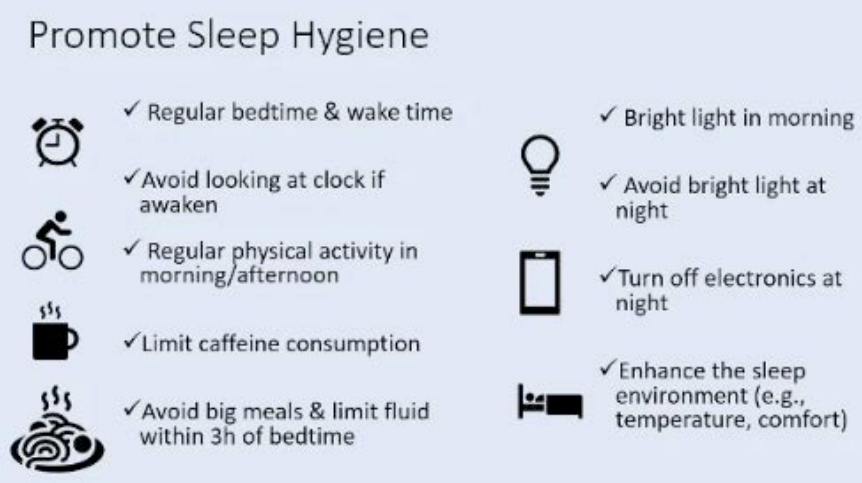
What this means to patients: Sleep is important to physical and mental health. Sleep disturbances are experienced by the large majority of prostate cancer patients and caregivers. More studies into the links between sleep and prostate cancer, as well as interventional studies to improve sleep in patients are needed to improve patient outcomes and quality of life.

Sleep disturbances—such as insomnia and obstructive sleep apnea—are common and are known to have both mental and physical health consequences. Several studies have investigated the relationship between sleep or circadian rhythm disturbances with prostate cancer risk, and many, but not all, suggest an association.

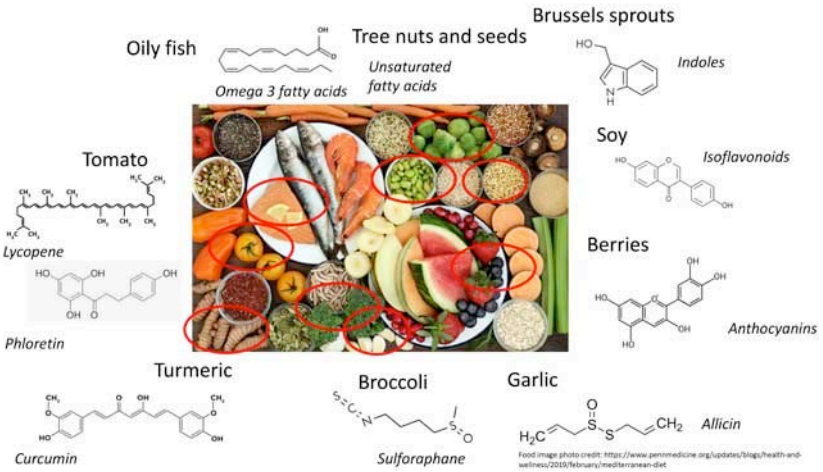
Dr. Loeb and team used a number of methods to study the links between sleep/circadian disruptions and prostate cancer. These include "social listening," a method that evaluates posts on online prostate cancer communities, surveys of patients and caregivers, and reviewing scientific studies.

Social listening studies found that sleep was a common concern among prostate cancer patients. Surveys found that sleep disturbances are very common among patients and caregivers, with 67% of patients and 88% of caregivers meeting cutoffs for poor sleep quality. However, a survey of urologists found that sleep is rarely discussed with patients and sleep quality is rarely measured.

The team recently initiated a trial that will test a 3-month digital sleep intervention in prostate cancer patients. Dr. Loeb's practical suggestions for improving sleep hygiene include:



The Complex, Natural Biochemistry of a Healthy Diet



Source:
27 Sept 2021
http://cure.pcf.org/site/R?i=ZcXjnTLP3Or2hS2ajpbjPlFJZyWHVp1g8tg-NfXYJ7XxGYcHYS2H5Q

During Prostate Cancer Awareness Month, PCF hosted a webinar, "The Science of Nutrition and Prostate Cancer." The accomplished nutrition researcher Prof. Richard Mithen, presented an overview of diets and foods that have been linked to a lower risk of cancer, and in some cases, prostate cancer. Mithen, professor of nutrition at the University of Auckland and PCF Challenge Award recipient, has been a leader in this field for decades.

Prof. Mithen began with the "Big Picture" by outlining the benefits of plant-based and Mediterranean diets, emphasizing the importance of eating a large variety of plant-based foods. Oily fish, such as salmon, is a healthier animal protein alternative. These general principles, along with regular exercise, offer a path to good overall health.

Prof. Mithen then went on to discuss a variety of fruits, vegetables, protein sources, and even spices that have the potential to affect health, and possibly prostate cancer specifically. Although it's tempting to believe that diet is an exact science, there is a lot of complex biochemistry associated with it. This is because unlike taking a medicine (which has a high concentration of one molecule made to target a specific protein or chemical reaction in the body), diet means that you eat small amounts of a large number of molecules, creating an intricate web of reactions with many changing variables. Broccoli, for instance, contains many phytochemicals and nutrients in addition to cancer-fighting glucoraphanin (more on that below), including: fiber, vitamins, and minerals. All of these affect the body in some capacity, and may differ somewhat from person to person.

Broccoli remains at the forefront of Prof. Mithen's research, as current evidence suggest that it offers meaningful potential to reduce prostate cancer risk or risk of cancer progression. This is because broccoli contains glucoraphanin, which is converted to the active molecule sulforaphane by the gut microbiota (a.k.a., "bugs in your gut"). Within a few hours, sulforaphane is absorbed throughout the body and accumulates in the prostate gland. Sulforaphane has general health benefits due to its ability to turn on hundreds of genes in the liver associated with anti-oxidant defense, anti-inflammation, and the excretion of foreign pollutants. Beyond that, sulforaphane may directly affect the prostate itself by fighting the growth of tiny cancers that have the potential to become larger. Prof. Mithen has developed new varieties of broccoli with different amounts of glucoraphanin, including those with up to 7 times higher than regular broccoli. In a PCF-funded study, men with localized prostate cancer on active surveillance consumed a "broccoli soup" weekly. After 12 months, men who ate the broccoli soup containing the highest amounts of glucoraphanin had reduced changes of expression in their prostate gland of genes that are thought to drive cancer progression, suggesting that glucoraphanin (sulforaphane) may indeed directly affect the risk of aggressive prostate cancer.

Mithen concluded his presentation by emphasizing that while there is no dietary magic bullet—not even broccoli—lifestyle changes including more plant-based foods, less red meat and dairy, and increased exercise lead to better health, and certainly will not cause harm. Examples of foods containing important phytonutrients include broccoli, turmeric, tomatoes, garlic, Brussels sprouts, and berries....and there are so many more to choose from.

ADT: What You Really Need to Know

Source:
By JANET FARRAR WORTHINGTON
https://www.pcf.org/c/adt-what-you-really-need-to-know/

Prostate Cancer Foundation
Curing Together.

The only people who really like androgen deprivation therapy (also called ADT, or hormonal therapy) are the drug companies that make billions of dollars a year selling the drugs. Doctors don't like it, and men don't like being on these drugs.

So why do it?

There are very few specific situations when ADT therapy is the right thing to do. These are the most common:

* *Intermediate-risk men who are given six months of ADT plus external-beam radiation;*

* *High-risk men who are getting radiation therapy*. This is a finite course of ADT, and this combination – two or three years of ADT plus external-beam radiation – has been proven to cure cancer in many men.

* *Men with metastatic prostate cancer*. ADT can make a big difference in these men, in relieving their symptoms and dramatically improving their quality of life. It can also extend life – some men have been on ADT for 20 years and are still going strong.

Who should not get ADT? Anybody else with prostate cancer. If you just have a rising PSA after radiation therapy or radical prostatectomy, that is not a good enough reason for a doctor to put you on ADT. If your doctor wants to put you on ADT to "shrink your prostate" before brachytherapy, that's not a good enough reason. [https://www.pcf.org/about-prostate-cancer/prostate-cancer-treatment/immunotherapy-prostate-cancer/]

ADT has never been shown to extend life if it's given too soon.

Why not just give it? At least it's doing something, rather than sitting around waiting for the cancer to spread. Well, that sounds good. Please refer to the previous paragraph, and read the last sentence again. Now, if you have a rising PSA, there are other things you can do that may help a lot. These include:

- Salvage surgery or radiation, if your doctor thinks the cancer is still confined to the "prostate bed," the area around the prostate. (Note: In this case, if you get salvage radiation, your radiation oncologist may want to put you on a limited course of ADT, which is one of the two specific acceptable situations for ADT; see above.)
- Immunotherapy; a vaccine such as Provenge, designed to boost your body's ability to fight off the cancer.
- Early chemotherapy.
- A clinical trial testing a promising new drug.

Don't get us wrong; we're not hating on ADT. If you need it, you need it. But it's not just like taking a vitamin supplement or getting a flu shot. There are serious side effects with long-term ADT – things that testosterone normally helps protect you from – including thinning of bones, loss of muscle mass, weight gain, loss of libido, hot flashes, mood changes, depression and, our main subject here, the risk of cognitive impairment.

Before we get into that, let's take a brief detour into the metabolic syndrome.

"Metabolic syndrome" includes an unholy cluster of bad things that can lead to a heart attack or stroke. Elevated blood pressure; unhealthy levels of blood sugar, cholesterol, and triglycerides; and abdominal fat – a big jelly donut of visceral fat, also known as "heart attack fat," right around your belly, a cardiac spare tire. A big gut equals a bigger risk for diabetes, heart attack and stroke.

All of this is magnified with ADT.

Maybe you already have some of these risk factors; maybe you've already had a heart attack, or you've got diabetes. If you need ADT, you need it.

(continued page 8)

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

But hear these words: You will need to fight what it's doing to do to the rest of your body, even as it saves you from your prostate cancer.

You will need to get mad at it. Work hard to take back your life – work doubly hard, because not only will it try to turn you into a tub of butter, but you might get mildly depressed. Your brain will tell you that you're too tired to exercise. It's deceiving you. *You must not listen to it. Exercise anyway.*

Here's what you're up against: Normally, if a man wants to lose a pound, he needs to burn 3,500 calories. A man on ADT who wants to lose that same pound needs to burn 4,500 calories. He's slogging upstream with ankle weights. His metabolism is slower, his sugar metabolism is messed up, his blood pressure may be higher, and for many reasons, he probably feels like crap. Maybe he stops taking care of himself. *This is the worst thing he can do.*

You need to be aware of this, because it might not be on your doctor's radar.

Just as important, you need to enlist your family and friends, NOT ONLY to help push you to exercise and eat right – cut way down on the carbs and sugar, especially – but to tell you if you seem depressed, because depression might have snuck up on you, and you might not have noticed it.

All of these things can be fought. However, if you just go back to the urologist or oncologist for a 5-minute appointment and another Lupron shot, you are probably not getting the monitoring you need. Depression may not show up in a brief doctor's visit. Even if the scale shows that you've put on weight, your doctor might say, "Well, that's common with ADT."

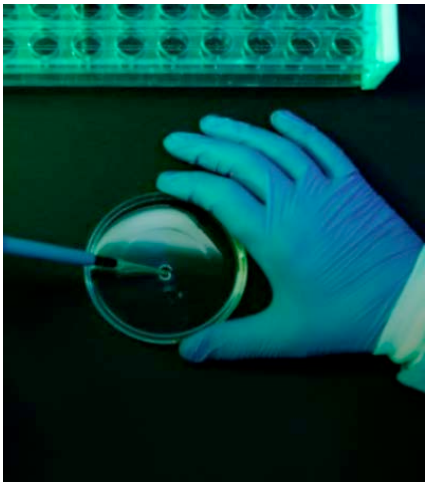
Years ago, when doctors first started using ADT, men didn't live that long. Now, men are living for years or even decades on ADT, and if that stops working, there are other drugs that can help, and exciting new types of drugs showing amazing results for some men in clinical trials. This is very good news; however, the downside is that doctors might just think, "hey, it's great, he's still alive and his PSA is not moving up."

But we know that weight gain is not only a common side effect of ADT; it's bad. *It's also something you can help prevent.* You need to exercise, with cardio (walking, swimming, riding a bike, aerobics, jogging, etc.,) plus weights for strength. These can be light weights; you don't need to turn into Arnold Schwarzenegger and bench-press a Volkswagon Beetle or anything like that. You just need to keep your muscles working. Exercise will help with depression, with the cardiac risks, and with the risk to your brain. As University of Colorado radiation oncologist E. David Crawford, M.D., recently put it, "What's heart healthy is usually prostate-cancer healthy... I've got a number of (patients on ADT) who are in great shape and they're tolerating [treatment] quite well. These are the people who are out there, who continue to lift weights, they continue to exercise, they watch their diet."

The metabolic syndrome that ADT causes may be a major reason – nobody knows for certain yet – why some men who are on ADT have cognitive impairment.

Andropause and the Treatment Nobody Talks About

Androgen Deprivation Therapy (ADT) causes andropause. It's like menopause, but it's the male version. Male hormones including testosterone are called androgens, and ADT basically shuts down all of those hormones. ADT has a lot of side effects, including a higher risk of cognitive impairment, but the biggest elephant in the room is the fact that men on ADT lose their sex drive and ability to have an erection. This is not ED, where the desire is there but the performance is difficult. No, this is a total lack of libido. Women are very good at talking about the problems they're experiencing with treatment for breast cancer, including their own hormonal therapy. Men, not so much. "Men don't like to talk about a flaccid penis with another man," says medical oncologist Jonathan Simons, M.D., CEO of the Prostate Cancer Foundation. "Men just generally don't talk about hormonal therapy with each other. And doctors – we don't talk about it the right way. This is not an easy topic: 'We're going to extend the quantity of your life and try to keep up the quality of life, but we're going to take away your maleness.' That's not an easy conversation." Also, Simons adds: "Most men won't complain. We don't always know what they're going through." Another wrinkle in ADT: men can have vastly different responses. "Around three to four percent can be on hormonal therapy with advanced disease for 10 years and not progress. For the vast majority of men, the benefit in controlling their cancer is between three and 12 months, and then the disease no longer responds to the treatment, and we have to add something else." However – and this is a big however – some men are "long-term exceptional responders to hormonal therapy," and are living for 20 years or longer with no apparent progression of their cancer. We don't know why this is. "We're not putting men on ADT just to make them miserable," says Simons. "We're doing it because the androgen receptor is a central part of what activates the 'on switch' for prostate cancer." That said, Simons, and the PCF, and many scientists around the world are working hard with the goal of not needing ADT anymore. Of finding another way to control or kill advanced cancer without needing to put men through these side effects. This is research the PCF is actively funding, and this is something we should be talking about.



Unlocking the Secrets of Sleeping Cancer Cells

Source:

22 Nov 2021
Health Report: Dr Norman Swan
https://www.abc.net.au/radionational/programs/healthreport/unlocking-the-secrets-of-sleeping-cancer-cells/13642138

Sometimes radiation and chemotherapy don't kill cancer cells. Instead cells go into a state of hibernation called senescence. We discuss what senescence is, what triggers cancer cells to 'reawaken', and potential treatments in the pipeline.

Guest:
Dr Shom Goel
Consultant Medical Oncologist

With a cancer diagnosis, after the initial shock and then the treatments, which each take their own toll, can come a new fear; will the cancer come back? Treatments like chemotherapy and radiation are designed to kill cancer cells, but sometimes they just send them into a dormant state called senescence, which researchers think might be behind some recurrent cancer.

A team at the Peter MacCallum Cancer Centre has just begun an eight-year project aiming to understand cancer senescence, figure out what might trigger senescent cells to reawaken, and develop treatments to target these sleeping cancers. I spoke to group leader Shom Goel earlier.

Shom Goel: So when you think about your body or tissues in your body all made out of cells, most of your cells are either sitting there quietly or dividing at any given time. But very occasionally when a cell gets damaged or injured in some way it can enter this unique cellular state known as senescence. And when a cell decides that it's going to go down that senescent path, it changes dramatically.

The first thing that is classical for a senescent cell is that it stops dividing. Secondly, it changes shape. It also changes its size. And most importantly, senescent cells switch off and on hundreds of genes which are not normally on and off in non-senescent cells, so they have a completely unique behaviour as well.

Most commonly, senescence has been studied in the context of ageing. As we age, even through the process of healthy ageing, our cells do sustain damage over the years. What my team is studying is cancer cells that have also entered this state of senescence, and trying to understand how that might impact the way cancers respond to treatment and the way cancers behave.

Tegan Taylor: Someone gets chemotherapy or radiation therapy, that's designed to kill the cancer cells, but sometimes it sends them into this senescent state where they are basically like Sleeping Beauty, they are not doing anything but they could be reanimated.

Shom Goel: That's exactly right, so all of our standard cancer treatments such as chemotherapy, radiation treatment and also some of the more modern cancer treatments that we use are, in an ideal world, supposed to kill a cancer cell, and hence eradicate a tumour. But far too often I think we all know that that doesn't happen, and then treatments work for a period of time and then stop working. And what's becoming clear is that one of the reasons for this is because some of the cancer cells, rather than die, enter this state of senescence in response to the injury that the treatment inflicts. And that might be considered all well and good because, as I mentioned, senescent cells are not dividing. However, what we have become clear on in recent years is that these senescent cells can be bad actors in the long-term and ultimately these cells, as you say, can reawaken, can start dividing again, and ultimately threaten the patient's life.

Tegan Taylor: So how would they be doing bad things to your body if they are not dividing?

Shom Goel: The most concerning thing about these senescent cells is that over time they will actually escape senescence. It's true that if these cells were put to sleep and stayed asleep forever, we might be able to live with that, but because we know these cells will almost invariably escape senescence, that's why we need to try and target them at the point when they are asleep so we don't have to worry about future problems.

Tegan Taylor: Can you tell that it's a cancer cell that's still there but it's just not dividing?

Shom Goel: The answer is yes. We are particularly good at identifying senescent cells in the research lab. So, often when we do experiments we are looking at cancer cells growing in a dish. Sometimes we're looking at cancers growing in mice, and when we treat those cancer cells with chemotherapy and radiation, it's very easy to see when they have entered senescence.

There is also good evidence and mounting evidence that the same exact process does occur in humans with cancer, although for purely practical reasons of not being able to perform biopsy after biopsy of a patient's tumour, we don't have as much evidence of senescence happening in human

(continued page 10)

cancer, but we are very confident that the same processes do take place.

Tegan Taylor: Okay, so your cell hasn't died, it's gone into this senescent state, and you say that there is this chance that it could reawaken and start growing again, which is obviously I'm guessing the biggest concern. What would trigger that?

Shom Goel: So that process of senescence escape, when a cell decides that it's time to reawaken, is a really poorly understood process, and one of the big goals of our research is to understand what is it that might make a cell after months or even years suddenly reawaken. What we think might be going on is that there are changes in the senescent cells, not DNA, but rather in what we call the senescent cell's epigenome, that lead it to start proliferating again. In other words, for some reason, certain genes that were switched off during senescence suddenly become switched on again, and they drive the cell to start proliferating, to start dividing. But the deeper question of why that happens is actually a black box at the moment and it's something we are very focused on understanding in the lab.

Tegan Taylor: So tell me about what your research is looking at now.

Shom Goel: So, our research is really focused on two things. The first is understanding the biology of senescent cancer cells in much more detail. At the moment our understanding of senescence in other contexts such as ageing is moving along quite nicely. Our understanding of the biology of senescent cancer cells is still very superficial.

The second big part of our research is to develop new treatments to kill these senescent cells. We don't think we can understand how we can kill them until we first understand what makes them tick.

Tegan Taylor: I was going to ask you what the solution is here, whether it is being able to identify them, or if it's about being able to kill them. If you just can kill them, does it even matter if you can identify them, in terms of treating patients?

Shom Goel: I think both aspects to this are really important. The way it typically plays out is that a lot of the work understanding when senescence happens and then how we can kill those senescent cells will take place in the lab. And once we have developed some novel therapies that can target these senescent cells, we will call them senotherapies, we would aim to move those pretty quickly into clinical trials in patients.

Tegan Taylor: How often is this happening where, instead of dying, these cancer cells are going into this state?

Shom Goel: We think it's a very common problem, and I think the most clear piece of evidence to suggest this is that we know when patients have cancer that has, for example, spread to other organs, our current treatments very rarely are able to cure that person's cancer. If our current treatments were able to eradicate all cancer cells from a patient's body at that point, we would think senescence is not a common problem. However, what we often see is that a cancer will respond to certain treatments, it will then sit in a stable state for some time, only to then regrow again in a more aggressive fashion. And the fact that that is so common lends us to believe that senescence is probably a common state in human cancer. The research that we will be doing initially will be focused on breast cancer, but we believe that the results we obtain are likely to be applicable to a broad range of cancers.

Treatment-Related Regret Common in Prostate Cancer

Source:
22 Nov 2021
Jody A Charnow
https://www.renalandurologynews.com/home/news/urology/prostate-cancer/men-with-localized-prostate-cancer-commonly-express-regret-about-treatment-decision/?utm_medium=email&utm_source=rasa_io

Treatment-related regret is driven in largely by a disconnect between patient expectations and outcomes, according to investigators.

Treatment-related regret is common among men with localized prostate cancer, especially among those who undergo surgery, new study findings suggest.

A disconnect between patient expectations and outcomes is a major contributor to regret, and appears to influence regret to a greater extent than factors such as disease characteristics, treatment modality, and patient-reported functional outcomes, according to investigators.

“Thus, improved counseling at the time of diagnosis and before treatment, including identification of patient values and priorities, may decrease regret among these patients,” a team led by Christopher J.D. Wallis, MD, PhD, of Mount Sinai Hospital in Toronto, Canada, concluded in a report published in *JAMA Oncology*.

Dr Wallis and colleagues conducted a population-based, prospective cohort study that included 2072 men with localized prostate cancer. Of these, 279 patients (13%) reported treatment-related regret at 5 years after their cancer diagnosis. The proportion of men who reported treatment-related regret was higher among those who underwent surgery (16%) and radiation therapy (11%) compared with those opted for active surveillance (7%) after adjusting for baseline differences.

Compared with active surveillance, surgery was significantly associated with 2.4-fold higher odds of regret, whereas radiation therapy was not significantly associated with higher odds of regret.

Results also showed that sexual dysfunction, and no other patient-reported functional outcomes, was significantly associated with regret, according to the investigators.

“Treatment preparedness that focuses on expectations and treatment toxicity and is delivered in the context of shared decision-making should be the subject of future research to examine whether it can reduce regret,” the authors concluded.

In an accompanying editorial, Randy A. Jones, PhD, RN, of the University of Virginia School of Nursing in Charlottesville, commented, “Considering multiple factors that are involved in providing patients with localized prostate cancer and the potential to enhance quality of life and decrease decisional regret, it is well worth the time for clinicians to assess and address patients’ treatment concerns.”

New Prostate Cancer Treatment Could Improve Outcomes for Advanced Patients

New implants could increase the sensitivity of prostate cancer cells to radiotherapy

Source: https://www.walesonline.co.uk/news/uk-news/new-prostate-cancer-treatment-could-22131345?utm_medium=email&utm_source=rasa_io

A new treatment to be used in combination with radiotherapy that could significantly improve treatment outcomes for men with locally advanced prostate cancer.

Researchers at Queen's University in Belfast have developed the treatment, which can make cancerous cells up to 30% more receptive to radiotherapy while simultaneously reducing adverse side-effects that limit quality of life.

Radiotherapy is extensively used to treat various localised cancers including prostate cancer, offering the best chance for curative intervention. However, approximately 30% of prostate cancer patients experience treatment failure leading to disease progression.

The research team at Queen's has developed a new nanomedicine comprised of tiny gold particles, coated in a small peptide called RALA. If these nanoparticles are present in tumour cells when treated with radiotherapy, they increase the cell-killing potential of this conventional treatment, helping to reduce the risk of disease relapse.

Professor Helen McCarthy, from the School of Pharmacy at Queen's University, said: "The peptide enables the gold nanoparticles to be delivered more efficiently to the tumour cells.

Dr Jonathan Coulter said: "Our research has shown that ultra-low concentrations of the RALA-gold nanoparticles effectively sensitise prostate tumour cells to radiotherapy. "Now we want to build on this work, to address the second major challenge, consistently delivering sufficient nanoparticles to the tumour throughout a patient's radiotherapy.

"We are delighted that Prostate Cancer UK is supporting our proposal to develop a biodegradable implant designed to provide sustained release of the gold nanoparticles. "Following insertion into the main tumour lesion, the biodegradable implant will consistently release the nanoparticles over time. This is opposed to current approaches that involve daily injections.

"Following consultation with a local prostate cancer patient focus group, we learned that a one-off implant would be better tolerated by patients than regular injections to the tumour."

The multi-disciplinary team has recently been awarded £376,000 from Prostate Cancer UK to evaluate the effectiveness of these implants at increasing the sensitivity of prostate cancer cells to radiotherapy.

Promising Treatments & New Methods

30 Nov 2021
https://www.pcf.org/c/pcf-scientific-retreat-2021-top-stories-for-patients-

PRINCE Trial Shows Promise For Combination of LuPSMA + Pembrolizumab

Shahneen Sandhu, MBBS

Peter MacCallum Cancer Centre, Australia

What this means for patients: LuPMSA is an emerging treatment for advanced prostate cancer that is anticipated to gain FDA approval in the next few months. The results of a recent clinical trial combining LuPSMA with pembrolizumab showed significant promise, with 73% of patients experiencing at least a 50% decline in PSA, and some patients having ongoing complete responses.

^{177}Lu-PSMA-617 (LuPSMA) is a groundbreaking new "seek and destroy" therapy that delivers a radioactive molecule to prostate cancer cells. It significantly improves overall survival in patients with metastatic castration-resistant prostate cancer (mCRPC). However, patients treated with LuPSMA eventually progress, and further optimization is needed. Radiation therapies are thought to cause cancer cells to die in a way that alerts the immune system, and thus may synergize with immunotherapy.

Dr. Sandhu and team led the PRINCE trial to test the combination of LuPSMA with the immunotherapy drug pembrolizumab in 37 patients with mCRPC. Results were encouraging, with 73% of patients seeing their PSA drop by at least 50%. At 24 weeks, 65% of patients had radiographic progression-free survival (rPFS; no worsening of disease on scans). Some patients have had deep and durable responses: For instance, one case was presented in which an 81-year old man experienced a complete response lasting over 60 weeks. Side effects were consistent with those observed for LuPSMA and pembrolizumab alone.

Further studies are needed to define the impact of adding pembrolizumab to LuPSMA on rPFS and overall survival.

Harnessing Immune Cells to Kill Prostate Cancer

Oliver Sartor, MD

Tulane University

What this means for patients: Bi-specific antibodies are a promising experimental class of treatments for advanced prostate cancer that leverage the body's immune system to kill tumor cells. Early-phase clinical trials show efficacy for several different bi-specific antibodies. Future studies will test new agents and address mechanisms of resistance.

There is a crucial need for effective treatments for metastatic castration-resistant prostate cancer (mCRPC). One option involves immunotherapy, helping the body's T cells to recognize, bind to, and kill cancer cells. Bi-specific antibodies are specially-designed proteins that have two (hence the "bi") parts and can bind to T-cells and tumor cells simultaneously. When these treatments are infused into the patient, they find their way to the tumor, bringing the T cells with them.

As one example, AMG 160 is a bi-specific antibody that binds to prostate-specific membrane antigen (PSMA) with one of its "arms" and T-cells with the other. In a phase 1 study, nearly 70% of patients had a reduction in PSA. Certain precautions are taken to lessen side effects associated with stimulating the immune system. AMG 160 is being tested in combination with other medicines, and a number of other therapies in this class are in early-stage clinical development for the

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

Prostate Heidelberg Cancer Support Group Meetings

Guest Speakers:

Tues 14 December 10:30am

Dr. Dixon Woon
Urological Surgeon, Uro-Oncologist
Senior Lecturer, Department of Surgery, University of Melbourne, Australia.
MBBS (Hons), DMedSc, FRACS (Urology)

"Prostate Cancer Treatment Progress over the last 10 years"

Dixon is a consultant urologist at Epworth Health, Austin Health, and Olivia Newton-John Cancer Wellness and Research Centre. He is also a senior lecturer at the Department of Surgery, University of Melbourne, Australia. He has a special interest in urological cancers.

Dixon completed his urological training at the Royal Australasian College of Surgeons (RACS) in 2016. He then went on to complete a further 2 years of fellowship training in robotic and open cancer surgery at the University of Toronto, at one of the largest cancer centres, Princess Margaret Cancer Centre. Dixon is now a member of the Society of Urologic Oncology (SUO), the peak North American body for urology cancer surgery.

In 2018, Dixon completed his Doctor of Medical Science degree through the University of Melbourne in advanced prostate cancer treatments and cancer immunology. As a result of his research in this field, he was awarded a number of grants and awards.

Dixon is passionate about research in the urology field, he has authored multiple peer-reviewed publications and has attended and presented at a number of international conferences.

TGA approves Australia's first PSMA PET/CT imaging agent

In a massive win for Australian men impacted by prostate cancer, Australia's Therapeutic Goods Administration has just approved Illuccix® for PSMA PET/CT imaging.

Made by world-leading global biopharmaceutical company Telix, headquartered here in Melbourne, Illuccix® enables the targeting of PSMA molecules in PSMA PET/CT scanning.

The game-changing agent labels with the radionuclide Ga-68 – allowing the imaging process to trace rogue cancer cells that may have spread throughout the body.

The TGA has granted Illuccix a broad clinical indication comprising:

1. Patients with prostate cancer who are at risk of metastasis and who are suitable for initial definitive therapy (also known as "primary staging"), and
2. Patients with prostate cancer who have suspected recurrence based on elevated serum prostate specific antigen (PSA) level (also known as "biochemical recurrence").

The approval means there is nothing to stand in the way of Medicare listing of PSMA PET/CT scanning for every man who needs it!

PCa Clinical Trials & Studies

For Further information on current and recruiting trials visit:

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

NINJA

The NINJA clinical trial aims to compare two emerging schedules of radiotherapy in the treatment of intermediate or high risk prostate cancer. Participants will be randomly assigned to one of two radiotherapy schedules as part of this study. In schedule 1 (called Stereotactic Body Radiotherapy) participants will receive 5 radiotherapy treatments over 2 weeks, and in schedule 2, (called Virtual High Dose Rate Boost), participants will receive Stereotactic Body Radiotherapy delivered in 2 treatments over 1 week followed by 12 treatments of conventional external beam radiotherapy over 2 and a half weeks. It is hoped this research will potentially improve the accuracy and quality of radiotherapy treatment in prostate cancer.

DASL-HICAP

The purpose of this study is to see if a new tablet drug, darolutamide, combined with the current best treatments, can improve outcomes for men with high risk prostate cancer that has not spread beyond the prostate area. Previous studies have shown promising results for darolutamide preventing disease progression and improving survival for men with advanced prostate cancer. This is a randomised controlled trial, which means that, in addition to best standard treatments, half the participants on the study will receive darolutamide, and the other half will receive placebo. The trial is being led from Australia by ANZUP in collaboration with the NHMRC Clinical Trials Centre. We plan to enrol 1,100 men from Australia, New Zealand, Canada, the US, Ireland, and the UK.

UPFRONTPSMA

Most prostate cancer cells have a molecule on their surface called prostate cancer specific membrane antigen (PSMA). PSMA can be targeted with Lutetium-177 PSMA (Lu-PSMA), a radioactive drug that kills prostate cancer cells anywhere in the body. This investigational drug is not approved for use in Australia by the Federal Government's Therapeutic Goods Administration (TGA). It is a new form of treatment that is effective in some patients with metastatic prostate cancer. It is a radioactive substance that, after injection into a vein, attaches to prostate specific membrane antigen (PSMA). The treatment enables delivery of highly targeted radiation to cancer cells. The emitted radiation only travels about 1mm, which means it mainly causes the killing of cancer cells, while avoiding healthy cells, and seems to be well tolerated with few side effects. This is called radionuclide therapy or theranostic therapy. The purpose of this randomised controlled clinical trial is to compare the effectiveness of Lu-PSMA therapy followed by docetaxel chemotherapy versus docetaxel chemotherapy on its own. Previous clinical trials have shown promising activity of Lu-PSMA in treatment of patients with metastatic prostate cancer. Docetaxel is a chemotherapy drug that is approved by the TGA to treat prostate cancer and has been used for many years in the treatment of metastatic prostate cancer. Since Lu-PSMA radiotherapy and docetaxel chemotherapy are both effective in treating metastatic prostate cancer, it is possible that using Lu-PSMA in addition to standard docetaxel chemotherapy at the beginning of the treatment course may improve patient outcomes when compared to treatment with docetaxel alone. A recent phase 2 clinical trial, showed the effectiveness of Lu-PSMA when used as a last treatment option and helped control disease progression. This study brings the use of Lu-PSMA forward as a first option to patients, with the hope of disease eradication and potential cure.

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

ENZA-P

Enzalutamide is a potent hormone therapy that prevents testosterone from reaching prostate cancer cells, thereby stopping cancer growth. It is already widely used in men with prostate cancer that has stopped responding to standard hormone treatments (castration-resistant prostate cancer). However, most cancers become resistant to enzalutamide over time, with almost 1 in 4 being resistant from the start of treatment. Many prostate cancers, in particular those that have spread or become resistant to hormonal therapies, have a substance on their cell surface called prostate specific membrane antigen (PSMA). Lutetium-177 PSMA (Lu-PSMA for short) is a new treatment in advanced prostate cancer. Lu-PSMA is a radioactive molecule that attaches to the surface of prostate cancer cells throughout the body. This drug is given as an injection through the vein and allows targeted radiation to be delivered directly to prostate cancer cells. Smaller pre-clinical studies have demonstrated synergistic effects by combining Lu-PSMA with enzalutamide. It is possible that Lu-PSMA can prevent early resistance to enzalutamide, extending the time that men benefit from treatment. The ENZAp clinical trial aims to compare the effectiveness of enzalutamide in combination with Lu-PSMA, versus enzalutamide alone for the treatment of prostate cancer. This is a randomised study, so half the men in this trial will be randomly allocated to receive Lu-PSMA and enzalutamide, and the other half will be randomly allocated to receive enzalutamide alone. We plan to enroll 160 participants across Australia.

GUIDE

The purpose of this study is to see if a prostate cancer marker in the blood (mGSTP1) can be used to guide chemotherapy treatment. Based on the level of this blood marker, some men may be able to have breaks in treatment rather than having chemotherapy continuously which is the current standard of care. This study will tell us if having these treatment breaks guided by mGSTP1 can improve how men feel during treatment while still treating the prostate cancer effectively.

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.

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

PHCSG Meetings 2021 10am – 12:30pm

Tues 16 Feb
Tues 16 March
Tues 20 April
Tues 18 May
Tues 15 June
Tues 20 July
Tues 17 August
Tues 21 September
Tues 19 October
Tues 16 November
Tues 14 December (including Xmas lunch – (subject to COVID)

Please note from Tues 16 November we are able to resume face-to-face meetings. Members will be able to log in via Zoom if they are unable to attend in person.

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

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 PC
- 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

September 2020
- ProtecT Trial showing patient outcomes after AM, RP & EBRT
- Changes in Penile Length after RP
- Active Surveillance for PC – is it right for you?
- The final part of The Perils and Pitfalls of "Treating PSA" in Advanced Prostate Cancer
- Managing Erectile Dysfunction – A Patient Guide

Prostate Cancer Trials
- Efficacy and Safety of Pembrolizumab (MK-3475) Plus Enzalutamide Plus Androgen Deprivation Therapy (ADT) Versus Placebo Plus Enzalutamide Plus ADT in Participants with (mHSPC)
- Navigate: An online treatment decision aid

October 2020
- World Osteoporosis Day
- Lifestyle Factors and Chronic Disease
- Hormone Therapy for PC
- Early ADT for Recurrent PC Challenged
- Unexpected aPC weakness can be targeted by drugs
- Hijacking an Epigenetic Program
- New PC Research: Immunotherapy; Gut Microbiome
- Veyonda New Research on Survival Rates

Prostate Cancer Trials
- MIndonline - mindfulness

November 2020
- Life insurance & Genetic Testing
- World First Surgery in NZ
- Melatonin increases survival
- SBRT disease control
- Public vs Private Hospitals
- Early ADT for Recurrent PC challenged
- Enzamet trial results

Prostate Cancer Trials
- Randomised Phase 2 of sequential 177Lu-PSMA & Docetaxel
- Exercise for Heart Health

December 2020
- ACTA Trial Award
- Rethinking Metastasis
- ESMO Phase 1 AMG160
- Five Ways to Get it Right
- Immunotherapy Offers Hope
- SBRT Doubles Pain Response
- Elevated Streess Hormone Levels

Prostate Cancer Trials