Prostate Heidelberg Cancer Support Group

Prostate Heidelberg

June 2022 Issue 219

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

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 face-to-face PHCSG Meeting
Tuesday 21 June 10am – 12:30pm

To join via Zoom: Copy link and paste into your browser
https://us02web.zoom.us/j/84453819052?pwd=YkFScHVYMEZieGVKVndBMVBSa0JuUT09

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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
BSB 083 256
Acct 583244292

Prostate Heidelberg Cancer Support Group Guest Speaker – 21 June

ADT – the Good, the Bad & the Ugly

Assoc Prof Joseph Ischia MB BS PhD FRACS (urology) is a urologist with a special interest in urologic oncology.

Joseph graduated medicine from Melbourne University in 1998 where he completed his PHD (2012) investigating the role of a neuroendocrine growth factor (gastrin-releasing peptide) and its precursor in the growth of renal cell cancer. He also completed a two year clinical and research post-doctoral uro-oncology fellowship at the Vancouver Prostate Centre in Vancouver, Canada.

You can listen to Joseph's podcasts https://www.talkingurology.com.au

Member News

Dry July 2022 The Long Run

Congratulations to David Campbell and his son Joel for completing the PCFA rally and raising over $6000 for Prostate Cancer. The final tally for the event was just under $200K, which will go towards prostate cancer research.

The PCFA have more fund raising events this year. Log on to their website to join and raise funds for a cause close to all our hearts. https://www.prostate.org.au

NEW PBS ITEM 61563 – PSMA PET scan

PSMA PET will be available on the PBS from 1 July 2022 but please note that this is a one off Medicare payment benefit in the patient's lifetime.

However this is great news for the initial staging of a patient with prostate cancer

Descriptor: 61563 Whole body prostate-specific membrane antigen PET study performed for the initial staging of intermediate to high-risk prostate adenocarcinoma, for a previously untreated patient who is considered suitable for locoregional therapy with curative intent. Applicable once per lifetime.

Indication: This item applies when requested by a specialist or consultant physician. The specialist or consultant physician is to record in the clinical notes and the request that the patient:

• has intermediate to high-risk prostate adenocarcinoma as defined below;

• has previously been untreated; and

• is considered suitable for locoregional therapy with curative intent.

Other requirements: • Patients with intermediate risk prostate adenocarcinoma can be defined as having at least one of the following risk factors in the absence of any high-risk features: PSA of 10-20 ng/ml, or Gleason score of 7 or International Society of Urological Pathology (ISUP) grade group 2 or 3, or Stage T2b.

• Patients with high-risk prostate adenocarcinoma can be defined as having at least one of the following risk factors: PSA >20 ng/ml, or Gleason score >7 or ISUP grade group 4 or 5, or Stage T2c or ≥T3.

• MBS fee: $1,300.00 Benefit: 85% = $1,212.10 75% = $975.00 Out of hospital Bulk billed benefit = $1,235.00

file:///Users/suelawes/Downloads/psma-pet-scans-medicare-benefits-schedule-items-1-july-2022-fact-sheet.pdf

Prostate Heidelberg Cancer Guest Speaker

Tues 16 Aug

Professor Avni Sali AMMBBS, PhD, FRACS, FACS, FACNEM
Member of the Order of Australia
Founding Director of the National Institute of Integrative Medicine.
Professor Avni Sali AM is often referred to as the father of Integrative Medicine in Australia. In 1996 he was the Founding Head of the Graduate School of Integrative Medicine at the Swinburne University in Melbourne. In 2009 he established the not-for-profit, charitable **National Institute of Integrative Medicine (NIIM)**, and became its founding Director. In the past he was also Head of the University of Melbourne Department of Surgery at Heidelberg Hospital.

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

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

My Cancer Care Record

Source: https://www.nemics.org.au/page/improving_cancer_care/My_Cancer_Care_Record/

NEMICS - *North Eastern Melbourne Integrated Cancer Service* have created a folder where PCa patients can keep details of their medical records.

My Cancer Care Record is a resource that helps people affected by cancer (patients, carers, families and support people) to manage the information related to their care and treatment. It has been developed by the Consumer Reference Group of the North Eastern Melbourne Integrated Cancer Service NEMICS). It provides tips on questions and information to ask health professionals. It can also assist you to record specific details that you may be frequently asked and find hard to remember.

The folder is aimed at helping with issues related to managing information when you have multiple treatments provided by multiple people, across different services and over long periods of time. It can assist you to be able to communicate across the variety of health care professionals involved in your care.

My Cancer Care Record can also help the clinicians working with you. It can provide easy access to information they often require such as:

- copies of your test results and letters from other hospitals/doctors
- current medication, medical and family history, current treatment schedules
- details of side effects and symptoms you might have had since your last appointment
- contact details of other clinicians involved in your care

The folder has nine key sections to help you organise your medical information: Health Summary; Medication; Contacts; Appointments; Tests and test results; Treatment; Support; Financial and legal; My Tab.
My Cancer Care Record can be used in either electronic or hard copy. More information on, including a link to request a free hard copy of the folder from NEMICS, is available here https://www.nemics.org.au/page/improving_cancer_care/My_Cancer_Care_Record/

Common Blood Test Results Explained

A/G RATIO (Albumin-Globulin Ratio) A measure of the relationship of the albumin and globulin proteins. Normally, albumin is greater than globulin.(0.8- 2.0)

ALBUMIN A blood protein manufactured by the liver. Marked changes may be related to liver abnormalities, poor diet or kidney disease. (3.5-5.4g/dl)

ALKALINE PHOSPHATASE A material in the blood related to liver and bone. Can show bone injury, pregnancy, or skeletal growth. Younger people have higher values Adults (39-117 U/L); Children (40-400 U/L)

ALT/SGPT/AST/SGOT (Serum Glutamic
Pyruvate Transaminase, Serum Glutamic Oxaloacetic Transaminase) Materials found in liver and muscle cells. Damage to these cells will increase the values. ALT (0-40), AST (0- 37)

AMYLASE An enzyme present in the pancreas. It may be elevated shortly after an attack of pancreatic inflammation. (I - 137 U/L)

APTT (ACTIVATED PARTIAL THROMBOPLASTIN TIME) A measure of one part of the clotting system known as the intrinsic pathway. Monitors anti-coagulant therapy. Related tests: PT, PTT, INR. (24 to 34 seconds)

B 12 (Cobalamin) One of the B vitamins the body needs to make bloodcells and to maintain a healthy nervous system. (400-900 pg/mL)

BASOPHILS and BASOPHIL COUNT Are WBC's usually involved in fighting parasitic infections. Known to carry histamine, heparin and serotonin. Increased levels found in allergic reactions. (0- 2%)

BICARBONATE/CO2 A body chemical, which is affected by a variety of medications. It reflects the acid-base balance in the body. (22- 29)

BILIRUBIN DIRECT A measure of liver or gallbladder abnormalities. (0.0 - 0.3)

BILIRUBIN TOTAL An indicator of bile pigment in the blood. Increases may be associated with liver abnormalities or breakdown of red blood eel.ls.
Slight increases may have no significance. (0.2 - 1.0)

BUN (BLOOD UREA NITROGEN) Measures the amount of nitrogen in your blood- that comes from the waste product urea. This test evaluates kidney function. (6 - 20 mg/di)

BUN/CREATININE RATIO A derived measure of two tests reflecting kidney function, normally varying from I 0: I to 20: I.

CALCIUM A material in the blood and bone. Abnormalities of the parathyroid glands or of bone may increase values. Poor nutrition, kidney disease or lack of vitamin D may decrease values. (8.8- 10.2 mEq/dl)

CHLORIDE A
body salt, which usually follows the same pattern as sodium. Changes are very frequent in normal people. (96 - I 08mEq/L)

CHOLESTEROL A blood fat, which is in part, related to eating animal fats. Increased values may indicate a tendency to hardening of the arteries (atherosclerosis). Values of 200 or lower are associated with decreased rate of heart attack. (< 200)

CO2 (Carbon Dioxide) Related to the respiratory exchange of carbon dioxide in the lungs. (20- 29 mEq/L)

CREATININE A waste product removed from the body by the kidneys. High values may be seen in muscle degeneration or kidney disease. (0.67-1.17 mg/di)

DIGOXIN Reflects the level of heart medicine in the blood. (0.5- 2.0 ng/ml)

EOSINOPHILS A type of leukocyte I (White Blood Cell) produced in bone marrow. These cells become active due to allergic reactions, infections and other medical conditions. (0.5- 0.50 K/uL)

(continued page 5)

Listed is an explanation of common blood test results that you may find on your own report, along with normal reference ranges.

Deviations from the normal range do not necessarily indicate problems and are not cause for alarm.

Healthy people often have variations from the ideal ranges. Ask your healthcare team to clarify what these tests mean for you.

And remember, you are entitled to receive copies of all your blood tests if you ask. They are your property so be insistent if you find any resistance in giving you copies of the tests.

For more information about the interpretation of test results see here

https://www.cancerabcs.org/understanding-your-medical-tests

ESR (ERYTHROCYTE SEDIMENTATION RATE) Shows inflammatory activity in your body. (0- 22mm/hour- Males; 0-29mm/hr - Females).

FOLIC ACID One of the many B vitamins needed to make red blood cells (RBC), white blood cells (WBC), platelets, DNA, and for normal growth. (2.7- 17.0ng/mL)

GGT (GAMMA-GLUT AMYL TRANSPEPTIDASE) Increased levels may be found in liver disease, bile duct obstruction, magnesium ingestion and alcoholism. Female (0-45U/L); Male (0- 65 U/L)

GLOBULIN A type of protein similar to albumin. The four major globulin The four major globulin groups are gamma, beta, alpha-2 and alpha-1. Globulins help fight infection and are related to immunity. Minor variations are common.

GLUCOSE A blood sugar. High values are seen in diabetes, stress, and tests performed soon after a meal. (55- 110 mg/di)

GLYCATED HEMOGLOBIN (HgbAIC) A measure of sugar control for the past three months. (4.0- 6.0)

GRAN (GRANULOCYTES) Are a type of white blood cell. Often increases with bacterial infection -there are three types - Neutrophils, Eosinophils and Basophils.

GRANPERCENTAGE Is the percentage of these cells in relation to all white blood cells.

HDL (HIGH DENSITY LIPOPROTEIN) The "protective" fraction of cholesterol. Values in excess of50mg/dl are desirable. (A minimum 12-hour fast is required for accurate determination). (>= 50)

HCT (HEMATOCRIT) Measures the percentage of red blood cells found in the whole blood. Low levels are associated with anemia. (38.3-48.5%)

Hgb (HEMOGLOBIN) The protein molecule that carries oxygen from the lungs to the body's tissues. (12.5-16.9 g/dl)

HTSH (HIGH SENSITIVITY THYROID HORMONE) the thyroid stimulating hormone level produced by the pituitary gland.

INR (INTERNATIONAL NORMALIZED RATIO) A test of blood clotting. It is used to regulate Coumadin or warfarin levels. Related tests: PT, PTT and APTT. (Recommended therapeutic range: 2.0-3.5)

LDL (LOW-DENSITY LIPOPROTEIN) The fraction of cholesterol that reflects the dangerous portion. Levels should be lower than 130. (A minimum 12-hour fast is required for accurate determination).(<= 100)

LDL/HDL A ratio of two cholesterol sub fractions sometimes used as a risk factor measurement. (4.4- 7.1)

LEUKOCYTES White Blood Cells that are part of the immune system helping to defend the body against infection. There are five types of leukocytes that are produced in the bone marrow.

LYMPHS (LYMPHOCYTES) a type of white blood cell often increased with viral infection. (0.09-3.50)
LYMPHS PERCENTAGE The percentage of lymphocytes in relation to all white blood cells. (18-48%) **MAGNESIUM** A chemical in the blood that is often increased in certain kidney diseases. (1.4- 2.0)

MID (MID SIZE) Are white blood cells

MID-PERCENTAGE the percentage of these cells in relation to all white blood cells.

MCH (MEAN CORPUSCULAR HEMOGLOBIN) A measure of red cell hemoglobin. (26.1- 33.7 pg)

MCHC (MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATE) CONCENTRATE) Measures the concentration of hemoglobin within the red cell. (32.2-35.1%)

MCV (MEAN CORPUSCULAR VOLUME) A measurement of red blood cell size. Higher ranges are found in newborns and infants. (79.5- 98.0 fl)

MONOCYTES –MONOCYTE COUNT Elevated levels are seen in tissue breakdown or chronic infections.(0-9%)
NEUTROPHILS AND NEUTROPHIL COUNT This is the main defender of the body against infection and antigens. (48-73%)
PLATELET COUNT Indicates one of the constituents of blood involved in clotting. (117- 361 thous/mcl)

PHOSPHORUS Required by the body for bone and teeth function. An essential mineral in human nutrition. (2.7- 4.5)

POTASSIUM A chemical in the blood affected by kidney disease and intestinal disease. (3.3- 5.1 mEq/L)

PSA (PROSTATIC SPECIFIC ANTIGEN) A prostate test that is elevated in infections, enlargement, inflammation, or prostate cancer.(< 3 ng/mL)

PT (PROTHROMBIN TIME) Evaluates the bloods ability to clot properly. Measures the time it takes the plasma of your blood to clot and can be used to detect bleeding disorders. Related tests: APPT, PTT and INR. (11- 13.5 seconds)

PTH (PARATHYROIC HORMONE) Measures the level of parathyroid hormone in blood. Helps to identify hyper and hypoparathyroidism or to find the causes of abdominal calcium levels. PTH controls calcium and phosphorus levels in the blood. (10- 55 pg/ml)

PTH- INTACT
Used in detecting parathyroid disease and for the differential diagnosis of calcium homeostasis disorders. Also used in monitoring individuals with renal disease. (15.0- 65.0)

PTT (PARTIAL THROMBOPLASTIN TIME) Used to measure the effect of anti-coagulant drugs on blood clotting. Related tests: PT, ATTP and INR. (30-45seconds)

RED BLOOD CELLS, HEMOGLOBIN, HEMATOCRIT Measurements of the red blood cell count. Low values are seen with anemia. RBC (4.07-5.74mill/mcl), Hemoglobin (12.5-16_-9 g/dl) Hematocrit (38.3- 48.5 %)

RHEUMATOID FACTOR Antibody measured in the blood that detects rheumatoid arthritis. High levels of Rh factor associated with more severe rheumatoid disease. Also present in other conditions such as

lupus, infectious hepatitis and tuberculosis(<= 14.0)

SED RATE (SEDIMENTATION RATE) or ESR (ERYTHROCYTE SEDIMENTATION RATE) Shows inflammatory activity in your body. (0- 22mrn/hour- Males; 0-29 mm/hour - Females).

SODIUM A body salt. It functions in the body to maintain osmotic pressure, acid-base balance and to transmit nerve impulses, Kidney disease, certain diseases of the adrenal gland, or dehydration may cause abnormal values. (133-145 mEq/L)

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PLEASE NOTE:
Terminology & normal test result ranges may vary in Australia. Please ensure you discuss your diagnosis and treatment options with your consulting specialist https://pathologytestsexplained.org.au

T4 TOTAL, T UPTAKE, FTI & **HTSH** Measures of thyroid function. The overall pattern is more important than any single value.

TOTAL CHOLESTEROL/HDL A ratio obtained by dividing total cholesterol by HDL. (Levels lower than 4.5:1 are desirable.)

TOTAL PROTEIN Indicates albumin and globulin, the two major protein types in the blood. Abnormalities may reflect a variety of illnesses. (6.6- 8.7g/dl)

TRIGLYCERIDES Blood fats related to total calorie intalce and consumption of starch and sweets. Alcohol will increase this value (< = 200)

TSH (THYROID STIMULATING HORMONE) A pituitary hormone that stimulates the thyroid to produce more thyroid hormone. (0.5- 5.0) (T4 and T3 thyroidhormone level s are often performed with this test).

UREA NITROGEN (See BUN) A measure of kidney function. Elevated levels are seen with dehydration and some cases of intestinal bleeding.

URIC ACID A material that, in excessive amounts, may deposit in the kidneys, causing stones, or in the joints, causing gout. (Male Ranges 3.5- 7.5 mg/di); (Female Ranges 2.5- 7.5 mg/di).

VLDL (VERY LOW DENSITY LIPROPROTEIN) a fraction of cholesterol often related to tryglycerides and LDL, which may play a minor role in the development of coronary artery disease. (A minimum 12-hour fast is required for accurate determination.)

VITAMIN D-25 HYDROXY The most accurate way to measure how much Vitamin D is in your body. (30.0- 74.0 ng/mL)

WBC (WHITE BLOOD CELLS) A measur e of the body's response to infection or nonspecific stress. Elevations are seen most commonly with bacterial infection.(4.8- 10.8 thous/mcl)

Glossary of Terms:

Prostate Cancer is full of acronyms.

To help you navigate all the terms we have produced a list on our Website:

www.prostateheidelberg.info

Don't allow Statistics to Dictate Your Treatment Choices – Understand What is Meant by Median Survival

When your doctor talks to you or you read about survival rates, for a type of cancer or a type of treatment you are being given a statistic. It is important to understand what statistics are and what they are not. They are nothing more than information about a group trend; they are NOT about you, an individual.

Never assume that a statistic will foretell your experience. It will not. You are one, an individual; you are not a group trend.

Researchers and the medical community talk about survival statistics, especially when they try to convey information to their patients and other professionals. They often do this by using a statistical term, or a group trend, including one, called the median.

Many people believe that a median is a different word for the average. Don't be confused as a **median is not the average**, and more importantly, it does not predict what will be the experience of an individual, in this case, you!

A median is nothing more than the number that falls in the exact middle of a list of numbers. One-half of the numbers are lower than the median and one-half of them are higher.

To illustrate this concept, take the following list of numbers representing how many months a group of nine people lived after receiving a cancer diagnosis: 1, 2, 3, 4, 6, 14, 20, 40, 65.

The survival number in the middle or the statistical median is six months because there are four numbers lower (1, 2, 3 and 4) and four numbers higher (14, 20, 40 and 65). The median survival for people in this example after having received a diagnosis was six months, but the average survival was 17.1 months.

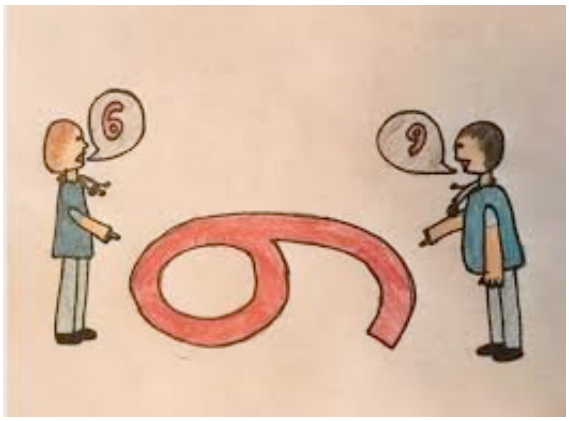
Note that there are four individuals (one-half of the group) of the nine who lived for many months beyond the median and three people lived well beyond the average of 17.1 months. Statistics do not tell you if you will be one of the 1-month survivors or if you will be the outlier of 65 months.

Scientists report the median, in this case, a 6-month survival, but the four individuals who beat the median lived much longer.

Don't let median survival numbers convince you that you will have a limited life or discourage you from having a treatment.

The IMPORTANT STATISTIC is the statistic of one, YOU. Statistics do not predict your story.

HTTPS://WWW.CANCERABCS.ORG/CANCER-AND-STATISTICS-UNDERSTANDING-WHEN-YOU-MIGHT-DIE



Getting a Second Doctor's Opinion

SOURCE:
https://www.cancerabcs.org/getting-a-second-doctors-opinion

Obtaining additional doctor's opinions is a CANCER PATIENT's standard of care. When you have cancer getting a second or third opinion from different doctors is not an option, it's a requirement. No matter how long you know your doctor, that your doctor is the best or that the doctor is world renowned, you need a second opinion.

Doctors have differences of ideas, they make mistakes, and they have different levels of experience, all of which leads to different recommendations. Some doctors are more conservative than others while others tend to be more aggressive. The approach to understanding and treating your cancer can differ from one doctor to another; for example, a surgeon can legitimately believe that surgery is the best course of action while a radiologist believes that radiation treatments are your preferred treatment protocol.

It is not unusual for findings and recommendations to differ dramatically. For this reason, you must always obtain second opinions after a cancer diagnosis.

There can be many benefits to getting a second opinion. Benefits can include peace of mind knowing that you are steering the correct course, developing a different treatment plan to even ending up with a different diagnosis.

It is easier if your second opinion just confirms what the primary doctor told you. A confirming second opinion will let you know that you have done everything you can to ensure that you have the correct diagnosis and treatment plan. Even in cases where the diagnosis is confirmed, a second opinion can open the potential of a different or additional treatment option that was not mentioned by the original doctor.

A second opinion will allow you to become more informed about what is available. Being fully informed will pave your way to better medical decisions, possibly a longer life, as well as an improved quality of life.

What Does Research Say About Second Opinions?

The need for having a second opinion has been borne out by scientific research. A study was done https://newsnetwork.mayoclinic.org/discussion/mayo-clinic-researchers-demonstrate-value-of-second-opinions/ at the Mayo Clinic in 2010 found that as many as 88% of patients looking for a second opinion will leave the second opinion doctor's office with a new or better-refined diagnosis. Meanwhile, 21% of patients will leave with a "distinctly different" diagnosis.

The Mayo study also showed that 12% of patients who had a second opinion left the doctor's office learning that the original diagnosis was not correct.

Another study conducted at the Johns Hopkins Hospital showed that medical errors rank as the third leading cause of death in the United States, supporting the need for you to have a second opinion.

When you do seek a second medical opinion, you should remember that an error is as likely to be coming from the second opinion doctor. Given this, if you find that the two doctors vastly disagree it would be wise to get a third opinion.

The key that should guide you is to keep digging until the diagnosis and treatment make sense to you. Anyone diagnosed with any cancer must get a second opinion. A cancer diagnosis is confusing, overwhelming, and it is a life-changing event.

No doctor, no matter how informed, is aware of the findings from every single study and clinical trial. Doctors are only human. That's why you need to advocate for yourself and to obtain as many additional medical opinions as you need. Getting other views will improve the likelihood that you will end up with the best possible treatment plan..

Second opinions should always come from doctors who are associated with different hospitals from each other. Like everything in life, hospitals have politics, and you want to be sure that your second opinion doctor isn't constrained by these types of concerns.

One of the most common concerns expressed, as a reason not to get a second opinion, has to do with insulting your doctor. Getting a second opinion is not an insult to your doctor, it is your standard of care. Oncologists expect that patients with cancer will get numerous medical opinions.

Most doctors welcome additional input because they recognize that they do not know everything. If they don't encourage you to have a second opinion you need to move on and find a different doctor. Only a primary doctor without confidence in their personal opinions fears a second opinion.

Remember, deciding to get a second opinion does not mean that you are difficult or that you are in denial about your diagnosis. Getting a second opinion is not only the standard of care you should be insisting upon; it means that you are smart.

As a Cancer [patient] you need to take an active part in your health care, getting a second opinion is an important component of the process.

Primary Care Use of FRAX: Absolute Fracture Risk Assessment

Source:
https://pubmed.ncbi.nlm.nih.gov/20107292/

Osteoporosis-related fractures (low-trauma or fragility fractures) cause substantial disability, health care costs, and mortality among postmenopausal women and older men. Epidemiologic studies indicate that at least half the population burden of osteoporosis-related fractures affects persons with osteopenia (low bone density), who comprise a larger segment of the population than those with osteoporosis. The public health burden of fractures will fail to decrease unless the subset of patients with low bone density who are at increased risk for fracture are identified and treated. Risk stratification for medically appropriate and cost-effective treatment is facilitated by the World Health Organization (WHO) FRAX algorithm, which uses clinical risk factors, bone mineral density, and country-specific fracture and mortality data to quantify a patient's 10-year probability of a hip or major osteoporotic fracture. Included risk factors comprise femoral neck bone mineral density, prior fractures, parental hip fracture history, age, gender, body mass index, ethnicity, smoking, alcohol use, glucocorticoid use, rheumatoid arthritis, and secondary osteoporosis. FRAX was developed by the WHO to be applicable to both postmenopausal women and men aged 40 to 90 years; the National Osteoporosis Foundation Clinician's Guide focuses on its utility in postmenopausal women and men aged >50 years. It is validated to be used in untreated patients only. The current National Osteoporosis Foundation Guide recommends treating patients with FRAX 10-year risk scores of > or = 3% for hip fracture or > or = 20% for major osteoporotic fracture, to reduce their fracture risk. Additional risk factors such as frequent falls, not represented in FRAX, warrant individual clinical judgment. FRAX has the potential to demystify fracture risk assessment in primary care for patients with low bone density, directing clinical fracture prevention strategies to those who can benefit most.

https://www.sheffield.ac.uk/FRAX/tool.aspx

Quality of Life in Men with Prostate Cancer Randomly Allocated to Receive Docetaxel or Abiraterone in the STAMPEDE Trial

Summary: This analysis of data from the randomised controlled STAMPEDE trial assessed patient-reported QOL (QLQ-C30 + PR25 questionnaire) outcomes in patients receiving abiraterone acetate plus prednisone or prednisolone (AAP) plus standard of care androgen deprivation therapy (ADT; n = 342) or docetaxel plus ADT (n = 173). Over 2 years, the mean mixed-model global-QOL score was higher by 3.9 points (95% CI 0.5-7.2; p = 0.022) in AAP plus ADT versus docetaxel plus ADT recipients. Global-QOL was higher with AAP plus ADT over the first year (5.7 points; 95% CI 3.0-8.5; p < 0.001), especially at 12 (7.0 points; 95% CI 3.0-11.0; p = 0.001) and 24 (8.3 points; 95% CI 4.0-12.6; p < 0.001) weeks.

Comment: Current systemic treatments for metastatic prostate cancer are seldom if ever curative and, with ever increasing survival from the sequential use of life-prolonging therapies, overall patient QOL is an important consideration. In this QOL study from the STAMPEDE trial, patients randomly allocated to abiraterone and standard of care ADT had consistently better global QOL measures compared to patients treated with docetaxel plus ADT, despite the time-limited use of chemotherapy (maximum 6 cycles/24 weeks) and need for continued steroid use with abiraterone out to the time of the primary endpoint measurement (24 months). Not surprisingly, differences in QOL favouring abiraterone were greater at earlier timepoints, when toxicity from chemotherapy would be expected to be at its greatest. Much to think about when choosing the first treatment.

Source:
Prostate Cancer Research Review
Authors: Rush HL et al.
Reference: Eur Urol Oncol. 2022;5(1):44-51

Penile Traction Therapy Improves Erectile Function, Penile Length After Prostatectomy

Source:
25 May 2022
https://www.practiceupdate.com/news/38057/32/3?elsca1=emc_conf_AUA2022Post-2&elsca2=email&elsca3=practiceupdate_uro&elsca4=202243_AUA2022Post-2&elsca5=conference&rid=NjYzMTc3OTU2NTUS1&lid=36656680

Radical prostatectomy poses a significant risk of developing erectile dysfunction and subjective penile shortening. Despite the advances in nerve-sparing surgical techniques, the incidence of erectile dysfunction has been reported to be as high as 78% to 87% in a large population study. Perioperative penile rehabilitation with PDE5 inhibitors and vacuum erectile devices has been standardized in many centers performing radical prostatectomy. However, studies have shown mixed results in terms of the protocol's efficacy in preserving erectile function. In addition, the patient adherence to such protocols has been reported to be poor due to the cost and perceived ineffectiveness.

In this study, the authors performed a randomized clinical trial on the efficacy of RestoreX penile traction therapy (RxPTT; PathRight Medical, Plymouth, Minnesota, USA) in improving the penile length and preserving erectile function post prostatectomy. Despite the low number of enrolled patients, this study demonstrated preserved sexual function and improvement of penile length with the use of RxPTT when compared with controls. Furthermore, the trial reported a high rate of satisfaction among patients using the traction device. Although the results are promising, patient adherence remains an expected challenge. The study protocol involves daily application (30–60 minutes) of the traction device for 5 months. In this trial, 82 men were randomized initially at 1 month, and, at 6 months post randomization, data were available on 55 men and on 42 men at 9 months. The findings of this study could be practice-changing, since this study demonstrated a durable response, especially if the protocol is applied early (4 weeks) post prostatectomy. A postulated mechanism, which has been recently reported in rat penile traction studies, may involve the tensile force-mediated release of nitric oxide and upregulation of nitric oxide synthase.

However, the results of this trial need to be externally validated on a larger set of patients before their clinical application. A new multicenter randomized controlled trial, NCT 05244486 (N = 200), has been initiated for both academic and nonacademic centers or providers for enrollment. Participants would receive a free RestoreX device and would be required to fill out related validated questionnaires during their treatment. No in-person visits are required. For further information, or if wishing to enroll prospective patients, please contact Email@peyronies.org.

NB This trial only appears to be in the US.

If validated, results would be significant advancement in the field

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.

SPPORT Trial: Whole Pelvic Salvage Radiation + Short-Term ADT after Failed Surgery

Source:
31 May 2022
https://www.prostatecancer.news/2022/05/spport-trial-whole-pelvic-salvage.html

In 2018, we saw the early results of the SPPORT randomized clinical trial. Now Pollack et al. has published the full results. To review:

They randomly assigned 1,792 men with a recurrence after prostatectomy in 2008-2015 at 460 locations in the US, Canada, and Israel to one of 3 therapies:

1. PBRT (prostate-bed radiation only)
2. PBRT + STADT (prostate-bed radiation + short-term ADT)
3. sWPRT + STADT (salvage whole pelvic radiation + short-term ADT)

- ADT consisted of 4-6 months of a combination of an anti-androgen and an LHRH agonist starting 2 months before salvage radiation.
- Radiation dose to the prostate was 64.8-70.2 Gy at 1.8 Gy per fraction.
- Radiation dose to the pelvic lymph nodes was 45 Gy at 1.8 Gy per fraction.
- The treated pelvic lymph node area was per RTOG guidelines and did not include the recently recommended expansion. (There is also an expansion of the prostate bed, as discussed here)
- The sample size was powered to detect progression-free survival, but not metastases, prostate cancer mortality, or overall survival. 8 years of follow-up is insufficient for those other endpoints.

The oncological results were:

- 8-year freedom from progression (biochemical or clinical) was 77% for sWPRT+STADT, 72% for PBRT+STADT, and 61% for PBRT (all significantly different, regardless of initial ADT, Gleason score, or stage). They used a nadir+2 definition of biochemical progression because it correlated best with clinical progression.
- At lower PSA (≤ 0.35), Group 3 did no better than Group 2, so widening the treatment area had no effect. Both groups did better than Group1, so ADT had a significant effect.
- At higher PSA (> 0.35), Group 3 was better than Group 2, but the difference was not statistically significant. Both groups did better than Group 1, indicating ADT effectiveness.
- 4 vs 6 months of ADT did not matter. It reduced the occurrence of local and regional metastases.
- Widening the treatment area reduced the long-term rate of local and regional metastases.
- 8-year incidence of metastases was 69 (12%) for PBRT (HR=0.71), 56 (10%) for PBRT+STADT (HR=0.74), and 41 (7%) for sWPRT+STADT (HR=0.52). sWPRT+STADT was significantly better than the other two.

The physician-reported acute toxicity results show some small early adverse effects of ADT and the wider treatment area:

- GI grade 2 or higher: 7% for sWPRT+STADT vs. 4% for PBRT+STADT vs. 2% for PBRT
- GU grade 2 or higher: 12% for sWPRT+STADT vs. 12% for PBRT+STADT vs. 9% for PBRT
- Bone marrow grade 2 or higher: 5% for sWPRT+STADT vs. 2% for PBRT+STADT vs. 2% for PBRT

The physician-reported late toxicity results show that late toxicity was not influenced by ADT or whole pelvic RT:

- GI grade 2 or higher: 9% for sWPRT+STADT vs. 10% for PBRT+STADT vs. 10% for PBRT
- GU grade 2 or higher: 40% for sWPRT+STADT vs. 35% for PBRT+STADT vs. 37% for PBRT
- Bone marrow grade 2 or higher: 4% for sWPRT+STADT vs. 2% for PBRT+STADT vs. 4% for PBRT

This RCT proved that whole pelvic salvage radiation with 4-6 months of ADT is the preferred salvage treatment.

In contrast to a previous trial (RTOG 9601) that told us that ADT can be safely avoided if PSA<0.7, this trial suggests at least 4 months of ADT and whole pelvic treatment. The reason for the difference in recommendations is due to the choice of endpoint. SPPORT is telling us that if we are willing to put up with 4 months of ADT and some extra short- term toxicity from the wider field of radiation, a cure is likely. RTOG 9601 tells us that if your PSA<0.7, you aren't likely to die if you don't get the extra short-term hormone therapy, but you may have to have lifelong ADT eventually. It will always be a managed disease. Patients should acknowledge these trade-offs and discuss with their doctors.

Results may possibly be improved further with:

- Better patient selection using PET scans (PSMA, Axumin, or NaF)
- Extra radiation to the prostate bed
- Boost doses to cancer detected with a PSMA PET scan (if PSA> 0.5 - but do not wait!)
- Selection of patients who would benefit from treatment intensification using a Decipher test
- Hormone therapy intensification in select patients

Persistent Testosterone Suppression after Cessation of Androgen Deprivation Therapy for Localised Prostate Cancer

Source
https://www.auajournals.org/doi/10.1097/JU.0000000000002619.02

INTRODUCTION AND OBJECTIVE:

Introduction: ADT plays a fundamental role in the treatment of localized prostate cancer. However, there is limited data regarding testosterone recovery in men who have received ADT for localized prostate cancer. Identification of T recovery profiles associated with ADT will facilitate personalization of ADT regimens and guide future treatment strategies to minimize the risk of T deficiency in patients following treatment for prostate cancer.

Objective: Temporary use of Androgen Deprivation Therapy (ADT) is a cornerstone in the treatment of localized prostate cancer. However, the ability for testosterone to recover after ADT is not well understood. The aim of this study was to investigate testosterone recovery in men with localized prostate cancer following ADT.

METHODS:

A global federated health research network (TriNetX) was used to identify men with a diagnosis of prostate cancer who underwent temporary use of ADT. Three cohorts were identified: Men who received LHRH antagonists, LHRH agonists, and men who received combined ADT (LHRH agonist and antiandrogens). Further stratification was based on treatment duration of 6 or 18 months to compare T recovery profiles 5 years after ADT cessation.

RESULTS:

A total of 17,884 men received LHRH agonists alone, 12,767 men received combined ADT, and 628 men received LHRH antagonist therapy alone. Eugondal mean baseline T level (>300 ng/dL) prior to starting ADT was an inclusion criterion for all men. Five years after ADT cessation, 36% of patients who received LHRH agonists recovered eugondal T levels, 26% recovered after LHRH antagonist therapy, and 36.8% recovered after combined ADT. Overall, more than half of men who received ADT failed to recover eugondal T levels even 5 years after treatment cessation.

CONCLUSIONS:

Five years after ADT cessation for localized prostate cancer, incomplete testosterone recovery persists in >50% of the men. Since, testosterone deficiency will lead to metabolically adverse changes in body composition, increased insulin resistance, impaired bone health, and poor quality of life, serum T levels need to be closely followed in men receiving ADT.

Testosterone recovery in patients after short term ADT

ADT modality	T recovery 5 year follow up	Mean testosterone at 5 years
LHRH Agonist	41.2%	296.265 ng/dL
LHRH Antagonist	63.6%	419.044 ng/dL
Combined ADT	43.6%	275.224 ng/dL

Testosterone recovery in patients after long term ADT

ADT modality	T recovery 5 year follow up	Mean testosterone at 5 years
LHRH Agonist	31.2%	271.209 ng/dL
LHRH Antagonist	10%	334.885 ng/dL
Combined ADT	30.3%	232.156 ng/dL

IMSK Scientists Identify New — and Very Common — Subtype of Prostate Cancer

Source
27 MAY 2022
https://www.mskcc.org/news/msk-scientists-identify-new-and-very-common-subtype-prostate

A previously unknown subtype of hormone-resistant prostate cancer accounts for about 30% of all cases, according to a new study from a team of scientists at Memorial Sloan Kettering Cancer Center (MSK) and Weill Cornell Medicine, published May 27, 2022, in the journal Science. The results could pave the way for targeted therapies for people with this subtype of prostate cancer.

Prior to this recent work, which was led by MSK physician-scientist and Human Oncology and Pathogenesis Program member Yu Chen, only two prostate cancer subtypes had been described: androgen dependent and neuroendocrine. Dr. Chen's team calls the newly characterized third type stem cell-like (SCL), because some of the genes that are turned on in the cells are reminiscent of those in stem cells.

To make their discovery, Dr. Chen and his team examined 40 different patient-derived models of prostate cancer obtained from people with cancer treated at MSK and Weill Cornell.

"We didn't know whether we were going to find additional subtypes," Dr. Chen says. "This is a field that's been studied for many years, by many investigators. So we were happy and surprised to find that there's this fairly large group of patients with tumors that haven't been characterized."

Innovative Technologies Enable New Insights

One reason the subtype may have alluded researchers is that there aren't enough good laboratory models for studying this type of cancer.

"Prostate cancer is uniquely difficult to propagate in the lab," Dr. Chen explains. "Whereas there are hundreds of cell lines of melanoma and lung cancer, there's only three or four prostate cancer cell lines that are useful."

To circumvent this problem, the team turned to a new technology called organoids. The organ-like structures are grown in the lab from pieces of a patient's tumor. They are a kind of "avatar" of patient's tumor and can be used to study its genetics and biochemistry.

In addition, the team made use of patient-derived xenografts — tumors removed from a patient and grown in a mouse — for a total of 40 different patient-derived models of prostate cancer.

With these patient-derived organoids in hand, they could then determine which genes are turned on or off in the cells. This information was used by the scientists to determine that a new subtype of prostate cancer exists.

Next, they looked to see if the SCL subtype was apparent in a biobank of 366 prostate cancer tumors. It was. In fact, it was the second most prevalent group, after the androgen-sensitive type.

Knowing the molecular drivers of this common subtype of prostate cancer opens the door to approaches that could target these drivers with drugs.

New Treatment Possibilities

"For the past 80 years, the backbone of treatment for prostate cancer has been hormone-deprivation therapy," Dr. Chen explains. "That's because essentially all prostate cancers when they are first diagnosed depend on testosterone signaling."

"Once patients become resistant to antigen deprivation," he continues, "it becomes a universally lethal disease."

This is where the new findings could help improve treatment options. The scientists found that there are experimental drugs currently being tested in humans that can block the growth of the SCL subtype in laboratory and animal models. They are currently working with several companies to establish a clinical trial of their drugs for people with this subtype of prostate cancer.

Key Takeaways

- Prostate cancer responds well to hormone-blocking therapy but often develops resistance.
- Scientists at MSK have identified a previously uncharacterized subtype of hormone-resistant prostate cancer.
- They used organoids and patient-derived xenografts to make their discoveries.
- Identifying the molecular drivers of this new subtype opens the door to better targeted therapy for this subtype.

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

Clinical Efficacy of Bipolar Androgen Therapy in Men with Metastatic Castration-Resistant Prostate Cancer and Combined Tumor-Suppressor Loss

Abstract

Bipolar androgen therapy (BAT) relies on oscillating levels of serum testosterone as a way to treat patients with metastatic castration-resistant prostate cancer (mCRPC). Aggressive-variant prostate cancers typically require combination chemotherapy and are frequently associated with loss-of-function mutations in tumor suppressor genes. Here we report clinical outcomes after BAT among patients with mCRPC harboring pathogenic alterations in at least two of three genes: *TP53, PTEN*, and *RB1*. In this setting, BAT induced a meaningful PSA50 response rate, progression-free survival and overall survival, particularly in patients without prior chemotherapy.

Patient summary

Bipolar androgen therapy, in which drugs are used to raise testosterone levels and then allow them to decrease again in a cycle, may be a safe and effective treatment for prostate cancer that is resistant to testosterone suppression and has mutations in tumor suppressor genes. A randomized study comparing this approach to chemotherapy is needed to confirm the findings.

Bipolar androgen therapy (BAT) is an emerging treatment strategy for patients with metastatic castration-resistant prostate cancer (mCRPC). During BAT, serum testosterone is cycled from supraphysiologic down to near-castrate levels every month. Multiple clinical trials have demonstrated the benefit of BAT as a single-agent strategy and its ability to resensitize patients to prior novel androgen receptor (AR)-targeted therapies.

Treatment resistance to AR-targeted therapies occurs through a variety of mechanisms, including lineage plasticity, a process by which the prostate cancer undergoes a series of molecular events resulting in less reliance on AR signaling. Loss-of-function mutations in tumor suppressor genes have been associated with lineage plasticity and the emergence of neuroendocrine prostate cancers or other AR-indifferent cancers. Thus, the presence of at least two mutations in *TP53, RB1*, and/or *PTEN* has been proposed as a prognostic biomarker associated with aggressive prostate cancer variants. These aggressive-variant prostate cancers are largely resistant to AR-targeted therapies, but may respond favorably to taxane and platinum doublet chemotherapy.

Even though prostate-specific antigen (PSA) production is directly stimulated by testosterone, we have shown that BAT can induce deep PSA responses in some patients with mCRPC harboring inactivating *TP53* or DNA-repair gene mutations. Given the efficacy of BAT in *TP53*-mutated mCRPC, we hypothesized that BAT may yield a clinical benefit in prostate cancers with an AR-indifferent phenotype, which would address an unmet medical need.

We identified 22 patients with aggressive-variant mCRPC, defined as inactivating mutations or genomic loss in at least two of three specific genes (*TP53*, *RB1*, and/or *PTEN*), identified via clinical-grade next-generation DNA sequencing or immunohistochemistry (IHC) of either a primary or metastatic tumor. These patients were treated with testosterone cypionate 400 mg intramuscularly every 28 d in one of three prospective clinical trials (NCT02090114, NCT03554317, and NCT02286921). All patients were also maintained on luteinizing hormone–releasing hormone agonist/antagonist therapy if not surgically castrated. Treatment status for mCRPC ranged from treatment-naïve to heavily pretreated patients with multiple novel AR-targeted therapies in addition to taxane chemotherapy. Here we report the clinical efficacy of BAT in terms of the PSA50 response rate (≥50% decline from baseline) and composite progression-free survival (PFS, defined as the first of either radiographic or clinical progression), and overall survival (OS). A full description of clinical and pathologic characteristics is provided in the supplement.

The PSA50 response rate in the cohort was 45.5% (*n* = 10/22; 95% confidence interval [CI] 24.4–67.8%; Fig. 1). An additional two patients experienced a decline in PSA from baseline that did not reach the PSA50 threshold (PSAany = 54.5%; *n* = 12/22). All patients who experienced PSA reductions on BAT had a pathogenic *TP53* mutation (while no PSA reductions were observed in patients with combined *PTEN*/*RB1* inactivation). No patients in this cohort had mutations in all three genes of interest. Interestingly, two patients who were previously resistant to enzalutamide were rechallenged following BAT. Both patients experienced a PSA50 response to enzalutamide retreatment. To better understand the duration of benefit, we estimated the median PFS on BAT, which was 4.8 mo (95% CI 2.8–8.5; Fig. 2A). Since this was a heterogeneous population with respect to prior therapies, we also

Source
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9168525/

(continued page 14

assessed the effect of prior chemotherapy on PFS. Patients who received prior taxane chemotherapy had a shorter median PFS in comparison to chemotherapy-naïve patients (8.4 vs 3.6 mo; log-rank $p = 0.04$; Fig. 2B). The median OS estimate for the whole cohort was 34 mo (95% CI 15–not reached; Fig. 2C). Similarly, patients without prior chemotherapy had a longer median OS in comparison to taxane-treated patients (38 vs 15.1 mo; log-rank $p = 0.04$; Fig. 2D).

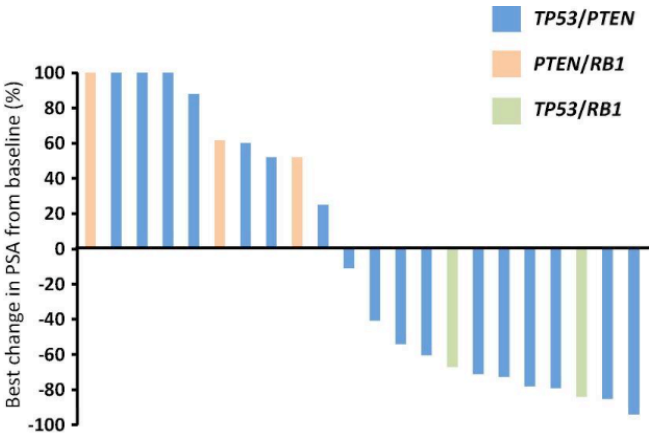


Fig. 1

Waterfall plot of prostate-specific antigen (PSA) response in patients with androgen receptor–indifferent metastatic castration-resistant prostate cancer treated with bipolar androgen therapy. The best change in PSA from baseline

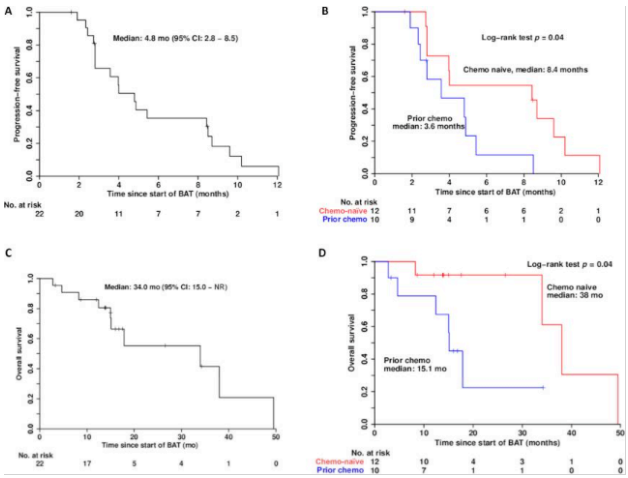


Fig. 2

Progression-free survival (PFS) and overall survival (OS) on bipolar androgen therapy. Kaplan-Meier estimates of (A) composite PFS in the entire cohort and (B) PFS stratified by prior chemotherapy exposure. PFS was longer for chemotherapy-naïve patients (red) than for patients treated with prior taxane-based chemotherapy (blue): 8.4 mo (95% CI 2.8–NR) versus 3.6 mo (95% CI 1.9–NR); log-rank $p = 0.04$. Kaplan-Meier estimates of (C) OS in the entire cohort and (D) OS stratified by prior chemotherapy exposure. OS was longer for chemotherapy-naïve patients (red) than for patients treated with prior taxane-based chemotherapy (blue): 38 mo (95% CI 34–NR) versus 15.1 mo (2.8–NR); log-rank $p = 0.04$. chemo = chemotherapy; CI = confidence interval; NR = not reached.

In a randomized trial, Corn et al compared carboplatin in combination with cabazitaxel versus cabazitaxel alone in men with mCRPC. A post hoc analysis revealed that patients with alterations in at least two of *TP53, PTEN*, and *RB1*, determined via circulating tumor DNA or IHC analysis, had an estimated PFS of 2.2 mo with cabazitaxel alone versus 6.0 mo with carboplatin-cabazitaxel ($p = 0.0003$). OS (17.4 vs 9.9 mo; $p = 0.002$) favored the carboplatin-cabazitaxel arm in this cohort with combined tumor-suppressor losses. In a separate study, an OS of ~14 mo was observed for patients with *TP53*−/*RB1*− mCRPC at the start of novel AR-targeted therapy. The median OS approached 3 yr following treatment with BAT in our analysis, suggesting potential long-term benefits in comparison to these prior studies. Similar to the study by Corn et al, our patient population was heavily pretreated, with chemotherapy-naïve patients experiencing the most favorable outcomes. Although chemotherapy remains the mainstay of treatment for this aggressive subtype of prostate cancer, grade ≥3 adverse events, including fatigue (20%) and neutropenia (16%), were observed with combination chemotherapy. In the largest randomized clinical trial using BAT, the majority of adverse events were of low grade (grade <2). Our data suggest that BAT may induce clinically meaningful responses in aggressive-variant prostate cancers with a more favorable safety profile in comparison to a taxane/platinum doublet. Although anecdotal, the observation that two patients achieved PSA responses to enzalutamide rechallenge suggests that BAT has potential for resensitization to novel AR-targeted therapies.

Several limitations of this analysis should be addressed. (1) The number of patients is small. The confidence intervals for the PFS and OS estimates are wide and broad conclusions are not prudent. (2) There was random sampling of patients. The data come from a small number of patients across different clinical trials for whom clinical-grade molecular analyses were available. It is likely that other patients in these studies may have had the requisite molecular profile. This approach may have led to unintended bias in the study. (3) We did not identify any patients with neuroendocrine features on pathology. Pathology review was conducted on archived tissue and may not have detected neuroendocrine transformation in late-stage disease. (4) The study includes patient with a molecular profile detected via IHC or next-generation sequencing. It is possible that clinical benefit may have been differentially affected by the technique used for eligibility. (5) The study is not randomized. Corn et al. reported significant PFS and OS differences across different treatment paradigms using the carboplatin-cabazitaxel combination. Although our PSA response rates and PFS and OS estimates suggest preliminary efficacy, a randomized study with BAT is necessary to derive further conclusions.

Our findings suggest that BAT may have a role in treatment of an aggressive molecular phenotype of mCRPC characterized by combined tumor-suppressor losses. Given the tolerability of BAT in comparison to combination chemotherapy, more patients may be eligible for this unique treatment paradigm. Further study of BAT in this clinical setting is warranted.

Three Steps Further

Can intense hormonal suppression before surgery keep potentially aggressive prostate cancer from coming back?

Source
18 April 2022
https://www.pcf.org/c/three-steps-further/

What does it take to make sure localized prostate cancer never comes back? A new Phase II clinical trial aims to find out, and it is noteworthy in two ways: One, as its principal investigator, medical oncologist Fatima Karzai, M.D., puts it, **"We're being aggressive in treating high-risk disease."** And two, with the help of serial DCFPyL PSMA PET/CT imaging, which can identify prostate cancer in the prostate and other places in the body, the investigators can **observe the effects of anti-cancer medications – three powerful forms of hormonal therapy, in addition to surgery – on the cancer in real time.**

Three steps further than surgery: **This is a no-holds-barred, all-out attack on localized prostate cancer that has the potential to be aggressive and to recur after treatment.**

Karzai and colleagues are taking high-risk cancer – even though it's localized – very seriously, and rightly so: more than half of patients diagnosed with high-risk prostate cancer have a recurrence, sometimes years later, and more than 20 percent of men with high-risk prostate cancer die of their disease within 15 years. Note: these numbers have not yet caught up with the use of PSMA-PET scanning, which can detect tiny bits of cancer that can't be seen on conventional scans – and if cancer can be seen, doctors believe it can be treated.

What will a short-course of triple hormone therapy do? The researchers hope this systemic (throughout the body) treatment before surgery, coupled with serial imaging scans with PSMA PET/CT, prostate MRI, and repeat prostate biopsy on treatment, will show how imaging can be incorporated into high-risk disease. They also hope that by striking any stray cancer cells while they are most vulnerable, they can reduce the risk of full-blown metastases later on. A similar trial showed promising results after more than three years of follow-up.

The trial is still recruiting patients. So far, Karzai says, the average participants are in their mid-60s, with Gleason scores of 8 or 9, but the trial is open to men of any age with high-risk or even intermediate-risk prostate cancer that has not spread to other parts of the body (up to clinical stage T4) who are planning to be treated with prostatectomy.

For six months before surgery, men in the trial undergo "intense androgen deprivation therapy," says Karzai, a PCF Young Investigator who serves as Clinical Director of the Genitourinary Malignancies Branch of the Center for Cancer Research at the National Cancer Institute. This includes: **Goserelin (Zoladex),** which shuts down testosterone, and two drugs that target the androgen receptor and testosterone production: **abiraterone** (given with prednisone), and **enzalutamide**. "We're really driving down the male hormones as low as we can." The cancer is imaged with prostate MRI and serial PSMA-PET scans – before treatment, two months after starting treatment, and again before surgery – and patients undergo an additional prostate biopsy two months into the study.

Note: The loss of testosterone is temporary! As the patients are recovering from surgery, testosterone starts to come back. "It takes six months to a year from the second shot (given midway through the study), and **all the patients will recover their testosterone. Their libido will be affected temporarily, but as they start to recover testosterone, libido will return."**

During the six months, "we see the PSA levels become very low or undetectable," says Karzai. She and colleagues are also looking for corresponding changes in the tissue (in biopsy samples and in the prostate tissue itself after surgery), studying genetic mutations in the cancer and – for the first time – observing how the effects of intense hormonal therapy are manifested on PSMA-PET imaging. "We are seeing some patients who are exquisitely sensitive to androgen deprivation, and some who aren't; the difference really has to do with the unique biology of their cancer." On PSMA-PET, "usually what we see is that the area that lights up initially becomes less intense. In some patients with disease that's pretty aggressive, it won't go away completely in six months," but it does diminish. "We're not looking to cure them completely with this treatment, but to get them to the surgery," and to **maximize their chances of cure.**

One goal of the study is to learn how to incorporate PSMA-PET scans into the treatment of men diagnosed with high-risk prostate cancer. "Right now, many men don't routinely get PSMA-PET scans. We're also trying to see, up front, if you do more androgen suppression, what does that mean for the overall outcome?" **Is it possible to hit aggressive cancer hard enough at the localized state to keep it from coming back?** "We'll follow these patients for a long time."

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.

Treatment Intensification in Metastatic Hormone-Sensitive PCa

Source
10 May 2022
Zachary Klassen, MD MSC
https://www.pcf.org/c/treatment-intensification-in-metastatic-hormone-sensitive-prostate-cancer/?utm_source=NewsPulse&utm_medium=email&utm_campaign=MAY22NP

If you've been diagnosed with **metastatic hormone-sensitive prostate cancer (mHSPC),** you have many treatment options. Doctors and researchers at the Advanced Prostate Cancer Consensus Conference held in April 2022 in Lugano, Switzerland reviewed the latest clinical trial information and highlighted key points for clinicians.

Metastatic prostate cancer means that prostate cancer has spread outside of the prostate gland to other organs, most commonly the lymph nodes and bones, but also not uncommonly to the liver and lungs. mHSPC may manifest in two main ways: the cancer is metastatic at the time of diagnosis ("de novo"), or it has recurred after treatment ("metachronous") for localized disease (typically after radiation or surgical removal of the prostate gland). Patients with mHSPC are started on androgen deprivation therapy (ADT or "hormone therapy"), and see their PSA drop as the tumor shrinks.

While historically testosterone was reduced (to very low or "castrate" levels) by removing both testicles (termed bilateral simple orchiectomy), today, most men who need ADT receive medications that accomplish this medically, rather than undergoing surgery. There is a complex signaling pathway between parts of the brain, the pituitary gland, and the testicles that leads to testosterone production. This pathway may be disrupted by medications that affect the signals between the brain and pituitary glands – groups of medications called GnRH (LHRH) agonists (i.e., leuprolide) or antagonists (i.e., degarelix).

Treatment Intensification: Doublet Therapy

Until recently, ADT alone was the mainstay of treatment. Starting in 2015, data from large clinical trials (the GETUG-AFU15, STAMPEDE and CHAARTED trials) demonstrated that adding chemotherapy (docetaxel) to ADT improved survival in men with mHSPC to nearly 4 years from their initial diagnosis. Shortly thereafter, other trials showed that adding other oral medications such as abiraterone (the STAMPEDE and LATITUDE trials), apalutamide (the TITAN trial), and enzalutamide (the ENZAMET and ARCHES trials) could also significantly prolong life beyond just ADT alone. Furthermore, additional data from the STAMPEDE trial showed that adding radiation therapy to the prostate gland, even in patients with metastatic disease, prolonged survival.

Currently, doublet treatment intensification is standard of care per all of the prostate cancer guidelines. However, in practice, data from the Veterans Affairs health care system, as well as from Medicare claims data suggests that ~45%-80% of men do not receive treatment intensification beyond ADT alone. The reason(s) for this lack of intensification are unknown and is an active area of research to provide further clarity.

Treatment Intensification: Triplet Therapy

Two recent clinical trials have provided further evidence that treatment intensification with "triplet therapy" may improve survival even beyond standard of care doublet therapy. The PEACE-1 trial tested mHSPC patients treated with the "triplet" of ADT + abiraterone + docetaxel and found that they were 25% less likely to die versus patients treated with docetaxel + ADT (no abiraterone). The addition of abiraterone also prolonged the time to cancer progression by 2.5 years. The second trial was the ARASENS trial which tested the "triplet" of ADT + docetaxel + darolutamide (another oral second-generation anti-androgen) versus ADT + docetaxel ("doublet" therapy) in mHSPC patients. This trial found that patients treated with triplet intensification had a 32% decreased risk of death compared to doublet therapy patients. These patients also had improved time to castration resistance (when the PSA increases and disease worsens, despite hormone therapy), time to pain progression, time to symptomatic skeletal related events (i.e., bone fractures, needing radiation to the bones, etc.), and time to next cancer therapy. Importantly, for both trials, these improved outcomes of triplet therapy intensification were associated with only a modest increase in adverse events.

The decision on which therapies to combine can be complex and is influenced by several factors, including:

- Whether the patient is newly diagnosed with metastatic prostate cancer or has prostate cancer recurrence
- The amount of metastatic disease, classified as low vs. high volume
- Other patient health factors

Ultimately, if you are newly diagnosed with metastatic prostate cancer or have a new recurrence of your cancer that has spread (mHSPC), talk to your doctor about your treatment options. Ask if additional therapies beyond ADT alone may be right for you.

On the Radar
Interesting research

Researchers Identify Key Factors Impacting Adaptive Therapy

Most cancer treatments are based on using the maximum tolerated dose of a drug to kill as many cancer cells as possible. While this approach has led to patients achieving good responses to therapy, most patients develop drug resistance and disease recurrence. Researchers in the Center of Excellence for Evolutionary Therapy at Moffitt Cancer Center have been investigating an alternative treatment approach called adaptive therapy that focuses on maintaining disease control instead of complete tumor cell elimination. In a new study published in *Communications Medicine*, the researchers used mathematical modeling to reveal that the spatial organization of a tumor is an important factor that governs how cells compete with one another and the effectiveness of adaptive therapy.

Source
9 May 2022-06-13
Science News
H. Lee Moffitt Cancer Center & Research Institute
https://malecare.org

Mediterranean Style Dietary Pattern with High Intensity Interval Training in Men with Prostate Cancer Treated with Androgen Deprivation Therapy: A Pilot Randomised Control Trial

Background: Androgen deprivation therapy (ADT) in prostate cancer has been shown to deteriorate body composition (reduced lean mass and increased body and fat mass) and increase the risk of cardiovascular morbidity. The Mediterranean style dietary pattern (MED-diet) and high intensity interval training (HIIT) may synergistically alleviate these side effects and improve quality of life in men treated with ADT.

Methods: Twenty-three men (65.9 ± 7.8 years; body mass index: 29.6 ± 2.7 kg/m2; ADT duration: 33.8 ± 35.6 months) receiving ADT for ≥3 months were randomly assigned (1:1) to 20 weeks of usual care or the MED-diet (10 nutrition consults) with HIIT (4 × 4 min 85-95% heart rate peak, 3× week, starting at 12 weeks). Results: The MED-diet with HIIT significantly improved cardiorespiratory fitness (+4.9 mL·kg-1·min, p < 0.001), and body mass (-3.3 kg, p < 0.001) compared to the usual care group at 20 weeks. Clinically meaningful (≥3 points) improvements were seen in quality of life and cancer-related fatigue after 20 weeks.

Conclusions: The MED-diet with HIIT increased cardiorespiratory fitness and reduced body weight in men with prostate cancer treated with ADT. Larger trials determining whether the MED-diet with HIIT translates to cardiovascular benefits are warranted.

Source
16 May 2022
https://www.urotoday.com/recent-abstracts/urologic-oncology/prostate-cancer/137197-mediterranean-style-dietary-pattern-with-high-intensity-interval-training-in-men-with-prostate-cancer-treated-

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.

PCa Clinical Trials & Studies

For Further information on current and recruiting trials visit:

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

Phase 1 trial of TLX591 (WA)

This is a Phase 1 trial of TLX591, a monoclonal antibody HuX591 conjugated with a DOTA chelator and radiolabelled with 177Lu (177Lu-DOTA-TLX591). TLX591 is being developed as a PSMA-targeting antibody to be radiolabelled with a therapeutic radiosotope for the treatment of PSMA-expressing tumours, therefore this study has been designed to assess the safety and tolerability, pharmacokinetics, whole body biodistribution and radiation dosimetry of 177Lu-DOTA-TLX591.

Detailed Description:

This multi-center prospective Phase 1 study is designed to evaluate the safety, tolerability, biodistribution and dosimetry of 177Lu-DOTA-TLX591 administered to patients together with best SoC with metastatic castration-resistant prostate cancer (mCRPC) that expresses PSMA and has progressed despite prior treatment with a novel androgen axis drug (NAAD).

PSMA positivity will be defined by 68Ga-PSMA-11 or [18F]DCFPyL PET/CT where at least one site of metastatic disease has an intensity of tracer uptake significantly greater than normal liver (i.e., standardized uptake value [SUV]max at least 1.5 times SUV of normal liver.

Approximately 25-50 eligible patients will be enrolled and evaluated for safety and tolerability as well as undergo imaging to allow determination of the biodistribution and dosimetry 177Lu-DOTA-TLX591. All patients will receive two single intravenous (IV) infusions of 76 mCi each (equivalent to 45 mCi/m2 in a standard 1.7m2 individual) of 177Lu-DOTA-TLX591, given notionally 14 days apart, plus best SoC.

Twenty-five patients will also be enrolled in a biodistribution and dosimetry sub-study, involving the collection of quantitative single-photon emission computerized tomography (SPECT) or single-photon emission computerized tomography/computerized tomography (SPECT/CT) data in a subset of patients.

SPECT/CT scans will be performed after administration of the drug, at the following timepoints: i) 4h ± 5 min, ii) 24 ± 4h, iii) 96 ± 4h, iv) Day 7± 4h and v) Day 13± 4h. These SPECT/CT scans will be conducted to further support safety monitoring and to understand exposure of tumour and healthy tissue to 177Lu-DOTA-TLX591.

Imaging data from the first five patients enrolled in the biodistribution study will be analysed and, if appropriate, the number of imaging timepoints will be reduced to three for subsequent patients. Qualitative comparison of 68Ga-PSMA-PET/CT images and 177Lu-DOTA-TLX591 images will also be undertaken for patients undergoing the biodistribution study to ensure equivalence of radiopharmaceutical localization to tumour sites and equivalent off-target localization.

Blood samples for pharmacokinetic analysis will be performed at 15 min ± 5 min before administration of the drug, and at the following timepoints after dosing: i) end of infusion ± 5min, ii) 60 min ± 5 min, ii) 4h ± 5 min, iii) 24 ± 4h, v) 48 ± 4h, vi) 96 ± 4h, vii) Day 7± 4h and viii) Day 13± 4h.

Collection of information on prior and concomitant medications and adverse events (AEs), as well as the review of temporary contra-indications and conditions for withdrawal, will occur at every patient interaction.

Contact
Nat Lenzo MD
62 02 8236 3300

Locations
Hollywood Private Hospital
Genesis Care, Medical Centre

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.

About the Patch Trial (UK)

Prostate cancer needs the male hormone testosterone to grow. Hormone therapy is usually used to lower the level of testosterone, which helps to control the growth of the cancer. LHRH agonists (luteinizing hormone-releasing hormone agonists) are the most common type of injection or implant. The pituitary gland in the brain makes a hormone called luteinising hormone (LH), and LHRH agonists work by interfering with this action and stop the testicles making testosterone. There are several available that all work in the same way, including:

goserelin (Zoladex®)

leuprorelin acetate (Prostap®)

triptorelin (Decapeptyl®)

Unfortunately, standard hormone treatment with injections or implants can cause a range of long-term side effects. They may cause bones to thin which might lead to them becoming fragile (osteoporosis) and more likely to break. They might also increase the chance of developing diabetes or heart disease.

An alternative way of giving hormone therapy is through the use of hormone patches. These patches, referred to as transdermal oestradiol, allow oestradiol (a type of hormone) to pass through the skin. Giving hormone therapy this way might be able to treat the cancer in a similar way as standard hormone therapy without causing some of these side effects.

The PATCH trial has already shown transdermal oestradiol can suppress testosterone as effectively as standard hormone therapy, while having a number of other potential benefits:

- It does not cause the bone to thin.
- Men treated with transdermal oestradiol generally reported better quality-of-life than those on hormone injections.
- Cholesterol and glucose levels increased in men on hormone injections but decreased in those on transdermal oestradiol.

The PATCH trial has been extended in order to look at whether transdermal oestradiol patches can control prostate cancer as well as standard hormone injections.

The Patch Trial is taking place in the UK

http://patch.mrcctu.ucl.ac.uk/about/

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

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PHCSG Committee

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

March 2022
- Will PSA Testing be Replaced? Novel Screening Approches
- How Bipolar Androgen Therapy Works
- Bipolar Androgen Therapy and the Immune System
- The Role of SBRT
- On Metabolic Syndome, Statin Drugs & PCa Progression
- Yoga Improves QoL in Men Newly Diagnoses with PCa
- The Trials & Tribulations of Managing Men with mHSPCa
- How Enzalutamide Impacts QoL in Metastatic Cancer
- Low-meat and Meat-free Diets associated with lower overall cancer risk
- Transdermal Oestradiol for Androgen Suppression
- PCa Test Cuts False Positives
- Trial to Evaluate Men Starting ADT
- Who goes on ADT with RT to Treat Intermediate Risk PCa
- Darolutamide & Surival in mHSPC
- Effect of High Dose Vitamin D on Bone Density & Strength
- How Important is Bone Mineral Density for Men on ADT
- Bipolar Androgen Therapy – A Patient's Guide
- The D-Health Trial – Effect of Vitamin D on Mortality
- Does Estradiol Improve Cognitive Function for men on ADT?
- SBRT or Convemntional RT for Macroscopic Prostate Bed Recurrence
- To continue ADT – or Not?
- Biochemical Definition of Cure with Brachytherapy of PCa
- New Radiotracer increases Accuracy
- Less Meat, Less PCa?
- PCa's Sweet Tooth
- RP vs RT in Ductal Carcinoma of Prostate
- Survival after RP vs RT in Nodai Positive PCa

April 2022
- Apalutamide no on the PBS
- The Benefit of Exercise
- Ex Med and Hospital exercise programs for patients wit PCa
- Gut Environment changes due to ADT
- Effect of Statins on Advanced PCa or abiraterone/enzalutamide
- Researchers identify five types of bacteria in men with aggressive PCa
- Curative treatments didn't work – what should I do?
- Molecular Mechanisms of Coffee on PCa
- ADT use & duration with RT for Localised PCa
- Association of Muscle Mass after RP
- Is ADT Necessary when you take Abiraterone?
- Obesity Linked to Improved Survival in Advanced PCa
- A Novel Oral Cytoskeleton Disruptor – experimental drug Sabizabulin
- Survival Benefit to Debulking with radiation
- QoL in mHSPC men taking Enzlautamide
- Cleveland Clinic Study Links Microbiome to Aggressive PCa
- Portable Method for PSA Screening
- Clinical Trials

May 2022
- Apalutamide no on the PBS
- The Benefit of Exercise
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- QoL in mHSPC men taking Enzlautamide
- Cleveland Clinic Study Links Microbiome to Aggressive PCa
- Portable Method for PSA Screening

June 2022
- My Cancer Care Record
- Common Blood Test Results Explained
- Don't Allow Statistics to Dictate Your Treatment
- Getting Second Doctor's Opinion
- Primary Care Use of FRAX / Quality of Life in in the Stampede Trial
- Penile Traction Therapy
- SPPORT Trial
- Persistent Testosterone Suppression after Cessation of ADT for localised PCa
- MSK SScientists Identify New Subtype of PCa
- Clinical Efficacy of Bipolar Androgen Therapy with MCRPCa
- Three steps Further
- Treatment Intensification in mHSPCa
- On The Radar /
- Mediterranean Style Dietary with High Interval Training
- Clinical Trials

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