Manitoba Prostate Cancer SUPPORT GROUP

Newsletter

Vol. 366 1,300 copies printed/e-mailed **August 2022**

Medical Advisors

Paul Daeninck M.D.
Medical Oncologist

Darrel Drachenberg
M.D. Urologist

Arbind Dubey M.D.
Radiation Oncologist

Piotr Czaykowski M.D.
Medical Oncologist

Thanks!

Next Meeting

Date: Wednesday, August 17, 2022

Speaker: Dr. Piotr Czaykowski
Medical Oncologist, CancerCare MB
Assistant Professor, Department of Surgery,
University of Manitoba

Topic: "*The times they are a-changing" ….*
and so is Drug therapy in prostate cancer

Location: The First Unitarian Universalist Church of Winnipeg, 603 Wellington Crescent, Winnipeg

Time: 7-9 pm
(First hour for general discussion; second hour for expert guest speaker)

Free Admission Everyone Welcome Plenty of free parking

Thought of The Day

"Look for something positive in each day, even if some days you have to look a little harder."

- Unknown

A Prostate Cancer Breakthrough Could Speed up Research by 10 Years

"This has never been seen before."

Prostate cancer growth is driven by male sex hormones called androgens. And so, lowering levels of these hormones can help slow the growth of cancer.

Hormone therapy has been successful in keeping metastatic, or advanced prostate cancer, under control. Patients with metastatic prostate cancer often receive treatment with anti-hormonal therapy, which inhibits the signal sent out by testosterone that stimulates tumor growth.

But eventually, the tumor cells could become resistant to it. An international team of researchers led by the Netherlands Cancer Institute has now unveiled an "unexpected potential" solution, not designed to fight cancer but to target proteins that regulate a cell's circadian rhythm.

(Continued on page 2)

SHARING IS CARING

The Manitoba Prostate Cancer Support Group offers support to prostate cancer patients but does not recommend any particular treatment modalities, medications or physicians ; such decisions should be made in consultation with your doctor.

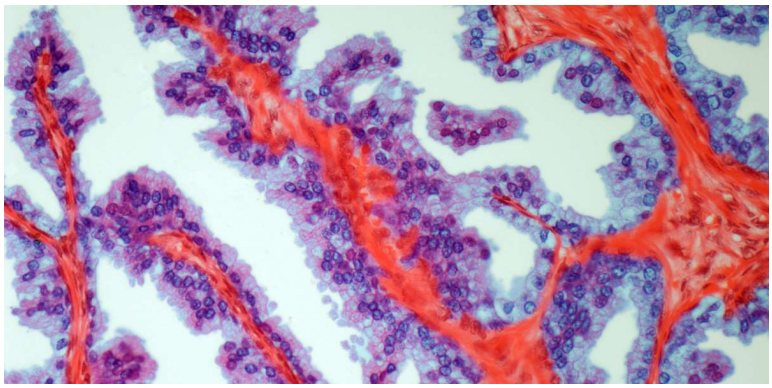
MPCSG – active since 1992.

(Continued from page 1)

The discovery has been published Monday in Cancer Discovery.

Proteins that dampen the effects of anti-hormonal therapy

While anti-hormonal therapy can keep prostate cancer under control, cancer manages to progress despite ongoing treatment, as the tumor cells have become resistant. This means that the greatest challenge in treating metastatic prostate cancer isn't to find drugs that inhibit tumor growth itself but to find drugs that can prevent resistance to hormonal therapy.



Using tissue from patients with prostate cancer who had been treated with testosterone-inhibiting drugs, scientists from the Netherlands Cancer Institute and Oncode Institute made a surprising discovery.

They discovered that an unexpected class of proteins, namely proteins that normally regulate the circadian clock, dampens the effects of anti-hormonal therapy. "Prostate cancer cells no longer have a circadian rhythm," said Wilbert Zwart, one of the research leaders, in a statement. "But these 'circadian clock' proteins acquire an entirely new function in the tumor cells upon hormonal therapy: they keep these cancer cells alive, despite treatment. This has never been seen before."

The study was based on tissue from 56 patients with high-risk prostate cancer, who had received three months of anti-hormonal therapy before their surgery. After which their tissue was examined at the DNA level. "We noticed that the genes keeping the tumor cells alive despite the treatment, were suddenly controlled by a protein that normally regulates the circadian clock," said researcher Simon Linder, who will receive his Ph.D. for his research in this study.

Now that they've figured out the tumor's escape route, the researchers will next work together with Oncode towards the development of novel strategies to block this process, ultimately increasing the efficacy of anti-hormonal therapy against prostate cancer even further.

'Has our full attention'

"Our discovery has shown us that we will need to start thinking outside the box when it comes to new drugs to treat prostate cancer and test medicines that affect the circadian clock proteins to increase sensitivity to hormonal therapy in prostate cancer," said Zwart. "Fortunately, there are already several therapies that affect circadian proteins, and those can be combined with anti-hormonal therapies. This lead, which allows for a form of drug repurposing, could save a decade of research."

This surprising discovery also creates new opportunities, because inhibition of this circadian protein was found to further increase sensitivity to anti-hormonal therapy in prostate tumor cells in the lab as well as in mice.

The results of this study might raise questions if disturbances to the body's circadian clock could increase the risk of therapy insensitivity in prostate cancer. "There is no evidence to support this," said medical oncologist André Bergman. "The circadian rhythm in prostate tumor cells is no longer functional, and the proteins have taken on an entirely new role. This new escape route of the tumor cell has our full attention now, and follow-up research will show whether inhibition of this process can improve prostate cancer treatment.," he adds.

Abstract:

In prostate cancer, androgen receptor (AR)-targeting agents are very effective in various disease stages. However, therapy resistance inevitably occurs and little is known about how tumor cells adapt to bypass AR suppression. Here, we performed integrative multi-omics analyses on tissues isolated before and after 3 months of AR-targeting enzalutamide monotherapy from high-risk prostate cancer patients enrolled in a neoadjuvant clinical trial. Transcriptomic analyses demonstrated that AR inhibition drove tumors towards a neuroendocrine-like disease state. Additionally, epigenomic profiling revealed massive enzalutamide-induced reprogramming of pioneer factor FOXA1 – from inactive chromatin sites towards active cis-regulatory elements that dictate pro-survival signals. Notably, treatment-induced FOXA1 sites were enriched for circadian clock component ARNTL. Post-treatment ARNTL levels associated with poor outcome, and ARNTL knockout strongly decreased prostate cancer cell growth. Our data highlight a remarkable cistromic plasticity of FOXA1 following AR-targeted therapy, and revealed an acquired dependency on circadian regulator ARNTL, a novel candidate therapeutic target.

By Deena Theresa Jun 27, 2022

Source: https://interestingengineering.com/prostate-cancer-breakthrough-speed-up-research

• • •

87% Survival – New Combined Therapy Greatly Improves Prostate Cancer Survival

A Cedars-Sinai cancer study indicates improved survival following a combination of hormone therapy and pelvic lymph node treatment
A combination of androgen deprivation therapy—a common hormone injection—and pelvic lymph node radiotherapy prevented prostate cancer from therapy in nearly 90% of clinical trial participants for five years, according to a ground-breaking study from Cedars-Sinai Cancer. The results were recently published in the peer-reviewed journal The Lancet.

The research also demonstrates that individuals with prostate cancer who did not get pelvic lymph node radiotherapy or androgen restriction treatment had a five-year survival rate of 70%.

"We can now confirm that pelvic lymph node treatment used together with androgen deprivation therapy, or even used as a stand-alone treatment option, greatly improves outcomes in patients with postoperative prostate cancer," said Howard Sandler, MD, chair of the Department of Radiation Oncology at Cedars-Sinai Cancer and senior author of the study. "These findings are an encouraging step forward, both for the medical community and for the patients and their loved ones seeking curative treatment options."

Between March 31, 2008, and March 30, 2015, 1,716 participants were recruited in the global Phase III clinical trial that formed the basis of The Lancet research. Three groups of participants were created.

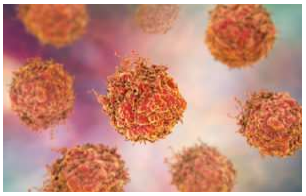
Salvage prostate bed radiotherapy was administered to Group 1; this kind of radiation is often directed towards the prostate's former location before it was surgically removed. The median five-year survival rate for these individuals was 71%.

The second group underwent androgen deprivation therapy in addition to the conventional radiation therapy. They had an 81^ median five-year survival rate.

The third group received salvage prostate bed radiotherapy, androgen deprivation therapy, and pelvic lymph node radiation. These patients had a five-year freedom from progression of just over 87%.

"The combined treatment approach proved to be the most beneficial approach," said Sandler, also the Ronald H. Bloom Family Chair in Cancer Therapeutics and professor of Radiation Oncology at Cedars-Sinai.

Prostate cancer is the most common non-skin cancer in the U.S., affecting 1 in every 6 to 7 men. While there are rarely early warning signs of the disease, there is a robust screening test that can catch the disease in its earliest stages. Diagnosis usually accompanies an elevated level of PSA, an acronym for prostate-specific antigen.



Many men diagnosed with prostate cancer will undergo a prostatectomy—the surgical removal of the prostate. After surgery, a man's PSA level should be near zero. However, some men start to see their PSA levels rise several years after surgery. This is typically an indication that radiation therapy is needed.

Sandler says men with postoperative prostate cancer can have excellent outcomes, especially if radiation is given early—when PSA levels are at their lowest—and in combination with proven therapies, as suggested in this new research.

"Improving and extending lives is at the heart of all we do at Cedars-Sinai Cancer," said Dan Theodorescu, MD, Ph.D., director of Cedars-Sinai Cancer, the PHASE ONE Foundation Distinguished Chair, and professor of Surgery and Pathology and Laboratory Medicine. "These pivotal clinical findings exemplify our mission while showcasing how ideas spur leading-edge research and treatment innovations."

This study was funded by grants U10CA180868 (NRG Oncology Operations), U10CA180822 (NRG Oncology Statistical and Data Management Center), UG1CA189867 (NCORP), and U24CA180803 (Imaging and Radiation Oncology Core).

Reference: "The addition of androgen deprivation therapy and pelvic lymph node treatment to prostate bed salvage radiotherapy (NRG Oncology/RTOG 0534 SPPORT): an international, multicentre, randomised phase 3 trial" by Professor Alan Pollack, MD, Professor Theodore G Karrison, Ph.D., Alexander G Balogh, MD, Professor Leonard G Gomella, MD, Professor Daniel A Low, Ph. D., Professor Deborah W Bruner, Ph.D., Jeffrey S Wefel, Ph.D., Professor Andre-Guy Martin, MD, Professor Jeff M Michalski, MD, Steve J Angyalfi, MD, Professor Himanshu Lukka, MBChB, Sergio L Faria, MD, Professor George B Rodrigues, MD, Marie-Claude Beauchemin, MD, R Jeffrey Lee, MD, Samantha A Seaward, MD, Professor Aaron M Allen, MD, Drew C Monitto, MD, Wendy Seiferheld, MS, Professor Oliver Sartor, MD, Prof Felix Feng, MD, Professor Howard M Sandler, MD, 14 May 2022, The Lancet.

DOI: 10.1016/S0140-6736(21)01790-6

By CEDARS-SINAI JULY 16, 2022

Source: https://scitechdaily.com/87-survival-new-combined-therapy-greatly-improves-prostate-cancer-survival/

• • •

Prognosis of Prostate Cancer Patients Improved

Summary:
Prostate cancer is a common form of cancer. Researchers have now discovered a faster and easier way to determine who has an aggressive form of cancer, and who has not.

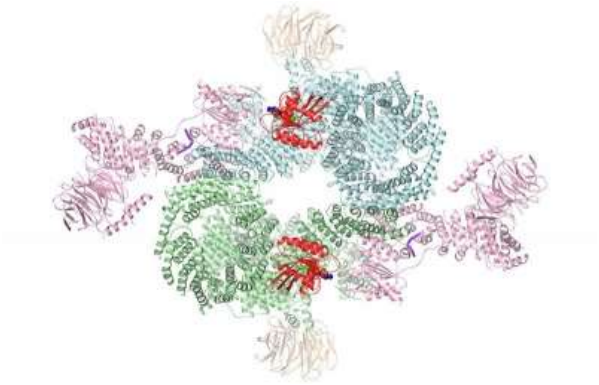
FULL STORY
Prostate cancer is the most common form of cancer in male in Sweden. Researchers at Umeå University in Sweden have now discovered a faster and easier way to determine who has an aggressive form of cancer, and who has not. "This may have great implications on precision medicine when treating prostate cancer, and on more cancer groups alike," says Maréne Landström, Professor of Pathology at Umeå University.

Over 10,000 men are annually diagnosed with prostate cancer in Sweden. Out of those, 2,300 of these lives cannot be saved, whereas many others can be cured or actually carry harmless tumours. The medical services are struggling with a balancing act between detecting as many cancers as possible in good time to start treatments early, and avoiding to diagnose men with cancer when the tumour is harmless as this causes unnecessary anxiety and negatively impacts the quality of life.

Consequently, intense work is carried out at research institutes to improve the methods distinguishing cancers that require treatment from cancers that should be left untouched, or preferably should not even be detected.

The research group of Professor Maréne Landström at the Department of Medical Biosciences is busy studying just that. In this project, they have also collaborated with a research group at Uppsala University.

The Umeå researchers have now discovered a new function in specific proteins in the transforming growth factor beta (TGF-β) signalling pathway, which is a significant path that affects how cancer cells grow and spread. This may have huge implications on the treatment of cancer since the discovery makes it possible to identify the men who are at risk of developing aggressive and life-threatening prostate cancer more easily, faster and early in the course of disease.



"We have found a new, previously unknown, function of the TGF-β type I receptor (TbRI), which is an important signalling protein in cancer cells. Previous studies have shown that TGF-β signalling is important in the development of several cancer forms," says Maréne Landström, Professor of Pathology at the Department of Medical Biosciences at Umeå University, continuing: "But with the use of this new discovery, we can put the men with prostate cancer whose prognosis is promising at ease, and those with high-risk prostate cancer can be offered treatment sooner. Our findings and the publication is significant for a large group of patients with prostate cancer, and there is reason to believe that further patient groups will benefit from this.

Jie Song, first author of the publication in EBioMedicine is tremendously pleased:

"Our discovery is a breakthrough in cancer research in the field of TGF-β signalling," she says whilst explaining the finding in more detail:

"We have discovered that AURKB, which is a kinase usually overexpressed in cancer cells, interacts with TβRI during cell division of both prostate cancer cells and neuroblastoma cells, which is a form of cancer common in children. We have also shown that TRAF6 - an enzyme which causes so-called K63-linked polyubiquitination -- ubiquitinates AURKB on two amino acids and thereby contributes to the kinase activity of AURKB when cancer cells proliferate. By in situ PLA technique, we show that K63-linked polyubiquitination of AURKB is present in lung adenocarcinoma, prostate cancer and clear cell renal cell carcinoma in patient tissue sections. Moreover, the AURKB-TβRI complex is correlated with the malignancy of prostate cancer. Our results suggest that the AURKB-TβRI complex may be a useful biomarker for early detection of advanced prostate cancer, which may be of huge clinical benefit in the development of precision drugs for treating prostate cancer," she says.

This finding is so unique that the researchers have applied for a patent through the pharmaceutical development company, MetaCurUm Biotech AB, as well as recruited further members to the research group. "We wish to really reach out with these news to the medical services as we think it can be of great use to patients with cancer, and to the medical services in general," says Maréne Landström.

"We will apply for funding from innovation agencies such as Vinnova and others to develop a method that the

(Continued on page 5)

(Continued from page 4)
medical services can use. We estimate that it will take us two to three years to develop a test based on our findings," says Maréne Landström.

What happens next?

"We are now investigating the molecular mechanisms for how these proteins can affect the activity of one another. This takes place in a research project in collaboration with Professor Magnus Wolf-Watz at the Department of Chemistry at Umeå University, and through studies of clinical samples from patients together with our clinical partners from the medical service.

~ ~ ~ ~

Materials provided by Umea University. Original written by Johanna Fredriksson. *Note: Content may be edited for style and length.*

Journal Reference:

Jie Song, Yang Zhou, Ihor Yakymovych, Alexej Schmidt, Chunyan Li, Carl-Henrik Heldin, Maréne Landström. The ubiquitin-ligase TRAF6 and TGFβ type I receptor form a complex with Aurora kinase B contributing to mitotic progression and cytokinesis in cancer cells. eBioMedicine, 2022; 104155 DOI: 10.1016/j.ebiom.2022.104155

July 18, 2022

Source: www.sciencedaily.com/releases/2022/07/220718094504.htm

• • •

Fast, Uncomplicated, and Specific: Diagnosis of Prostate Cancer From Blood Samples

Early detection of prostate cancer, one of the most common types of cancer in men, is often achieved with PSA tests. However, this blood test for prostate-specific antigens gives many false positive results, causing unnecessary biopsies and overtreatment. In the journal Angewandte Chemie International Edition, a Chinese research team now introduces a highly specific, non-invasive alternative to biopsy: the "thermophoretic AND gate operation" assay, abbreviated as Tango, quickly and reliably detects prostate cancer directly in blood samples.

The Tango assay is based on the analysis of circulating extracellular vesicles, which are membrane-bound "nanobubbles." These come from all cells of the body, circulate in the bloodstream, and contain numerous biomarkers typical of the cells in which they originated. Isolation and accumulation of the heterogeneous vesicles in complex samples requires complex and expensive pre-treatments. The new method developed by a team headed by Fei Tian, Bo Dai, and Jiashu Sun combines accumulation with a logical AND gate operation in a single step for the identification of the desired tumor vesicles.

The concentration process is based on thermophoresis, the movement of particles based on a temperature gradient. The sample is placed into a specially designed microchamber that is locally heated with an IR laser. The vesicles preferentially move toward the heated spot. Polyethylene glycol is also added to form a concentration gradient, which amplifies the effect. This results in a 2800-fold accumulation around the laser spot.

To identify the desired vesicles unequivocally and specifically, they must contain two proteins that occur in high concentrations in prostate tumors: prostate-specific antigen (PSMA) and epithelial cell-adhesion molecule (EpCAM). The team introduced two probes based on aptamers, which are short, single strands of DNA with a "programmed" 3D structure that specifically binds to a target molecule. In this case the two targets are PSMA and EpCAM. Each of the probes has fluorescence dye.

In order to only detect vesicles that contain both tumor markers, the team developed a logical AND operation. Both of the probes have a little molecular "anchor" that specifically binds to the end of a DNA connector. If both of the target proteins are found on a vesicle membrane, both types of probe are linked by the DNA connector and the two fluorescence dyes come close enough to each other for an energy transfer. The one dye absorbs light and transfers part of the energy to the other without radiation (Förster resonance energy transfer, FRET), the second dye then emits light. The intensity of this FRET fluorescence is a measure of the number of vesicles containing both tumor markers.

The Tango assay was able to identify patients with prostate cancer out of a group with inconclusive PSA results with 91 % accuracy in 15 minutes. It should also be possible to develop tango tests for other types of cancer, according to the team from the National Center for Nanoscience and Technology (Beijing) and Fudan University (Shanghai).

by Angewandte Chemie International Edition

JULY 20, 2022

Source: https://phys.org/news/2022-07-fast-uncomplicated-specific-diagnosis-prostate.html

• • •

Large Dose of Iron Could be Used to Kill Off Drug-Resistant Prostate Cancer, Scientists Believe

This could be especially prevalent since while there are a variety of treatments, and these usually work at first, some cancers develop resistance after 18-24 months and that dramatically limits the available options.

However, a team of scientists led by Dr. Chunhong Yan of the Medical College of Georgia are hoping to use iron to fight this stubborn disease in a process called ferroptosis, taken from the Latin word for Iron (ferro) and the word for cell death "optosis."

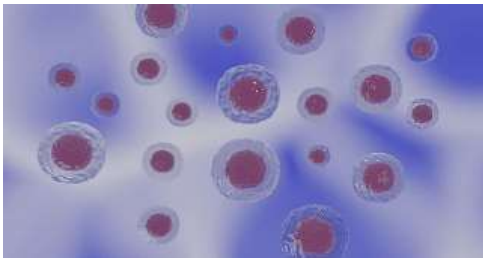
Iron is important for red blood cells carrying oxygen around the body but large amounts of it can be lethal to cells.

It produces a lot of toxic free radicals, or reactive oxygen species (ROS) which damage the fat component of the cellular membrane.

Lipids, or fats, are important for energy storage and for internal cell signaling. Free radicals cause them to lose their flexibility and efficiency until the cell dies, though exactly why is unclear.

Prostate cancer cells are unusually resistant to this destruction because their lipids are already changed to have the energy they need to grow and spread.

But Dr. Yan's team has found a gene called ATF3 that can lower the stress threshold of prostate cancer cells and make them more vulnerable to a new iron compound called JKE-1674 which induces ferroptosis.
"When the cell takes up iron, it goes through different processes, which generate a lot of ROS," said Dr. Yan. "What we are trying to do is take advantage of this side effect to treat prostate cancer."



Working on a $1.1 million idea development award from the U.S. Department of Defense, his team has also found that combining a chemotherapy drug with one of the body's natural mechanisms can also help kill prostate cancer cells.

The drug is called bortezomib and it helps activate the ATF3 gene while the compound JKE-1674 inhibits a process called glutathione peroxidase 4, which separates iron and free radicals and allows cells to repair.

Dr. Yan said clinical trials have shown that bortezomib is not very effective at treating prostate cancer on its own but that when combined with JKE-1674 it becomes a powerful weapon.
The next steps are to conduct experiments on mice and see whether advanced prostate cancer can be neutralized using ferroptosis.

The scientists have a genetically engineered mouse that produces more ATF3 and they want to see whether this makes prostate cancer cells more vulnerable to ferroptosis as well.

Dr. Yan wants to develop a therapy that could progress quickly from the lab to a clinical trial and help combat what is one of the most common form of cancers in men across the world.

By Andy Corbley Jul 21, 2022

Source: www.goodnewsnetwork.org/large-doses-of-iron-could-be-used-to-kill-off-drug-resistant-prostate-cancer-cells-scientists-believe/

• • •

Prostate Cancer Drug So Effective Trial Stopped

Michael Wells at his home in Oakville , Ca. on Friday June 1, 2012. A new drug for advanced prostate cancer has proven to be so effective that researchers stopped the clinical trial early to give patients a chance to receive the life-extending medication, a UCSF study showed. Michael Wells was diagnosed with prostate cancer in 2000 which had already spread to his hip bone, has been on the trial since 2009. The drug has shown success in the treatment of his cancer.

A new drug for advanced prostate cancer patients has proved so effective that researchers stopped the clinical trial early to give all patients a chance to receive the life-extending medication, according to a UCSF-led study released Saturday.

The hormone treatment, Johnson & Johnson's Zytiga, when added to a standard steroid therapy doubled the time it takes for the disease to progress in patients treated with the standard therapy alone, said the lead researcher, Dr. Charles Ryan, associate professor of clinical medicine at the UCSF Helen Diller Family Comprehensive Cancer Center.

The U.S. Food and Drug Administration last year approved Zytiga, also known as abiraterone, for use in men whose prostate cancer had spread to other parts of their body and had already been treated with chemotherapy. The FDA will have to approve it for patients who have not

(Continued on page 7)

(Continued from page 6)
had chemotherapy before it can be marketed for broader use.

This trial focused on patients whose cancer had metastasized, may have been treated with other hormone therapies but had not yet gone through chemotherapy. The interim results are to be presented Saturday at the American Society of Clinical Oncology's annual meeting in Chicago.

"If the FDA looks favorably upon the data ... it will really change the standard of care in advanced prostate cancer away from chemotherapy toward a well-tolerated, oral therapy," Ryan said from Chicago. "It opens up the possibility of this life-prolonging therapy being given to a larger population of patients."

A treatable disease
Prostate cancer, the second most common form of cancer in males after only lung cancer, is diagnosed in about 200,000 men in the United States each year. And while it is generally treatable, the disease kills nearly 30,000 men a year.

Because their disease is often slow-growing, about a third of patients diagnosed with prostate cancer won't be treated. Another third will undergo successful treatment, which could include surgery, various hormone therapies or chemotherapy.

Still, a third of patients will have recurrent or aggressive disease that may have been caught too late. Ryan said men tend to die when the cancer spreads outside the prostate, mostly to bone, and the patient becomes resistant to hormonal therapy. The cancer cells rely on testosterone to exist, so typically doctors treat patients with testosterone-blocking hormone therapy.

But patients become resistant when the cancer cells develop the ability to make their own hormone and learn to survive even in the face of the testosterone-blocking drugs, giving the disease the ability to progress, Ryan said.

Zytiga is the first FDA-approved drug that can go inside the cancer cell and block it from making its own testosterone.

The trial involved 1,088 men who were being treated by 151 cancer centers in 12 countries. Each was given a low dose of the steroid prednisone, which works to combat the cancer, but some received Zytiga while others were given a placebo.

All participants receiving the placebo drug were allowed in March to start taking Zytiga. Not only did they notice a slowdown in the progression of the disease, but patients also reported reduced pain and went longer before having to resort to chemotherapy.

If the FDA extends Zytiga's approval to include patients who have not yet gone through chemotherapy, more health insurers will cover the drug. The final results of the trial are expected next year.

Hopeful drug for many
Dr. Mark Scholz, executive director of the Prostate Cancer Research Institute in Los Angeles, said the drug may not work for all patients, but he's seen remarkable results in many.

"When you treat cancer patients you sometimes run out of options," he said. "We've had some patients like that since Zytiga was released that looked like the game was up, and they've been able to regain their lives."

One trial participant, Michael Wells, 65, was diagnosed in 2000 with prostate cancer that had spread to his hip bone. He was treated with various hormone therapies, but the disease kept recurring.

During the trial, the Oakville resident didn't know whether he was taking the real drug or the placebo, but he guessed about a year into it that he was on Zytiga. He was tired, his blood pressure went up - a potential side effect of the drug - and his cancer stayed in check.

Wells guessed right and continues to take the drug. He was pleased but not surprised by the trial results because his disease has remained under control.

Rodolfo Chavez, 83, who was diagnosed with prostate cancer in 1997 and learned it had spread in 2006, calls Zytiga his miracle drug. Chavez, who has had chemotherapy, started taking it last year after it was approved.

"After that first bottle, my pain went away and I just felt like my life was turning around," said the former longshoreman from San Pedro (Los Angeles County). "I'm still taking them. I'm on my 10th bottle and supposed to get another bottle today."

Victoria Colliver June 2, 2012

Victoria Colliver is a San Francisco Chronicle staff writer.

Source: www.sfgate.com/health/article/Prostate-cancer-drug-so-effective-trial-stopped-3603973.php

• • •

Return undeliverable Canadian addresses to
Manitoba Prostate Cancer Support Group
315-971 Corydon Ave.,
Winnipeg R3M 3S7

Publications Agreement
40037332

MANITOBA PROSTATE CANCER SUPPORT GROUP TAX DEDUCTIBLE DONATION

NAME:_____

ADDRESS:_____ POSTAL CODE_____

THIS GIFT IS IN MEMORY/HONOUR OF_____ PLEASE SEND NOTIFICATION TO:_____

NAME:_____

ADDRESS:_____ POSTAL CODE_____

***Make payment to**: Manitoba Prostate Cancer Support Group;*
Box 315 – 971 Corydon Ave., Winnipeg, Manitoba, R3M 3S7
**A tax deductible receipt will be issued. Charity number: 88907 1882 RR0001*

***Credit Card** donations can be made by going to our website at: www.manpros.org and clicking on the donate tab.*
Canada Helps will issue a tax receipt. ***Amount:*** *$25 $50 $75 $100 Other_____*

Thank-you to all our sponsors

Manitoba Motorcycle Ride For Dad

MCSC Manitoba Community Services Council Inc.

Gold Wing Road Riders Association
Manitoba District · Region K
http://mb-a.regionk.ca/

The Winnipeg Foundation

CancerCare Manitoba Foundation
All funds raised stay in Manitoba.

Email - manpros@mts.net ALL MEMBER INFORMATION IS KEPT CONFIDENTIAL
Answering Machine - (204) 989-3433 ***Help us lower our costs :***
Receive this newsletter by email ~ Please notify us and we'll make the changes. ***Thank-you***

FUTURE MEETINGS 2022

22-Sep This is our highlight event of the year, the September Prostate Cancer Awareness Evening

Dr. Jeff Saranchuk will address multiple topics including an update of progress on the Urologic Health Center at HSC in Winnipeg; impact of the covid pandemic on prostate cancer diagnosis and treatment in Manitoba; as well as take questions from the audience

This meeting will be held at the Caboto Centre, 1055 Wilkes Avenue, Winnipeg

Please note that this meeting is on a **Thursday** instead of our usual Wednesday. Don't miss it

19-Oct (To be confirmed) This meeting will focus on self-help action that prostate cancer patients can take to complement the specialist treatment provided by medical professionals. Such actions can have a big impact on quality of life for cancer survivors

16-Nov Xmas party. Details to follow

21-Dec No meeting this month

MPCSG BOARD

Betty O'Grodnik – Secretary	(204) 661-8549
Jos Borsa - Chair	(204) 219-7726
Don Murray - Vice Chair	(204) 487-0822
Liz Feschuk - Special Projects	(204) 654-3898
Ernie Schade – Meeting Convener	(204) 489-1648
Pat Feschuk – Special Events	(204) 654-3898
Wally Jackson - Member-at-large	(204) 668-1222
Deloris Ankrom - Member-at-large	(204) 795-4361
Cam Thompson - Member-at-large	(204) 941-0419

Volunteers On Committees

Irek Iskat — membership

For general information please contact Jos Borsa at number listed above

This newsletter is a
Bottom Line Computer Services
publication
Bottom Line Computer Services is not responsible for content
www.misterpete.com