Prostate Examiner Summer Newsletter

Visit us at www.prostatecancersupporttbay.org

Prostate Cancer Support

Looking for Support?

Men available to talk to you

Harold Alanen	807 252-9067
Mike Aldrich	807 630-3744
Marc Breton (en francais)	807 628-9944
Bill Everitt	807 767-5768
Bill Horde	807 767-1490
Bill Komar	807 627-2424
Ed Long	807 628-6915
Milton Marion	807 475-0760
Keith Moore	807 632-6055
Mark Sypus	807 768-5009

Women available to talk to you

Carmen Marion	807 475-0760
Susan Aldrich	807 628-7150
Lise Pollard (en français)	807 623-3102

Northwestern Ontario Region

Atikokan
Ron Speck 807-597-2219

Hearst
Marcel Girouard 705-362-8154 (en français)

Terrace Bay/ Schreiber
Mike Regis 807 825 9696

Geraldton
Ron Adams 807 854 1476

Summer Beef on a Bun Event

Another happy Bordercats 50/50 winner

2026 Walk for Dad Event

Email us at info@prostatecancersupporttbay.org

New form of hereditary prostate cancer can cause aggressive disease at a young age

University of British Columbia July 9 2026

Researchers at the University of British Columbia have identified a new form of hereditary prostate cancer that, while rare, can cause aggressive disease at a young age. The discovery paves the way for genetic testing programs that could help identify at-risk families and support early cancer detection.

While most cancers are caused by genetic changes that accumulate over a person's lifetime, about five to 10 per cent are linked to inherited mutations that can be passed from one generation to the next.

The best-known examples are mutations in the BRCA1 and BRCA2 genes, which greatly increase the risk of developing certain cancers, including breast, ovarian, pancreatic and prostate cancer. Genetic testing for BRCA mutations has transformed cancer care, helping people at risk access early screening and prevention programs.

The new study, published in Cancer Discovery, identifies another gene-called CDK12-that could provide a similar warning sign for aggressive prostate cancer. The researchers analyzed genetic data from more than 4,500 people with aggressive prostate cancer and identified five unrelated men carrying inherited CDK12 mutations. All five developed metastatic prostate cancer between the ages of 44 and 62.

> *"What's striking is that every patient we identified with this inherited mutation had already developed metastatic disease by the time they were diagnosed. The opportunity now is to identify these families earlier and give people the chance to benefit from enhanced screening, when there are still curative treatment options."*

Uncovering a hidden hereditary risk

The study was an international collaboration between researchers at UBC, BC Cancer, Vancouver Coastal Health Research Institute and the University of Washington, as well as institutions in Australia, the Netherlands, Spain and Belgium.

Until now, scientists believed harmful CDK12 mutations only occurred spontaneously within tumour cells and could not be inherited. To confirm that the inherited mutations were causing the cancer, the researchers looked for a distinctive genetic fingerprint left behind when CDK12 stops working.

"The tumours provided us with a genetic signature that pointed directly back to CDK12," said lead author Dr. Sofie Tolmeijer, a postdoctoral fellow in the Wyatt lab. "It gave us compelling evidence that these inherited mutations were playing a direct role in causing their cancer."

Although inherited CDK12 mutations were found in only about one in every 1,000 people with aggressive prostate cancer, the researchers estimate they could affect hundreds of families worldwide.

"While this mutation is extremely rare, the discovery could be lifesaving for the families who carry it," said Dr. Tolmeijer. "Finding one person with an inherited mutation gives us the opportunity to identify other family members at risk and help them take action before cancer develops or spreads."

Expanding genetic testing for families at risk

The researchers say the findings support adding CDK12 to standard genetic testing panels for hereditary prostate cancer. Unlike many newly discovered cancer biomarkers, existing clinical technology can already detect CDK12 mutations, providing a potentially faster and more cost-effective path to expanded testing.

"One of the most exciting aspects of this discovery is that we already have the technology needed to act on it," said Dr. Wyatt, who is also a senior research scientist at the Vancouver Prostate Centre and senior scientist at BC Cancer. "Adding CDK12 to existing genetic tests is relatively straightforward, which means this discovery could move from the research lab into clinical care much more quickly."

The study also raises the possibility that inherited CDK12 mutations may increase the risk of ovarian cancer. Several of the patients had family histories of ovarian cancer, and the researchers identified an additional person with ovarian cancer who carried an inherited CDK12 mutation and whose tumour showed the same characteristic genetic changes.

"With the right genetic testing and screening programs, we envision a future where no one who inherits one of these mutations dies from cancer," said Dr. Wyatt.

Journal references:

Tolmeijer, S., et al. (2026). Germline CDK12 variants in aggressive prostate cancer. Cancer Discovery. DOI: 10.1158/2159-8290.CD-26-0084. https://aacrjournals.org/cancerdiscovery/article-abstract/doi/10.1158/2159-8290.CD-26-0084/786384/Germline-CDK12-variants-in-aggressive-prostate?redirectedFrom=fulltext

Prostate Cancer Support Thunder Bay

September 2026

Scientists identify new therapeutic target for deadly prostate cancer

Rockefeller University Press — May 28/2026

Researchers at Columbia University Irving Medical Center have identified a gene that drives the development of neuroendocrine prostate cancer (NEPC), an aggressive form of the disease. The study, to be published May 28 in the Journal of Experimental Medicine (JEM), shows that genetic or pharmacological inhibition of Sirtuin 1 prevents the growth of NEPC tumors in mice, and lays the groundwork for future clinical studies aimed at developing new treatments for NEPC in humans.

One in every six men will be affected by prostate cancer in their lifetime. The current standard of care is androgen deprivation therapy (ADT). However, it is well documented that ADT will eventually fail, leading to tumor recurrence and development of the ADT-insensitive aggressive prostate cancer variant, NEPC. The process through which ADT-responsive tumors transition towards NEPC tumors-a phenomenon known as lineage plasticity-remains unknown. "Elucidating the mechanisms governing this process may improve treatment by overcoming plasticity-associated drug resistance," says Cory Abate-Shen, a professor at Columbia University Vagelos College of Physicians and Surgeons, who co-led the new JEM study with fellow professor Andrea Califano.

Abate-Shen's team performed a genetic screen in mice looking for mutations that recurred across multiple independent prostate cancer tumors. They identified 75 candidate NEPC-promoting genes, the most promising of which was Sirtuin 1 (Sirt1). Sirt1 encodes an enzyme with a broad range of functions, including control of gene expression and metabolism. The group first looked to a human prostate cancer cell line to characterize the role of Sirt1. In these cells, the induction of NEPC produced an increase in the expression of genes predicted to be activated by SIRT1 and a corresponding decrease in those predicted to be downregulated by this protein. Confirming these results, the group found that activation of Sirt1 in cells with low SIRT1 expression levels led to a robust increase in key NEPC markers.

Recapitulating their cell line data, the team found that silencing of Sirt1 profoundly reduced tumor growth in mice with NEPC, indicating that Sirt1 is indeed a promising target for NEPC treatment. They also treated the tumors with the FDA-approved SIRT1-inhibitor, Selisistat, which was originally developed for treatment of Huntington's disease. Excitingly, the researchers saw that Selisistat administration significantly reversed the NEPC phenotype.

Journal reference:
Nunes de Almeida, F., et al. (2026). A forward genetic screen identifies Sirtuin1 as a driver of neuroendocrine prostate cancer. Journal of Experimental Medicine. DOI: 10.1084/jem.20241484. https://rupress.org/jem/article/223/7/e20241484/282639/A-forward-genetic-screen-identifies-Sirtuin1-as-a

"Our findings demonstrate that SIRT1 plays a pivotal role in promoting NEPC, revealing a context-dependent function that extends beyond general tumor growth to the regulation of lineage plasticity and neuroendocrine differentiation, this highlights SIRT1 as an attractive and clinically actionable target for lethal prostate cancer that warrants

UPCOMING EVENTS

Sept 13th **Prostate Cancer Health Fair at 55 Plus 12:00noon to 4:30 PM**

Sept 17th **Dr. Nicholas Timothy Holzapfel, a Medical Oncologist, specializing in using chemotherapy, immunotherapy, and targeted agents to treat a wide range of cancers, will address our monthly meeting at 55 Plus Centre at 7:00 PM**

Prostate Cancer Support Thunder Bay

September 2026

PSMA PET detects high-risk prostate cancer bone metastases

Society of Nuclear Medicine and Molecular Imaging June 1 2026

Compared to conventional imaging techniques, prostate specific membrane antigen (PSMA) PET imaging provides superior detection of bone metastases in prostate cancer patients a critical indicator of a patient's long-term survival. Outcomes data revealed that patients with even just one bone metastasis experience faster disease progression and worse overall survival. This research was presented at the Society of Nuclear Medicine and Molecular Imaging 2026 Annual Meeting.

Prostate cancer commonly spreads to bone, and for decades physicians have relied on bone scans and CT scans to detect this. These tools, however, have limitations, frequently missing small deposits of cancer that are already present and already changing a patient's prognosis. Newer PSMA PET scans use a radioactive tracer that attaches directly to a protein on prostate cancer cells, making it far more sensitive than conventional imaging. As a result, PSMA PET has become the gold standard for staging prostate cancer at major cancer centers.

> *"We know that PSMA PET scans are effective in detecting bone metastases, but we don't have the data to show what that means in terms of overall outcomes. Our study sought to determine what happens to patients over time when PSMA PET finds just one to five bone metastases, yet the conventional scan looks completely normal."*
> *Surekha Yadav, MBBS, resident in the Department of Radiology and Biomedical Imaging, University of California, San Francisco*

The retrospective study followed 36 patients across two academic centers who had between one and five bone lesions on PSMA PET at initial diagnosis. Conventional imaging was reviewed to determine upstaging rates. Time to biochemical recurrence, castration-resistant prostate cancer, composite endpoints, and overall survival over a median of 25 months were calculated. Treatment patterns were also recorded.

More than 80 percent of patients had completely normal conventional imaging, despite having bone lesions detected on PSMA PET. Compared to the 984 patients in the same cohort with no bone metastases on PSMA PET, patients with even one to five bone lesions had more than five times the risk of progressing to treatment-resistant cancer, and nearly four times the risk of death.

"Treatment decisions made at the time of diagnosis have lasting consequences. If a patient's conventional imaging looks negative, they may be managed with a less intensive approach as if their cancer hasn't spread," said Yadav. "Our study shows that when PSMA PET finds bone metastases that conventional imaging misses, those patients are not in a gray zone. Their cancer behaves aggressively, they progress faster, and they die sooner. Many of them are currently being treated based on a bone scan that says everything looks fine."

According to Yadav, patients don't have to wait to take benefit from PSMA PET; it is already FDA-approved and available at major academic cancer centers, including the University of California, San Francisco and the University of California, Los Angeles. "What this research changes is not access to the scan, she said, it's what we do with the results. Until now, oncologists have had limited outcome data to guide how aggressively to treat patients whose PSMA PET finds bone metastases that conventional imaging misses. Our study provides that evidence."

Prostate Cancer Support Thunder Bay

September 2026

Study links ceramide levels to prostate cancer drug response differences

Wiley May 26

Ceramides-lipid molecules in cells that affect many physiological functions including cell differentiation, migration, and death-and their metabolites have been implicated in the development of cancer and other conditions. New research indicates that different ceramide metabolism in Black and white individuals with metastatic castration-resistant prostate cancer may help explain why they tend to experience different responses to anti-prostate cancer androgen receptor pathway blocking medications. The findings are published by Wiley online in CANCER, a peer-reviewed journal of the American Cancer Society. In two previous clinical studies, investigators observed differences in response between Black and white individuals who were treated with androgen receptor pathway inhibitors (which decrease or block the effects of hormones such as testosterone) for metastatic castration-resistant prostate cancer. This cancer is a form of advanced prostate cancer that continues to grow and spread even when testosterone is suppressed to castrate levels.

Because certain genetic ancestry-related variants of ceramide metabolism genes were linked to indicators of faster cancer progression in one of those studies, the researchers analyzed ceramide metabolism prior to and during androgen receptor pathway inhibitor therapy in Black and white participants in the two studies. The team focused on what's called the carbon acyl chain length of ceramides because evidence suggests that ceramides with a 24-carbon acyl chain length promote cell survival while ceramides with a 16-carbon acyl chain length induce cell death. The ratio of 24- to 16-carbon acyl chain length ceramides can influence cancer cells, with higher ratios protecting cells and lower ratios promoting cell death.

When the researchers analyzed trial participants' blood, they found that pretreatment total ceramide levels were lower among Black patients compared with white patients. Among pretreatment ceramides, Black patients had higher values of C24- to C16-ceramide ratios compared with white patients. An opposite trend seen during treatment, with lower C24- to C16-ceramide ratios among Black patients, whereas higher C24- to C16-ceramide ratios were observed among white patients. Also, certain C16-, C20-, and C24-ceramides were associated with shorter time to cancer progression or worse survival, with differences between Black and white patients treated in the studies.

"The two previous clinical studies we conducted were unique with respect to including equal numbers of Black and white patients and collecting trial participants' blood. This created an unprecedented opportunity to explore potential biomarkers that may associate with patient self-reported race and genetic ancestry and treatment outcome. The findings from this research offer valuable observations into ceramide metabolism, including genetic ancestry-related ceramide metabolism, and its potential relationship with prostate cancer outcomes. Continued investigation of genetic ancestry-related ceramide metabolism has the potential to improve prostate cancer outcomes for all patients and mitigate prostate cancer disparities."
Jennifer A. Freedman, PhD, senior author, Duke University School of Medicine

Source:
Wiley
Journal reference:
Piwarski, S. A., et al. (2026). Genetic ancestry–concordant ceramide metabolism and response to androgen receptor pathway inhibition in metastatic castration-resistant prostate cancer. Cancer. https://doi.org/10.1002/cncr.70371. https://acsjournals.onlinelibrary.wiley.com/doi/abs/10.1002/cncr.70371

Memberships

Time to purchase your $15.00, 2026 PCS membership
Can be purchased at the monthly general meeting or
send an E Transfer to Prostate Cancer Support using
info@prostatecancersupporttbay.org
(no password required)

Prostate Cancer Support Thunder Bay

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PRESIDENTS MESSAGE

I'm hoping everybody has enjoyed this warm summer after the terrible winter we just had!
July 28th we set up our booth at Applebee's to spread the word about Prostate Cancer. There was a large crowd all day and we had lots of good interaction.
Applebee's have been very supportive of our group and we really appreciate their continued support.
I continue to recover from surgery and will be good as new soon.
Don't forget our Health Fair at 55 Plus September 13th. It begins at 12 noon and goes until 4:30PM.
Hope to see you there. Enjoy the rest of the summer.

Sincerely
Your President

Keith Moore

PROSTATE CANCER HEALTH FAIR

Sunday **September 13th** at 55 Plus Centre
12:00 pm to 4:00 pm
Free PSA blood test (**by Lifelabs**)
as well as Urologists on site
Numerous **speakers** on relevant topics
Door prizes

IN PERSON MONTHLY MEETINGS AT 55 PLUS

The multi purpose room has been booked for the third Thursday of every month from 7 PM till 9 PM

DONATIONS

Prostate Cancer Support Thunder Bay is a charitable organization that relies entirely on donations to remain in operation.
donations can be e-transferred to info@prostatecancersupporttbay.org

Prostate Cancer Support Thunder Bay

September 2026

PCS THUNDER BAY

CONTACT US AT

1100 MEMORIAL AVE- SUITE 374

THUNDER BAY ONTARIO P7B 4A3

PHONE:

807 627 0333

EMAIL

info@prostatecancersupporttbay.org

WEB SITE:

prostatecancersupporttbay.org

Recently diagnosed with Prostate Cancer ?

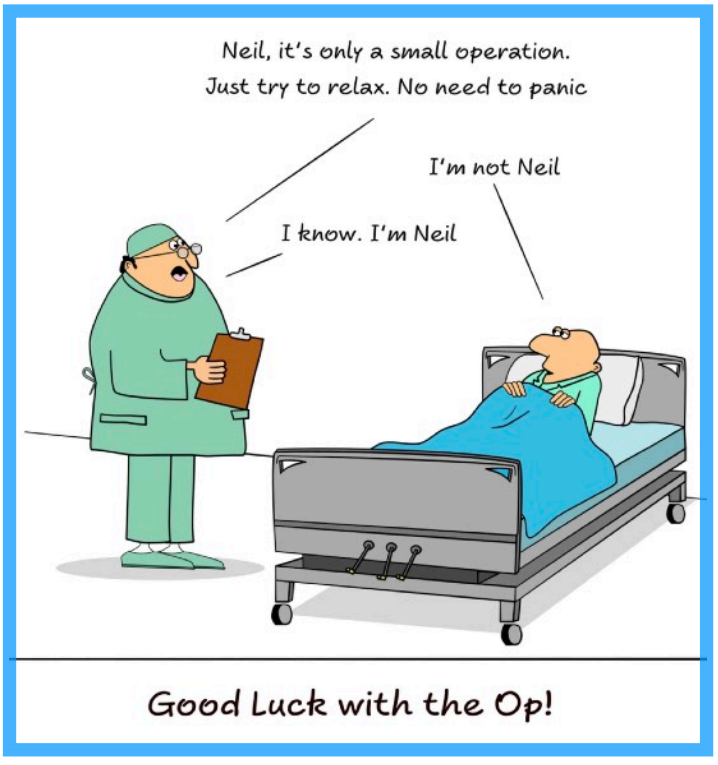
NEED SOMEONE TO TALK TO?

Please feel free to call anyone listed on the left side of the front page of this newsletter.

They have been where you are now and will be happy to listen to your concerns and questions.

Prostate Examiner Monthly News

Please forward photos or information that benefits communication to Prostate Cancer Support Thunder Bay members to the attention of Mike Aldrich.
email: mraldrich@tbaytel.net



Prostate Cancer Support Thunder Bay

September 2026

GET YOUR PSA TESTED

Its important

We believe in it so strongly that

we will reimburse you for your PSA test !!!

The PSA test is a key step in early diagnosis of prostate cancer

Early Detection Saves Lives
Get Informed!

Talk to your health care professional! Get your blood work done!

Send us the receipt

Address below or check us out on our website

Has been extended to December 31 2026.Available for men in NWO.

PCS Thunder Bay Members, please share the above message !

Inform your family, relatives, friends and neighbours to request a

PSA Test

Awareness Support Research

Telephone 807 627 0333

Email info@prostatecancersupporttbay.org

Web www.prostatecancersupporttbay.org

1100 Memorial Ave, Suite 374

Thunder Bay ON, P7B 4A3