



# Prostate Cancer Support Association of New Mexico

Celebrating  
30 years of  
supporting men  
and their families

PCSANM Quarterly  
April 2021  
Volume 28, Issue 2

## LIFELINE

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### Support Group Meetings

Due to COVID-19, PCSANM offers web-based meetings from 12:30-2:45 p.m. on the first and third Saturday of most months.

In-person support group meetings have been suspended until further notice.

Please call 505-254-7784 or email [pchelp@pcsanm.org](mailto:pchelp@pcsanm.org) for information. Meeting topics by date may be found at:

<https://www.pcsanm.org/meetings/>

## Exercise & Diet Helped Me Live Over 20 Years with Stage 4 Prostate Cancer

By Dennis Presthold, PCSANM Member

*The editors encourage articles from our members about their prostate cancer experiences. We cannot guarantee they will be published, but they will be considered.*

In May 1997, at the age of 53, I was diagnosed with stage 4 prostate cancer. I had a PSA over 160 and a bone scan that looked like a lit-up Christmas tree. My urologist informed me that I had advanced stage 4 prostate cancer that had gone to the bone. With no treatment, I would be expected to live less than one year. With hormone therapy, he estimated I would live approximately 18 months, maybe as long as two years. So I immediately started intermittent triple hormone therapy. After two months of treatment, my PSA had dropped from over 160 to below 0.1. At this point I decided to get much more involved with my own treatment.

I started a major investigation of prostate cancer on the Internet. I discovered that with advanced prostate cancer like I had, the hormone therapy would fail after approximately one year. After that, the cancer would become hormone insensitive/independent and continue to grow even while on hormone therapy, with an average survival of less than one year. Therefore, I started a major investigation into alternative approaches in addition to my hormone treatment to see if I could lengthen my survival time and improve my quality of life.

I very quickly learned that a cocktail approach using hormone therapy, along with diet and exercise to fight the cancer, would work the best. Using this cocktail approach, I was able to lengthen the time until my cancer became hormone insensitive from the standard time of less than 18 months to over 10 1/2 years.

Continuing to use my cocktail approach, after six months my PSA started to rise while still on hormone therapy. In February 2008, I joined a phase II clinical trial of a new second line hormone therapy medication called Abiraterone. It is now known as Zytiga. Initial results of the phase I study of Abiraterone showed very promising results with 61% of the men having declines in their PSA levels of greater than 50%. A few were experiencing PSA declines greater than 90%, and a median increase in survival of over 4 months.

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Special thanks to Presbyterian Healthcare Services for its generous support of this newsletter.

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*In Memory of*  
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 this name:  
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#### PCSANM *Lifeline*

A quarterly newsletter addressing issues of prostate cancer

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# Exercise & Diet Helped Me Live Over 20 Years with Stage 4 Prostate Cancer

By Dennis Prestholdt

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Continuing to use my approach of diet and exercise along with Abiraterone, I was able to reduce my PSA to an undetectable level of  $< 0.01$  within 6 weeks of starting Abiraterone. Of the men that started the phase II study of Abiraterone in the USA, I was the only man whose PSA became undetectable and the only man from the study that is still living.

It has now been over 13 years since I started Abiraterone and my PSA is still undetectable. I contribute a major portion of my success to the cocktail approach using diet and exercise along with the medical prostate cancer treatments.

The following is a summary of my exercise and diet:

### Exercise:

Along with reducing stress levels, improving muscle mass and bone density, exercise has been shown to reduce the growth rate of prostate cancer. The vast majority of published articles on exercise have shown that exercise can lower the levels of chemicals (such as prostaglandin) in the body that increase the growth of prostate cancer. Finally, laboratory studies have shown that when prostate cancer cells are exposed to blood taken from men that exercised, the cancer growth is slowed. But if the same cancer cells are exposed to blood taken from men that have not exercised, the growth of the cancer is increased.

Based on this information, I established this daily exercise and yoga/stretching program:

### **Monday, Wednesday, Friday:**

- 30-40 minutes fast walking at 3.4 miles/hour
- 30 minutes of yoga/stretching
- 15 minutes of upper body weight lifting using only 5 to 10 lb weights and 25 reps each (to reduce injury while on therapy)

### **Tuesday, Wednesday, Saturday:**

- 30-40 minutes of bicycle riding
- 30 minutes of yoga/stretching
- 15 minutes of lower body weight lifting

### **Sunday:**

- Day of rest and recovery

I did these exercises each day in local gym until COVID-19 closed the gyms. Then I switched to walking outside and riding a recumbent bicycle in my neighborhood.

See Diet on page 4

## Exercise & Diet Helped Me Live Over 20 Years with Stage 4 Prostate Cancer

Continued from page 3

### Diet:

UCLA scientists reported that 11 days of daily exercise and a low-fat, high fiber diet induce prostate cancer cells to die.

The University of South Carolina along with the University of Massachusetts reported that plant-based diets decrease the rate of PSA increase and slowed the rate of prostate cancer progression.

The University of Buffalo studies reported that low-fat, vegetarian diets inhibit the growth of prostate cancer cells in mice in the laboratory.

Several studies published in the Journal of National Cancer Institute showed a strong association between vegetable consumption, drinking green tea and reduced prostate cancer growth rate with the strongest effect from eating cruciferous vegetables (broccoli, cauliflower).

A research study presented at the American Association of Cancer Research showed a strong link between dairy product consumption and increased prostate cancer growth. Studies have shown that the dairy protein casein (found in milk and cheese) can greatly increase the growth of prostate cancer. These are only a few examples of the many studies that show consistent evidence that dietary fat (especially saturated fats and omega-6 fats), meats, and dairy are related to increased prostate cancer growth, while fruits, and vegetables, omega-3 fats and fiber are related to reduced prostate cancer growth.

Based on a review of many studies, I established the following diet as part of my cancer-fighting approach:

- Vegan (no meat or dairy with the exception of fish with high levels of Omega-3)
- Ultra-low fat diet with less than 10-15% of my daily calories coming from fat
- Lots of fruits and vegetables everyday
- Lots of low-fat soy foods (including soy cheese without casein)
- Lots of low-fat tomato-based foods
- One or more meals of salmon per week (wild caught)
- At least 3 cups of white or green tea a day
- Lots of cruciferous vegetables (broccoli sprouts are extremely beneficial)

The following is an example of a typical day's set of meals (cup of white tea with each meal):

- **Breakfast:**
  - Simple corn flakes with low-fat soy milk and blueberries or blackberries
  - Glass of orange juice (fortified with vit D and calcium)
- **Lunch:**
  - Cook a meal from Purple Carrot meal kit delivery company– they deliver fresh, measured ingredients for vegan meals with cooking instructions. Examples: smoky two-bean chili, sesame orange tofu, lemon grass soup
- **Dinner:**
  - Wild caught Salmon filet cooked with herbs and garlic
  - Steamed broccoli with half a baked potato covered with fat-free dressing

So over 20 years ago I was told I may only have 18 months to live. Using a cocktail of hormone therapy, Abiraterone, diet, and exercise, my PSA is presently undetectable. I have no symptoms of prostate cancer, and I'm living a full, healthy, unrestricted life.

*This is one PCSANM member's experience and may not work as effectively for everyone.*

Newsire: January 16, 2021

# The Circadian Clock and Prostate Cancer

Thomas Jefferson University

Our biological or circadian clock synchronizes all our bodily processes to the natural rhythms of light and dark. It's no wonder then that disrupting the clock can wreak havoc on our body. In fact, studies have shown that when circadian rhythms are disturbed through sleep deprivation, jet lag, or shift work, there is an increased incidence of some cancers including prostate cancer, which is the second leading cause of cancer death for men in the U.S. With an urgent need to develop novel therapeutic targets for prostate cancer, researchers at the Sidney Kimmel Cancer - Jefferson Health (SKCC) explored the circadian clock and found an unexpected role for the clock gene CRY-1 in cancer progression. The study was published on January 15th in Nature Communications.

"When we analyzed human cancer data, the circadian factor CRY-1 was found to increase in late stage prostate cancers, and is strongly associated with poor outcomes," explains Karen Knudsen, MBA PhD, executive vice president of oncology services for Jefferson Health and enterprise director of SKCC, and senior author of the study. "However, the role CRY-1 in human cancers has not been explored."

A common therapy for prostate cancer involves suppressing the male hormone androgen and/or the androgen receptor, as prostate tumors require androgens to develop and progress to advanced disease. With their collaborators in the U.S. and Europe, the researchers found that CRY-1 is induced by the androgen receptor in prostate tumor tissue obtained from patients, thus explaining in part the high levels of CRY-1 observed in human disease.

"This was a clear indication of CRY-1's link to prostate cancer," says Ayesha Shafi, PhD, a postdoctoral researcher in Dr. Knudsen's lab and first author of the study. "As we looked further into the role of CRY1, we unexpectedly found that the circadian factor was altering the way that cancer cells repair DNA."

Cancer treatments aim to damage the DNA in cancer cells and cause defects in repair mechanisms; eventually the cells self-destruct when the damage is severe. The researchers probed CRY-1's possible role in DNA repair in cultured cells, animal models and tissue harvested from prostate cancer patients. They first induced DNA damage by exposing cancer cells to radiation and found that CRY-1 levels became elevated, indicating that it was responding to this type of damage. They also found that CRY-1 directly regulates the availability of factors essential for the DNA repair process, and alters the means by which cancer cells respond to DNA damage. The findings suggest that CRY-1 may offer a protective effect against damaging therapies.

"The fact that CRY-1 is elevated in late-stage prostate cancer may explain why androgen-targeting treatments become ineffective at those later stages," says Dr. Shafi. "It also tells us that if a tumor has high levels of CRY-1, DNA repair targeting treatments may be less effective for them."

"Looking ahead, the team plans to explore how best to target and block CRY-1 and what other existing therapies may work synergistically to hinder DNA repair in prostate cancer cells. They also plan to study more circadian rhythm genes and determine how circadian disruption may affect cancer treatment.

"It's been shown that circadian disruptions can affect efficacy of treatment, but also that aligning treatment with the body's natural rhythms or giving therapy at certain times of the day can be beneficial," explains Dr. Knudsen. "Our findings open up a multitude of important research questions exploring the link between the circadian clock and cancer."

Prostate Cancer Foundation: January 28, 2021

# What's New in Inflammation and Prostate Cancer

Janet Farrar Worthington

## Anti-Inflammatory Drugs

If [inflammation can lead to prostate cancer](#), could anti-inflammatory agents help protect against it? Johns Hopkins epidemiologist and friend to Prostate Cancer Foundation (PCF) Elizabeth Platz, Sc.D., has been intrigued by this possibility for many years. She is senior author of a [new study](#) on the use of aspirin and statins, published in *Cancer Prevention Research*.

The study, of men in the placebo arm of the Prostate Cancer Prevention Trial, doesn't answer this question once and for all – but adds more weight to the idea that, for **lowering the risk of developing potentially fatal prostate cancer, fighting inflammation is a good thing.**

Evidence from observational studies has suggested that **when taken regularly over time, aspirin may lower the risk of prostate cancer.** These drugs block enzymes that play a key role in the body's inflammatory response. Other studies have linked long-term use of statins, prescription drugs that are used to lower cholesterol but that also are anti-inflammatory, to a lower risk of advanced and metastatic prostate cancer.

In this most recent study, the investigators looked for **inflammation markers** in benign prostate tissue samples. “We compared the use aspirin and statins with the presence and extent of inflammation in the prostate tissue,” says Platz. They also looked at prostate biopsy slides for the presence of certain immune cells that are involved in inflammation.

“Of 357 men, 61 percent reported aspirin use, and 32 percent reported statin use,” Platz continues. “Aspirin users were more likely to have low FoxP3, a T regulatory cell marker, and statin users were more likely to have a low CD68, a macrophage marker.”

“Our results suggest these medications may **alter the immune environment of the prostate.** A next step is to determine whether these immune alterations may underlie the epidemiologic observations that taking an aspirin or statin may protect against getting advanced prostate cancer, and dying from it.”

## Prostate Cancer Loves Fats

Here's some more recent research out of Johns Hopkins, a neat bit of basic science that may help explain the findings of Platz's recent study: “Our work is mechanistic,” says investigator Marikki Laiho, M.D., Ph.D., director of the Division of Molecular Radiation Sciences, “and provides insight into **how the tumor microenvironment senses the excess load of lipids (fats).** Diet and statins obviously relate to the amount and regulation of the lipids, and have shown those clear correlations to prostate cancer. However, we need to understand *why* to be able to correct the problem. Our work provides at least one explanation how the lipids fuel cancer.” One step “was just to feed the prostate cancer cells with cholesterol, which made them more invasive.”

**It turns out that even on a cellular level, prostate cancer gravitates to its own kind of junk food – the tiny version of deep-fried Oreos with a side of chili cheese fries.** Laiho and colleagues have just figured out *how* the body enables prostate cancer's terrible diet.

The key is a lipid-regulating protein called CAVIN1, the scientists [reported](#) in the journal, *Molecular Cancer Research*. In lab studies, when CAVIN1 was removed from cells in and around the prostate tumor, the fatty acid that was in those cells spilled into the tumor's microenvironment. The effect on prostate cancer cells was dramatic: **the cancer cells soaked up the lipids, which then acted as turbo fuel to make the cancer spread more aggressively.**

“In every prostate cancer cell line we tested,” says research fellow Jin-Yih Low, Ph.D., the study's first author, “**tumor cells universally had an appetite for the lipids,** using them to strengthen the protective membrane around the cell, synthesize proteins and make testosterone to support and fuel the cancer's growth. The tumor cells then behaved more aggressively, exhibiting invasive and metastatic behavior. Just having access to the lipids gave the tumor cells more power; the tumor's behavior changed.”

See But Wait on page 7

Prostate Cancer Foundation: January 12, 2021

## Prostate Cancer Foundation (PCF)-Funded Research Inhibits Prostate Cancer Growth

New research, just published in [Cancer Discovery](#), describes a new therapy with promising preclinical activity that is now being tested in clinical trials for very advanced prostate cancer patients. Early studies suggest this treatment has promising activity for the significant proportion of patients with advanced prostate cancer whose tumors remain driven by the AR (androgen receptor), but no longer respond to the newer AR-targeted therapies.

Why is this so important for patients? Advanced prostate cancer that has developed resistance to hormone therapy has no cure. It's like a car engine that continues to run when the "fuel supply" (testosterone) is cut off by ADT, or even by newer androgen directed therapies. Treatments are available, the disease often continues to progress, and more options are urgently needed for such patients.

A PCF-funded team led by Dr. Johann de Bono (Institute of Cancer Research, London and Royal Marsden NHS Foundation Trust) and Dr. Karen Knudsen (Thomas Jefferson University) has identified a promising new therapeutic target in prostate cancer (called p300/CBP), and are credentialing this target biologically and in clinical trials. p300/CBP is critical to the activity of the androgen receptor (AR), the main driver of prostate cancer growth.

For the first time, researchers were able to show that blocking p300/CBP with a new experimental treatment causes a decrease in AR signaling and slows tumor growth. This inhibitor molecule – referred to as CCS1477 – is currently in a [phase 1 clinical trial](#) of patients with advanced prostate cancer. To confirm that CCS1477 is able to block AR activity in patients, the team also looked at markers of AR activity in the blood and biopsy tissues of patients in the trial. Results from the trial on the optimal dosage, safety, and preliminary efficacy of CCS1477 are not yet available.

Taken together, the results show that CCS1477 merits further study as a possible treatment for castration-sensitive and castration-resistant prostate cancer, both alone and in combination with existing medications.

This discovery was funded in part by a PCF Challenge Award led by Dr. de Bono and Dr. Knudsen. We congratulate the entire team on their careful, deliberate work to discover a new, potentially "druggable" target in advanced prostate cancer.

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Continued from page 6

**But wait! There's more: nearby cells changed, too. Deprived of their regular type of lipids, normal stromal cells started to churn out inflammatory molecules, adding fuel of their own to the fire.**

Laiho's team then confirmed their findings in mouse models, comparing tumors with and without CAVIN1 in the stromal cells. In the mice, Laiho says, "although the presence or absence of CAVIN1 did not affect the speed of tumor growth, lack of CAVIN1 definitely caused the cancer to spread. All of the mice with tumors that lacked CAVIN1 had a twofold to fivefold increase in metastasis. The tumors also had a fortyfold to hundredfold increase in lipids and inflammatory cells."

**The investigators were surprised at these results,** Laiho adds. "We suspected CAVIN1 was important, but we didn't realize how important. **The tumor's microenvironment matters, and the amount of lipids matters a lot.**" **Just changing the level of lipids "created a situation of rampant metastasis."**

What could come from this research? One possibility is development of a new biomarker: a loss of CAVIN1 in local or locally advanced cancer, for example, could signal a higher risk of metastasis. The next step is to understand more about the inflammatory process in the tumor's microenvironment. "We want to understand why the inflammation brings in macrophages, immune cells that further exacerbate the inflammatory process, instead of T cells, which should attack the cancer."



Medscape Medical News: January 29, 2021

# Prostate Cancer Drug Equally Effective in Black and White Men

M. Alexander Otto, PA, MMS

In an open-label trial, 93% of Black men with metastatic hormone-sensitive [prostate cancer](#) (mHSPC) experienced a response to treatment with [enzalutamide](#); the response rate was nearly identical to the 94% response rate among non-Black men.

The finding is important because it shows that Black men, who historically have been underrepresented in prostate cancer studies but who constituted 41% of the current trial, also benefit from newer treatments.

Seventy-one men with mHSPC were randomly assigned in a 1:1 ratio to receive either enzalutamide 160 mg daily or [bicalutamide](#) 50 mg daily. Enzalutamide is a recently approved second-generation androgen receptor blocker; bicalutamide is a first-generation blocker that was approved in [1995](#). Patients who received either drug had undergone [testosterone](#) suppression with a [luteinizing hormone](#)–releasing hormone analogue.

Overall, 30 of 32 patients who received enzalutamide (94%) met the primary endpoint by achieving a prostate-specific antigen (PSA) level <4 ng/mL after 7 months. That endpoint was a surrogate of overall survival. Of the patients who received bicalutamide, 17 of 26 men (65%) met the primary endpoint ( $P = 0.008$ ). PSA wasn't measured at 7 months for the remaining 13 men.

Enzalutamide is one of several newer androgen receptor axis–targeting (ARAT) agents. The benefit of enzalutamide in comparison with bicalutamide has been demonstrated before in phase 3 trials, but almost exclusively in White men. In the new trial, 29 men (41%) were Black; the other patients were White, and there was one Asian man in the four-center study.

"To our knowledge, this is the only randomized clinical trial comparing bicalutamide with enzalutamide in a Black patient population.... The favorable risk benefit profile makes it crucial to strongly consider the addition of enzalutamide to

ADT [androgen deprivation therapy] in Black patients with mHSPC," say the investigators, led by genitourinary oncologist [Ulka Vaishampayan, MD](#), professor at the University of Michigan, Ann Arbor, Michigan.

The trial was [reported](#) in *JAMA Open Network*.

## Differences in Black and White

Importantly, only 42% of Black men experienced a response to bicalutamide; 93% of those patients experienced a response to enzalutamide, the newer agent. Among non-Black patients, the response to the older agent was more comparable — 86%, vs 94% with enzalutamide. "Racial differences in bicalutamide efficacy are overcome by using contemporary [ARAT agents] such as enzalutamide" in advanced prostate cancer, say the authors. "ADT plus bicalutamide is inadequate therapy in all cases, but especially for Black patients," they note.

On a level playing field, several investigations have reported better overall survival among Black men than White men in metastatic castration-resistant prostate cancer treated with contemporary options, including [abiraterone](#), [enzalutamide](#), [sipuleucel-T](#), and [docetaxel](#). Genetics might play a role, the editorialists comment.

## Black Recruitment Is Key

Vaishampayan and her colleagues analyzed tumor samples to gain insight into race and response.

They found that high baseline ERG protein levels and changes in androgen metabolism enzymes and genetic resistance pathways might play a role in the difference in response to bicalutamide between their Black and non-Black patients, but they found nothing conclusive. They said that "further investigations exploring molecular biomarkers to help guide therapy are highly recommended".



Medscape Medical News: February 12, 2021

## Drop in PSA Screening, Increase in Metastatic Prostate Cancers

Sharon Worcester

The incidence of metastatic prostate cancers at diagnosis increased as prostate-specific antigen (PSA) screenings across U.S. states decreased, registry data show.

Between 2008 and 2016, the age-adjusted overall mean percentage of prostate cancers that were metastatic at diagnosis increased significantly from 6.4 to 9.0 per 100,000 men. During the same period, the overall mean percentage of men undergoing PSA screening decreased from 61.8% to 50.5%, [Vidit Sharma, MD](#), reported in a poster session at the 2021 Genitourinary Cancers Symposium ([Abstract 228](#)).

A random-effects linear regression model demonstrated that longitudinal reductions across states in PSA screening were indeed associated with increased age-adjusted incidence of [metastatic prostate cancer](#), said Sharma, the lead author of the study and a health services fellow in urologic oncology at the University of California, Los Angeles.

Sharma and colleagues had reviewed [North American Association of Central Cancer Registries](#) data from 2002 to 2016 for each state and extracted survey-weighted PSA screening estimates from the Centers for Disease Control and Prevention's [Behavioral Risk Factor Surveillance System](#). The researchers noted wide variations in screening across states, but they said across-the-board declines were evident beginning in 2010, marking a "worrisome consequence that needs attention."

[Robert Dreicer, MD](#), deputy director of the University of Virginia Cancer Center, Charlottesville, agreed, noting in a [press statement](#) that the findings suggest reduced PSA screening may come at the cost of more men presenting with metastatic disease. "Patients should discuss the risks and benefits associated with PSA screening with their doctor to identify the best approach for them," Dreicer said.

PSA screening has been shown to reduce [prostate cancer](#) metastasis and mortality, but it has also been linked to overdiagnosis and overtreatment of prostate cancer. As a result, the [U.S. Preventive Services Task Force](#) (USPSTF) "found insufficient evidence to recommend PSA screening in 2008 and later recommended against PSA screening in 2012," Sharma said.

Several studies subsequently showed a rise in metastatic [prostate cancer diagnosis](#), but the role of PSA screening reductions in those findings was unclear. In 2018, the USPSTF updated its recommendations, stating that men aged 55-69 years should make "an individual decision about whether to be screened after a conversation with their clinician about the potential benefits and harms." The task force recommended against PSA screening in men aged over 70 years.



Newsire: November 18, 2020

## Prolonged Use of Common Prostate Cancer Treatment May Impair Cardiorespiratory Fitness, Increase Risk of Cardiovascular Death

Prolonged androgen deprivation therapy (ADT) can impair cardiorespiratory fitness and increase risk of cardiovascular death in prostate cancer patients with high risk of cardiovascular disease, according to a study in *JACC: CardioOncology*. The findings contribute further data supporting the need for cardiovascular disease (CVD) monitoring in patients who are living longer after successful cancer treatment.

Approximately 1 in 9 men will be diagnosed with prostate cancer during their lifetime, and it is the second leading cause of cancer death for men in the United States. Furthermore, CVD is a leading cause of death in men who have a history of prostate cancer.

ADT with radiation therapy is a standard primary treatment for prostate cancer as an alternative to surgery and is frequently used in patients with metastatic, recurrent and localized high-risk tumors. More prolonged use of ADT in certain patients with prostate cancer is increasingly employed following studies that demonstrated improved cancer outcomes compared to short-term ADT exposure. However, whether ADT is associated with increased CV mortality remains controversial. Authors of this study set out to study the association between ADT exposure and CV mortality and cardiorespiratory fitness (CRF), which is a known independent predictor of CV mortality, in patients with prostate cancer.

Researchers of this study evaluated 616 patients from a single center, retrospective cohort who underwent an exercise treadmill test for clinical indications a median of 4.8 years after their prostate cancer diagnosis. CV risk assessment was determined by a patient's demographics, indication for exercise treadmill test (such as chest pain), medical history and medication usage at the time of the treadmill test. Prostate cancer

treatment regimens used before and after a patient's exercise treadmill test were looked at. Researchers also examined ADT treatment, including the therapy used and duration of ADT exposure prior to the treadmill test. ADT exposure was categorized as short-term (less than or equal to six months) versus prolonged (greater than six months). CRF was calculated from peak treadmill speed and grade achieved during a patient's exercise treadmill test.

Almost one-quarter of the patients (150) received ADT prior to their treadmill test, with 51 patients exposed to long-term ADT use. There were 504 patients (81.8%) out of the study cohort who had two or more CV risk factors, such as diabetes mellitus and hypertension. Most patients with prolonged exposure to ADT (92.2%) had two or more CV risk factors.

The rate of reduced CRF was considerably higher among patients with ADT exposure compared to those without the treatment (48.7% versus 32.6%). Prolonged ADT exposure was significantly linked to reduced CRF. Long-term ADT exposure was associated with an almost four-fold increased adjusted risk of CV mortality.

"This study highlights that patients with prostate cancer and high baseline CV risk are at increased risk of reduced CRF and CV mortality when exposed to prolonged ADT regimens," said John D. Groarke, MBBCh, MSc, MPH, cardiologist and author of this study. "While prolonged ADT certainly plays a role in the treatment of prostate cancer, these findings emphasize the need to consider CV surveillance/risk modification during and after ADT exposure."

Medscape Medical News: February 5, 2021

# MRI-Guided Prostate Biopsy Prevails in PRECISE Trial

Neil Osterweil

Here's welcome news for men of a certain age: new results support a less invasive approach to investigations for suspicion of [prostate cancer](#).

An approach using MRI of the prostate followed by targeted biopsy (TB) in men with images suggesting a high risk beat the conventional approach of using transrectal ultrasound (TRUS)-guided 12-core systematic biopsy.

The results come from the randomized phase 3 PRECISION trial, and were [published](#) online in *JAMA Oncology*. "What the trial showed is that by taking an imaging-first strategy, you could reduce the number of patients needing a biopsy by about 40% and actually find more significant cancer (35% vs 30%) and reduce the diagnosis of grade group 1 cancers that we don't want to find by more than half," lead author Laurence Klotz, CM, MD, from the Sunnybrook Health Sciences Centre in Toronto, Canada, said in an interview with *Medscape Medical News*.

These results from the PRECISE trial support and slightly improve upon findings from the European-based [PRECISION](#) trial. That European trial had already provided "compelling evidence in favor of MRI and targeted biopsy," notes Olivier Rouvière, MD, PhD, from the University of Lyon, Lyon, France, writing an [accompanying editorial](#). But he argues that it was worth duplicating the trial, as "it must not be forgotten that in science, testing the robustness of an effect and the factors influencing it is as important as demonstrating this effect in the first place."

The results from both trials suggest that, instead of replacing TRUS biopsy entirely, MRI results could be used to guide patients to the appropriate diagnostic pathway, Rouvière comments.

"Using only MRI findings to decide which patients should undergo biopsy is probably insufficient," he adds. "Most likely, MRI findings will be used in conjunction with other biomarkers such as PSA density to select, among the patients with positive MRI findings, those who need targeted biopsy (and

those who may safely avoid it), and among the patients with negative MRI findings, those who may still deserve systematic biopsy," he writes.

## Details of PRECISE Findings

The Canadian PRECISE study was developed as a trial in coordination with the European PRECISION study, but the Canadian version had several additional features: It added risk-based eligibility, systematic follow-up of all patients for 2 years, a repeat MRI in all untreated patients, investigation of fluid- and tissue-based biomarkers in the cohort, and an economic analysis.

The patients randomly assigned to MRI underwent an MRI-targeted biopsy (MRI-TB) only if a lesion with a Prostate Imaging Reporting and Data System (PI-RADS) score of 3 or greater was identified, whereas all men in the other arm of the trial underwent a systematic TRUS-guided 12-core biopsy.

The MRI approach identified more clinically significant cancers. Grade 2 or higher tumors were found in 79 (35%) of 227 men allocated to MRI-TB, vs 67 (30%) of 225 men who underwent TRUS biopsy.

MRI also reduced the need for a biopsy. Of 221 men who were randomly assigned to the MRI group, 83 (37%) had a negative MRI result and avoided biopsy. In contrast, all men in the TRUS group had a biopsy. In addition, MRI was associated with a marked reduction in the diagnosis of clinically insignificant International Society of Urological Pathology (ISUP) grade group 1 cancers (10% with MRI-TB vs 22% with TRUS). Detection of such early cancers, under conventional protocols, often leads to unnecessary therapies or invasive procedures with significant side effects.

These results led the researchers to conclude that the strategy of MRI followed by MRI-guided biopsy only in men at risk of prostate cancer "offers substantial advantages over an initial systematic biopsy."



# Prostate Cancer Support Association

of New Mexico

**PCSANM Lifeline Newsletter**  
**Celebrating 30 years of supporting men  
and their families**

**Prostate Cancer Support Association  
of New Mexico, Inc.**  
**2533 Virginia St. NE, Suite C**  
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## A Message from the Chairperson

**April 2021**

Dear Lifeline Reader: Whether you are a prostate cancer patient or survivor, an interested or concerned family member of any sort, or simply a friend, here's a challenge. The topic is fundraising and you. No, keep reading. You can become a much-appreciated PCSANM fundraiser and it needn't cost you anything but several minutes of time (well, you might want to chip in a modest amount). During 2020, two individuals familiar with our group did just that in recognition their birthdays.

What do you do? You set up a free Facebook Birthday Fundraiser. You will need a Facebook account and preferably a collection of Facebook Friends. Go to the Birthday Fundraiser setup page ([facebook.com/help/search/?query=birthday fundraiser](https://facebook.com/help/search/?query=birthday+fundraiser)) and follow the directions. You will need to name the nonprofit you want to support, the amount of money you hope to raise, the date your fundraiser will begin (your birthday) and end (generally a week or two later). Finally, title your fundraiser and why you are interested in and a supporter of PCSANM.

Got questions? Send me an email at [pchelp@pcsanm.org](mailto:pchelp@pcsanm.org). And as always, thank you for your support.

A final note — hopefully you read our lead article authored by a long-time PCSANM member. We are eager to publish member stories about their journeys. If you've got an idea, please email me or LIFELINE editors Lou Reimer or Ann Weinberg at the email address listed above.

A handwritten signature in cursive script that reads "Rod Geer".

**Rod Geer**

**Chairperson of the Board, PCSANM**