Newsletter

Prostate Cancer 101, Inc.

http://prostatecancer101.org

▸May, 2014

The Prostate Cancer Information and Support Group of the Mid-Hudson

Special Event
Tuesday, May 6, 2014: Dr. David Shaffer

On Tuesday, May 6, 2014, in lieu of our regular First Tuesday newly diagnosed meeting, Dr. David Shaffer of New York Oncology Hematology will be making a presentation on **"Advances and Treatments in Advanced Prostate Cancer."** We will be meeting in the auditorium at our usual 4:30 p.m. time frame. Bring a friend and a list of any questions, but mostly, bring yourself to absorb at least some of the knowledge that Dr. Shaffer can impart.

Dr. Shaffer was born in Louisiana – wonder if he can cook as well as Paul Prudhomme or is a Zydeco aficionado? He is a talented and well respected medical oncologist with New York Oncology Hematology in Albany. You may remember that Dr. Michael Kolodziej was a partner in the same practice when he left last year to pursue a career with Aetna. Dr. Shaffer has ably taken care of the men who were seeing Dr. K and we're hearing much praise about him from those patients. It's always a good thing to hear positive reports about a doctor.

Dr. Shaffer was educated and trained in Louisiana, Oklahoma, Texas, North Carolina and even in New York; Louisiana State University, University of Tulsa, University of Houston, University of North Carolina and even Indiana University for a Master's program in Slavic languages. His Internship and Residency were in St. Louis, MO at Barnes-Jewish Hospital; Clinical & Research Fellowships at Memorial Sloan-Kettering, NYC. He was principal investigator at the Laboratory of Developmental Genetics, Pier-Paolo Pandolfi in NYC.

He is an Assistant Professor in the Department of Medicine at Albany Medical Center and had been an Instructor at both Memorial Hospital and Weill Medical of Cornell University. The good doctor is a member of several Scientific and Medical Societies and the recipient of many honors and awards.

One could say he is a man for all seasons and certainly a man to be trusted with issues pertaining to prostate cancer. From all reports he is a kind, compassionate and most excellent physician and one on whom we can call in confidence.

So be at the Hurley Reformed Church on Tuesday, May 6 by 4:30 p.m. and be prepared to expand your horizons.

Selenium and vitamin E supplements 'increase prostate cancer risk'

A new study recently published in the *Journal of the National Cancer Institute* suggests that taking high doses of selenium and vitamin E supplements may increase the risk of prostate cancer, depending on a man's selenium levels prior to taking the supplements.

The research team, including first author Dr. Alan Kristal of the Public Health Sciences Division of the Fred Hutchinson Cancer Research Center in Seattle, WA, analyzed 1,739 patients with prostate cancer and 3,117 matched controls from the Selenium and Vitamin E Cancer Prevention Trial (SELECT).

According to the investigators, previous research has suggested that men who already have an adequate intake of selenium would not benefit from supplements of the nutrient.

Therefore, the researchers took selenium measurements from the toenails of participants at the baseline of the study.

Selenium is a chemical element most commonly found in seafoods and organ meats, such as liver. Other food sources of selenium include muscle meats, cereals and dairy products.

The National Institutes of Health state that selenium is nutritionally essential for humans and plays roles in reproduction, thyroid hormone metabolism and DNA syntheses, as well as protects against oxidative damage and infection.

According to the Food and Nutrition Board, the recommended dietary allowance for both males and females aged 14 years and over is 55 mcg per day.

For the study, the researchers wanted to determine whether taking daily high doses of vitamin E (400 IU) and/or selenium (200 mcg) may reduce the risk of prostate cancer.

Vitamin E is a group of fat-soluble compounds that act as an antioxidant in the body. The vitamin is commonly found in foods such as nuts, seeds, vegetable oils, green leafy vegetables and fortified cereals.

Dietary supplements 'not necessarily helpful or innocuous'

SELECT began in 2001 and was scheduled to carry on for 12 years. But in 2008, the study was called to a halt on the grounds that no protective effects were found from selenium supplements and vitamin E supplements were thought to increase the risk of prostate cancer.

However, although the men stopped taking the supplements in 2008, the researchers continued following them in order to monitor their prostate cancer risk.

The findings revealed

that men who had high selenium levels at the beginning of the study had a 91% increased risk of high-grade prostate cancer. According to the researchers, the levels of selenium for these men became toxic.

The investigators also found that for men with low selenium levels at the baseline of the study, vitamin E increased total prostate cancer risk by 63%, while high-grade prostate cancer risk increased by 111%.

"Many people think that dietary supplements are helpful or at the least innocuous. This is not true," says Dr. Kristal.

"We know from several other studies that some high-dose dietary supplements - that is, supplements that provide far more than the daily recommended intakes of micronutrients - increase cancer risk.

We knew this based on randomized, controlled, double-blinded studies for folate and beta carotene, and now we know it for vitamin E and selenium."

He adds that people taking vitamin E or selenium supplements should stop because there is no evidence that they produce any health benefits - only risks.

Dr. Kristal says that even standard multivitamins - which he says have yet to demonstrate any risk - could be harmful in high doses.

"Taking a broad view of the recent scientific studies, there is an emerging consistency about how we think about optimal intake of micronutrients," he adds.

"There are optimal levels, and these are often the levels obtained from a healthful diet, but either below or above the levels there are risks."

Of late, there have been many studies questioning the health benefits of vitamin supplements. *Medical News Today* recently reported on a study suggesting that vitamin C and E supplements may hinder athletes' training, while other research suggests that multivitamins are a waste of money and have no health benefits.

Written by Honor Whiteman

Contributions

Our thanks for your help so we can continue our outreach

William & Sydna Byrne
Jay Dorin
Arnold Jaffe
Andrew Pletch
Bill & Arlene Ryan
Craig Smith

Prostate Cancer 101 is a 501 (c) (3) IRS approved non-profit organization. Your ***tax deductible*** donations should be mailed to:

Prostate Cancer 101
c/o Diane Sutkowski, Treasurer
8 Alcazar Avenue
Kingston NY 12401-4302

Cialis May Not Prevent Impotence in Men Treated for Prostate Cancer By Steven Reinberg-*HealthDay Reporter*

Study sees little value in taking the drug during radiation treatment

April 1, 2014 (HealthDay News) -- Taking the erectile dysfunction drug Cialis while receiving radiation therapy for prostate cancer doesn't seem to help men's sexual function after treatment, a new study finds.

About 40 percent of men undergoing radiation therapy for prostate cancer suffer from erectile dysfunction afterward, according to the study. The researchers wanted to find out whether impotence could be prevented by having patients take Cialis (tadalafil) during the course of treatment.

But there was very little difference in outcome when Cialis was compared to a placebo pill.

"There is no indication to use Cialis in men about to undergo radiotherapy for prostate cancer," said lead researcher Dr. Thomas Pisansky, a professor of radiation oncology at the Mayo Clinic.

"Cialis should be reserved for the treatment of erectile dysfunction if and when it occurs," he added.

The report was published April 2 in the *Journal of the American Medical Association* and partially funded by Eli Lilly & Co., the maker of Cialis. The study also received funding from the U.S. National Cancer Institute.

Dr. David Samadi, chairman of urology at Lenox Hill Hospital, in New York City, said, "Radiotherapy is the most common treatment for prostate cancer, but erectile dysfunction is a common side effect in a large number of patients."

This study clearly shows that there is no support for use of medications such as Cialis, Viagra and Levitra to prevent erectile dysfunction after radiation therapy, said Samadi, who was not involved with the research. "All treatments come with side effects, and a good discussion with a urologist and the radiation oncologist about those side effects, upfront, is part of the decision-making process," Samadi said.

For the study, Pisansky's team randomly assigned 242 men with prostate cancer to receive daily doses of Cialis or a placebo for 24 weeks, starting when radiation therapy began.

The researchers found that at 28 and 30 weeks after the start of radiation therapy, 79 percent of those who received Cialis maintained erectile

function compared with 74 percent of those who received placebo -- a difference of 5 percent.

After a year, there was still not a significant difference between the Cialis and placebo groups, with 72 percent of men who took Cialis and 71 percent who took the placebo able to maintain an erection.

Moreover, Cialis was not associated with an improvement in overall sexual function or satisfaction. Likewise, the partners of men who took Cialis saw no significant effect on sexual satisfaction, the researchers noted.

Dr. Bruce Gilbert, director of reproductive and sexual medicine at North Shore LIJ Health System in Great Neck, N.Y., took issue with the study.

"We have a problem in this study. The data that they are looking at is the patient's subjective response to whether or not their erections are good. We don't know if the patient had real problems with erections, only what he said about it," Gilbert said.

The real question boils down to the damage radiation therapy causes. If the damage is to nerves, then drugs like Cialis won't work because they only affect the blood circulation, Gilbert explained.

"Whether you have radiation or surgery you are going to have some impairment in your erections. When you are treating a cancer, you are treating the cancer. The side effects can be dealt with after," he said.

Gilbert said that treatments are available. "With sexual function, we can get most people working again," he said. "We use a variety of medications, possibly injected medications, or other alternatives that we have."

Source: prostate cancer foundation-Health Day

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To be added to this list contact:
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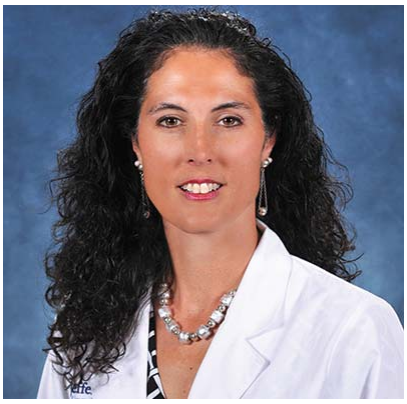
or 8 Alcazar Avenue, Kingston, NY 12401-4302

PCF researchers discover molecular crosstalk that prostate cancer cells use to survive radiation therapy; plan steps to halt that “conversation”

Prostate Cancer Foundation-funded researchers published two studies this fall that help explain why androgen deprivation therapy improves survival when given with radiation therapy; ability to repair broken DNA implicated. Findings may predict which men are most likely to benefit from combination therapy, and could lead to novel treatments for advanced prostate cancer.



"This research identifies the DNAPK enzyme as a very viable therapeutic target against prostate cancer." - Karen Knudsen, PhD

March 28, 2014 -- A 2011 study in the *New England Journal of Medicine* provided definitive evidence that adding a short course of androgen deprivation therapy (ADT) to radiation therapy in men being treated for prostate cancer increased their chances of survival. “The addition of hormone therapy can cure you vs. not cure you,” says William Polkinghorn, MD, a radiation oncologist at Memorial Sloan-Kettering Cancer Center in New York, and a Prostate Cancer Foundation-funded researcher. “So we knew this combination worked, but no one knew why it worked,” he adds. Now, thanks to two side-by-side studies recently published in *Cancer Discovery*, one by Polkinghorn and colleagues, the other led by Karen Knudsen, PhD, of Thomas Jefferson University in Philadelphia, who is also a Prostate Cancer Foundation-funded researcher, the fix is in: ADT retards the ability of cancer cells to repair DNA damage caused by the radiation therapy.

Radiation injures cells by causing severe DNA damage. Cancer cells then try to survive this insult by firing up DNA-repair mechanisms. It works like this: radiation-injured cells sense damaged DNA and turn on DNA repair pathways, involving many specialized enzymes that perform functions such as pasting broken DNA strands back together. If DNA can be repaired, the cells will go on to survive and divide. But if not, cells have embedded suicide programs that force them to die in order to avoid passing on mutations to future generations of cells. Such cell-suicide programs function to block cancer growth.

One pathway to cancer growth is when this programed cell death in response to DNA or genome damage fails to occur. Just as “survival of the fittest” is used to describe Darwin’s theory of natural selection in the evolution of species, the ability of cancer cells to cheat death when their DNA is damaged allows them to acquire even more mutations, leading to the evolution and rapid outgrowth of the most competitive cells. Understanding how cancer cells can survive DNA-damaging insults such as radiation therapy will lead to more effective treatments for cancer patients and the prevention of

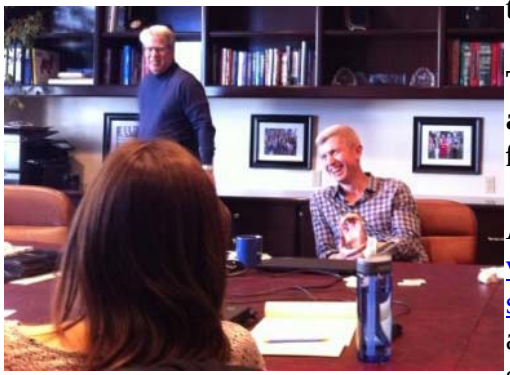
aggressive cancers that are highly resistant to radiotherapy and other treatments.

Prostate cancer cells are highly reliant on the androgen receptor (AR) protein for growth and survival. Blocking AR activity via androgen deprivation therapy (ADT) is a primary treatment against prostate cancer. Over time however, the cancer cells that remain even after ADT acquire new mutations, and patients will all too commonly suffer from recurrences with aggressive tumors that can grow even under androgen-depleted conditions. It is vitally important to more fully understand how prostate cancers grow despite AR depletion, in order to cure prostate cancer once and for all.

The primary roles of AR in prostate cancer are to turn on genes that promote growth and survival of cancer cells, and to produce proteins that are normally secreted by prostate cells, such as prostate specific antigen (PSA). Is has long been known that simultaneously blocking AR with ADT, while damaging DNA with radiation therapy, leads to a synergistic killing of prostate cancer cells that is powerful enough to even cure a fraction of patients. The effectiveness of this bilateral punch hinted that AR had yet-unknown functions in managing responses to DNA damage. With the goal of improving therapy for prostate cancer patients, both Polkinghorn and Knudsen set out to unravel this mystery.

Solving the mystery of why radiation therapy plus hormone therapy cures more men

Dr. Polkinghorn began his investigation by identifying the genes that are turned on or turned off in prostate cancer cell lines when AR is shut down with ADT. Surprisingly, he found that when AR was turned off, so was the expression of a large number of genes involved in repairing damaged DNA. Next, Polkinghorn studied the genes expressed in primary tumors from prostate cancer patients, and identified a set of 32 DNA-repair genes that appear to be directly regulated by AR. (He found 144 DNA-repair genes in total that have related but less direct connections to AR activity.)



Above: During a visit to the Prostate Cancer Foundation this winter, Dr. William Polkinghorn shares the details of his latest research with staff. Left: Dr. Polkinghorn (Left) with Dr. Soule, chief science officer, PCF.

His study then confirmed that AR is a necessary DNA-repair governor in prostate cancer cells—suppression of AR by ADT led to an accumulation of broken DNA, and when ADT was combined with radiation treatment, DNA damage was super-enhanced and many more cells died. Or in other words: the androgen receptor gooses the activity of genes that repair DNA. Lowering AR levels with hormone therapy, will fudge up the ability of cancer cells to fix their damaged DNA, leading to greater cancer cell death.

Based on these findings, Polkinghorn concluded that, "AR acts like a sunscreen, to protect prostate cancer cells from radiation. These results indicate that a subset of prostate cancer patients -- those with more sunscreen, i.e., higher levels of AR activity and consequentially higher levels of DNA repair gene expression—may preferentially benefit from the addition of ADT to radiation therapy."

The DNAPK enzyme and the androgen receptor protein function as partners in crime

At the 2014 AACR - PCF Advances in Prostate Cancer Research Conference, held in January in San Diego, CA, Dr. Knudsen presented findings from several studies including research from her *Cancer Discovery* study published alongside Polkinghorn's, in which she followed a different route to uncover AR's "sunscreen" function, as well as

exactly how AR acts as a DNA-repair governor to protect tumor cells from radiation therapy-induced DNA damage and cell death.

Knudsen's team found that DNA damage to prostate cancer cells caused by radiation actually activates AR, which then causes AR to turn on expression of a major DNA-repair enzyme, DNAPK. This enzyme in turn, enhances the activity of AR—a positive feedback circuit. Similar to what Polkinghorn observed, when Knudsen co-treated prostate cancer cells or mice with the disease, with radiation therapy plus ADT, the combination led to better killing of tumor cells than either treatment alone.

Importantly, Knudsen demonstrated that activation of the DNAPK enzyme by AR was a critical factor in the synergy between these two therapies. Radiation therapy causes DNAPK and AR to essentially amplify one another, to fix broken DNA, enabling prostate cancer cells to survive radiation therapy. Radiation plus ADT puts the breaks on this collusion, and is the reason why this combination is so much more effective in killing tumor cells than radiation alone. These studies also indicate that DNAPK itself may be an important therapeutic target.

Men whose tumors become resistant to ADT will eventually succumb to their disease. Knudsen made a very clinically important observation when she treated mice with ADT-resistant tumors: the combination of radiation and ADT led to super-enhanced tumor cell killing, while as expected, ADT had no effect on its own. Knudsen says that while ADT-resistant tumors no longer need AR for growth and survival, some still rely on AR to turn on DNA-repair programs in order to survive radiation therapy. Combining ADT with radiation therapy may be an effective treatment strategy for patients with late-stage ADT-resistant disease, whom in the past might have been considered poor candidates for such a regimen.

Intriguingly, in a study published in *Cancer Discovery* in 2012, Knudsen had discovered a similar dual-amplification paradigm between AR and another DNA-repair gene that has an infamous oncogenic role in breast and other cancers, PARP1. Polkinghorn's study identified PARP1 as a gene that is directly turned on by AR, while Knudsen's research found that PARP1 not only functions in DNA-repair, but promotes the activity of AR. According to Knudsen, "these dual functions of PARP1 [DNA-repair *and* promoting AR activity] can be leveraged to improve outcome for advanced prostate cancer." Based on these studies, Knudsen and others have initiated a series of clinical trials with PARP-inhibitors in various combinations with AR-targeting therapies. (PARP-inhibitors on their own show positive results in early results of Phase I clinical trials underway.)

At the AACR-PCF Conference, Knudsen presented additional support for the theory that aberrant DNA-repair programs contribute heavily to the development of late-stage prostate cancer. Knudsen discovered that DNAPK levels in prostate tumors predicted the subsequent development of cancer spread, or metastasis—the higher the levels of this enzyme in tumors, the more likely men were to have their cancer recur. She also observed that as prostate cancers transition into ADT-resistant states, mutations are acquired in many genes that mediate DNA repair. These mutations enhanced expression or function of DNA repair enzymes. For example, p53, a DNA-repair gene that typically acts as a tumor suppressor and is deleted in many cancers, develops unique mutations that instead appear to spur development of ADT-resistant disease. This indicates that when AR is scrubbed from prostate cancer cells by ADT, cancer cells still can survive by evolving bypass mechanisms to maintain the integrity of their genome. And an intact genome means better cell survival.

Understanding this molecular crosstalk leads to new clinical trial designs

Scientists are constantly uncovering new functions for genes that have been studied for decades. This of course is exciting

for biologists, but these discoveries are of tremendously greater importance when centered on genes critical in diseases such as cancer: new understandings invariably pave the way toward novel treatments for patients. “We need to start thinking of AR as having three functions,” says Knudsen. “One, a prostate secretory function (i.e. secretion of PSA); two, a tumor cell proliferation and survival function; and now, three, as a regulator of DNA repair and resistance to DNA damage.” Together, Polkinghorn and Knudsen’s findings indicate that this third, “sunscreen” function of AR as a DNA-repair governor, is critical to the development of drug-resistant, lethal prostate cancer.

“AR-DNA-repair crosstalk is a targetable, critical effector of disease progression,” says Knudsen. Targeting DNA-repair genes, such as PARP1, DNAPK, mutant p53 molecules, or any other of Polkinghorn’s 144 AR-associated genes, in combination with AR-inhibiting therapies and/or radiation therapy may achieve significantly greater therapeutic impact. Knudsen revealed that in addition to ongoing prostate cancer clinical trials with PARP1 inhibitors, trials with candidate agents that target DNAPK are being planned. Results from these trials are eagerly awaited.

Key Points

Two Prostate Cancer Foundation-funded researchers have uncovered the mystery of why adding a short course of androgen deprivation therapy (ADT) to radiation therapy improves outcomes.

Also, a new gene signature was discovered that may help identify which men most likely to benefit from combination radiation therapy and ADT.

In addition, an enzyme involved in repairing DNA damage (DNAPK) identified as a highly promising target for novel drug development. (Another DNA-repair enzyme PARP1, is currently in clinical trials against prostate cancer.)

The androgen receptor (AR) drives prostate cancer growth. Researchers discover “cross-talk” between AR and DNAPK enables cancer cells to better survive radiation therapy. Blocking AR with ADT interferes with this “crosstalk” during treatment with radiation therapy, resulting in increased cancer cell death.

Blocking the DNAPK enzyme may also boost cancer cell death, and researchers plan to investigate immediately. Blocking both AR and DNAPK may cure even more men undergoing radiation therapy.

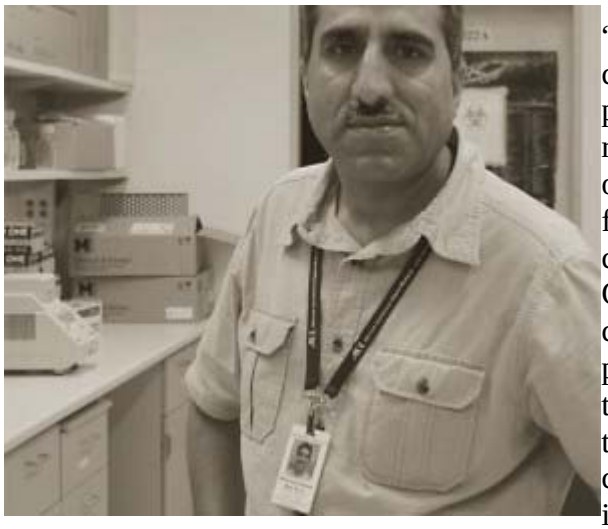
Polkinghorn WR, Parker JS, Lee MX, Kass EM, Spratt DE, Iaquinta PJ, Arora VK, Yen WF, Cai L, Zheng D, Carver BS, Chen Y, Watson PA, Shah NP, Fujisawa S, Goglia AG, Gopalan A, Hieronymus H, Wongvipat J, Scardino PT, Zelefsky MJ, Jasin M, Chaudhuri J, Powell SN, Sawyers CL. Androgen receptor signaling regulates DNA repair in prostate cancers. Cancer Discov. 2013 Nov;3(11):1245-53. doi: 10.1158/2159-8290.CD-13-0172. Epub 2013 Sep 11.

Goodwin JF, Schiewer MJ, Dean JL, Schrecengost RS, de Leeuw R, Han S, Ma T, Den RB, Dicker AP, Feng FY, Knudsen KE. A hormone-DNA repair circuit governs the response to genotoxic insult. Cancer Discov. 2013 Nov;3(11):1254-71. doi: 10.1158/2159-8290.CD-13-0108. Epub 2013 Sep 11.

Schiewer MJ1, Goodwin JF, Han S, Brenner JC, Augello MA, Dean JL, Liu F, Planck JL, Ravindranathan P, Chinnaiyan AM, McCue P, Gomella LG, Raj GV, Dicker AP, Brody JR, Pascal JM, Centenera MM, Butler LM, Tilley WD, Feng FY, Knudsen KE. Dual roles of PARP-1 promote cancer growth and progression. Cancer Discov. 2012 Dec;2(12):1134-49. doi: 10.1158/2159-8290.CD-12-0120. Epub 2012 Sep 19.

Source: Prostate Cancer Foundation http://www.pcf.org/site/c.leJRIROrEpH/b.5780289/k.D2E4/Research.htm

Institute researcher's work highlighted nationally



Department of Defense honors Dr. Saleem's cutting-edge prostate cancer research

Promising research at The Hormel Institute focused on effectively attacking a form of prostate cancer resistant to chemotherapy is being recognized nationally by the U.S. Department of Defense.

Dr. Mohammad Saleem, leader of the Molecular Chemoprevention and Therapeutics research section at The Hormel Institute, University of Minnesota had work recently featured as one of three prostate cancer research projects included in the Department of Defense's "**2014 Research Highlights**" section.

Saleem, an assistant professor who brought his research to The Hormel Institute four years ago from the University of Wisconsin, is the recipient of a New Investigator Award from the Department of Defense Prostate Cancer Research Program. Saleem and his team have provided compelling evidence that a gene known as BMI1 plays a crucial role in determining the fate of tumors treated with chemotherapy.

"I am greatly honored and encouraged to know that the Department of Defense views my research as a potential key to overcoming the molecular barrier for treating aggressive prostate cancer in men," Saleem said.

Castration-resistant prostate cancer (CRPC) is a deadly form of prostate cancer that is difficult to treat, and little is known about the reasons contributing to it, according to the DOD. Given that, identifying one or more critical molecules involved in conferring chemoresistance would be a significant advancement in developing new ways to prevent or treat that form of prostate cancer.

Recent advancements in cancer genomics have paved a way to understanding the causes and mechanisms underlying chemoresistance and the survival of prostate tumor cells during and after chemotherapy, the DOD says. Recent reports indicate involvement of several genes, including a group known as the Polycomb gene repressor family, which has been associated with several types of cancer, including prostate cancer. The emergence of BMI1 – a specific member of the Polycomb family – as a gene involved in cancer stem-cell renewal makes it an attractive target for further investigation.

Saleem and his researchers have identified a unique mechanism through which BMI1 rescues tumor cells from chemotherapy by regulating the activity and binding of the TCF4 transcription factor to the control region of BCL2, an anti-apoptotic gene. They also noticed that BMI1 is capable of driving normal cells to an over-active growth state by breaking their normal process of aging and death, suggesting that BMI1 possibly plays a vital role in breaking the sleeping mode of tumor cells and driving them toward a rapid increase in numbers.

"The fact that BMI1 expression is not influenced by androgen, which is the target of most chemotherapy treatments, further suggests that BMI1 might play a role in driving indolent disease to a more aggressive, androgen-independent disease state," according to the DOD report on Saleem's research.

Work by Saleem's section also tested the ability of BMI1 blockade to enhance the results of docetaxel treatment, a commonly used therapy that can lower PSA levels in prostate-cancer patients by more than 50 percent but is not able to completely eliminate the tumor. Researchers at The Hormel Institute showed that targeting BMI1 in chemoresistant cells sensitizes them

to docetaxel therapy, and that the success of docetaxel therapy is highly dependent on the BMI1 level.

"This suggests that targeting BMI1 should be part of a therapeutic strategy to combat chemoresistant cancer, and BMI1-specific interventions may provide opportunities to enhance the efficacy of chemotherapy in a large group of prostate cancer patients," the DOD says.

The Hormel Institute, University of Minnesota, is a world-renowned medical research center in Austin, Minn., focused on discoveries leading to the prevention and control of cancer. In July 2014, The Hormel Institute will undergo a major expansion that will about double the size of its state-of-the-art facility, adding 20 laboratories and needed space for its International Center of Research Technology. The Hormel Foundation is providing a $13.5 million match to the State of Minnesota's bonding funding for the expansion. The Hormel Foundation is committing an additional $9.5 million to recruit top scientists, equip labs with state-of-the-art technologies, and create a cutting-edge environment for progressive cancer research.

Source : CDMRP-Department of Defensehttp://cdmrp.army.mil/pcrp/

Our Clinical Trials

Memorial Sloan Kettering

In addition to providing excellent care, Memorial Sloan Kettering is constantly working to improve treatment for prostate cancer. Our clinical trials enable us to deliver state-of-the-art care, and our physicians lead a large number of trials of innovative approaches to treatment for men at all clinical states of prostate cancer. These trials test new drugs and drug combinations, surgical and radiation therapy techniques, innovative diagnostic technologies, and strategies for preserving quality of life for men undergoing treatment.

Choosing Memorial Sloan Kettering for your care may give you access to new treatment options before they are widely available elsewhere. Our experts can help to determine which clinical trials are right for you. For more information about our clinical trials, call our Physician Referral Service at 800-525-2225, or talk with your doctor.

Memorial Sloan Kettering is the coordinating center for the Prostate Cancer Clinical Trials Consortium (PCCTC), a national organization that includes 13 leading academic centers. The PCCTC is engaged in the development of new medications and new combined-modality approaches against aggressive disease that will affect lifespan or quality of life. We have long been at the forefront of bringing needed new drugs to patients quickly. Our medical oncologists have leadership positions within the consortium and other national clinical trial groups focusing on prostate cancer, and created the infrastructure for the development and evaluation of new prostate cancer drugs.

The following prostate cancer clinical trials at Memorial Sloan Kettering are currently enrolling new patients. To learn more about a study, choose from the list below. See the following URL for a list of clinical trials: http://www.mskcc.org/cancer-care/adult/prostate/clinical-trials

Source: Memorial Sloan Kettering Cancer Center

Prostate Cancer 101, Inc.
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1st	3rd
Tuesday	TBA

4:30 p.m. monthly

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Meetings are held the First Thursday of the month at the Central Hudson Auditorium on South Road in Poughkeepsie, starting at 6:30 p.m. Various doctors and speakers are on the agenda and one on one help is available after the meeting.

Contact
Paul Totta 845 297-7992
or Jim Kiseda 223-5007

If you need or want to help:

PCa 101 Seminar
First Tuesday of every month

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