Prostate Cancer Support Association of New Mexico

LIFELINE

PCSANM Quarterly July 2015 Volume 22, Issue 3

Issue Highlights

Our website address
www.pcsanm.org

e-mail
pchelp@pcsanm.org

Meeting Place:
PCSANM is meeting at Bear Canyon Senior Center, 4645 Pitt St NE in Albuquerque. This is two blocks from Montgomery and Eubank; go north one block to Lagrima de Oro St, and east one block to Pitt, and left 50 yards to the Bear Canyon parking lot. We are in room 3, at the west end of the building. Meetings are usually the first and third Saturdays of the month; from 12:30-2:45 pm.
Map: http://binged.it/1baQodz

This information comes to us from the Urology Group of New Mexico Office of Dr. Fred Snoy and Mr. Jim Taylor, 4161 Montgomery Blvd, NE, Albuquerque, NM 87109

Prostate Cancer Registry

Become part of the biomedical research community at:

AccumetRx/Urology Group of New Mexico!

Medical advancement requires participation from interested patients.

We are seeking as many people as possible to sign up for our Prostate Cancer Registry (requires only a simple phone call).

Once on the registry, you will have access to:

- **Opportunities to participate in prostate cancer research trials and surveys.**
- **Access to innovative treatment through the clinical trials.**
- **Insights into the success the medical community is having in the fight against prostate cancer.**

Your information is confidential.

Signing up for the prostate cancer registry does not commit you to becoming a participant in any study.

We have two studies that are looking for patients who have castrate-resistant prostate cancer (CRPC).

- **Study 1 is seeking patients who are castrate resistant and their cancer has not spread elsewhere.**
- **Study 2 is seeking patients who are castrate-resistant and are not responding to other castration treatments.**

If you are interested in the registry or want to find out more information about the studies, please call one of our friendly coordinators:

Jeremy at 505-872-4091, ext. 118

Heather at 505-872-4091, ext. 117

FOUNDER Rae Shipp, established 1991

Board Members

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Silver City	David Schwantes or Walt Hanson	(575) 388-2331 (575) 388-1817

In Memory of

We have no names to honor this issue.

For that, we are thankful.

Save the Date

Saturday, September 19, 2015
9:00 to 4:30
South Broadway Cultural Center

Our Annual FREE Seminar on PCA

Multiple Speakers
Confirmed as of this pub date.

Dr. Peter Lindberg

Dr. Margaret Gallegos

Dr. Paul Anthony

Dr. Larry Massie

Bernadette Goodman

PCSANM *Lifeline*

A quarterly newsletter addressing issues of prostate cancer
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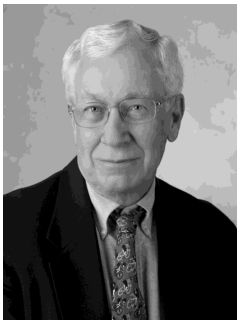
Jerry Cross, Dave Ball

MEETINGS Lou Reimer

Dr. Lindberg's Take



Dr. Peter Lindberg is accepting new patients. See below for current information.

2 MAJOR meetings with breaking news in prostate cancer, will be formally presented at ASCO, the American Society of Clinical Oncologists meeting.

A British trial of 7000 men showed an average improvement of almost one year of life when Docetaxol was added to hormone therapy in men whom surgery or radiation had failed to cure. The chemo was given for only 6 months and the hormones for up to 3 years. Exactly the form of the hormone therapy was not mentioned. PSA on average was 63 and Gleason Score was >7. A previously reported large American trial demonstrated up to 18 months improved survival in men with a heavy tumor burden (>5 bone mets, liver mets etc.) when Docetaxol was added to initial hormone treatment.

A European trial with a different set of criteria did not show chemo helping but when I listened to presentations at GU/ASCO I was convinced of the value of earlier chemotherapy. This also matches my experience in treating men with prostate cancer. As noted in my last written article, I believe "triple therapy®" as done by Robert Leibowitz is the best form of hormone treatment. Evan Yu, an outstanding researcher and clinician at the University of Washington, has been quoted as stating that chemo should be considered when starting hormone therapy, even in men who do not have metastatic disease after the usual testing with bone and CT scans.

Dr. Mark Scholz has recently written in his blog prostatesnatchers.com update about 2 under-reported complications of radical or robotic surgery for prostate cancer. At the AUA (American Urological Association) meeting, Dr. Mulholl reported that 17% of men may develop Peyronie's Disease of the penis, scarring and inflammation, which result in a distorted erection. Previously, Dr. Mulholl found in his research that after a radical prostatectomy, 20% of men when they climax or have orgasm lose urine control at exactly the same time.

Also at the AUA meeting, Dr. Hans Lilija from Memorial Sloan Kettering Hospital in New York City discussed the new 4K score and how to use this test to avoid unnecessary prostate biopsy. A high score can indicate a bad high risk cancer while a low score indicates that if cancer is found it is low risk and does not need treatment. Dr. Lilija states that if a psa test on a man sixty year of age is below 1 and if no worrisome lump is felt on rectal, maybe psa annual testing can be stopped. Also most prostate cancer deaths occur in men with a psa above 2 at some point. Then if psa is > 3.0, do the 4K test available if your clinic registers to do the test.

Also at the AUA meeting, the Prolaris test on the prostate biopsy tissue can offer guidance as to which man is a good candidate for active surveillance.

In other news, Tasquinimod, a unique agent and form of immunotherapy, after initial EXCITING results failed in a large confirmatory test of 1200 men to provide enough benefit to be moved to a standard approved treatment.

Another exciting treatment: ARN-509, similar but much more powerful than Enzalutamide (Xtandi), is now in a large trial to see if it can be given by itself to control prostate cancer. ARN-509 does NOT lower testosterone and should allow better and longer life in men with prostate cancer.

Dr. Lindberg is in practice at New Mexico Cancer Center 4901 Lang Ave NE, Albuquerque, NM 87109 Phone 505-842-8171 http://www.nmcancercenter.org/

The Future of Cancer: Closer to a Cure

By Craig B. Thompson April 26, 2015

Sloan Kettering CEO Craig Thompson on the revolution under way in cancer prevention and treatment From WSJ.com

Decades into the declared modern war on cancer, scientists and clinicians are excited by what we are learning. Yet patients and families are too often frustrated by the lack of progress in prevention and treatment.

To understand this seeming paradox, we have to consider what has been learned about the biology of cancer and how we are putting this knowledge to use.

Viewed in this light, there is tremendous hope for the future, both in decreasing an individual's lifetime risk of getting cancer and in increasing the success of treating those cancers that do arise.

Most people don't acquire a significantly higher risk of cancer from the genes that they inherit from their parents. Instead, cancer arises as a result of copying errors (mutations) in the inherited genes, as our bodies make new cells to maintain our various organs. A recent widely quoted publication suggested that these errors are an inevitable consequence of trying to copy three billion bits of information as a cell divides.

That may be true, but it doesn't mean getting cancer is inevitable. The fastest and most extensive rates of cell division occur when we are developing as embryos. Billions upon billions of cells are produced each day, yet cancer in newborns is exceedingly rare. In contrast, cell division in each of our tissues slows as we grow older, while the incidence of cancer increases with age.

We damage ourselves

What accounts for this discrepancy? One overlooked factor is that during pregnancy, both the mother and the placenta protect the developing embryo from environmental exposure. In contrast, we constantly put ourselves in harm's way as we age. We are exposed to viruses and bacteria that damage our tissues. And it isn't just invading pathogens that wreak havoc; we do most of the damage ourselves. Through sunburns, smoking, environmental pollutants and overeating, we constantly damage our tissues, forcing restorative cell proliferation to occur in a war zone of damage.

It is in this inhospitable environment that most cancers arise. We have known for some time that many of these environmental exposures damage DNA, making it harder to copy and resulting in more mutations as cells divide. Recently, we have come to appreciate that during regeneration of damaged tissue, the rest of the body pitches in to keep every cell in the damaged tissue alive.

Not just the healthy cells, but also the ones that have acquired mutations that render them unfit. Our immune system, which usually detects and destroys cells with excess mutations, is turned off. The body produces growth factors that stimulate the survival of cells with deleterious mutations, and our habit of overeating maintains an excess supply of nutrients that compounds the damage.

These are scientific facts we didn't appreciate even a decade ago. But how can this information be harnessed into better cancer treatment and prevention? Some of it is clear. Don't smoke, use sunscreen, avoid unnecessary radiation exposure, get vaccinated. Sometime this decade, it is expected that obesity, driven in large part by excess sugar intake, will surpass tobacco exposure as the No. 1 cause of preventable cancer in the U.S. Already, in terms of population health, we are putting this new scientific knowledge to use. Earlier this year, new guidelines to reduce the percentage of sugar in our diet were added.

Precision medicine

This new information is also revolutionizing cancer care. Until recently, cancer was treated based on the tissue in which it originated. Increasingly, effective cancer therapy is empowered by knowing the precise mutations a patient's tumor has acquired, independent of where in the body the cancer arose. This approach is called precision medicine.

Patients whose cancer bears specific mutations are now more effectively treated with drugs designed to selectively reverse the effects of those mutations. Such drugs are termed targeted therapeutics. The downside of this class of drugs is that they usually don't have any benefit in treating cancers that don't carry that specific mutation. While we don't yet have many therapies that target cancer-causing mutations, the results can be dramatic when such drugs are available.

We have learned that the number of mutations a cancer cell has acquired also matters. Our immune system is designed to recognize cells with new properties, as when infected with a virus. The mutations building up in a cancer cell are exactly what our immune system should respond to. The more mutations there are, the more likely it is that the immune system can recognize and destroy the cancer cells.

A recent discovery has unlocked this potential. Immunologists have found that our immune system has a built-in "off switch," a checkpoint that shuts down an immune response a few weeks after it is initiated. A new and expanding class of cancer therapeutics have been developed that have the ability to block this normal shut-off switch and thus augment the ability to recognize and destroy cells carrying mutations. In the right situation, "immunotherapy" can have stunning efficacy. Some patients with widely metastatic cancer have been rendered cancer-free with therapies aimed at increasing the body's own ability to fight cancer.

Missed signals

Our knowledge of cancer is still expanding. We have begun to appreciate that cancer is initiated primarily in the cells that endow us with the ability to repair our tissues. When a tissue is damaged, so-called progenitor cells expand in number to fill the damaged area, and once that is accomplished, the cells differentiate to form tissue much like the original. Some of the mutations discovered in cancer cells result in the signal to differentiate not being received or acted upon. As a result, progenitor cells keep on reproducing.

Treatments that focus on restoring a cell's ability to differentiate, thus stopping the excessive proliferation, are already being used successfully in the clinic. These therapies avoid the toxic side effects of traditional chemotherapy and can effectively eliminate cancer cells even when they have spread throughout the body.

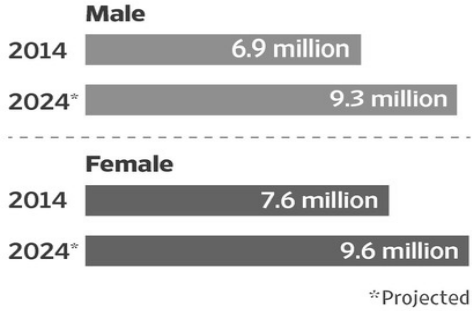
Why is finding a cure for cancer taking so long? A major reason lies in the fact that cancer is not one disease, but many. Each tissue has its own unique progenitor cells, and each tissue uses only a subset of the genes we inherit from our parents; each tissue is exposed to environmental insults differently. We are just beginning to understand the interplay of all these factors in the origin of the many forms of cancer. Understanding these issues will ultimately allow us to optimize the treatment approach to each patient's disease.

While we aren't yet ready to put cancer on the extinction list along with "simpler" diseases like smallpox and polio, it is clear that with more science—the lessons learned from cancer research over the past two decades—we face the future with less fear.

Dr. Thompson is president and chief executive officer of Memorial Sloan Kettering Cancer Center in New York. He can be reached at reports@wsj.com

Promise in the War on Cancer

The number of cancer survivors in the U.S.



The number of survivors in the U.S. for the most common cancers

Male	2014	2024*
Prostate	3.0 mil.	4.2 mil.
Colon and rectum	621,430	789,950
Melanoma	516,570	698,040
Urinary bladder	455,520	577,780
Non-Hodgkin lymphoma	297,820	390,170

Female	2014	2024*
Breast	3.1 mil.	4.0 mil.
Uterine	624,890	756,980
Colon and rectum	624,340	771,070
Melanoma	528,860	696,280
Thyroid	470,020	645,330

*Projected

Note: A cancer survivor is anyone who has been diagnosed with cancer, from the time of diagnosis through the balance of life.
Source: National Cancer Institute via American Cancer Society

THE WALL STREET JOURNAL.

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The Cancer Center
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Phone 505-559-6100

Cleveland Clinic now offers Online Prostate Cancer second opinions

Recognizing the challenges surrounding the arrangement of travel, time off of work and time away from family, the My**Consult** program offers patients an easy, effective, and secure option for obtaining a second opinion from our medical experts without having to leave the comfort of home.

Our physician experts provide comprehensive, individualized second opinion reports that include:

A thorough review of the patients' medical records, history and test results
Answers to questions submitted by the patient
Treatment options or alternatives
Recommendations regarding future care needs

A registered nurse from the My**Consult** Clinical Support Team is available to answer questions or help walk patients through the process. To contact the My**Consult** Clinical Support Team, read patient success stories, or to begin the process of getting an online medical second opinion, visit eclevelandclinic.org/myconsult

As you know, there are few decisions in life as important as those that impact your health. Seeking an expert second opinion from Cleveland Clinic through My**Consult** can provide additional education and awareness, confidence, and peace of mind that your members are making the right choices for their health care.

Our team is available to provide webinars outlining the process of obtaining a second opinion through MyConsult.

Phone
1.216.444.3223
1.800.223.2273 ext 43223
Email
eClevelandClinic@ccf.org

Hours
Monday - Friday
8:00 a.m. - 5:00 p.m. EST , Excluding national holidays

Editor note: Our Board Chairman, Lou, has this to add: It is not yet covered by insurances and costs $745, including a rereading of your biopsy score. You would need to evaluate if this is something you might want to consider. We have some of their printed materials in our office.

Prostate Cancer: The Price of Fitness?

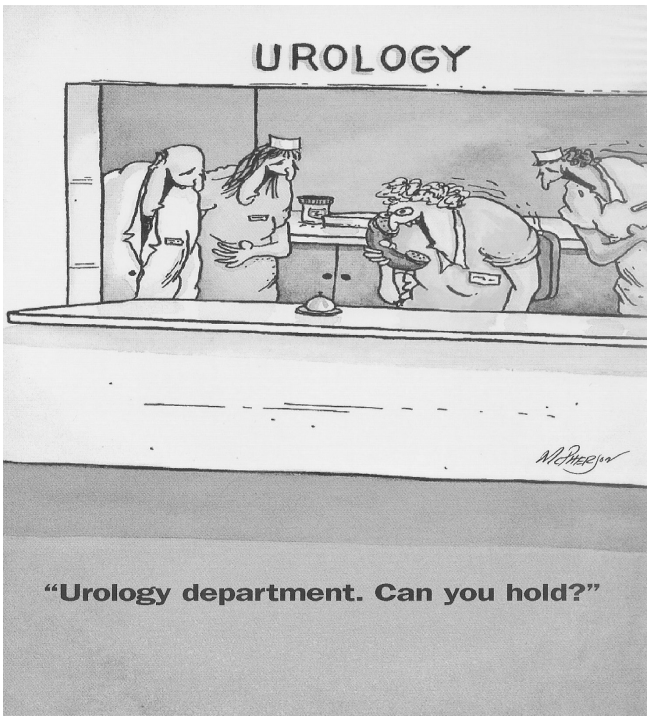
The men in the best shape had significantly lower rates of lung and colorectal cancer but, surprisingly, had higher rates of prostate cancer.

Although physical fitness is widely known to prevent cardiovascular disease, its impact on cancer is less clear.

In an observational cohort study published in *JAMA Oncology* (2015; published online ahead of print) nearly 14,000 men underwent a treadmill-based fitness assessment and were then observed for an average 6.5 years. The men in the best shape had significantly lower rates of lung and colorectal cancer but, surprisingly, had higher rates of prostate cancer (PCa)—even after adjusting for several potentially confounding variables.

The researchers hypothesized that physically fit men were more likely to undergo screening tests, presumably resulting in PCa over diagnosis. Fortunately, the fittest men also had the lowest overall cancer and cardiovascular mortality, so even if exercise did somehow contribute to PCa risk, its benefit still prevails.

By Jaime Landman, MD and Christopher R.Kelly, MD May 2015 Issue of RenalAndUrologyNews.com



This Prostate Cancer Treatment May Impair Thinking

Men with a particular gene mutation suffered most, study finds

Men undergoing hormone therapy to treat prostate cancer may experience impaired mental function within the first six months that persists for at least a year, a new study suggests.

Moreover, the risk of memory, learning and concentration problems associated with hormone therapy was greatest for men with a particular gene mutation, researchers from the University of South Florida in Tampa found.
Hormone therapy is used to lower the level of testosterone, thus preventing growth of prostate cancer cells.

"There is something about the treatment that seems to be associated with worse mental function," said lead researcher Brian Gonzalez, a postdoctoral fellow at the Moffitt Cancer Center in Tampa.

But the association seen in the study does not prove a cause-and-effect relationship.

For the study, Gonzalez's team evaluated 58 prostate cancer patients before they began hormone therapy and six months and 12 months later. The investigators compared them with 84 men who had their prostate gland surgically removed and 88 men without prostate cancer.

Fuzzy mental functioning was worse for men receiving hormone-depletion therapy. But men with the gene mutation rs1047776 were 14 times more likely to have mental problems related to hormone therapy than men without this mutation, Gonzalez said.

"Men who are considering hormone therapy for prostate cancer should be aware of the possible mental side effects," Gonzalez said.

Gonzalez suspects altering testosterone levels might cause thinking impairments. But men on hormone therapy also experience fatigue and depression, which might affect their mental abilities, too, he said.

The report was published online May 11 in the *Journal of Clinical Oncology*.

Other experts aren't so sure about the extent of impairment -- or whether hormone therapy is even warranted for prostate cancer patients.

"This is a small study . . . that needs to be verified," said Dr. Anthony D'Amico, chief of radiation oncology at Brigham and Women's Hospital in Boston, who had no role in the study.

What is needed, he said, is a randomized study that compares similar men receiving hormone therapy with those not receiving this treatment. In addition, the duration of hormone therapy needs to remain constant, he said. D'Amico does believe, however, that a biological reason exists for thinking and memory problems among men on hormone therapy.

He pointed to an earlier study that found long-term hormone therapy affected men's mathematical ability. "There is a basis for it. I don't discount it. I believe that these problems are probably true, but not to the extent reported in the current study," D'Amico said.

Current practice is to give hormone therapy for only a short time, he said. "I am not positive that short-course hormone therapy has any impact on mental function. I am fairly convinced that long-term therapy does," D'Amico said.

Also, whether mental function returns to normal after therapy ends isn't known, he said. "There are lot of questions that need to be raised," D'Amico added.

Dr. David Samadi, chairman of urology and chief of robotic surgery at Lenox Hill Hospital in New York City, doesn't think men with prostate cancer should have hormone therapy.

"Hormone therapy is absolutely not necessary," he said. "Not only do you have to worry about the side effects of fatigue, male menopause and depression but also heart problems," Samadi said.

"And what's not good for the heart is also not good for the brain," he said. "These mental issues are another reason to walk away from hormone therapy."

Samadi believes that with state-of-the-art surgery, men can have their prostate gland removed and not suffer from incontinence or sexual side effects.

WebMD News from HealthDay By Steven Reinberg
From WebMD.com May 12, 2015

Role of Genetic Mutations in Metastatic Castrate-Resistant Prostate Cancer Treatment

By Andrea S. Blevins Primeau, PhD, MBA April 23, 2015 from CancerTherapyAdvisor.com

A summary of sessions related to genetic mutations in metastatic castrate-resistant prostate cancer at the 2014 AACR annual meeting.

Genetic mutations are a major driver for the development and progression of metastatic castrate-resistant prostate cancer (mCRPC) and were the topic of discussion at the "Metastatic Prostate Cancer Precision Medicine: Challenges and Solutions" session at the 2014 American Association for Cancer Research (AACR) Annual Meeting.

High Frequency of Mutations in mCRPC

Over 90% of sequenced specimens in a multicenter cohort of men with mCRPC had clinically actionable genetic mutations, according to Arul M. Chinnaiyan, MD, PhD, of the University of Michigan in Ann Arbor, MI.[1]

As expected, 60% were located within the androgen receptor (AR); however, mutations were also detected in PI3 kinase, DNA repair modulators such as *BCRA1* and *BRCA2* (>20%), *BRAF*, the cell cycle pathway (20%), Wnt pathway members (18%), and those of the germline (8% to 10%).

"Germline mutations suggested that these patients could benefit from genetic counseling," said Dr. Chinnaiyan and mutations in Wnt pathway members were particularly intriguing because "this is suggestive that these patients may actually be susceptible to porcupine or hedgehog inhibitors."

"Probably the most compelling and clinically actionable findings from this initial cohort were actually in the DNA repair pathway," highlighted Dr. Chinnaiyan. Both somatic and germline mutations in the DNA repair pathway were identified, and the highest rate of mutations was located in the *BRCA2* gene, with mutations also identified in *BRCA1* and *ATM*, as well as in other genes.

"This was quite intriguing because this suggests that these patients may be potentially susceptible to PARP inhibitors, especially with the high frequency of BRCA and ATM inactivation."

AR Variants as a Clinical Target

Genetic mutations in the AR occur frequently and tend to be specific to mCRPC. In addition, there are multiple splice variants of AR. "The good news is, all you only need to focus on, in my opinion, is AR-V7," said Jun Luo, PhD, of Johns Hopkins Medical Institutions in Baltimore, MD.[2]

Developed by Luo's group, a simple, rapid blood test combined with reverse transcriptase polymerase chain reaction (RT-PCR) can identify AR-V7 in a variety of different settings, such as in patients receiving different types of therapy.

According to Dr. Luo, AR-V7 expression levels can be similar to that of the full-length transcript of the AR (AR-FL), and in vitro data from the VCaP and LNCaP95 cell lines suggest that AR-V7 does not form heterodimers with AR-FL.

The importance of AR-V7 detection has recently been demonstrated in a study published last year in the *New England Journal of Medicine*,[3] in which poorer prostate-specific antigen (PSA) response and progression-free survival were associated with AR-V7 positivity in patients with mCRPC. In addition, patients who were initially AR-V7-negative and became positive for AR-V7 demonstrated outcomes between those of patients who were positive or negative for the variant transcript.

"If you detect AR-V7 before therapy, you can actually predict that patient's response to abiraterone and enzalutamide," Dr. Luo said. There was a 68% PSA response rate in patients who were AR-V7-positive compared with 0% in patients who were AR-V7-negative.[3]

"It is certainly good that we have multiple therapies, all of which have therapeutic benefit, but on the other hand, we have a problem here because you don't know which one to choose, how to sequence, when to start, when to stop. All of these decisions are difficult for clinicians," stated Dr. Luo.

Therefore, a predictive marker would be clinically valuable. AR-V7 may have predictive value for response to abiraterone and enzalutamide.

"I do think it's possible that there are additional variants out there and we could modify the test to detect them," stated Dr. Luo.

Role of DNA Repair Pathways

In addition to AR variants, aberrant expression of other genes have been demonstrated to be associated with mCRPC, but not hormone-sensitive prostate cancer, such as PARP1 and DNAPK.[4]

PARP1 regulates AR function, and in an animal model system, PARP inhibitors suppress AR-dependent or regulated genes.[5]

Indeed, single-agent treatment of explants from a radical prostatectomy by a PARP1 inhibitor resulted in decreased cell proliferation and survival. Yet, a phase 1 study that evaluated the PARP1 inhibitor niraparib demonstrated that the antitumor response occurred regardless of the genetic mutation.

Another molecule that appears to be important in mCRPC is DNAPK, according to Karen E. Knudsen, PhD, of the Sidney Kimmel Cancer Center in Philadelphia, PA. Dr. Knudsen's recent work found that in men with mCRPC, "DNAPK is the top deregulated kinase in this cohort, indicating to us that DNAPK is highly enriched in these tumors."

Studies have demonstrated that AR activation promotes double-stranded DNA break repair, and the AR binds to the *DNAPK* gene in response to DNA damage.

As a result, Dr. Knudsen proposed that the AR promotes the upregulation and activation of DNAPK, resulting in repair of double-stranded DNA—leading to tumor cell survival. In addition, DNAPK induces the transcription of genes involved in metastatic signaling, cell migration, and cell invasion.

However, AR-independent in vitro studies demonstrated that suppression of DNAPK resulted in a reduction of cell migration and invasion.

Furthermore, pretreatment of mice with the DNAPK inhibitor NU7441 did not decrease tumor growth, but did decrease the formation of metastases. In another model, treatment of AR-positive tumors with a DNPK inhibitor significantly decreased the occurrence of lung metastases (P=0.0227), but did not affect primary tumor growth.

In a cohort of men with mCRPC, "those tumors that were identified as having high levels of DNAPK were associated with a significantly worse prognosis, if you look at freedom from recurrence, freedom from metastasis, or overall survival," Dr. Knudsen said.

Improved Trial Design Needed

"Genomic studies embedded in early clinical trials decrease the risk of drug development and accelerate successful pivotal studies," stated Johan S. De Bono, MB, ChB, PhD, of the Institute of Cancer Research in Sutton, United Kingdom.[6]

One way to improve clinical trial design is to use an adaptive design for phase 2 trials, such as the TOPARP trial, which evaluated olaparib in mCRPC. "What we did in TOPARP was hypothesize from the outset that PARP inhibition would work in a subset of patients with prostate cancer," Dr. De Bono said.

In the TOPARP trial, patients who demonstrated a high response rate to olaparib were randomly assigned as part of an unselected population. Patients who demonstrated an intermediate response defined as 10% to 50% were assigned according to a biomarker-guided selection.

In the TOPARP trial, the role of predictive biomarkers was evaluated in heavily pretreated patients with end-stage prostate cancer who received olaparib. In the single-agent trial, complete response, stable disease, partial response, and progressive disease were observed, as well as a decrease in PSA levels. Overall, the response rate was 30%, and patients with mutations in BRCA2 or ATM demonstrated a greater sensitivity to olaparib.

Dr. De Bono stated this drug design "has allowed us to prove that this drug has activity in the overall prostate cancer population, and we can now focus on the biomarker-selected patients and a targeted administration approach."

Quality of Life Outcomes Differ Among Patients with Low-Grade Prostate Cancer

Jason Hoffman, PharmD, RPh April 10, 2015
from CancerTherapyAdvisor.com

Urinary and sexual health-related quality of life outcomes were worse for low-grade prostate cancer patients who underwent radical prostatectomy.

No differences in mental health outcomes were observed between patients with low-risk prostate cancer who received active surveillance or radical prostatectomy, but urinary and sexual health-related quality of life (HRQoL) outcomes were worse for patients who underwent radical prostatectomy for up to 3 years, a new prospective study published online early in the journal *Cancer* has shown.

For the study, researchers sought to longitudinally compare HRQoL in a prospective, contemporary, and racially diverse cohort of patients with low-risk prostate cancer who underwent radical prostatectomy or active surveillance.

Researchers surveyed 288 patients no older than 75 years of age with low-risk prostate cancer who underwent radical prostatectomy and 77 who underwent active surveillance for initial disease management. Participants were followed for 3 years.

Results showed that patients in the surgery group had significantly worse sexual function, sexual bother, and urinary function compared with patients in the active surveillance group; however, there were no significant differences in mental health outcomes between groups for 3 years.

Ask Dr. Myers

If you have not looked at Dr. Charles (Snuffy) Myers videos once in a while, you are really missing out on some very good information.

They can be found at https://askdrmyers.wordpress.com/

He puts a new one up every few days.

On the May 11 post, he talks about aging with prostate cancer.
The April 28 was about Cancer drug Interactions.

They are free, and you can even sign up for email notifications at https://www.prostateforum.com/

His Facebook page is at https://www.facebook.com/

Our Affiliate organization, **Cancer Support Now**, will be hosting their 6th annual Survivor and Caregiver Picnic on Sunday August 9, at Elena Gallegos Picnic Area Kiwanis Shelter, making it a rainproof event. Free food, games, entertainment, and activities for the whole family. Watch for our email or look for the flyer on their website http://www.cancersupportnow.org/ in order to sign up.

From pcf.org glossary of terms Differences in clinical trials

phase I trial: The first step in testing a new treatment in humans. These studies test the best way to give a new treatment (for example, by mouth, intravenous infusion, or injection) and the best dose. The dose is usually increased a little at a time in order to find the highest dose that does not cause harmful side effects. Because little is known about the possible risks and benefits of the treatments being tested, phase I trials usually include only a small number of patients who have not been helped by other treatments.
phase II trial: A study to test whether a new treatment has an anticancer effect (for example, whether it shrinks a tumor or improves blood test results) and whether it works against a certain type of cancer.
phase III trial: A study to compare the results of people taking a new treatment with the results of people taking the standard treatment (for example, which group has better survival rates or fewer side effects). In most cases, studies move into phase III only after a treatment seems to work in phases I and II. Phase III trials may include hundreds of people.
phase IV trial: After a treatment has been approved and is being marketed, it is studied in a phase IV trial to evaluate side effects that were not apparent in the phase III trial. Thousands of people are involved in a phase IV trial.

Advanced Viral Gene Therapy eradicates prostate cancer in preclinical experiments

From ScienceDaily.com May 11, 2015 from Virginia Commonwealth University

Even with the best available treatments, the median survival of patients with metastatic, hormone-refractory prostate cancer is only two to three years. Driven by the need for more effective therapies for these patients, researchers at VCU Massey Cancer Center and the VCU Institute of Molecular Medicine (VIMM) have developed a unique approach that uses microscopic gas bubbles to deliver directly to the cancer a viral gene therapy in combination with an experimental drug that targets a specific gene driving the cancer's growth.

Recently published in the journal ***Oncotarget***, this new study is the most recent in a long line of studies led by Paul B. Fisher, M.Ph., Ph.D., investigating the use of viral gene therapy to treat a variety of cancers. The treatment strategy uses a novel "cancer terminator virus" (CTV), which replicates exclusively in cancer cells delivering the cancer-specific, toxic cytokine gene mda-7/IL-24 directly to the tumor. The researchers added an experimental drug known as BI-97D6, which targets MCL-1 and other members of the Bcl-2 gene family that protect cancer cells from therapeutic agents, resulting in enhanced prostate cancer cell death while sparing healthy prostate epithelial cells in preclinical experiments involving advanced mouse models of prostate cancer. The therapy not only killed cells at the primary tumor site, but also in distant metastases by "bystander" antitumor activity driven by the secreted MDA-7/IL-24 protein.

"We are at a point in our research where we have validated the efficacy of this combination treatment approach in preclinical animal models, and we now need to define its safety through toxicology and pharmacology studies," says Fisher, Thelma Newmeyer Corman Endowed Chair in Cancer Research and co-leader of the Cancer Molecular Genetics research program at VCU Massey, chairman of VCU School of Medicine's Department of Human and Molecular Genetics and director of the VCU Institute of Molecular Medicine. "We are hopeful that this research will culminate in the development of a phase 1 clinical trial that will test the safety of this novel approach and potentially lead to an effective new therapy for advanced prostate cancer."

When viruses attack their hosts, they introduce their genetic material into the host cell. This process essentially hijacks the cell in order to produce more copies of the virus. The CTV used in the study, Ad.tCCN1-CTV-m7, is a modified adenovirus--the kind of virus that typically causes mild respiratory infections. The scientists removed the genes controlling viral replication and that cause disease, and they added part of the controlling element of a gene known as CCN1 to cause the virus to replicate selectively in cancer cells. The scientists then engineered the virus to deliver the tumor-suppressing and -cell-death inducing gene mda-7/IL-24 into the cancer cells, generating a CTV. As the CTV continues to replicate, it causes the cells to produce and secrete mda-7/IL-24.

The mda-7/IL-24 gene was originally discovered by Fisher, who showed in previous studies that it prevents tumor growth and inhibits tumor blood vessel formation, promotes anti-tumor immune effects and stimulates a form of cell suicide known as apoptosis. The gene has also been shown to synergize with other cancer treatments. In the present study, the scientists demonstrated that the drug BI-97D6 increased cancer cell death caused by mda-7/IL-24, and it also helped defend against resistance to the viral gene therapy.

Critical to the therapy is the stealth delivery technique known as ultrasound-targeted microbubble destruction (UTMD). If injected directly into the bloodstream by itself, the CTV may get trapped in the liver or be removed by the body's immune system. UTMD uses microscopic, gas-filled bubbles that can be paired with viral therapies, therapeutic genes and proteins, and imaging agents. The bubbles are released in a site-and target-specific manner via ultrasound, and, with appropriate modification of the therapeutic virus, can be imaged in real-time to track the delivery of the CTV to the tumor. Fisher and his colleagues are pioneering this approach and have already reported success in preclinical experiments utilizing UTMD technology and mda-7/IL-24 gene therapy in prostate and colorectal cancer models. UTMD has also been used elsewhere in clinical trials testing therapies for patients with heart disease.

"This approach holds promise for the treatment of many different cancers ," says Fisher. "Our team is collaborating with researchers at Massey and at other institutions to move this research forward. We even plan to open a phase 1 clinical trial next year testing a different CTV expressing mda-7/IL-24 in patients with recurrent brain cancer."

"Age, fitness and body weight have a far bigger effect on survival than PSA, Gleason score, or any type of treatment. The diagnosis of prostate cancer should be taken as a helpful wake-up call encouraging men to improve their diet and start exercising." Dr. Michael Kattan (medical statistician, nomogram developer, now with MSK); as published in the Journal of Urology, Jan-06

PCSANM *Lifeline* Newsletter
July 2015

Prostate Cancer Support Association
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Chairman's Message, April 2015
September is Men's Health and Prostate Cancer Month

Your Chairman is pleased to see that PCSANM continues to help both the newly diagnosed and continuing patient by being a clearinghouse for prostate cancer information. We remind our members that we exist to supply information about this disease and are a place for patients to share their experiences and successes with treatments.

As part of our efforts to meet these objectives, PCSANM will hold our fourth annual conference on the diagnosis and treatment of prostate cancer on Saturday, September 19, 2015. It will be held at the South Broadway Cultural Center/Library from 9 am to 4:30 pm. This date was chosen because it coincides with men's health month and prostate cancer month.

We will have speakers on the new topics of laser ablation, MRI as a diagnostic tool, and genetic testing for staging of prostate cancer. We will have a review of prostate cancer staging and how the pathologist develops the Gleason score. Wrapping up the conference, we will have breakout sessions during which you can talk with various physicians regarding any particular concerns about your diagnosis or treatment. I urge you to take advantage of this program to learn more about prostate cancer.

Confirmed speakers so far:

Dr. Margaret Gallegos on Use of MRI in locating tumor;
Dr. Peter Lindberg on Advanced PCa treatments;
Dr. Larry Massie on Pathology review/Gleason
Dr. Paul Anthony on Radiation;
Bernadette Goodman on Laser Ablation treatment;
And a speaker from Genomic Health on Genetic Testing;
And more speakers we are awaiting confirmation on.

I wish all our members good health and well-being.


Lou Reimer Chairman of the Board