



# Newsletter

## Prostate Cancer 101, Inc.

<http://prostatecancer101.org>  
**November, 2006**

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

### Our November 21 Guest Speaker is Dr. Elizabeth Tapen

### Genetic code of cancers mapped

By Jonathan Bor Sun reporter

Mark your calendar for Tuesday, November 21<sup>st</sup>, 4:30 p.m., at the Hurley Reformed Church, Main St., Hurley, NY.

This is the date we are having Dr. Elizabeth Tapen,



Medical Director Ulster Radiology Oncology Center at the Benedictine Hospital speaking about What

is Currently Available for Prostate Cancer and also What is Planned for the Future. She will include a discussion of IMRT and IGRT (Image Guided Radiation Therapy) and the parameters used for treatment decisions.

Prior to her present position Dr. Tapen worked in San Jose and Berkley, California at their cancer institutes and

has experience in prostate brachytherapy, specializing in HDR (temporary high dose radiation). A native of Paterson, NJ, she obtained her undergraduate education from Rutgers University in New Brunswick, NJ in 1990 and her graduate education at Robert Wood Johnson Medical School in Camden, NJ. She is Board Certified in Radiation Oncology and did her residency in St. Mary's Hospital, San Francisco, CA. Dr. Tapen has participated in Investigator Initiated Trials and has published for the American Journal of Clinical Oncology. At present she serves as a Board Member for the American Cancer Society in Kingston, NY.

Please make every effort to attend this informative meeting.

In a step that could lead to new ways of detecting and treating cancer, Johns Hopkins researchers reported yesterday that they had deciphered the genetic code of breast and colon tumors.

The discovery, described in the online version of the journal Science, is the first time anyone has spelled out the complete genetic makeup - or genome - of a human cancer. In doing so, the scientists identified close to 200 genes whose mutations play a role in the formation and spread of the disease.

Dr. Bert Vogelstein, one of three Hopkins University researchers who spearheaded the effort, said the discovery gives cancer researchers precise targets for new drugs and screening tests. Some in the field

said the study might have opened a new era in cancer research, showing scientists the methods to crack the genomes of other malignancies as well.

"It's like viewing the enemy's game plan," said Vogelstein, a professor of oncology, adding that the team will next turn its attention to mapping brain and pancreatic cancers.

"There are numerous ways the information can be exploited. The first is viewing the enemy, seeing what its weak points are and formulating an attack plan."

Also leading the effort were Dr. Kenneth W. Kinzler and Dr. Victor E. Velculescu. Together, the three head a lab that is widely regarded as one of the most fertile in cancer research.

The report comes less than a year after the National Institutes of Health announced a three-year, \$100 million project to begin mapping cancer's genetic underpinnings. The agency plans to announce two "milestones" in the effort next week.

Though it dovetails with the federal program, the Hopkins project was conducted independently and largely with private money. Still, federal officials lauded the Hopkins program, saying it demonstrates the feasibility of the larger effort.

"We anticipate that as [the federal program] scales up, we may be able to identify the majority of genetic changes that cause the most important and common forms of the major cancers," Dr. Francis Collins, director of the National Human Genome Research Institute, said in a written statement.

"In fact, the large number of mutations reported in this paper offers a glimpse of what is yet to come and provides exciting new directions for drug discovery in breast and colon cancer."

In both a historical and practical sense, the Hopkins study was an outgrowth of federal and private programs that, by 2003, mapped the composition of the approximately 20,000 genes that make up each human cell.

Genes contain the blueprint for the proteins that make up every tissue in the body.

Each cell in the body contains the same complement of genes, though some acquire mistakes - or mutations - that give rise to improvements or abnormalities. Unlike normal cells that die and are replaced, a cancer cell lives on, multiplying into a tumor that can spread to other parts of the body.

The Hopkins scientists studied 11 breast tumors and 11 colon tumors of patients who had undergone

treatment.

To identify the genes that had gone awry, they determined the chemical sequence of the tumors' DNA - and compared that information with the sequence of normal cells. The differences led scientists to identify the cancer genes.

Specifically, the team discovered 69 colon cancer genes and 122 breast cancer genes. Only two altered genes appeared both in colon and breast tumors.

To become cancerous, a cell needs to accumulate only some of these mutations. Vogelstein said the precise number, though not yet known, could be in the neighborhood of 20.

Though some people might inherit genes that predispose them to cancer, most mutations are acquired during a person's lifetime, he said. Mutations can be caused by diet or environmental influences.

"From the first mutation to a full-blown malignancy, it takes literally 20 to 40 years," said Vogelstein, noting that only a small number of cancers occur during childhood. "This data could explain why it takes so long."

One colon cancer patient, for instance, might have accumu-

lated 20 of the 69 altered genes that are known to play a role in the disease. Another patient might have accumulated a similar number, with only some overlap between that patient and the other. The differences could explain why cancers that seem alike behave differently in different patients.

Dr. Kurtis E. Bachman, a breast cancer specialist from the University of Maryland Greenebaum Cancer Center who collaborated in the study, said some mutations might figure into the early formation of a tumor, while others might trigger tumor growth.

Still others could influence the ability of tumor cells to break off, enter the bloodstream and invade other parts of the body.

"Some may be very instrumental in cancer being resistant to therapy," Bachman said. "We have cases where tumors look the same, but one person will respond to therapy while another won't. This opens up the possibility of explaining why they respond differently."

Earlier studies have led to the identification of a relative handful of cancer genes. Those discoveries stemmed from a cruder method - so-called linkage studies - in which scientists find mutations shared by family members who have developed the same type of cancer.

Dr. Anna D. Barker, deputy director of the National Cancer Institute, noted that those studies have already yielded drugs such as Herceptin for breast cancer and Gleevec for certain types of leukemia and gastrointestinal cancer.

The Hopkins study and others like it will expand the number of targets for new drugs that could be more effective and less toxic than those now available, she said.

As the era of tumor mapping unfolds, scientists will have to learn how the genes function before they develop strategies of taming the disease. But Vogelstein said the answers could yield ways to detect cancer before it causes symptoms, as well as new drugs and vaccines that target the genes behind the cancer.

Though the large number of genes found in breast and colon tumors points to the disease's complexity, scientists said many of the genes might work in common pathways. Rather than targeting specific genes, a dizzying task given their sheer number, researchers hoping to fashion new drugs might be able to target the smaller number of shared pathways.

The federal government's plan

to develop a Cancer Genome Atlas attracted general praise when it was announced in December, but some criticism too. Some scientists said the project, with its hefty price tag, could drain resources at a time when the federal cancer research budget is shrinking.

But Dr. Stephen J. Elledge, a Harvard cancer researcher who co-authored a critical letter last year to *Science*, said he was encouraged by the Hopkins study because on a smaller scale it demonstrated the effort is feasible.

"It's a very well done study, very impressive, done about as well as you could do it," he said.

Elledge, however, said he hopes the government doesn't pour money into the sequencing of cancer genes at the expense of efforts to figure out how the genes function.

In the meantime, he said, it's too early to predict when, if ever, gene-mapping discoveries will yield practical results.

"Without functional studies, you can't know what the significance of these mutations really is," he said. "In 10 years, 15 years from now, we'll know a lot more."

*Source: jonathan.bor@baltsun.com*

## Thank you for your Contributions

Kenneth Adin  
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## Life-Threatening Prostate Cancer Detectable by Measuring PSA Velocity During a "Window of Curability"

November 1, 2006. How fast the amount of PSA (prostate-specific antigen) in a man's blood increases is an accurate gauge of tumor aggression and danger, according to new research from Johns Hopkins University School of Medicine. Even when PSA levels are so low as to not warrant a biopsy, a fast rate of rise (high PSA velocity or PSAV) is a marker of aggressive disease.

Findings of this Hopkins study of PSAV, in this month's Journal of the National Cancer Institute, may add a new level of predictive accuracy to prostate cancer testing, the value of which has remained controversial under currently accepted guidelines, the investigators say.

"Our data provide a further argument for PSA testing that begins relatively early in life, when PSA levels are usually lower and prostate enlargement is not a confounding factor in diagnosis," says H. Ballentine Carter, M.D., the director of the Johns Hopkins Division of Adult Urology at the Brady Urological Institute

and lead author of the study.

"We would recommend that men at around age 40, not 50, have their PSA checked to develop a baseline against which to compare future changes (velocity), since even a slight rise in PSA may indicate a potential for cancer down the road," Carter said.

An estimated 234,460 men in the United States will be diagnosed with prostate cancer this year, according to the American Cancer Society.

"The main debate over how to use PSA has centered on the choice of the level that is used to trigger a biopsy," says Carter, a professor at the Johns Hopkins University School of Medicine. "Lowering the level that triggers a biopsy leads to detection of more harmless cancers, and higher levels could miss the opportunity to detect an important cancer early. We have found that the rate at which a man's PSA rises may be more important than any absolute level for identifying men who will develop life-threatening cancer

while their disease is still curable. In addition, PSA velocity could be a useful method for identifying those men with a prostate cancer that could be safely monitored - an approach termed 'active surveillance'."

PSA is a protein found in the bloodstream of men, produced by the prostate gland and found at increased levels in those with prostate cancer. In previous research, PSA velocity in the year before prostate cancer diagnosis has been shown to identify men who are likely not to be cured by surgery. However, Carter's latest findings show that PSA velocity can also identify men with life-threatening disease at a time when it is still curable.

Using serum samples dating as far back as 1958, frozen as part of an ongoing randomized health study of men, Carter and his team determined PSA velocity in 980 of those study participants (856 without prostate cancer, 104 with the disease and 20 who died from it) up until May of 2005. They found that the PSA velocity determined at a time when PSA levels would not have triggered a biopsy were predictive of death from prostate cancer 20 to 30 years later.

Those men whose PSA velocity was lower had a 92 percent chance of not dying of prostate cancer 25 years later; whereas

those with a higher PSA velocity had a 54 percent chance of not dying of prostate cancer. The rates of prostate cancer death were 1,240 in 100,000 for subjects with a higher velocity compared to 140 in 100,000 for those with lower velocities.

Carter emphasizes that an important difference between the current research and previous studies is that the subjects in the current study were not selected, but rather taken at random from a large, ongoing study, thus more accurately representing the U.S. population.

His research was supported by the Intramural Research Program of the National Institutes of Health, National Institute on Aging.

## **Sources and links:**

### **[Detection of Life-Threatening Prostate Cancer With Prostate-Specific Antigen Velocity During a Window of Curability](#)**

H. Ballentine Carter ,  
Luigi Ferrucci , Anna  
Kettermann , Patricia  
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*Journal of the National Cancer Institute, November 1, 2006.*

David C. Miller, John T. Wei, Rodney L. Dunn, James E. Montie, Hector Pimentel, Howard M. Sandler, P. William McLaughlin and Martin G. S a n d a .  
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*Source: psa rising*

## Long-Term Outcomes For Prostate Cancer Show IMRT Curative: 89 Percent Disease-free 8 Years Later

Results from the largest study of men with prostate cancer treated with high-dose, intensity modulated radiation therapy (IMRT) show that the majority of patients remain alive with no evidence of disease after an average follow-up period of eight years. The 561 prostate cancer patients treated with IMRT at Memorial Sloan-Kettering Cancer Center were classified into prognostic risk groups.

After an average of eight years, 89 percent of the men in the favorable risk group were disease-free and none of the men in any group developed secondary cancers as a result of the radiation therapy. This report, published in the October 2006 issue of *The Journal of Urology*, is the first description of long-term outcomes for prostate cancer patients using IMRT.

"Our results suggest that IMRT should be the treatment of choice for delivering high-dose, external beam radiotherapy for patients with localized prostate cancer," said Dr. Michael J. Zelefsky, Chief of the Brachytherapy Service at Memorial Sloan-Kettering. "We were able to

show long-term safety and long-term efficacy in a very diverse group of prostate cancer patients that we followed - many for as long as ten years. Despite the fact that some patients had an aggressive form of their disease with high Gleason scores and PSA (prostate specific antigen) levels, the overwhelming majority of patients had good tumor control with neither recurrence of their original cancer nor development of second cancers, which one might have expected from the high doses of radiation," he added. Pre-treatment diagnostic evaluations were performed for all of the patients to better define their clinically localized prostate cancer. They were classified into prognostic risk groups as defined by the National Comprehensive Cancer Network guidelines ([www.nccn.org](http://www.nccn.org)).

These are based on clinical characteristics including age, T stage, Gleason score, PSA level, and pre-treatment with neoadjuvant androgen deprivation.

Between April 1996 and January 2000, 561 patients with a median age of 68 (ranging from 46 to 86 years old) were treated with

IMRT, an improved form of three-dimensional conformal radiation therapy (3D-CRT), also used in radiotherapy. IMRT uses enhanced planning treatment software that more precisely targets the prostate, allowing the beam of radiation to deliver a high dose (81 Gy) to the tumor target while sparing the adjacent bladder and rectum from exposure to the higher amounts of radiation. Perhaps because of this, the eight-year results show urinary continence was maintained for all patients, and only 1.6 percent of the five hundred sixty-one patients experienced rectal bleeding. The high-dose radiotherapy was curative for the majority of the patients in all three prognostic risk groups, with 89 percent of the favorable, 78 percent of the intermediate, and 67 percent of the unfavorable group alive after an average period of eight years. Of those men who were potent prior to IMRT, erectile dysfunction developed in 49 %.

"This study confirms that we can improve patients' quality of life by reducing the side effects

# Banishing Blind Spots

Robert Langreth 11.13.06

of radiotherapy while maintaining disease-free survival," said Dr. Zelefsky. "However, there is still room for improvement. We are incorporating image-guided approaches that may continue the excellent tumor control but further limit the area we are irradiating and reduce side-effects."

The study's co-authors are Heather Chan, Margie Hunt, Yoshiya Yamada, MD, Alison M. Shippy, and Howard Amols, PhD, of Memorial Sloan-Kettering.

Memorial Sloan-Kettering Cancer Center is the world's oldest and largest institution devoted to prevention, patient care, research, and education in cancer. Our scientists and clinicians generate innovative approaches to better understand, diagnose, and treat cancer.

Our specialists are leaders in biomedical research, and in translating the latest research to advance the standard of cancer care worldwide. Contact: Joanne Nicholas  
[Memorial Sloan-Kettering Cancer Center](#)

Article URL :  
<http://www.medicalnewstoday.com/medicalnews.php?newsid=52907>

Source: Medical News Today

## Genetic breakthroughs and an unlikely drug offer hope for a disease ravaging the eyes of an aging population

By age 75, one in three Americans shows early signs of the disease. A grayish blotch expands ever so slowly in the center of the victim's vision. Sufferers may have to turn their head or get up close to the subject to see clearly. In the disease's end stages, abnormal blood vessels form in the back of the eye and start to leak fluid, rapidly causing blindness.

The disease, macular degeneration, has become a modern scourge of an aging population. By 2020, 3 million Americans will have advanced cases, up from not quite 2 million now. "We are outliving our eyes," says Johns Hopkins ophthalmologist Julia Haller. "Otherwise healthy people are losing their independence because they cannot read or drive."

Until very recently little was known about what caused the disease, and treatments were few and far between. Its hallmark is yellowish deposits of debris called drusen that grow at the back of the eye, damag-

ing the macula, the tiny but crucial section of the retina responsible for central vision.

But in the last year and a half giant strides have been made on both fronts. Two breakthrough drugs from Genentech offer hope for patients with the more severe "wet" form of age-related macular degeneration, or AMD. For the more common "dry" form a series of genetic discoveries have offered the first good clues to its causes, providing leads for new drugs and genetic diagnostic tests.

At least 18 companies are now pursuing macular degeneration treatments, including stem-cell treatments, anti-inflammatory medicines and implants that slowly release protective proteins to stave off cell damage. "When I started out in the ophthalmology business 20 years ago, you barely heard anything about macular degeneration," says Neurotech Pharmaceuticals Chief Executive Edward Danse. "Now the whole world is on fire for drugs for macular degeneration."

Doctors are most excited so far

about the two Genentech drugs. Lucentis, approved in June for wet AMD, is essentially a fragment of the monoclonal antibody that was used to make the company's colon cancer drug Avastin. Lucentis halts blood-vessel growth when injected into the side of the eye. In two large-scale trials it stopped vision loss in 95% of patients, and improved vision in one-third--results that a *New England Journal of Medicine* editorial deemed "miraculous." The downside of Lucentis is its cost: \$1,950 per monthly dose.

Many doctors are turning to Avastin itself to treat wet AMD. The cancer drug has never been rigorously tested in patients with macular degeneration, but in uncontrolled trials it appeared to work well. And Avastin costs only \$50 or so for a small ocular dose. Darwin Holian, a retired surgeon in Santa Barbara, Calif., first noticed a big blurry spot in his left eye in 2004. After other treatments failed, his doctor injected a single dose of Avastin into the eye in July 2005. "Four days later it was a miracle. I had almost a 100% return of vision," he says. He has needed only one additional Avastin shot since then. Noting Lucentis' stiff price, he adds: "How they can justify that is beyond me."

Genentech Chief Medical Officer Hal V. Barron argues that patients

would be wise to stick with Lucentis, since insurance usually covers all but \$50 of the cost. Avastin degrades more slowly inside the body and might cause more side effects. "Until something is proven to be absolutely equivalent or better, you shouldn't mess around with something as important as vision," he says.

The company has no plans to compare the two drugs. To resolve the debate, the federal National Eye Institute plans a giant Avastin-versus-Lucentis head-to-head trial. But results aren't expected for years. Meanwhile, various firms such as Acuity Pharmaceuticals and Sirna Therapeutics are testing blood-vessel blockers for the eye that might not need to be given as frequently as Lucentis, limiting unpleasant eye injections.

An even bigger long-term need is to find a treatment that could help millions of patients with the far more common dry form of the disease. This one often precedes the wet form, so the goal is to devise pills, drops or other treatments that could be used at the earliest sign of trouble. One proven risk reducer is a blend of high doses of vitamins C and E, beta carotene and zinc. A 4,757-patient government study found that people at high risk who took the vitamins reduced their chance

of getting advanced forms of the disease by 25%. Quitting smoking and eating a healthy diet may also reduce risk.

In the meantime a handful of treatments for the dry form are in clinical trials. Neurotech Pharmaceuticals is testing a tiny polymer capsule that is implanted in the back of the eye and continuously pumps out a nourishing protein for six months or longer. The hope is that this will prevent crucial light-sensing "cones" inside the retina from degenerating. In a test on ten patients with retinitis pigmentosa, an inherited disease involving retinal degeneration, vision improved significantly. A trial in 48 patients with advanced dry amd should start soon.

The ultimate treatment might be using healthy stem cells to replace damaged cells in the macula. StemCell Ventures is working with the University College London's Institute of Ophthalmology to devise a patch containing adult-eye stem cells that could be implanted in the eye in hopes of restoring vision. In rats with inherited eye defects "it works perfectly," says Roger Ashby, the firm's chief executive. Stem-cell trials in humans are roughly three years off.

Until recently researchers had few molecular clues on how to improve AMD treatments. But the

## NCI Releases Preliminary Data on Genetic Susceptibility for Prostate Cancer

landscape has shifted significantly with the discovery of several common genes that dramatically raise the risk of the disease. The mutated form of a gene called complement factor H may cause the immune system to go awry, resulting in too much inflammation in the eye. That may be a cause of macular degeneration. Roughly one-third of the genes scanned in a recent study had this mutation. People who have one or more copies of the bad gene have at least double the risk of the disease. Early efforts are under way at specialty pharmaceutical firm Allergan and elsewhere to develop anti-inflammatory treatments for the back of the eye.

A second gene is implicated in both the wet and dry forms and is intriguing because it codes for a type of enzyme called a protease; this class has proven very amenable to drug development. People who are unlucky enough to inherit the bad version of both genes have a thirtyfold higher risk of macular degeneration, says University of Utah geneticist Kang Zhang, who helped nail down the second gene after Yale University researchers first spotted it. "Absolutely, it is going to be a great target for the next generation of macular degeneration drugs," says Zhang.

*Source: Forbes 11/13/06*

The National Cancer Institute (NCI), part of the National Institutes of Health, has released new data from the Cancer Genetic Markers of Susceptibility (CGEMS) study on prostate cancer.

This information could help identify genetic factors that influence the disease and will be integral to the discovery and development of new, targeted therapies. This is also the first public release of a whole genome association study of cancer -- such studies examine the entire genome, with no assumptions about which genetic alterations cause cancer.

"Knowing which genes are most likely to lead to cancer will greatly enhance our ability to diagnosis the disease at its earliest stages, as well as develop therapies to treat cancer when it is most vulnerable to attack," said NCI Director John E. Niederhuber, M.D.

Launched in February 2006, CGEMS is the largest comprehensive initiative to identify genetic risk factors for breast and prostate cancers, which are two of the most frequently diagnosed cancers in the United States. By finding genetic variations that differ in frequency between patient and control groups, researchers can identify the location of multiple inherited genes that increase or decrease the risk of prostate

cancer.

"The immediate sharing of this data with scientists in the cancer research community will allow individual researchers to compare existing and developing information with CGEMS data to identify new genes associated with increased prostate cancer risk," said NCI Deputy Director for Advanced Technologies and Strategic Partnerships Anna D. Barker, Ph.D. "The CGEMS database will provide information we need to develop new strategies for the early detection and prevention of a cancer that takes the lives of nearly 27,000 American men each year."

The genetic samples of prostate cancer came from more than 1,100 men with the disease and 1,100 men who have not developed prostate cancer. The samples include more than 680 million individual genotypes, or genetic markers, including 310,000 genetic variants.

"CGEMS represents one of the first of a new generation of studies made possible by the Human Genome Project," added Gilles Thomas, M.D., Ph.D., lead scientist of CGEMS. "Through immediate sharing of data, we hope to encourage other teams to make similar studies in cancer and other diseases rapidly accessible to speed progress in understanding the inherited causes of cancer."

Prostate cancer is the third-leading

## Protoxin trial for recurrent Prostate Cancer

cause of cancer-related death in men. In 2006, there will be an estimated 234,460 new prostate cancer cases in the United States.

"NCI is leveraging its resources to make this valuable dataset immediately accessible to all interested scientists," said Stephen Chanock, M.D., director of the NCI Core Genetics Facility and co-director of CGEMS.

Similar data on breast cancer, the second-leading cause of cancer-related death in women, are now being generated and are anticipated to be released in early 2007. When the data is released, the CGEMS database will contain close to 2.5 billion genotypes. All data from the CGEMS study will be available through NCI's caBIG™ (Cancer Biomedical Informatics Grid™), at <http://caIntegrator.nci.nih.gov/cgems/>.

"CGEMS represents the best of collaborative science, with geneticists and epidemiologists pooling resources to make publicly available the first database of inherited genetic variants associated with a major cancer," said David Hunter, M.D., an NCI Eminent Scholar who is co-director of CGEMS and professor of cancer prevention at the Harvard School of Public Health.

For more information on NCI's Cancer Genetic Markers of Susceptibility (CGEMS) initiative, please visit

<http://cgems.cancer.gov>.

Protoxin, also called PRX302, is a drug being tested on men **with localized recurrent prostate cancer** in Phase I clinical trials in Texas and Vermont.

Patients considering this trial must have experienced recurrence after completing a full course of definitive external beam radiation or definitive brachytherapy (but not both) as primary therapy for diagnosed prostate cancer at least one year prior to enrollment

Our main news about Protoxin is here:

<http://www.psa-rising.com/prostatecancer/protoxin1106.htm>

A reader asked for a simpler, clearer explanation of what's going on with this drug. Here's what we know at the moment:

In Texas, one trial enrolled the first patients in May this year and is hoping to recruit 36 men with localized recurrent prostate cancer. Patients must have recurred after EBR or brachytherapy, have PSA level less than 20 ng/mL and PSA doubling time longer than 3 months.

They must NOT be taking hormone drugs. Also, they must NOT have signs of metastatic disease including no bone metastases on bone scan, or any lymph node, lung, liver or soft tissue.

A link to info about that trial is here:

<http://clinicaltrials.gov/ct/gui/show/NCT00379561?order=2>

The 3 cancer centers so far are:

1) Scott and White Cancer Center, Temple, Texas:

[http://www.sw.org/sw/portal/\\_pagr/105/\\_pa.105/161](http://www.sw.org/sw/portal/_pagr/105/_pa.105/161)

2) M. D. Anderson Cancer Center

3) University of Vermont (contact Dr. Mark Plante).

If you think you would want to be in this trial take this info to your doctor.

This Phase I trial aims to find out:

- 1)whether the drug is safe
- 2)whether it has side effects
- 3)whether it beats back the cancer.

So far they have reached the third "dose level" in testing the drug in the Temple, TX trial, which began in May and are moving forward to recruit more patients there and at

the 2 other centers.

What they know so far is that PRX302 is activated (switched on) by PSA. It reduces the size of a prostate cancer tumor by destroying cancer cells. It does this by making a hole (or pore) in the cell membrane (the surface covering each prostate cancer cell).

PRX302 is injected into the prostate under ultrasound guidance.

PRX302 is expected to have fewer side effects than traditional or current systemic treatments because it is injected directly into the prostate.

The high level of PSA being produced by prostate cancer cells switches on the drug, the drug forms pores on the cell's surface, and the cancer cells die.

This is the plan. Whether it will really work on men with localized recurrent prostate cancer, and whether it will keep working permanently or for a long time, no one will know for sure till the trial is further along and/or completed.

If it works reasonably well they will take it to a Phase II and then a Phase III clinical trial. Ultimately this might create a whole new treatment for men with early stage localized prostate cancer.

Another Phase I trial testing Protonix for BPH (benign overgrowth of the prostate) is under way in Vancouver, Canada.

## Study Shows Men With Erectile Dysfunction Favor Treatment With Vardenafil

Research published in The Journal of Sexual Medicine and presented at the 12th World Congress of the International Society for Sexual Medicine in Cairo, Egypt is unique in that the data is from a head to head trial of PDE5 inhibitors used to treat patients with [erectile dysfunction](#) (ED), designed to minimize bias toward either study drug.

The study was a randomized, double-[blind](#), crossover, head-to-head clinical trial that compared vardenafil and [sildenafil](#) treatment in men with ED and diabetes, [hypertension](#), and/or hyperlipidemia. The results demonstrated that vardenafil achieved nominal statistical superiority over sildenafil for several frequently used efficacy measures, and non-inferiority of vardenafil to sildenafil as measured by various assessments of patient satisfaction and patient preference.

A total of 1,057 men participated in the study, which involved treatment using each drug for four weeks, with a one-week washout period in between. Patients were asked: "Overall, which medication do you prefer?" along with 11 other preference questions relating to their ED treatment. Additional efficacy assessments using established scales were also used in analysis.

Data showed that 38.9% preferred vardenafil compared to 34.5%

sildenafil (26.6% had no preference). Vardenafil was significantly superior to sildenafil in terms of [erectile](#) function, intercourse satisfaction and overall satisfaction. There were also a significant higher percentage of positive responses for vardenafil with regards to erection hardness for penetration, maintenance of erection, maintenance until completion, and erection confidence.

"This study represents an important step forward in our understanding of the clinical differences between PDE5 inhibitors, confirming the efficacy of vardenafil for men with erectile dysfunction," explains Irwin Goldstein, study co-author and Editor-in-Chief of The Journal of Sexual Medicine.

There are currently three PDE5 inhibitors available to treat ED: sildenafil, tadalafil and vardenafil, all of which have previously demonstrated efficacy and tolerability in a range of patient populations, according to researchers.

Data from head-to-head [clinical trials](#), like this one, are scarce. However, results from studies such as this should help clinicians to differentiate among sildenafil, vardenafil, and tadalafil and to select the most appropriate for individual patients.

This study is published in The Journal of Sexual Medicine. The

*Source:psa rising*

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Source: Science Daily

**If you need or want to help:**

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