Newsletter

Prostate Cancer 101, Inc.

http://prostatecancer101.org

April, 2009

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

Come meet and hear Jillian Quinn, Radio Show Host, Author, Speaker. Tuesday, April 21 at 5:00 pm Hurley Reformed Church

The Secrets of the Bulletproof Spirit

How to Bounce Back from Life's Hardest Hits. Come meet and hear Jillian Quinn,, Author, Radio Show Host, Speaker. Tuesday, April 21 at 5:00 pm at the Hurley Reformed Church

We are delighted and fortunate to have Jillian Quinn come share a prescription with us on how to learn to think in a new way to help overcome the adversities that visit us. No one is exempt, but a negative response to trials can keep us stuck in grief, anger and pain. Come open the door and allow yourself to respond with ease and grace and increase your emotional resilience.

This is not just a talk for cancer survivors, but for all who journey along the path called "Life" with its trials, tribulations, joys, sorrows and questions. Prepare to lift your spirits and don't forget to invite a friend who may just need a little more resilience in their life.

In talking with Jillian over the past few months, I have found her to be a positive, joy filled and spiritual person. She is certainly a woman who lifts the level of consciousness when she is in a room or in a conversation. I, for one, can't wait to meet her in person and share her insights and story.

Jillian lives in Dutchess County with her husband and three young children. She is a radio host on WHVW 950AM and is a seasoned speaker, teacher and workshop facilitator, author and all around delightful person.

She is also an Associate Minister at the Interfaith Temple in New York City. In other words – she's a genuine and multi-faceted person.

Copies of her new book – ***The Secrets of the Bulletproof Spirit*** will be available for sale and maybe she'll even sign one for you. You can find her websites at www.jillianquinn.com and www.bulletproofspirit.com

Be there – April 21, Tuesday at 5:00pm. We'll be looking for you!

Diane Sutkowski

Weight Gain Triggers BPH

Benign prostatic hyperplasia (BPH) is the most common benign (noncancerous) growth process in men. About one in four men experiences BPH-related symptoms by age 55; by age 75, half of men have BPH symptoms.

What triggers BPH is not well understood, but aging and testosterone (the predominant male sex hormone) are believed to be the primary influences on its development. Animal studies suggest that the female sex hormone estrogen (produced in small amounts in men) also may play a role, perhaps when a man's testosterone production declines and the balance of the two hormones is altered.

Now there are new insights as to the causes of BPH. A study reported in The Journal of Urology (Volume 177, page 1395) found that men who are overweight -- especially those with excess fat around the middle -- are more likely to develop BPH than men of normal weight.

Researchers examined the risk of BPH among 5,667 men age 55 or older who were in the placebo group of the Prostate Cancer Prevention Trial. They considered potential risk factors such as race, ethnicity, body mass index, and waist circumference. The researchers assessed the men for the development of BPH annually for seven years.

The study results revealed that the risk of developing BPH increased 4% for each additional year of age and that black and Hispanic men had a 41% higher risk of BPH than did white men. BPH risk was higher for all men with a body mass index greater than 25 (the cutoff point defining "overweight"), with the increased risk ranging from 13-29%. Men with abdominal obesity -- defined as a waist-to-hip ratio (waist measurement divided by hip measurement) of 0.95 or greater -- were also at higher risk. For men with a ratio of 1.05 or higher, the risk of developing severe BPH was increased by 45%.

Although the study was not designed to address the impact of weight loss on BPH, these findings suggest that if you're overweight, you might be able to reduce your risk by dropping excess pounds.

Posted in Enlarged Prostate on March 31, 2009

Source: Johns Hopkins Medicine HEALTH ALERTS

Thank you all for your Contributions

Howard & Gisa Adriance
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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
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Notices to Membership

1. Membership Lists – If you no longer want to be on the mailing list or you do not want your name on the distributed list which we give to the newly diagnosed so they can talk to men who have taken the journey before them, you must notify Diane Sutkowski by mail or email. Updates on doctors with whom you have had additional treatment would be helpful too.

2.New Mailing and Street address – If you move or change your email address, you must notify Diane Sutkowski if you want to continue receiving newsletters. All too often we waste postage when newsletters are returned due to the expiration of the forwarding time. So ***if you want to receive the newsletter, let us know where you are!***

3. DVD's of most prior lectures – contact Yavuz Birturk if there is any program you missed that you would like to see. You will have to pay postage and mailing.

4. Officers and Board of Directors – If you have any interest in serving on the Board or as an officer, please contact any of the current members serving. Help is needed with programs, advertising, newsletter and other administrative areas. Can't you manage to give even an hour of your time?

Officers and Directors of Prostate Cancer 101, Inc.

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Family History of Prostate Cancer Does Not Affect Some Treatment Outcomes

American Society for Therapeutic Radiology and Oncology | 01.02.2009

In a first of its kind study, a first-degree family history of prostate cancer has no impact on the treatment outcomes of prostate cancer patients treated with brachytherapy (also called seed implants), and patients with this type of family history have clinical and pathologic characteristics similar to men with no family history at all, according to a January 1 study in the International Journal of Radiation Oncology*Biology*Physics, the official journal of the American Society for Radiation Oncology.

"This information is relevant for both physicians and patients with new diagnoses as they embark on complex treatment decisions," Christopher A. Peters, M.D., lead author of the study and a radiation oncologist at Northeast Radiation Oncology Center in Dunmore, Pa. (chief resident at Mount Sinai School of Medicine at the time of the study), said. "Now patients with a family history of prostate cancer can be confident that they have the same outcomes as patients with sporadic disease, regardless of the treatment modality they chose."

According to the American Cancer Society, prostate cancer is the most common cancer in men behind skin cancer. Many patients diagnosed with prostate cancer have some type of family history of the disease and men with a family history do have an increased risk of developing the disease, but there is conflicting data on how family history impacts treatment outcomes.

In the study, researchers at the Departments of Radiation Oncology and Urology at the Mount Sinai School of Medicine in New York sought to determine if having a familial history of prostate cancer, which is defined as a clustering of prostate cancer cases within a family, had an impact on the prognosis of men treated with brachytherapy for clinically localized prostate cancer patients.

Researchers followed 1,738 prostate cancer patients, of which 187 had a family history of prostate cancer in a first-degree relative, for a median follow-up time of 60 months. They found that in the low-, intermediate- and high-risk groups, a family history of prostate cancer had little to no prognostic significance in men treated with brachytherapy. Previous studies done with prostate cancer patients receiving external beam radiation therapy or radical prostatectomy had similar findings.

ASTRO is the largest radiation oncology society in the world, with more than 10,000 members who specialize in treating patients with radiation therapies. As the leading organization in radiation oncology, biology and physics, the Society is dedicated to improving patient care through education, clinical practice, advancement of science and advocacy. For more information on radiation therapy, visit http://www.rtanswers.org/. To learn more about ASTRO, visit http://www.astro.org/.

Demand For All Inclusive Prostate Cancer Care Treatment Centers In High Demand Among Urologists Who Diagnose Prostate Cancers On
Press Release: Oncology Med, Inc. | 01.09.2009

Source: Zero-The Project to End Prostate Cancer

Folic Acid Doubles Risk of Prostate Cancer

A study led by researchers at the University of Southern California (USC) found that men who took a daily folic acid supplement of 1 mg daily had more than twice the risk of prostate cancer compared with men who took a placebo.

The finding came from a secondary analysis of the Aspirin/Folate Polyp Prevention Study (AFPP), a placebo-controlled randomized trial to determine the impact of aspirin and folic acid on colon polyps in men and women who were at high risk for the disease. The results appear in the March 10 online issue of the Journal of the National Cancer Institute.

Folic acid (folate) is a B vitamin found in many vegetables, beans, fruits and whole grains. While evidence of its ability to reduce neural tube defects in infants while taken by the mother before or during pregnancy has been well documented, its effects on other conditions are unclear.

"We know that adequate folate levels are important in the prevention of several cancer types, cardiovascular and neurological diseases," says lead author Jane Figueiredo, Ph.D., assistant professor of preventive medicine at the Keck School of Medicine of USC. "However, little has been known about its role in prostate cancer. Our objective was to investigate the relationship between folic acid supplements and dietary folate and risk of prostate cancer."

The AFPP study was conducted between 1994 and 2006 and found that aspirin reduced the risk of colon polyps while folic acid had a negative effect and increased the risk of advanced and multiple polyps. The first analysis did not address the impact of folic acid supplements on prostate cancer risk. Previous observational studies have been inconsistent. Some studies suggest that increased folate in the diet or in supplements might actually lower the risk of prostate cancer, and others have suggested no effect or even a potential harmful effect.

In the secondary analysis, researchers looked at prostate cancer incidence among 643 men who were randomly assigned to 1 mg daily folic acid supplements or placebo in the AFPP study and who enrolled in an extended follow-up study. The estimated prostate cancer risk was 9.7 percent at 10 years in men assigned to folate, compared with 3.3 percent in men assigned to placebo.

By contrast, dietary folate intake and plasma folate showed a trend toward reduced risk of prostate cancer, although the difference did not reach statistical significance. It remains unclear why dietary and circulating folate among non-multivitamin users may be inversely associated with risk, Figueiredo says.

"The synthetic form of folate, folic acid, found in supplements, is more bioavailable compared to folate from dietary sources and we know the amount of folate available is critical," she says. "Adequate levels of folate may be beneficial, but too much folate is unlikely to be beneficial."

Alternatively, these results may be due to chance, and replication by other studies is needed, she notes.

"These findings highlight the potentially complex role of folate in prostate cancer. The possibility of different effects from folic acid-containing supplements versus natural sources of folate definitely merits further investigation."

The study was supported by the National Cancer Institute and the National Institutes of Health.

Source:NewsMax.com

Bisphosphonate Side Effects and a New Clinical Trial

By Jaquie Strax

Recent stories on bisphosphonate side effects might be signaling the advent of a new, superior drug, but will Halozyme's rHuPH20 enzyme solve the problem of jaw necrosis?

Drug development companies operate within the overall consumer culture. We all want better drugs, better everything. Generic Fosamax (alendronate) now costs just $4 at Wal-Mart, Kroger and other retail pharmacies. What might make right now a better than usual time to get word out to the masses that Fosamax carries some dreadful, if quite rare, risks?

As we just reported, U.S. Food and Drug Administration official Diane Wysowski reveals in the current New England Journal of Medicine that the FDA has received 23 reports of esophageal cancer possibly linked to Fosamax between its October 1995 debut and May 2008. Of these patients, according to her report, so far 8 have died. Is there any ulterior reason in the fact that the FDA is publicizing this risk just now? Is anything better on the horizon?

Checking our good friend SEARCH, indeed there is. Just last month, December 2008, a Texas company, Healthcare Discoveries, began recruiting patients for a Phase I clinical trial of a new, subcutaneously injectable bisphosphonate drug made by Halozyme Therapeutics. Halozyme's bisphosphonate uses an enzyme called rHuPH20 (recombinant human hyaluronidase PH20). Searching "bisphosphonate" at clinicaltrials.gov, brings up rHuPH20 top of a list of 195 bisphosphonate trials.

Halozyme Therapeutics's immediate need as of December is for 72 people at risk for osteoporosis, who have not taken a bisphosphonate in the past 6 months and are willing to take a new type of osteoporosis drug. Some of these people would receive bisphosphonate alone, and some would receive it with rHuPH20. The company reported results of preclinical animal studies of rHuPH20 in April 2008.

Let's step back a moment from cynicism — not a good spirit in which to start a new year. Or maybe turn it around. The question is not just why is FDA publicizing 23 cases of esophageal cancer now. It's, what took them so long to report this as a rare side effect of Fosamax? And how sure can they be that just 23 people developed this cancer out of the millions who take Fosamax or other oral drugs in the same family (Boniva and Actonel)?

In May 2004, Salvatore Ruggiero, DMD, MD, reported 63 cases of another serious side effect of bisphosphonates, necrosis of the jaw. Dr. Ruggiero has continued to work on this problem since. In an article scheduled for publication January 9, he writes:

Bisphosphonate therapy has been considered standard therapy in the management and care of cancer patients with metastatic bone disease and patients with osteoporosis. The efficacy of these drugs is due to their ability to inhibit osteoclast-mediated bone resorption. However, the postmarketing experience with intravenous and, to a much lesser extent, oral bisphosphonates has raised concerns about potential side effects related to profound bone remodeling inhibition and osteonecrosis isolated to the jaws. We review the risk factors, incidence, pathogenesis, prevention strategies, and management of this new complication.

Every new drug development brings a ray of hope and a whole new set of questions. Halozyme's enyzme treated bisphosphonate is designed, as an injectable, to bypass 2 obstacles to patients' treatment with this class of drugs: risk from taking the pill form and inconvenience or hardship of reaching a center to receive infusion. The company writes:

Bisphosphonates are a class of molecules that bind to mineralized bone matrix and inhibit bone resorption. Currently, there

are oral and intravenous bisphosphonates. Oral Bisphosphonates often cause gastrointestinal side effects and require a cumbersome dosing regimen. The gastrointestinal side effects of oral bisphosphonates is a significant cause of patient noncompliance to prescribed therapy. Certain bisphosphonates are indicated for the treatment of osteoporosis and skeletal metastases, but can only be administered today by intravenous infusion. As such, patients often have to travel to an infusion center or see a specialist to receive their intravenous bisphosphonate infusion. Subcutaneous injections of bisphosphonates are not considered feasible due to injection site toxicity in the skin and/or impractical injection volumes.

The recombinant protein, rHuPH20, is a human hyaluronidase enzyme that increases the dispersion and systemic absorption of locally injected drugs by temporarily degrading hyaluronan under the skin.

This new injectable format will likely completely resolve the problem of gastrointestinal effects and also any risk of cancer of the espohagus. The injection will not irritate or involve any part of the digestive tract. But whether this investigational drug will help in any way with the other problem, of bisphosphonates' anti-healing effects on injured bone (such as the jaw after dental extraction) remains to be seen.

Source:psa rising

Generic Casodex: Tentative US Approval

On December 24, 2008 the US Food & Drug Administration granted tentative approval to Accord Healthcare's bicalutamide.

Accord is a subsidiary of Intas Pharmaceuticals, Ltd., an Indian company. Accord's function is licensing and marketing.

According to the FDA, this is the definition of "tentative approval":

"If a generic drug product is ready for approval before the expiration of any patents or exclusivities accorded to the reference listed drug product, FDA issues a tentative approval letter to the applicant. The tentative approval letter details the circumstances associated with the tentative approval. FDA delays final approval of the generic drug product until all patent or exclusivity issues have been resolved. A tentative approval does not allow the applicant to market the generic drug product."

According to drugpatentwatch.com, AstraZeneca's Casodex was approved in the US Oct 4, 1995. The patent expires Apr 1, 2009.

Source: psa rising

'I . . . feel like a man again'

By Judie Foreman

Manny Hamelburg, 68, a retired businessman from Holbrook, had fought prostate cancer for years. First he tried radiation, then a drug with side effects that nearly killed him, and finally Lupron, a drug that blocks production of testosterone, the hormone that can fuel prostate cancer.

The cancer disappeared. But life was miserable. Without normal levels of testosterone, Hamelburg said he had no energy, and "zero libido for seven years. I was like a eunuch. I was chemically castrated. Sex was just hugs."

So three years ago, with his cancer undetectable and his oncologist and urologist cautiously on board, Hamelburg made a decision that many doctors consider anathema: He took testosterone supplements.

So far, says Hamelburg, "The cancer hasn't come back, but my libido has, my sense of being alive. It's like a fog cleared. It's being aware of things, being more vibrant."

For decades, the idea of giving testosterone to a man who had

had prostate cancer was forbidden - "verboten" in the words of Hamelburg's urologist, Dr. Abraham Morgentaler of Beth Israel Deaconess Medical Center.

"It would have been considered heresy, or malpractice," says Morgentaler.

But that thinking is changing, due in part to Morgentaler, and his new book, "Testosterone for Life." Morgentaler argues that, while depriving tumors of testosterone does make them shrink, other evidence is beginning to suggest that it may be safe to give testosterone to men who have been successfully treated for prostate cancer, and appear to be cancer free.

One revolutionary aspect of Morgentaler's theory is the observation that prostate cancer is often found in men with low testosterone levels, not high ones, underscoring the idea that taking it may not be an added risk.

It's not surprising that Morgentaler - who has received honoraria and research funding from companies selling testosterone-related products - has generated controversy with his ideas.

"To say that testosterone replacement therapy is safe because we have no evidence it's harmful is making an assertion on faith, not facts," said Dr. Ian Thompson, chairman of the department of urology at the University of Texas Health Science Center at San Antonio, echoing the view of other doctors who disagree with Morgentaler.

But amid often-confusing testosterone research results, there are hints that Morgentaler and like-minded physicians may be on to something. In the test tube, prostate cancer cells have been shown to grow faster when testosterone is added, but only up to a point. Then the growth plateaus, even if more testosterone is added.

In 2006, Morgentaler co-wrote a study on 345 men with low testosterone. The study - published in the journal Urology and not industry funded - showed prostate cancer risk was higher in men with the lowest testosterone, a finding supported by a handful of other small-scale studies using human subjects. That was contrary to findings suggested by the Physicians' Health Study in 1996, a discrepancy doctors can not fully explain.

And last February, an analysis of data from 18 studies around the world involving nearly 4,000 men with prostate cancer, and more than 6,000 without, showed no correlation between high testosterone levels and cancer risk. The study was published in the Journal of the National Cancer Institute.

Understanding the pros and cons of testosterone replacement is not easy.

An estimated 2 million to 6 million American men have low testosterone, and the benefits of replacement therapy can be huge: revival of sagging libido, better mood, more energy, more muscle mass, better bone density, more red blood cells.

But there are also risks, in large part because many seemingly healthy men have undetected prostate cancer, which could be stimulated by taking testosterone. Indeed, studies suggest prostate cancer is lurking in as many as 25 percent or more of men 50 and older.

Only when a man has a "clean" biopsy - an invasive procedure in which snippets of the prostate are surgically removed and tested - can a doctor confidently say the man doesn't have cancer.

As an extra measure of safety, Morgentaler says he biopsies men over 50 before he prescribes

testosterone for them. But most doctors don't, says Dr. Marc Garnick, a cancer specialist at Beth Israel Deaconess Medical Center and editor in chief of Harvard Medical School's publication, Perspectives on Prostate Disease.

Even with apparently healthy men, "Nobody has proven that it is completely safe" to give testosterone," says Dr. Philip Kantoff, head of the Prostate Cancer Program at Dana-Farber Cancer Institute.

So what's a guy to do?

The traditional recommendations are to steer clear of testosterone supplementation if you have prostate or breast cancer; or if you meet one of several criteria: your physician can feel a nodule on the prostate during a digital rectal exam; your PSA (a marker of potential cancer) score is higher than 3 nanograms per deciliter; your hematocrit (red blood cell count) is greater than 50 percent; you have untreated sleep apnea, severe urinary tract symptoms or heart failure.

These standards are set by the Endocrine Society, a professional group of doctors who study and treat patients with hormones.

And if you have had prostate cancer that appears to be gone?

Proceed with caution. "Most physicians consider testosterone replacement therapy contraindicated for men with a history of prostate cancer," says Dr. Matthew Smith, director of genitourinary medical oncology at Massachusetts General Hospital Cancer Center.

But if you do wish to explore testosterone supplements, it's smart, given the controversy, to get a second opinion. Grill your doctors on how serious your prostate cancer was to start with - that is, how high your PSA was, and how many gland segments contained cancer. Also, keep being monitored for cancer recurrence.

Hamelburg is glad he eventually opted for testosterone. "My body was my enemy," he says. "Now, I just feel like a man again."

Steve Drouin, 56, a mason in Northfield, N.H., who has also had prostate cancer, echoes that view. "He's not tired all the time," says his wife, Jean. And has their sex life improved? "Yeah," she says. "It has."

Source: Boston Globe

Viagra May Shield the Heart from Damage from High Blood Pressure

Written by j strax

First evidence that the Impotence Aid Helps a Signaling Protein Protect the Heart

The first direct evidence in lab animals that the erectile dysfunction drug Viagra (sildenafil) amplifies the effects of a heart-protective protein has been reported today by a team of researchers at three leading US medical centers, Johns Hopkins in Baltimore, Tufts Medical Center in Boston and University of North Carolina.

Published in the Journal of Clinical Investigation online, the findings help explain why sildenafil has already been shown to improve heart function and may one day have value in either treating or preventing heart damage due to chronic high blood pressure.

The key, investigators say, is sildenafil's effects on a single protein, RGS2, newly identified in the latest study as an essential link in the chain reactions that initially protect the body's main blood-pumping organ from spi-

raling into heart failure.

Experimenting in mice, the team of heart experts first established that after a week of induced high blood pressure, the hearts of animals engineered to lack RGS2, or regulator of G-protein signaling 2, quickly expanded in weight by 90 percent. Almost half the mice died of heart failure. In mice with RGS2, by contrast, the dangerous muscle expansion, known as hypertrophy, was delayed, growing only 30 percent, and no mice died.

Subsequent tests treating hypertensive mice that had RGS2 with sildenafil showed enhanced buffering, with less hypertrophy, stronger heart muscle contraction and relaxation, and as much as 10 times lower stress-related enzyme activity compared to their untreated counterparts. In mice lacking RGS2, sildenafil had no effect.

"Sildenafil clearly prolongs the protective effects of RGS2 in mouse hearts," says study senior investigator and cardiologist David Kass, M.D.

According to Kass, a professor at the Johns Hopkins University School of Medicine and its Heart and Vascular Institute, RGS2 is stimulated by an enzyme, protein kinase G, whose action is, in turn, raised by countering the activity of another enzyme, phosphodiesterase 5 (PDE5A). Sildenafil's ability to block PDE5A was shown by Kass and his team in 2005 to be responsible for blunting hypertrophy due to high blood pressure in mice and offsetting similar, adrenaline-stimulated heart stress in people.

Kass says RGS2 "acts like a short-term reset mechanism in the heart," recoupling G proteins that if left alone stimulate the heart's response to high blood pressure. And without the "reset", a cascade of reactions known as Gq signaling leads to scar tissue formation, hypertrophy and heart failure.

Currently, physicians use so-called ACE inhibitor and ARB inhibitor drugs to block Gq signaling. These classes of drugs are the most common treatment for heart failure, which afflicts more than 5 million Americans, killing over a quarter million of them each year.

"The evidence is piling up that unbridled Gq signaling is driving a central biological chain reaction in heart failure," says Kass, "and that by extending the protective effects of RGS2 or by developing a test for its presence, researchers can develop new therapies or improve existing ones, including ACE inhibitors and possibly sildenafil, for people with heart failure who will benefit most."

Until recently, scientists thought RGS proteins, which are found only in small quantities in the heart -- a thousand times less than other, more common proteins, such as myosin and metabolic proteins -- played no key role in heart function. Previous tests in mice, Kass says, had shown no harmful effects to the heart from knocking out production of RGS2, though the protein was known to have a role in maintaining smooth muscle function in blood vessels.

But studies by co-investigators at Tufts Medical Center in Boston had shown that RGS2 activity was upped by protein kinase G, leading Kass and others to look for stronger links between these biological pathways and hypertrophy.

The latest study involved more

than a half-dozen experiments, all performed within the last three years and designed to zero in on the role played by RGS2 in stalling hypertrophy.

In one experiment for the current study, researchers artificially stimulated the Gq chemical pathway in mice lacking RGS2, worsening the effects of Gq signaling, including hypertrophy and widened heart chambers.

In another experiment in mice with and without RGS2, researchers analyzed the cardiac response to the physical stress of twice daily swimming exercises lasting 90 minutes each, a stress not known to affect Gq signaling. After six weeks of testing, both sets of mice showed similar increases, at 30 percent, in heart mass and no signs of impaired heart function.

Subsequent protein analysis for enzymatic action common to heart failure showed the same results for both sets of mice, confirming to researchers that RGS2 proteins were responsible for protecting the heart from hypertrophy linked to Gq signaling.

More tests with pressure overload showed that when RGS2 was stimulated by protein kinase G, both proteins moved together from inside the cell to its outer cell walls. This effect was then stabilized in RGS2 mice treated with sildenafil, solidifying evidence of the biological chain reactions between the drug and the protein.

"Our results offer among the first insights into the biology of the RGS2 protein in heart cells during hypertrophy," says study lead investigator Eiki Takimoto, M.D., Ph.D. "This greatly expands our understanding of how high blood pressure affects the heart and helps break down the disease equation into its molecular components for subsequent clinical testing."

Takimoto, an assistant professor at Hopkins, says the team's next plans are to look at other potential consequences of increased RGS2 activity within the cell and to zero in on what other proteins or factors boost its action.

PDE5A is involved in the breakdown of a key molecule, cyclic guanosine monophosphate, which helps control stresses and limit overgrowth in the heart. PDE5A is also the biological pathway blocked in the penis by sildenafil to promote the relaxation of blood vessels and maintain erections.

Funding for the reported study was provided by the National Institutes of Health, the Peter Belfer Laboratory Foundation, and the American Heart Association.

Besides Kass and Takimoto, other Hopkins researchers involved in this study were Norimichi Koitabashi, M.D., Ph.D.; Steven Hsu; Elizabeth Ketner, M.S.; Manling Zhang, M.D., Ph.D.; Takahiro Nagayama, Ph.D.; Djahida Bedja, M.S.; and Kathy Gabrielson, D.V.M., Ph.D. Additional assistance was provided by Michael Mendelsohn, Robert Blanton, at Tufts Medical Center in Boston; and by David Siderovski, at the University of North Carolina at Chapel Hill. Kass is also the Abraham and Virginia Weiss Professor of Cardiology at Hopkins.

SOURCE:

Regulator of G protein signaling 2 mediates cardiac compensation to pressure overload and antihypertrophic effects of PDE5 inhibition in mice
Eiki Takimoto, et al.

Source: psa rising

Prostate Cancer 101, Inc.
8 Alcazar Avenue
Kingston, NY 12401-4302

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SEMINAR For Newly Diagnosed

3rd Tuesday

Distinguished Lecturer Series

4:30 p.m. monthly

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Our brothers in support and education

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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