The Manitoba Prostate Cancer Support Group NEWSLETTER

Prostate Cancer Canada Network Winnipeg

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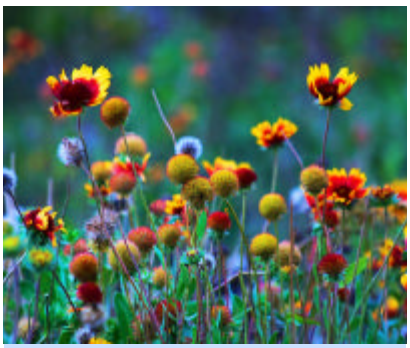

NEXT MEETING:

THURSDAY, MAY 19, 2011 7 - 9 P.M.

Greg Harochaw, Pharmicist

Topic: "**Erection, Misdirection, Penile Rehabilitation & Treatments for Erectile Dysfunction**"

Location: AUDITORIUM of the Seven Oaks General Hospital - Leila & McPhillips

Medical Advisors to The Manitoba Prostate Cancer Support Group

=> Paul Daeninck M.D.
Pain Management

=> Darryl Drachenberg
M.D. Urologist

=> Graham Glezerson
M.D. Urologist

=> Ross MacMahon
M.D. Urologist

=> John Milner
M.D. Urologist

=> Jeff Sisler M.D.
Family Practitioner

Thanks!

The Manitoba Prostate Cancer Support Group encourages wives, loved ones, and friends to attend all meetings.

Feel free to ask basic or personal questions without fear of embarrassment. You need not give out your name or other personal information.

The Manitoba Prostate Cancer Support Group does not recommend treatment modalities, medications, or physicians. All information is however freely shared.

THOUGHT FOR THE DAY

ABOUT GROWING OLDER...

You know you are getting old when everything either dries up or leaks.

Will Rogers Wisdom

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R3M 3S7

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The Manitoba Prostate Cancer Support Group operates on your donations. Have you used any of Newsletter - General Meetings - Hospital visits -One-on-one visits – Speakers ?

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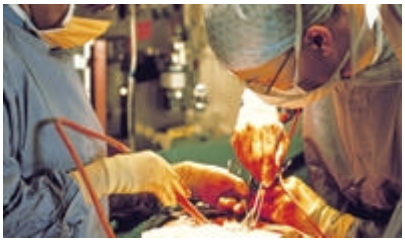
Erectile Dysfunction After Prostate Cancer

Amanda Daniel and Sara Haddow, MSA, PA-C

March 01 2011



Erectile dysfunction occurs most often when prostate cancer is treated surgically.

At a glance

=> Erectile dysfunction (ED) occurs more frequently when prostate cancer is treated surgically rather than medically.

=> Phosphodiesterase type 5 inhibitors are often used initially for ED following radical prostatectomy.

=> Address the medical and psychological issues of ED in conjunction with treatment options early in the disease course.

=> Reasons for not seeking treatment include embarrassment, financial instability and threatened masculinity.

Prostate cancer is the most commonly detected and diagnosed solid tumor in American men. [1] It is expected that a male born in the United States has a 17% probability of developing prostate carcinoma later. [2] Thanks to the improved education of providers, extensive screening, and overall public awareness, many men are being diagnosed when asymptomatic, with the tumor localized to the prostate. [3]

With the diagnosis of a localized tumor, many patients choose complete eradication of the cancer by means of radical prostatectomy. In fact, it is generally recognized as the favored treatment for localized prostate cancer in young, otherwise healthy men with high probability of significant life expectancy. [4] While this treatment reduces mortality, distant metastasis, and tumor recurrence, it is also associated with possible lifelong side effects. Although it is essential that men with prostate cancer are effectively treated, primary-care providers must keep in mind the possible complications and preservation of function after treatment.

Pathology and epidemiology

Erectile dysfunction (ED) is one of the most devastating long-term obstacles following treatment of prostate cancer, regardless of the management chosen by the patient. ED occurs more frequently when prostate cancer is treated surgically rather than medically. [5] Even seven years postoperatively, more than 75% of men struggle with problems related to ED.6 The rate of ED following radical prostatectomy is unpredictable because of such variables as time of assessment, baseline function, and use of pharmacologic or surgical treatments. [7] With the increasing use of radical prostatectomy to eradicate localized prostatic carcinoma, providers need to be more aware of this common and distressing adverse effect. It was proposed in a landmark trial that the basis of ED was damage to the cavernous nerves that transmit autonomic neuroregulatory function to the proximal penis and deep pelvis. [8] Vascular and smooth-muscle damage during surgery plays a role in pathogenesis as well. [9] Despite efforts to reduce incidence (e.g., bilateral nerve-sparing prostatectomy), most men battle with ED after prostatectomy. [10]

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Treatment options

Easy-to-use and inexpensive, phosphodiesterase type 5 (PDE-5) inhibitors are often prescribed following radical prostatectomy. A trial of sildenafil (Viagra) is a sound initial choice for prostate-cancer survivors presenting with ED after surgical treatment. In men treated with the nerve-sparing prostatectomy procedure, sildenafil and other oral PDE-5 inhibitors have been effective. [11] Unfortunately, these agents are less effective in prostate-cancer patients following non-nerve-sparing surgical intervention. [10] More aggressive management may be needed. Increased dosages of PDE-5 inhibitors have not been found to improve the desired effect of an erection. [12] Propionyl-L-carnitine and acetyl-L-carnitine have parallel mechanisms of action to the PDE-5 inhibitors and can be added to sildenafil to increase its effectiveness. [12]

More invasive treatments such as penile-injection therapy, vacuum devices, and penile-prosthetic surgery have better efficacy in assisting production of erections. [10] Penile-injection therapy consists of the introduction of such vasoactive substances as prostaglandins and phentolamine via intracavernosal injection. [13] A small study found that penile injections improved not only sexual function in terms of erection after prostatectomy, but satisfaction with the sexual relationship and sexual confidence as well. [13] Success rates of penile injections lie within the 75% to 80% range. [13]

Vacuum devices and penile implants allow anywhere from 90% to 100% of patients to achieve successful sexual intercourse. [14] Early use of vacuum-constriction devices after radical prostatectomy allows quicker return to sexual activity. [15] Studies have shown early return of erectile function in patients undergoing both nerve-sparing and non-nerve-sparing intervention. [11] The penile prosthesis was introduced in the early 1970s by Brantley Scott. [16] The basis for Scott's implant is still in use today, with increased reliability, longevity, and improved surgical outcomes attributable to its modifications.

The receptiveness toward more invasive treatments is less than that of oral PDE-5 inhibitors.[10] Many prostate-cancer survivors consider vacuum devices and implants unnatural. [17] For these men, the recently introduced sural-nerve grafting has shown promising results. Unfortunately, nerve grafting is a technically difficult procedure and has varied reproducibility. [18] This surgical procedure employs the concept of a neural conduit to improve the recovery of erectile function. [19]

Unilateral loss of neurovasculature with subsequent sural-nerve grafting allowed a 78% chance of recovery of erectile function; bilateral neurovasculature loss with sural-nerve grafting procedure allowed a 58% chance. [18] Such invasive procedures are not usually within the scope of primary care but are available options to discuss with patients presenting with persistent ED (Table 1).

Patient education

Providers must realize that ED may be a lifelong complication following prostate-cancer treatment, despite the availability of acceptable management options. After radical prostatectomy, almost 60% of men are unable to maintain firmness for intercourse, and about 44% are completely unable to have erections. [1] Providers need to address the medical and psychological issues of ED in conjunction with treatment options early in the disease course. Successfully coping with diagnosis, treatment, and adverse effects can drastically alter personal aspects of quality of life for patients.

From a primary-care standpoint, patient education plays a large part in the treatment and management of a prostate-cancer patient. A survey of prostate-cancer survivors indicated a need for more information concerning the side

(Continued on page 4)

TABLE 1. Treatment options for ED following radical prostatectomy

Treatment Option	Role	Efficacy
Phosphodiesterase type 5 inhibitors (tadalafil [Cialis], vardenafil [Levitra], sildenafil [Viagra])	First-line	70%-80% (nerve-sparing) 0%-15% (non-nerve-sparing)
Penile injections	Second-line	75%-80%
Vacuum devices	Second-line	90%-100%
Penile implants	Third-line	95%-100%
Sural-nerve grafting	Third-line	78% (unilateral loss) 58% (bilateral loss)

Adapted from Burnett AL. Erectile dysfunction following radical prostatectomy. *JAMA*. 2005;293:2648-2653; Albaugh JA, Ferrans CE. Impact of penile injections on men with erectile dysfunction after prostatectomy. *Urol Nurs*. 2010;30:64-77; and Donatucci CF, Greenfield JM. Recovery of sexual function after prostate cancer treatment. *Curr Opin Urol*. 2006;16:444-448.

(Continued from page 3)

effects of the cancer itself and its treatments. [20] Patients surveyed also placed an importance on sexual function postoperatively. The fact that information concerning management of ED ranked seventh on a list of top unmet needs shows that the side effect and its treatment were not sufficiently presented to a significant number of patients. [20] ED might be included on a list of side effects presented to patients in the preoperative period, but discussion of the side effect itself and options for its treatment are often not readily explored.

It has been found that patients would trade as much as a 10% or greater advantage in five-year survival to avoid ED following prostate-cancer treatment. [21] Men consistently value sexual function almost as much as a positive postoperative prognosis. Providers ought to think about how to better support the information needs of their patients. [20] Primary-care clinicians have the ability to intervene and provide much needed information, options, and support to prostate-cancer patients and survivors who face ED.

Patients who forgo treatment

The complex, personal struggle with ED can be difficult for many men to discuss with a clinician. By way of illustration, only 30% of prostate-cancer survivors utilize a treatment for ED.[22] Thus, a number of men are left with a life-altering condition that has approved options for management. Reasons for not seeking treatment include embarrassment, financial instability, belief that treatment may be risky or harmful, and threatened masculinity. [23] In fact, the most common initial reaction to ED is a sense of emasculation. [24]

Patients with ED who are not treated or counseled can suffer from relationship issues and depression, leading to decreased desire to correct the problem. [24] Of those men who have discussed ED with health-care provider and begun treatment, most had longstanding ED and/or presence of other comorbidities. [25] These patients make more health-care visits and may have stronger clinician-patient relationships. The small number of patients seeking treatment management for ED desired to improve their self-esteem, hear about the available therapies, and presented at the insistence of their partner. [23] Awareness of the negative beliefs surrounding outcomes of ED helps providers understand the importance of opening a discussion of the condition and the possibility of beginning treatment. Increased quality of life and positive results following treatment can ease patients' minds and support the choice to initiate therapy.

Nearly 95% of men surveyed said they would be willing to have a one-on-one consultation with a health-care provider concerning ED and its management. However, this was decided only after being approached about the issue. [10] Men visit clinicians less frequently than women and will more often play a submissive role in the provider-patient relationship. [26] Men are receptive to a discussion of ED, so it must be adequately addressed in the primary-care setting. With informative intervention, positive changes in sexual function and receptiveness to medical or surgical management for ED are achievable. [10] Effective treatment of ED after radical prostatectomy will improve a man's mental and physical quality of life. [24]

Ms. Daniel is a second-year student in the physician assistant program at the Medical College of Georgia in Augusta, where Ms. Haddow is an assistant professor and director of education.

References

http://www.clinicaladvisor.com/erectile-dysfunction-after-prostate-cancer/article/197330/

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One Type of Stem Cell Creates a Niche for Another Type in Bone Marrow

ScienceDaily (Aug. 12, 2010) — Hematopoietic stem cells (HSCs) have two unique abilities that are prized by medical researchers: to self-renew and to develop into any kind of blood cell, which enables them to replenish the entire blood and immune system. Scientists have traced these qualities to a distinct locale or niche within the bone marrow that HSCs home in on, but the identity and function of the niche-forming constituents have not been clearly defined.

Now, the precise source of HSC maintenance and regulation within the bone marrow has been discovered by Cold Spring Harbor Laboratory (CSHL) researchers and members of a multi-institutional team. In a study to be published in Nature on August 12th, the collaborators report that the HSCs retain their unique features in response to signals from another stem cell population, the mesenchymal stem cells (MSCs), which create a supportive bone marrow niche for the HSCs.

"This is the first demonstration that one type of stem cell can regulate another type of stem cells," says CSHL scientist Grigori Enikolopov, Ph.D. "Having a detailed

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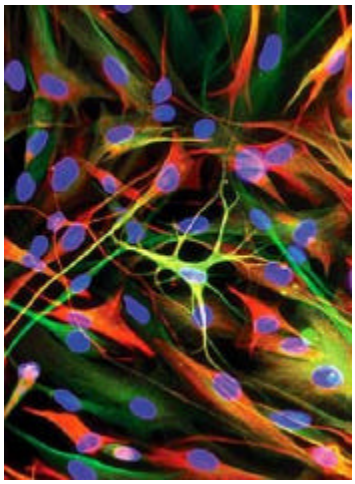
understanding of how HSCs are maintained within the niche microenvironment offers new opportunities to better exploit these cells for therapeutic use."

The path to this discovery originates from Enikolopov's efforts to develop reliable ways of distinguishing stem cells from other cell types. Previously, his group found that various types of stem cells express a protein called nestin. In recent years, they have detected and analyzed stem cell populations located in niches throughout the body using mice genetically engineered such that nestin-expressing cells also produce a fluorescent marker, making it possible to visually track these cells.



One location where the CSHL researchers found these nestin-expressing cells was the bone marrow, but these cells proved to not be HSCs. To find the identity and function of these mystery cells, they teamed up with a multi-institutional group led by Paul Frenette, M.D., of Mount Sinai School of Medicine, and which also included scientists from Harvard Medical School and the Albert Einstein College of Medicine.

Examining bone marrow sections from the nestin-expressing mice and based on the presence of various molecules on the populations of cells found within, the team identified the nestin-expressing cells as MSCs. These cells, which outnumber the HSCs 10:1, were either in direct contact with the HSCs or in clusters around them.

"This close proximity suggested that there might be some kind of molecular signaling or cross-talk going on that might control HSC maintenance," explains Enikolopov, who has been investigating the consequence of this kind of cell-cell dialogue during tissue and organ development. "One hypothesis is that this interaction directs and regulates growth by teaching different types of cells where they ought to locate themselves and what they should do there."

This seems to be the case, at least in the bone marrow, as the collaborators found that altering the numbers of MSCs has a corresponding effect on the numbers of HSCs. Increasing the numbers of MSCs by injecting mice with a growth hormone caused HSCs to double in number. Inhibiting MSC proliferation via infusions of growth-inhibiting proteins, on the other hand, was followed by a dip in HSC numbers.

The collaborators also explored the consequence of completely eradicating bone marrow MSCs using genetic tools developed in Enikolopov's lab. The CSHL scientists created mice in which only nestin-expressing cells -- the MSCs -- were programmed to carry a receptor for a toxin molecule. Injecting mice with the toxin thus selectively wiped out the MSCs. As a result, the scientists observed a four-fold reduction in the numbers of HSCs in the bone marrow of these mice. In the absence of bone marrow MSCs, large numbers of HSCs injected into the mice also failed to make their way to the bone marrow.

The collaborators found that genetic factors that are essential for HSC maintenance are highly concentrated within the neighboring MSCs. "The expression of these factors are least 500-fold higher in the MSCs than in other bone marrow cells, which shows why HSCs are so highly dependent on the MSCs," says Enikolopov.

"These results are a definitive indication that the MSCs are required for HSC maintenance as well as their homing to the bone marrow," says Tatyana Michurina, Ph.D., a research investigator in Enikolopov's laboratory who is an expert on both MSCs and HSCs. "After studying them as separate entities for years, it was such a surprise to discover that it was the MSCs that helped the HSCs to remain stem-like and primitive." "These findings suggest that if we can control the niche, we can also manipulate the HSC population within," says Enikolopov. Pharmacological targeting of the niche might help enhance stem cell production for use in regeneration therapies or might help stave off adverse events like leukemia or other disorders where stem cells proliferate out of control.

The CSHL researchers were supported by a grant from the National Institute of Mental Health and a generous contribution by the Ira Hazan Fund.

Story Source:
The above story is reprinted (with editorial adaptations by ScienceDaily staff) from materials provided by Cold Spring Harbor Laboratory, via EurekAlert!, a service of AAAS.

Journal Reference:
Simon Mendez-Ferrer, Tatyana V. Michurina, Francesca Ferraro, Amin R. Mazloom, Ben D. MacArthur, Sergio A. Lira, David T. Scadden, Avi Ma'ayan, Grigori N. Enikolopov & Paul S. Frenette. Mesenchymal and haematopoietic stem cells form a unique bone marrow niche. Nature, 2010; 466 (7308): 829 DOI: 10.1038/nature09262

Cold Spring Harbor Laboratory (2010, August 12). One type of stem cell creates a niche for another type in bone marrow. ScienceDaily. Retrieved April 22, 2011, from http://www.sciencedaily.com / releases/2010/08/100812101020.htm

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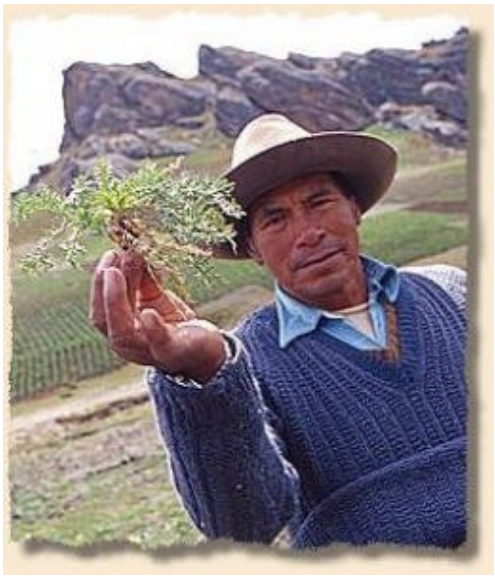
Prostate.net
Healthy Living for Men
Maca Root Benefits for Sexual and Prostate Health

by Editor on September 27

Prostate.net

The highlands of Peru are the home of maca (*Lepidium meyenii)*, a cruciferous plant that can count broccoli, cauliflower, and kale among its relatives, and a plant some claim is a boost to men's sexual health. A new review published in *BMC Complementary and Alternative Medicine* (August 2010) presents what has been discovered about maca root and its effect on libido and erectile dysfunction in the limited number of randomized controlled studies conducted so far. (1)

Use of maca root goes back a long way when, according to Incan folklore, warriors took the plant before they went into battle because it increased physical strength. Maca also elevated their libido, a situation Incan leaders found unacceptable for their purposes so they eventually prohibited their soldiers from taking it during wartime.



In recent years, scientists have explored the biochemistry behind the claims of enhanced sexual function and stamina associated with maca. Because maca belongs to the Brassicacae family, which is known for its anticancer properties, there is also speculation that it may be helpful in protecting against prostate cancer or other prostate conditions.

One of the more recent studies comes from Italy, where investigators in the department of urology at Morgagni-Pierantoni Hospital conducted a double-blind trial. Fifty men who had mild erectile dysfunction were enrolled: half were given 2,400 mg maca root daily and the other half received placebo. After 12 weeks, men in the maca-treatment group experienced a more significant increase in the International Index of Erectile Function (IIEF-5) than men in the placebo group. The scientists concluded that maca supplements provide a small but significant effect on sexual health and well-being in men who have mild erectile dysfunction. (2)

Maca may also benefit the prostate, and in this case color matters. Maca comes in red, yellow, and black, and in a 2009 study researchers compared the impact of each one on prostate size in rats with induced benign prostatic hyperplasia, a condition in which the prostate is enlarged. Red maca appears to have a prostate-reducing effect, while yellow offers a mild impact and black has none. (3)

Three other studies are also worth noting. In one, researchers evaluated the effect of 1,500 mg and 3,000 mg of maca compared with placebo in a 12-week trial. After 8 weeks, men who took maca reported an improvement in sexual desire in one while men in the placebo group did not. (4) In another, maca increased sperm count, sperm motility, and seminal volume, which could improve fertility. (5) The third study was conducted at Massachusetts General Hospital and involved depressed men and women. Participants in this study who took 3,000 mg maca daily reported an improvement in sexual dysfunction. (6)

The ability of maca to improve sexual health may be attributed to several different components. Maca contains higher levels of glucosinolates than its cruciferous cousins, and substances hydrolyzed from these glucosinolates

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reportedly inhibit certain types of cancer in rats. [7] Maca's ability to enhance libido is attributed to its polyunsaturated fatty acids. However, experts are not certain how maca impacts erectile dysfunction without affecting levels of testosterone, estradiol, and other hormones. One idea is that it acts on the receptors for these hormones rather than on the hormones themselves. [8]

Although the amount of research on maca is limited, so far it appears to have a positive effect on sexual health and prostate health. Maca is available as capsules, powder, tablets, and an extract. Several studies have shown 3,000 mg daily to be an effective and safe dose, but it is best to take maca according to package directions and to consult a knowledgeable healthcare provider.

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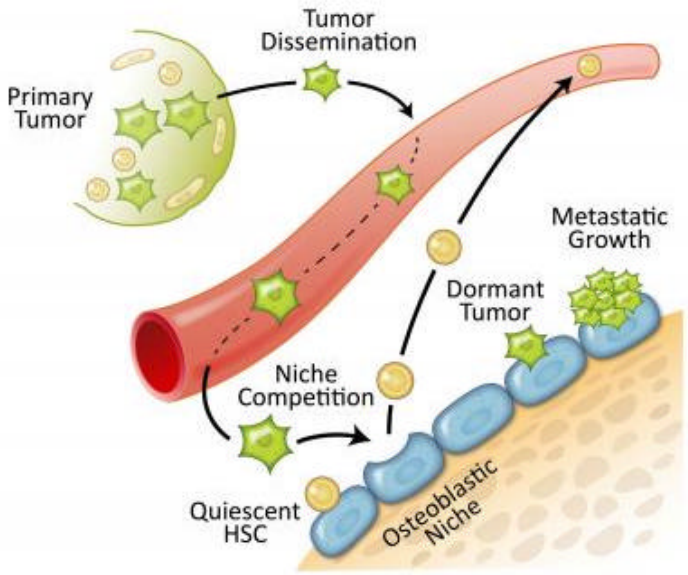
Prostate Cancer Spreads To Bones By Overtaking The Home Of Blood Stem Cells

ANN ARBOR, Mich.-Like bad neighbors who decide to go wreck another community, prostate and breast cancer usually recur in the bone, according to a new University of Michigan study.

Now, U-M researchers believe they know why. Prostate cancer cells specifically target and eventually overrun the bone marrow niche, a specialized area for hematopoietic stem cells, which make red and white blood cells, said Russell Taichman, professor at the U-M School of Dentistry and senior author of the study.

Once in the niche, the cancer cells stay dormant and when they become active again years later, that's when tumors recur in the bone. The implication is that this may give us a window as to how dormancy and recurrence take place. Taichman and a team of researchers looked in the bone marrow and found cancer cells and hematopoietic stem cells next to one another competing for the same place. The finding is important because it demonstrates that the bone marrow niche plays a central role in bone metastasis - cancers that spread into the bone - giving researchers a new potential drug target.



This is a drawing of prostate cancer cells in the bone marrow niche.

University of Michigan

Drugs could be developed to keep the types of cancers that likely recur in the bone from returning, Taichman said. For example, these drugs could either halt or disrupt how the cancer cells enter or behave in the niche, or keep the cancer cells from out-competing the stem cells.

Cancer cells act a lot like stem cells in that they must reproduce, so the U-M research group hypothesized that prostate cancer cells might travel to the niche during metastasis. One of the jobs of the niche is to keep hematopoietic stem cells from proliferating - which may be the case for cancer cells, as well, the researchers found.

So why does cancer recur? Say a person has a tumor and surgeons cut it out or do radiation, but it recurs in the bone marrow five years later, Taichman said. Those cancer cells had been circulating in the body well before the tumor was discovered, and one place those cancer cells hid is the niche.

"So what have the cancer cells been doing during those five years? Now we have a partial answer - they've been sitting in this place whose job it is to keep things from proliferating and growing," Taichman said.

"Our work also provides an explanation as to why current chemotherapies often fail in that once cancer cells enter the niche, most likely they stop proliferating," said Yusuke

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Shiozawa, lead author of the study. "The problem is that most of the drugs we use to try to treat cancer only work on cells that are proliferating."

Metastases are the most common malignant tumors involving the skeleton, and nearly 70 percent of patients with breast and prostate cancer have bone involvements. Roughly 15 percent to 30 percent of patients with lung, colon, stomach, bladder, uterus, rectum, thyroid or kidney cancer have bone lesions.

Researchers aren't quite sure how the cancer cells out-compete the stem cells in the niche. However, they do know the stem cells were displaced because when cancer cells were in the niche scientists also found evidence of immature blood stem cells in the blood stream, instead of in the marrow where they were supposed to be, Taichman said.

"Eventually the entire blood system is going to collapse," he said. "For example, the patient ultimately becomes anemic, gets infections, and has bleeding problems. We really don't know why people with prostate cancer die. They end up dying from different kinds of complications in part because the marrow is taken over by cancer."

The next step is to find out how the tumor cell gets into the niche and becomes dormant, and exactly what they do to the stem cells when they are there. Researchers also want to know if other types of cancer cells, such as breast cancer, also go to the niche.

The study, "Prostate Cancer Metastases Target the Hematopoietic Stem Cell Niche to Establish Footholds in Marrow," appears online in the Journal of Clinical Investigation.

Co-authors are: Elizabeth Pedersen, Aaron Havens, Younghun Jung, Anjali Mishra, Jeena Joseph, Jin Koo Kim, Anne Ziegler, Michael Pienta, Jingcheng Wang, Junhui Song and Paul Krebsbach of the U-M School of Dentistry; Lalit Patel, Chi Ying, Robert Loberg and Kenneth Pienta of the departments of Urology and Internal Medicine at the U-M Medical School.

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2011 SPEAKERS:

May 19, 2011
Greg Harochaw, Pharmacist
Topic: "Erection Misdirection: Penile Rehabilitation & Treatments for Erectile Dysfunction"

June 16, 2011
Dr. Chris Jensen
Topic: Non-Traditional Prostate Cancer Assessment & Treatment in Canada

July 21, 2011
"Members speak out"
Member's stories ....
Radical Prostatectomies, HIFU, Active Surveillance, Radiation, Brachytherapy and more.
SNACKS included

All meetings are held at
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Everyone welcome

M.P.C.S.G. Executive

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Len Bueckert - Newsletter	782-4086
Tom Boomer - Recording Sec./ New Member	663-1351
June Sprott - Corresponding Sec.	668-6160
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