

“Something is happening in the Department of Urology at Pittsburgh,” I overheard a urologist say recently at a national meeting in California. “They are going great guns.”
Great guns, indeed!

We have been busy. In 1999, when I came from Johns Hopkins to the University of Pittsburgh to be the first chairman of the newly formed Department of Urology, there were five faculty members in the department. Today there are 21: 12 clinical urologists and nine basic scientists. We have started four new fellowship programs, including pediatric urology, neurourology, endourology, and laboratory research. We attract physicians and scientists from around the world who compete for a place to work and study as part of the group.

It was clear to me when I first started here, and it is even more obvious now, that we have a tremendous opportunity to make this department the premier program in the world for clinical care and research. The institutions—UPMC and the University of Pittsburgh—have been unparalleled in their support. Patients seeking our care come from across the United States, in significant numbers. Just the other day, someone in Argentina asked about our prostate cancer program.

A world-class department must be strong in both clinic programs and laboratory research. This facilitates a pragmatic approach to helping our patients: the physician is kept abreast of the newest research findings, and the scientist is reminded of the real problems patients face. To encourage this interaction, we specifically built our laboratories close to our clinics. Discoveries in our laboratories often get their first tests in our clinics.

Over the last four years, our research program has grown exponentially. Our grant funding has grown from approximately \$300,000 to nearly \$4 million in the last fiscal year. We currently have 22 grants funded by the National Institutes of Health (NIH).

Welcome to the FOREFRONT

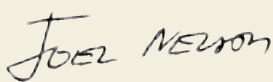


DR. JOEL B. NELSON
CHAIRMAN, DEPARTMENT OF UROLOGY
UNIVERSITY OF PITTSBURGH

In this premier issue of *ForeFront*, several articles describe the work of these investigators and the highlights of their research.

This is an exciting time. Our trajectory has captured national attention. What really matters, however, is what we do for our patients. As you will see in these stories that follow, we are focused on improving the lives of our patients. By the end of 2003, I will have performed more than 700 radical prostatectomies in Pittsburgh. I look forward to the day when—through our department’s research efforts—we will no longer need to do that operation.

One of the best things about Pittsburgh is that patients often become our supporters and friends. The goal of this report is to give you a better idea of our research activities. I trust you will share in my pride at our accomplishments.



JOEL B. NELSON, MD

FEATURING

Australian Scientists
Come to Pittsburgh
to Explore New Ideas
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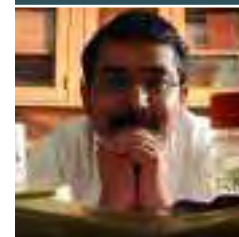
Reducing Post-
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Can Diet Cause
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Australian Scientists

Come to Pittsburgh to Explore

New Ideas



DEAN BACICH, PHD AND DENISE O'KEEFE, PHD

PSMA Studied as Possible Factor

Making great strides in prostate cancer research requires the right people and the right resources. All experiments, whether chemical, animal, or human trials, require skilled scientists, adequate laboratory space, and state-of-the-art technology if an innovative idea is ever to become a new medical treatment.

Equally vital is a supportive environment, one that encourages freethinking investigations into far-reaching ideas. This environment exists at the University of Pittsburgh's Department of Urology and it's what drew Dean Bacich, PhD and Denise O'Keefe, PhD to join the faculty.

The husband-and-wife team are basic science researchers from Australia, who had previously worked at Memorial Sloan-Kettering Cancer Center and the Cleveland Clinic. They came to Pittsburgh in

March 2003 to join the growing group of young investigators working under Dr. Joel Nelson's supportive leadership.

"Dr. Nelson is interested in our research, and is willing to take risks. The department is vibrant and exciting," said Dr. O'Keefe. "Our doctors make up the youngest department at the University, but we also have the most PhDs of any urology department. Here you can take a new idea and really run with it."

The fantastic resources at the University of Pittsburgh impressed Dr. Bacich. "I've got access to a great tissue bank and top-notch technology. The department's quality leadership makes it a group that is going places," he said.

The University of Pittsburgh's reputation extends around the world, earning the respect and cooperation of other research centers around the country in sharing

findings. "Here at the University, we get unheard of access to normal and abnormal prostate tissues for study," Dr. Bacich added.

Though prostate cancer is a disease that affects many men, it remains silent in many more. Forty percent of men in their 40s and 60 percent of men in their 60s have prostate cancer and never know it, because it never reaches the level of clinical detection.

"Why do so many men get it and almost all animals do not? If we can understand that, we may be able to prevent it," said Dr. Bacich.

Dr. Bacich's work could lead to a link between prostate-specific membrane antigen (PSMA) and prostate cancer. PSMA is a protein found in high levels in prostate cancer tissues, but not in normal blood. Dr. Bacich genetically engineered mice to express human PSMA, and found they developed

enlarged prostate glands containing precancerous protein lesions, similar to PIN (prostatic intraepithelial neoplasia), the precancerous cell changes found in men. Mice that did not express PSMA did not show these changes.

This research is essential for advancement in medical treatment of prostate cancer. If PSMA is determined to be a factor in cancer progression, the next step will be to develop a suitable inhibitor.

Envisioning connections between research studies in disparate fields is another characteristic of the progressive scientist. In another experiment, Dr. Bacich created mice lacking in PSMA and induced strokes in them. The non-PSMA mice showed 30 percent less stroke damage than the control group, were less susceptible to nerve damage, and also recovered faster. This could lead to the development of PSMA inhibitors that would minimize nerve damage and speed the recovery of radical prostatectomy patients.

“Silent Genes” May Indicate Prostate Cancer

Dr. Denise O’Keefe was in high school in Australia when she learned that human DNA genes switch on and off, creating different effects in different cells. She has been exploring this line of cutting-edge research, on the very vanguard of genetics, ever since. At the University of Pittsburgh, she is pursuing the consequences of gene methylation (the shutting off or silencing of a gene’s influence) on prostate cancer.

In studying most cancers, scientists have found that each has specific mutated genes. But researchers haven’t found such muta-

tions in prostate cancer. “Normally, we see genes that look one way in normal tissue and different in cancer tissue. Using the tissue bank, I am able to see the genes that are methylated, and how cancer congregates in certain cells,” said O’Keefe.

The University’s tissue bank is unique because it has ten years’ worth of samples, and follows each patient through his disease, allowing genetic differences to be correlated with disease progression.

This research is essential for advancement in medical treatment of prostate cancer.

Dr. O’Keefe has noted that in 90 percent of prostate cancer tumors, the GSTPI gene is hyper-methylated. GSTPI is a “gate-keeper” of the cell, a detoxifying agent. If its activity is silenced, it may allow toxins to enter the cell and cause increased DNA damage.

By studying high-grade PIN biopsies, Dr. O’Keefe hopes to isolate the five to 10 genes that are silenced in prostate cancer via methylation. This knowledge could help scientists reach one of the prime goals of cancer research: better genetic markers for patients with prostate cancer.

“If we learn how shutting off a gene makes a cancer more aggressive, we can work toward more specific treatment,” she said. “Developing rational approaches to therapy begins with understanding how the molecular mechanisms affect disease progression.” **F**

DEAN BACICH, PHD, joined the department in 2003. He received his Ph.D. in the Faculty of Medicine in Obstetrics and Gynaecology at the University of Adelaide, Australia, in 1997. In July of 1996, he undertook postdoctoral studies on the characterization and genomic organization of the prostate-specific membrane antigen gene (PSMA) at Memorial Sloan-Kettering Cancer Center, New York. In 1999, he was promoted to Research Associate and awarded a New Investigator Grant from the National Cancer Institute—designated Comprehensive Cancer Center Clinical Nutrition Research Unit, to study the role of PSMA on prostate carcinogenesis and progression. This was performed in a transgenic mouse model that he had generated that specifically expresses PSMA in the mouse prostate. He continued this work at the Cleveland Clinic in 2000, and was awarded an American Foundation for Urological Disease Post-Doctoral Fellowship.

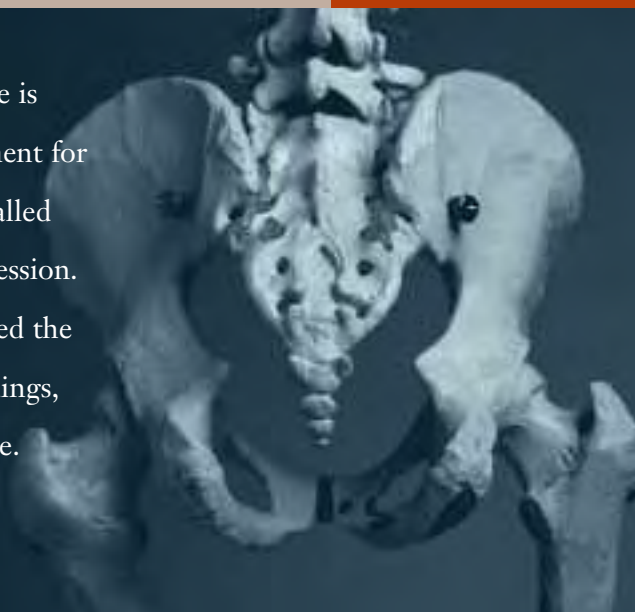
DENISE O’KEEFE, PHD, received her doctorate in Medicine from the University of Adelaide, Australia in 1996. She then studied genetic imprinting in Beckwith-Wiedemann Syndrome and Wilm’s tumor as a postdoctoral researcher at Columbia University in New York City. At Memorial Sloan-Kettering Cancer Center, she studied the role of prostate-specific membrane antigen (PSMA) in prostate cancer. She is co-author of an international patent describing the uses of the PSMA-like gene, a homolog of PSMA. In addition, Dr. O’Keefe was an AFUD fellow and supported by a DOD fellowship-training grant. She came to the University of Pittsburgh in 2003, following two years as a project scientist at the Cleveland Clinic Foundation. She plans to examine the epigenetic mechanisms occurring in prostate cancer.

in Cancer Progression

Delaying Prostate Cancer Progression in Bone

FOREFRONT
RESEARCH

To date, the only reliably effective treatment for prostate cancer growing in bone is hormonal therapy. Dr. Joel Nelson has been studying atrasentan as a new treatment for men with advanced prostate cancer. Atrasentan blocks the activity of a protein called endothelin-1, which Dr. Nelson has shown to be active in prostate cancer progression. In large clinical trials involving more than 800 men worldwide, atrasentan delayed the progression of prostate cancer in men with bone metastases. Based on these findings, it is hoped that atrasentan will be a new treatment for men with advanced disease.



REDUCING

Post-Surgical Troubles:

Research Suggests Nerve Proteins Can Restore Function

Some 179,000 American men will learn that they have prostate cancer this year. Because of new medical and surgical treatments, the majority will survive, but many will be left with a degree of impotency, unable to resume normal sexual relations.

“Erectile dysfunction (ED) is common after any treatment for prostate cancer,” said Michael Chancellor, MD, professor of urology, and director of Neurourology and Urinary Incontinence Programs at the

Impotency and Incontinence

Dr. Chancellor’s pioneering research could someday reduce the prevalence of ED by spurring new growth within the damaged nerves, and possibly speed the recovery of sexual functioning after surgery. In experiments on rats, he has frozen the cavernous nerves (those nerves that cause an erection), inducing a state of artificial impotence. He then injects nerve growth factors, known as neurotrophic proteins, into the area around the cavernous nerves. The proteins are

aging, and, in men, prostate surgery. The weak muscles cannot constrict to prevent urine leakage.

Though there are various medical and surgical treatments that can help—such as collagen injections and pubovaginal sling surgery—Dr. Chancellor’s research takes a new approach: asking the body to heal itself.

“We envision harvesting stem cells from the adult patient, usually through a biopsy of the small muscles of the upper arm,” said Dr. Chancellor. “Stem cells have the ability to repair damaged muscles. They perform a healing function.”

Dr. Chancellor emphasized that this procedure does not involve stem cells from any fetal or umbilical cord tissue. But because a biopsy can only supply about 100 adult stem cells, researchers have established a commercial biotech facility to culture the sample—and produce up to 10 million muscle-derived cells.

These cells would then be purified and injected into the patient’s sphincter, causing it to rebuild muscle tissue and rejuvenate itself. The stronger muscle would regain function and control leakage.

In using this technique on rats, Dr. Chancellor has improved their sphincter contraction by 88 percent. He hopes to start phase one of a human clinical trial in 2004. “I believe we can be the first to do this clinically,” he said. “There is little risk because the patient will be getting his or her own cells, and the procedure is noninvasive.”

In addition, this technology has already spun off a new biotech company, which could further the business development goals of the University’s Office of Technology Transfer, generate patents and licenses, and bring new job opportunities to Pittsburgh. **F**

“A man’s fear of losing his potency may keep him from seeking help early, and that can impinge on outcomes.”

carried to the nerve by a modified, safe, herpes virus. “Because herpes loves to live in the nerves, it was a good virus to carry these proteins,” said Dr. Chancellor.

Researchers then compared the pressure of the erections in rats receiving the regenerative proteins to those animals that did not. They found significant differences: the nerve growth factors led to a more rapid return to nearly normal erection.

Dr. Chancellor’s team certainly has regulatory hurdles to overcome before the procedure can be tested on humans, particularly because the herpes virus is involved, but he foresees a time when the injection will be given routinely, at the time of surgery, to forestall ED before it begins.

Reducing Incontinence Through Stem Cell Engineering

Stress incontinence is a problem that affects 13 million Americans, mostly women. This occurs when the urethral sphincter muscles are weakened as a result of pregnancy,

DR. CHANCELLOR earned his medical degree from the Medical College of Wisconsin. He completed his urology residency at the University of Michigan and a fellowship in neurourology and female urology at Columbia University. Before joining the University of Pittsburgh Medical Center, he was Associate Professor of Urology at Thomas Jefferson University in Philadelphia. His clinical interests include neurourology, urinary incontinence, and reconstructive surgery of the urinary tract. A prolific author and researcher, Dr. Chancellor is pioneering research in tissue engineering and gene therapy for the treatment of bladder and prostate pain and urinary incontinence.



MICHAEL CHANCELLOR, MD

University of Pittsburgh. “But with new nerve-sparing surgical techniques, many men will find that the dysfunction is temporary. ED can also be caused by cardiovascular disease, diabetes, high blood pressure, and neurological disease.”

And it can also be brought on by stress, depression, anxiety, and guilt, as well as certain medications. While Viagra® has brought ED out of the closet, the little blue pill is not effective for everyone, and long-term alternative treatment is certainly needed.

Many men have heard that radical prostatectomy—surgical removal of a cancerous prostate gland—may damage the nerves controlling erections. They are afraid that if the nerves don’t heal, they may be unable to achieve and maintain an erection. A man may worry about how this would affect his relationships, marriage, and self-esteem.

“A man’s fear of losing his potency may keep him from seeking help early, and that can impinge on outcomes,” Dr. Chancellor added.



GENE ANALYSIS OF PROSTATE TISSUES:

RETHINKING OLD PARADIGMS

ROBERT GETZENBERG, PHD

Investigating Different Forms of BPH Reveals Common Patterns

The two most common conditions to hit a man's prostate are prostate cancer, occurring in one out of every six men, and benign prostate hyperplasia (BPH), seen, to one degree or another, in nearly every man as he ages. These processes often co-exist but are thought to have different causes: BPH doesn't cause cancer and cancer doesn't cause BPH. It appears, however, that the genetic bases of these conditions are much more similar than previously thought.

BPH, or an enlarged prostate gland, creates problems with urination for the majority of aging males. The enlarged prostate causes the urethra to constrict, making it difficult to empty the bladder completely, and resulting in urinary frequency. It is as if you filled up your car's gas tank every time it reached half-empty: you would visit the service station twice as often. So, too, for men who don't empty their bladders completely. They need to wake up to urinate several times during the night, or are unable to start or control their urine flow—common manifestations of this condition.

Robert Getzenberg, PhD, director of urological research for the University of Pittsburgh Department of Urology, is conducting cutting-edge genetic research to help scientists better understand BPH, develop markers for the disease, and improve treatment strategies. "Up until now, our evidence has been very subjective and based largely on clinical observations. Prostates were either big or small; frequent urination is either present or absent. We knew little genetically about BPH," he said.

Using DNA microarrays, a computerized bank of 42,000 separate genes, Dr. Getzenberg compared several types of prostate tissues, including normal glands, BPH both with and without severe symptoms, and BPH next to prostate cancer. "The microarrays allow us to integrate the data and look for common genetic patterns," he said.

"We found BPH is not the same," said Dr. Getzenberg. "The most severe form is genetically different." One gene, JM-27, known to be over-expressed in prostate cancer tissues, is also 18 times more prevalent in patients with severe BPH.

BPH and Prostate Cancer May Have Genetic Similarities

When looking at BPH samples from men with prostate cancer but no symptoms of BPH, Dr. Getzenberg's team found something startling: "The genes expressed were similar to those found in severe BPH, making us think that BPH and prostate cancer may have similar pathways at the genetic level," he said. These findings, published in the prestigious journal *Proceedings of the*

National Academy of Sciences, USA, are making scientists and clinicians rethink the common dogma about BPH. "Not only may this lead to a genetic marker for severe BPH, it may give us clues about the early genetic changes in prostate cancer."

Dr. Getzenberg is also leading a national consortium involved in a clinical trial of 3,000 men sponsored by the National Institutes of Health. Seven health centers in the United States are testing drugs to see if they slow the progression of BPH more effectively when used in combination. "We can then access the serum samples of the men who are most responsive to this treatment. Based on our work with gene arrays, this could lead to more personalized treatments and help us better monitor the efficacy of the drugs," he said. **F**

DR. GETZENBERG is the director of urological research in the Department of Urology of the University of Pittsburgh. He is an associate professor of urology, pathology, and pharmacology, and associate director, Cellular and Molecular Pathology Graduate Program. He earned his PhD in biochemistry and cellular and molecular biology at the Johns Hopkins University School of Medicine, Baltimore. He completed a postdoctoral fellowship in cancer pathology at the Yale University School of Medicine. Since Dr. Getzenberg joined the University of Pittsburgh Medical Center in 1994, his widely published research in prostate and bladder cancer has garnered numerous grants from a variety of agencies and foundations, including the National Institutes of Health.

"BPH and prostate cancer may have similar pathways at the genetic level."

CAN DIET CAUSE PROSTATE CANCER?

Researchers Study a Possible Link



UDDHAV P. KELAVKAR, PHD

Identifying Which Patients May Benefit from Low-Fat Foods

One of the hottest debates in cancer treatment and prevention is the question of whether a high-fat diet can increase the risk of getting prostate cancer. Groundbreaking research by Uddhav Kelavkar, PhD, assistant professor of urology at the University of Pittsburgh, could someday determine if diet actually affects some people more than others and may help slow the progression of this widespread disease.

Dr. Kelavkar has identified a particular lipid-metabolizing enzyme, 15-lipoxygenase-1 (15-LO-1), that could play an important role in the formation of prostate cancer tissue. His studies show that 15-LO-1 rises to high levels in human prostate cancerous cells (increasing with increasing Gleason grades) compared with normal prostate tissue. He has also produced aggressive tumors in mice by implanting them with human cancer cells high in 15-LO-1.

Dr. Kelavkar first became interested in this research when he examined the dramatic differences in prostate cancer prevalence between Europeans and Americans vs. Asians and Japanese. “I thought that diet might play a role, since Western diets are higher in omega-6 fatty acids and linoleic acid. Some studies have hinted that these dietary factors increase the risk of prostate cancer,” he said. Omega-6 is found in red meat, while linoleic acid is found in polyunsaturated fats, like sunflower and safflower oils.

“My research is showing for the first time that linoleic acid may be the culprit,”

said Dr. Kelavkar. “I want to find out if it has a stronger effect on individuals who have high 15-LO-1 expressions as compared with the general population. This could become a marker indicating which patients can lower their cancer risk by avoiding a high-fat diet,” Dr. Kelavkar said.

Why Do Normal Cells Turn Cancerous?

Prostate cancer develops at different grades (called Gleason grades) in different patients. When Dr. Kelavkar confirmed

This is a significant development in the path toward better understanding this disease and in tailoring treatment to specific patients based on their known genetic makeup.

that 15-LO-1 levels are higher in more advanced cases, he advanced the theory that it is the 15-LO-1 that makes normal cells turn into cancer cells. This is a significant development in the path toward better understanding this disease and in tailoring treatment to specific patients based on their known genetic makeup. It may even lead to the creation of a 15-LO-1 inhibitor to slow the progression of prostate cancer.

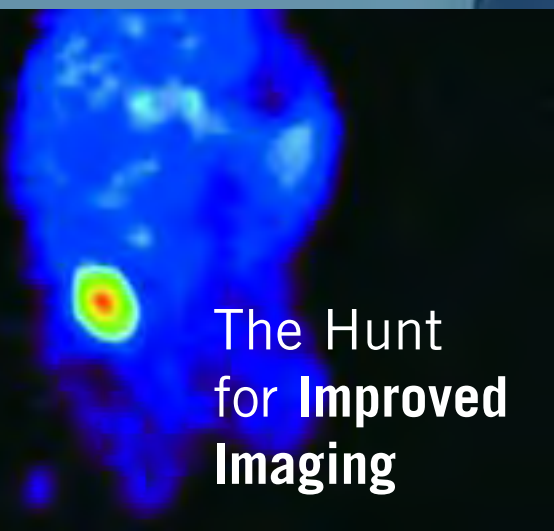
Following radical prostatectomies, some patients still have detectable PSA levels. This type of biochemical failure means that the cancer is still present and, perhaps, may have spread within the body. “Perhaps 15-LO-1 is the driver that keeps the cancer active,” said Dr. Kelavkar. “By inhibiting it, we could suppress the formation of additional tumors for these patients as well.”

Dr. Kelavkar used the University of Pittsburgh’s DNA microarrays, a computerized bank of 42,000 genes, to compare different types of prostate tissues. This valuable resource helps investigators narrow down the important genes and find a pathway that is common in cancer patients. **F**

UDDHAV P. KELAVKAR, PHD, received his doctorate in microbiology from Maharaja Sayajirao University of Baroda, India, in 1993. He did postdoctoral studies on translational control of gene regulation in eukaryotes at the University of Louisville School of Medicine, Kentucky; physical and transcriptional mapping of human chromosome 17 at the Human Genome Laboratory, Clark Atlanta University, Georgia. He was assistant professor at Emory University, Atlanta, and studied the anti-inflammatory activities of 15-lipoxygenase (15-LO). He has obtained funding from the American Cancer Society and the National Institutes of Health to continue his studies at the University of Pittsburgh on the link between 15-LO-1 over-expression and biology of prostate cancer.

Investigating Prostate Cancer PREVENTION

Considering that one man in six will be diagnosed with prostate cancer in his lifetime, studies to find an effective method of preventing prostate cancer are a priority in the Department of Urology. We participate in several large national trials, such as SELECT, which is looking at the effects of vitamin E and selenium on prostate cancer prevention. In addition, the department is conducting clinical studies of the anti-estrogen agent toremifene. In these studies, men are given toremifene prior to radical prostatectomy, and investigators look for changes in the genes and proteins of the prostate gland—changes that are consistent with preventing cancer. Shivandra Singh, PhD, is conducting similar studies of PEITC—an active agent found in watercress and other cruciferous vegetables.



The Hunt for Improved Imaging

Radiographic studies, such as bone scans and CT scans, are often too insensitive to detect the microscopic spread of prostate cancer. Beth Pflug, PhD, an investigator in the Department of Urology, has been studying the potential of positron emission tomography (PET) imaging as a better tool for tumor detection. A PET scan targets overactive biological processes in cancer cells. In comparing normal prostate tissues with prostate cancers, Dr. Pflug has found that prostate cancers have increased production of fatty acids. In tumors grown in mice, the agent ^{11}C acetate targets this increased fat production and can be detected using microPET imaging (see image, above). With refinement, this technique may be able to detect microscopic prostate cancer in men.

Tobago: **HOTBED OF PROSTATE CANCER**

Afro-Caribbean men on the small, idyllic island of Tobago have the highest rates of prostate cancer in the world. Working with epidemiologist Clare Bunker, PhD, surgeons in the Department of Urology have performed 41 radical prostatectomies in a one-room operating suite in Tobago—including the first ever performed there. Rather than basking in the sun, surgeons typically do five operations in two days, and hold clinics late into the evenings. Studies of these men and their prostate tumors will aid our understanding of why prostate cancer is more common and lethal in African-American men.

Drs. Dosumo, Chung, Pound, and Patrick in Tobago



When Potty-Training Goes Awry

By their very nature, urinary problems in children are kept behind closed doors. Although bed-wetting generally resolves itself over time, it is only one of a host



of urinary problems children and their parents face every day. Fortunately, there is help. Pediatric urologist Steven Docimo, MD, recruited by Dr. Nelson

from Johns Hopkins, is an expert in childhood voiding dysfunction. Through biofeedback and other techniques, doctors in a special clinic are helping many of these families. Hsi-Yang Wu, MD, another recently recruited pediatric urologist, is performing laboratory studies of bladder muscle to determine the causes—and to devise better management—of these common problems.

Tumor Banking Pays Huge Dividends

“TAKE IT. WHAT GOOD IS IT TO ME?” This is frequently the response when men are asked to donate their prostate gland to the University of Pittsburgh Tumor Bank. Those specimens, it turns out, are invaluable to our research team. The prostate glands are carefully dissected, frozen, and stored. Acting as the “honest broker,” pathologist Rajiv Dhir, MD, tracks the computer-based patient information, and keeps strict confidentiality for all follow-up studies. All investigators in the department’s cancer laboratories use these tissues as part of their research. The tumor bank, stored adjacent to the laboratories, is among the largest and most comprehensive in the world.

How You CAN

Help

This mission is not for surgeons and scientists alone. **To cure prostate cancer and urologic diseases, we need your help.**

Remember the early computer game called Pong? Two white rectangles representing paddles knocked a small square “ball” between them against an empty black field. Today, computer games are three-dimensional, presenting interactive stories with colorful characters and complex plots.

The revolution in technology that changed computer games has also radically altered science. Research techniques, particularly for those working with DNA in molecular biology, are completely different from what they were even 10 years ago. Scientists who trained more recently are often more knowledgeable about the newest scientific methods than their experienced supervisors. Just as your children or grandchildren probably know more about computers than you do, the younger scientists have more research skills at their disposal than anyone else.

As you’ve seen in the pages of *ForeFront*, we at the University of Pittsburgh Department of Urology favor young, recently trained investigators. Guided by physician–scientists who have expert understanding of the clinical problems, these young, enthusiastic investigators represent the future of our program.

No matter how promising and talented, young investigators are still young, and promise doesn’t pay the bills. Research funding is competitive, and grants go to those with a track record and preliminary results, not great ideas. As a result, most young investigators spend their first two to three years in the laboratory proving that their ideas are worthy of funding by the National Institutes of Health or other

government agencies. As reviewed on page one, the Department of Urology has been very successful in gaining research funding. However, our goal is to find cures and not simply to obtain research money. So we must rely on outside, independent support during this period, both to develop ideas and to keep on the task of translational research.

What is translational research? As our campaign slogan states: “We are on the Mission of Cure.” Just as engineering is the practical application of physics and mathematics, translational research is the practical application of biology to the diseases we see every day. Although scientific discovery is exciting and rewarding to the discoverers, we do not conduct research for its own sake. If we could cure disease by doing poetry, we would fill the labs with laureates, but we do laboratory and clinical research because this is the route to a cure.

This mission is not for surgeons and scientists alone. To cure prostate cancer and urologic diseases, we need your help. We’ve assembled an outstanding team of young researchers in brand-new laboratories to tackle these diseases. However, salaries and top-notch equipment must be paid for. To accomplish this goal, the department is in the midst of a campaign to raise \$15 million; so far, we have over \$4 million in pledges. **Please join us in this important and lifesaving effort. You will find details on how to make a contribution enclosed with this issue of *ForeFront*.** **F**



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You will find details on how to make a contribution enclosed with this issue of *ForeFront*, or please contact:

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