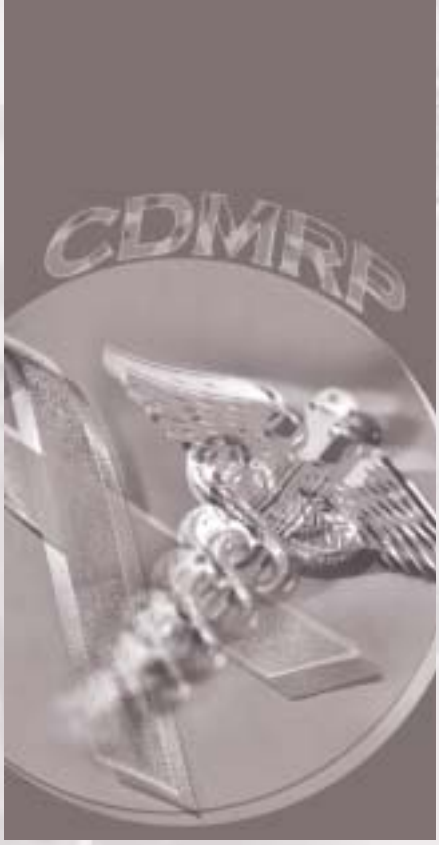


Section IV Prostate Cancer Research Program

Vision: To conquer prostate cancer.

Mission: To promote innovative, multidisciplinary, and regionally focused research directed toward eliminating prostate cancer.

Congressional Appropriations for Peer-Reviewed Research: $210M in FY97–00, $100M in FY01, and $85M in FY02

Funding Summary: 439 awards from the FY97–00 appropriations; 212 awards from the FY01 appropriation; ~200 awards anticipated from the FY02 appropriation

THE DISEASE

Cancer of the prostate is the most commonly diagnosed cancer in men and is the second most common cause of cancer death in men in the United States. In 2002, approximately 30,200 will die from this disease, and an estimated 189,000 men in the United States will be diagnosed with prostate cancer. African American men continue to have higher incidence rates of prostate cancer and are over twice as likely to die from prostate cancer compared to white men.[1] Currently, there is no cure for locally advanced or metastatic prostate cancer.

PROGRAM BACKGROUND

The Department of Defense (DOD) Prostate Cancer Research Program (PCRP) was established in fiscal year 1997 (FY97) by Appropriations Conference Committee Report No. 104-863, which provided $45 million (M) for research in prostate cancer. At that time, the U.S. Army Medical Research and Materiel Command convened a meeting of expert scientists, clinicians, and consumer advocates drawn from academia, urology and oncology organizations, consumer advocate organizations, industry, military, and cancer research funding agencies. This group defined the goals and areas of emphasis of the program and identified underrepresented avenues of research and novel applications of existing technologies. The mission of the PCRP is to promote innovative, multi-institutional, multidisciplinary, and regionally focused research directed toward eliminating prostate cancer.

From FY97–02, Congress appropriated a total of $395M to fund peer-reviewed prostate cancer research through the PCRP. A total of 651 awards have been made through FY01 across the categories of research, training/recruitment, and infrastructure. The PCRP has developed a multidisciplinary research portfolio that encompasses both basic and clinical research aimed at preventing, detecting, treating, and improving the quality of life of those afflicted with prostate cancer. Each fiscal year's investment strategy focused on the program's vision to conquer prostate cancer. Appendix B, Table B-2, summarizes the congressional appropriations and the investment strategy executed by the PCRP for FY01–02. Additional details of the FY97–00 programs may be found in the DOD Congressionally Directed Medical Research Programs Annual Reports of September 1999, September 2000, and September 2001.

"The dedication of the scientists and clinicians funded by the PCRP, the passion of the consumer advocates who are an integral part of this program, and the commitment of the CDMRP staff and contractors who make it all happen, are truly remarkable. It is an honor to be part of this alliance as we strive to reach the PCRP's goal of eradicating prostate cancer."

—Leo Giambarresi, Ph.D., PCRP Program Manager

FY01 PROGRAM

Congress appropriated $100M in FY01 to continue the peer-reviewed DOD PCRP. The FY01 PCRP challenged the scientific community to design innovative prostate cancer research that would foster new directions, address neglected issues, and bring new investigators into the field. Awards were made in areas that represent underinvestigated avenues of research or novel applications of existing technologies. The programmatic vision was implemented by requesting proposals in three award categories: (1) research, (2) infrastructure, and (3) training/recruitment. Table IV-1 provides a summary of the FY01 PCRP award categories and mechanisms in terms of number of proposals received, number of awards, and dollars invested. As illustrated in Figure IV-1, the portfolio of research supported by the FY01 PCRP is diverse.

[1] *American Cancer Society - Cancer Facts and Figures 2002.*

Table IV-1. Funding Summary for the FY01 PCRP

Category and Award Mechanism	Number of Proposals Received	Number of Awards	Investment
Research			
Clinical Trial	*21*	*5*	*$5.8M*
Idea Development	*394*	*102*	*$53.3M*
New Investigator	*286*	*69*	*$22.6M*
HBCU Collaborative Partnership Awards	*2*	*2*	*$1.2M*
Health Disparity Research-Prostate Scholar Awards	*6*	*1*	*$.4M*
Infrastructure			
Consortium Development	*16*	*5*	*$.7M*
Training/Recruitment			
Health Disparity Training Prostate Scholar Awards	*3*	*2*	*$.4M*
Postdoctoral Traineeship	*54*	*26*	*$2.4M*
Total	***782***	***212***	***$86.8M***

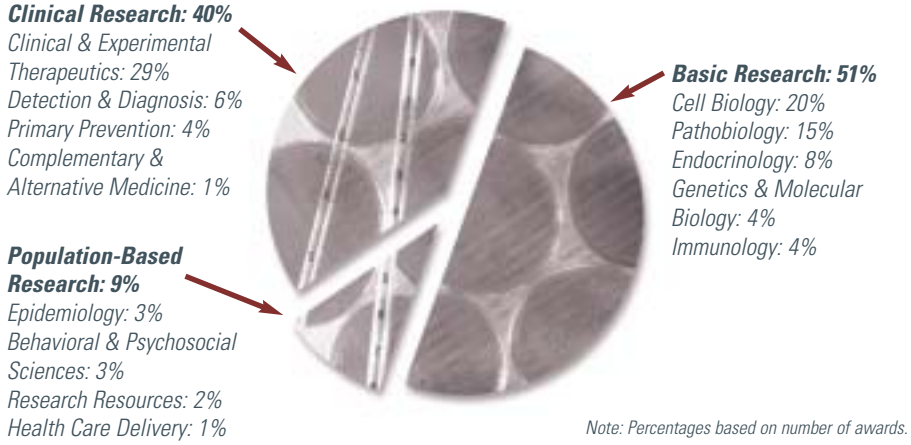
The PCRP continued its emphasis on innovation by awarding 171 Idea Development and New Investigator grants. Both awards aim to stimulate and reward creative research ideas that represent the start of something new, or create or introduce a unique or unusual approach to the study of prostate cancer.

In FY01, an exciting new infrastructure award mechanism was launched–Consortium Awards. This award mechanism is intended to support major, coordinated, goal- and product-driven synergistic research efforts that involve the nation's leading researchers. Consortium Awards are being executed in two phases spanning both FY01 and FY02.

Another award mechanism offered for the first time in FY01 was the Prostate Cancer Clinical Trial Award. This award mechanism is intended to support prospective Phase 1 and Phase 2 clinical trials focused on new prostate cancer therapies or treatments. Five clinical trials were supported by the FY01 PCRP. See related box story on pages IV-6 and 7 for highlights of the FY01 PCRP-funded clinical trials.

To address the disparate prostate cancer incidence, morbidity, and mortality among African Americans, two new award mechanisms were developed in FY01 to provide training and research support in this area: Health Disparity Training – Prostate Scholar Awards and Health Disparity Research – Prostate Scholar Awards.

A combined total of 3 awards were made in these award mechanisms to provide training and research opportunities that focus on important questions on prostate cancer disparity. Additionally, HBCU Collaborative Partnership Awards were offered for the first time in FY01 to provide support at the institutional level between an applicant HBCU and collaborating institution with the goal of increasing the number of HBCU scientists and clinicians studying prostate cancer. As with the Prostate Scholar Awards, the focus of the HBCU Collaborative Partnership Awards is on the ethnic disparity in prostate cancer. Learn more about the PCRP's emphasis on prostate cancer disparity by reading the box story on page IV-4.



Note: Percentages based on number of awards.

Figure IV-1. FY01 PCRP Portfolio by Research Area

Encouraging Research on Prostate Cancer Disparity

While cancer strikes all ethnic and economic groups in the United States, its burden falls especially hard on minority and medically underserved populations, especially African Americans. The reasons for these disparities in incidence and mortality are not entirely clear, nor are the best ways to prevent or treat cancers in any U.S. minority population well understood. To address these issues, the PCRP has developed special funding mechanisms to support research about minority populations. Some of these funding mechanisms provide training and research efforts focused on the disparate burden of prostate cancer in African Americans. For instance, in FY01, the PCRP offered Health Disparity Research-Prostate Scholar Awards and Health Disparity Training-Prostate Scholar Awards to provide young investigators the opportunity to establish research careers focused on prostate cancer in the African American Community. Investigators supported under these award mechanisms include:

Marva Price, R.N., Dr. P.H., *Duke University: Dr. Marva Price is using her background in public health issues to identify factors that inhibit African American men from seeking screening for prostate cancer. This research is being conducted to narrow the disparate gap in early identification and treatment of prostate cancer among African American men.*

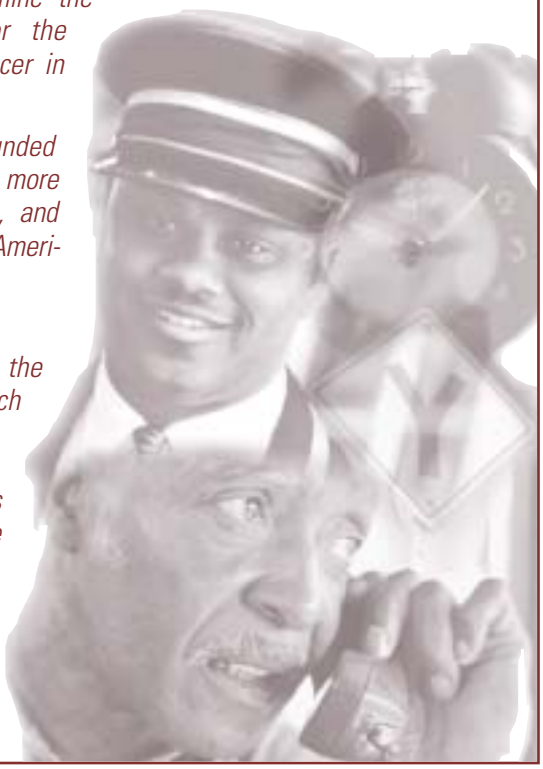
Lorrie Powel, Ph.D., *Edith Nourse Rogers Memorial Veterans Hospital: Under the mentorship of Dr. Jack Clark, Dr. Powell will be extending her postdoctoral studies to investigate the emotional impact of post-prostatectomy urinary incontinence by identifying the socio-cultural and personal meaning that African American men attach to prostate cancer.*

Matthew L. Freedman, M.D., *Dana Farber Cancer Center: Under the mentorship of Dr. David Altshuler. Dr. Freedman will be conducting research to determine the underlying genetic basis for the increased risk of prostate cancer in African American men.*

Results from these PCRP-funded studies will continue to lead to more effective screening, prevention, and treatment strategies for African Americans with prostate cancer.

Need More Information?

Additional information about the CDMRP's minority research programs can be obtained by contacting Dr. Barbara Terry-Koroma, Special Populations Program Manager, at the CDMRP, 1077 Patchel Street, Fort Detrick, MD 21702-5024; phone: 301-619-7071; or by visiting the CDMRP web site at http://cdmrp.army.mil.



The Vision for the FY02 Program

Congress continued the program in FY02 with an $85M appropriation. The FY02 PCRP continued its emphasis on innovative, high-risk/high-gain research by supporting Idea Development and New Investigator Awards. Additionally, support for training was encouraged and the second phase of the Consortium Awards was executed.

A total of 699 proposals were received electronically, as detailed in Table IV-2. Scientific peer review was conducted in July and August 2002, and programmatic review is scheduled for October 2002. Approximately 200 awards are anticipated.

Scientific Outcomes and Advances

PCRP award outcomes are exciting and present promise for the future. The outcomes of PCRP-funded research can be gauged, in part, by the number of resultant publications, abstracts/presentations, and patents/licensures reported by awardees. This information is summarized in Table IV-3. The following highlighted projects represent a sampling of some of the exciting advances in prostate cancer research. This broad portfolio of research is laying the foundation for increasing basic knowledge about prostate cancer, treating prostate cancer, improving the lives of individuals affected by this

Table IV-2. Award Mechanisms Offered and Proposals Received for the FY02 PCRP

Category and Award Mechanism	Number of Proposals Received
Research	
Idea Development	487
New Investigator	141
Health Disparity Research-Prostate Scholar Awards	10
Infrastructure	
Consortium Development	5
Training/Recruitment	
Health Disparity Training-Prostate Scholar Awards	2
Postdoctoral Traineeships	54
Total	**699**

disease, and preventing prostate cancer. These examples represent the research of dedicated investigators working to support the program's vision of conquering prostate cancer.

African Americans, Genetic Diversity, and Prostate Cancer. *Rick A. Kittles, Ph.D., National Human Genome Center, Howard University:* African American men are known to have a higher risk of developing prostate cancer, present with disease at a higher stage, and have a worse outcome from the disease than non-African American men. The debate continues within the medical field about whether this disparity is due to biological, environmental, or behavioral factors, or a combination of these factors. As part of larger projects in African American genomic research, scientists at Howard University's National Human Genome Center have been studying the role of gene polymorphisms in prostate cancer incidence and severity in African American men. A slight change in sequence in the control region of the CYP17 gene, which plays an important role in androgen biosynthesis, was found to be significantly associated with increased prostate cancer risk and clinically advanced disease in African American men compared to control groups of either Nigerian men or European-American men. Studies with a different gene involved in testosterone deactivation, CYP3A4, initially found correlations between a mutation in a control region of this gene and prostate cancer. However, when the data were corrected for contributions of different ethnic groups that may be a part of the ancestry of the African American, no significant correlation was found between the CYP3A4 mutation and prostate cancer. The researchers at the National Human Genome Center are continuing these and other studies to find the influence of genomic variation on prostate cancer and other diseases that affect African Americans.

Life and Death Decisions in Prostate Cancer Cells. *Anning Lin, M.D., Ph.D., The University of Chicago:* Programmed cell death, or apoptosis, is an important mechanism used to kill foreign cells in the body. In the healthy body, apoptosis is a normal part of development, such as for the formation of an embryo's fingers and toes, or to maintain normal cell function. Complex networks of cellular factors working in synergy induce apoptosis to occur under normal situations. A more delicate balance between apoptosis and growth of cells is needed in this

Table IV-3. FY97-99 PCRP Award Outcomes

Number of Awards	**297**
Publications in Scientific Journals	**>450**
Abstracts/Presentations at Professional Meetings	**>250**
Patents/Licensures (including applications)	**>25**

"In the war against prostate cancer, the DOD PCRP has proven to be an extremely effective and innovative means of attacking the disease. Through novel approaches to funding, it has brought countless new investigators' innovative translational programs to bear on curing the disease. Clearly, when the history of the fight against prostate cancer is concluded, the DOD will have played a decisive role in the successful campaign."

—Ralph de Vere White, M.D., Director, University of California Davis Cancer Center, FY02 PCRP Integration Panel Chair

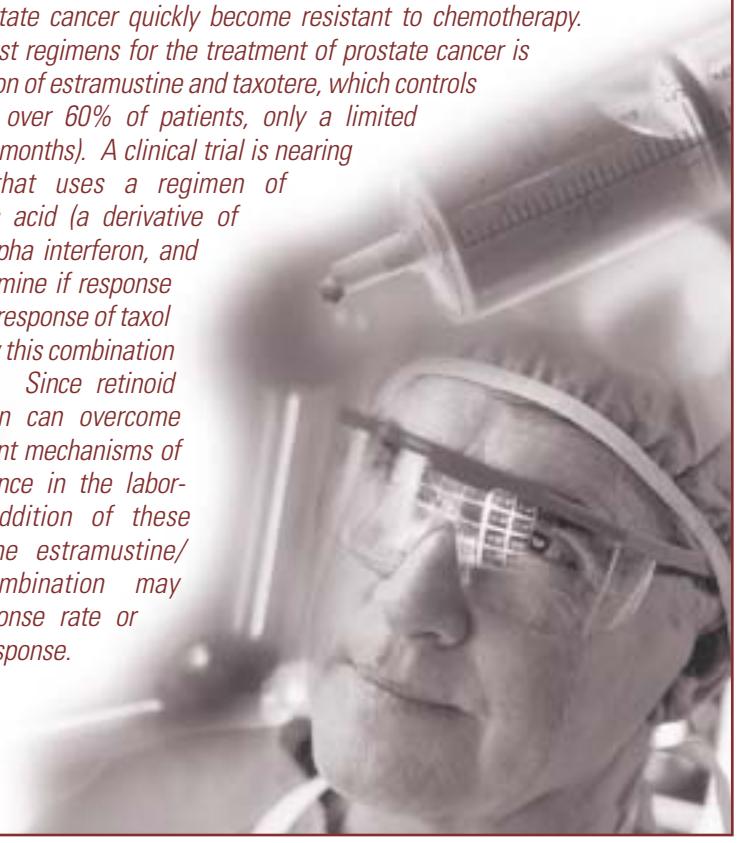
pathway when the survival of normal cells or death of malignant cells needs to be determined. Dr. Anning Lin of the University of Chicago has been examining tumor necrosis factor-alpha (TNF-α), a cytokine that is known to both induce cell death and allow cells to survive. Interestingly, many prostate cancer cells are not as sensitive to apoptosis caused by TNF-α as normal prostate cells. He and his colleagues have meticulously dissected two chemical pathways used by TNF-α to allow cells to either live or die following TNF-α exposure. Understanding the processes allowing enhanced survival of prostate cancer cells to apoptosis may allow development of targeted therapies that can pinpoint and suppress the cell's ability to survive apoptosis, therefore allowing the cancer cells to succumb to traditional therapies.

Developing Alternative Treatment Options to Prostatectomy for Prostate Cancer Treatment. *Wadih Arap, M.D., Burnham Institute:* For many men diagnosed with prostate cancer, the treatment options are limited to prostate removal, or prostatectomy, radiation, hormone therapy, or "watchful waiting." All of these treatment options present either side effects with treatment, prolonged recovery times, or potential quality of life issues with their use. Many men choose prostatectomy over other options presented by their physician because it removes the source of the problem. However, surgery may not be right for all prostate cancer patients. Treatment options that eliminate the need for surgical intervention and decrease

FY01 PCRP Clinical Trials Recipients – Moving Us Closer to New Prostate Cancer Therapies

Kim Chi, M.D., *Vancouver General Hospital: A Phase 1/2 Study of the Combination of Neoadjuvant Hormone Therapy and Weekly OGX-011 Prior to Radical Prostatectomy for the Treatment of Localized Prostate Cancer: Androgen ablation (AA) therapy has been shown to prolong life in men with advanced prostate cancer, and is frequently used prior to surgery and radiation in patients at high risk of relapse. Unfortunately, despite high initial response rates, remissions are temporary in advanced disease because of the survival of resistant cancer cells. An increase in the expression of survival genes is a mechanism of resistance to treatment. Clusterin, a survival gene, has been shown to increase in expression following AA therapy. Dr. Chi has been funded to begin a Phase 1/2 clinical study to evaluate the clinical, pathologic, and biologic effects of OGX-011, an antisense oligonucleotide directed against clusterin, given in combination with AA in patients with prostate cancer prior to radical prostatectomy. In preclinical studies, OGX-011 was able to selectively inhibit the expression of clusterin resulting in an improved response to AA, radiation, and chemotherapy. The changes observed in clusterin expression before and after treatment will be used to guide drug development and improve understanding of the biology of prostate cancer.*

Robert DiPaola, M.D., *Robert Wood Johnson Medical School, University of Medicine and Dentistry of New Jersey and The Cancer Institute of New Jersey: A Phase 1/2 Trial of 13-Cis Retinoic Acid, Alpha Interferon, Taxotere, and Estramustine (R.I.T.E.) for the Treatment of Hormone Refractory Prostate Cancer: Prostate cancer that has spread beyond the prostate is only temporarily controlled with lowering testosterone through surgical or medical castration. Patients with hormone-resistant prostate cancer quickly become resistant to chemotherapy. One of the best regimens for the treatment of prostate cancer is the combination of estramustine and taxotere, which controls the tumor in over 60% of patients, only a limited duration (6–8 months). A clinical trial is nearing completion that uses a regimen of 13-cis-retinoic acid (a derivative of vitamin A), alpha interferon, and taxol to determine if response or duration of response of taxol is improved by this combination of treatment. Since retinoid and interferon can overcome some important mechanisms of tumor resistance in the laboratory, the addition of these agents to the estramustine/taxotere combination may improve response rate or duration of response.*

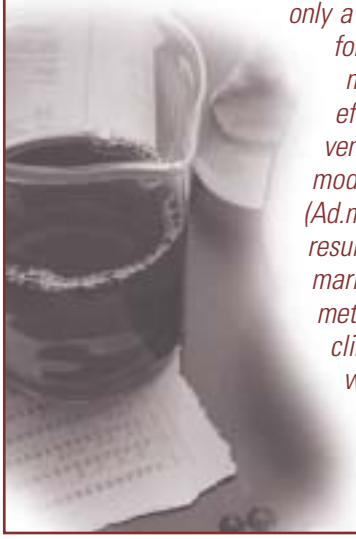


FY01 PCRP Clinical Trials Recipients – Moving Us Closer to New Prostate Cancer Therapies

Mitchell Sokoloff, M.D., *University of Chicago: Neoadjuvant Anti-Angiogenesis Therapy for Prostate Cancer: Some men develop prostate cancer that is likely to fail surgery or radiation therapy due to characteristics inherent to their cancers. These characteristics, including elevated preoperative prostate specific antigen (PSA) levels, high Gleason grades, and large volume disease felt during digital rectal examination. Men with these characteristics are considered high risk where failure rates after surgery or radiation therapy approach 30% to 50%. This trial will target men with prostate cancer who are at a high risk for recurrence after surgery or radiation therapy and treat them with an anti-angiogenic drug prior to surgery (neoadjuvant). This drug is designed to kill tumor cells by choking off their blood supply, combined with androgen ablation therapy, aiding in our ability to control prostate cancer.*

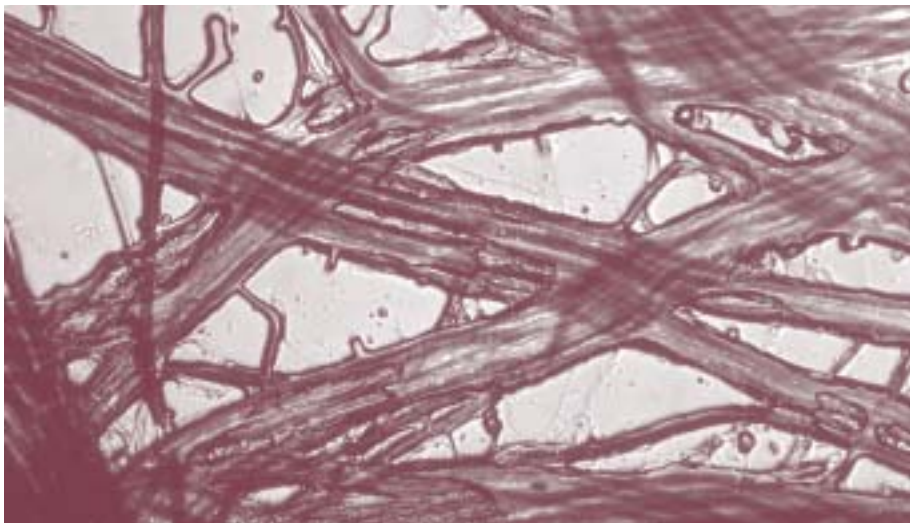
Eugene Kwon, M.D., *Mayo Clinic, Rochester, and Eric Small, M.D., University of California, San Francisco: A Multi-Institutional Phase 2 Immunotherapeutic Trial for the Treatment for Advanced Prostate Cancer: AA therapy (Lupron, Zolodex, Flutamide, Casodex, and/or castration) has long served as a mainstay for patients with advanced prostate cancer. However, this form of therapy is typically not curative, and many of the 35,000 patients who will die of prostate cancer each year will do so after failing AA therapy. Immune-based therapies, such as vaccination with prostate cancer vaccines, antibodies, or other immune boosters that can act in combination with AA therapy, may help to elicit a curative rather than transient response to treatment. Hence this clinical trial is designed to examine the capability of AA therapy to "jump-start" a prostate-specific immune response that could then be greatly amplified by an antibody called MDX-CTLA-4, which blocks a receptor on patient T cells and keeps the T cells in an active state. Thus, T cells triggered by AA therapy could then be stimulated by MDX-CTLA-4 to evoke a more complete treatment response than would be observed with AA therapy alone, since many of the T cells are likely able to recognize prostate tumor cells, eliminate them, and overall improve prostate cancer treatment.*

Simon Hall, M.D., *Mount Sinai School of Medicine: Phase 1 Trial of Adenovirus-Mediated IL-12 Gene Transduction in Patients with Radiorecurrent Prostate Cancer -Radiation therapy fails in many men, especially in those cancers with aggressive pathological characteristics or high tumor burden (e.g., serum PSA levels >10). With only a few options most patients are treated with some form of hormone therapy. Cytokine gene therapy may be able to provide both local and systemic effects without the toxicity associated with intravenous delivery of recombinant cytokines. In a mouse model of prostate cancer, adenovirus-mediated (Ad.m) transduction of the cytokine IL-12 (Ad.mIL-12) resulted in local prostate cancer growth suppression, marked survival enhancement, and inhibition of metastases. The use of Ad.mIL-12 in patients with clinically localized radiorecurrent prostate cancer will be examined in a Phase 1 trial, which is designed to control prostate cancer in patients with few viable options.*



recovery time following treatment would be a welcome addition to the choices that these men need to make, especially for older prostate cancer victims where surgery may present more risks than benefits. PCRP-supported studies were done with mice that develop prostate cancer and were given small peptides that specifically target prostate blood vessel structures. In mice treated with these peptides, there was marked destruction of the prostate gland but no damage to other organs. In addition, the peptide-treated mice showed a delay in prostate cancer development equivalent to several years of a human's life. The mice treated in this way also remained fertile after the treatment despite the destruction of the prostate. This treatment option may give both younger and older men diagnosed with prostate cancer treatment options that preserve sexual function and improve quality of life without invasive surgery.

Prostate Cancer Nerve Interactions in Metastasis. *Gustavo E. Ayala, M.D., Baylor College of Medicine:* In prostate cancer, the most common way that cancer cells escape from the prostate (metastasize) is by traveling along the nerve cells. This process, termed perineural invasion, is similar to cars traveling on a highway; the prostate cancer cells travel along nerves that form a path or highway of least resistance. Beyond this purely mechanistic explanation, little is known about how this process occurs. Researchers at the Baylor College of Medicine are trying to understand this process by studying how prostate



cancer cells interact with neural cells. They have demonstrated, with cultured cells, that prostate cancer cells actually cause nerve branches to grow directly toward them, establishing contact between the two cell types. Once in contact, the cancer cells travel along the nerve branches back to the main body of the nerve where they can metastasize to other parts of the body. It is noteworthy that these cultured cells wrap around nerve cells in the same manner that human prostate cancer wraps around nerves in the prostate. The researchers also observed that nerves grow more quickly in the presence of cancer cells, and that the prostate cancer cells grow more quickly in the presence of nerves. Such interactions are most likely controlled through chemical signaling pathways that have yet to be discovered. The researchers intend to further investigate if nerve-epithelial cell interactions are exclusive to prostate cancer, or if they can occur either in cancers of other organs (e.g., the pancreas or colon), or in benign conditions such as those associated with benign prostate hyperplasia. Understanding the specific mechanisms of these cancer cell/nerve cell interactions is key to developing therapeutic strategies that target the chemical factors that define the ability of prostate cancer to metastasize.

Painting Pictures of Prostate Cancer Genetic Mutations. *Jeremy Squire, Ph.D., Ontario Cancer Institute:* Medical treatment of prostate cancer patients is based on assessment of the aggressiveness of the cancer. Patients with dormant prostate cancer may not need to undergo the more extensive and potentially debilitating treatments required by patients with more aggressive cancer. However, upon initial diagnosis, it is often difficult to immediately determine how fast a tumor may grow or spread. New laboratory methods are needed that allow early detection of aggressive forms of prostate cancer. Investigators at the Ontario Cancer Institute are working on new techniques to identify genetic and chromosomal changes in early cancers that may indicate aggressive types of prostate cancer. One of these techniques is known as interphase fluorescent in situ hybridization, or IFISH, which incorporates 24 different fluorescent colors, resulting in "painted" chromosomes uniquely identified by color. Advanced computer technology is used to rapidly identify and detect minor changes to the number or structure of the chromosomes in each cell analyzed. The investigators tested the ability of IFISH on histological specimens of cancer in patients with high-grade prostatic intraepithelial neoplasia (HPIN) to predict progression to prostate cancer. HPIN currently is believed to be the earliest stage of prostate cancer, but current techniques cannot predict whether HPIN will progress to aggressive prostate cancer. In a patient population

divided into those initially diagnosed with HPIN but after 3 years of follow-up developed either benign tumors or progressive prostate cancer, IFISH analysis of the initial biopsies from these patients showed that patients who eventually developed progressive prostate cancer tended to have a greater number of chromosome abnormalities in the prostate cells. It is hoped that further advances in the use of IFISH, and complementary technology under development in the investigators' laboratory, will soon allow early detection of aggressive prostate cancers.

Herpes Simplex Virus as a Therapeutic Agent for Prostate Cancer.
Ian Mohr, Ph.D., New York University School of Medicine: Genetic engineering techniques have made it possible to use viruses to combat cancer by infecting and killing tumor cells without damaging normal cells or causing viral-related disease. Unfortunately, the genetic modifications that limit viruses' ability to overcome the body's normal defenses also limit replication of viruses in tumor cells. As a result, antitumor viral agents often do not completely eradicate tumor cells. Investigators at New York University School of Medicine recently have isolated through genetic selection in cancer cells a mutant of herpes simplex virus-1, the virus that causes cold sores, with enhanced antitumor properties. This mutant, called a suppressor virus, replicated at greater levels in prostate cancer cells and was an effective antitumor agent in an animal model of human prostate cancer. In fact, treatment with suppressor virus appeared to eliminate prostate tumor cells in some animals. More importantly for the overall health of an individual, the mutations that enhanced antitumor effectiveness in the suppressor virus did not produce an increased risk of widespread viral infection. By determining the genetic components that confer these properties to the mutant suppressor virus, the investigators have formed a foundation for the design of more effective and safer viral antitumor agents. The greatest benefit of this work ultimately will come when this advance is translated into the development of therapeutic agents for the treatment of human prostate cancer.

"Having been aware of the "War Against Cancer" for over 25 years, it was important to get a view from the `front!' Understanding the complexities of the research process and seeing that actual progress is being made lends credence to the belief that we WILL end cancer in my lifetime. The PCRP brings the amorphous vision of `research' down to a tangible form of names and places and how we are moving to defeat the disease that has brought carnage to so many lives in our communities. We need to be more aggressive in presenting the message of this program in order to provide hope and accountability to those who need to know—our society."
—Virgil Simons, FY02 Ad Hoc Member of the Integration panel

Summary

Since 1997, the DOD PCRP has been responsible for managing $395M in congressional appropriations, resulting in 651 awards for FY97–01 that are directed toward eliminating prostate cancer. The diverse portfolio of funded research is already making important contributions to understanding, preventing, detecting, diagnosing, and treating prostate cancer.

FY02 Integration Panel Members

Chair, Ralph DeVere White, M.D.: Medical Director of the Cancer Center, and Professor and Chair of Urology, University of California, Davis.

Chair Elect, Frederic Waldman, M.D., Ph.D.: Professor, Department of Laboratory Medicine, University of California, San Francisco. Director, DNA Cytometry Service and Director, Molecular, Cytogenetics Core, University of California San Francisco Cancer Center.

Chair Emeritus, Carl Olsson, M.D.: Professor and Chairman, Department of Urology, College of Physicians and Surgeons, Columbia University. Director of Urological Service, The Presbyterian Hospital and the Squier Urological Clinic.

Thomas E. Carey, M.D.: Director, Laboratory of Head and Neck Cancer Biology, Department of Otolaryngology Laboratory, University of Michigan.

Donald Coffey, Ph.D.: Catherine Iola and J. Smith Michael Distinguished Professor of Urology, Professor of Oncology, Professor of Pharmacology and Molecular Sciences, and Professor of Pathology, The Johns Hopkins School of Medicine. Director of Research Laboratories, Department of Urology.

Robert Dreicer, M.D.: Director, Urologic Oncology, Department Hematology and Oncology, The Cleveland Clinic Foundation.

Winston Dyer: Consumer; Member of the Board of Directors, CapCURE.

Phillip Kantoff, M.D.: Director, Prostate Cancer Program at the Dana-Farber Harvard Cancer Center and Director, Lank Center for Genitourinary Oncology, Harvard University.

Ronald Lieberman, M.D.: Program Director, Prostate and Urologic Cancer Research at the National Cancer Institute.

Monica Liebert, Ph.D.: Director, Office of Research, American Urological Association.

Ronald Morton, Jr., M.D.: Chief of Urology, Houston Veterans Affairs Medical Center. Director of Laboratories, Baylor Prostate Center, Baylor College of Medicine.

Gail S. Prins, Ph.D.: Professor of Physiology, Departments of Urology, Physiology, and Biophysics, University of Illinois at Chicago.

Mack Roach III, M.D.: Associate Professor, Radiation Oncology, Medical Oncology, and Urology, University of California, San Francisco.

Howard R. Soule, Ph.D.: Executive Vice-President and Chief Science Officer, CapCURE.

Nicholas Vogelzang, M.D.: Fred C. Buffet Professor of Medicine and Surgery (Urology), University of Chicago. Director, Genitourinary Program. Director, University of Chicago Cancer Research Center.