Newsletter

Prostate Cancer 101, Inc.

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

New Test Improves Assessment of Prostate Cancer Risk, Study Says

By ANDREW POLLACK

A new test can help distinguish aggressive prostate cancer from less threatening ones, potentially saving many men from unneeded operations for tumors that would never hurt them, researchers are reporting.

The test, developed by Genomic Health, could triple the number of men who could confidently monitor their tumors rather than undergo surgery or radiation treatments, according to the company and to researchers.

Results of a study assessing the test's performance will be presented Wednesday at the annual meeting of the American Urological Association in San Diego.

Many of the 240,000 cases of prostate cancer diagnosed each year in the United States are considered to pose a low risk of hurting or killing the man. But sometimes those assessments are wrong. So many men, reluctant to take the chance, undergo treatments that can cause impotence and incontinence.

"It's very hard to tell a surgeon 'I'd like to leave a cancer in place,' " said Dr. Jonathan Simons, president of the Prostate Cancer Foundation, a research and advocacy organization. "Having objective information is going to help a lot of patients make that decision."

Dr. Simons, who was not involved in the study, said the development of new genetic tests like the one from Genomic Health represented a "watershed," akin to going from pulse rate measurements to electrocardiograms in cardiology.

Still, some experts said it was too early to assess how accurate the test really was and whether it would make a difference in men's decisions. Insurers are going to want to know that before deciding to pay for the test, which will be available starting Wednesday at a list price of $3,820.

Even the senior investigator of the study, Dr. Peter R. Carroll, said he was not sure.

"Certainly for a group of men it will have an impact," Dr. Carroll, who is chairman of urology at the University of California, San Francisco, said in an interview.

"The question is how many men and how many physicians."

The new test, which is called the Oncotype DX Prostate Cancer Test, is one of more than a dozen coming to market that use advanced genetic methods to help better manage prostate cancer. The most direct competitor to the Oncotype test is likely to be the Prolaris test, introduced last year by Myriad Genetics.

But Genomic Health's test has attracted attention because of the company's track record. It already sells a similar test for breast cancer, also Oncotype DX, that is widely used to help women decide whether they can forgo chemotherapy after their tumor is surgically removed.

Some analysts say that with the breast cancer test facing intensified competition, the company's future growth could hinge on the prostate test, which could take time to gain acceptance. Genomic Health's stock closed Tuesday at $33.87, up 1 percent.

The test looks at the activity level of 17 genes in the biopsy sample and computes a score from 0 to 100 showing the risk that cancer is aggressive.

To see how well the test worked, testing was performed on archived biopsy samples from 412 patients who had what was considered low or intermediate-risk cancer but then underwent surgery.

In many such cases, the tumor, which can be closely studied after it is surgically removed, turns out to be more aggressive than thought based on the biopsy, which looks at only a tiny sample of the tumor.

The researchers found that the Oncotype test predicted such unfavorable pathology more accurately than existing methods, which depend mainly on the Gleason score based on how the biopsy sample looks under the microscope.

Genomic Health said that 26 percent of the samples were classified as very low risk by its test, compared to only 5 to 10 percent for the existing methods. In some cases, however, the new test showed the cancer to be more aggressive than the existing methods.

Some experts not involved in the study were cautiously optimistic.

"They showed a pretty good correlation with the score and how it predicts things," said Dr. E. David Crawford, a professor of urology, surgery and radiation oncology at the University of Colorado. He has consulted for Myriad Genetics and said he might become a consultant to Genomic Health.

Dr. Stacy Loeb, assistant professor of urology at New York University, said, "I think it will help — they definitely showed it improves upon what we are using now." She said it was not clear, however, how the Genomic Health and Myriad tests compared to each other.

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New Test Improves Assessment of Prostate Cancer Risk, Study Says.

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Building a Better Mousetrap to catch Prostate Cancer

New urine test for prostate cancer improves utility of the PSA blood test, increases doctors' ability to pick out high-risk tumors from low-risk tumors in patients, and may help tens of thousands of men avoid unnecessary biopsies.

Drs. Scott Tomlins and Arul Chinnaiyan of the University of Michigan discovered a gene fusion abnormality known as T2:ERG that is present in half of all prostate cancers and is thought to be an initiator of this disease. A new urine test, based in part on detecting T2:ERG, is now available to aid in early detection of prostate cancer.

When scientists and doctors disagree over the finer points of our medical care, the best the rest of us can do is talk with our own doctors, read about the debate so we are as informed as possible, and perhaps do a bit of advocacy to fund the research needed to solve the debate. In May of last year, one such debate erupted when a group of physicians and scientists convened by the U.S. government to make health care recommendations for the general public changed course from prior years and called for an end to the routine use of a blood test that screens men for prostate cancer.

Currently there is sharp disagreement over that recommendation within the medical profession. For example, a survey of doctors conducted by Johns Hopkins Medicine found "serious pushback" from primary care doctors over ending these routine cancer screens. Formal evidence of pushback against the governmental task force's recommendation to end routine screening came in May of this year, with the American Urological Association's issuance of their own set of guidelines that called for selective routine screening.

The test in question is called PSA for prostate-specific antigen; it measures the blood level of the PSA protein that is produced by the prostate gland in all men. As PSA levels rise, so do the chances a man has prostate cancer; but benign conditions can also cause high levels of PSA in the blood. This means that the PSA protein is tissue specific in nature—only the prostate gland can make the protein—but not cancer specific.

Because higher levels of PSA in the blood—generally greater than 4.0 ng/ml—are considered suspect for a cancerous prostate gland, most doctors have routinely recommended a needle insertion biopsy for men with PSA levels of 4.1 or greater to test, more definitively, for the presence of cancer. While less than half of those biopsies find cancerous cells, the cancers found are identified early, at a stage when cure is likely. (Research has also shown that 15% of men with PSA levels of 0 to 4.0 ng/ml have prostate cancer and of those men, 15% have high-risk disease, thus PSA is far from perfect at capturing clinically relevant cancers.) And even in men whose PSA levels are high

enough to trigger a biopsy, accurate results are not assured: biopsy tests may miss some cancers (on average less than 1% of the prostate gland is sampled at biopsy). And because up to 44% of those PSA-induced biopsies find cancerous cells that are non-lethal—meaning they'd not shorten the lifespan of the man—many men are overtreated for prostate cancer.

Thus, overall, the PSA test has serious drawbacks. In essence, it leads to finding too many prostate cancers and, ironically, not enough.

From its inception in the mid 1980s, to its widespread use as a screening tool for asymptomatic men in the early 1990s, the PSA test has been incredibly effective at reducing the U.S. death rate from prostate cancer in much the same way routine Pap smears reduced the death rate of cervical cancer in women after the test's mainstream introduction in mid 20th century. (A study published last August in the journal Cancer calculated that without widespread PSA testing in use today, the number of men diagnosed with advanced, metastatic prostate cancer would triple.) Now the challenge is to reduce the treatment rate of what doctors call indolent prostate cancers—those that do contain abnormal prostate cells, but whose abnormalities would likely never morph into aggressive, lethal, symptomatic forms of the disease—while simultaneously assuring that high-risk disease is not undertreated. One way to accomplish that would be to introduce a test that is prostate-cancer specific as opposed to prostate-tissue specific, with the aim of better informing patients and their doctors of the need for biopsy after PSA screening.

Sampling urine too

A new urine test has been developed that is based on two extremely prostate-cancer specific biomarkers and is now available to patients through the University of Michigan MLabs. The test measures two molecular markers that are distinct to prostate cancer and only prostate cancer. One marker is a measurement of RNA made from the PCA3 gene; prostate cancer cells churn out extremely high levels of this PCA3 RNA which can be detected in urine and more than 95% of all prostate cancers overexpress PCA3 RNA. (A recent study by the National Cancer Institute showed that a positive PCA3 test predicted a positive biopsy 80% of the time at initial biopsy; and, for men undergoing repeat biopsy, a negative urine test predicted a negative biopsy 88% of the time.) The second marker is a measurement of RNA made from the fusion of two genes—TMPRSS2 and ERG. Normally the ERG gene, a potent oncogene, is turned off in prostate cells and its protein is not expressed or produced. But in 50% of prostate cancer foci, the ERG gene fuses abnormally with the TMPRSS2 gene, which is located close by on the same chromosome—number 21. When this happens, ERG is turned on and high levels of a slightly shortened form of the ERG protein is made. When this aberrant fusion RNA is detected in men's urine at high levels it is ultra specific for the presence of prostate cancer.

PCF-funded Scott Tomlins, MD, PhD, an assistant professor of pathology and urology at the University of Michigan and a co-discoverer in 2005 of what is now commonly known as the TMPRSS2:ERG fusion, says that evidence shows that if TMPRSS2:ERG is detectable at high levels in urine, a man likely has prostate cancer, whether or not his biopsy is positive for cancer. "If you wanted to design a way to cause prostate cancer, this is what you'd do: fuse a gene that is normally turned on in the prostate (like TMPRSS2) with an oncogene that is normally turned off (ERG)," says Tomlins. (In 2007, the Safeway Foundation provided unrestricted funding to the Prostate Cancer Foundation for biomarker research. The Foundation also provided PCF-Young Investigator funding for Dr. Scott Tomlins, who at the time was just finishing his PhD training at the University of Michigan. Also, it was Tomlins and colleagues, including Dr. Daniel Rhodes also of the University of Michigan, who developed a novel bioinformatics algorithm called the Cancer Outlier Profile Analysis, or COPA, that led to the discovery of the overexpression of ERG and the TMPRSS2:ERG fusion.)

In February of 2012, the FDA granted the California-based biotech company Gen-Probe approval to offer the PCA3 urine test to men who are considering repeat biopsy after an initially negative result. Its trade name is PROGENSA PCA3.

While that was a welcome milestone for men, research has shown that testing urine for both PCA3 and TMPRSS2:ERG levels is an even better way to stratify men suspected of having prostate cancer. Incorporating both PCA3 and TMPRSS2:ERG will significantly improve upon PSA testing as a means to predict if a man has prostate cancer, says Tomlins. It will be an especially important tool for men with PSA levels below 10, to determine if they may be able to delay invasive biopsy testing, and opt for a program of monitoring their PSA and TMPRSS2:ERG and PCA3 levels over time for signs of progression.

The combination test may also offer men additional information on the size of their tumors which can be an indication of how aggressive that tumor would become if not surgically removed.

Gen-Probe has collaborated with the University of Michigan and the National Cancer Institute—under an initiative of that institution (Early Detection Research Network, or EDRN—to determine the best way to utilize TMPRSS2:ERG in combination with PCA3 and other markers for better patient management. "The ongoing NCI/EDRN study will complement the efforts at University of Michigan by providing independent validation of urine test clinical utility for men with elevated PSA," says Dr. Jack Groskopf, director of Oncology Research and Development at Gen-Probe. In addition to PROGENSA PCA3, the company is also developing a commercial urine test that targets both PCA3 and TMPRSS2:ERG.

Data sets on the TMPRSS2:ERG and PCA3 urine tests

In August of 2011, Dr. Tomlins as first author and Dr. Arul M. Chinnaiyan (Chinnaiyan is also PCF-funded) as senior author published a paper in Science Translational Medicine detailing the ability of urine testing for the TMPRSS2:ERG fusion (both singularly or in combination with PCA3) to stratify prostate cancer risk in men with elevated PSA blood levels.

The researchers found that testing urine levels of both TMPRSS2:ERG and PCA3 was more effective at predicting prostate cancer than testing blood PSA levels alone. Looking at urine samples for 1,312 men with elevated PSA levels who had gone on to have either a biopsy or surgery to remove their prostate gland, the researchers found the highest rates of cancer in men with the highest levels of TMPRSS2:ERG and PCA3 in their urine. The men in the study were stratified into three groups based upon the levels of TMPRSS2:ERG and PCA3 in their urine: low, intermediate and high levels, or scores. Cancer was diagnosed in each of the groups respectively: 21%, 43%, and 69%. High-grade prostate cancer, defined in the study as a Gleason score greater than 6, also occurred at different frequencies in the three groups with 7%, 20%, and 40% diagnosed in each group respectively. (Gleason scores are based upon a microscopic examination of biopsied prostate tissue; the higher the score the more likely a cancer is or will become aggressive.)

In addition, adding the urine test to a standard method now in employ to calculate prostate cancer risk—the Prostate Cancer Risk Calculator, a tool that combines a patient's clinical and family history with PSA levels to estimate a man's risk of developing prostate cancer—significantly improved its predictive ability.

And in a study published in the American Journal of Clinical Pathology, with Tomlins as senior author, the researchers demonstrated that the amount of TMPRSS2:ERG in urine samples from men suspected of having prostate cancer correlated highly with the size of their tumor (known as tumor burden) when removed at prostatectomy. (This applies only to tumors positive for the fusion.) "So if a man has lots of TMPRSS2:ERG in his urine, these men turn out to have a large fusion-positive tumor focus or high tumor burden, and in general, these larger tumors are the ones that are more aggressive," says Tomlins. Their data also suggest that in men who have a high urine TMPRSS2:ERG score, but are found to have low tumor burden on biopsy may have been under-sampled during biopsy and might be well served by repeat biopsy.

Not the whole solution, but strong headway

Tomlins admits their combination urine test is far from the be-all-end-all in determining how aggressive a prostate tumor is or will become. (A study in Cancer Epidemiology, Biomarkers & Prevention reported finding no association with having a tumor positive for TMPRSS2:ERG and recurrence of the cancer after removal of the prostate, although the study only looked for the presence of the genetic

marker, not how much the tumor had produced or whether it could be detected in urine. It also did not report on long-term outcomes in men with TMPRSS2:ERG positive tumors who deferred surgery.) Prostate Cancer Foundation President and CEO, Dr. Jonathan W. Simons, says that being positive for the gene fusion seems to act as a tipping point to prostate cells becoming cancerous but it's not the later instigator of transition to aggressive, deadly disease. This is seemingly borne out by research that shows metastatic prostate cancer sites tend to be either uniformly TMPRSS2:ERG positive or TMPRSS2:ERG negative, thus the mutation doesn't tend to occur after the initial development of the cancer and is unlikely to be necessary to seed metastatic cancer sites. In the urine test, TMPRSS2:ERG functions as a highly-specific marker of cancer cells shed after physical manipulation of the prostate gland via a digital rectal examination. Larger, more invasive tumors may shed more cells, and this could explain the association between higher urine TMPRSS2:ERG scores and high-volume, high-grade disease.

"This combination test is not designed to say definitively whether you have aggressive prostate cancer at diagnosis," says Tomlins. Yet, the test, he says, can provide men with a more accurate estimate of the likelihood that they do in fact have cancer, and the likelihood that they have an aggressive cancer."

Dr. Howard Soule, the chief science officer at PCF says, "The hope with this new urine test is that it will lead to a more informed decision tree for men and their doctors without upticks in detection of incidental prostate cancers."

The department of Pathology at the University of Michigan, under the Michigan Center for Translational Pathology and MLabs, have just began offering the a combined test, termed MiProstate Score (MiPS), that incorporates serum PSA, urine PCA3 score and urine TMPRSS2:ERG score to provide a patient with their individualized risk of having prostate cancer on biopsy. The formulas used to derive MiPS were developed and validated on over an additional 1,900 pre-biopsy urine specimens. The MiPS varies from 0 to 100, with the score equivalent to the risk of having cancer (i.e., a MiPS of 30 indicates a 30% risk of having cancer). A separate score is provided that indicates the risk of having high Gleason score prostate cancer (Gleason score > 6).

For example, a man may receive a report that gives him a 15% risk of having prostate cancer, and a 7% risk of that cancer being high grade. That man might then feel more comfortable forgoing an immediate biopsy, instead opting for a less invasive program of blood and urine monitoring over time for rising levels of PSA, TMPRSS2:ERG and PCA3. Conversely, another man whose report shows an

85% risk of having cancer and a 45% risk of having high grade cancer, may feel more confident that immediate biopsy is the right choice for him. It is entirely feasible that both men might have started out with the same PSA level.

In an editorial that accompanied their study in Science Translational Medicine, the author wrote: "Here, Tomlins et al. improve on the PSA test by taking a new twist on a known gene fusion….demonstrating more accurate, individualized stratification of men at high risk for developing clinically significant prostate cancer." The editorial concludes that the combination TMPRSS2:ERG + PCA3 urine test may help men and their doctors better estimate how urgently a follow-up biopsy is needed after a suspect PSA test.

Dr. Simons of the Prostate Cancer Foundation says, "To our knowledge and from our perspective, this could be the best new use of urine for diagnostics since the pregnancy test."

The combined MiPS test is now available from the University of Michigan MLabs to provide patient-specific prostate cancer risk assessment. For questions on How to Send a Specimen, please call MLabs at 800-862-7284. You may also visit us at www.mlabs.umich.edu

PDF of PCFs comments in response to the U.S. Preventive Services Task Force's (USPSTF) request to the Prostate Cancer Foundation to review the draft Recommendation Statement against prostate-specific antigen (PSA)-based screening for prostate cancer.
Source: http://www.pcf.org/site/c.leJRIROrEpH/b.8827819/k.DADA/Building_a_Better_Mousetrap_to_catch_Prostate_Cancer.htm

Thank you all for your Contributions

Gerard Brice
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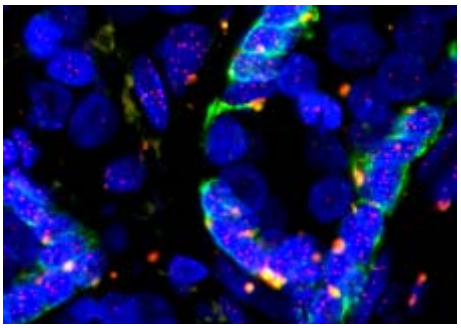
And if any of you would like to participate further by helping with newsletter, administration or becoming an officer, we welcome the addition to our volunteer staff – all of which you see listed.

Thanks for your anticipated assistance. Diane

In Prostate Cancer Prognosis, Telomere Length May Matter

Release Date: 09/26/2013



Fluorescent telomeres (pink) in prostate tissue, including normal, cancer and stromal cells.

-- Alan Meeker, Johns Hopkins

Like the plastic caps at the end of shoelaces, telomeres protect -- in their case -- the interior-gene containing parts of chromosomes that carry a cell's instructional material. Cancer cells are known to have short telomeres, but just how short they are from cancer cell to cancer cell may be a determining factor in a prostate cancer patient's prognosis, according to a study led by Johns Hopkins scientists.

"Doctors are looking for new ways to accurately predict prostate cancer patients' prognoses, because the current methods that use disease stage, Gleason score, and PSA are not perfect," says Alan Meeker, Ph.D., assistant professor of pathology at the Johns Hopkins University School of Medicine and its Kimmel Cancer Center. "Telomere shortening is common in cancer, but the degree of shortening varies from one cancer cell to another within each patient, and this variability may give us a better idea of how prostate cancers behave."

In the study, described in the October issue of Cancer Discovery, the scientists studied tissue samples from 596 men surgically treated for prostate cancer thought to be confined to the prostate and who were participants in a long-term follow-up study on men's health. Then, they used images of prostate cancer cells and nearby cells called stroma, which include smooth muscle and fibroblast cells, taken from surgery-tissue samples of each patient.

Meeker and his team used a technique they developed called telomere-specific fluorescent in situ hybridization (TELI-FISH) to measure telomere length in cancer and stromal cells. The technique uses fluorescent-labeled probes specific for particular locations in DNA, and is commonly used to detect or confirm gene or chromosome abnormalities. In the new study, a fluorescent probe specific for telomere regions was added to the cells, enabling the scientists to identify these specific chromosomal locations under a microscope and measure the level of fluorescence that corresponds to telomere length.

After determining telomere length for more than 40,000 cells among the samples, disease-pattern experts at Johns Hopkins then corre-

lated telomere length measurements in the cancer and stromal cells with each patient's survival.

"Men who had a combination of more variable telomere length among cancer cells and shorter telomere length in stromal cells were more likely to develop metastatic disease and die sooner from their prostate cancer than other men," says Elizabeth Platz, Sc.D., M.P.H., professor of epidemiology at the Johns Hopkins Bloomberg School of Public Health and the Martin D. Abeloff Scholar in Cancer Prevention at the Johns Hopkins Kimmel Cancer Center.
In the group of 98 men with more variable telomere length in cancer cells and shorter telomeres in stromal cells, 20 died of their prostate cancer an average of 8.4 years after diagnosis. Accounting for standard prognostic factors, these men were 14 times more likely to die of their prostate cancer compared with another group of 98 men whose telomeres had less variable length among cancer cells and were longer in stromal cells. In this group, only one man died, and that was after 16.5 years.

"Our studies strongly suggest that the combination of telomere length in stromal cells and its variability among prostate cancer cells could be a marker for prostate cancer prognosis," says Platz.

Meeker and Platz are continuing to study additional groups of patients and are now using an automated fluorescence microscope and computer software to speed the collection of tissue images and extract telomere data.

Funding for the study was provided by the Department of Defense, the National Institutes of Health's National Cancer Institute (CA58236, CA55075, CA72036, CA133891, CA141298) and National Heart, Lung, and Blood Institute (HL35464), the Seraph Foundation, and the Prostate Cancer Foundation.

Tissue samples used for the study were taken from men enrolled in Harvard's Health Professionals Follow-Up Study.
Scientists contributing to the research include Christopher M. Heaphy, Ghil Suk Yoon, Sarah B. Peskoe, Corinne E. Joshu, Thomas K. Lee, Jessica L. Hicks, and Angelo M. De Marzo at Johns Hopkins; and Edward Giovannucci, Stacey A. Kenfield, Lorelei A. Mucci, and Meir J. Stampfer at Harvard School of Public Health.

Media Contacts:
Vanessa Wasta
410-614-2916, wasta@jhmi.edu
Amy Mone
410-614-2915,
amone@jhmi.edu

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New prognostic tool forecasts survival of patients with advanced prostate cancer

Published on October 19, 2013 at 2:42 AM

For men with advanced prostate cancer that has progressed after taking hormones and undergoing chemotherapy, getting an accurate prognosis is critical to determine the next steps for treatment.

But a good prognostic tool has been lacking in this setting, particularly since a new chemotherapy called cabazitaxel has been approved by the U.S. Food and Drug Administration as another line of treatment.

Now researchers at the Duke Cancer Institute have developed a tool for doctors to forecast the potential survival of individual patients, enabling faster, more accurate information on whether to try additional rounds of treatment or seek clinical trials.

The findings are published online in the Journal of the National Cancer Institute.

"Our findings provide a prognostic tool that relies on information that is routinely collected in clinical practice and should be readily available," said Susan Halabi, Ph.D., professor of biostatistics and bioinformatics at Duke and lead author of the study.

"For patients with metastatic prostate cancer who are appropriate candidates for second-line chemotherapy, this model can be helpful for guiding care. It could also be used during clinical trials to assign patients in risk groups based on measurable criteria."

In their study, Halabi and colleagues developed and validated the new prognostic tool using two different clinical trials of prostate cancer patients whose cancer returned after they had undergone a regimen of docetaxel, the standard first-round chemotherapy that is used after hormone treatments have been ineffective.

The researcher's approach provides an understanding of the complex interactions between the host, the tumor factors and clinical outcomes.

By plugging in 17 variables - including pain intensity, measurable disease, race, age, body mass index and others - the researchers determined that certain key factors were relevant to overall survival.

Of the 17 variables, nine were determined to be predictive of survival: how a patient's physical performance is rated on a scale of 0-2; the length of time since the first chemotherapy ended; how extensive the disease is; whether the disease has spread to the liver, lungs or other organs; how much pain the patient is experiencing; the duration of hormone use; and levels of hemoglobin, prostate specific antigen and alkaline phosphatase.

Two of those factors had not previously been used in prognostic models - the duration of hormone therapy and the amount of time since the first-round docetaxel treatment.

"Several new treatments have been developed in recent years that prolong life for men with metastatic prostate cancer," Halabi said. "As a result, it's increasingly important to provide a clear prognostic picture that can help guide both doctors and patients to the best options."

Source: National Institutes of Health as reported by psa-rising.com

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or Jim Kiseda 223-5007

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Fred Bell 845 338-1161
Fwbelljr1@aol.com

Gene Groelle 338-1805
Gro226@aol.com

Website & Newsletters
http://prostatecancer101.org
Walt Sutkowski 331-7241
wsutkowski@hvc.rr.com

Greeters/Church Hall Setup
Bob Miggins 382-1305
GD7M37@verizon.net

Programs
Arlene Ryan 338-9229
Aryan@hvc.rr.com

Diane Sutkowski 331-7241
dsutkowski@hvc.rr.com

Membership & Administration
Diane Sutkowski 331-7241
dsutkowski@hvc.rr.com