Newsletter

Prostate Cancer 101, Inc.

http://prostatecancer101.org

July, 2016

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

Study links normal stem cells to aggressive prostate cancer

February 29, 2016

A study that revealed new findings about prostate cells may point to future strategies for treating aggressive and therapy-resistant forms of prostate cancer.

The study proved that the prostate basal cell layer contains adult stem cells which possess a unique gene expression profile resembling the deadliest form of prostate cancer. The research was led by The University of Texas MD Anderson Cancer Center with findings published in the Feb. 29, 2016 online issue of *Nature Communications*.

"It has become very controversial as to whether the human prostate contains adult stem cells or not and where they are located within the basal or luminal cell compartments," said Dean Tang, M.D., Ph.D., professor of Epigenetics and Molecular Carcinogenesis. "Our study provided definitive evidence that the prostate basal cell layer harbors self-renewing adult stem cells that are enriched in stem-cell genes."

Tang and his team headed by Dingxiao Zhang, Ph.D., an instructor in Tang's lab, show that the findings point to a "theoretical rationale for combining Pol-I and MYC inhibitors to treat highly aggressive forms of prostate cancer which are resistant to endocrine therapy." Pol-I is an enzyme involved with DNA replication and MYC is a regulator gene that plays a role in cell death and transformation.

The prostate gland contains basal and luminal cells, both of which have been identified as "cells-of-origin" for prostate cancer in recent mouse studies. However, the question of whether and where stem cells were present in the human prostate has been largely a medical mystery and a constant debate until now.

Tang's team completed a genome-wide analysis of human benign prostate basal and luminal cells using RNA sequencing and found that they expressed genes differently and that some basal cells represented self-renewing adult stem cells.

"Strikingly, we found that basal stem cells also expressed a large cohort of 'proneural' genes that are normally involved in regulating the nervous system development," said Tang. "These proneural genes seem to play important functions in conferring stem cell-like properties upon some basal cells."

This finding is important because a subset of prostate cancers (less than 5 percent) are highly aggressive and do not respond to current anti-prostate cancer treatments such as endocrine therapy.

"Surprisingly, these hard-to-treat cancers also express a gene signature that overlaps with our normal basal stem cell gene expression profile, suggesting that basal stem cells may represent the cell-of-origin for these prostate cancers," said Tang. "Of significance, the basal stem cell gene expression profile is also linked to endocrine therapy-resistant cancer which is lethal

to virtually all advanced prostate cancer patients."

Tang's team also found that basal stem cells are enriched in a genetic component that is partially regulated by MYC which offers hope that the deadliest of prostate cancers and therapy-resistant prostate cancers may have a new therapeutic option.

"Our studies establish that therapy that combines Pol-I and MYC inhibitors may be a potential new line of treatment for highly metastatic and endocrine therapy-resistant prostate cancer," said Tang.

Explore further: Researchers find cancer aggression differences in different types of prostate cells

More information: Dingxiao Zhang et al. Stem cell and neurogenic gene-expression profiles link prostate basal cells to aggressive prostate cancer, *Nature Communications* (2016). DOI: 10.1038/ncomms10798

Journal reference:

Nature Communications

Provided by: University of Texas M. D. Anderson Cancer Center

Source: http://medicalxpress.com/news/2016-02-links-stem-cells-aggressive-prostate.html?_ga=1.77717075.611357903.1454440878#

FDA Approves Radioactive Imaging Agent Axumin in Recurrent Prostate Cancer

Tony Berberabe, MPH @OncBiz_Wiz
Published Online: Friday, May 27, 2016

Libero Marzella, MD, PhD

The radioactive imaging agent, fluciclovine F18 (Axumin), was approved by the FDA for use in positron emission tomography (PET) scans in men with suspected recurrence of prostate cancer whose prostate-specific antigen (PSA) levels rise following prior treatment.

With the approval of the agent, clinicians hope to better determine the location of the recurrence in the prostate, as this defines optimal choice of therapy. Standard imaging tests used to determine identification of sites of recurrence have low diagnostic accuracy, so more accurate techniques are needed.[1]

“Imaging tests are not able to determine the location of the recurrent prostate cancer when the PSA is at very low levels,” said Libero Marzella, MD, PhD, of the FDA’s Center for Drug Evaluation and Research and director of the Division of Medical Imaging Products, in a statement. “Axumin is shown to provide another accurate imaging approach for these patients.”

It is estimated that up to one-third of patients who receive curative treatment after a diagnosis of primary prostate cancer will experience recurrence within 10 to 15 years following primary treatment.[2]

The approval was granted based on 2 safety and efficacy studies involving Axumin scans in men with suspected recurrent disease. In the first study, 105 Axumin scans were compared with the histopathology samples obtained by prostate biopsy and by biopsies of suspicious imaged lesions in patients with suspected recurrent disease. In the blinded study, radiologists read the scans initially, with subsequent review by 3 independent radiologists.

A second study determined the agreement between Axumin scans and C11 choline, which is an approved PET imaging agent, involving patients with median PSA values of 1.44 ng/mL. To determine consistency, on-site radiologists read the scans initially. Their diagnoses were confirmed by 3 independent, blinded radiologists who read the scans in this second blinded study. The researchers report that the independent scan readings were generally consistent with one another, confirming the results of the on-site scan readings. Both studies supported the safety and efficacy of the agent for imaging prostate cancer in men with elevated PSA levels following prior treatment.

PET scans are a well-established non-invasive imaging technique that uses the radiotracer [18F]flouro 2-deoxyglucose (FDG) for many

tumor types. However, this agent is not widely used in imaging prostate cancer because of poor uptake and high urinary excretion limiting imaging quality.3

The other PET radiotracer that is available, C11 choline, has been shown to improve detection in men with biochemical recurrent prostate cancer, but its 20-minute half-life limits use to medical centers with on-site C11 production capability.

Axumin is a synthetic amino acid that is actively transported into prostate cancer cells by amino acid transporters. It is not metabolized, nor is it incorporated into newly synthesized proteins. Imaging studies have demonstrated that Axumin is preferentially taken up into prostate cancer compared with surrounding normal tissue and visualization is not obscured by bladder uptake.

The agency advises that the agent should be handled with appropriate safety measures to minimize radiation exposure to patients and healthcare providers during administration. Image interpretation errors can occur with Axumin PET imaging. A negative image does not rule out the presence of recurrent prostate cancer and a positive image does not confirm the presence of recurrent prostate cancer. Clinical correlation, which may include histopathological evaluation of the suspected recurrence site, is recommended.

The most commonly reported adverse events in patients are injection site pain, redness, and a metallic taste in the mouth.

1. Choueiri TK, Dreicer R, Paciorek A, Carroll PR, Konety B. A model that predicts the probability of positive imaging in prostate cancer cases with biochemical failure after initial definitive local therapy. *J Urol. 2008;179(3):906–910.*
2. Freedland SJ, Moul JW. Prostate specific antigen recurrence after definitive therapy. *J Urol*. 2007;177(6):1985–1991.
Abdellaoui A1, Iyengar S, Freeman S. Imaging in prostate cancer. *Future Oncol*. 2011;7(5):679 –691.

- See more at: http://www.onclive.com/web-exclusives/fda-approves-radioactive-imaging-agent-axumin-in-recurrent-prostate-cancer#sthash.nubqE5zu.dpuf

CONTRIBUTIONS

Our thanks to those who help us continue our reach out

Tom & Marianne Collins
Frank Guido
John Hayes
Joseph & Helen Sullivan
Your Cause – AT & T - Anon
Your Cause – Price Waterhouse
JC Penney –Change for Better

A special thanks to:

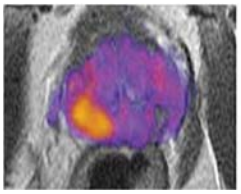
Mid- Hudson Valley FCU for their generous donation.

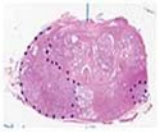
Prostate Cancer 101 is a 501 (c) (3) IRS approved non-profit organization. Your ***tax deductible*** donations should be mailed to:

Prostate Cancer 101
C/o Diane Sutkowski, Treasurer
8 Alcazar Avenue
Kingston NY 12401-4302

Imaging biomarker distinguishes prostate cancer tumor grade

June 1, 2016





The image on the left is a magnetic resonance image (MRI) of a prostate enhanced with restriction spectrum imaging (RSI). Higher grade tumor is indicated by orange and yellow. The image on the right is a digitized section of the prostate …more

Physicians have long used magnetic resonance imaging (MRI) to detect cancer but results of a University of California San Diego School of Medicine study describe the potential use of restriction spectrum imaging (RSI) as an imaging biomarker that enhances the ability of MRI to differentiate aggressive prostate cancer from low-grade or benign tumors and guide treatment and biopsy.

"Noninvasive imaging is used to detect disease, but RSI-MRI takes it a step further," said David S. Karow, MD, PhD, assistant professor of radiology at UC San Diego School of Medicine and the study's senior author. "We can predict the grade of a tumor sometimes without a biopsy of the prostate tissue. This is taking all that's good about multiparametric MRI and making it better."

The addition of RSI to a pelvic MRI added between 2.5 to 5 minutes to scanning time making it a fast and highly accurate tool with decreased risk compared to contrast MRI which involves injecting patients with dye, said Karow.

In the study, published online June 1, 2016 in *Clinical Cancer Research*, the authors said RSI-MRI corrects for magnetic field distortions found in other imaging techniques and focuses upon water diffusion within tumor cells that exhibit a high nuclear volume fraction. By doing this, the ability of imaging to accurately plot a tumor's location is increased and allows for differentiation between tumor grades. The higher the grade, the more aggressive the cancer. Patients can have more than one tumor with different grades, however. Karow said RSI-MRI can be used to guide treatment or biopsy to target the region of highest-grade cancer.

An early diagnosis of prostate cancer typically improves a patient's prognosis. According to the National Cancer Institute, prostate cancer is the second leading cause of cancer death in men in the United States, with more than 26,000 estimated deaths this year and 180,890 new diagnoses predicted. The average age at the time of diagnosis is 66.

At UC San Diego Health, more than 1,000 patients have been imaged with RSI-MRI since 2014 and a subset have subsequently undergone MR-fused ultrasound guided prostate biopsy, said J. Kellogg Parsons, MD, MHS, UC San Diego School of Medicine associate professor of surgery and study co-author.

"Previously, we relied completely on systematic—but random—biopsies of the prostate to diagnose cancer, which has been the standard practice in our field for years. Now, we use RSI-MRI to precisely target specific areas of concern and enhance the accuracy of our diagnosis," said Parsons, surgical oncologist at Moores Cancer Center at UC San Diego Health.

"Greater accuracy means improved care tailored to each individual patient. With RSI-MRI, we are better able to identify which cancers are more aggressive and require immediate treatment, and which ones are slow growing and can be safely observed as part of a program called active surveillance."

Although this study focused on 10 patients, more than 2,700 discrete data points were evaluated. Next steps include introducing the technology to other hospitals and to study whether it can be used in isolation from other screening tools. In prior papers published in the journals *Abdominal Radiology* and *Prostate Cancer Prostatic Diseases*, the same authors reported that RSI-MRI in-

creases detection capability and can perform better than traditional multi-parametric MRI when used in isolation.

These data suggest that RSI-MRI could eventually serve as a stand-alone, non-contrast screening tool that would take 15 minutes compared to a normal contrast-enhanced exam lasting 40 to 60 minutes.

"What our evidence shows so far is the imaging benefit is coming from RSI-MRI," said Karow. "I think this technique could become standard of care and mainstream for the vast majority of men who are at risk for prostate cancer. Full contrast MRI is expensive and risky for most men. This is the kind of exam that could be done on a routine clinical basis."

Anders Dale, PhD, professor of radiology and neurosciences and co-director of the Multimodal Imaging Laboratory at UC San Diego, and Nate White, PhD, assistant professor of radiology, initially co-invented RSI-MRI to characterize aggressive brain tumors.

"RSI-MRI could be a transformational imaging technology for oncologists in the same way CT scans altered the way effects of treatment are quantitated from plain X-rays," said Jonathan W. Simons, MD, Prostate Cancer Foundation president and Chief Executive Officer. "Based on the investigations at UC San Diego, this is a particular promise that needs more validation. Now testable is the hypothesis that RSI-MRI could identify oligometastatic prostate cancer that became curable through its identification by RSI-MRI."

Explore further: MRI-guided biopsy for brain cancer improves diagnosis

More information: G. Yamin et al, Voxel Level Radiologic-Pathologic Validation of Restriction Spectrum Imaging Cellularity Index with Gleason Grade in Prostate Cancer, *Clinical Cancer Research* (2016). DOI: 10.1158/1078-0432.CCR-15-2429

Journal reference: Clinical Cancer Research

Provided by: University of California - San Diego

Source: ***http://medicalxpress.com/news/2016-06-imaging-biomarker-distinguishes-prostate-cancer.html***

Our Business Owners

Please support the efforts of our members.

Discount Beverages
Van Loan's Discount Beverages
327 Broadway, Box 1181, Port Ewen
Since 1974 – all brands beer – all sizes
Micro brews and craft beers
soda, cigars, cigarettes, chips
845 331-8808

Finances
Income Tax Services
Lou Kolano, Enrolled Agent
106 Hurley Ave., Kingston
845 340-0010
lou@loukolano.com
www.loukolano.com

Per$onal Financial Advocate
Financial consulting for individuals,
Families, and small, closely held Businesses.
845 657-8454 Jac Conaway
Jacpath@gmail.com

Restaurant & Catering
Frank Guido's Little Italy – Kingston
845 340-1682
Mariner's Harbor – Kingston
845 340-8051
Frank Guido's Port of Call – Catskill
518 943-5088
Ole Savannah
88E Strand-Kingston
845-331-4283

To be added to this list contact:
dsutkowski@hvc.rr.com

Inherited defects in breast and ovarian cancer genes predispose to prostate cancer with worse outcomes

A new study finds inherited defects in DNA repair genes are prevalent in advanced prostate cancer patients regardless of family history, and are associated with poorer responses to hormonal therapy and shorter survival times. May warrant genetic testing in all hormone therapy-refractory patients.

At the 2016 American Association for Cancer Research (AACR) Annual Meeting, Dr. Joaquin Mateo, a medical oncologist at the Institute for Cancer Research, UK, presented results from a new study which assessed hormone therapy-resistant prostate cancer patients for the prevalence of hereditary alterations in DNA repair genes.

An estimated 50-60% of prostate cancers are thought have an inherited component, making the disease among the most heritable cancer types. The most common inherited genetic alterations associated with increased prostate cancer risk are mutations in the BRCA2 gene, which are also linked with increased risk for breast, ovarian, and pancreatic cancer. However, outside of people with a family history of these cancers or a young age at diagnosis, genetic testing for inherited BRCA2 alterations is not common, as they only occur in <2% of the overall population.

In a recent study by the Prostate Cancer Foundation (PCF)-funded International Prostate Cancer Dream Team, tumors from 20-30% of metastatic hormone therapy-resistant prostate cancer patients were found to harbor mutations in genes which function to repair damaged DNA. Defects in BRCA2 were the most frequent of these alterations. The tumor mutations were either hereditary or had developed in the tumor cells over time. Unfortunately, patients whose tumors carry BRCA2 mutations often experience worse outcomes, with more frequent relapses, poorer responses to therapy, and shorter survival times.

Fortunately, a number of PCF research projects are focused on improving the outcomes for men with mutated DNA repair genes. "We now have drugs in development or are available, which may be particularly relevant for the treatment of these patients" said Mateo, who is also a member of the PCF International Prostate Cancer Dream Team.

In a study led by Mateo that was published last year in the *New England Journal of Medicine*, prostate cancer patients with tumors carrying mutations in DNA repair genes were found to be selectively responsive to treatment with the PARP1-targeting agent olaparib (Lynparza®).

This landmark trial was the first successful demonstration of precision medicine in prostate cancer patients. Olaparib received FDA approval in 2013 for the treatment of breast and ovarian cancer patients whose tumors carry BRCA1/2 mutations. Platinum chemotherapies such as cisplatin, may also be effective against prostate tumors with DNA damage repair alterations, though this awaits validation in clinical trials.

In the current study, Mateo followed patients diagnosed with hormone therapy-resistant prostate cancer to determine the prevalence of hereditary mutations in DNA repair genes and their association with clinical outcomes. Of 150 men with seemingly sporadic (non-hereditary) prostate cancer, **22 (14.7%) were actually found to have hereditary mutations** in one of 10 DNA repair genes (53 genes were examined in total). The most common mutations were in the BRCA2, ATM, and PALB2 genes.

Men who were carriers of hereditary DNA repair mutations, BRCA2 in particular, experienced a significant reduction in median overall survival (76 months for all carriers and 68 months for BRCA2-mutant carriers) when compared with non-carriers (106 months).

Additionally, median time to resistance to primary hormonal therapy was shorter in mutation carriers (12.52 months) compared with non-carriers (14.8 months). BRCA2 mutation carriers fared worst, with all patients experiencing hormone therapy resistance by ~20 months (median of 10.98 months).

Interestingly, the men selected for this study were initially thought to be sporadic cases of prostate cancer, prompting Mateo to question whether a review of their family history would have indicated the presence of a hereditary mutation.

Upon review, Mateo discovered that while a family history of prostate cancer in first or second-degree relatives increased the chances that patients would carry a hereditary mutation, **more than half (59%) of patients with hereditary mutations had no family history of prostate cancer**, and a third had no family history of any of the cancers associated with BRCA mutations (breast, ovarian, prostate, or pancreas).

This translates to 9% of all hormone therapy-resistant prostate cancer patients with no family history *actually being hereditary mutation carriers*. Dr. Mateo also did not find any difference in age at diagnosis between prostate cancer patients with and without hereditary mutations.

"Do not think that only young patients with mothers and sisters who have breast or ovarian cancer will have these mutations. We are seeing these mutations across the spectrum of prostate cancer patients," Mateo warned.

The National Comprehensive Cancer Network (NCCN) has published a set of guidelines to warrant genetic testing for heritable BRCA1/2 mutations for breast and ovarian cancer patients, including young age at diagnosis and family history.

"Many of these patients would not have been tested based on current criteria and would not have received genetic counseling," Mateo said. Although this is worrisome, he further suggests that, "these risk grading systems were not developed for prostate cancer patients." Currently, there are no genetic/familial high-risk assessment guidelines on the NCCN website for prostate cancer patients.

"I think we would recommend genetic testing for all patients who develop castrate resistant prostate cancer," said Mateo. "The results from the test may have implications for the patient's treatment and for his family."

"We are just trying to raise the flag," Mateo continued. "In breast and ovarian cancer, testing for BRCA mutations is a question that every patient asks. But in prostate cancer, it's just not common because the common knowledge was that these mutations were very rare. But when we look in patients with advanced disease, they are actually very common."

Every person has two copies of each gene -- one copy inherited from each parent. While the patients with hereditary mutations had inherited only one mutated gene copy, the majority of these patients acquired a "second hit" in their tumor -- i.e., the second copy of the gene was lost or mutated. Tumors from 18% of hormone therapy-resistant prostate cancer patients without any inherited defects in these genes had also gained these mutations, indicating that these mutations are critical drivers of the development and progression of lethal prostate cancer.

Several ongoing clinical trials are examining the sensitivity of tumors with DNA repair mutations to various therapies including platinum chemotherapy and drugs targeting DNA repair genes. The results from all of these studies will collectively accelerate precision medicine for prostate cancer patients and improve patient outcomes.

This work was funded in part by the Prostate Cancer Foundation (PCF).

The 2016 AACR Annual Meeting was held from April 16-20 in New Orleans, Louisiana.

Source: ***http://www.pcf.org/site/c.leJRIROrEpH/b.9391453/k.BE71/Inherited_defects_in_breast_and_ovarian_cancer_genes_predispose_to_prostate_cancer_with_worse_outcomes.htm?msource=MAY16NP&tr=y&auid=16727323***

New clinical trial endpoint may speed the development of new therapies for patients with localized prostate cancer

Prostate cancer can be slow to progress, frustrating the development of new therapies for patients with early, high-risk disease. A new clinical trial endpoint has been identified that will shorten the time taken to conduct trials, enabling the development of novel therapies that can be administered at the earliest — and potentially curative — stage possible.

At the 2016 American Society of Clinical Oncology (ASCO) Annual Conference, Dr. Christopher Sweeney presented results from ICECaP (Intermediate Clinical Endpoints in Cancer of the Prostate), a Prostate Cancer Foundation (PCF)-supported initiative, that will halve the time required to assess new therapies for aggressive prostate cancer.

"We need to move trials for new therapies earlier, as this is the time when patients with lethal but potentially curable cancer have the best chance of being cured," said Sweeney, principal investigator of the ICECaP project, medical oncologist at the Dana-Farber Cancer Institute, and Associate Professor in Medicine at Harvard Medical School. "We already have many new treatments that extend the lives of patients in the late stage metastatic setting but aren't curative. If we can use them earlier, it is likely we can improve upon proven successes in the adjuvant setting."

"These findings will make a huge impact on prostate cancer drug development," said Dr. Howard Soule, executive vice president and chief science officer of PCF. "Using intermediate clinical endpoints in clinical trials will shorten the time to approval for new treatments and will motivate development of therapies in early disease settings where prostate cancer might be curable. We applaud the ICECaP team, and especially Dr. Sweeney's leadership, for coordinating this huge data collection and analysis program."

When diagnosed early, prostate cancer is highly treatable, but approximately 20% of patients will experience disease recurrence. However, due to the slow nature of prostate cancer progression, it can take up to 10-15 years before patients who relapse succumb to the disease. Of the approximate 29,000 deaths per year in the U.S. resulting from prostate cancer, an estimated 17,000 are patients initially diagnosed with localized disease, but who ultimately experience progression.

Finding ways to prevent recurrence in these patients would pay enormous dividends toward reducing deaths from prostate cancer. Unfortunately, only a fraction of clinical trials for new therapies are conducted in patients presenting with localized disease. The overwhelming majority of trials are in recurrent or metastatic prostate cancer, when patients are essentially incurable.

The dearth of trials in early prostate cancer stems from the difficulty in conducting them. In order for international regulatory agencies such as the FDA to approve a new therapy, an improvement in length or quality of life due to the therapy must first be demonstrated in clinical trials. The "overall survival" (OS) endpoint, which measures the length of time from randomization to death from any cause, is the gold standard for measuring the impact of a treatment on length of life. In localized prostate cancer, reaching an OS endpoint can require 10-15 years — a prohibitive timeframe for pharmaceutical companies. This fact has translated into only limited improvements being made in the treatment of early, aggressive prostate cancer in the last decade.

PCF identified this issue as a

critical unmet need, and in 2012, supported the establishment of the ICECaP Working Group, an international collaborative initiative led by Sweeney, and funded in partnership with Astellas/ Medivation, Janssen, Millenium/Takeda, Sanofi and Sotio. The goal of ICECaP is to undertake the arduous task of identifying an intermediate clinical trial endpoint that functions as a "surrogate" for OS. A successful surrogate would accurately predict overall survival but could be obtained much earlier in the course of the disease. The information on the surrogate endpoint would then be forwarded to regulatory agencies and drug companies around the world in order to hasten clinical trials and regulatory approvals for new therapies for early prostate cancer patients.

To accomplish this goal, Sweeney and team assembled data from 21,140 patients from 24 randomized clinical trials in early stage prostate cancer for which long-term clinical follow-up information was available. A statistical analysis plan was created, in which a candidate surrogate endpoint was required to meet two conditions to be considered suitable: 1) the surrogate must correlate with the true endpoint (i.e., overall survival), and 2) the effects of the treatment on overall survival and on the surrogate endpoint must be correlated (i.e., the treatment must affect both endpoints to similar degrees).

The group found that "disease-free survival" (DFS) rates at 5-years (a measure of the length of time from randomization until local/regional progression, distant metastasis, or death from any cause) significantly correlated with overall survival rates at 8 years (correlation of 0.86). In addition, the treatments were found to similarly affect the OS and DFS endpoints (correlation of 0.73). (A correlation value of 0.70 or greater is considered a reliable surrogate.)

"We are providing the information which regulatory authorities will be able to use as documentation that **disease-free survival is a strong surrogate for overall survival**. We are currently also assessing the more refined endpoint of metastasis-free-survival as a surrogate for overall survival and will present the results of this analysis later in 2016," Sweeney said. "This project will inform drug developers on how to design their studies. They will then use this to petition regulatory authorities to see if it meets their metrics for approval."

To accelerate regulatory agency acceptance of the surrogate endpoint in lieu of overall survival, the ICECaP Working Group has solicited feedback and guidance from regulatory science experts from around the world including the U.S. (FDA), Canada, the U.K., mainland Europe, Ireland, Australia, and New Zealand.

"Although our work is not yet done, we have completed the most difficult task of collecting the data as part of a magnificent and inspiring data-sharing collaboration. I am confident the final product will be able to guide the next generation of clinical trials," said Sweeney. "This will help us to more expediently evaluate new therapies and apply all of the advancements we've been making in drug development to decreasing the death rate from prostate cancer."

The 2016 ASCO Annual Meeting was held from June 3-7, in Chicago, Illinois.

Source: http://www.pcf.org/site/c.leJRIROrEpH/b.9395681/k.8FB0/New_clinical_trial_endpoint_may_speed_the_development_of_new_therapies_for_patients_with_localized_prostate_cancer.htm?msource=JUNE16NP&tr=y&auid=16803702

John Grisham Turns Promoter for Focused Ultrasound
Takes a break from legal thrillers to tout device's benefits
by Parker Brown

Staff Writer, MedPage Today

Best-selling author John Grisham has a side job.

When the legal thriller writer is taking a break from stories of tort reform and multi-million dollar trials, or of small town justice gone awry and blackmailed jurors -- or any of the other legal topics he's covered in the roughly one book a year he's written since 1988 -- he thinks about high-intensity focused ultrasound.

Or rather, he's busy raising money and awareness for the technology as part of the Focused Ultrasound Foundation, where he sits on the board. He "still doesn't understand the technology" behind focused ultrasound, he said in a recent TEDx Talk in Charlottesville, Va., but nevertheless advocates for it because he's found "no other cause, issue, non-profit, or charity that can potentially save so many lives."

High-frequency focused ultrasound, or HIFU, is sometimes used to treat uterine fibroids through non-invasive heating of damaged or diseased tissue. But the foundation -- established by John Grisham's neighbor, Neal Kassell, MD, a professor of neurosurgery at the University of Virginia -- advocates for the technology and its expansion to treat a variety of diseases, including several types of cancer and neurological diseases. Late last year, HIFU was cleared for use by the FDA to treat prostate cancer even though the agency had previously expressed skepticism.

Grisham's latest effort to get more eyes on, and more money for, the foundation is *The Tumor: A Non-Legal Thriller,* a 67-page book that tells the story of a man with glioblastoma. It includes diagrams of the procedure and pictures of an actual glioblastoma, and it's available for free as an Amazon Single, where it was sitting at #16 on the most popular free Kindle Single list at the time of writing.

The foundation did not hire Grisham to write the book, a public relations representative said in an email to *MedPage Today*. "It was John Grisham's idea to write a book about focused ultrasound. He has been on our board for several years so he has seen the Foundation grow and the technology expand as new applications are being researched," the email stated. "He got involved because he sees this as a once-in-a-lifetime opportunity for him to help millions of people."

The book tells the story of Paul, and it's not too much of a spoiler to say that Paul dies. He has glioblastoma, and he undergoes two difficult surgeries and then passes away at age 36, leaving behind a wife and three kids under the age of 8. But that's just half the story.

In a not so subtle line, Grisham emphasizes in boldface: "The total cost of his treatment and care is approximately $300,000." Grisham then channels his inner Philip K. Dick to create an alternative universe, one in which Paul was born 10 years later and gets glioblastoma in 2025, when focused ultrasound therapy is available. This time, there are no difficult surgeries. He undergoes two procedures, his life is extended, and he enjoys life with his kids.

"This is the most important book I've ever written," said Grisham, adding -- as if writing a research paper -- that "Much more research is needed." The story is interspersed with images of real people who have died from glioblastoma, like Ted Kennedy and Lee Atwater, and of illustrations of the procedures.

Using a star writer to bring attention to a certain subset of research is only one of the ways in which researchers have sought funding outside of the usual channels. A crowdfunding campaign on IndieGoGo last year, for example, raised $60,000 for research on a cure for viral infections.

And, if the promotional blurbs on the book's Amazon page are anything to go by, the Focused Ultrasound Foundation's latest effort to raise money could be successful.

"*The Tumor* gives readers a better picture for what the future of medicine can look like with focused ultrasound," wrote U.S. Sen. Mark Warner (D-Va.).

"By increasing awareness, *The Tumor* can help make focused ultrasound available sooner to countless patients worldwide with a host of medical conditions," wrote the CEO of Tupperware Brands, Rick Goings.

And even Ed Miller, MD, the former CEO of Johns Hopkins Medicine, weighed in. "Grisham's book is changing the game," he wrote. "He paints a great picture of how sound waves may shape the future of medicine."

Source:
http://www.medpagetoday.com/Radiology/TherapeuticRadiology/55917

More Men With Early Prostate Cancer Are Choosing to Avoid Treatment

By GINA KOLATAMAY 24, 2016

Bruce Perry of Charleston, W.Va., said his doctors never discussed active surveillance when he received his diagnosis. Credit Raymond Thompson Jr., for The New York Times
Seemingly overnight, treatment of men with early-stage prostate cancer has undergone a sea change. Five years ago, nearly all opted for surgery or radiation; now, nearly half are choosing no treatment at all.

The approach is called active surveillance. It means their cancers are left alone but regularly monitored to be sure they are not growing. Just 10 percent to 15 percent of early-stage prostate cancer patients were being treated by active surveillance several years ago. Now, national data from three independent sources consistently finds that 40 percent to 50 percent of them are making that choice.

In recent years, major research organizations have begun to recommend active surveillance, which for years had been promoted mostly by academic urologists in major medical centers, but not by urologists in private practice, who treat most men. In 2011, the National Institutes of Health held a consensus conference that concluded that it should be the preferred course for men with small and innocuous-looking tumors. Last year, the American Society of Clinical Oncology issued guidelines with the same advice.

The data includes a large new national registry established by the American Urological Association involving 15,000 men nearly all treated by urologists in private practice through 2015; a national registry of 45 mostly private urology practices; and a Michigan registry of mostly private urology practices. In addition, preliminary 2016 data from the urology association indicates that the numbers are growing, with even more than 50 percent of patients choosing active surveillance.

“Things are changing very, very quickly,” said Dr. Matthew R. Cooperberg, a urologist and epidemiologist at the University of California, San Francisco, who has been helping collect data for the new American Urological Association database.

Half of all men with newly diagnosed prostate cancer have low-risk tumors, which pathologists, using a scoring system that looks at the appearance of cells under a microscope, rate as Gleason 6 or less on a commonly used scale. Their risk of dying from prostate cancer in the next 10 years is less than 1 percent, whether they have aggressive treatment or whether they choose active surveillance, research shows.

Nothing, though, is straightforward in the cancer world. Is everyone with a cancer scored as Gleason 6 or lower a candidate for active surveillance? It is not clear.

It is an easier decision for older men with a life expectancy of 10 to 15 years because most prostate cancers that grow do so very slowly. But what about men in their 50s or early 60s?
These men will need to have regular biopsies for long periods of time so doctors can look for signs their cancer might be growing. Biopsies, though, can result in infections that can be serious and can miss more aggressive cancers because they examine only tiny snippets of the prostate.

Mike Steskal, who lives just outside Philadelphia, chose active surveillance after seeing different doctors, most of whom recommended aggressive treatment. Credit Charles Mostoller for The New York Times

That leads to a dilemma for younger men, said Dr. Jonathan I. Epstein, a pathologist at Johns Hopkins Hospital. They have a longer life expectancy during which their tumor might grow and become more aggressive. Yet, he said, “it is the young men who really want to avoid radical treatment for their cancer as the complications, such as impotence and incontinence, affect them to a much greater degree.”

Dr. William Catalona, a professor of urology at the Northwestern University Feinberg School of Medicine, said he worried that some younger men may find out too late that their cancer has become incurable. Active surveillance, he warned, “is a tragic mistake for some.”
Yet some doctors are beginning to question whether these tumors should even be called cancers. Dr. Cooperberg argues that the lowest-risk tumors need a name that conveys their languorous growth and generally benign nature.

“There has to be some terminology that doesn’t say it’s normal but doesn’t use the C-word,” he said.

Others, like Dr. Justin E. Bekelman, an associate professor of radiation oncology at the Perelman School of Medicine at the University of Pennsylvania, are not ready to drop the word “cancer.”
“They have an excellent prognosis, but it is still cancer and we have to follow it carefully,” he said.

Without the word “cancer,” Dr. Epstein said, men may not take seriously the need for regular biopsies and other tests. He and his colleagues at Johns Hopkins proposed a grading system to make it clear that Gleason 6 cells are less frightening than higher-grade tumors, but not necessarily benign.

In the Gleason system, which involves a pathologist’s assessment of how ominous the prostate cells look, 6 is actually pretty much the lowest score for cells that are cancer, despite the Gleason scale officially starting at 2. The highest is a 10. But many men, hearing that their cancer is a 6, assume the worst.

In the new system, which has been endorsed by the World Health Organization, instead of calling the cells Gleason 6, they will be called Group 1 in a scale that goes from 1 to 5.
One issue complicating the active surveillance questions, said Dr. Alan J. Wein, the chief of urology at the Perelman School of Medicine, is that the long-term outcomes are unclear.
Sign Up for the Science Times Newsletter
Every week, we'll bring you stories that capture the wonders of the human body, nature and the cosmos.
“We need follow-up of at least 10 to 15 years to be sure we are not hurting these people,” he said. “The problem is we’ve been in the active surveillance business only since about 2000, and everyone started off very, very slowly. No one really has a number of patients who have gone for years and years.”

Dr. Wein advocates active surveillance for most men with low-risk cancers. But he emphasizes that they should have a second biopsy within a year, followed by regular biopsies every year or two and regular PSA tests, a blood tests that looks for a protein linked to prostate cancers.
“I tell patients, this is a bet,” he said. “You are betting that the disease is not going to progress, or if it progresses, you will be able to tell before it progresses to a situation where it is less treatable. You enhance the chances of winning the bet by doing a confirmatory biopsy.”

Mike Steskal, a commodities trader who lives and works just outside Philadelphia, decided to take the bet. He is 55 and was told last summer that he had prostate cancer after his doctor ordered a blood test for flulike symptoms that happened to include a PSA test. His PSA level was slightly elevated, which led to more tests, including a biopsy that showed a Gleason 6 cancer.
Mr. Steskal spent months seeing different doctors, most of whom recommended aggressive treatment because he is so young. He also researched prostate cancer on the Internet and talked to men who had gone through various treatments. Finally, he chose active surveillance.

As part of the surveillance, he had two more PSA tests. They came back with levels so low that no one would ever have suspected prostate cancer. His higher level last summer was probably due to an infection, which can cause PSA levels to rise.
“It was pure chance” that he got a prostate cancer diagnosis, Mr. Steskal said. “That’s another thing that went into my decision.”

Bruce Perry of Charleston, W.Va., who was a similar age when he received a diagnosis of prostate cancer, said he wished he had had a chance to make that bet.

He was 57 when he learned he had a Gleason 6 cancer in 2010. “I’m like, ‘Oh my God. That is a bad cancer,’” he said. “The problem with men, when they find out they have something like this, it’s like, ‘Get it out now, I don’t want it in me.’ With prostate cancer, I now know that’s not the right attitude.”

The operation was not so bad, but its consequences were difficult, he said: “It took almost a year for me to feel confident about going out and doing anything without wearing protection.”
His doctors never discussed active surveillance.
“The sad part is that that wasn’t really offered as an alternative,” Mr. Perry said.

Source: http://www.nytimes.com/2016/05/25/health/prostate-cancer-active-surveillance-surgery-radiation.html?_r=2

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

1st Tuesday

3rd TBA

4:30 p.m. monthly

SEMINAR For Newly Diagnosed

Distinguished Lecturer Series

Hurley Reformed Church Hall, Hurley, NY

Poughkeepsie Men to Men Group
Our brothers in support and education

Meetings are held the First Thursday of the month at the Central Hudson Auditorium on South Road in Poughkeepsie, starting at 6:30 p.m. Various doctors and speakers are on the agenda and one on one help is available after the meeting.

Contact
Jim Kiseda 223-5007

If you need or want to help:

PCa 101 Seminar
First Tuesday of every month

Fred Bell 845 338-1161
Fwbelljr@aol.com

Gene Groelle 419-5128
Gro226@aol.com

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

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