Informed Prostate Cancer Support Group Inc.
"A 501 C 3 CORPORATION ID # 54-2141691"

Thursday, September 29,

OCTOBER 2022 SPECIAL NEWSLETTER

P.O. Box 420142 San Diego, CA 92142

Volume 15 Issue 10a

IPCSG HAS SOME EXCITING NEWS!

We will now be meeting in-person for IPCSG meetings!

Sanford Burnham Prebys Medical Discovery Institute has informed us we can use their auditorium for all upcoming meetings (this is the same auditorium we previously held our meetings at prior to Covid-19).

In celebration of this great news, IPCSG will be providing lunch and beverages (in the foyer) immediately after the October 15th meeting.

The October meeting starts at 10:00AM PT. IPCSG's president Dr. William Lewis Ph.D. will be presenting:

"Surviving [Aggressive] Prostate Cancer,".

"Lessons learned from 7 years of living with Gleason 9 cancer that metastasized to a hundred places in the bone. Options for anyone living with prostate cancer, from active surveillance to focal treatments, to radiation or surgery, and on to new treatments. Suggestions for thriving while minimizing cancer growth. Nuggets from summarizing IPCSG monthly meetings for six years. Preview of a book in progress about prostate cancer."

Please be advised that no livestream will be available for this meeting as this is only an in-person meeting.

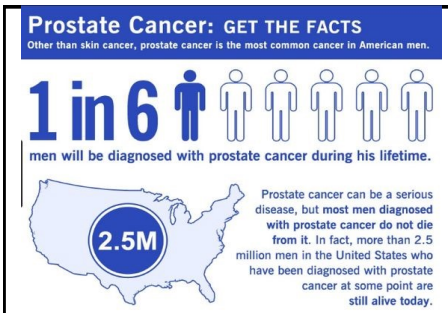
Meeting Location: (see page 10 map)

Sanford Burnham Prebys Medical Discovery Institute (Building 12)
10905 Rd to the Cure, San Diego, CA 92121

For more information, please visit our website https://ipcsg.org/meetings
As always, spouses/partners are welcome and encouraged to attend!
For more information, you can call **Bill** at (619) 591-8670 or **Gene** at (619) 890-8447

Looking forward to seeing you all!

IPCSG



Organization

a 501c3 non-profit organization - all positions are performed gratis

IPCSG

Officers

Bill Lewis President

Additional Directors

Gene Van Vleet

Aaron Lamb

Bill Manning

Honorary Directors

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Aaron Lamb, ...Meeting Set-up
Stephen Pendergast ..... Editor

NEWSLETTER
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PROSTATE CANCER—*2 WORDS, NOT A SENTENCE*

What We Are About

Our Group offers the complete spectrum of information on prevention and treatment. We provide a forum where you can get all your questions answered in one place by men that have lived through the experience. Prostate cancer is very personal. Our goal is to make you more aware of your options before you begin a treatment that has serious side effects that were not properly explained. Impotence, incontinence, and a high rate of recurrence are very common side effects and may be for life. Men who are newly diagnosed with PCa are often overwhelmed by the frightening magnitude of their condition. Networking with our members will help identify what options are best suited for your life style.

Meeting Video DVD's

DVD's of our meetings are available for purchase on our website at https://ipcsg.org/purchase-dvds and are generally available by the next meeting date.

Join the IPCSG TEAM

If you consider the IPCSG to be valuable in your cancer journey, realize that we need people to step up and HELP. Call **President** Bill Lewis @ (619) 591-8670 ; or **Director** Gene Van Vleet @ 619-890-8447.

From the Editor

With this special issue, we celebrate the return to in person meetings. This will give you the chance to interact informally with fellow members and with the speaker. Our normal issue with the summary of the last meeting will follow later this month. In this issue we provide several articles of interest.

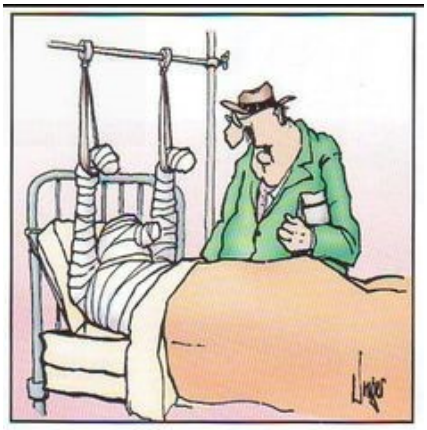
In this issue:

1. How prostate cancer may begin -- ScienceDaily—Scientists found that 'normal' prostate cells in men who had prostate cancer had more mutations (changes in the DNA) than 'normal' prostate cells from men without prostate cancer.
2. Researchers identify drug resistance factors for advanced prostate cancer -- ScienceDaily—researchers identified critical genomic changes in response to abiraterone acetate/prednisone,
3. Coffee Might Give Some Men an Edge Battling Prostate Cancer | Cooking with Kathy Man—a new study finds an association between a genotype that metabolizes caffeine quickly and longer survival from prostate cancer.
4. How are Medicare benefits changing for 2023?(extract)
5. UCSF Pilot Award to Help Develop Improved Method for 225Ac Radioimmunotherapy of Prostate Cancer (extract)

On the Lighter Side

"You can't be expected to get it right the first time!"

"Can't you hum something else?"



"You're looking a lot better today, Ralph."

"I always wear my lucky hat for job interviews."

"What is it with your profession! You're the 39th doctor this year to tell me I'm a hypochondriac."

"You phoned me and said you had amnesia; don't you remember?"

Articles of Interest

How prostate cancer may begin -- ScienceDaily

https://www.sciencedaily.com/releases/2022/09/220921210011.htm

Researchers at the University of East Anglia have made an important discovery about how prostate cancer may start to develop.

A new study published today reveals that the prostate as a whole, including cells that appear normal, is different in men with prostate cancer.

It suggests that tissue cells throughout the whole prostate are primed and ready to develop prostate cancer.

This means that it may be better to treat the whole prostate rather than only the areas in the prostate that have cancer.

The team hope their work could help scientists better understand the causes of prostate cancer, and even prevent it altogether.

Lead researcher Prof Daniel Brewer, from UEA's Norwich Medical School, said: "Prostate cancer is the most common cancer in men and kills one man every 45 minutes in the UK.

"Often, when men are diagnosed with prostate cancer, groups of cancer cells can be found in more than one location within the prostate.

"We wanted to know if this is because of changes in 'normal' prostate cells throughout the prostate."

Cancer is driven by changes in DNA, the genetic code of life, that appear in every cell. The team studied the DNA code in 121 tissue samples from 37 men with and without prostate cancer.

Prof Brewer said: "The samples we studied included tissue that comes from the cancer and tissue from elsewhere in the prostate, which looks normal down the microscope.

"This produces a massive amount of data and by applying a large amount of computer power we can determine the differences that have occurred in the DNA, giving us insight into how the cancer grows.

"We found that 'normal' prostate cells in men who had prostate cancer had more mutations (changes in the DNA) than 'normal' prostate cells from men without prostate cancer.

"Based on the genetics of the samples analysed, we created maps to understand where the different mutations occurred. And we showed that in most men, the mutations in normal cells are different to mutations in cancer cells.

"The 'normal' prostate cells in men who have prostate cancer appear to provide a beneficial environment for prostate cancer cells to develop and grow.

"In other words, the whole prostate is primed and ready to develop prostate cancer driven by an, as yet unknown, biological process.

"This work has improved our knowledge of how prostate cancer first starts to develop and might one day give us clues as to how to prevent or treat it.

"And it shows that it may be better to treat the whole prostate rather than only the areas in the prostate that have cancer," he added.

Dr Hayley Luxton, Senior Research Impact Manager at Prostate Cancer UK, said: "This exciting new research shows for the first time how normal cells in the prostate can facilitate the growth and spread of prostate cancer.

"The researchers found that normal prostate cells in men with prostate cancer have specific genetic changes that make them act like a rich compost, providing the perfect environment for prostate cancer cells to grow and develop. These findings give us important new insights into the early development of

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prostate cancer, which might one day give us clues as to how to prevent it."

This research was led by UEA, in collaboration with the University of Cambridge, The Institute of Cancer Research, London, the Wellcome Sanger Institute, the Universities of Oxford, St Andrews, York, Manchester, Tampere (Finland), and University College London -- as well as Cambridge University Hospitals NHS Foundation Trust, Royal Marsden NHS Foundation Trust, HCA Healthcare UK Laboratories and the Earlham Institute.

It was funded by Cancer Research UK, the Dallaglio Foundation, and a Prostate Cancer UK Movember Training, Leadership & Development Award.

The project has also received support from Prostate Cancer Research, Big C Cancer Charity, Bob Champion Cancer Trust, The Masonic Charitable Foundation successor to The Grand Charity, The Alan Boswell Group, The King Family and The Hargrave Foundation.

Researchers identify drug resistance factors for advanced prostate cancer -- ScienceDaily

sciencedaily.com

In a new study published in *Molecular Cancer Researc*h, Mayo Clinic researchers identified critical genomic changes in response to abiraterone acetate/prednisone, a standard treatment option for men with progressive, incurable and castration-resistant prostate cancer.

"We defined a potential strategy for both responders and nonresponders of the drug that may help men overcome resistance and prolong survival," says Liewei Wang, M.D., Ph.D., the Bernard and Edith Waterman Director, Pharmacogenomics Program, Mayo Clinic's Center for Individualized Medicine. Dr. Wang is the corresponding author of the study.

Dr. Wang explains that while several drug choices are available to control disease progression, many questions remain over which drugs to use in individual cases. Also, predictive biomarkers for drug resistance and sensitivity remain primarily unknown.

Abiraterone acetate is a standard treatment option for men with castration-resistant prostate cancer. However, the response rate is limited, no known biomarkers predict prognosis, and alternative therapies for those who failed treatment are unavailable.

In the Prostate Cancer Medically Optimized Genome Enhanced Therapy study, also known as PROMOTE, Mayo researchers revealed DNA sequences associated with response to abiraterone acetate to identify additional treatment options for men with advanced prostate cancer resistant to all standard therapies. They identified an 11-gene drug panel that provided a new tool to individualize treatment for abiraterone acetate. A genetic testing panel is a laboratory test that looks at a select group of genes. The 11-gene panel predicted a worse prognosis for a subset of primary or metastatic patients enrolled in the study.

In the next step of their analysis in this prospective study, the researchers analyzed whole-exome sequencing and RNA sequence data from 83 patients with metastatic biopsies before and after 12 weeks of abiraterone acetate/prednisone treatment. They identified genomic alterations associated with acquired resistance after 12 weeks of this treatment.

"We analyzed the posttreatment genomic landscape of metastatic biopsies in these patients with metastatic castration-resistant prostate cancer to identify mechanisms of acquired resistance," says Hugues Sicotte, Ph.D., a Mayo Clinic bioinformatician and lead author of the study. "These results may assist with selecting alternative therapies in a subset of abiraterone acetate-resistant patients with the highest risk of having the poorest outcome."

Dr. Sicotte says biomarkers based on the stage-specific landscape of genomic changes in prostate cancer are under investigation.

"Further studies will be needed to test these drug treatments to overcome abiraterone acetate/

prednisone resistance and define subgroups of nonresponders," says Dr. Sicotte. "Our goal is to incorporate these into clinical practice for physicians and patients with castration-resistant prostate cancer."

Prostate cancer is the most commonly diagnosed solid organ malignancy in the U.S., with more than 268,490 new diagnoses annually and an estimated 34,500 deaths. It is the second leading cause of cancer deaths among men, according to the National Cancer Institute's Surveillance Epidemiology and End Results Program.

The PROMOTE study is a collaboration of Mayo Clinic's Center for Individualized Medicine and the Mayo Clinic Comprehensive Cancer Center.

Story Source:

Materials provided by **Mayo Clinic**. Original written by Colette Gallagher. *Note: Content may be edited for style and length.*

Coffee Might Give Some Men an Edge Battling Prostate Cancer | Cooking with Kathy Man

cookwithkathy.wordpress.com

Cara Murez wrote . . . . . .

For some men battling prostate cancer, drinking coffee may offer not just a quick pick-me-up but longer survival.

Research is still in the early phases, but a new study finds an association between a genotype that metabolizes caffeine quickly and longer survival from prostate cancer. That genotype is called CYP1A2 AA.

"I'm very excited about this work because each time we're digging in deeper. I think it has some really interesting findings that say, 'Hey, there may be something here.' We need to look more into what could be going on in terms of coffee and impact on people's lives, and especially those who are diagnosed with cancer," said lead study author Dr. Justin Gregg. He is a urologic oncologist at the University of Texas MD Anderson Cancer Center in Houston.

Gregg said one of the most frequent questions he hears in his work is how can someone slow down a cancer or even prevent one from developing.

While there is a lot of interest in how diet and activity affect cancer risk, there aren't many specific recommendations, especially for patients already diagnosed with cancer, Gregg said.

Past research on coffee and its potential health benefits, with antioxidants that may affect inflammation, made it an interesting subject. Gregg said he was further intrigued by another study that looked at differing genotypes and the speed at which they metabolize caffeine.

This new study included data for prostate cancer cases across studies that were in the PRACTICAL Consortium, which stands for Prostate Cancer Association Group to Investigate Cancer Associated Alterations in the Genome. It included over 5,700 cases from seven studies.

Patients included those on active surveillance, where their cancer isn't treated while it's watched for change; those who were treated for their prostate cancer; and some patients who had cancer that had metastasized.

Limitations included that patients were asked to recall their own food and drink consumption and data was from seven different sites, which asked patients to recall consumption dating back varying lengths of time.

The researchers compared levels of coffee consumption, such as those who were considered high intake at two or more cups per day and those who were low intake at three or more cups per week.

High coffee intake was linked to longer prostate cancer-specific survival in men who had the CYP1A2 AA genotype, the investigators found.

"There's a chance in the future, with additional research, that looking at things like what to do in

(Continued from page 6)

your diet based on certain patient groups could be something that is used to augment the care of men who are diagnosed with prostate cancer," Gregg said. "It may become part of a number of things that clinicians and others look at when they're treating men with prostate cancer."

The findings were published online recently in the journal *European Urology Oncology*.

Dr. William Dahut, chief scientific officer for the American Cancer Society, said what he took away from the study was that if a person is a coffee drinker and has prostate cancer, there's no reason to stop drinking coffee and there may be some benefit.

Oftentimes people will live for many years, even decades, with a prostate cancer diagnosis, and people's coffee habits may change, so there may be factors that confound the data, Dahut said. The authors are upfront about the potential shortcomings, too, he added, commending them for pulling all of the data together.

"There's at least a theoretical reason why it's helpful, but there's not enough information out there for us to say, 'If you have prostate cancer, you need to start drinking coffee' either," Dahut said.

Other studies have looked at whether different foods such as tomatoes or milk might help prevent or slow cancer, but it can be hard to control for differences in how the items are cooked or mixed with other foods, he noted.

"Food likely actually has an impact on cancer. We certainly know there's correlations with high BMI [body mass index] and multiple cancers, but it's very difficult to study," Dahut said.

About 268,000 men in the United States will be diagnosed with prostate cancer this year, according to the American Cancer Society, and about 34,500 will die.

Cases vary, but many men have their conditions monitored with what's called active surveillance, testing for changes in the cancer but not treating it. Sometimes there is never a need to intervene, Dahut said.

Those who may have greater risks because of genetics or for other reasons typically have surgery to remove the prostate or undergo radiation.

"I do think these large-scale population studies are important because they do give us clues about where science should go," Dahut said.

Source: HealthDay

Filed under: Drink, Health, News and Articles, Study | Tagged: Coffee, Prostate Cancer |

How are Medicare benefits changing for 2023?(extract)

medicareresources.org

How are Medicare benefits changing for 2023?

Louise Norris September 17, 2022 Reviewed by our health policy panel.

Key takeaways

- The standard Part B premium is expected to remain at $170.10 for 2023, or possibly decrease. (This won't be finalized until November 2022.)
- The Part B deductible is $233 in 2022, and is projected to remain at that level in 2023.
- Part A premiums, deductible, and coinsurance are projected to increase in 2023.

(Continued on page 8)

- The income brackets for high-income premium adjustments for Medicare Part B and D start at $91,000 for a single person, but this threshold is expected to increase to $97,000 in 2023.
- Medicare Advantage enrollment is expected to continue to increase in 2023.
- The maximum allowable out-of-pocket cap for Medicare Advantage plans is increasing to $8,300 in 2023 (but most plans have lower out-of-pocket caps).
- Part D donut hole no longer exists, but a standard plan's maximum deductible will increase to $505 in 2023, and the threshold for entering the catastrophic coverage phase (where out-of-pocket spending decreases significantly) will increase to $7,400. But the Inflation Reduction Act will ensure that Part D enrollees no longer have to pay for covered vaccines, and will have access to insulin for no more than $35/month.

But there are also changes to Original Medicare cost-sharing and premiums, the high-income brackets, and more.

The standard premium for Medicare Part B is $170.10/month in 2022. And although we won't know the 2023 premium until November 2022, the Medicare Trustees Report projects that it will remain at $170.10/month. There's also a possibility that it could decrease, due to Medicare's lower-than-expected spending on Aduhelm, the new Alzheimer's drug that drove a significant portion of the Part B rate increase in 2022. (The standard Part B premium increased by nearly $22/month in 2022 — it had been $148.50/month in 2021 — which was the largest dollar increase in the program's history.)

Although the Part B increase for 2022 was substantial, the 5.9% Social Security cost-of-living adjustment (COLA) for 2022 was also historically large, and more than covered the increase in Part B premiums for beneficiaries who receive Social Security retirement benefits. For 2023, the Social Security COLA is expected to be even larger. And unlike 2022, when a chunk of seniors' COLA had to be used to cover the additional Part B premiums, the Part B premium is not expected to increase in 2023. So the COLA will be available for retirees to use to cover other living expenses, which have increased sharply in 2022.

(If a Social Security recipient's COLA isn't enough to cover the full premium increase for Part B, that person's Part B premium can only increase by the amount of the COLA. That's because Part B premiums are withheld from Social Security checks, and net checks can't decline from one year to the next. That was not an issue in 2022, however, due to the size of the COLA, and will not be an issue in 2023 due to the projected large COLA and lack of a Part B rate increase.)

Some enrollees have supplemental coverage that pays their Part B deductible. This includes Medicaid, employer-sponsored plans, and Medigap plans C and F. But since the beginning of 2020, Medigap plans C and F have no longer been available to newly-eligible enrollees (people can keep them if they already have them, and people who were already eligible for Medicare prior to 2020 can continue to purchase them). The ban on the sale of Medigap plans that cover the Part B deductible for new enrollees was part of the Medicare Access and CHIP Reauthorization Act of 2015 (MACRA). It's an effort to curb utilization by ensuring that enrollees incur some out-of-pocket costs when they receive medical care.

Many Medicare Advantage plans have low copays and deductibles that don't necessarily increase in lockstep with the Part B deductible, so their benefits designs have had different fluctuations over the last few years. (Medicare Advantage enrollees pay the Part B premium plus the Advantage plan premium if the plan has a separate premium — many do not, so the enrollees just pay the Part B premium. Medicare Advantage plans wrap Part A, Part B, usually Part D, and various supplemental coverage together into one

plan, with out-of-pocket costs that are different from Original Medicare.)

Louise Norris is an individual health insurance broker who has been writing about health insurance and health reform since 2006. She has written dozens of opinions and educational pieces about the Affordable Care Act for healthinsurance.org. Her state health exchange updates are regularly cited by media who cover health reform and by other health insurance experts.

UCSF Pilot Award to Help Develop Improved Method for 225Ac Radioimmunotherapy of Prostate Cancer(extract)

University of California's, San Francisco (UCSF) advanced targeted alpha therapy (TAT) agent called 225Ac-DOTA-YS5, can treat prostate cancer while allowing verification of its activity in prostate cancer models, according to Kondapa Naidu Boppa, Ph.D., assistant professional researcher in UCSF's Department of Radiology and Biomedical Imaging. In the case of the UCSF advancement, the TAT uses radioimmunotherapy, helping to deliver high doses of highly lethal alpha participants to the targeted tumors. At UCSF, thus far, this particular agent has been used to target an antigen highly expressed in prostate cancer known as CD46. Now, UCSF researchers have received a pilot reward to develop further and improve the imaging and therapeutic agent targeting prostate cancer. Specifically, Kondapa Naidu Bobba, and Bin Liu, Ph.D., a professor in the UCSF Department of Anesthesia, secured a UCSF Resource Allocation Program (RAP) contribution: a "Pilot Award in Precision Imaging of Cancer and Therapy" for the formal study titled "Development of an improved method for 225Ac radioimmunotherapy of prostate cancer."

What follows is a brief breakdown of this UCSF breakthrough.

This UCSF advancement falls into a category of imaging and therapeutic agents helping to better target and destroy tumors while minimizing the associated toxicity. For example, there is an emerging technology in which radiation is selectively targeted to tumors using molecular targeting radioligand therapy. This approach enables effective tumor treatment with minimal toxicity as compared to before.

TAT is an example of a highly targeted approach, delivering a highly lethal payload of alpha particles targeting tumors.

The grant-winning Dr. Bobba's research is conducted in UCSF's Radiology department's Molecular Imaging Lab, also called the Flavell lab. This lab's PI is Robert Flavell, MD, Ph.D., chief of Molecular Imaging & Therapeutics.

Members of this laboratory integrate new chemistry, chemical biology, imaging, and clinical methods with the aim of improving new methodologies to help advance patient care.

Lead Research/Investigator

Kondapa Naidu Boppa, Ph.D., assistant professional researcher in UCSF's Department of Radiology and Biomedical Imaging.

Bin Liu, Ph.D., professor in the UCSF Department of Anesthesia.

NETWORKING

Please help us in our outreach efforts. Our speakers bureau consisting of Gene Van Vleet and Bill Lewis is available to speak to organizations of which you might be a member. Contact Gene 619-890-8447 or gene@ipcsg.org or Bill 619-591-8670 (bill@ipcsg.org) to coordinate.

Member John Tassi is the webmaster of our website and welcomes any suggestions to make our website simple and easy to navigate. Check out the Personal Experiences page and send us your story. Go to: https://ipcsg.org/personal-experience

FINANCES

We want to thank those of you who have made special donations to IPCSG. Remember that your gifts are tax deductible because we are a 501(c)(3) non-profit organization.

We again are reminding our members and friends to consider giving a large financial contribution to the IPCSG. This can include estate giving as well as giving in memory of a loved one. You can also have a distribution from your IRA made to our account. We need your support. We will, in turn, make contributions from our group to Prostate Cancer researchers and other groups as appropriate for a non-profit organization. Our group ID number is 54-2141691. Corporate donors are welcome!

Directions to Sanford-Burnham-Prebys Auditorium

10905 Road to the Cure, San Diego, CA 92121

Take I-5 (north or south) to the Genesee exit (west).

Follow Genesee up the hill, staying right.

Genesee rounds right onto North Torrey Pines Road.

Do not turn into the Sanford-Burnham-Prebys Medical Discovery Institute or Fishman Auditorium

Turn right on Science Park Road. Watch for our sign here.