# PCCN Markham

Newsletter

Volume 19 Issue 9 May, 2018

## NEXT MEETING

## *Tuesday, May 8, 2018 - 7:30PM*

***St. Andrews Presbyterian Church – Main St Markham***

***Upstairs Hall***

***(Free Parking off George St)***

## GUEST SPEAKER

**Dr. Mateya Trinkaus MD, Medical Oncologist**

**Topic: Seeing a Medical Oncologist … What to expect**

**Spouses, Family, Friends - Always Welcome**

**Coffee, Treats & Mingle @ 7:00pm**

## IN THIS ISSUE …

# PCCN Markham

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**Volume 19 Issue 9** **May, 2018**

## MHSPC: Increased Survival with Added Docetaxel Confirmed Long-Term

### 13-month advantage in median overall survival

by Pam Harrison, Contributing Writer, MedPage Today April 16, 2018

**Action Points**

- Docetaxel plus androgen-deprivation therapy (ADT) significantly improved long-term overall survival (OS) in men with metastatic hormone-sensitive prostate cancer (mHSPCA), compared with those who received ADT alone.
- Note that longer follow-up in this randomized clinical trial confirmed the interim analysis that found the effect of docetaxel was more pronounced for patients with high-volume disease.

For men with metastatic hormone-sensitive prostate cancer (mHSPC), significant long-term improvement in overall survival (OS) has been confirmed for those who received docetaxel plus androgen-deprivation therapy (ADT), compared with those who received ADT alone.

As an interim analysis of the Chemohormonal Therapy Versus Androgen Ablation Randomized Trial for Extensive Disease in Prostate Cancer (CHAARTED) had demonstrated, the survival benefit in favor of the chemohormonal arm was reported for the study population as a whole, but the greatest benefit occurred among men with high-volume disease. At a median follow-up of 28.9 months, there was a 13-month difference in median OS among men randomized to have docetaxel plus ADT, at 57.6 months compared with 44.0 months for men treated with ADT alone.

At the time of the interim analysis, 101 patients had died in the chemotherapy-plus-ADT arm compared with 136 patients in the ADT-alone arm.

Now, as shown in the new study in the *Journal of Clinical Oncology* by Christos Kyriakopoulos, MD, of the University of Wisconsin School of Medicine and Public Health in Madison, and colleagues, with 188 deaths in the combination arm and 211 in the ADT-alone arm, median OS was 10.4 months longer in the chemohormonal arm at the same 57.6 months after a median follow-up of 53.7 months, compared with 47.2 months in the hormonal-alone arm alone (hazard ratio [HR], 0.72; 95% CI, 0.59 to 0.89; *P*=.0018).

"This is a practice-changing, confirmatory follow-up study that tells us that front-line treatment with docetaxel plus ADT will be one of the new standards for high-risk metastatic prostate cancer, although this does not seem to apply to low-risk disease," Derek Raghavan, MD, PhD, president of Carolinas Health Care System's Levine Cancer Center in Charlotte, N.C., a genitourinary cancer specialist not affiliated with the study, affirmed via email to *MedPage Today*.

Until the CHAARTED results were released, it was not clear whether giving chemotherapy upfront along with ADT could delay the inevitable transformation of mHSPC into castrate-resistant prostate cancer (CRPC) and improve both quality of life and OS. Kyriakopoulos et al randomized 790 participants with mHSPC to either ADT in combination with docetaxel at a dose of 75 mg/m$^2$ for up to six cycles of ADT alone. The primary endpoint of the study was overall survival.

The results were additionally analyzed by prospectively defined low- and high-volume disease subgroups. In the study, high-volume disease was defined as the presence of visceral metastases either with or without four or more bone metastases, one of which had to be outside the vertebral column and pelvis. A total of 513 participants were classified as having high-volume disease, with roughly equal numbers of patients receiving the combination of docetaxel plus ADT and ADT alone.

"Longer follow-up confirmed that the effect of docetaxel was more pronounced for patients with high-volume disease," the team reported.

At the same median follow-up of 53.7 months, median OS in the high-volume disease subgroup was 16.8 months longer in the combination arm. at a median of 51.2 months compared with 34.3 months for the ADT-alone arm -- HR 0.63 (95% CI, 0.50 to 0.79; *P*<.001). In comparison, median OS among men with low-volume disease treated with the same combination was 63.5 months, at a median follow-up of 53.8 months compared with a median survival that had not yet been reached for the ADT-alone arm at an HR of 1.04 (95% CI, 0.70 to 1.55; *P*=.86), the researchers added.

Time to the development of CRPC and time to clinical progression were also assessed. For the overall cohort, the time to CRPC was 39% longer, at 19.4 months in the docetaxel-plus-ADT arm, versus 11.7 months in the ADT-alone arm (HR, 0.61; 95% CI, 0.52 to 0.73; *P*<.001). Median time to clinical progression, again for the overall cohort, was also 38% longer, at 33.0 months in the combination arm versus 19.8 months in the ADT-alone arm, at an HR of 0.62 (95% CI, 0.51 to 0.75; *P*<.001).

Other major findings:

- For patients with high-volume disease, the median time to CRPC was 42% longer, at 14.9 months for the chemohormonal arm compared with 8.6 months for the ADT-alone arm, at an HR of 0.59 (95% CI, 0.47 to 0.71; *P*<.001)
- For men with low-volume disease, the median time to CRPC was 30% longer, at 31.0 months among men in the combination arm compared with 22.7 months for those treated with ADT alone, at an HR of 0.70 (95% CI, 0.50 to 0.96; *P*=.03)
- For men with high-volume disease, the median time to clinical progression at 27.3 months was 47% longer for those treated with the combination compared with a median of 13.0 months, at an HR of 0.53 (95% CI, 0.42 to 0.67; *P*<.001) for those in the ADT-alone arm
- For men with low-volume disease, the median time to clinical progression was virtually identical in both treatment groups, at 42.5 months in the combination arm versus 44.3 months in the ADT-alone arm, at an HR of 0.86 (95% CI, 0.60 to 1.25; *P*=.43)

  "With longer follow-up, the clinical benefit observed with chemohormonal therapy was confirmed for patients with high-volume disease regardless of whether they had relapsed after [prior local therapy] of the prostate with or without curative intent," the researchers concluded. "In contrast, the subgroup with low-volume disease showed no evidence of survival benefit when docetaxel was added (HR, 1.04 with 100 deaths), despite the [earlier] analysis showing a nonsignificant HR of 0.60 with 44 deaths."

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The team cautioned that the results need to be viewed in light of recent guidelines issued by the American Society of Clinical Oncology indicating that both docetaxel and abiraterone (Zytiga) represent two separate standards of care for mHSPC, since the addition of either drug to ADT significantly improved OS in men with newly diagnosed metastatic noncastration-resistant prostate cancer. Raghavan also emphasized that it is not yet clear whether abiraterone or enzalutamide (XTANDI) plus ADT have equivalent efficacy in this context, although the combination approach seems to be superior to ADT alone, as he indicated.

"Added considerations in this comparison will include toxicity, ease of delivery, patient compliance, and cost," he said. In addition, "fiscal toxicity has become a much bigger factor [in cancer therapy, even though] it is pleasing to see meaningful increases in median- and long-term survival from these new treatment algorithms."

https://www.medpagetoday.com/hematologyoncology/prostatecancer/72373

## Prostate cancer diagnosis breakthrough hailed

April 23, 2018

A new ultrasound process offering more successful diagnosis and management of prostate cancer has been identified by Dundee University researchers.

The university said non-invasive shear wave elastography (SWE) offers "much greater accuracy and reliability" than current testing and is less expensive.

Prostate cancer is the most common cancer in men in the UK.

Former Dundee University rector Stephen Fry, who underwent surgery for prostate cancer, called the research "exciting".

Over 47,000 new cases of prostate cancer are diagnosed in the UK every year.

The most common tests for the disease include the PSA blood test, a digital rectal examination (DRE), MRI scans and a biopsy.

The university said each of these carried "significant problems".

**"Unnecessary treatments"**

The new method targets the prostate with ultrasound. The Dundee University study involved about 200 patients.

Cancerous tissue is stiffer than normal tissue, so the shear waves are slowed as they pass through it.

The technology was able to detect 89 per cent of prostate cancers and could identify more aggressive cancers and those beginning to spread outside the prostate.

Ghulam Nabi, professor of surgical uro-oncology at the university, said, "Prostate cancer is one of the most difficult to pinpoint.

"We are still in a position where our diagnosis of prostate cancer is extremely inefficient, leading to unnecessary treatments for many patients."

Prof Nabi said the new treatment was "like someone has turned the lights on in a darkened room."

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He said: "We have had cases where the SWE technique has picked up cancers which MRI did not reveal.
"We can now see with much greater accuracy what tissue is cancerous, where it is and what level of treatment it needs."
Image caption Stephen Fry said news of the new technique was "doubly, triply exciting"
Stephen Fry underwent surgery for prostate cancer in January.
He said it was "doubly, triply exciting" to hear of the new techniques.
He said: "Anyone who has been in my position will know that when it comes to this pernicious disease early screening and diagnosis is the absolute key to a successful outcome.
"The news of this breakthrough comes at a time when prostate cancer is being pushed to the forefront of our consciousness in the UK, not least because of the disturbing upward trend in its prevalence.
"So hurrah for Dundee University and Medical School and a huge thank you to Professor Nabi and his team for their work in developing this new weapon in the war against a deadly killer."
The project was funded by Prostate Cancer UK with support from the Movember Foundation.
http://www.bbc.com/news/uk-scotland-tayside-central-43864875

## Research shows diabetes drug's promise in zapping prostate cancer

**By Delthia Ricks** delthia.ricks@newsday.com Updated April 22, 2018 8:22 PM

The compound, marketed as metformin, apparently undercuts what certain aggressive cancer cells rely on to produce energy and sustain themselves.

Dr. Lloyd Trotman in his lab on April 9 at the Cold Spring Harbor Laboratory. Photo Credit: Joseph D. Sullivan

Lloyd Trotman journeys daily into the inner sanctum of cancer cells.
He analyzes their molecular machinery and potent energy production, but he does not stop there: The Cold Spring Harbor Laboratory cancer biologist's explorations are revealing how a low-cost diabetes drug ultimately may quell the most lethal malignancies of the prostate.
In studying tumors that invade the walnut-sized gland, Trotman discovered a key genetic flaw in the most virulent forms of prostate cancer — those with a missing or mutated protein called PTEN.
When present, the protein slams the brakes on cancer growth.

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"There is a loss of PTEN in many men who have aggressive variants of prostate cancer," Trotman said. "So that loss is synonymous with a life-threatening form of the disease."

The situation, he said, is found in about half of the men who die of prostate cancer. Many patients with aggressive forms of the disease also lack another brake, a key tumor suppressor called p53.

Trotman then found a way to make those cancers destroy themselves when exposed to a compound that first was isolated from French lilacs during the Middle Ages, now marketed as the top-selling diabetes drug, metformin.

The medication apparently undermines the mechanisms that aggressive prostate cancer cells rely on to produce energy and sustain themselves. It costs only pennies per dose.

If repurposed as a cancer treatment, it would be among the least expensive in an escalating pricing drama in which some cancer medications cost more than $450,000 for a one-time treatment.

Prostate cancer is a growing global health concern and a leading cause of cancer deaths among men. The American Cancer Society estimates that one in every 41 men in the United States dies of the disease, defining it as the second-leading cause of cancer deaths among men nationwide, after lung cancer.

Some prostate tumors, especially in older men, are indolent, growing so slowly that most men are more likely to die with the disease than of it.

It rarely is diagnosed before the age of 40, though some groups are at elevated risk. African-American men, for example, are at higher risk than white men. Others at high risk include anyone with a family history of the cancer.

Anecdotal evidence had long suggested that metformin might play a role in cancer therapy, because diabetics who were on the drug had lower rates of certain cancers.

But no one knew the precise mechanism of how metformin works in cancer cells until Trotman and his colleagues revealed their research on PTEN, published earlier this month in the journal "Cell Reports."

It was a breakthrough for the prizewinning professor who received a 2014 Pershing Square Sohn Prize for Young Investigators in Cancer Research, given annually to six investigators in the greater metropolitan area exploring groundbreaking new paths.

The discovery that the absence of PTEN made prostate cancers more aggressive was the first piece in a large and complex puzzle, Trotman said.

When present and healthy, PTEN functions as a master tumor suppressor, keeping cancer at bay. But without it, combating prostate cancer becomes decidedly difficult, and sometimes futile.

"We were asking this question: How could we most effectively kill cells that have the mutation while sparing cells that do not? So we screened a lot of drugs and tested them in an unbiased way," Trotman said. "What we found is that there is one type of drug that selectively kills those cells that have lost PTEN" — and that the drug leaves healthy cells unharmed.

Metformin, with its long and storied past, hails from a drug class known as mitochondrial inhibitors.

Trotman and his colleagues discovered the precise molecular mechanisms by which the drug forces aggressive prostate cancer cells to burn all of their energy and destroy themselves.

Mitochondria are tiny, bean-shaped powerhouses in most human cells (they are not in red blood cells). They provide the chemical energy necessary for every human activity — walking, running, lifting, even reading the words on this page.

Aggressive prostate cancer cells, lacking PTEN proteins, not only possess mitochondria, they covet them and go to great lengths to preserve them.

"Cells in general have two ways of generating energy," Trotman said. "One is through the mitochondria, and the other is burning sugar. When you inhibit the mitochondria with metformin, cells are forced to burn sugar."

Once cancer cells have burned all their fuel, they die, he said.

Missing PTEN suppressors have been found in other cancers, and small, inconclusive clinical studies have been conducted under the auspices of the National Cancer Institute to explore the role of metformin in cancer therapy.

The aims of these investigations have been to determine if metformin might be used as a cancer preventive in obese patients at high risk for cancer of all kinds, and who also had elevated blood sugar. Those doctors have not yet reported their results.

Dr. Gerald Bernstein of Lenox Hill Hospital in Manhattan, who was not involved with Cold Spring Harbor Lab's research, said metformin probably works against cancer for many of the same reasons it helps to control diabetes.

"Metformin, at the present time, is the most commonly prescribed diabetic medication in the world," said Bernstein, an endocrinologist and coordinator of the Friedman Diabetes Program at Lenox Hill. It was developed in Europe and prescribed for years before being approved by the U.S. Food and Drug Administration in the mid-1990s.

In diabetes, metformin reduces the amount of glucose — sugar — produced by cells in the liver. It also acts directly on the mitochondria by inhibiting respiration in the tiny bean-shaped pods.

"Most people who are on metformin, including myself, take the generic because it is perfectly effective and very inexpensive," Bernstein said. The drug also is sold under the brand name Glucophage. Sixty tablets costs about $4 at prescription drug discounters, such as Walmart.

"Some of us think it should be in baby's milk," Bernstein said jokingly.

He added that metformin helps improve the integrity of blood vessels. And clinical studies also have shown that it can lower the risk of advancing from pre-diabetes to full-blown disease.

Trotman, meanwhile, is delving into the next part of the puzzle involving missing PTEN proteins, metformin and prostate cancer.

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A looming scientific question is when it should be administered. Trotman noted that the drug probably would be best given when patients' glucose levels are low, a point when there is little fuel left and no hope for cancer cells to cling to life.
https://www.newsday.com/news/health/prostate-cancer-diabetes-metformin-1.18199913

## Evolutionary history of tumor helps predict severity of prostate cancer

April 20, 2018

Findings from Canadian Prostate Cancer Genome Network (CPC-GENE) researchers and their collaborators, published today in *Cell*, show that the aggressiveness of an individual prostate cancer can be accurately assessed by looking at how that tumor has evolved. This information can be used to determine what type and how much treatment should be given to each patient, or if any is needed at all.

The researchers analyzed the whole genome sequences of 293 localized prostate cancer tumors, linked to clinical outcome data. These were then further analyzed using machine learning, a type of statistical technique, to infer the evolutionary past of a tumor and to estimate its trajectory. They found that those tumors that had evolved to have multiple types of cancer cells, or subclones, were the most aggressive. Fifty-nine per cent of tumors in the study had this genetic diversity, with 61 per cent of those leading to relapse following standard therapy.

"By incorporating time into the context of the existing knowledge we have about where a tumor is at diagnosis we were able to very accurately identify those patients whose prostate tumours needed no treatment, those men who could be cured by existing treatments, and those men who had very aggressive tumours and may have benefitted from novel therapeutic options," says Dr. Paul Boutros, Principal Investigator, Ontario Institute for Cancer Research and leader of CPC-GENE.

"Clinical decision making in treating prostate cancer can be very difficult. These findings pave the way for a new tool to improve our ability to determine the best approach for each individual patient, including sparing patients from unnecessary treatment or over-treatment and the associated side effects," says Professor Robert Bristow, Director of the Manchester Cancer Research Centre at the University of Manchester U.K., formerly of the Princess Margaret Cancer Centre in Toronto.

"Tumors are a community of related cancer cells, and by examining their DNA using machine learning, we can gain insight into how they evolved from normal cells. In this paper, we show that the past evolutionary history of a tumor helps predict whether that tumor will progress into an aggressive form," says Dr. Quaid Morris, Associate Professor, The Donnelly Centre for Cellular and Biomolecular Research, University of Toronto, who collaborated with the CPC-GENE team on the study.

"Prostate cancer is the most common cancer among men," says Reza Moridi, Ontario's Minister of Research, Innovation and Science. "Ontario congratulates this research team, whose work is pointing the way toward improved testing and treatment."

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The study's findings are not its only contributions to prostate cancer research. The sequencing data generated during the course of the study are now freely available online to researchers worldwide to carry out further analyses, becoming the largest prostate cancer genomics resource available to-date.

CPC-GENE is a team of multidisciplinary researchers from across Canada working to crack the genetic code of prostate cancer. Through funding of approximately $20 million, research of this magnitude has been made possible through a partnership between the Movember Foundation, Prostate Cancer Canada, and the Ontario Institute for Cancer Research. Dr. Stuart Edmonds, Vice-President of Research, Health Promotion and Survivorship at Prostate Cancer Canada, has released the following statement:

"From the tireless work of researchers to the selfless giving of donors, we applaud the efforts of everyone who has played a role in helping make CPC-GENE possible. Since its beginnings as an ambitious undertaking that was massive in scope, the goal of this project has been to greatly improve personalized care for men with prostate cancer. The findings published in *Cell* - widely considered one of the most prestigious and highest impact medical journals - represent a monumental stride towards that goal. Together, we will continue to advance this important work on behalf of the one in seven Canadian men who will be diagnosed with prostate cancer and their families."

https://www.news-medical.net/news/20180420/Evolutionary-history-of-tumor-helps-predict-the-severity-of-prostate-cancer.aspx

## Brachytherapy – What is it?

Brachytherapy delivers radiation internally. There are 2 main types: low-dose seed implant brachytherapy and high-dose rate brachytherapy (HDR).

Low-dose seed implant brachytherapy

- Usually recommended to men with lowergrade cancers that are contained within the prostate gland.
- Between 80 and 100 radioactive seeds, the size of a grain of rice, are implanted directly into the prostate.
- Each seed releases low-energy level radiation steadily over several months.

HDR

- Reserved for patients with high-grade cancers.
- High-dose radiation is received through approximately 15 needles in the prostate, concentrating on the cancerous areas.

### What is done?

Low-dose seed implant brachytherapy

- The seeds are inserted through the skin in the perineum.
- Procedure is performed under either general or spinal anesthesia and lasts approximately 1 hour.

HDR

- Under anesthesia, approximately 10–15 needles are inserted through the perineum.
- These needles are wired to the radiation source that delivers a high radiation dose to the prostate.
- The needles are then removed.

- The treatment takes 10–20 minutes.

**What to expect?**

Low-dose seed implant brachytherapy

- A catheter may be used for a short time for urine drainage.

HDR

- Often preceded or followed by a few weeks of external beam radiation.
- Sometimes HDR treatments are given over a few days and the external beam radiation is not needed.

**Side effects and risks**

- Side-effects of brachytherapy are similar to those of external beam radiation.
- Brachytherapy differs slightly in the following ways:
  - Dominant short-term side-effect is irritation to the bladder and urethra
  - Acute urinary r etention may develop
  - Bowel irritation is relatively uncommon
  - Side effects may last months

**Long term side effects**

- Very rare risk of incontinence of urine or chronic urinary obstruction (both less than 5%).
- Erectile Dysfunction may occur, though many men will be able to achieve an erection with the use of prescription medication.

http://www.prostatecancer.ca/Prostate-Cancer/Treatment/Brachytherapy

## The Best Nutrition to Combat Prostate Cancer

*by Jenny Holt | March 26, 2018*

Recent reports from the Nature Genetics state that a high-fat diet can significantly increases the likelihood of prostate cancer. Given that every 18 minutes another American man dies from prostate cancer, that's certainly food for thought. If you are a man facing a prostate cancer diagnosis, read on to learn what you can do to optimize your nutrition and give your body its best chance of fighting back?

**If you've just been diagnosed**

Try to stay positive; the good news is that the 5-year survival rate for most men with localized prostate cancer – that's cancer that hasn't spread – is almost 100%. Though there are many risk factors for prostate cancer that are beyond our control – such as age, family history, ethnicity and so on – there are various lifestyle choices that you can make to improve your chances.

**Take a look at your diet**

While no one is suggesting that there is a diet out there that is capable of curing prostate cancer, ensuring optimum nutrition can help strengthen the immune system. Try to include a wide variety of fruits and vegetables into your diet. Several studies suggest that diets high in certain vegetables may be linked with a lower risk of prostate cancer; cruciferous vegetables are said to be particularly good – so make a beeline for

broccoli, cauliflower and cabbage. According to the American Cancer Institute, tomato consumption has been linked to lower instances of prostate cancer – thanks to the antioxidant lycopene, that they contain.
When choosing carbohydrates, try to stick to wholegrain varieties, be it brown rice, wholegrain bread or whole-wheat pasta. Nuts and seeds are good sources of healthy fats and nutrients, including the antioxidant vitamin E. Anything to avoid? Some studies have found that diets rich in red meat, dairy products, and animal fat may raise prostate cancer risk – so it might be wise to limit consumption of those foods.
Additionally, prostate cancer's ability to spread appears to be affected by what you eat too, according to recent research contained in journal Nature Genetics. Cancer cells can become more aggressive and spread beyond the prostate itself when they have plentiful access to fat – suggesting that a low-fat diet is the way to go.

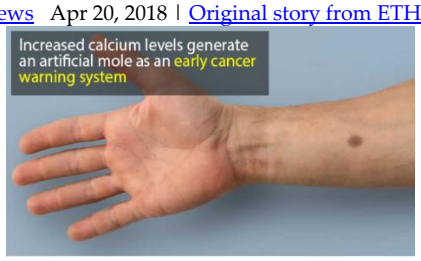
### What else can you do?

You might consider adding supplements to ensure your diet is providing you with the optimum level of all the nutrients you need. This can be helpful if there are gaps in your diet, but be wary of any 'magic' supplements that appear to promise the world. It's important to avoid nutritional myths so you don't waste money on supplements that won't do you any real good.
It's important to stay physically active. Keeping to a healthy weight has been shown to reduce the risk of getting the most dangerous, aggressive form of prostate cancer; exercise can help with mood too.
The bottom line is, get checked early. Digging your head in the sand will not help. Far better to learn all you can about your condition so you are in a position to make informed decisions and take positive action.
https://zerocancer.org/blog/best-nutrition-combat-prostate-cancer/

# NOTABLE

## An Artificial Mole As An Early Warning Sign

News Apr 20, 2018 | Original story from ETH Zurich




*Credit: ETH Zurich*

Alongside cardiovascular disease, cancer has become the top cause of death in industrialised countries. Many of those affected are diagnosed only after the tumour has developed extensively. This often reduces the chance of recovery significantly: the cure rate for prostate cancer is 32 percent and only 11 percent for colon cancer. The ability to detect such tumours reliably and early would not only save lives, but also reduce the

need for expensive, stressful treatment.

Researchers working with Martin Fussenegger, Professor at the Department of Biosystems Science and Engineering at ETH Zurich in Basel, have now presented a possible solution for this problem: a synthetic gene network that serves as an early warning system. It recognises the four most common types of cancer – prostate, lung, colon and breast cancer – at a very early stage, namely when the level of calcium in the blood is elevated due to the developing tumour.

### Derailed calcium balance triggers melanin production

The early warning system comprises a genetic network that biotechnologists integrate into human body cells, which in turn are inserted into an implant. This encapsulated gene network is then implanted under the skin where it constantly monitors the blood calcium level.

As soon as the calcium level exceeds a particular threshold value over a longer period of time, a signal cascade is triggered that initiates production of the body's tanning pigment melanin in the genetically modified cells. The skin then forms a brown mole that is visible to the naked eye.

The mole appears long before the cancer becomes detectable through conventional diagnosis. "An implant carrier should then see a doctor for further evaluation after the mole appears," explains Fussenegger. It is no reason to panic. "The mole does not mean that the person is likely to die soon," stresses the ETH professor. It simply means that clarification and if necessary treatment are needed.

The researchers used calcium as the indicator of the development of the four types of cancer, as it is regulated strongly in the body. Bones serve as a buffer that can balance out concentration differences. However, when too much calcium is detected in the blood, this may serve as a sign for one of the four cancers.

### Early detection increases survival rate

"Early detection increases the chance of survival significantly," says Fussenegger. For example, if breast cancer is detected early, the chance of recovery is 98 percent; however, if the tumour is diagnosed too late, only one in four women has a good chance of recovery. "Nowadays, people generally go to the doctor only when the tumour begins to cause problems. Unfortunately, by that point it is often too late."

The implant also has an additional advantage: "It is intended primarily for self-monitoring, making it very cost effective," explains the ETH professor. However, for those who would prefer not to deal with the constant stress, an implant can also be used that develops a mark visible only under a red light. "This regular check could be carried out by their doctor."

The disadvantage is that the service life of such an implant is limited, as Fussenegger has found in literature. "Encapsulated living cells last for about a year, according to other studies. After that, they must be inactivated and replaced."

### A reliable prototype

So far, this early warning implant is a prototype; the associated work recently published in the journal Science Translational Medicine is a feasibility study. The researchers have tested their early warning system in a mouse model and on pig skin. It functioned reliably during these tests. Moles developed only when the

calcium concentration reached a high level.

The Basel-based scientists still have a long way to go before human testing can begin. “Continued development and clinical trials in particular are laborious and expensive, which we as a research group cannot afford,” says the ETH professor. However, he would like to promote the translation of his developments, so that one day they will lead to applicable products. He estimates that bringing such a cancer diagnosis implant to market maturity will take at least ten years of research and development.

The concept of the “biomedical tattoo”, as Fussenegger describes this new finding, would also be applicable to other gradually developing illnesses, such as neurodegenerative diseases and hormonal disorders. In principle, the researchers could replace the molecular sensor to measure biomarkers other than calcium.

This article has been republished from materials provided by ETH Zurich. Note: material may have been edited for length and content. For further information, please contact the cited source.

**Reference:**Tastanova A, Folcher M, Müller M, Camenisch G, Ponti A, Horn T, Tikhomirova MS, Fussenegger M. Synthetic biology-based cellular biomedical tattoo for detection of hypercalcemia associated with cancer. Science Translational Medicine 10, eaap8562 (2018) 18 April 2018. DOI: 10.1126/scitranslmed.aap8562

https://www.technologynetworks.com/diagnostics/news/an-artificial-mole-as-an-early-warning-sign-299911

## QUOTABLE

“Attitude is a little thing that makes a big difference.” Sir Winston Churchill

“Healing takes courage, and we all have courage, even if we have to dig a little to find it. “ Tori Amos

"Worrying is like a rocking chair: it gives you something to do, but doesn't get you anywhere." Van Wilder

# PCCN Markham

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***PCCN Markham***

***Prostate Cancer Support Group***

**Meets the 2nd Tuesday**
**Every month**
**September – June**
**St. Andrew's Presbyterian Church**
**143 Main St Markham**

**The Markham PCCN Prostate Support Group is generously supported by Dr. John DiCostanzo, Astellas Pharma, PCCN, St. Andrews Presbyterian Church, and the Canadian Cancer Society.**

The group is open to all; survivors, wives, partners, relatives and those in our community who are interested in knowing about prostate health. Drop by St Andrews Presbyterian Church 143 Main Street Markham at 7:30PM, the 2nd Tuesday every month from September to June. The information and opinions expressed in this publication are not endorsements or recommendations for any medical treatment, product, service or course of action by PCCN Markham its officers, advisors or editors of this newsletter.

**Treatment should not be done in the place of standard, accepted treatment without the knowledge of the treating physician.**

The majority of information in this newsletter was taken from various web sites with minimum editing. We have recognized the web sites and authors where possible.

PCCN Markham does not recommend treatment, modalities, medications or physicians. All information is, however, freely shared.

Email *markhampccn@gmail.com*

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