

### New Cellular Immunotherapy for Cancers

A new study published in the journal Nature Communications on 22 October 2019 reports a new approach to cancer treatment that increases the immune capability of T lymphocytes against malignant cancers. This allows the T cells to successfully infiltrate and destroy these tumors. Animal trials have been completed and the researchers are planning clinical trials in human beings next.

#### What is immunotherapy?

Immunotherapy has been the start of a

new age of cancer treatments, including immune checkpoint blockade against inhibitory receptors like PD-1 (programmed cell death-1), to increase T cell responses. T cells are the lymphocyte subset directly involved in cellular attack on foreign cells and particles, including malignant cells. Another mechanism that has been exploited is the direct suppression of signaling pathways that reduce T cell responses to tumor-related neoantigens. Both these pathways result in T cell activation, which is accompanied by

regulators that prevent excessive inflammation and autoimmunity due to hyperactive T cell responses.

#### The study – Rasal1

The present study takes note of the role of a T cell molecule called Rasal1 (Ras protein activator-like 1 protein) that appears after T cell activation. This molecule binds to the T cell receptor (TCR) complex that is responsible for initiating all T cell responses to foreign antigens. The effect of Rasal1 binding

*(Continued on page 2)*

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*Thanks!*

*Next Meeting:*

#### **Wednesday, 20 November 2019**

*This is the wind-up event for the 2019 program.  
There's no speaker but there is a potluck  
smorgasbord along with live musical entertainment.  
Don't miss it.*

**Location:** The First Unitarian Universalist Church of Winnipeg,  
603 Wellington Crescent

**Time:** 7 – 9 pm.

*Free Admission    Everyone Welcome  
Plenty of free parking    ☆ Door prizes ☆*



The Manitoba Prostate Cancer Support Group offers support to prostate cancer patients but does not recommend any particular treatment modalities, medications or physicians ; such decisions should be made in consultation with your doctor.

**MPCSG – active since 1992.**

*Thought of The Day*

*Nobody trips over mountains. It is the small pebble that causes you to stumble.  
Pass all the pebbles in your path and you will find you have crossed the mountain.*

*~Author Unknown*

(Continued from page 1)

to the TCR is to inhibit further activation pathways within the T cells, causing T cell anergy or unresponsiveness towards cancer antigens.

### Two pathways of T cell inhibition via Rasal1

The experiments showed that T cell activation by the binding of anti-CD3 antibodies to the TCR led to an increase in both Rasal1 at 24 hours and 48 hours, in both CD4 and CD8+ cells. The anti-CD3 antibodies caused the Rasal1 to move from the cytoplasm to the membranes of the activated T cells. It then associates with the ZAP-70 which is a protein kinase enzyme linked to T cell activation, and suppresses its activity, causing inhibition of T cell activity.

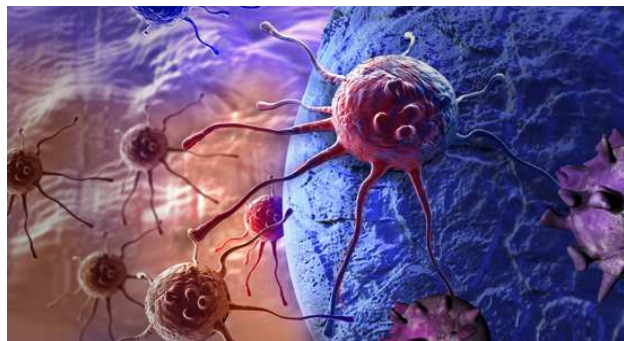
This molecule also acts on extracellular-activated kinase (ERK) of the Ras-Raf-MEK-ERK pathway that activates T cells. Overall, the Rasal1 binding to the TCR causes reduced proliferative activity of T cells in proportion to the degree of receptor binding by the antigen. When the expression of Rasal1 within the T cells is reduced, T cells proliferate more actively and also have a higher cell expansion capacity.

### Limiting tumor growth via Rasal1 downregulation

These findings were confirmed in a mouse model. When T cells with low levels of Rasal1 activity were transferred into mice with metastatic melanoma in the lung, the tumor's growth was slowed. This was associated with a threefold increase in the number of T cells with CD8+ antigens on the cell surface. The tumor-infiltrating lymphocytes from these mice also showed a greater number of CD8+ cells with granzyme B and interferon  $\gamma$ -1 (IFN- $\gamma$ 1), two molecules that are responsible for cancer cell

destruction by cytotoxic T cells.

When T cells with low Rasal1 levels were injected into another mouse model, this time with a solid lymphoma, tumor size reduced significantly from a mean of 570 mg to 370 mg, while the number of infiltrating T cells in the tumor increased fourfold, indicating a robust immune attack on the tumor cells. Molecular markers that signpost T cell activation were also increased, along with the effector molecules granzyme B and IFN- $\gamma$ 1. Thus T cell proliferation and activation level was increased by suppressing the expression of Rasal1, along with a slowing of the growth of the solid tumor.



### Conclusion and implications

The current study picked up a novel T cell pathway that could help increase the effectiveness of cancer immunotherapy. This pathway involves the molecule Rasal1, which is shown to reduce T cell activity against cancer cells. Rasal1 seems to be only weakly present in inactive T cells and may therefore be upregulated only after the initial activation of T cells, to prevent inappropriate immune responses which could damage the host.

The inhibitory effect of Rasal1 on T cell activation was found to occur both in the cell cultures and in animal models. It occurs via two main pathways, one involving the ZAP-70

protein and the other via the p21-ERK pathway. On the other hand, T cells that do not express Rasal1 actively reduced the growth rate of metastatic lung melanomas solid lymphomas in mouse models.

T cells without Rasal1 expression have a greater anti-tumor effect. This is associated with a significant rise in the number of tumor-infiltrating T cells with the CD8 antigen. There is also an accompanying marked rise in the expression of killer effector molecules granzyme B (GZMB) and IFN- $\gamma$ 1, which directly kill tumor cells. This makes it potentially useful in cancer immunotherapy by inhibiting Rasal1, or to develop specific T cells that have higher anti-tumor immunity via already established approaches like CART.

Researcher Christopher E. Rudd says, "Hyperactivation of T cells enables them to penetrate and attack tumors. This discovery demonstrates that modulation of the identified [T cell] protein can activate the immune system and lead to destruction of the cancer cells."

By Dr. Liji Thomas, MD  
Oct 26 2019

Source:

<https://nouvelles.umontreal.ca/article/2019/10/24/cancer-une-grande-decouverte-en-immunotherapie-realisee-par-un-chercheur-de-l-hmr/>

<https://www.news-medical.net/news/20191026/New-cellular-immunotherapy-for-cancers.aspx>

Journal reference:

GTPase-activating protein Rasal1 associates with ZAP-70 of the TCR and negatively regulates T-cell tumor immunity. Youg Raj Thaker, Monika Raab, Klaus Strebhardt & Christopher E. Rudd. Nature Communications 10, Article number: 4804 (2019).  
<https://doi.org/10.1038/s41467-019-12544-4>.  
<https://www.nature.com/articles/s41467-019-12544-4#article-info>

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## Urine Test Predicts Prostate Cancer Progression, Need for Treatment

A urine test developed by researchers at University of East Anglia and Norfolk and Norwich University Hospital in the U.K. appeared to accurately predict whether men with prostate cancer will need treatment as many as 5 years earlier than current methods.

“Current practice assesses a patient’s disease using a PSA blood test, prostate biopsy and MRI. However, up to 75% of men with increased PSA levels are negative for prostate cancer on biopsy. Meanwhile 15% of patients who do not have a raised PSA are found to have prostate cancer — with a further 15% of these cancers being aggressive,” Shea P. Connell, PhD student and researcher at the University of East Anglia’s Norwich Medical School, said in a press release.

“A policy of ‘active surveillance’ has been developed to combat this uncertainty, but it requires invasive follow-ups and constant reminders that a patient has a cancer with an uncertain natural history,” he added. “This results in up to 50% of men on active

surveillance self-electing for treatment — whether they need it or not. There is a considerable need for additional, more accurate tests.”

*HemOnc Today* spoke with Connell about what prompted the development of this test, the results of the study and subsequent research underway.

**Question:** What prompted the development of this test ?

**Answer :** We are unsure about what to do with men with low-grade prostate cancer, with watch and wait the current norm for these men. Disease progression triggers treatment, but we have no way to predict this. In the U. K., men diagnosed with low-risk, early-stage disease are invited to receive surveillance. They then usually undergo an MRI and a repeat biopsy and are assigned to PSA surveillance and maybe a repeat biopsy 1 year after. Every year, these men are reminded that they have prostate cancer and that we do not know how to treat them.

**Q:** Can you describe how the test works?

**A:** This test appears to be able to predict prostate cancer progression up to 5 years in advance from a single urine sample. In theory, a man diagnosed with early-stage prostate cancer can provide one urine sample and we can tell him if he will be relatively safe for another 5 years without annual follow-up tests. We assess cell-free RNA with NanoString technology, which is similar to a barcode. After this, we look at the expression pattern of 35 different genes and use this in a regression analysis to produce the Prostate Urine Risk, or PUR, risk score.

Shea P. Connell    October 24, 2019

source: <https://www.healio.com/hematology-oncology/prostate-cancer/news/online/%7B523d560b-464c-4a8f-b347-3fa08f4e5888%7D/urine-test-predicts-prostate-cancer-progression-need-for-treatment>

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## Apalutamide Linked to OS Benefit in Nonmetastatic CRPC

The apalutamide effect on OS was observed across patient subgroups, especially in those <65 years (HR, 0.33; 95% CI, 0.14-0.74), no prior radical prostatectomy or local radiation (HR, 0.82; 95% CI, 0.60-1.11), N1 stage disease (HR, 0.52; 0.29-0.94), prior bone-sparing therapy (HR, 0.55; 95% CI, 0.27-1.11), and PSADT >6 months (HR, 0.57; 95% CI, 0.34-0.95). The OS benefit was also seen with apalutamide, despite patient crossover from the placebo arm.

Of those who initiated cytotoxic chemotherapy (n = 197), 14% (n = 115) of patients were on apalutamide and 20% (n = 401) were on placebo. The time to initiation of chemotherapy was not reached in either arm (HR, 0.60; 95% CI, 0.45-0.80). “Per protocol sequential testing, time to initiation of chemotherapy was not formally testing for statistical significance since the OS analysis was not significant,” Smith said. “None the less, it appears that apalutamide is associated with a delay in the time to initiation of chemotherapy.”

Moreover, apalutamide was also found to significantly extend PFS2 compared with placebo, with a median PFS2 of 55.5 months and 43.8 months, respectively (HR, 0.55; 95% CI, 0.45-0.68; P <.0001). Sixty-nine percent of those on the placebo arm and 40% of patients on apalutamide received subsequent therapy, which mainly consisted of abiraterone acetate plus prednisone.

Regarding safety, apalutamide’s profile was found to be consistent with prior reports. The median duration of treatment was 31.4 months with the AR inhibitor and 11.5 months with placebo. AEs were reported in 97.3% and 93.7% of apalutamide- and placebo-treated patients, respectively. Grade 3/4 AEs were reported in 53.1% of patients on the apalutamide arm and 36.7% of those on placebo, and serious AEs occurred in 33.5% and 24.9%, respectively. The treatment discontinuation rate was nearly doubled with apalutamide (13.6%) compared with placebo (7.3%). AEs that led to death occurred in 2.1% and 0.5% of patients on apalutamide and placebo, respectively.

Discontinuation rates due to disease progression were 34% in the apalutamide group compared with 74% in the placebo group, Smith explained.

Karim Fizazi, MD, PhD, commented on the OS data as an invited discussant following Smith’s presentation.

“It is true when you’re looking at the hazard ratio, it is 0.5, but as very honestly announced by Dr. Matthew Smith, it is a very nonsignificant difference at this point. The P value is about .02 at the moment, and the boundary to claim [OS significance] is .0121. At the moment, we cannot say that apalutamide prolongs survival in this setting. [With PFS2], there is a more meaningful difference we are seeing; this is utterly important because abiraterone was provided as a potential

salvage treatment in both arms. In other words, it is comparing deferred AR-targeting, at least in some patients, and at the same time, it is making a difference in [PFS2].”

In September 2019, the FDA granted a second approval to apalutamide for the treatment of patients with metastatic castration-sensitive prostate cancer.

### References

1. Smith MR, Saad F, Chowdhury S, et al. Apalutamide and overall survival in patients with nonmetastatic castration-resistant prostate cancer: updated results from the phase III SPARTAN study. Presented at: 2019 ESMO Congress; September 27 to October 1, 2019; Barcelona, Spain. Abstract 843O. Small EJ, Saad F, Chowdhury S, et al. Apalutamide and overall survival in non-metastatic castration-resistant prostate cancer. *Ann Oncol*. 2019;0:1-8. doi: 10.1093/annonc/mdz397.
1. Small EJ, Saad F, Chowdhury S et al. SPARTAN, a phase 3 double-blind, randomized study of apalutamide (APA) versus placebo (PBO) in patients (pts) with nonmetastatic castration-resistant prostate cancer (nmCRPC). *J Clin Oncol*. 2018;36(suppl 6s; abstract 161). doi: 10.1200/JCO.2018.36.6\_suppl.161.

Gina Columbus @ginacolumbusonc

Friday, Sep 27, 2019

source: <https://www.onclive.com/conference-coverage/esmo-2019/apalutamide-linked-to-os-benefit-in-nonmetastatic-crpc>

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## Panama Approves New Drug to Fight Prostate Cancer

Panama is the fourth country in Central America to endorse the use of apalutamide, a medicine for men with prostate cancer that has not spread (metastasis) and is resistant to conventional treatment (hormonal), with high probabilities of progression

and extension to other organs. According to figures from the National Oncology Institute (ION), between 2009 and August 2019, 3,401 new cases of prostate cancer were detected in Panama

Apalutamide approval was given after the safety and efficacy results of the Spartan Phase III study, which included more than 1,200 patients, have been found, in which it is proven that adding this molecule to

(Continued on page 5)



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conventional therapy reduces the risk by 72% of developing metastases, while managing to delay its appearance or more than two years.

" Apalutamide not only slows the progression of the disease but allows

patients to improve their quality of life," said Dr. Óscar González, Janssen Medical Manager, developer of the treatment.

The drug has also been approved by the Dominican Republic, Honduras and Nicaragua.

26/10/2019

source: <https://www.newsroompanama.com/health/panama-approves-new-drug-to-fight-prostate-cancer>

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## Apalutamide Survival Benefits Unclear for Castration-Resistant Prostate Cancer

Despite prolonged progression-free duration, a 25% reduction in risk of death associated with nonsteroidal anti-androgen agent apalutamide (Erleada) fell short of statistical significance in patients with non-metastatic, high-risk castration-resistant prostate cancer (nmCRPC). This was shown in an updated interim analysis 1 from the SPARTAN trial which was presented at the 2019 European Society of Medical Oncology (ESMO) meeting in Spain and in the *Annals of Oncology*.

Median overall survival (OS) has not been reached and definitive conclusions about apalutamide-associated survival benefits in nmCRPC patients depends on longer-term follow-up with more mature data, study authors reported. The study was funded by Janssen Research & Development.

"Because the difference in OS between groups did not cross the pre-specified O'Brien-Fleming boundary for statistical significance [of  $P = .0121$ ], the final OS analysis will occur when 427 deaths are observed," reported Eric J. Small, MD, of the UC San Francisco Helen Diller Family Comprehensive Cancer Center in California and coauthors.

A total of 1207 patients with nmCRPC were randomly assigned 2:1 to receive androgen deprivation therapy (ADT)

plus either apalutamide (240 mg daily) or a placebo in the randomized, double-blind, placebo-controlled phase III trial.



At median follow-up of 41 months, 4-year overall survival rates were 72.1% for patients receiving apalutamide vs 64.7% in the placebo arm (hazard ratio [HR] 0.75; 95% CI: 0.59-0.96;  $P = .0197$ ). The statistically nonsignificant numeric survival benefit associated with apalutamide was "consistent" across all pre-specified subgroups analyzed.

"The [numerical] OS difference favoring apalutamide was observed despite a crossover of 19% of patients from placebo to apalutamide, and a higher use of subsequent life-prolonging therapy in the placebo group (69% versus 40%)," the study authors wrote.

Twenty-eight percent of apalutamide group patients and 37% of patients in the placebo group experienced disease

progression during second-line therapy. Apalutamide was associated with an 11.8-month longer time to second disease progression (PFS2, an exploratory endpoint) than placebo (55.6 vs 43.8 months; HR 0.55; 95% CI: 0.45-0.68;  $P < .0001$ ).

Fifty-three percent of patients receiving apalutamide experienced grade 3 or 4 adverse events, and 33.5% experienced serious adverse events, with 13.6% of patients discontinuing treatment because of toxicities. The most frequent grade 3 or 4 adverse event was hypertension (16%). Other common adverse reactions included fatigue, arthralgia, rash, decreased appetite, fall, weight loss, hot flush, diarrhea, and fracture. Grade 3 or 4 anemia was reported for 70% of patients on apalutamide; 47% experienced leukopenia, and 41% experienced lymphopenia.

Bryant Furlow October 3, 2019  
ESMO, Prostate Cancer

### References:

1. E J Small, F Saad, S Chowdhury, S Oudard, B A Hadaschik, J N Graff, D Olmos, P N Mainwaring, J Y Lee, H Uemura, P De Porre, A A Smith, K Zhang, A Lopez-Gitlitz, M R Smith, Apalutamide and overall survival in non-metastatic castration-resistant prostate cancer, *Annals of Oncology*, <https://doi.org/10.1093/annonc/mdz397>

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## Prostate Cancer Salvage Combo Benefits Confirmed

A post-hoc analysis of a phase 3 trial demonstrated that adding 6 months of androgen deprivation therapy to salvage radiotherapy for recurrent prostate cancer following radical prostatectomy improves progression-free survival at 120 months.

Final results of a phase 3 trial confirm the progression-free survival (PFS) benefit of adding short-term androgen suppression to radiotherapy as salvage treatment for men who experience rising PSA following radical prostatectomy for prostate cancer (PCa), investigators concluded in a paper published in *Lancet Oncology*.

In a post-hoc analysis of men in the GETUG-AFU 16 trial, the 120-month PFS rates were 64% for men treated with radiotherapy plus 6 months of androgen deprivation therapy with goserelin compared with 49% for those who received radiotherapy alone. The combined treatment was significantly associated with a 46% reduction in the risk of disease progression—the study's primary end point—

compared with radiotherapy alone. The investigators, led by Christian Carrie, MD, of Léon Bérard Center and the University of Lyon in France, defined PFS as the time from randomization to documented biologic recurrence or clinical progression (or both), death from any cause, or censoring at the date of last follow-up.

In addition, the investigators found that the combined treatment offered an advantage in terms of metastasis-free survival (MFS), a prespecified secondary end point of the trial. The 120-month MFS rates were 75% for the combined treatment arm compared with 69% for the radiotherapy alone arm. The combined treatment was significantly associated with a 27%

decreased risk of metastasis compared with radiotherapy alone.

Overall survival rates at 120 months did not differ significantly between the combined treatment and radiotherapy alone arms (86% vs 85%), Dr Carrie's team reported.

When adding androgen suppression to salvage radiotherapy, 14 patients would need to be treated to spare 1 patient from experiencing metastasis or death in the 10 years after salvage therapy, according to the investigators.



Subgroup analyses found that the beneficial effect of the combined treatment on PFS was significantly greater for patients with low-risk than high-risk cancer. Compared with radiotherapy alone, the combined treatment was significantly associated with a 53% decreased risk of progression in the low-risk patients compared with a 44% decreased risk in the high-risk group. The investigators found no significant difference in MFS between treatment arms when they stratified patients according to risk group.

“Our results confirm the benefit of adding short-term androgen deprivation therapy to salvage radiotherapy on metastasis-free survival in patients with

biological recurrence after radical prostatectomy,” the authors wrote.

Dr Carrie and his colleagues noted that the FDA recently approved MFS as an end point for patients with PCa enrolled in clinical trials, and MFS “has been described as a strong surrogate of overall survival.”

### Related Articles

Radiation Therapy After Prostate Surgery Offers No Benefit

Radiation + ADT Superior for Prostate Cancer Salvage Therapy

The GETUG-AFU 16 trial enrolled

patients from October 19, 2006 to March 30, 2010. Dr Carry and his collaborators randomly assigned 743 men with to receive radiotherapy alone (374 patients) or radiotherapy in addition to goserelin (369 patients). One patient in the radiotherapy alone group later withdrew consent, leaving 373 patients in that group. The median follow-up was 112 months. In the first analysis of the trial, which was published in 2016, only the first

progression event was collected, so MFS could not be analyzed, the authors noted.

### Reference

Carrie C, Magné N, Burban-Provost P, et al. Short-term androgen deprivation therapy combined with radiotherapy as salvage treatment after radical prostatectomy for prostate cancer (GETUG-AFU 16): a 112-month follow-up of a phase 3, randomised trial [published online October 16, 2019]. *Lancet Oncol*. doi: 10.1016/S1470-2045(19)30486-3

Jody A. Charnow October 21, 2019

source: <https://www.renalandurologynews.com/home/news/urology/prostate-cancer/prostate-cancer-salvage-combo-benefits-confirmed/>

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## A Dairy-Heavy Diet Has Been Associated With A Higher Risk Of Prostate Cancer

Researchers have reviewed 47 studies published since 2006, comprising of more than one million participants, in an attempt to better understand links between prostate cancer and plant or animal-based foods.

Accordingly, it was discovered that high consumption of dairy products, like milk and cheese, appears to be connected with an increased risk of prostate cancer, which typically affects men over the age of 50.



"Our review highlighted a cause for concern with high consumption of dairy products," stated lead author Dr.

John Shin, a Mayo Clinic oncologist. "The findings also support a growing body of evidence on the potential benefits of plant-based diets."

There was no clear association of increased risk of prostate cancer in relation to other animal-based foods, including red and white meat, processed meats and fish. But the researchers did identify a decreased risk of prostate cancer associated with plant-based diets.

Dr. Shin and his team now want to conduct a further investigation into the topic.

And the doctor noted that prior studies have reported that dairy products are the primary source of calcium in Western countries, where rates of prostate cancer are high, while there are lower rates of the disease in Asian countries, where dairy intake is low.

Prostate cancer develops in the prostate, a gland in the male reproductive system. Symptoms include an increased need to urinate and a feeling like the bladder has not fully emptied. Men should contact their GP immediately if they are concerned.

source: <https://www.msn.com/en-za/health/medical/dairy-heavy-diet-linked-to-higher-risk-of-prostate-cancer/ar-AAJpxRf>

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## iMedicalApps: Predict Prostate Cancer

Web app for shared decision making for non-metastatic prostate cancer

In the U.K., researchers created a prognostic tool to aid patients and providers regarding watchful waiting (conservative management) vs radical management (surgery and/or hormone treatment). This group used several large databases to derive and then validate the tool.

Again, this is only for non-metastatic prostate cancer and some groups are not well-represented in their cohorts. Nonetheless, the tool enables patients and providers to put in individual patient data (age, PSA, Gleason score, etc.) to help them decide between treatment approaches. Information is presented both verbally and graphically to include side effects such as erectile dysfunction, incontinence, etc.

The web app takes the externally validated Predict Prostate prognostic model and adapts it to mobile browsers.

The prognostic model has been extensively researched with ongoing studies to assist patients and providers in determining the "best" treatment for non-metastatic prostate cancer. The model has been endorsed by NICE and the University of Cambridge Academic Urology Group.

The Predict Prostate web-based prognostic tool brings evidence-based decision making for non-metastatic prostate cancer to any device with an internet browser. The app takes individual patient data to provide patients and their providers' information on prognosis with conservative management vs radical treatment including potential side effects. If you treat patients with prostate cancer, then this prognostic tool is worth a look.

Despite the web-only format, the app is easy to complete and view results on mobile browsers.

### Likes

- Evidence-based prediction tool with details on derivation/validation
- Detailed information on pros/cons including side effects
- Numerous links to PubMed articles, resources for patients

### Dislikes

- No dedicated app, only a web app
- Some information may be challenging for patients to input/understand
- Some graphics may be difficult to view on smaller devices

This post appeared on iMedicalApps.com

by Douglas Maurer DO, MPH, FAAFP  
October 25, 2019

<https://www.medpagetoday.com/blogs/iltifathusain/82957>

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**FUTURE MEETINGS 2019 - 2020**

**18 Dec. NO MEETING**

**15 Jan. 2020**

Speaker: Dr. Premal Patel  
 (Specialist in infertility and sexual medicine)  
 Topic: Sexual dysfunction and urinary  
 incontinence after prostate cancer  
 treatment

**19 Feb. 2020 Watch for it**

All meetings (except September) will be held at :  
 The First Unitarian Universalist Church of Winnipeg, 603  
 Wellington Crescent

All meetings are 7 – 9 pm.  
 (First hour for general discussion;  
 second hour for expert guest speaker)

Everyone Welcome Plenty of free parking

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