

### Medical Advisors

Paul Daeninck M.D.  
Medical Oncologist

Darrel Drachenberg  
M.D. Urologist

Arbind Dubey M.D.  
Radiation Oncologist

Piotr Czaykowski M.D.  
Medical Oncologist

*Thanks!*

### *Thought of The Day*

“Every challenge,  
every adversity,  
contains within it  
the seeds of  
opportunity and  
growth.”

- Roy Bennett

### Public meetings cancelled until 2021

#### **Covid Spike in Manitoba Forces Action on Newsletter Mailouts**

Bad news. The current spike in daily covid cases in Manitoba, coupled with the rising trend in such cases, indicates that we probably will not be able to resume our regular activities for a few more months. It now seems likely that this crisis will not end until a vaccine arrives and is used to confer immunity to the general public. Only then will the restrictions on assembly be lifted. This prolonged shutdown has not only prevented us from holding our regular monthly meetings, it has also severely reduced our financial revenues. That is bad news indeed, and we have to take steps to conserve our financial reserves, so that we can resume normal operations when better times arrive.

Our financial reality requires us to take action to reduce our operating costs. Towards that end the board has made the decision that, until such time as circumstances permit, our monthly newsletter will be available only in electronic version with no hardcopy mailouts. This is because the main cost of the

newsletter flows from the need to print and mail the hardcopies, which amounts to the better part of a dollar a copy, while the e-versions are distributed at essentially no cost. The electronic version will continue to be made available on our website (manpros.org).

***To minimize the loss of contact with our newsletter recipients we urge everyone who has access to the internet to migrate away from the hardcopy version to the e-version. Not only will this allow you to continue receiving the newsletter, you will also enjoy the higher quality full color presentation of the e-version.*** To register for the e-version simply send an email to manpros@mts.net, with your name, home address and email address. Watch the website for further developments. Thank you for your patience and understanding. Stay safe.

*The board.*

*Manitoba Prostate Cancer  
Support Group*



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.**

## In Prostate Cancer, ADT After RT Better Than Before RT

For patients with localized prostate cancer who undergo radiotherapy, giving androgen deprivation therapy (ADT) in the adjuvant setting leads to better oncologic outcomes than occur with up-front neoadjuvant ADT, which is how it is usually given.

This new finding comes from an analysis of individual patient data from two large randomized trials, and it could change a clinical practice that has been going on for a decade, if not longer, say experts.

In one of the two trials, ADT was initiated before radiotherapy and was continued during it (neoadjuvant); in the other, ADT was given after radiotherapy or was started during it but not before (adjuvant).

In both settings, the duration of ADT was similar.

"Thus, it's really a question as to whether you should start ADT months before radiation or whether you should start it when you start radiation and continue on afterwards," explained lead author Daniel Spratt, MD, an associate professor of radiation oncology at the University of Michigan, Ann Harbor, Michigan.

"The hypothesis of this study was that sequencing of ADT with radiotherapy independent of ADT duration will have a clinically meaningful impact on oncologic outcomes just as it does in other disease sites," he told Medscape Medical News.

"We found that progression-free survival, biochemical recurrence, distant metastases, and metastases-free survival were all significantly improved with adjuvant compared with neoadjuvant ADT sequenced with prostate-only radiation, and we believe this analysis currently serves as the highest level of evidence to support the importance of

sequencing of ADT with radiotherapy," he concluded.

Spratt presented the new data at the virtual annual meeting of the American Society for Radiation Oncology (ASTRO).

This is a "startling" finding and one that challenges the way physicians have combined ADT and radiotherapy over the past decade or even longer, commented Alison Tree, MBBS, MD, consultant clinical oncologist, the Royal Marsden Hospital and the Institute of Cancer Research in London, the United Kingdom. She was not involved in the study and was approached for comment.

"In a large group of patients, all recruited to high-quality randomized trials and with long follow-up, starting ADT with radiotherapy significantly improved meaningful outcomes for patients, compared to starting ADT months before radiotherapy," she told Medscape Medical News in an email.

This benefit was achieved at no cost, Tree added. Side effect rates appear to be no different from those when starting ADT earlier.

**This finding has the potential to make a big difference to many men receiving curative treatment for prostate cancer. *Dr Alison Tree***

"This finding has the potential to make a big difference to many men receiving curative treatment for prostate cancer," Tree underscored.

### Lowers Testosterone

Explaining the rationale behind the study, Spratt, who is also chair of the genitourinary clinical research program at the University of Michigan's Rogel

Cancer Center, noted that ADT systemically lowers testosterone to starve cancer cells. "If you have two different therapies, one being hormone therapy and the other radiation, it probably doesn't matter if you use them together or one before the other or one after the other, because each are just giving their independent effects if they are simply additive," he said.

However, if the effect of the two therapies is synergistic — which, for example, is how chemotherapy works in tandem with radiotherapy in other cancers — then sequencing should matter.

Spratt explained that when radiotherapy is given for patients with prostate cancer, it upregulates the androgen receptor, which is what testosterone binds to and which is the driver of prostate cancer.

"The androgen receptor controls a lot of the DNA repair genes that fix the radiation damage, so when you radiate prostate cancer cells, it upregulates the androgen receptor, and then that receptor tells it to repair that radiation damage," Spratt noted.

However, radiation damage can continue long after the radiotherapy itself has been completed.

"So by keeping the androgen receptor inhibited or suppressed by hormone therapy, you can suppress that DNA repair mechanism for months, and this is why I think adjuvant ADT is a very important component to kill prostate cancer cells," Spratt told Medscape Medical News.

Spratt added, however, that some men have very aggressive locally advanced tumors. For these men,

*(Continued on page 3)*

(Continued from page 2)

hormone therapy can shrink the prostate, making treatment safer.

In this setting, neoadjuvant ADT may be intentionally given not for any oncologic benefit but to achieve cytoreduction of the tumor and, potentially, to alleviate symptoms if the tumor is pushing on the rectum or bladder.

This point was emphasized by Marc Garnick, MD, professor of medicine at Beth Israel Deaconess Medical Center and Harvard Medical School, Boston, Massachusetts, who was approached for comment. The whole intent of giving neoadjuvant therapy for prostate cancer has been to shrink the tumor, allowing radiation oncologists to deliver appropriate doses of radiotherapy to a smaller gland, he explained.



"There were also some data to suggest that androgen deprivation actually enhanced the sensitivity of radiation therapy's anticancer activity," he said.

However, the authors of this new analysis are suggesting that there may be some independent effects of radiotherapy that could allow ADT given afterward to be even more effective than it would be in the neoadjuvant setting.

"The study certainly should serve as a basis for a formal randomized study to prospectively establish the superiority of this sequence," Garrick added. If proven to be the case, "this would be practice changing," he emphasized.

However, there will be patients for whom physicians might want to initiate ADT immediately, so each case would

have to be individualized, he added.

### Details of the Combined Analysis

For their study, Spratt and colleagues carried out the first combined individual patient analysis of two phase 3 randomized trials to determine the optimal timing of ADT with radiotherapy for patients with localized prostate cancer. In the neoadjuvant group, ADT was initiated a few months prior to starting radiotherapy, and it was continued during radiotherapy; in the adjuvant group, ADT was given after radiotherapy or was initiated during it.

Data from 1065 patients were analyzed. The neoadjuvant and the adjuvant groups had the same number of

patients. The two cohorts were extremely well matched, Spratt commented: more than 50% of both

groups had Gleason 7 disease; the majority had palpable disease (T2 or T3); and baseline prostate-specific antigen levels were greater than 10 ng/mL in the majority of patients.

The primary endpoint was progression-free survival (PFS); median follow-up was 14.9 years.

For all oncologic outcomes with the exception of prostate cancer-specific survival and overall survival, adjuvant ADT was statistically superior to neoadjuvant ADT, Spratt reported. There was a 25% relative improvement in PFS with adjuvant ADT compared with neoadjuvant ADT, which translated into a 7% absolute improvement at 15 years' follow-up.

"Biochemical recurrence was significantly lower with adjuvant ADT, with a 37% relative improvement over

neoadjuvant ADT and a 10% absolute improvement again at 15 years," Spratt observed.

The cumulative incidence of distant metastases was also significantly lower with adjuvant ADT, with a 40% relative reduction compared with neoadjuvant ADT and a 6% absolute reduction at the same follow-up point.

Although the difference in prostate cancer-specific mortality between the adjuvant and neoadjuvant ADT groups did not reach statistical significance, it was numerically lower in the adjuvant group, at a 29% relative reduction at 15 years' follow-up, Spratt noted. The difference in overall survival between the adjuvant and the neoadjuvant ADT groups was also not significant.

"These improvements were accomplished without an increase in late grade 3 to 5 genitourinary and gastrointestinal toxicity," he observed, "and the cumulative incidence of late-grade toxicity was low regardless of ADT sequencing."

Spratt concluded: "We demonstrate for the first time that sequencing of ADT with radiotherapy significantly impacts long-term oncologic outcomes in localized prostate cancer, favoring an adjuvant rather than neoadjuvant-based approach, without increasing late toxicity."

*Spratt has disclosed no relevant financial relationships. Garnick is editor-in-chief of Harvard Prostate Knowledge and Harvard Medical School's Annual Report on Prostate Diseases.*

*American Society for Radiation Oncology (ASTRO) 2020 Annual Meeting: Abstract 32, presented October 24, 2020.*

Pam Harrison October 30, 2020

Source: <https://www.medscape.com/viewarticle/940049>

• • •

## Toward a New Staging System for Prostate Cancer, and Why it Matters

The U-M-led development and validation of a staging system for non-metastatic prostate cancer could help doctors and patients assess treatment options, as well as improve clinical trials.

Doctors and biostatisticians at the University of Michigan Rogel Cancer Center have led the development and validation of a staging system to better predict outcomes and inform treatment decisions for men diagnosed with non-metastatic prostate cancer.

Although it is one of the most common cancers worldwide, prostate cancer remains one of the few major cancers for which the familiar, numerical staging system — ranging from stage 1 to stage 4 — has not been adopted into national guidelines for treatment or for the testing of new medicines in clinical trials.

The new proposed system — dubbed STAR-CAP — which appears in *JAMA Oncology*, draws on patient, tumor and outcomes data from nearly 20,000 patients from 55 centers in the U.S., Canada and Europe to create a robust model with strong prognostic power.

“Localized prostate cancer is sometimes less aggressive, sometimes more — and whether we’re patients, physicians or researchers, we all want to know as best we can how aggressive a particular cancer is likely to be,” says study co-first author Robert Dess, M.D., an assistant professor of radiation oncology at Michigan Medicine. “That information helps with our conversations with patients, it helps with clinical trial design and it is particularly valuable when you can make those estimates based off of standard information that you would collect when you first see a patient to discuss their treatment options.”

The system assigns patients to a particular stage through a point system based on several key variables. These include the patient’s age, tumor category, Gleason grade of cell abnormality and prostate-specific antigen levels, also known as PSA levels. And STAR-CAP uses more granularity in these categories than many of the previous models, the authors note.

**"This is the kind of information that can give patients and doctors more confidence when discussing treatment options and expected outcomes"**  
*Robert Dess, M.D.*

The model divides patients into nine stages of non-metastatic prostate cancer based on their point score — from stage 1 to stage 3, with each stage split into substages of A, B and C.

STAR-CAP’s predictions outperformed or equaled previous, non-validated models, including the current American Joint Committee on Cancer staging system, the study notes. And for a significant number of patients, the new model would reclassify them as having less advanced disease — 22% of patients, for example, who would be classified as stage 3A under the AJCC’s 8th edition criteria would be classified as stage 1C using the STAR-CAP system, a downgrade of four classification steps.

“This is the kind of information that can give patients and doctors more confidence when discussing treatment options and expected outcomes,” Dess says.

Several years ago, the AJCC

established criteria to evaluate prediction models for the staging of prostate cancer — however, since no models met the criteria, the most recent staging designations were based on the consensus of experts in the field, says study co-senior author Daniel Spratt, M.D., the Laurie Snow Endowed Research Professor of Radiation Oncology at Michigan Medicine.

“None of the previous models evaluated met the criteria, so none of them could be used,” Spratt says. “So we said, ‘Well, let’s make one.’ We wanted it to be transparent, robust and validated, so that we can start moving closer to communicate using a common staging system, similar to other cancers. Right now we primarily categorize people as low risk, intermediate risk or high risk — which is a fairly blunt and imprecise system.”

Moreover, the new scoring system is designed to be able to be used worldwide with information that is commonly gathered about a patient and their cancer.

“We’re leveraging a backbone of more than three decades of research,” Dess says. “And we wanted to do it in a formal way and provide the best validated prognostic system we could come up with that was simple, easy to use, and that relied on readily available information.”

The team has made the scoring system available to doctors and researcher worldwide via a web-based app at STAR-CAP.org.

“We know that some of the newest tools that we have that are just coming online like genomics or molecular imaging may improve upon

*(Continued on page 5)*

(Continued from page 4)

this system, but we wanted to create the best, most widely accessible model based on the data we currently have — understanding that new tools may help us develop even better models in the future,” Dess says.

Both Dess and Spratt stressed that the effort would not have been possible without co-first author Krithika Suresh, Ph.D., a former biostatistics graduate student, and co-senior author Matthew Schipper, Ph.D., a research professor of biostatistics at the School of Public Health and research associate professor of radiation oncology at Michigan Medicine, who led the work’s complex statistical analyses. Elizabeth Chase, a doctoral candidate in biostatistics was also instrumental, helping to design and develop the online web application, they said.

Nor would the work have been possible without the participation of numerous national and international collaborators.

“This work doesn’t get done unless you

have the collaborative spirit of investigators across the country and around world,” Dess adds.

The research was supported by grants to Spratt from the Prostate Cancer Foundation, the National Institutes of Health (CA186786, CA240991-05, CA231219) and generous philanthropic gifts from patients.

Additional authors include: Michael J. Zelefsky of the Memorial Sloan Kettering Cancer Center; Stephen J Freedland of the Samuel Oschin Comprehensive Cancer Institute; Brandon A. Mahal of Harvard University; Matthew R. Cooperberg and Peter R. Carroll of the Helen Diller Family Comprehensive Cancer Center; Felix Y Feng of the Helen Diller Family Comprehensive Cancer Center and University of California, San Francisco; Brian J. Davis, Bradley J. Stish and Thomas M. Pisansky, Vidit Sharma and R. Jeffrey Karnes of the Mayo Clinic; Eric M. Horwitz of the Fox Chase Cancer Center; Martha K. Terris of the Medical College of Georgia; Christopher L. Amling of Oregon Health and Science University; William J. Aronson of the University of California, Los Angeles; Christopher J. Kane of the University of California, San Diego;

William C. Jackson, Jason W. D. Hearn, Yilun Sun, Rohit Mehra, Samuel D. Kaffenberger and Todd M. Morgan of U-M; Curtiland Deville, Theodore L. DeWeese, Stephen Greco, Todd R. McNutt, Daniel Y. Song and Phuoc T. Tran of Johns Hopkins University; Paul L. Nguyen of the Dana-Farber Cancer Institute; Nicholas G. Zaorsky of the Penn State Cancer Institute; Fabio Ynoe Moraes of Queen’s University, Ontario, Canada; Alejandro Berlin and Antonio Finelli of Princess Margaret Cancer Centre and the University of Toronto, Ontario, Canada; Nicola Fossati, Giorgio Gandaglia and Alberto Briganti of the University Vita-Salute San Raffaele Hospital, Milan, Italy; Michael W. Kattan of the Cleveland Clinic Foundation.

*Paper cited: “Development and Validation of a Clinical Prognostic State Group System for Nonmetastatic Prostate Cancer Using Disease-Specific Mortality Results from the International Staging Collaboration for Cancer of the Prostate,” JAMA Oncology. DOI: 10.1001/jamaoncol.2020.4922*

Source: <https://labblog.uofmhealth.org/lab-report/toward-a-new-staging-system-for-prostate-cancer-and-why-it-matters>

• • •

## Newly Identified Biomarker Linked to Metastatic Prostate Cancer Development

Researchers from Rutgers University have identified human gene markers that lead to the development of metastatic prostate cancer or cancer that spreads past the prostate.

Prostate cancer is the second leading cause of cancer-related death in men in the United States, and metastatic prostate cancer has a five-year survival rate of 30%.

Exploring prostate cancer cells in both humans and mice, the Rutgers investigators found a connection between 16 genes that lead to metastasis development, which can introduce treatment challenges. These gene markers may be able to predict if a patient has a high probability of developing metastasis.

The biomarkers were initially discovered via analyses of bone metastasis on mice,

revealing distinct molecular profiles tied to patterns of subclonal branching from the primary tumor. Integrating those data from both mouse and human datasets with functional studies in vivo confirmed a co-activation signature among these genetic markers that was associated with prostate cancer metastasis.

“People diagnosed with prostate cancer should now be screened for the protein markers discovered to help determine their risk of developing metastatic prostate cancer, which can help inform more personalized therapy,” coauthor Antonina Mitrofanova, PhD, research member at Rutgers Cancer Institute, said in a press release. “Our results show that molecular profiling at the time of diagnosis can help inform more personalized therapy, leading to better outcomes for those with this advanced form of disease.”

The team went on further to identify a gene signature in humans with prognostic value for time to metastasis and predictive of treatment response in patients undergoing androgen receptor therapy, commonly used to treat metastatic disease. The researchers are hopeful that the biomarker will be able to decrease multiple treatment rounds for patients by identifying early who is at risk for treatment failure.

*The study was published in Nature Cancer.*

By Rebecca Araujo October 29, 2020

Source: <https://www.docwirenews.com/condition-center/precision-medicine-in-prostate-cancer-precision-medicine-in-prostate-cancer-picks/newly-identified-biomarker-linked-to-metastatic-prostate-cancer-development/>

• • •

## Advanced Prostate Cancer Has An Unexpected Weakness That Can Be Targeted By Drugs

### Researchers identified that SUCLA2-deficient prostate cancer cells can be selectively treated with thymoquinone

#### Summary:

Researchers reported that the SUCLA2 gene is frequently involved in the deletion of the tumor suppressor gene RB1 in advanced prostate cancer. RB1 deletion makes cells resistant to hormone therapy but SUCLA2 deletion induces a metabolic weakness. The study showed that thymoquinone selectively killed SUCLA2-deficient prostate cancer cells in vitro and in vivo. The findings highlight a vulnerability of advanced prostate cancer cells that can be targeted by drugs.

The compound thymoquinone (TQ) selectively kills prostate cancer cells at advanced stages, according to a new study published in

Oncogene. Led by researchers at Kanazawa University, the study reports that prostate cancer cells with a deletion of the SUCLA2 gene can be therapeutically targeted. SUCLA2-deficient prostate cancers represent a significant fraction of those resistant to hormone therapy or metastatic, and a new therapeutic option for this disease would have immense benefits for patients.

Hormone therapy is often chosen for the treatment of metastatic prostate cancer but nearly half of patients develop resistance to the treatment in as little as 2 years. A mutation in RB1, a tumor suppressor gene that keeps cell growth under control, has been pegged as a particularly strong driver of treatment resistance and predicts poor outcome in patients.

"Mutations in tumor suppressor genes are enough to induce initiation and malignant progression of prostate cancer, but so far we haven't been able to directly target these mutations with drugs to treat prostate cancer," says the lead author Susumu Kohno. "We wanted to find a genetic aberration associated with that of a tumor suppressor gene which we could target therapeutically."



In the genome, SUCLA2 neighbors RB1. An analysis of prostate cancer cells showed that cells with a RB1 deletion were also missing SUCLA2, pairing up the SUCLA2 deletion with the RB1 deletion present in advanced stage prostate cancer. Kohno and colleagues analyzed prostate cancer tissue and found that 11% of cases were missing both SUCLA2 and RB1.

The researchers screened compounds to identify drugs that would selectively kill cells with a SUCLA2 deletion. Out of around 2,000 compounds, TQ emerged as a hit compound. TQ already has known anti-cancer effects and was shown to be safe in a phase I clinical trial. Kohno and colleagues applied the TQ treatment to a mouse model of SUCLA2-deficient prostate cancer and TQ selectively suppressed tumor growth.

"These findings show that TQ treatment could be an effective therapy for treating prostate cancer cells that harbor SUCLA2 deficiency" says the senior author Chiaki Takahashi.

In a search of genetic databases from patients with prostate cancer, the researchers found that the frequency of SUCLA2 loss was almost perfectly aligned with RB1 loss at every disease stage -- meaning the SUCLA2 deletion could identify people with prostate cancer needing advanced therapy.

Finding this drug-targetable vulnerability opens a crack in the barrier of treatment resistance for prostate cancer. More work needs to be done to improve efficacy of TQ and identify patients that would benefit from this

type of treatment, but the compound provides a promising route for new treatment options for advanced prostate cancer.

#### Story Source:

Materials provided by Kanazawa University. Note: Content may be edited for style and length.

#### Journal Reference:

Susumu Kohno, Paing Linn, Naoko Nagatani, Yoshihiro Watanabe, Sharad Kumar, Tomoyoshi Soga, Chiaki Takahashi. Pharmacologically targetable vulnerability in prostate cancer carrying RB1-SUCLA2 deletion. *Oncogene*, 2020; 39 (34): 5690 DOI: 10.1038/s41388-020-1381-6

October 7, 2020 Kanazawa University

Source: <https://www.sciencedaily.com/releases/2020/10/201007123053.htm>

• • •

## Fluciclovine PET/CT Improves Prostate Cancer Salvage Radiation Outcomes

Use of fluciclovine (18F) positron emission tomography/computed tomography (PET/CT) in addition to conventional imaging to guide radiation therapy for men with recurrent prostate cancer (PCa) following radical prostatectomy is associated with improved outcomes compared with conventional imaging alone, investigators reported during the American Society for Radiation Oncology (ASTRO) virtual annual meeting.

The finding is from the EMPIRE-1 (Emory Molecular Prostate Imaging for Radiotherapy Enhancement) trial, the first randomized trial comparing how radiotherapy decisions guided by the 2 modalities affect outcomes among men with post-prostatectomy PCa recurrence. Investigators randomly assigned 165 patients to receive external beam radiation therapy (EBRT) based on conventional imaging (bone scan plus CT or magnetic resonance imaging [MRI] of the abdomen and pelvis) or conventional imaging plus fluciclovine PET/CT.

To be enrolled in the study, patients needed to have detectable PSA following radical prostatectomy, negative bone scans, and no extra-pelvic metastases found on CT or MRI scans of the abdomen or pelvis. The treatment arms were balanced with respect to age, race, PSA level, androgen deprivation therapy use, and pathologic characteristics.

The median follow-up duration was about 2.5 years overall and 3.0 years for the failure-free patients. A total of 125 patients had a minimum follow-up duration of 3 years. The 3-year failure-free survival rate, the study's primary endpoint, was significantly greater in the PET/CT group than in the control



group (75.5% vs 63.0%), co-principal investigator Ashesh B. Jani, MD, of the Winship Cancer Institute at Emory University School of Medicine in Atlanta, Georgia, reported in a late-breaking abstract session. The 4-year failure-free survival rate also was significantly greater in the PET/CT group (75.5% vs 51.2%). On multivariable analysis, patients in arm 2 were twice as likely to have failure-free survival compared with controls.

“What this research has found is that integrating advanced molecular imaging into the treatment planning process allows us to do a better job of selecting patients for radiation therapy, guiding radiation treatment decisions and planning and ultimately, keeping patients’ cancer under control,” Dr Jani said in an ASTRO press release.

Radiation treatment decisions for the

PET/CT group were determined strictly by PET. Patients with extra-pelvic fluciclovine uptake received no radiation, whereas those with pelvic uptake received radiation to the pelvis and prostate bed, according to the investigators. Uptake only at the prostate bed led to radiation treatment directed at that site. Radiation also was directed to the prostate bed when no uptake was detected anywhere.

Toxicity was similar in both study arms, suggesting the PET-

guided treatment was well tolerated, the investigators reported.

### Reference

Jani A, Schreibmann E, Goyal S, et al. Initial report of a randomized trial comparing conventional- vs conventional plus fluciclovine (18F) PET/CT imaging-guided post-prostatectomy radiotherapy for prostate cancer. Presented at: ASTRO 2020 virtual annual meeting, October 23-29, 2020. Abstract LBA 1.

By Jody A. Charnow

Source: <https://www.renalandurologynews.com/home/news/urology/prostate-cancer/fluciclovine-positron-emission-tomography-ct-improves-salvage-radiotherapy/>

• • •

**MANITOBA PROSTATE CANCER SUPPORT GROUP TAX DEDUCTIBLE DONATION**

NAME: \_\_\_\_\_  
 ADDRESS: \_\_\_\_\_ POSTAL CODE \_\_\_\_\_  
 THIS GIFT IS IN MEMORY/HONOUR OF \_\_\_\_\_ PLEASE SEND NOTIFICATION TO: \_\_\_\_\_  
 NAME: \_\_\_\_\_  
 ADDRESS: \_\_\_\_\_ POSTAL CODE \_\_\_\_\_

**Make payment to:** Manitoba Prostate Cancer Support Group;  
 Box 315 – 971 Corydon Ave., Winnipeg, Manitoba, R3M 3S7

\*A tax deductible receipt will be issued. Charity number: 88907 1882 RR0001

**Credit Card** donations can be made by going to our website at: [www.manpros.org](http://www.manpros.org) and clicking on the donate tab.  
 Canada Helps will issue a tax receipt. **Amount:** \$25 \$50 \$75 \$100 Other \_\_\_\_\_

Gold Wing Road Riders Association  
 Manitoba District - Region K  
<http://mb-a-regionk.ca/>

☆☆☆☆☆☆☆☆☆☆  
 ☆ Thank-you to ☆  
 ☆ all our ☆  
 ☆ sponsors ☆  
 ☆☆☆☆☆☆☆☆☆☆

MANITOBA COMMUNITY SERVICES COUNCIL INC.

**Email - [manpros@mts.net](mailto:manpros@mts.net) ALL MEMBER INFORMATION IS KEPT CONFIDENTIAL**  
 Answering Machine - (204) 989-3433 **Help us lower our costs :**  
**Receive this newsletter by email ~ Please notify us and we'll make the changes. Thank-you**

**MPCSG BOARD**

Betty O'Grodnik – Secretary .....	(204) 661-8549
Jos Borsa - Chair .....	(204) 219-7726
Liz Feschuk - Special Projects .....	(204) 654-3898
Ernie Schade – Meeting Convener .....	(204) 489-1648
Pat Feschuk – Special Events .....	(204) 654-3898
John O'Grodnik - Vice Chair .....	(204) 661-8549
Wally Jackson - Member-at-large .....	(204) 668-1222
Deloris Ankrom - Member-at-large .....	(204) 667-4156
Don Murray - Member-at-large .....	(204) 487-0822

**Volunteers On Committees**

Irek Iskat — membership

*For general information please contact Jos Borsa at number listed above*

**Our public meetings will not  
 resume until the covid-19  
 restrictions are lifted.**

**Watch this space for information  
 on the latest status.**



This newsletter is a  
**Bottom Line Computer Services**  
 publication

Bottom Line Computer Services is not responsible for content  
**[www.misterpete.com](http://www.misterpete.com)**