PROSTATE CANCER ACTION GROUP (S.A.) INC

Affiliated with
Prostate Cancer Foundation of
Australia

ABN 26 499 349 142

NEWSLETTER

FIND US AT www.pcagsa.org.au

APRIL 2006

Chairman's Report April 2006

My report for this month is very brief due to being Interstate on holidays. As a result I did not attend several activities that took place during the month and these will be reported on by Trevor Hunt.

Awareness Evenings

Blackwood

As previously advised the presentation will take place on Wednesday 10th May at the Blackwood Over 50's Club – 4 Young Street, Blackwood commencing at 7 p.m.

The key speaker for the Evening will be Dr Peter Sutherland, Head of Urology at the Royal Adelaide Hospital. Dr Graham Lyons, a Researcher with University of Adelaide, will also give a presentation. In addition two survivors of prostate cancer from our Group will share their experiences.

Previous mention had been made of a possible DVD recording of the two main speakers at the Evening, to be arranged by the Prostate Cancer Foundation of Australia, but this will not be taking place.

Registrations for the Evening can be made with the Blackwood Hospital on 8278 0400. This is a free presentation and all are welcome to attend.

Distribution of flyers promoting the Evening have commenced and details are listed on our website at www.pcagsa.org.au

A Grant from the City of Mitcham has enabled our Group to conduct this Event.

Clare

No further developments have taken place in regard to this presentation which will take place on the 21st August 2006.

Mitcham Prostate Cancer Support Group

The attendance at the meeting held on the 23rd March was 15 with several regular members absent. The speaker was Ken Hancock, a member of the Mitcham Group and he gave an excellent presentation that was well received by members.

The next meeting will be on Thursday 27th April when the guest speaker will be John Mayes, Vice President and Research Librarian for the Adelaide Support Group. John will highlight some ways men can assist themselves with diet and lifestyle using himself as an example.

For more information contact Jeff Roberts on 8277 3424 or visit the link on www.psaadelaide.org

Jeff Roberts

THE GOOD OIL ON PROSTATES

A preparation containing an olive-oil extract suppresses the growth of prostate cancer cells and induces those cells to self-destruct, a new study has shown.

In a laboratory, the dietary supplement Zyflamend has been found to reduce prostate cancer cell growth by as much as 78% and even can induce cancer cell death, or "apoptosis", scientists have reported in the journal of Nutrition and Cancer. *(Sydney Morning Herald, 23/2, health & Science, p3)*

FRED GOES TO SEMAPHORE

On 19th March, Trevor, Coralie, Ian and Rob, accompanied by Fred B. A. Man conducted our information stall at the 3rd Man Alive! event at Semaphore. The weather was very kind to us, with a beautiful blue sky with a few fluffy "cotton wool" clouds, and a very gentle breeze. One wondered whether it could get any better, and why would you want to be anywhere else?

This event has been conducted for 3 consecutive years, and our Group has participated in each of those years. The 2006 event continued the previous trend of improvement, and this year's event had better crowds, better entertainment, and a better all-round response. It worth noting that this year's event was fully self-funded, but that this may not be possible next year. Surely, the results of this year's event should convince potential sponsors or government departments that it is worth supporting this event. After all, how many men's health events really do get any support from the State government?

Another feature of the 2006 event was that, through judicious manipulation of the entertainment programme, the organizers were able to hold a good crowd throughout the afternoon. As a result, it is believed that our Group handled more enquiries than at any previous Man Alive! event. As from this event, we have introduced a "score sheet" to enable a more accurate record of genuine, valid enquiries and distribution of literature. This year, we had 92 such enquiries – i.e., 1 genuine enquiry every 4 minutes of the event, plus contact with many men and/or partners where prostate cancer has already been diagnosed.

In addition, we found new opportunities for networking, further opportunities for public speaking engagements, and were able to observe ideas for fund-raising, and, possibly, another contact to be followed up for co-operation with the Prostate Cancer Foundation of Australia.

In all, a very worthwhile day, which illustrated that our presence was appreciated by the public, and the organizers of the event. Should we go again? Was it worth our effort? Are we providing a needed service? The answer to all questions is a resounding "YES".

VIRUS MAY HAVE LINKS TO PROSTATE CANCER

A new virus has been identified in human prostate tumors, but the virus's link to prostate cancer is unclear and requires more research, researchers say.

"This is a virus that has never been seen in humans before. This is consistent with previous epidemiologic and genetic research that has suggested that prostate cancer may result from chronic inflammation, perhaps as a response to infection," researcher Dr. Eric Klein, head of urologic oncology at the Cleveland Clinic's Glickman Urologic Institute, said in a prepared statement.

The virus, which is closely related to viruses found in mice, has never before been detected in humans. Researchers at the University of California, San Francisco, and the Cleveland Clinic found it using the same DNA-hunting "virus chip" used three years ago to confirm the identity of the SARS virus.

The finding was reported Friday at a prostate cancer symposium co-sponsored by four organizations: the American Society of Clinical Oncology, the American Society for Therapeutic Radiology and Oncology, the Prostate Cancer Foundation, and the Society of Urologic Oncology. A full report will be published in the journal /PloS Pathogens/.

The virus was found more often in human prostate tumors with two copies of the RNASEL gene mutations than in tumors with at least one normal copy of the gene. RNASEL is a gene that serves as an important defense against viruses. Scientists have previously speculated that a virus may be involved in some types of prostate cancer in men with mutated RNASEL genes.

Klein and his colleagues say the finding further validates the use of the virus chip to discover previously unknown viruses, and to learn more about viral causes of disease. "The power of the virus chip resides in its ability to simultaneously screen for all viruses, without preconceptions or bias. In the case of these prostate tissues, no one would have suspected a virus of this class," UCSF researcher Joe DeRisi, who developed the chip, said in a prepared statement.

SOURCE: University of California, San Francisco, news release, Feb. 24, 2006

The need for a multidisciplinary care approach toward prostate cancer that can improve both clinical outcomes and quality of life has never been more important. Advances in both diagnosis and treatment have resulted in men with prostate cancer living longer with the disease, making dialogue between and among urologists, radiation oncologists and medical oncologists ever more critical to the implementation of individualized management strategies.

For the second consecutive year, the Prostate Cancer Foundation (PCF) has teamed up with three professional medical societies for the 2006 Prostate Cancer Symposium to bring together key thought leaders in each of the different subspecialty areas. In February 2006, nearly 1,500 practicing physicians, biopharmaceutical executives and government policy makers joined the PCF, the American Society of Clinical Oncology (ASCO), the American Society of Therapeutic Radiology and Oncology (ASTRO) and the Society of Urologic Oncology (SUO) for three days of discussions on issues critical to the care of men with prostate cancer.

Finding the Optimal Treatment Approach in Clinical Practice

A recurring theme at the meeting was the need to individualize treatment for men at all stages of disease. Thankfully, additional and innovative treatment approaches are being studied daily, enabling men to find the treatment approach that's right for them.

New research demonstrates that men with early-stage disease have more choices than ever to cure the disease. Data from Massachusetts General Hospital in Boston show that equivalent and excellent outcomes can be seen with both brachytherapy radiation "seeds" and external beam radiation therapy, while data from the University of Toronto show that active surveillance encompassing periodic PSA (prostate-specific antigen) tests and biopsies can be an effective option for men at low risk for progression.

In men with more advanced disease, the need to optimize quality of life is often taken into account when selecting a therapeutic approach; new research demonstrates that there is a broadening array of options for men at this stage of disease. Data from the large ASCENT trial show that "intermittent" chemotherapy-a strategy that allows men to take short breaks in the chemotherapy cycles-can provide some relief from the intense regimen without compromising clinical outcomes, while data from the large TAX-327 trial show that the use of docetaxel chemotherapy can provide effective symptomatic relief in addition to improving clinical outcomes.

Yet, as many presenters and discussants at the meeting reminded the participants, it is only through the coordinated efforts of urologists, radiation oncologists, medical oncologists and other health care providers that these important advances in research centers can be implemented in the clinic to help men with prostate cancer live longer and better lives.

Debating the Best Practices in Prevention, Diagnosis and Treatment

The advent of new preventive, diagnostic and therapeutic strategies has greatly contributed to the ability of men with prostate cancer to live longer and better lives. But it remains unclear how best to implement some of these strategies in clinical practice, as evidenced by the debates at the meeting on these and other important prostate cancer management issues.

Results from the landmark Prostate Cancer Prevention Trial (PCPT) released in 2003 showed that preventing prostate cancer with finasteride might be feasible - but potentially at the risk of promoting the growth of more aggressive cancers in a small percentage of men. Similarly, data obtained from the mandatory end-oft-study biopsies in PCPT showed that prostate cancer can he found even in men with low PSA levels, prompting the question of whether the threshold for "normal" levels of PSA should be lowered so as to more easily identify men with earl-stage disease.

In men with progressing disease, the issue of when and how to initiate additional therapy has been debated since the early 1990s. Here, too, most studies show mixed results, and the question of the benefits outweighing the risks must be addressed. In some men, earlier initiation of hormone therapy can delay the onset of metastatic disease, but the loss of testosterone can significantly affect quality of life. Similarly, earlier implementation of chemotherapeutic strategies might provide more effective tumor control in some men, but the rarity of effective second- and third-line therapies that might be necessary should the cancer continue to progress makes some physicians reluctant to initiate early chemotherapy.

Although no clear consensus was reached in debating these issues, the participants strongly advocated for continued research on these important topics; for greater enrollment in clinical trials by men at all stages of disease; for coordinated care among specialists in both academic medical centers and community practices; and, most importantly, for an individualized approach toward the prevention, diagnosis and management of prostate cancer.

TREATMENT "HOLIDAYS" EFFECTIVE IN HORMONE-REFRACTORY PROSTATE CANCER

According to results presented at the 2006 prostate Cancer symposium in San Francisco, a treatment strategy involving cessation of chemotherapy until disease progression achieves cancer control in men with hormone-refractory prostate cancer.

Chemotherapy is standard treatment for HRPC. However, patients are not able to tolerate continuous, ongoing chemotherapy. In order to find a tolerable regimen, researchers evaluated intermittent administration of chemotherapy, called chemotherapy 'holidays'; with this schedule, patients take a break from treatment and resume at a specified point when their disease starts to progress.

Researchers from Oregon Health and Science University recently conducted a study to evaluate several therapy holidays in men with HRPC who were undergoing treatment with chemotherapy. This study included a subgroup of 45 patients involved in a larger clinical trial referred to as the ASCENT trial in which treatment with Taxoteree (docetaxel) plus Calcitriol (high-dose vitamin D) was compared to Taxotere plus placebo (inactive substitute).

The 45 men who underwent treatment holidays had achieved a prostate specific antigen (PSA) level of no greater than 4 ng/ml and had no detectable progression of disease while on chemotherapy. These patients then opted for no treatment (holiday) until their PSA levels increased by 50% or increased by 2 ng/ml at which point they resumed treatment with chemotherapy.

The following results were achieved after chemotherapy resumed following a treatment holiday:

* Half of the patients achieved a 50% decline in their PSA.
* PSA levels were stabilized for at least 3 months in 35% of patients.
* Disease progression occurred in 15% of patients.
* The median duration of the first holiday was nearly 17 weeks.
* Even after several treatment holidays, chemotherapy continued to produce anticancer activity.

The researchers concluded that treatment holidays provide relief as well as effective therapy in patients with HRPC undergoing treatment with chemotherapy. Further study is necessary to determine precisely which patients benefit from chemotherapy holidays.

Reference: Beer, TM, et al. Intermittent Chemotherapy In Metastatic Androgen-Independent Prostate Cancer (Ai pc): Initial Results From ASCENT. proceedings from the 2006 Prostate Cancer Symposium. Abstract #139.produced by cancerConsultants.com <http://www.cancerconsultants.com/>

PROSTATE TEST TO GET "EASIER"

An Australian company is confident of developing a much more accurate laboratory test to diagnose prostate cancer, based on semen rather than blood, within a year.

Proteome Systems Ltd.'s chief executive Stephen Porges said he expected the non-invasive test to be cheaper and more effective than diagnostic methods already in use.

The test measures levels of a cancer marker known as Human Carcinoma Antigen in semen. (The Advertiser, 17/3, p24)

THE UNPALATABLE TRUTH

Leslie Michaelson does not have prostate cancer, but, as chief executive of the Prostate Cancer Foundation in the US, he knows all too well how bad the disease is. So he changed his diet.

Michaelson is one of a growing number of people who are turning to diets for protection from cancer. Cancer patients, doctors say, almost always ask what to eat to reduce their chances of dying. Yet scientists say they really do not know whether dietary changes make a difference. It is turning out to be much more difficult than anyone expected to discover if diet affects cancer risk. Most people want some sort of control, a way to prevent the disease from ever striking them or, if it does, to keep it from recurring. Many think of diet as a strategy. *(Sydney Morn. Herald, 23/2, Health & Science,p9)*

IS IT SAFE TO EAT ANYTHING?

A compound formed when meat is charred at high temperatures – such as barbecues – encourages the growth of prostate cancer in rats, researchers say.

The study, presented to the American Association for Cancer Research yesterday, may help explain the link between eating meat and a higher risk of prostate cancer. The compound called PhIP is formed when met is charred, Dr. Angelo De Marzo of Johns Hopkins University in Baltimore reported. *(The Advertiser, 4/4, p27*)

SURGEON EXPERIENCE AFFECTS OUTCOMES FOLLOWING PROSTATECTOMY

According to results recently presented at the 2006 Prostate Cancer Symposium in San Francisco, patients with prostate cancer have improved results when their prostatectomy is performed by surgeons who have performed a larger number of prostatectomies compared to surgeons who have performed fewer such procedures.

Results from some clinical studies have indicated that surgeons who are experienced in specific surgical procedures may provide their patients with better outcomes than surgeons who are less experienced in the procedure. Researchers from Memorial Sloan-Kettering Cancer Center conducted a clinical trial to evaluate outcomes of prostatectomies and surgeon experience with the procedure.

This study included 7,849 men with early prostate cancer who underwent a prostatectomy between 1987 and 2003 at one of the following four cancer centers: Cleveland Clinic, Wayne State University, Baylor college of Medicine, or Memorial Sloan-Kettering Cancer Center. None of the patients had received chemotherapy, hormone therapy, or radiation therapy prior to surgery. This study included 74 surgeons.

Patients had better outcomes if their prostatectomy was performed by a surgeon experienced in the procedure:

*At 5 years, progression-free survival was 88% for patients whose surgeons had performed 250 or more prostatectomies.

*At 5 years, progression-free survival was 79% for patients whose surgeons had done only 10 prostatectomies.

*The rate of patients experiencing a rise in prostate-specific antigen (PSA) levels following surgery increased significantly among patients whose surgeons had performed fewer prostatectomies.

The researchers concluded that patients who undergo a prostatectomy by a surgeon who has performed a greater number of these procedures have improved outcomes compared to patients whose surgeon has performed fewer procedures. They stated that surgeons who performed 250 or more prostatectomies produced the best outcomes.

Men with early prostate cancer who are considering a prostatectomy may wish to ask their physician who will be referring them to a surgeon or their surgeon about the number of prostatectomies he or she has performed.

Reference: Bianco, FJ, et al. Outcomes Measurement Influence of the Surgeon on Cancer Control After Radical prostatectomy. proceedings from the 2006 Prostate Cancer Symposium. Abstract 272. _- produced by cancerConsultants.com <http://www.cancerconsultants.com/> copyright _2005 CancerConsultants.com <http://www.cancerconsultants.com/>. All Rights Reserved.

STUDIES REVIEWING TOMATO, LYCOPENE AND THE INCIDENCE OF PROSTATE CANCER

Dietary factors are considered an important risk factor for the development of prostate cancer in addition to age, genetic predisposition, environmental factors, and other lifestyle factors such as smoking. Some studies have found a lower incidence of prostate cancer in populations that consume large amounts of tomatoes and tomato products; thus, consuming tomatoes and tomato products may decrease the risk for developing prostate cancer. Lycopene, one of over 600 carotenoids, is one of the main carotenoids found in human plasma. It is responsible for the red pigment found in tomatoes and other foods (guava, red grapefruit, and watermelon) and is absorbed well into the human body. Lycopene is a natural pigment synthesized by plants and microorganisms but not animals. It is one of the most potent antioxidants, with a singlet-oxygen-quenching ability twice as high as that of beta-carotene and 10 times higher than that of alpha-tocopherol (2).

Studies based either on (a) dietary/dietary supplement intake or (b) blood/tissue measurements of carotenoids have been the primary methods to determine lycopene's role in lowering the risk of developing or enhancing the growth of prostate cancer cells. The following studies provide an historic review of lycopene and prostate health as well as offering a look at more recent studies.

Dietary intake studies

An important study examining specific dietary and lifestyle habits and the risk of developing prostate cancer was published by Mills et al. in1989. In the six-year study involving 14,000 men, only tomato, bean, lentil and pea consumption were related to lowered prostate cancer risk.

Intake of beta-carotene-rich foods, such as carrots, was unrelated to risk (3). In a large, comprehensive study at the Harvard School of Medicine involving 47,894 prostate cancer-free male health professionals, dietary intake of various carotenoids was assessed using a detailed questionnaire (4). Of all of the carotenoids (including beta-carotene), only high lycopene consumption had a statistically significant 21 percent risk reduction. Of the 46 food items that contained carotenoids, three of the four significantly associated with a lower risk of prostate cancer contained lycopene -- tomato sauce, tomatoes and pizza. Those subjects that consumed more than 10 servings of tomatoes and tomato-based products per week -- accounting for an estimated 82 percent lycopene intake -- had a 35 percent reduced risk of prostate cancer compared to those that consumed fewer than 1.5 servings per week. For more advanced or aggressive prostate cancers, which are more likely to cause death, the apparent protective effects of lycopene were even higher (47 percent). Of all the food items analyzed, tomato sauce provided the maximum protection (66 percent).

A population-based, case-control study carried out in Auckland, New Zealand during 1996-1997 also investigated associations between prostate cancer risk and dietary intake of the carotenoids beta-carotene and lycopene and their major plant food sources, including carrots, green leafy vegetables, and tomato-based foods. The study reviewed 317 prostate cancer cases and 480 controls. The authors found that dietary intake of beta-carotene and its main vegetable sources was largely unassociated with prostate cancer risk, whereas intake of lycopene and tomato-based foods was weakly associated with a reduced risk. These results suggest that in contrast to the findings regarding many types of cancers, vegetables rich in beta-carotene are not protective against prostate cancer. However, lycopene from tomato-based foods was found to be associated with a small reduction in risk (5).

Researchers also examined the protective effect of vegetables, fruits, and legumes against prostate cancer in a multicenter case-control study of 1619 African-American, white, Japanese, and Chinese men with histologically confirmed cases of prostate cancer and 1618 controls. Intake of legumes, yellow-orange vegetables, and cruciferous vegetables were inversely associated with prostate cancer. However, in this study, intake of tomatoes and fruits was not related to risk (6),

A recent study suggests dietary supplementation with lycopene in a pill may decrease the growth of prostate cancer (7). In the study, 26 men with newly-diagnosed, clinically-localized prostate cancer were randomly assigned to receive 15 mg of lycopene (n=15) two times a day or no supplementation (n=11) for three weeks before a radical prostatectomy. Researchers found that lycopene supplementation may increase prostate tissue levels of lycopene, positively affect biomarkers of growth and differentiation, and decrease clinical signs for aggressiveness of the prostate cancer. These findings suggest that lycopene may have a role in the prevention of prostate cancer.

Blood and Tissue Measurement Studies

Lycopene has been shown to concentrate in prostate tissues (8). Among the carotenoids present in the prostate gland, lycopene levels appeared to be highest. Thus, it has been hypothesized that lycopene may lower the risk of prostate cancer. Studies at the University of Toronto found that prostate cancer patients have lower serum and prostate tissue lycopene levels compared to control subjects (9). In a cell culture study, lycopene, when combined with Vitamin E, prevented the growth of prostate cancer cells (10). These data provide further evidence that increased consumption of tomato products and other foods containing lycopene could lower the risk of prostate cancer. **

One recent study evaluated how prostate levels of antioxidants relate to plasma levels and self-reported usual dietary intake (11). Levels were measured in 47 men undergoing radical prostatectomy or transurethral prostatectomy at Loyola University Medical Center in Chicago. The levels of tocopherols and carotenoids in the prostate were significantly correlated with plasma levels; the strongest correlations were associated with lycopene, beta-carotene, and gamma-tocopherol. The researchers note that this finding supports their potential to provide better estimates of internal dose, and thus target organ exposure, than reported intake.

In a study conducted on 65 patients with prostate cancer and 132 cancer-free controls, significant inverse relationships with prostate cancer were observed with plasma concentrations of lycopene. An 83% reduction of prostate cancer risk was observed in the group with the highest plasma concentration of lycopene in comparison with individuals with the lowest concentration. The strength of the association was directly related to dose; with increasing concentrations of plasma lycopene, the risk of prostate cancer was decreased (12).

Studies suggest that lycopene from various tomato products is indeed associated with the lowered risk of several types of cancers (13). This further supports the current dietary recommendations to increase consumption of fruits and vegetables, including tomatoes and tomato products, as part of a healthy diet to possibly reduce the risk of prostate cancer. Additional studies are needed to continue to investigate the relationship between dietary lycopene, levels of lycopene in human tissues, and the prevention and treatment of prostate cancer. In particular, more large-scale studies must be conducted before any definitive conclusions can be made.

References

1. American Cancer Society, Cancer Facts & Figures 2001: 5; 16-17

2. Sanjiv A and Rao, AV. Tomato lycopene and its role in human health and chronic diseases. Canadian Medical Association Journal, Volume 163(6):739-744, September 19, 2000

3. Mills PK, Beeson WL, Phillips RL, Fraser GE. Cohort study of diet, lifestyle and prostate cancer in Adventist men. Cancer 1989; 64:598-604

4. Giovannucci E, Ascherio A, Rimm EB, Stampfer MJ, Colditz GA, Willett WC. Intake of carotenoids and retinol in relation to risk of prostate cancer. Journal of the National Cancer Institute 1995; 87:1767-1776

5. Norrish, Alan E.; Jackson, Rodney T.; Sharpe, Susan J.; Skeaff, C. Murray, Prostate Cancer and Dietary Carotenoids. American Journal of Epidemiology. 151(2):119-123, January 15, 2000

6. Kolonel LN, Hankin JH, Whittemore AS, Wu AH, Gallagher RP, Wilkens LR, John EM, Howe GR, Dreon DM, West DW, and Paffenbarger, Jr. RS. Vegetables, Fruits, Legumes, and Prostate Cancer: A Multiethnic Case Control Study, Cancer Epidemiology & Prevention Vol. 9, 795-804, August 2000

*7. *Kucuk O, Sarkar FH, Sakr W, Djuric Z, Pollak MN, Khachik F, Li Y, Banerjee M, Grignon D, Bertram JS, Crissman JD, Pontes EJ, Wood, Jr. DP. *Cancer Epidemiology Biomarkers & Prevention Vol. 10, 861-868, August 2001*

8. Clinton SK, Emenhiser C, Schwartz SJ, Bostwick DG, Williams AW, Moore BJ, Erdman JW Jr. Cis-trans lycopene isomers, carotenoids, and retinol in the human prostate. Cancer Epidemiology, Biomarkers & Prevention 1996; 5:823-833

9. Rao AV, Fleshner N, Agarwal S. Serum and tissue lycopene and biomarkers of oxidation in prostate cancer patients: a case-control study. Nutrition and Cancer 1999; 32:159-164

10. Pastori M, Pfander H, Boscoboinik D, Azzi A. Lycopene in association with alpha-tocopherol inhibits at physiological concentrations proliferation of prostate carcinoma cells. Biochemical and Biophysical Research Communications 1998; 250:582-585

11. Freeman VL, Mohsen M, Yong S, Pyle J, Wan Y, Arvizu-Durazo R, Liao Y. Prostatic Levels of Tocopherols, Carotenoids, and Retinol in Relation to Plasma Levels and Self-Reported Usual Dietary Intake, Am. J. Epidemiology, Vol 151(2):109-118, January 15, 2000

*12. *Lu Q-Y, Hung J-C, Heber D, Go VLW, Reuter VE, Cordon-Cardo C, Scher HI, Marshall JR, and Zhang Z-F. *Cancer Epidemiology Biomarkers & Prevention Vol. 10, 749-756, July 2001*

13. Giovannucci E. Tomatoes, tomato-based products, lycopene and cancer: review of the epidemiological literature. Journal of the National Cancer Institute 1999; 91:317-331

Find weeds out cancerous stem cells

Dan Box 27febO6

AUSTRALIAN scientists have overcome a hurdle in the evolution of embryonic stem cell technology by devising a means of weeding out cells that are potentially cancerous. The research, published in the March edition of Nature magazine, represents a breakthrough for the technology, whose critics have used this instability to argue stem cell research be halted.

While the use of human embryonic stem cells, which can develop into any human cell, may offer great opportunities in medicine, the research has proved controversial with some arguing it is morally unacceptable.

A spokeswoman for the Australian Stem Cell Centre in Victoria, Michelle Singe, said the discovery would allow scientists to identify which stem cells were beginning to develop the abnormalities that preceded the cell becoming cancerous. "It doesn't mean that we have control, but it means that we have a big piece of the puzzle," Ms Singe said. "At least now we can see the cell is changing and we can ask what it is in the culture or the environment causing that change.

"The reason that it's so significant is the people who are opposed to embryonic stem cell research often quote the fact that scientists have no control over stem cells when they develop and a lot of them develop tumours."

Led by the centre's Professor Martin Pera, the research team also believes the discovery may allow abnormal cells to be identified and then purged, maintaining the health of stem cell cultures. The scientists, including researchers from Monash Institute of Medical Research and Monash University, have identified how a cellular marker known as CD30 -- present on all embryonic stem cells -- changes when those cells develop abnormalities.

The research comes after scientists from around the world met in Cambridge to agree on a set of international guidelines to ensure the ethical practice of stem cell technology. This was the first time researchers from the 14 countries engaged in the research had met to establish guidelines by which they agreed to abide.

In the past, international co-operation has been hindered by conflicting national rules.*(The Australian, 25/3/06)*

BLOOD TEST MAY SPOT AGGRESSIVE PROSTATE CANCER

A reliable screen would help doctors match treatments to individual patients
**By E.J. Mundell*/HealthDay Reporter/*

Ideally, the type of prostate cancer a man has -- aggressive or slow-growing -- should guide decisions as to whether he needs intensive treatment or simply "watchful waiting."

Trouble is, there's currently no quick, reliable means of gauging just how deadly a particular prostate malignancy might be. That's why researchers are excited about new findings for a blood protein "biomarker" called MDM2.

In a preliminary study, relatively high blood levels of MDM2 were associated with three distinct signs of aggressive prostate cancer. If the findings are replicated in a larger trial, an MDM2-based test might someday become a routine part of patient care, experts say.

"To be able to say which tumors are likely to progress and become problematic vs. those that are much more indolent or slow-growing would be very helpful in allowing a patient to make a very important decision: whether he wants active treatment or not," explained Dr. Durado Brooks, director of prostate and colorectal cancer at the American Cancer Society.

Brooks believes MDM2 "has a lot of potential" as a viable prognostic indicator, but he added that more study is needed. The findings were reported Monday at the American Society for Therapeutic Radiology and Oncology annual meeting in Denver.

In their study, researchers at the Fox Chase Cancer Center in Philadelphia tracked the outcomes of 469 prostate-cancer patients for an average of nearly six years. At the same time, they routinely tested for levels of MDM2 and other biomarkers in the men's blood. All of the men received standard treatments such as radiation and short- or long-term hormone-deprivation therapy.

The researchers looked at the relationship between blood biomarkers and three signs of aggressive prostate cancer: biochemical failure (rising PSA levels), the spread of malignancy to sites outside the prostate, and death.

While other markers were correlated with one or two of these outcomes, only high blood levels of MDM2 were linked to all three, the researchers reported. Men with high concentrations of MDM2 were much more likely to display aggressive, metastatic disease than men with low levels.

Besides helping to spot those patients needing aggressive therapy, a good prognostic blood test could also spare men with less-threatening, slower-growing tumors troublesome treatments.

"For example, if the cancer is very slow-growing and a man has other medical problems or has an anticipated lifespan of less than seven or eight years, it's sometimes not worthwhile to operate or provide radiation for that prostate cancer," Brooks said.

Lead researcher Dr. Alan Pollack noted that any test that could reliably determine prostate cancer type would also aid research. "We can learn about which types of cancers are anticipated to respond to [specific] treatments, for what reason," he said.

Pollack, who is chief of radiation oncology at Fox Chase, said MDM2 is also being investigated as a potential therapeutic target. "MDM2 interferes with p53, a very important molecule in regulating cancer cell death in response to radiation and perhaps hormone therapy," he explained. "We want cancer cells to die and MDM2 interferes with that-- if MDM2 is high, then the cells seem to be protected from radiation and hormone therapy."

Already, he said, drug companies are developing compounds that may thwart the proteins' attempt to shield cancer cells from destruction. "The results are very promising that by knocking out MDM2 you might be able to improve responses to radiation and hormone therapy," Pollack said.

In the meantime, work on MDM2 as a prognostic indicator continues, with a larger trial the obvious next step, according to Brooks. "It has to be looked at in a larger pool of men with various stages of prostate cancer," he said.

SOURCES: Durado Brooks, M.D., M.P.H., director, prostate and colorectal cancers, American Cancer Society; Alan Pollack, M.D., Ph.D, chairman, radiation oncology, Fox Chase Cancer Center, Philadelphia; Oct. 17, 2005, presentation, American Society for Therapeutic Radiology and Oncology annual meeting, Denver

MY HORMONE TREATMENT – A CASE HISTORY

Eight years ago, in the New Year of 1995/1996, I retired with a pension from Volvo. I saw a lovely time ahead of me, where I could please myself and enjoy life however I wished. But that was not the way it turned out.

At the same time, I got the word that I had prostate cancer of an aggressive type (PSA=150), which had already spread itself in secondary growths around my body. This obviously came as a great shock both for me and my wife Barbro. Our bright gleaming future together suddenly disintegrated before our eyes. We struggled to the conclusion that this was a disease that we had to deal with together and I have had great support from Barbro throughout this difficult time.

A total ignorance of what prostate cancer was, and how it was supposed to be treated, found us at a loss and with no idea of how we should deal with the situation Gradually, the situation became clearer and we came to accept that salvation was not going to come by way of an operation, or radiation, given that the cancer had spread outside the gland.

We repeatedly returned to the question of hormone treatment. Hormone treatment, which is aimed at stopping the male sex hormone testosterone. This can be done in two ways. You either remove the testicles, where the testosterone is generated, by a surgical operation; or you can do it medically by inducing the pituitary gland to send signals to the testicles to stop producing testosterone. Both methods are seen as being equally valid but at the time(eight years ago), surgical castration was regarded as the safer option. Today, the decision may have been different. Surgical castration is irreversible whilst the medical option can be terminated.

Going under the knife was actually fairly straightforward and without any complications. It took place in a clinic with multi-disciplinary functions. The treatment was completed with an anti-androgen (nilutamide– 'Anandron') which, amongst other things, was meant to block a testosterone like substance which is produced by the adrenal glands.

And what was the outcome of all this treatment, all this shifting from pillar to post? The PSA (Prostrate Specific Antigen) - value plummeted and, on examination, my prostate gland was soft and rubbery to the touch. The PSA-value lay at between 1 and 2 for a lengthy period and the doctor stopped my anti-androgen medication. Nothing changed after this. The value stayed the same. Then, without warning, my worst fears came to reality. Throughout 2002 the PSA rose disturbingly from 1.4 to 3.7. Different cancer cells react differently to hormone treatment and there is a great risk that you can become hormone resistant after several years of treatment. I was convinced that this was the case. I was started on another anti-androgen (bicalutamide – 'Casodex') and, once again, I responded well. Today, the PSA-value is down at 0.03 and I am now, after more than eight years feeling great.

I have had body scans on three occasions, in the intervening years which have shown that my secondary growths remain, but are now inactive. There have of course been side effects from the treatment. These began with swelling and hot flushes which started almost immediately. My chest started to swell and became extremely tender. No more playing with the grandchildren and hugging them, or letting them bounce on your chest. It was just too painful. Sadly, I had no idea that it was possible, in the pre-treatment stage, to have the chest treated with some short periods of radiation to guard against this. This was tried later but to no avail.

I became impotent and my libido just disappeared. It is so important to discuss the implications of this with your partner. Barbro and I did just that. After a long and great life together, we managed to work out alternatives so that we could continue to enjoy our life together.

I became incontinent, but this has meant nothing worse than having to wear a small bag (which I get free from the local clinic). I can move about at will and get on with things as normal. I can play golf, for example, but 18 holes is a long and roundabout way to the toilet! I think it is important to engage with those around you and tell them about the disease you have – with relatives, friends, workmates, in a natural way. That prostate gland under your belt does not have to be a taboo subject. But be careful and diplomatic in the way you deliver the news. There are those who are diplomatically challenged – like the young; our grandchildren for example. With them, what you see and hear, is what you get.

We have a summer house on Orust. It is a small house where, amongst other things, we have to shower in an outhouse. There I was with my grandson, Jesper, who was then 4½. It was a beautiful summer's day. The sun shone and all was good with the world. We splashed and sprayed water on each other and our laughter echoed around the surrounding hills. I had not one thought about the time bomb ticking away in my body. Life was great. That old saying "grandchildren are life's dessert menu" really held true in those moments.

Then, suddenly, he stands up, looks at me and says: "Granddad, what a huge big chest you have."

The sun was devoured by a big black cloud, and reality set in. In the time it takes to utter a word, the joyful atmosphere had vanished. Now, out of the blue, I was faced with having to be much more careful, shower alone and otherwise always wear a T-shirt. Things were different. Time to face it. But then after a pause he carried on:

"But, do you know what? 'Birds' have much bigger ones."

And then came the punch line that floored me. "And their ones hang down!!!!"

Suddenly there were birds everywhere. The sun came out again over Orust and we carried on drenching each other and our laughter filled the skies.

http://www.prostateline.com/prostatelinepat/10250_13073_8_5_0.aspx

SCANDINAVIAN STUDY SHOWS PROSTATECTOMY PATIENTS DO BETTER, LIVE LONGER

When men with curable prostate cancer disease that has not spread widely beyond the prostate undergo radical prostatectomy, they are much less likely to have the cancer come back, and much less likely to die of the disease than men who don't have surgery.

This has been illustrated dramatically by a large Scandinavian trial published three years ago, and by a follow-up report, published recently in the New England Journal of Medicine. The results of both publications have rocked the way prostate cancer is perceived in Sweden, Finland, and Iceland, where the mainstay of treatment traditionally has been watchful waiting and where, sadly, most men with prostate cancer in those countries eventually die of it. In the first report, nearly 700 men were randomly assigned either to radical prostatectomy or to watchful waiting. The results provided the first concrete evidence of something American doctors had known anecdotally for years - that treating localized disease reduces deaths from prostate cancer. During the average follow-up of six years, twice as many men in the watchful waiting group died of prostate cancer, which meant, the scientists concluded, that radical prostatectomy can reduce prostate cancer deaths by about half. That study brought hope that treatment can make a difference, and the elated scientists anticipated that with a longer follow-up, the differences in cancer deaths between these two groups would become even more clear.

They were right. At 10 years after the study began (the results published in the second paper), half of the men in the watchful waiting group had died from prostate cancer. Radical prostatectomy reduced the likelihood of dying from prostate cancer by 40 percent. And the overall survival (including all causes) was significantly better in the men who underwent radical prostatectomy. Surgery was of greatest benefit to men who were younger than age 65 at the time their cancer was diagnosed. In that age group, after 10 years, 19 percent of the watchful waiting patients had died of prostate cancer, but fewer than 9 percent of the men who underwent surgery had died. Also, surgery reduced the risk of local recurrence of cancer by 67%, and of the cancer's spread to distant sites by 40 percent. "The impact on distant metastases all the more impressive here," notes Patrick C. Walsh, M.D., University Distinguished Service Professor of Urology, "because hormonal therapy was given more often to the men in the watchful waiting group than to the men who underwent radical prostatectomy." The study's authors concluded: "We expect the benefits of this surgery will increase during longer periods of follow-up."

One important note about this study: Most, 75 percent, of the Scandinavian men were diagnosed with cancer advanced enough to be felt during a physical exam, with an average PSA of 13 ng/ml. This is in sharp contrast to the United States today, where 75 percent of men are diagnosed, on average, five years earlier, and at a much more curable stage, with non-palpable cancer, detected because of a change in PSA.

However, says Walsh, "Although these men had more advanced disease than we commonly see today in the United States, they are very similar to the men who underwent surgery in the early 1990s, before the wide-spread use of PSA screening."

In 1992, 104,000 men underwent a radical prostatectomy in the United States, Walsh continues. "If we apply the outcome from the recent Scandinavian trial to these figures, we would expect that there would be at least 5,000 fewer men dying of prostate cancer 10 years later, which is close to what we have experienced." In applying the findings of the Scandinavian study to today's patients, who are diagnosed with smaller cancers, detected much earlier, the authors note that it may take much longer to see the difference in survival and quality of life, "but the removal of small tumors may facilitate surgery and result in fewer side effects."

Just before the New England Journal Of Medicine study was published, an investigation by researchers at the University of Connecticut and McGill University in Canada appeared in the Journal of the American Medical Association. The article made headlines with its authors' conclusion that their findings do "not support aggressive treatment for localized low-grade prostate cancer." However, the JAMA study's patient population was limited in several ways: First, 60 percent of the patients were diagnosed with low-grade tumors found during transurethral resection of the prostate, a treatment for benign prostate enlargement. "Today these low-grade (Gleason 2-4) tumors are rare," notes Walsh, "because with the availability of medical therapy, fewer men are undergoing surgery for an enlarged prostate. I haven't operated on a patient with Gleason 2-4 disease in the last 10 years. What the authors' data supported, and what they should have stated in their conclusion, was that men with Gleason scores greater than 4 - the vast majority of all men diagnosed today - have a significant risk of dying from prostate cancer, and may benefit from treatment. Also, this paper did not accurately describe the natural history of untreated prostate cancer, because 42 percent of the patients received hormonal therapy within six months of diagnosis. And finally, because many of the study's patients also had serious, chronic health problems when the paper was written, only 6 percent of the patients in the study were still alive, and most had died from other causes. The results aren't helpful to an otherwise healthy man trying to decide on the best course of treatment for cancer."

<http://www.usnews.com/usnews/health/best-hospitals/rankings/specihqurol.htm>

Watching the PBS waistline

*The Treasurer has runaway drug subsidy costs in his sights, and medicines makers are worried. Health editor **Adam Cresswell** reports*

A CLOUD is hanging over the minds of Australia's drug company bosses - a long white one. Radical proposals to drive down the cost of Australia's $6 billion a year Pharmaceutical Benefits Scheme by incorporating elements of the New Zealand system have got the industry up in arms.

Drug makers claim the consequences of such a move could make things worse, not better, for patients - by making market conditions uneconomic and ensuring some more expensive medicines used by small patient groups never reach these shores.

But it seems some senior members of the federal Government suspect this to be grandstanding, and will not be put off. Ministers are convinced there are savings to be had - and this time they could flow both to patients and the Government.

Federal Cabinet met this week in part to consider a range of reform proposals for the PBS - although it's understood no agreement was reached at that meeting. But the stalemate has not killed off the debate. Just three days ago Treasurer Peter Costello was making the case for reform once again, saying further changes had to be made.

His comments banged the cost-cutting drum noticeably more loudly than other remarks made the same day in Sydney by Health Minister Tony Abbott, who said as long as extra spending was judged cost-effective, "we can be confident that cost increases in the future will have more than commensurate health outcomes".

Meanwhile, drug industry insiders warn that any attempt to copy the New Zealand system too directly will kill off innovation here and drive jobs overseas.

The stakes in this game are high.

The PBS pays subsidies for expensive medicines, meaning patients fork out less than $30 for drugs sometimes costing up to $5000 for a single supply The scheme cost taxpayers comfortably less than $3 billion in 1997-8; in 2004-5 it cost a fraction over $6 billion, after a decade of frightening growth rates that often made double digits and once hit 22 per cent, in 2000-01.
Last year the federal Government made a number of changes - including increasing the co-payments, or out-of-pocket charges patients have to pay at the pharmacist's counter, by 21 per cent for both general patients (up to $28.60) and concession-card holders (who then paid $4.60).

The effect was immediate: two million fewer prescriptions were filled in the first three months of 2005. Growth rates for the year were also effectively crimped.

On January 1 this year the co-payments were increased again, in line with normal indexation - up 3.2 per cent to $29.50 for general patients, and up 2.2 per cent to $4.70 for concessional patients.

Then there was the policy introduced later last year, whereby the Government announced it would slice 12.5 per cent from the price it would pay for drugs in each "therapeutic class" of medicines that worked in a similar way, once the patent of any of them expired and a generic manufacturer brought a copycat version to market.

These policies have already brought growth rates down from their previously heady heights, but the Treasurer evidently wants more. Costello told the National Press Club in Canberra the cost of the PBS was rising faster than the economy, and that was "not fully sustainable". And he fingered generic medicines as likely to offer the biggest savings.

"We have to see if we are getting the best price for off-patent pharmaceuticals and whether we can release to the community therapeutic pharmaceuticals, which have the same effect and the same safety at a cheaper price," he said. "Cheaper to the consumer, cheaper to the taxpayer."

The bit about cheaper for the consumer is a new departure: previous PBS-trimming attempts have only been about saving money for the Government, not the punters who collect the medicines from the chemist.

That's one of the reasons why the uptake of generics is relatively low in Australia: according to a federal parliamentary briefing paper, generic medicines accounted for about 25 per cent of the PSS in 2001-02, less than half the rates in some other countries such as Britain.

Although doctors are free to indicate on a prescription form that a pharmacist can dispense a generic product, patients often prefer the brand name. Sometimes that's because the branded drug is the one they are used to, and might be confused by a switch.

But another possible explanation is that even though there is no difference in efficacy compared to a generic, the patient might simply believe that the branded drug is "better" or doing them more good - an advantage which, after all, is the reason why manufacturers invest in brand names in the first place.

But even when patients do agree to a generic, under Australia's existing PBS arrangements it doesn't save the taxpayer nearly as much money compared to choosing a brand name drug as it does overseas, because the price the PBS pays is not enormously different

Writing in *The Australian* in January, Queensland Libera1 MP Andrew Laming, a former doctor, said the drug-purchasing system was flawed and as a result "we pay $800 million too much for plain-label medications; each year".

He said two generics companies, Alphapharm and Sigma Arrow, controlled 86 per cent of the generics market and "get top dollar from the PBS" because unlike brand-name manufacturers they did not have huge research and development costs to recoup. Laming claimed they handed "30 per cent to 70 per cent to pharmacists as loyalty discounts".

In New Zealand, companies are asked to tender to supply a drug to the government-funded scheme; the company that offers the lowest price gets the deal, in exchange for exclusivity.", which often means just one drug available for each condition or disease.

A carbon-copy of the New Zealand Pharmac scheme is considered a political impossibility in Australia, as it would amputate too many of the 2600-plus medicines funded under the PBS, which is *also* highly popular and has been going for over 50 years. But some form of tendering is being seriously looked at; Laming describes it as "the common denominator" of reform proposals.

Under one plan, whenever a brand name drug's patent expires - the point at which generic manufacturers are able to start offering copycat versions - a tendering process would begin to find the generic maker willing to provide it at the lowest price.

While the more expensive brand name versions would still be available, the PBS would share the savings with patients, who would be encouraged to ask their doctor and pharmacist for the generic by a dramatically lower co-payment (which could be cut by as much as half, saving around $15 a pop for general patients).

The generic maker might even be given an exclusivity deal that would exclude other generics for a given period, thereby guaranteeing the winning drug higher market share.

At the moment, the price the PBS pays for all other drugs that act in the same way (so-called "therapeutic classes") also falls by 12.5 per cent when a generic comes onto the scheme. Under this new plan, prices of the other drugs might be dragged down too, or this price link might be cut.

But even if the other drugs' prices were not affected, it's likely their makers would still suffer due to loss of market share.

As might be expected, Australia's brand-name manufacturers are horrified by any of these ideas. Their umbrella body, Medicines Australia, is arguing for another plan that would offset price cuts with price increases for brand name drugs.

While the PBS would pay 20 per cent less for generic drugs, the link to the similar brand name drugs would be cut and prices for them allowed to drift up by the same proportion. Overall, this would save slightly over $1 billion over five years.

Rather more surprisingly, the generics industry body, the Generic Medicines Industry Association, also

opposes tendering, describing it as a "disaster for Australian consumers".

GMIA's president. John Montgomery, says such a scheme would prove a red carpet to third-world generics manufacturers, who would have "nothing to lose by offering to supply a drug to the Government at an impossibly tow price".

"Australian companies that invest in the economy in terms of R&D, infrastructure, capital and employment costs, can in no way compete with these offshore companies," Montgomery says.

"Prices would be so low that product margins would be insufficient to continue the investment needed for a manufacturing operation. This is the situation in New Zealand, from where a large number of companies have withdrawn. What could happen in Australia is what's happened in New Zealand: local manufacturing could cease, the jobs of Australians could go offshore. and R&D could be wound back.'" *(The Australian, 4/3)*

Fatty acids help fight prostate cancer

Eating foods rich in omega-3 fatty acids such as oily fish may help to prevent the spread of prostate cancer, research has revealed.

Omega-3 and omega-6 are essential fatty acids that work together to promote good health. The body cannot make them, so eating a diet rich in the substances is important. Fish and certain oils such as canola and flaxseed are sources of omega-3 while raw nuts and seeds contain omega-.6.

In laboratory studies, scientists at the Paterson Institute at the Christie Hospital in Manchester, England found that omega-3 fats could block the spread of cancerous cells.

Dr Mick Brown, chief scientist in the research group, said the results of the research published in the British Journal of Cancer suggest a diet rich in omega-3 fatty acids might help to inhibit the spread of the disease in men with early prostate cancer. Because omega-3 and omega-6 work together, it is important to maintain a balance of the two for good health.

"Omega-6 fats, found in vegetable oils, nuts and seeds, increased the spread of tumor cells into bone marrow. This invasion was blocked by omega 3 fats - the ones found in oily fish," Brown said in a statement. "It is possible to have a healthy balance of these two types of fat - we only need about half as much omega-3 as omega-6 - that will still stop cancer cells from spreading," he added.

The researchers believe omega-3 fatty acids interfere with functions of omega-6, which cancer cells may use as a source of energy, and prevent them from spreading beyond the prostate gland.

Prostate cancer is more treatable and has a better survival rate if it is diagnosed and treated in its earliest stages.

"Some tumors develop slowly in the prostate without producing symptoms and sometimes when symptoms do develop, it is because the cancer has already spread.

"Eating a diet with the right balance of omega 3 and omega 6 fats may well help to keep prostate cancer within the prostate gland where it may be monitored safely or more easily treated with surgery or radiotherapy," said Noel Clarke, a co:-author of the study.

Prostate cancer is the third most common cancer in men worldwide, with 543,000 new cases each year, according to the international Agency for Research on Cancer in Lyons, France.

Professor John Toy, medical director of the charity Cancer Research UK, which publishes the journal, said the role of diet in prostate cancer is not fully clear and that more studies were needed. "Cancer Research UK advises people to reduce their risk of cancer by eating a healthy diet, high in fibre, fruit and vegetables and low in red and processed meat," Toy said*. (The Australian, 22/3/06)*

THE AGE • WEDNESDAY, MARCH 22, 2006
theage.com.au

HEALTH

Sack cancer chief: MP

FEDERAL Labor MP Wayne Swan has called for the sacking of the Cancer Council of Australia's chief executive, Professor Alan Coates.

Mr Swan, a prostate cancer survivor, attacked Professor Coates on SBS's *Insight* program last night.

"He ought to be removed from his current position because what he is advocating is his ignorance," Mr Swan said. "His advice against testing will, for some men, lead to an early and painful death. Professor Coates is effectively telling men to stick their head in the sand."

The Age : 19th March 2006 +

News 7

Carleton calm about cancer

TELEVISION current affairs veteran Richard Carleton has a handy knack of overcoming obstacles, whether in his *60 Minutes* duties or with his health, but now he's confronted with a fresh challenge — prostate cancer.

Carleton, 62, was diagnosed with the disease last year, beginning a course of hormone treatment before checking into hospital last week for a common surgical procedure.

Richard Carleton

But the *60 Minutes* stalwart, who has a history of heart problems, including a bypass 18 years ago and a heart attack in 2003, said the latest health scare wasn't any cause for hysteria.

"I'm the fourth person (with it) from *60 Minutes* in nearly 20 years. It's not quite the common cold, but it's not much more special than that," he said.

Carleton's approach to tackling the disease has not included being in denial. "I just do as I'm told (by the doctors)," he said.

ANTHONY STAVRINOS

Board Directors

- 5 Positions; Inaugural Board

The Cancer Council South Australia

The Prostate Cancer Alliance is a joint-venture of The Cancer Council South Australia and ProstateSA. The purpose of this newly formed organisation is to achieve a greater focus on prostate cancer in terms of fundraising for research and programs relevant to all aspects of prostate cancer control.

The formation of the Prostate Cancer Alliance has led to the rare opportunity for the appointment of a brand new Board. This is clearly a very exciting chance for appropriately experienced Directors to make a real difference to the ongoing success of the new organisation and the impact of prostate cancer control in the community.

With the imminent commencement of the new organisation, we are seeking 5 new Board Directors to work in conjunction with the appointed independent Chairman, Mr. Ray Blight and an existing Cancer Council SA Board Member as part of a 7-person Board.

The appointees will give their time voluntarily in providing corporate governance through advocacy, leadership, fundraising activities and influencing prostate cancer control in South Australia. Both associated organisations have enormous community support and key South Australian urologists have assisted in the creation of the amalgamated body through their expertise in treating prostate cancer.

If you are passionate about improving prostate cancer control in the community your Directorship of the new Board will-be rewarded by immense personal and professional satisfaction.

Appointees will display excellent commercial acumen, appropriate experience and the personal qualities to show significant empathy, combined with excellent interpersonal communication skills including the ability to engage with key stakeholders of the new organisation.

***Please forward your application* to *success@stillwellmanagement.com.au quoting Ref. No. 006/1416 in the subject heading. Telephone enquiries* to *Daryl S$tillwell are welcome*. From The Advertiser , 1/4/06, p10**

AVOIDING THE BARBS OF PROSTATE CANCER

By Jill Margo (Australian Financial review, 30/3/06)

In the bush, when you help a mate through a three-strand barbed wire fence, you hold the middle wire down with your foot and lift the top wire so he can climb through the enlarged space.

Don Baumber likes to use this analogy to describe what should happen with prostate cancer. But he says there is no one holding the wires apart. Those who should be are so busy arguing about the risks of being snagged that men are left to squeeze through alone.

Baumber, a businessman who invented the Zero Weeding Wand and sold millions in Australia and overseas, is a prostate-cancer survivor. He now helps others get through the confusion surrounding this disease.

For most men, the confusion begins with the debate about testing. He says the debate is puzzling because opposing sides are at cross-purposes.

"They are talking about different things and in the meantime 2500 Australian men die every year. Every day they spend disagreeing, another seven families bury a loved one, often after years of suffering" he says. ,

Baumber's involvement with prostate cancer is intense. He runs self-help groups, participates on national scientific cancer committees and is engaged in the politics of the disease. He keeps abreast of the latest research and has taught himself to evaluate medical evidence.

The debate over testing is largely between those who study diseases in populations and those who study diseases in individuals. The epidemiologists, who study populations, say they don't have evidence to show that mass-screening for prostate cancer improves survival. They are concerned that if they recommend screening for all men aged over 50, they might worry men unnecessarily and could waste public funds implementing testing and following through with expensive treatments.

Although studies that could provide some evidence will be completed in five years, there will be many more years of professional argument and bureaucratic wrangling before decisions are made.

While similar studies for breast cancer began in the 1960s, those for prostate cancer began only in the late 1990s.

"We are a good 30 years behind in our evidence and it is understandable that the statisticians need to play it safe until they get the facts," Baumber says. "But what the community wants is quite different. It wants the right to early

> Prostate cancer can only, be cured in men whose tumours are localised."

detection of prostate cancer even if the population survival benefit and the economic evidence is not available."

Baumber says Australia is a signatory to international charters on health in which it is an uncontested fact that early detection of cancer leads to better outcomes.

"Men want the right to find their cancer early," he says. "In Australia, they have this individual right but they have to exercise it against a background of negative sentiment based on the lack of evidence for population screening."

But he admits that it is not all simple. If a man takes a test and finds cancer, it is not always easy to tell if the cancer is active and aggressive. It could be so slow-growing that it could do more harm to eradicate it than leave it alone.

But Baumber says prostate cancer can be cured only in men whose tumours are localised and amenable to surgery or radiation. No curative therapy exists after such tumours have metastasised and the way to find localised tumours is through early detection. This means making an individual decision.

He says the value of early detection is highlighted by the comparison between advanced prostate cancer in the United States and the UK. In the US, where there is widespread-testing of men over 50, only I per cent of all new cases of prostate cancer are advanced.

In the UK, where there is a strong policy recommendation against testing, 40 per cent of new cases are advanced.

"It's a no-brainer, he says. "There is no point talking about population screening because it is still too many years out. We need to be talking about individual choice."

When he was diagnosed in 1994, Baumber hadn't even considered testing. At.58, he was visiting the doctor for a sporting injury when he casually mentioned he had a urinary problem and asked for a script for the newly released drug Proscar.

The doctor insisted on examining him and didn't like what he felt. Within a week, Baumber's cancer was confirmed and it was advanced and aggressive.

The choice was simple, he could have surgery or radiation. But he was about to leave on a prolonged business trip and thought of another possibility. . "I asked for hormone therapy to hold the situation while I was away. I think this was the beginning of my taking some control of my condition."

He was given drugs to block his testosterone and seven months later had his prostate removed. Although nervesparing surgery was available, Baumber decided against it because with his .grade and stage of disease, he wanted the cancer removed with the widest margins possible.

Afterwards, his continence returned swiftly but he fell into a sexual void. The months of hormone blockade had drained his libido and the operation had removed his capacity for a spontaneous erection. "Giving up potency didn't worry me

Don Baumber says men should have the right to individual choice Photo: ROBERT ROUGH

at first but a few years later, when 1 became involved with support groups and all the guys were talking about what they were doing to overcome the problem, 1 started to take an interest." he says.

"I tried various things and although injections worked, I gave up because of the lack of spontaneity."

About 10 years ago, when Baumber was approached to train as a support group leader, he was hesitant because of his non-medical background. But he was encouraged to hear that "wounded warriors" usually do it best because for them, it's a matter of practice not theory. He took the challenge.

Cancer spread protein identified

From correspondents in London 30marO6

SCIENTISTS have identified and blocked the action of a protein linked to the spread of breast, prostate and skin cancer cells to the bones.

The molecule called RANKL is produced in bone marrow. In studies of mice, researchers from Austria and Canada showed that inhibiting the protein could stop the cancerous cells from migrating to the bones.

“RANKL is a protein which tells tumour cells to come to it," said Professor Josef Penninger, of the Austrian Academy of Sciences in Vienna. "It sits on the bones and when tumour cells circulate in the body then RANKL attracts them into the bones," he said.

Once a cancer has spread beyond its original site in a process known as metastasis, it becomes much more serious and difficult to treat. An estimated 70 per cent of patients with progressive breast cancer and 84 per cent of advanced prostate cancer sufferers develop bone metastases.

The findings, reported in the journal *Nature,* explain the puzzle of why certain cancers spread to the bones and how interfering with the process could help to prevent the spread of the disease.

When the researchers gave mice with skin cancer a drug that blocked RANKL, the rodents had fewer tumours in their bones than animals that were not treated. But the drug did not slow the spread of the cancer to other sites in the body.

Prof Penninger and his colleagues stressed that the research was done in mice but they added that drugs that interfere with RANKL are in development which could be used test their findings and show it the same holds true for humans.

"This is an idea that can be directly tested," Prof Penninger said.

Nearly all breast cancer tumours in women have the receptor for RANKL which Prof Penninger said is an indirect indication that the findings are relevant to humans.

"Since there are novel inhibitors of RANKL far along in clinical development, the idea is that people who have cancer that is known to spread to bone can start taking this drug when they are diagnosed," said D. Holstead Jones, of the University of Toronto and the lead author of the study.

"It would inhibit how much bone metastases they would have," he said in a statement.

Every year an estimated one million people develop metastases to the bones, particularly women with breast cancer. Drugs that inhibit RANKL may also help to alleviate the severe pain that metastases can trigger and improve the quality of life of patients.

Breast and prostate are among the most common cancers.

More than a million new breast cancer cases occur worldwide each year and half a million men are diagnosed with prostate cancer, according to the International Agency for Research on Cancer in Lyon, France. *(from The Australian, 30/3/06)*

Wonder ingredient not the good oil once touted

Adam Cresswell, Health editor25marO6

THE supposed wonder ingredient fish oil, the much-touted omega 3 fats, may not be so wondrous after all.

Despite long-standing recommendations that Australians eat more fish - largely for the omega 3 fat it contains - a review of medical evidence has failed to show that people with a high omega3 intake live any longer.

Previously, experts had thought consumption of either the "long-chain" omega 3 fatty acids, found mainly in oily fish, or "short-chain" omega 3 found in some plant oils gave protection against heart disease.

But a review of almost 90 studies, published online yesterday by the British Medical Journal, pooled the results and found no proof that eating omega 3 cut deaths overall, or reduced the incidence of heart attacks, strokes or cancer.

Of the 89 individual studies in the review, 48 were the type of trials considered to be the most reliable.

However, even in these the margin of error was wide. Taken together they appeared to show a 2 per cent reduction in the risk of early death, but this could mean a 30 per cent reduction in mortality risk, a 36 per cent increase, or anything in between.

"It is not clear whether long-chain or short-chain omega 3 fats (together or separately) reduce or increase total mortality, cardiovascular events, cancer or strokes," the authors said.

However, they added the findings "do not rule out an important effect of omega 3 fats", because even after combining all the individual trials, there were still too few people who had suffered heart attacks or other adverse outcomes to be sure the variation was not due to chance.

Dr. Tim Crowe, lecturer in nutrition at Victoria's Deakin University, said the findings were "a surprise" but the recommendation to eat fish regularly should stand. "The study has shown no harm (from omega 3), and there's emerging evidence that fish oil may be beneficial particularly in colon and breast cancer - and there's a link with omega 3 and depression as well," he said.

"People shouldn't be changing conservative recommendations to be eating fish at one to two meals per week."

The official Dietary Guidelines for Australian Adults, endorsed by the National Health and Medical Research Council in 2003, said omega 3 fats had "been shown to provide specific health benefits, notably in relation to brain development and function and cardiovascular health".(*The Australian, 25/3/06)*

TESTOSTERONE TREATMENT LINKED WITH PROSTATE CANCER

by Will Boggs, MD | Reuters Health | 08.12.2005

NEW YORK - Prostate cancer developed in 20 men within months to a few years after they began testosterone supplementation to correct a deficiency of the hormone, investigators report.

"There are several anecdotal case reports, small studies, and observational studies like ours which raise concern but do not provide conclusive evidence yet," Dr. Franklin D. Gaylis said.

The issue is a concern because prostate cancer is usually driven by testosterone.

Gaylis, from the University of California at San Diego Medical Center, and colleagues report this series of patients "in whom clinically significant prostate cancer developed and was presumed to be related to exogenous testosterone use," in the Journal of Urology.

The men were identified in six different urology practices. Prostate cancer was detected within 2 years of the start of testosterone replacement in 11 of these men, seven of them within the first year, the authors report. The others were diagnosed after 28 months to 8 years.

Eleven men had normal prostate exams before testosterone supplementation was begun, the report indicates, and the average PSA level of the 17 men tested before treatment was 3, although the range was 0.9 to 15. The threshold for further evaluation is usually 4.

"It is our belief that men, especially those with a family history of prostate cancer, should not receive a prescription for testosterone supplementation without careful, informed consultation regarding the risks and benefits of such treatment," the investigators conclude.

"I would hope that guidelines would be developed by experts in the field to help us appropriately and carefully prescribe testosterone replacement to men who clearly need it and who would benefit from it, and then monitor them for potential adverse outcomes, e.g., the development of prostate cancer," Gaylis said.

While the study has flaws, writes Dr. E. Darracott Vaughan, Jr. from Weill Medical College of Cornell University, New York, in a related editorial, it "can be taken as a 'shot across the bow' for urologists and other physicians. We need to be extremely careful before beginning testosterone therapy."

SOURCE: Journal of Urology, August 2005.

Compiled by Trevor Hunt