PROSTATE CANCER ACTION GROUP (S.A.) INC

Affiliated with
Prostate Cancer Foundation of
Australia

ABN 26 499 349 142

NEWSLETTER

Website – www.pcagsa.org.au

JULY 2005

Chairman's Report – July 2005

Awareness Evenings

Port Augusta

An Information Evening was held at the Yacht Club, Port Augusta on the 7th July. The Evening was promoted by Abbott Australasia through Vesna Jankovic with support from the Corporation of the City of Port Augusta. Approximately 50 people attended which was considered quite a good number as it was apparently a cold, wet night. The speakers included Dr Agnelo De Sousa, Dr Don McQuistan (local GP), Rob Kitto and Bill Toop from our Group. Congratulations to Vesna for organising a successful Event.

Yorke Peninsula

Promotion for the Kadina visit is getting under way with flyers being prepared and forwarded to both the Northern Yorke Peninsula Health Service at Wallaroo and the Lions Club at Bute. Some excellent news – following a Grant application being completed on behalf of the Lions Club of Bute and ourselves, a Grant of $500 has been approved by the Northern Yorke Peninsula Health Service YP SPOKE and these funds will be applied to the costs of the Evening. Press, Community Radio and possibly TV advertising will be arranged shortly.
To refresh everyone's memory the date is the 19th August at the Farm Shed Museum and Tourist Centre – 50 Moonta Road Kadina from 7.00p.m.-9.30p.m. The key speaker is the visiting urologist to the area, Dr Zenon Herzberg. A free Evening for men, women and/or their partners and we are hoping for a good attendance.

Adelaide Metropolitan Area

Our Group will be conducting an Awareness Evening on the 14th September in collaboration with The Cancer Council South Australia who will be funding the presentation. The venue will be the Function Room in the Cancer Council premises – 202 Greenhill Road Eastwood. I had a further meeting with Margaret Ryan from The Cancer Council on the 27th June when distribution of flyers was discussed and speakers confirmed. As mentioned previously Prof. Marshall has agreed to be our key speaker. Presentations will be given by Dr Elizabeth Isenring (Flinders University) on nutrition and Dr Linda Foreman (The Cancer Council South Australia) on the GP Education Program.
2 members of our group will also speak and the final session will be a panel discussion. Everyone is welcome to attend this free Evening with tea/coffee etc provided.
Much of the promotion will be linked with the Prostate Cancer Call-In to be held on the 8th September but some separate advertising will take place. So, put the 14th September in your diary as this will be an interesting and informative Evening.

Clare

Following a discussion Trevor had with a men's health worker based at Clare, consideration will be given for an awareness evening to be conducted at Clare during the first half of 2006.

Grant Applications

Advice was received from the Department of Health and Ageing that our Grant application was unsuccessful. I phoned and enquired if this was due to any particular reason and was told preference was given to support groups. There is

likely to be another round of grants in 2005/06 and I feel we should enquire as to the criteria before submitting an application.

Prostate SA
At our June Meeting some of our members expressed interest in joining the Education and Community Committee of Prostate SA. I advised Amber Doyle (Prof. Tilley's P/A) and she thanked our Group for our interest. We should be contacted shortly on this matter.

Mitcham Support Group Meeting
The 2nd meeting of the Group was held at the Colonel Light gardens RSL Club on Thursday 23rd July. The attendance of 24 was very pleasing. At the Meeting I mentioned the generosity of the RSL Club who allowed free use of their facilities. During the Evening the Vice-President, John O'Leary announced the Group could have free use of the venue for 6 months – very much appreciated.
Gary Bowes (Chairman of the Association of Prostate Cancer Support Groups SA) spoke and mentioned the association would contribute funds to assist the Group being established. Apart from these funds, grant applications will be made to the City of Mitcham and The Cancer Council South Australia.
During the Evening John Francis and Roland Harris agreed to join myself as signatories to a cheque account.
Mention was made of the great assistance received from the City of Mitcham in particular Councillor Bob Marshall, Kerry Hallett – Manager Community Services and Julie Lamond, Communications Officer.
Details of our June Meeting were placed on the City of Mitcham website.
An article will probably appear in the August Edition of the Mitcham Community News.
Reg Mayes and Ian Fisk have promoted the Group in the PSA Newsletter.
The guest speaker for the Evening was Graham Lyons, nutrition researcher at the University of Adelaide. Graham spoke on "Diet and prostate cancer". His talk included types of cancer studies and discussion on anti-cancer foods and supplements. Graham gave his usual excellent presentation which was very well received. Many questions were asked by the audience.
Next Meeting Thursday 28th July. Anyone is welcome to attend. Tea/coffee will be provided.
Guest speaker will be Ian Fisk with a presentation on his brachytherapy treatment.
For more information check our website at www.pcagsa.org.au

Meeting of the Association of Prostate Cancer Support Groups (SA) Inc.
I attended a Meeting held on the 25th June. Among matters discussed were the following:
The receipt by the Association of the Grant from the Department of Health and Ageing (congratulations on this success)
The possible involvement in Prostate SA
The proposed Prostate Tissue Bank

Vietnam Veteran's Day/Men's Health Expo
The Vietnam Veteran's Day Council is organising a Men's Health Expo to take place on the Torrens Parade Ground on 21st August 2005.
Our Group will be manning a booth with a display of pamphlets and we hope many people will stop to have a chat and take plenty of literature. From what I have heard the Event will be well organised and likely to draw a large attendance. It would be great if our Groups could support the Event
Further information will be provided at our August Meeting.

Jeff Roberts

PROSTATE POSER

The belief that men are more likely to die with prostate cancer than from it has been called into question.

Queensland Cancer Fund researchers have produced an age-related risk of death among men diagnosed with prostate cancer. It shows younger men with the disease are more likely to die early than those diagnosed at an older age.
A man aged 50 diagnosed with prostate cancer has a 60% chance of dying prematurely from it before age 80, while a man aged 70 at diagnosis has a 38% chance of early death. Men diagnosed at 60 have a 50% risk of early death. *(Herald Sun 2/5/05, p11)*

STATE GOVERNMENT MAKES AMAZING DISCOVERY

Why are men reluctant to seek medical advice? Why do they often wait until the situation is urgent? Well, it is my considered opinion that there are many men around who could provide some very blunt and frank answers to those questions.

According to a newsletter “The Reynell Report” published by the Labor member for Reynell, Gay Thompson MP, JP, a Men’s Health Taskforce has been set up by the State Government to try to answer these questions and encourage men to think more about their health. She has asked that any person who would like to take part should contact her office.

It appears that men’s health has been finally discovered by this government. I wonder just how much research it took before they came up with this amazing discovery. It couldn’t be that we are heading into an election campaign, could it?

WEBSITES

www.patienthealthinternational.com	Astra Zeneca site for patients. Good interactive learning module
www.nature.com/pcan	Prostate cancer and prostatic diseases Journal of interest to surgeons
www.ejwhittenfoundation.com	E.J. (Ted) Whitten Foundation
www.afud.org/conditions/forwomen	American Foundation for urologic Disease Information on prostate disease to share with the Men in your life
www.aafp.org/afp/20030915/1075.html	American Family Physician. Serum Tumour Markers. Includes PSA testing.
www.osbon.com	Vacuum pump, cryotherapy experiences
www.novartisclinicaltrials.com	Learn about clinical trials
www.patientINFORM.org	Making sense of medical research (under construct.)

PROSTATE CANCER – ANTIOXIDANT BLOOD LEVELS KEY TO MnSOD GENE

Greater levels of selenium, vitamin E and the tomato nutrient lycopene have been shown to reduce prostate cancer in one out of every four Caucasian males - those who inherit a specific genetic variation that's particularly sensitive to oxidative stress.

Conversely, if carriers of this genetic variant have low levels of these vitamins and minerals, their risk of aggressive prostate cancer increases substantially, as great as 10-fold, over their cohorts who maintain higher levels of these nutrients. These results, published in the March 15 issue of the journal Cancer Research, were based on the analysis of 567 men diagnosed with prostate cancer between 1982 and 1995, and 764 cancer-free men from the Physicians Health Study (PHS).

'"This large prospective study provides further evidence that oxidative stress may be one of the important mechanisms for prostate cancer development and progression, and adequate intake of antioxidants, such as selenium, lycopene and vitamin E, may help prevent prostate cancer," said Haojie Li, M.D., Ph.D., a researcher at the Brigham and Women's Hospital and Harvard Medical School.

Destructive molecules known as "free radicals" have been shown to team up with oxygen in the human body resulting in oxidative stress and what some scientists believe is an assortment of age-related ailments. As a result, many believe that consumption of antioxidants can slow that process.

"Our study, as well as many other epidemiological studies, encourages dietary intake of nutrients such as lycopene from tomato products, or supplements for vitamin E and selenium to reduce risk of prostate cancer," said Li. The initial goal of the PHS study was to assess the effect of aspirin and beta carotene on men's health. Since blood samples collected in 1982 were available from many of the study's participants, the research team decided to review variants for the gene that codes for manganese superoxide dismutatase (MnSOD), *an* important enzyme that works as an antioxidant in human cells to defend against disease. The MnSOD gene is passed from parents to offspring in one of three forms: W, VA or AA.

"Compared with men with the MnSOD W or VA genotype, people with the AA genotype seem to be more sensitive to the antioxidant status," said Li. "Men with the AA genotype are more susceptible to prostate cancer if their antioxidant levels are low."

The study's results found that a quarter of the men in the study carried the MnSOD AA genotype, half carried the VA genotype, and the remaining quarter carried the VV genotype. The results indicated that the VA and W men were at equivalent risk for developing prostate cancer across all levels of antioxidants in their blood. Compared to MnSOD W or VA carriers with low selenium - those men in the lowest quartile of the study group - MnSOD AA males had an 89 percent greater risk for developing aggressive prostate cancer if blood levels for selenium were low.

On the other hand, MnSOD AA carriers with high selenium - those men in the highest quartile - had a 65 percent lower risk than the MnSOD W or VA males who maintained low levels of selenium. "The levels of selenium in the highest quartile of these men are not abnormally high," Li said. "Our range is neither extremely high nor extremely low."

While similar trends were observed for lycopene and vitamin E when tested independently, the contrast in relative risk was most

pronounced for the men who had high blood levels for all three antioxidants combined.

"Among men with the MnSOD M genotype, we observed a 10-fold difference in risk for aggressive prostate cancer, when comparing men with high versus low levels of antioxidants combined," said Li. "In contrast, among men with the W or VA genotype, the prostate cancer risk was only weakly altered by these antioxidant levels."

Similar interactions between dietary antioxidants and the variations in the MnSOD gene have previously been linked to risk for breast cancer. *(from http://www.aacr.org)*

TINY ROBOT TO HELP CANCER DIAGNOSIS

By Julie Clothier for CNN

British scientists are developing a tiny robot to fit inside MRI machines, which will improve the accuracy of biopsies taken to screen for prostate cancer. Needle biopsies are currently taken from the prostate using ultrasound technology to detect where the prostate gland is.

But Dr Alex Zivanovic, of Imperial College London, told CNN that ultrasounds provided poor image quality, which makes knowing where to take the biopsy *from* difficult. "Several samples are usually taken; but it's a lucky dip as to whether you've got a relevant one. The tumor could be quite small and you could miss it altogether," he said.

Zivanovic and a group of scientists at the mechatronics in medicine laboratory at the college's department of mechanical engineering are now developing a system involving magnetic resonance imaging (MRI) scanners.

The project is being funded by the National Health Service (NHS) Prostate Cancer Programme, and the machine will be developed within the next year and a half.

"MRI machines are the best way to image soft tissue inside the body; but although using MRI is preferable, to do so involves a very tight space inside the scanner tunnel. There's not much space in there, which makes it difficult," Zivanovic said.

The scientists will build a robot that will be controlled outside the scanner, while the patient will lie inside the tunnel as normal. The MRI technology will enable them to know exactly where the biopsy needs to be taken from.

"It needs to be small enough to fit inside the tunnel but the prostate is fairly large. so it needs to be able to move around,"

Zivanovic said. "It's a way of being more sure of catching a cancer." He said disadvantages of using MRI included the high cost of the scanners. "But detecting more cancers early on will mean fewer patients."

Because the magnetic fields inside MRI scanners are so strong, the scientists will .need to. find an alternative to electricity to power the robot and ensure it can be moved around.

Piezo-ceramic actuators, or ultra sonic, technology -- similar to that used to power the zoom on some cameras -- is being investigated, he said. Scientists at the laboratory have been researching and developing medical robots *for* about 15 years.

Zivanovic said the robots were able to perform high-precision tasks on a small scale. 'They're not being developed to replace surgeons, they're giving surgeons extra skills. It's about combining the advantages of robots over humans together with the advantages of' humans have over robots.

Other projects at the college include a. knee surgery robot, which has proved successful in clinical trials and is now being developed into a commercial product.

Another is a training system to teach surgeons how to perform knee surgery. The robot creates a "virtual" knee so that the operation can be performed in a simulated environment. At St. Mary's Hospital in London, meanwhile, robot technology was used recently to treat a patient suffering from atrial fibrillation, a common persistent heart rhythm disorder.
(http://www.cnn.com/2005/TECH/03/14/spark.prostate)

NEW RISK DATA ON PROSTATE CANCER

By Janelle **Miles**

Australian researchers have questioned' medical opinion that "men are more likely to die with prostate cancer than from prostate cancer", particularly for those in their 50s and 60s.

Queensland Cancer Fund researchers have for the first time produced age-based prostate cancer risk data and found the age at which a man is diagnosed greatly affects his chances of dying from the disease. Using population-based data, they found Australian men with prostate cancer at

age 50 had a 60 per cent likelihood of dying prematurely from the disease compared with 38 per cent for men diagnosed at aged 70.

"This suggests that the oft-used statement [about men dying with the cancer] is misleading, " the researchers wrote in the *Medical Journal of Australia.* They've used their research to produce an information card for general practitioners to assist men in making more informed decisions about whether or not to be screened for the cancer.

Because prostate cancer is normally slow-growing, testing men with a life expectancy of less than 10 years is not normally recommended.

Screening for the cancer involves a blood test, to measure the levels of prostate-specific antigens, and a rectal examination. But even if these prove suspicious, a prostate biopsy would then be needed to indicate cancer.

General practitioner Claire Jackson said, "It's a very complex area compared with a mammogram or a pap smear which you can fairly quickly and easily explain to people. "There's a lot more pros and cons. We're not quite as sure whether there are benefits long-term from screening."

Results of randomised controlled trials in the United States and Europe to measure whether prostate cancer screening can reduce death rates on a population basis are not due *for* several years.

Until then, Professor Jackson said GPs needed to provide men with as much information as possible to help them make their own decisions about whether *to* be screened. Men with a brother or father who've had the cancer diagnosed before the age of 60 have at least double the risk of being diagnosed with the cancer than other men.

Treatment for prostate cancer, including surgery and .radiation therapy, can greatly affect quality of life with side-effects such as impotence and sometimes urinary incontinence and bowel problems.

Queensland Cancer Fund community services director Suzanne Steginga said that before the age-based research, GPs could only give men life-time risk estimates. "Our research means a man can get risk information that's as specific to him as is possible," she said.

Prostate cancer survivor Bill McHugh, who was diagnosed six years ago at age 65, urged men in their 50s and 60s to be screened.
"We find out about our cholesterol, we find out about our heart and all sorts of things," he said. "This is another piece of information that if you're looking after your health, it's a good piece of information to have. "[My prostate cancer] was caught at a stage where it could be cured". The information cards will be available to all Australian GPs from June.

BLOCKING TESTOSTERONE CAN PUT BRAKES ON PROSTATE CANCER

A course of hormone-blocking drugs immediately after diagnosis can cut by one-third the likelihood of dying from advanced prostate cancer, and stop its spread to other parts of the body, according to Australian research that experts say will change the standard treatment for the disease.

The trial compared survival and recurrences in 818 Australian and New Zealand men. They were randomly assigned to receive either radiation alone – the usual treatment – or radiation at the end of a three- or six-month course of drugs to block testosterone. Testosterone is the male hormone that prostate cancer requires to grow and spread.

All the men, whose average age was 68 at diagnosis, had inoperable cancers that had spread throughout their prostate but not to other parts of the body. After 5 years of follow-up, 11% of the men who had six months medication before receiving radiation had further spread of the disease within the prostate, compared with 28% of those on radiation alone.

The results of the study – the largest in the world into hormone treatments for advanced prostate cancer – were presented to the Trans-Tasman Radiation Oncology Group annual general meeting in Darwin. Alan Coates, chief executive officer The Cancer Council Australia paid tribute to the researchers for undertaking the study, which had the potential to reduce the duration of men's drug treatment. *(Advertiser, 20/5, p27)*

GREEN TEA PREVENTS PROSTATE CANCER IN HIGH-RISK MEN

By Serena Gordon

A supplement containing antioxidants from green tea was 90 percent effective in preventing prostate cancer in men at high risk for the disease.

That's the conclusion of an Italian study that found after a year of taking green tea catechins, only one man in a group of 32 who were at higher risk of prostate cancer actually developed the disease, while nine men in a group of 30 high-risk men who took a placebo developed prostate cancer.

"To our knowledge, this is the first study showing that green tea catechins (GTC) have potent chemoprevention activity for human prostate cancer," said study author Saverio Bettuzzi, an associate professor of biochemistry in the School of Medicine at the

University of Parma in Italy. Findings from the study were presented April 20 at the American Association for Cancer Research annual meeting, in Anaheim, Calif.

Other than skin cancer, prostate cancer is the most common cancer affecting men. More than 230,000 American men are diagnosed with this disease each year, according to the American Cancer Society. Since many prostate cancers are found in their early stages, about 99 percent of those diagnosed can expect to live at least five years, while up to 92 percent survive for at least 10 years after their diagnosis. However, prostate cancer can be deadly. The disease claims the lives of more than 30,000 men in the United States annually, making it the second largest cancer killer in men.

Bettuzzi explained that while other studies, including his own previous work, had shown that green tea could inhibit prostate cancer cell growth in laboratory models, the researchers wanted to know if it would work in humans.

They recruited 62 men at high risk of developing prostate cancer because they already had precancerous lesions, which often turn into cancer within a year.

The men were between the ages of 45 and 75. The researchers excluded vegetarians because they may already have a lower risk of developing prostate cancer, men who already consumed green tea, and men taking antioxidant supplements or hormone therapy.

Thirty two of the men were asked to take a 200-milligram pill containing green tea catechins three times daily for a year; the other 30 men were given a placebo.

Biopsies were conducted at six months, and then again a year later. Remarkably, only one man in the treatment group was diagnosed with prostate cancer, while nine men in the control group developed the disease.

"A projection of our data suggests that up to 90 percent of chemoprevention efficacy could be obtained by GTC administration in men prone to developing prostate cancer such as the elderly, African-Americans and those with a family history of prostate cancer," Bettuzzi said.

However, Bettuzzi isn't recommending that men start treating themselves with green tea or green tea supplements. He said to consume an amount equivalent to that used in the study, you would have to drink 12 to 15 cups of tea daily, and that while supplements are commercially available, their quality cannot be assured and they may contain caffeine, or more alarmingly, pesticides or other contaminants.

"This is a very interesting observation that deserves to be studied further," said Dr. Jay Brooks, chairman of hematology and oncology at the Ochsner Clinic Foundation Hospital in New Orleans. But, he added, "Personally, I am not recommending that my patients do this." Bettuzzi also said his findings need to be confirmed in a larger study.

In the meantime, Brooks said that if you're concerned about prostate cancer, be sure you maintain a healthy body weight because obesity increases your risk. And, he said, make sure you go to your doctor for proper prostate cancer screening.

In other prostate cancer news presented at the same meeting, researchers from the Fox Chase Cancer Center in Philadelphia announced that the trace mineral selenium, in combination with other cancer-fighting agents, may make an even more powerful therapy. When a selenium metabolite, dubbed MSA, was combined with a chemotherapy known as TRAIL, more cancer cells underwent self-inflicted cell death (apoptosis). TRAIL alone can induce apoptosis in malignant cells, but some cancer cells are resistant to this therapy.

"The combination of TRAIL and MSA may be a novel strategy for the development of innovative therapeutic modalities targeting apoptosis-resistant forms of prostate cancer," said lead researcher Dr. Vladimir Kolenko.

For more information on tea's potential cancer prevention abilities, go to the National Cancer Institute <http://www.cancer.gov/cancertopics/factsheet/tea-and-cancer-prevention>. (*from www.forbes.com)*

CALCIUM MAY PROTECT AGAINST PROSTATE CANCER

US researchers have found that, contrary to expectations, calcium supplements may actually help protect men against the development of prostate cancer.

Noting that a high calcium intake has previously been linked to the development of prostate cancer, the team, led by Dr. Maria Grau from Dartmouth Medical Centre in Lebanon, New Hampshire, examined data from 672 men participating in a colon cancer prevention trial. All the participants were assigned to receive either daily supplements of 3g. of calcium carbonate or a dummy pill (placebo) for 4 years.

During the 10-year monitoring period, 33 calcium-treated men and 37 placebo-treated individuals were diagnosed with prostate cancer. Further analysis revealed that up to 2 years after the participants stopped taking the supplements, there were significantly fewer cases of prostate cancer in patients given calcium than in those taking the placebo.
In addition, blood samples collected at the beginning of the study and after 4 years showed that prostate-specific antigen levels – an indicator of prostate cancer risk – did not rise significantly among the calcium-treated individuals compared with those taking the placebo. Measurements taken at the start of the study also indicated that levels of dietary calcium and vitamin D were not associated with risk of prostate cancer.

Writing in the journal *Cancer Epidemiology Biomarkers and Prevention*, the team concludes: "In this… clinical trial, there was no increase in prostate cancer risk associated with calcium supplementation and some suggestion of a protective effect." *(www.patienthealthinternational.com*)

MEANWHILE, back in Adelaide

Adelaide researchers are investigating how to use calcium and hormones in the battle against prostate cancer. Flinders Medical Centre's Professor Greg Barritt is exploring ways to kill prostate cancer cells using calcium. He said that when prostate cancer cells die, calcium is part of the process. "So what we're doing is almost mimicking a natural process," he said. Professor Barritt said they were developing ways to make sure the calcium attacked only prostate cancer cells which could lead to a treatment to complement existing therapies.
And a team at Flinders University has discovered an enzyme which could affect the growth of prostate cancer cells. Early stage prostate cancer cells need male hormones called androgens to grow. Researchers have found an enzyme which helps remove these hormones from the prostate. *(Advertiser, 11/5, p23)*

PROSTATECTOMY STUDY

Radical prostatectomy significantly reduces the risk of metastasis and local tumour progression compared with watchful waiting, but the mortality benefits are less clear, a study has found. The procedure reduced the risk of distant metastasis by 40% and local progression by 67%, but this came with side-effects such as impotence and incontinence.

The findings add further information to the complex picture of prostate cancer management, but do not provide clear answers, the authors said. *(New England Journal of Medicine 2005, 352; 1977-84, Australian Doctor, 20/5)*

Breast Cancer Drug Could Benefit Prostate

By MARILYNN MARCHIONE *The Associated Press*

May. 14,2005 - A new study gives encouraging signs that a hormonal drug used to fight breast cancer might help prevent abnormal prostate growths from turning into cancers.

Men who took low doses of the drug for a year cut their chances of developing prostate cancer roughly in half, doctors reported Saturday at meeting of the American Society of Clinical Oncology.

The findings need to be tested in larger studies, specialists say. But this is the first time any drug has been shown to prevent a precancerous condition from forming a tumor.

As many as 50,000 men each year are diagnosed with such growths, and then suffer constant worry and frequent biopsies to see whether cancer has developed. "Before, we had nothing to offer them. Now you may have something," said Dr. Len Lichtenfeld, deputy medical director of the American Cancer Society, which had no role in the research.

The drug is toremifene, sold as Acapodene for treating advanced breast cancer. It selectively blocks some of the effects of estrogen, a hormone men have but in much smaller quantities than women. For decades, prostate cancer prevention and treatment has focused on blocking the male hormone, testosterone. Targeting estrogen "opens up a new area," said the cancer society's medical director, Dr. Harmon Eyre.

Men who have abnormal growths called prostatic intraepithelial neoplasia, or PIN, have about a 30 percent chance of developing prostate cancer within a year and about a 65 percent chance within two years.

"This is a significantly worse prognosis than, say, patients with just an elevated PSA," a blood protein used to measure prostate cancer risk, said Dr. David Price, a Shreveport, La., urologist who led the study. He consults for Memphis-based GTx Inc., which sells toremifene and paid for the study.

It involved 514 men with the growths at 64 sites across the country who were given either fake pills or 20, 40 or 60 milligrams of toremifene for a year. Biopsies were done at six months and a year after treatment started. Cancer rates were similar among the groups at six months, possibly because initial biopsies had missed some cases that were found the second time around.

But after a year, 24.4 percent of those on the drug had developed cancer versus 31 percent of those on fake pills.

That means that for every 100 patients who took the drug for a year, seven cancers were prevented, Price said. The benefit was greatest for those who took the lowest dose for a full year. Their cancer risk was 48 percent lower than men who didn't get the drug.

Side effects were similar for those on the drug and those given fake pills: 1 to 4 percent reported headaches, hot flashes, fatigue, nausea, dry eye or problems with sex.
A larger study testing the lowest dose is enrolling 1,500 men now. If it confirms that the drug can prevent prostate cancer, it would be "an important step" because there's little agreement now about how to treat the disease once it's found, said Dr. Peter Greenwald, director of cancer prevention at the National Cancer Institute.

Two years ago, a huge study showed that a testosterone-blocking drug called finasteride cut the risk of developing prostate cancer by 25 percent in men at high risk of the disease because of family history or other factors. Toremifene would be the first drug to prevent progression to cancer once abnormalities had appeared.

Also at the conference, three studies added to evidence that cholesterol-lowering statin drugs like Lipitor and Pravachol may help prevent various cancers.

Researchers looked at medical records for 1.4 million patients treated at 10 Veterans Affairs centres in Louisiana, Mississippi, Texas and Arkansas found that those taking statins had rates of breast, prostate and lung cancer that were 51%, 54%, and 48% lower, respectively, than those who hadn’t taken such drugs. These are the largest studies on statin use and cancer risk in the United States. Other studies from Europe and Israel reported similar results.

In lab experiments, statins curb cell growth and tumour invasiveness, “so there’s at least a logic to why they should work” against cancer, said Dr. Barnett Kramer, associate director for disease prevention at the National Institutes of Health.

However, this needs to be tested in big experiments that give statins to some and not to others, and watch to see how many cancers develop, he said.
(American Society of Clinical Oncology: http://www.asco.org People Living with Cancer: http://www.PLWC.org)

Prostate cancer linked to mothers' genes (The Associated Press)

Can men inherit risk for a uniquely male disease from their moms? New research raises that possibility. Scientists think they have found a gene that predisposes men to prostate cancer in parts of a cell that come exclusively from mothers, who obviously don't have prostates.
The find gives scientists a different place to look for cancer genes, and it could help biologists better understand what causes prostate cancer, the most common type of tumor in America.

Inherited risk

More than 99 percent of our genes are contained in the nucleus, but a very small number are in tiny structures called mitochondria, little energy factories in cells. Mitochondria are inherited from mothers.

Dr. John Petros and others at Emory University in Atlanta analyzed tissue samples from about 260 prostate cancer patients and found abnormalities in a mitochondrial gene called CO1. The gene helps regulate whether harmful substances that can set the stage for cancer are produced in a cell. Researchers then examined the gene in around 50 healthy men.

They found, the gene was abnormal in 12 percent of those with prostate cancer but in only one man without the disease. "This is a significant difference," said Dr. William Sellers, a cancer genetics expert at Dana-Farber Cancer Institute in Boston who had no role in the study.

The Atlanta group also found a pattern of inheritance of mitochondrial genes that seems to predispose men to prostate and kidney cancer. These may someday give a way to screen for these diseases, Petros said.

Dr. Cornelia Polyak, another Dana-Farber scientist who was among the first to discover cancer-related mutations in mitochondria - a colon cancer gene in 1998 - said the Atlanta findings need to be verified by other studies.
But if true, "it would be very exciting," not just because of the oddity of the location of the gene but also because it may help biologists unravel what processes lead to prostate cancer and how to treat it, she said.

It also might help determine whether men with borderline-high PSA scores need treatment, Sellers said.

"The big thing we're trying to do now in prostate cancer is really focus on people at higher risk," and having a gene signature would allow development of a more accurate index of worry, he said.
URL: http://www.msnbc.msn.com/id/7575018/

BETTER PROSTATE CANCER SURVIVAL WITH SURGERY?

This story is part of WebMD's coverage of the American Urological Association's 2005 Annual Meeting.
Men with aggressive prostate cancer may get a survival advantage from surgical removal of the prostate and surrounding tissue (radical prostatectomy), compared with other options.

Three treatment options were compared by researchers including Ashutosh Tewari, MD, director of the Robotic Prostatectomy and Prostate Cancer-Urologic Oncology Outcomes at Cornell University's medical school. Their findings were presented in San Antonio at the American Urological Association's 2005 Annual Meeting.

"Radical prostatectomy seems to confer a survival advantage over watchful waiting and radiation therapy in patients with high-grade (Gleason greater than or equal to 8) prostate cancer," writes Tewari, who is also an associate professor of urology and public health at Cornell. The Gleason scale ranks prostate tumors; it looks at biopsied cells of the prostate gland and examines the difference between healthy cells and those that are malignant (cancerous). The higher the Gleason score, the more aggressive the prostate tumor is believed to be.

Prostate Cancer Treatment Comparison

The study included 453 men with high-grade prostate cancer (Gleason score 8 out of 10) who were followed for about 4.5 years, on average. About 58% were white and 42% were black; they were treated between January 1980 and December 1997.

Three types of treatment were compared:
Watchful waiting. Observing the disease without treatment (197 men).
Radiation therapy. Using radiation to kill cancer cells (137 men).

Radical prostatectomy. Removing the whole prostate gland and surrounding tissue (119 men)

The study was observational; that is, the men's type of treatment wasn't assigned by the researchers Instead, each group's survival rates were noted by Tewari and colleagues.

Lower Death Risk With Prostatectomy
At the study's last follow-up, 124 patients (27%) had died from prostate cancer. Death rates were lowest in those that underwent radical prostatectomy.

"The risk of overall death following radical prostatectomy was 32% lower than watchful waiting 42% lower than radiation therapy," write researchers. In terms of cancer-specific death, the post-prostatectomy risk was 68% lower than watchful waiting and nearly half as low (49%) as radiation therapy.

There was no significant difference in disease-specific survival between radiation therapy and watchful waiting, according to the study. *(http://my.webmd.com/content/Article/106/108152.htm?printing=true*

BEATING THE BIG C

EVEN mention of the word "cancer" creates fear in some, but the long-held belief that a cancer diagnosis is a death sentence should be waning. And, perhaps more importantly, the view that people always catch cancer by accident is also being challenged.

Cancer is the leading cause of death in Australia, with almost 38,000 deaths from malignant cancer each year. But survival rates are improving steadily each year and there are measures people can take to lower the risk of developing many types of the disease.

According to the Cancer Council Australia, at least one in three cancer cases are preventable, and the number of cancer deaths could be almost halved by the implementation of effective cancer prevention programs.

More than 15,500 cancer deaths each year are due to smoking, sun exposure, poor diet, alcohol, inadequate exercise or being overweight, the council says.

AIHW statistics suggest that of the 90,000 new cases of malignant cancer per year, 12.5 per cent are attributed to smoking and 3.2 per cent to excessive alcohol consumption.

The Cancer Council Australia's Nutrition and Physical Activity Committee spokesman, Steve Pratt, is even more positive: he believes at least half of all cancers could be prevented with a healthy lifestyle.

"Over the long term, there's potential for 44,000 less cases of cancer and 18,000 lives saved every year," Pratt says. "Stop smoking, be physically active every day, aim for a healthy body weight, choose a varied diet with plenty of fruit and vegetables, be sun smart, avoid alcohol, and monitor your body for any changes."

Pratt says in recent years science has lost some of the links between specific foods and cancer prevention, and studies are more often disproving the links. "We just can't say any more that broccoli will prevent x cancer. But we know that generally speaking a good diet helps.

"It is unlikely there will be a silver bullet, but more an interaction and combination of a whole lot of things - but genetics and bad luck have a role to play too."

There is evidence that not eating enough fruit and vegetables probably increases the risk of oesophagal, lung, stomach and bowel cancer, and may also increase the risk of mouth, pharynx, larynx, kidney, ovary and bladder cancer. Eating processed meats and large amounts of red meat increases the risk of bowel cancer and eating large amounts of salt and/or salty foods probably increases the risk of bowel cancer, he says.

Currently there is no evidence of a link between fat intake and cancer, but eating high fat foods contributes to weight gain and being overweight or obese is a risk factor for several cancers, he says.

The Cancer Council Australia recommends a diet consistent with the Australian Guide to Healthy Eating - eat plenty of vegetables, legumes (pulses such as kidney beans, lentils and chickpeas) and fruit; eat plenty of cereals (including breads, rice, pasta and noodles), preferably wholegrain; choose foods low in fat and salt; and eat meat in moderation.

There is also evidence that regular physical activity reduces the risk of breast and bowel cancer and research also suggests regular physical activity may reduce the risk of endometrial, kidney, oesophagus and prostate cancers.

Prostate cancer:
Almost one man in ten will develop prostate cancer at some time in his life and it is the second largest cause of male cancer deaths behind lung cancer. There are 10,000 men diagnosed with the condition every year and 2600 die of the disease annually.*(Latest figures available-2001 – 11,191 men diagnosed, with 2,718 deaths, Ed.)*

"We still don't have a good handle on what causes prostate cancer, but it is fair to say it seems to be a western society thing rather than an eastern society thing - so the types of food we eat may be a key determinant," says Phillip Stricker, the chairman of the department of urology at Sydney's St Vincent's Hospital. "Too much saturated fats, excessive calories, and not enough soy protein could be problematic."

Phyto-estrogens in soy protein seem to be a preventative and there are products containing extracts of phyto-estrogens on the market, associate professor Stricker says.

"It is very hard to prove or disprove their worth at this stage because we have to have a person taking the substance for 20 years before we can say it is a preventative.

"People who get very little sunlight appear to be at higher risk, so vitamin D may have a role to play in prevention and people deficient in the mineral selenium also appear to have an increased incidence of prostate cancer.

"It appears obesity increases your chances of developing the cancer - that may explain why Japanese do not get it. But Italians don't either, which has led to research suggesting tomatoes cooked in oil can be a beneficial preventative."

Skin cancer:
More than 380,000 Australians are treated every year for skin cancer. It is the most expensive burden on the health system of any cancer. Australia has the highest rate of skin cancer in the world, but the prevention of skin cancer is simple and well publicised: sun protection. Skin cancer is caused by overexposure to ultraviolet (UV) radiation, says Craig Sinclair, chairman of the Cancer Council's skin cancer committee.

Protection is needed to prevent skin cancer even when UV levels are moderate. In some parts of Australia (for example, the tropical north) this is most of the day, all year round. In southern Australia, it is safe to go without sun protection in June and July, Sinclair says - unless you are in the Southern Alps, on the water, or out all day.

"When UV is forecast as moderate, wear a broad spectrum sunscreen with an SPF of 30-plus and use one teaspoon per limb," Sinclair says. "Create shady environments and check if the medication you are taking could increase your sensitivity to the sun."

Lung cancer:
Lung cancer has the largest number of deaths of any cancer because most cases are detected in the later stages. But the rates are decreasing and statisticians predict other cancers will take over its top deaths status in the coming decade, says Andrew Ellerman, chairman of the Cancer Council's tobacco issues committee.
Prevention is "obvious", Ellerman says. "Smoking is responsible for 80 per cent of all lung cancers.

"Atmospheric pollution, asbestos, radon, are relevant but insignificant in comparison to smoking, and there is no clear evidence to suggest any other factor will help prevent lung cancer." Smoking increases your risk of cancer in many other body parts. The list of smoking-related cancers includes: head (nose, nasal sinus, mouth, lip, tongue, throat, voice box); oesophagus (gullet); pancreas; kidney; bladder; stomach; liver; rectum; anus; cervix; vulva; penis; and blood (leukaemia). And quitting smoking has plenty of other health benefits. Smoking contributes to heart, respiration and circulatory disease; asthma; and low birth weight infants.

Bowel cancer:
Bowel cancer is the second most common cause of cancer-related death in Australia. Almost 5000 people die from the cancer each year. According to the Cancer Council, around 25 per cent, or more than 3000 of the 13,000 bowel cancers diagnosed in Australia every year, could be prevented if people maintained a healthy body weight, ate a healthy diet and engaged in daily physical activity.

A National Health and Medical Research Council guide on bowel cancer says 66 to 75 per cent of bowel cancer cases could be prevented by eating a healthy diet and exercising regularly. "Vegetables have been shown to provide strong protection against bowel cancer, therefore you should eat a wide variety, especially cruciferous vegetables such as bok choy, broccoli, brussels sprouts, cabbage, cauliflower, swedes and turnips," the NHMRC report says.

Eat plenty of fibre, especially wholegrain cereal fibres and wheat bran, and eat a low fat, low calorie diet. And cut back on red and processed meats.

High consumption of red and processed meats over a long period of time is associated with an increased risk of bowel cancer. It could also be beneficial to ensure you get the daily recommended dose of between1000mg to 1200mg of calcium, as some studies have shown a link between calcium and bowel cancer prevention, while others have been unable to find a link.

Several studies have suggested that there is a link between a substantial intake of alcohol and bowel cancer. The US National Cancer Institute bowel cancer prevention website says among populations that consume a diet high in fat, protein, calories, alcohol, and meat (both red and white) and low in calcium and folate, colorectal cancer is more likely to develop than among populations that consume a low-fat, high-fibre diet. A diet low in vitamin D may also increase the risk of colorectal cancer.

And some studies have shown that the use of non-steroidal anti-inflammatory drugs may be associated with a reduced risk of colorectal cancer.
(edited version from W/E Australian, 8-9/7/05)

E.J.WHITTEN FOUNDATION

Around this time of the AFL season, every year, we hear about the “Legends” game, the proceeds of which go to the E.J. Whitten Foundation. This foundation was established to commemorate the memory of the late Ted Whitten, who died from prostate cancer in August 1995. The foundation is set up to raise funds for research and create awareness regarding prostate cancer

So I wondered why we don’t hear much more about the foundation, and what it is achieving with the funds raised by the “Legends” game each year.

According to the website (www.ejwhittenfoundation.com) the foundation was established in 1995, and has raised nearly $800,000 by running numerous functions throughout the year, e.g., E.J. Whitten Legends game, E.J. Whitten Grand Final Luncheon, Brownlow Medal Dinner, Caduceus Club Memorial function, and others that attract good support. There have been golf days and a Gala Ball is planned. The foundation also receives bequests and donations from families and friends of prostate cancer sufferers Around $250,000 is raised per year.

Money raised each year is donated to the Cancer Council Victoria through the E.J. Whitten Fellowship, The Alfred Foundation and Garvan Institute Prostate Cancer Research Centres – all funded by the E.J .Whitten Foundation. The E.J. Whitten Prostate Assessment Centre was launched and funded by the E.J. Whitten Foundation.

They are also creating awareness about prostate cancer, particularly for males who are over 50 years.

There is not much detail as to what type of research has been funded, or the type of awareness activities conducted. The website appears not to be up to date, and short on detail about exactly what the funds are really funding.

Promising Lead in Prostate Cancer Diagnosis, New Discovery

Medical News Toady 6.11.05

Researchers led by Dr Shiv Srivastava from the Center for Prostate Disease Research (CPDR), Uniformed Services University of the Health Sciences (USU), report the ground breaking discovery of the ETS-Related Gene (ERG) as one of the frequent proto-oncogene overexpressions in prostate cancer cells. This discovery provides a very promising addition to a select group *of* genes; whose expression is frequently altered in prostate cancer cells and could provide novel molecular targets for diagnosis, 'prognosis or therapy of prostate cancer in the future.

The report by Dr. Gyorgy Petrovics et al showing ERG expression alterations in a large fraction *of* prostate cancer cells is published in the latest issue (May 26, *2005)* of leading cancer research journal Oncogene. Using laser capture microdissected prostate epithelial cells from malignant and benign prostate tissues and GeneChips, researchers identified ERG as the first proto-oncogene that is commonly overexpressed in early-phase prostate cancer.

The cancer-causing genes known as oncogenes and tumor suppressor genes have long been known to be major factors in the development *of* cancer cells, and mutation or altered expression *of* these genes has been identified in diverse human cancers. However, for prostate cancer, which is the most common non-skin cancer and the second leading cause of cancer-related deaths among men in the U.S., the identification of oncogenes and tumor suppressor genes that are altered in most prostate cancer cells have eluded scientists thus far.

Dr. Charles Bieberich, a leading prostate cancer researcher at the University of Maryland, Baltimore Campus, who is studying the regulation and function of a prostate tissue-specific tumor suppressor, the NKX3.1 homeobox gene, stated: "I *find* it very exciting that overexpression of a proto-oncogene transcription factor like ERG is associated with prostate cancer at such a high frequency." Dr. Beiberich's group also studies interactions *of* NKX3.1 with a prostate tissue-derived ETS factor (PDEF). "These novel observations warrant further studies looking into the functional effects of ERG overexpression in model systems of prostate cancer. It is likely that additional prostate cancer-relevant gene alterations also will be discovered by the microgenomics approach used in this study."

CPDR researchers went on to assess the cancer association of ERG change in combination with other prostate cancer marker genes. They found that when they combined ERG with two other genes, DD3 and AMACR, which are described by other laboratories as commonly overexpressed in prostate cancer, the three-gene panel exhibited cancer association in 98% of the prostate cancer patients tested. This result shows promise for prostate cancer diagnosis. Intriguing correlations of ERG overexpression features also have been noted for PSA recurrence-free survival of prostate cancer patients after radical prostatectomy.

Due to the previously established oncogenic functions of ERG, CPDR researchers also propose to explore ERG as a potential therapeutic target in prostate cancer treatment.

"This is a significant new finding, but it will take time to translate this into clinically useful products. The most likely first potential clinical application could be improved detection of prostate cancer by providing a novel and functionally relevant biomarker. However, the exploration of the therapeutic potential of ERG is equally important," said Dr. Robert Vessella from the University of Washington, Seattle, who is one of the pioneers in developing new diagnostic and prognostic cellular and molecular markers for prostate cancer. Dr. Vessella is one of the primary leaders of a research effort at the University of Washington that is defining mechanisms of prostate cancer metastasis.

This discovery was the result of a highly coordinated effort by urologists, pathologists and cancer biologists from Walter Reed Army Medical Center (WRAMC), USU, the Armed Forces Institute of Pathology (AFIP), the Walter Reed Army Institute of Research (WRAIR) and the National Human Genome Research Institute (NHGRI). *(from www.pcacoalition.org)*

Abbott to Give Cancer Drug to some **Patients**

CHICAGO (Reuters) - Abbott Laboratories said on Thursday it has received U.S. regulatory approval to make its experimental prostate cancer drug Xinlay available to certain patients even though it is not yet approved.
The U.S. Food and Drug Administration has given Abbott permission to make Xinlay available in the United States to men with late-stage prostate cancer who do not respond to hormone therapy under a program known as expanded access.

An Abbott spokeswoman said the company will distribute information about the drug to investigators in its clinical trials and to patient advocate groups. Abbott will be required to report safety information to the FDA.
The drug will be made available later this summer.

"We believe that today's news is a signal that the FDA considers Xinlay to be safe and effective enough to be used on patients," Banc of America Securities analyst Glenn Novarro said in a research note. "This gives us more confidence that the drug will receive approval by year-end." He believes about 200 prostate cancer patients will receive treatment under the program.

If approved in late 2005, Novarro expects Xinlay sales to reach $300 million in 2006 and ultimately reach annual sales of $1.5 billion to $2 billion.

An FDA advisory hearing on Xinlay is set for September. The current application is for patients whose cancer has spread to the bone and who have failed to respond to standard treatments

Abbott is conducting clinical trials to expand approval for use of the drug earlier in the disease.
Xinlay is an oral, once-a-day, non-chemotherapy cancer agent. It belongs to a class of compounds known as selective endothelin-A. receptor antagonists, a protein thought to be involved in the spread of cancer cells*. (Reuters, from www.pcacoalition.org)*

ASCO UNVEILS LATEST TREATMENT STRATEGIES

19/05/2005 - In the wake of the latest American Society of Clinical Oncology's (ASCO) annual conference, drug companies from around the world showcased potential drug treatments, with a number of new molecular targets as well as therapy strategies for cancer.

The ASCO <http://www.asco.org> conference, which took place in Orlando, Florida between May 13-17, took the opportunity to present the best of clinical and translational cancer research. Many drug companies presented new drug candidates at the show as DrugResearcher.com presents a round up of the latest research presented at the meeting.

Just ahead of the start of ASCO, OncoGenex Technologies<http://www.oncogenex.ca> announced results from a second Phase 1 study of OGX-011, the company's lead product candidate. OGX-011 is a second-generation antisense inhibitor of clusterin, a cell-survival protein that is over-expressed in many cancers and is associated with treatment resistance and poor clinical outcome.

At the 2004 ASCO meeting, OncoGenex and Isis reported that OGX-011, in a dose-dependent fashion, achieved effective drug concentration in prostate cancer tissue and produced up to a 91 per cent dose-dependent decrease in clusterin expression.
Results from the Phase 1 clinical trial also demonstrated that the inhibition of clusterin was associated with the predicted pharmacological outcome, the death of prostate cancer cells.

A second Phase 1 study, also featured at this year's ASCO annual meeting, was designed to determine recommended dose of OGX-011 in combination with Taxotere in various solid tumours. In preclinical animal studies, OGX-011 improved the potency of traditional chemotherapies by more than 10-fold in prostate cancer with no increase in toxicity. OncoGenex plans to initiate Phase 2 clinical trials of OGX-011 in patients with lung, breast and prostate cancers this year.

New data presented by Pfizer <http://www.pfizer.com> suggests that its investigational new drug Sutent/SU11248 (sunitinib malate) extends overall survival in gastrointestinal stromal tumours (GIST). Sutent is a highly selective, multi-targeted tyrosine kinase inhibitor that tumours of blood and nutrients needed for growth and simultaneously starves and kills cancer cells that make up tumours.

Long-term follow-up data from the Phase I/II GIST study that served as the basis for the larger Phase III trial demonstrated that Sutent extended overall survival to nearly 20 months in patients whose cancer had progressed despite treatment with other standard therapies. In addition, the median time to tumour progression in this study was 7.8 months for all patients, with some specific subtypes of patients benefiting even more dramatically than would be expected with Gleevec.

Results from a double-blind Phase III study of more than 300 GIST patients resistant to or intolerant of the standard treatment Gleevec (imatinib mesylate) showed Sutent significantly prolonged the time to tumour progression (6.3 months on Sutent vs 1.5

months for controls) and reduced the risk of death by approximately 50 per cent compared to placebo.

"These results substantiate the concept that multi-targeted molecular therapy can overcome resistance to other targeted drugs in cancer," said Dr George Demetri of Harvard University's Dana-Farber Cancer Institute in Boston, the lead investigator on the Sutent trial for GIST.
"We think that Sutent may have a broad spectrum of activity for many different forms of cancer beyond what we have seen in patients with GIST. We believe that Sutent is an important step forward in cancer therapy," he added.

According to DataMonitor <http://www.datamonitor.com>, the oncology market is the third largest pharmaceutical market, behind the cardiovascular and CNS therapy areas, worth an estimated $40 billion ($32 billion) in 2004. Datamonitor projects the sector to grow to $53 billion by 2008, yielding a compound annual growth rate (CAGR) greater than 10 per cent over this period.

In 2004, the top 20 cancer drugs in each of the seven major pharmaceutical markets generated combined sales exceeding $27 billion, with the US accounting for 67 per cent of this total, Japan 12 per cent and the five EU countries 21 per cent (Midas, IMS Health, April 2004). Collective sales in these markets represent approximately 70 per cent of global oncology revenues.

Over the next 10 years, Datamonitor predicts that of the cancer drugs that currently hold a top 20 position in one or more of the seven markets, only those in the innovative and supportive care classes will maintain a positive CAGR. Conversely, cytotoxics and antihormonals are expected to experience declining sales.

US bioscience company, Kosan Biosciences <http://www.kosan.com>, presented favourable interim data from two Phase Ib clinical trials evaluating KOS-862 (Epothilone D) in combination with carboplatin (Paraplatin) and gemcitabine (Gemzar) in patients with advanced solid tumours. Both trials were open-label, dose-escalation, two-centre studies designed to determine the maximum tolerated dose, toxicity profile, pharmacokinetics, and recommended Phase II dose of the combination therapies.

In the trial investigating KOS-862, the twelve patients that were treated with the KOS-862/carboplatin combination therapy, one patient with ovarian cancer experienced a complete response, and one patient with hepatocellular carcinoma demonstrated a 41 per cent decrease in aFP (alpha feto-protein) and stable disease for 21 weeks, Pharmacokinetic data revealed no apparent drug-drug interactions between the two agents.

In the trial investigating KOS-862 and gemcitabine, out of the fourteen patients that were treated with the KOS-862/gemcitabine combination therapy, one patient was observed to have a partial response, and two patients had disease stabilisation for more than three months, suggesting evidence of anti-cancer activity. Pharmacokinetic data revealed no apparent drug-drug interactions between the two agents.

KOS-862 is a polyketide that inhibits cancer cell growth in vitro by a mechanism similar to paclitaxel, and pre-clinical models have shown the compound to be effective against paclitaxel-resistant tumours. KOS-862 continues to be evaluated in Phase II monotherapy trials in breast and prostate cancer, as well as Phase Ib combination trials with Gemzar, Paraplatin and Herceptin.

It wasn’t just cancer drug trials that featured in the meeting. The ASCO conference took the opportunity to inform a worldwide audience about new techniques and methods that could be used to prevent cancer development or reduce the number of incidences.

A new study suggested that detecting a genetic deletion during the initial evaluation of children with neuroblastoma might indicate to physicians that they should recommend a more aggressive regimen of chemotherapy to fight the cancer. It is generally accepted that when certain genes are deleted on a particular section of chromosome 11, the result is an aggressive form of the childhood cancer neuroblastoma.

Edward Attiyeh, a paediatric oncology fellow at The Children's Hospital of Philadelphia <http://www.chop.edu>, reported on a study of 915 patients that an abnormal increase in the number of copies, of a cancer-causing gene called MYCN heralds a high-risk, aggressive cancer. However, a significant number of neuroblastomas are aggressive without having amplified MYCN. "The deletion of genetic material on chromosome 11 may account for a significant percentage of these high-risk neuroblastomas," said Dr Attiyeh.

It is unknown what causes the deletion of genes on chromosome 11, at a location designated chromosome 11q23. However, the loss of material at that site apparently removes the protective effect of a tumour suppressor gene, and thereby allows the tumour to grow. Patients in the study with the chromosome deletion had a three-year overall survival rate of 66 per cent, compared to 83 per cent for patients without the deletion.

Neuroblastoma, which accounts for 10 per cent of all paediatric cancers, often occurs as a solid tumour in a child's abdomen or chest. Some cases of neuroblastoma are low risk, and resolve after surgeons remove the tumour. Other cases are more aggressive, and are more likely to resist initial treatment, or to cause a relapse. Identifying the correct risk level allows doctors to treat aggressive cancers appropriately, while not subjecting children with low-risk cancer to over treatment.
<http://www.DrugResearcher.com/feedback/index.asp?type=4&page=%2Fnews%2FprintNewsBis%2Easp%3Fid%3D60113>

AVOCADO EXTRACT INHIBITED PROSTATE CANCER CELL GROWTH

"Although the avocado is known as a rich source of monounsaturated fatty acids, there has been far less attention given to its content of other bioactive substances including carotenoids, which might contribute to cancer preventive properties similar to those attributed to other fruits and vegetables," investigators in the United States reported. "The yellow-green color of the avocado prompted us to study the carotenoid content of this fruit using established methods in our laboratory. The California Hass avocado (Persea americana Mill.) was selected for study, because it is the most commonly consumed variety in the southwest United States," explained Q.Y. Lu and colleagues, University of California at Los Angeles.

"These avocados were found to contain the highest content of lutein among commonly eaten fruits as well as measurable amounts of related carotenoids (zeaxanthin, alpha-carotene, and beta-carotene). Lutein accounted for 70% of the measured carotenoids, and the avocado also contained significant quantities of vitamin E," the research team discovered.

Scientists said, "An acetone extract of avocado containing these carotenoids and tocopherols was shown to inhibit the growth of both androgen-dependent (LNCaP) and androgen-independent (PC-3) prostate cancer cell lines in vitro. Incubation of PC-3 cells with the avocado extract led to G2/M cell cycle arrest accompanied by an increase in p27 protein expression."

The researchers concluded, "In common with other colorful fruits and vegetables, the avocado contains numerous bioactive carotenoids. Because the avocado also contains a significant amount of monounsaturated fat, these bioactive carotenoids are likely to be absorbed into the bloodstream, where in combination with other diet-derived phytochemicals they may contribute to the significant cancer risk reduction associated with a diet of fruits and vegetables."

Lu and colleagues published their study in the Journal of Nutritional Biochemistry (Inhibition of prostate cancer cell growth by an avocado extract: role of lipid-soluble bioactive substances. J Nutr Biochem, 2005;16(1):23-30).

(URL: http://www.cancercompass.com/cancer-news/1,8843,00.htm)

GLEASON PATTERN COMBINATION HELPS GUIDE PROSTATE CANCER

NEW YORK JUN 03, 2005 (Reuters Health) - Following radical prostatectomy, the use of the combined percentage of Gleason patterns 4 and 5 appears to be the best predictor of cancer progression, according to Indianapolis-based researchers.

"Our findings may have impact on understanding the biology of prostate cancer and the development of an effective prognostication scheme that influences therapeutic regimens," lead researcher Dr. Liang Cheng told Dr. Cheng and colleagues at Indiana

University School of Medicine examined specimens from 364 prostate cancer patients who had undergone radical prostatectomy. None had received preoperative androgen therapy.

Employing analytical techniques including the percentages of Gleason patterns 4 and 5, Gleason score, preoperative prostate-specific antigen (PSA) score and other factors, the team found that the combined percentages of Gleason patterns 4 and 5 and total tumor volume were significant predictors of PSA recurrence ($p < 0.0001$).

The presence of high-grade elements may dictate the biologic behavior of tumors, the investigators note in the May 1st issue of the Journal of Clinical Oncology. As Dr. Cheng pointed out, "the worst cancer grade is most closely linked to the biological aggressiveness of prostate cancer."

The investigators established that the combined percentages of Gleason patterns 4 and 5 appear are superior to conventional Gleason score in identifying patients at increased risk.

They recommend that "the amount of high-grade cancer in a prostatectomy specimen should be taken into account in therapeutic decision making and assessment of patient prognosis." SOURCE: * Journal of Clinical Oncology http://medlineplus.gov/ also <http://www.nacme.org/> http://www.bread.org

NEW PROSTATE CANCER STUDIES LOOK AT HORMONES and PROTEIN CONNECTION

Two researchers at McMaster University's Faculty of Health Sciences have been awarded research grants from the Prostate Cancer Research Foundation of Canada.

Prostate cancer is the second most deadly form of cancer in men after smoke-related lung cancer. It is estimated by the Prostate Foundation of Canada that one in eight Canadian men will develop the disease, and one in four of them will die from prostate cancer.

Damu Tang, assistant professor, medicine, has received $120,000 to research the newly identified protein PTEN, to see how important it is in preventing prostate cancer progression.

In its early stage, prostate cancer requires the hormone androgen to grow, until it progresses to a point where androgen is no longer needed. Clinical observations demonstrate clearly that 50% of advanced prostate cancers have no PTEN.

It is believed that the loss of PTEN removes a crucial protection, which facilitates prostate cancer progression into the androgen-independent stage.

Sujata Persad, assistant professor, Biochemistry and Biomedical Sciences, was awarded $60,000 to study Beta-catenin, a protein highly associated with prostate cancer initiation, progression and metastases. Beta-catenin plays two important roles inside cells: it functions to glue cells to each other and it promotes cell growth and survival.

Beta-catenin can be modified by having sugar molecules attached to its structure. Persad's team will investigate whether modifying beta-catenin affects its role in promoting cell growth and survival. Modification happens less frequently in prostate cancer cells than in normal prostate cells, and using sugar modification may regulate the function of beta-catenin to prevent tumor formation in prostate cells. *This article was prepared by Biotech Law Weekly editors from staff and other reports. My CancerCompass <http://www.cancercompass.com/index.htm>*

What Happens Next?

My mate Jimmy had an appointment to see the urologist. The waiting room was full with patients.
The nurse was a large, unfriendly woman who looked like a sumo wrestler, and he gave her his name. In a very loud voice the nurse said “Yes, Mr. Jim, you want to see the doctor about your impotence, right?”
All the people in the waiting room turned their heads to look at the very embarrassed Jim. He recovered quickly and in an equally loud voice replied “No, I’ve come to enquire about a sex change operation, but I don’t want the same doctor who did yours”. (Ross Tait, in Queensland Prostate Cancer News)

Compiled by
Trevor Hunt