



The Vitamin & Herb Stores

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Editors Top Five:

Not enough research to justify a top five yet.

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Public release date: 8-Jan-2009

Maslinic acid provides a natural defense against colon cancer

Researchers from the University of Granada and the University of Barcelona have shown that treatment with maslinic acid, a triterpenoid compound isolated from olive-skin pomace, results in a significant inhibition of cell proliferation and causes apoptotic death in colon-cancer cells. Maslinic acid is a novel natural compound and it is able to induce apoptosis or programmed death in human HT29 colon-cancer cells via the intrinsic mitochondrial pathway. Scientifics suggest this could be a useful new therapeutic strategy for the treatment of colon carcinoma.

This study is the first to investigate the precise molecular mechanisms of the anti-tumoral and pro-apoptotic effects of maslinic acid against colon-cancer. Chemopreventive agents of a natural origin, often a part of our daily diet, may provide a cheap, effective way of controlling such diseases as cancer of the colon. A wide range of studies in recent years has shown that triterpenoids hinder carcinogenesis by intervening in

pathways such as carcinogen activation, DNA repair, cell cycle arrest, cell differentiation and the induction of apoptosis in cancer cells.

Triterpenoids are compounds present in a wide range of plants used in traditional medicine and known to have antitumoral properties. Low concentrations of maslinic acid are to be found in plants with medicinal properties, but its concentration in the waxy skin of olives may be as high as 80%.

The results of the study could contribute to the development of maslinic acid for use as cancer chemotherapeutic or chemopreventive agents.

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Chemopreventive agents in black raspberries identified

PHILADELPHIA – A study published in Cancer Prevention Research, a journal of the American Association for Cancer Research, identifies components of black raspberries with chemopreventive potential.

Researchers at the Ohio State Comprehensive Cancer Center found that anthocyanins, a class of flavonoids in black raspberries, inhibited growth and stimulated apoptosis in the esophagus of rats treated with an esophageal carcinogen.

"Our data provide strong evidence that anthocyanins are important for cancer prevention," said the study's lead author, Gary D. Stoner, Ph.D., a professor in the department of internal medicine at Ohio State University.

Stoner and his team of researchers fed rats an anthocyanin-rich extract of black raspberries and found that the extract was nearly as effective in preventing esophageal cancer in rats as whole black raspberries containing the same concentration of anthocyanins. This study demonstrates the importance of anthocyanins as preventive agents in black raspberries and validated similar in vitro findings. It is among the first to look at the correlation between anthocyanins and cancer prevention in vivo.

Stoner and his colleagues have conducted clinical trials using whole berry powder, which has yielded some promising results, but required patients to take up to 60 grams of powder a day. "Now that we know the anthocyanins in berries are almost as active as whole berries themselves, we hope to be able to prevent cancer in humans using a standardized mixture of anthocyanins," said Stoner.

"The goal is to potentially replace whole berry powder with its active components and then figure out better ways to deliver these components to tissues, to increase their uptake and effectiveness. Ultimately, we hope to test the anthocyanins for effectiveness in multiple organ sites in humans," said Stoner.

Public release date: 8-Jan-2009

Study shows California's autism increase not due to better counting, diagnosis

Institute has found that the seven- to eight-fold increase in the number children born in California with autism since 1990 cannot be explained by either changes in how the condition is diagnosed or counted — and the trend shows no sign of abating.

Published in the January 2009 issue of the journal Epidemiology, results from the study also suggest that research should shift from genetics to the host of chemicals and infectious microbes in the environment that are likely at the root of changes in the neurodevelopment of California's children.

"It's time to start looking for the environmental culprits responsible for the remarkable increase in the rate of autism in California," said UC Davis M.I.N.D. Institute researcher Irva Hertz-Picciotto, a professor of environmental and occupational health and epidemiology and an internationally respected autism researcher.

Hertz-Picciotto said that many researchers, state officials and advocacy organizations have viewed the rise in autism's incidence in California with skepticism.

The incidence of autism by age six in California has increased from fewer than nine in 10,000 for children born in 1990 to more than 44 in 10,000 for children born in 2000. Some have argued that this change could have been due to migration into California of families with autistic children, inclusion of children with milder forms of autism in the counting and earlier ages of diagnosis as consequences of improved surveillance or greater awareness.

Hertz-Picciotto and her co-author, Lora Delwiche of the UC Davis Department of Public Health Sciences, initiated the study to address these beliefs, analyzing data collected by the state of California Department of Developmental Services (DDS) from 1990 to 2006, as well as the United States Census Bureau and state of California Department of Public Health Office of Vital Records, which compiles and maintains birth statistics.

Hertz-Picciotto and Delwiche correlated the number of cases of autism reported between 1990 and 2006 with birth records and excluded children not born in California. They used Census Bureau data to calculate the rate of incidence in the population over time and examined the age at diagnosis of all children ages two to 10 years old.

The methodology eliminated migration as a potential cause of the increase in the number of autism cases. It also revealed that no more than 56 percent of the estimated 600-to-700 percent increase, that is, less than one-tenth of the increased number of reported autism cases, could be attributed to the inclusion of milder cases of autism. Only 24 percent of the increase could be attributed to earlier age at diagnosis.

"These are fairly small percentages compared to the size of the increase that we've seen in the state," Hertz-Picciotto said.

Hertz-Picciotto said that the study is a clarion call to researchers and policy makers who have focused attention and money on understanding the genetic components of autism. She said that the rise in cases of autism in California cannot be attributed to the state's increasingly diverse population because the disorder affects ethnic groups at fairly similar rates.

"Right now, about 10 to 20 times more research dollars are spent on studies of the genetic causes of autism than on environmental ones. We need to even out the funding," Hertz-Picciotto said.

The study results are also a harbinger of things to come for public-health officials, who should prepare to offer services to the increasing number of children diagnosed with autism in the last decade who are now entering their late teen years, Hertz-Picciotto said.

"These children are now moving toward adulthood, and a sizeable percentage of them have not developed the life skills that would allow them to live independently," she said.

The question for the state of California, Hertz-Picciotto said, will become: "What happens to them when their parents cannot take care of them?"

"These questions are not going to go away and they are only going to loom larger in the future. Until we know the causes and can eliminate them, we as a society need to provide those treatments and interventions that do seem to help these children adapt. We as scientists need to improve available therapies and create new ones," Hertz-Picciotto said.

Hertz-Picciotto and her colleagues at the M.I.N.D Institute are currently conducting two large studies aimed at discovering the causes of autism. Hertz-Picciotto is the principal investigator on the CHARGE (Childhood Autism Risk from Genetics and the Environment) and MARBLES (Markers of Autism Risk in Babies-Learning Early Signs) studies.

CHARGE is the largest epidemiologic study of reliably confirmed cases of autism to date, and the first major investigation of environmental factors and gene-environment interactions in the disorder. MARBLES is a prospective investigation that follows women who already have had one child with autism, beginning early in or even before a subsequent pregnancy, to search for early markers that predict autism in the younger sibling.

"We're looking at the possible effects of metals, pesticides and infectious agents on neurodevelopment," Hertz-Picciotto said. "If we're going to stop the rise in autism in California, we need to keep these studies going and expand them to the extent possible."

Public release date: 12-Jan-2009

Hormone therapy linked to brain shrinkage, but not lesions

ST. PAUL, Minn. – Two new studies show that hormone therapy for women is linked to brain shrinkage, but not to the small brain lesions that are the first sign of cerebrovascular disease. The studies are published in the January 13, 2009, print issue of *Neurology*[®], the medical journal of the American Academy of Neurology.

Earlier studies showed that estrogen with or without added progestin increased the risk for developing dementia and cognitive decline, or difficulty with thinking skills and memory in women age 65 and older.

These new studies aimed to look at how the hormones might affect memory and thinking skills. The studies involved participants of the Women's Health Initiative hormone therapy clinical trials who also agreed to participate in a substudy called the Women's Health Initiative Memory Study. These studies were stopped earlier than planned when researchers found that the hormone therapy increased health risks and failed to prevent heart disease.

Researchers took MRI brain scans of 1,400 women ages 71 to 89 one to four years after the Women's Health Initiative hormone studies ended. They found women who had taken estrogen with or without progestin had smaller brain volumes in two areas of the brain than the women who had taken a placebo.

Brain volume was 2.37 cubic centimeters lower in the frontal lobe in the women taking estrogen and .10 cubic centimeters lower in the hippocampus. Both areas are involved in thinking and memory skills, and loss of volume in the hippocampus is a risk factor for dementia.

"These effects were most apparent in women who may already have had some memory problems before they started taking hormones," said study author Susan Resnick, PhD, of the National Institute on Aging in Baltimore, MD. "This suggests that estrogen may adversely affect thinking skills among women whose brains may already be beginning a neurodegenerative disease process."

In the second study, researchers found that hormone therapy was not linked to an increase in volumes of small vascular lesions in the brain or "silent strokes" that are often the first sign of cerebrovascular disease. "This was not what we expected to find," said study author Laura H. Coker, PhD, of Wake Forest University Health Sciences in Winston-Salem, NC. Coker said the negative effects of hormone therapy on cognitive skills may not be related primarily to vascular disease but to neurodegeneration, which is supported by the first study's findings of brain atrophy.

Public release date: 12-Jan-2009

Vitamin D is the 'it' nutrient of the moment

MAYWOOD – Vitamin D is quickly becoming the "it" nutrient with health benefits for diseases, including cancer, osteoporosis, heart disease and now diabetes.

A recent review article published by researchers from Loyola University Chicago Marcella Niehoff School of Nursing concluded that adequate intake of vitamin D may prevent or delay the onset of diabetes and reduce complications for those who have already been diagnosed. These findings appeared in the latest issue of Diabetes Educator.

"Vitamin D has widespread benefits for our health and certain chronic diseases in particular," said Sue Penckofer, Ph.D., R.N., study co-author and professor, Loyola University Chicago Marcella Niehoff School of Nursing. "This article further substantiates the role of this nutrient in the prevention and management of glucose intolerance and diabetes."

Many of the 23 million Americans with diabetes have low vitamin D levels. Evidence suggests that vitamin D plays an integral role in insulin sensitivity and secretion. Vitamin D deficiency results in part from poor nutrition, which is one of the most challenging issues for people with diabetes. Another culprit is reduced exposure to sunlight, which is common during cold weather months when days are shorter and more time is spent indoors.

One study examined for this review article evaluated 3,000 people with type 1 diabetes and found a decreased risk in disease for people who took vitamin D supplements. Observational studies of people with type 2 diabetes also revealed that supplementation may be important in the prevention of this disease.

"Management of vitamin D deficiency may be a simple and cost-effective method to improve blood sugar control and prevent the serious complications associated with diabetes," said Joanne Kouba, Ph.D., R.D., L.D.N., study co-author and clinical assistant professor of dietetics, Loyola University Chicago Marcella Niehoff School of Nursing.

Diet alone may not be sufficient to manage vitamin D levels. A combination of adequate dietary intake of vitamin D, exposure to sunlight, and treatment with vitamin D2 or D3 supplements can decrease the risk of diabetes and related health concerns. The preferred range in the body is 30 - 60 ng/mL of 25(OH) vitamin D.

"People at risk for diabetes should be screened for low vitamin D levels," said Mary Ann Emanuele, M.D., F.A.C.P., study co-author and professor of medicine, division of endocrinology and metabolism, Loyola University Health System. "This will allow health care professionals to identify a nutrient deficiency early on and intervene to improve the long term health of these individuals."

Vitamin D deficiency also may be associated with hyperglycemia, insulin resistance, hypertension and heart disease. In fact, Penckofer recently published another study in Circulation that reported on the role of chronic vitamin D deficiency in heart disease. The Circulation study authors included Glen W. Sizemore, MD, emeritus professor of Medicine, Division of Endocrinology and Metabolism, Loyola University Chicago Stritch School of Medicine, and Diane E. Wallis, MD, Midwest Heart Specialists, Downers Grove, Ill.

Public release date: 12-Jan-2009

Most heart attack patients' cholesterol levels did not indicate cardiac risk

A new national study has shown that nearly 75 percent of patients hospitalized for a heart attack had cholesterol levels that would indicate they were not at high risk for a cardiovascular event, according to current national cholesterol guidelines.

Specifically, these patients had low-density lipoprotein (LDL) cholesterol levels that met current guidelines, and close to half had LDL levels classified in guidelines as optimal (less than 100 mg/dL).

"Almost 75 percent of heart attack patients fell within recommended targets for LDL cholesterol, demonstrating that the current guidelines may not be low enough to cut heart attack risk in most who could benefit," said Dr. Gregg C. Fonarow, Eliot Corday Professor of Cardiovascular Medicine and Science at the David Geffen School of Medicine at UCLA and the study's principal investigator.

While the risk of cardiovascular events increases substantially with LDL levels above 40 mg/dL, current national cholesterol guidelines consider LDL levels less than 100 mg/dL acceptable for many individuals. The guidelines are thus not effectively identifying the majority of individuals who will develop fatal and non-fatal cardiovascular events, according to the study's authors.

Researchers also found that more than half of patients hospitalized for a heart attack had poor high-density lipoprotein (HDL) cholesterol levels, according to national guidelines.

Published in the January issue of the American Heart Journal, the study suggests that lowering guideline targets for LDL cholesterol for those at risk for cardiovascular disease, as well as developing better treatments to raise HDL cholesterol, may help reduce the number of patients hospitalized for heart attack in the future.

"The study gives us new insight and intervention ideas to help reduce the number of heart attacks," said Fonarow, who is also director of the Ahmanson-UCLA Cardiomyopathy Center.

"This is one of the first studies to address lipid levels in patients hospitalized for a heart attack at hospitals across the entire country."

The research team used a national database sponsored by the American Heart Association's Get with the Guidelines program. The database includes information on patients hospitalized for cardiovascular disease at 541 hospitals across the country.

Researchers analyzed data from 136,905 patients hospitalized for a heart attack nationwide between 2000 and 2006 whose lipid levels upon hospital admission were documented. This accounted for 59 percent of total hospital admissions for heart attack at participating hospitals during the study period.

Among individuals without any prior cardiovascular disease or diabetes, 72.1 percent had admission LDL levels less than 130 mg/dL, which is the current LDL cholesterol target for this population. Thus, the vast majority of individuals having their first heart attack would not have been targeted for effective preventative treatments based on the criteria used in the current guidelines.

The team also found that half of the patients with a history of heart disease had LDL cholesterol levels lower than 100 mg/dL, and 17.6 percent of patients had LDL levels below 70 mg/dL, which are guideline targets for LDL cholesterol in those at fair risk and at high risk for cardiovascular disease, respectively.

The study also showed that HDL cholesterol, or "good cholesterol," levels have dropped in patients hospitalized for heart attack over the past few years, possibly due to increasing rates of obesity, insulin resistance and diabetes.

Researchers found that 54.6 percent of patients had HDL levels below 40 mg/dL. Developing more effective treatments to boost HDL levels may help reduce the number of patients hospitalized for heart attacks, according to the authors.

"We found that less than 2 percent of heart attack patients had both ideal LDL and HDL cholesterol levels, so there is room for improvement," said Fonarow.

Fonarow said that only 59 percent of patients in the database had their lipid levels checked upon admission, which should be increased, since these early measurements can often help guide treatment decisions.

He also noted that only 21 percent of patients in the study were taking lipid-lowering medications before admission, despite almost half having a prior history of cardiovascular events, which would prompt treatment.

Ralphs Note - So how many people had heart attacks, with high cholesterol. Well lets see, if 75% heart attacks occurred with people that had cholesterol in the normal range. That would make those that had cholesterol out of range only 25% of the heart attacks. Hm m m m m seems safer to have high cholesterol, than normal, why?

Public release date: 13-Jan-2009

Misuse of Vicks VapoRub may harm infants and toddlers

Vicks® VapoRub®, the popular salve used to relieve symptoms of cough and congestion, may be harmful for infants and toddlers. New research appearing in the January issue of CHEST, the peer-reviewed journal of the American College of Chest Physicians (ACCP), shows that Vicks® VapoRub® (VVR) may stimulate mucus production and airway inflammation, which can have severe effects on breathing in an infant or toddler. Research findings are consistent with current VVR labeling which indicates the product should not be used on children under 2 years of age.

"The ingredients in Vicks can be irritants, causing the body to produce more mucus to protect the airway," said Bruce K. Rubin, MD, FCCP, the study's lead author from the Department of Pediatrics at Wake Forest University School of Medicine, Winston Salem, NC. "Infants and young children have airways that are much narrower than those of adults, so any increase in mucus or inflammation can narrow them more severely."

Dr. Rubin and his colleagues at Wake Forest became interested in the effects of VVR on small children after they cared for an 18-month-old girl who developed severe respiratory distress after VVR was put directly under her nose. The research team then launched an investigation to determine the effects of VVR on the respiratory system. Using ferrets, which have an airway anatomy and cellular composition similar to humans, the team conducted tests that measured the effects of VVR on mucus secretion and build up in the airways, and fluid build up in the lungs. Healthy ferrets and ferrets who had induced tracheal inflammation (simulating a person with a chest infection) underwent testing after they were exposed to VVR through intubation.

Results showed that in vitro VVR exposure increased mucus secretion 59 percent over baseline, while in vivo VVR exposure increased mucus secretion 14 percent in normal airways and 8 percent in the inflamed airway, in addition to the increase in secretion due to the inflammation. Mucus clearance, as measured by ciliary beat frequency in the trachea, also decreased by 36 percent during in vitro testing.

VVR is not indicated for patients under age 2. However, Dr. Rubin realizes that some parents are still choosing to use VVR to relieve their sick young child's symptoms, usually rubbing the salve on the feet or chest.

"I recommend never putting Vicks in, or under, the nose of anybody—adult or child. I also would follow

the directions and never use it at all in children under age 2," said Dr. Rubin. Even when directions are followed, VVR may make people with congestion feel more comfortable, but it does nothing to increase airflow or actually relieve congestion. "Some of the ingredients in Vicks, notably the menthol, trick the brain into thinking that it is easier to breathe by triggering a cold sensation, which is processed as indicating more airflow. Vicks may make you feel better but it can't help you breathe better." Dr. Rubin also feels that although the study only tested Vick's VapoRub, similar products, including generic brands, could cause the same adverse reaction in infants and toddlers.

In addition to VVR, decongestants are not recommended for young children; however, there are other treatments that are safe and effective.

"Cough and cold medicines and decongestants are dangerous and neither effective nor safe for young children. Medications to dry up nasal passages also have problems," said Dr. Rubin. "The best treatments for congestion are a bit of saline (salt water) and gentle rubber bulb suction, warm drinks or chicken soup, and, often, just letting the passage of time heal the child." Dr. Rubin also notes that if a child is struggling to breathe, it is a medical emergency and would require the child to be seen by a doctor as quickly as possible.

"Parents should consult with a physician before administering any over-the-counter medicine to infants and young children," said James A. L. Mathers, Jr., MD, FCCP, President of the American College of Chest Physicians. "Furthermore, the American College of Chest Physicians and several other health-care organizations have concluded that over-the-counter cough and cold medicines can be harmful for infants and young children and are, therefore, not recommended."

Ralph's Note - Thanks to one inquisitive man, many small lives will be saved.

Public release date: 13-Jan-2009

HHS Report Slams FDA's Conflict of Interest Oversight

By Emily P. Walker, Washington Correspondent, MedPage Today

WASHINGTON, Jan. 12 -- The FDA fails to ensure that scientists conducting clinical trials on investigational products disclose financial conflicts of interest, found a review by the Department of Health and Human Services.

An analysis by the Office of Inspector General (OIG), covering all 118 marketing applications for products approved by the FDA in 2007, showed that 42% were missing the required financial disclosures on the investigators.

"Financial relationships between researchers and medical companies may compromise the safety of human subjects and the integrity of research data," the OIG report said.

The report recommended that trial sponsors submit financial disclosures as part of the pre-trial process, rather than after the trial's completion.

For every study submitted to the FDA as part of a marketing application, the agency's regulations require sponsors to submit financial disclosure information on each investigator.

Sponsors have the option of not including financial information if they tried and failed to obtain it. The report found that more than one-quarter of the marketing applications used that "due diligence" exemption.

Most often, the sponsors said the investigators could not be located or failed to return the financial form.

For those marketing applications that disclosed financial conflicts of interest, FDA reviewers and sponsors failed to take action to remedy the conflict in 20% of all cases, the report said.

For about one-third of all marketing applications, FDA reviewers didn't even document that they checked the financial disclosure.

"If FDA reviewers fail to document a review, division directors may overlook disclosed financial interests and their potential impact on data integrity," the report said.

Even when disclosure forms were filed, the number reporting potential conflicts was surprisingly low.

The OIG's investigation revealed that among forms filed for 29,691 clinical investigators, only 206 -- less than 1% -- indicated at least one financial interest, contrasting sharply with independent estimates.

For example, a figure of one-quarter of academic researchers having financial ties to medical companies was published last year in the Journal of the American Medical Association, the report noted.

The OIG recommended that the FDA compile a central list of clinical investigators and their financial disclosures in order to accurately and easily look for conflicts of interest for future trials.

In addition, the FDA should check that sponsors have submitted all required attachment and financial forms along with their marketing applications, and it should update guidance on the due diligence exemption, the report said.

FDA spokeswoman Karen Riley said in a statement that the recommendations were reasonable, with one exception.

The agency opposes requiring investigator disclosures as part of the pre-trial process, she said.

"This recommendation could have the unintended effect of adding to the complexity and cost of the clinical trial enterprise with no commensurate gain in the protection of human subjects or the quality of the data," Riley said.

She argued that a pre-trial submission requirement could create significant unnecessary paperwork, since data from a given study "may never be submitted in support of a marketing application."

Riley said clinical investigators' financial incentives are only one type of bias that might exist in a trial. She said it has not been established that increasing the level or frequency of disclosure "would solve a recognized problem or enhance subject protection."

The agency takes at face value the OIG's finding that fewer than 1% of investigators reported potential conflicts, according to Riley.

On that basis, Riley said, as well as the fact that only 8% of products entering phase I testing eventually gain final approval, "restructuring FDA's review process to accommodate this recommendation does not seem warranted."

Additional source: Office of the Inspector General in the Department of Health and Human Services

Public release date: 13-Jan-2009

Smoking during pregnancy may impair thyroid function of mom and fetus

Chevy Chase, MD—Cigarette smoking during pregnancy is associated with potentially harmful changes in both maternal and fetal thyroid function, according to a new study accepted for publication in The Endocrine Society's Journal of Clinical Endocrinology & Metabolism (JCEM).

"We studied the influence of cigarette smoking on thyroid function of two groups of women at different stages of pregnancy – one in the first trimester and the other in the third trimester," said Dr. Bijay Vaidya, Ph.D., of Peninsula Medical School at Royal Devon and Exeter Hospital in the United Kingdom, and coauthor of the study. "In both groups we found that smoking during pregnancy is associated with changes in the mothers' thyroid hormone levels."

Optimal maternal thyroid function during pregnancy is vital for a successful pregnancy outcome, said Dr. Vaidya. The adverse outcomes associated with thyroid dysfunction during pregnancy include increased risk of miscarriage, premature birth, low birth weight and impaired neuropsychological development of the baby.

Dr. Vaidya and his colleagues also measured thyroid hormone levels in the umbilical cord of babies born to smoking mothers and found that smoking-related changes in thyroid function extend to the fetus. Dr. Vaidya believes that impaired thyroid function in the fetus could have potentially harmful biological consequences.

The study also found that in mothers who stopped smoking during pregnancy their thyroid hormone levels were comparable to levels found in non-smokers, which suggests that changes in thyroid function are rapidly reversible.

There is currently no definitive explanation for how smoking affects thyroid function, but Dr. Vaidya suggests that smoking may influence thyroid hormone levels by affecting the enzyme which converts the active form of thyroid hormone to an inactive form.

Public Release: 13-Jan-2009

Greater Quadriceps Strength May Benefit Those with Knee Osteoarthritis

Studies on the influence of quadriceps strength on knee osteoarthritis (OA), one of the leading causes of disability among the elderly, have shown conflicting results. In some studies, decreased quadriceps strength is associated with greater knee pain and impaired

function, while other studies show mixed results on the effect of quadriceps strength on the structural progression of knee OA.

Most studies to date have used X-rays to indirectly measure cartilage loss in knee OA and have focused on the tibiofemoral joint (the main joint in the knee where the thigh and shin bones meet). A new study has examined the effect of quadriceps strength on cartilage loss (measured using magnetic resonance imaging [MRI]) at both the tibiofemoral joint and the patellofemoral joint (where the thigh bone and knee cap meet) as well as on knee OA symptoms. The study was published in the January issue of *Arthritis & Rheumatism* (<http://www3.interscience.wiley.com/journal/76509746/home>).

Led by Shreyasee Amin, M.D., M.P.H., of the Mayo Clinic, the study involved 265 men and women participating in a 30-month study of symptomatic knee OA. At the beginning of the study, participants underwent MRI of their more painful knee and measurement of quadriceps strength for the same knee. They were also asked to rate the severity of their knee pain and their physical function was assessed. The knee MRI and assessments of their knee OA symptoms were repeated at 15 and 30 months. A measurement of knee alignment was also performed.

The results showed that greater quadriceps strength had no influence on cartilage loss at the tibiofemoral joint even in those with knees that were out of alignment. However, stronger quadriceps were shown to protect against cartilage loss in the lateral compartment (outer part) of the patellofemoral joint, a site of frequent cartilage loss, pain and disability in patients with knee OA. The study also showed that those with the greatest quadriceps strength had less knee pain and better physical function than those with the least strength.

Previous studies had also shown no overall protective effect of greater quadriceps strength on cartilage loss at the tibiofemoral joint. The protective effect against cartilage loss at the lateral compartment of the patellofemoral joint is a new finding that needs to be confirmed in future studies, but does provide evidence as to the benefit of having strong quadriceps muscles in patients with knee OA. “Our findings, which also include an association of greater quadriceps strength with less knee pain and physical limitation over followup, suggest that greater quadriceps strength has an overall beneficial effect on symptomatic knee OA,” the authors state. This effect may be due to a strengthening of the vastus medialis obliquus (a quadriceps muscle that pulls the kneecap inward), that may stabilize the kneecap and help prevent cartilage loss behind part of the knee cap.

Although the study did not involve exercise training to strengthen the quadriceps, there have been several short-term studies that show that improving quadriceps strength has a beneficial effect on knee pain and function. “While our findings suggest that maintaining

strong quadriceps is of benefit to those with knee OA, further work is needed to determine the type and frequency of exercise regimen that will be both safe and effective," the authors conclude.

Public release date: 16-Jan-2009

Seasonal variation in blood pressure

A French study reported in the 12th January issue of Archives of Internal Medicine has found a strong correlation between blood pressure and outdoor temperature in a large sample of the elderly.(1) As a result, the investigators advise that, during periods of extreme temperatures, careful monitoring of blood pressure and antihypertensive treatment "could contribute to reducing the consequences of blood pressure variations in the elderly".

The study, which monitored 8801 participants over the age of 65 in the French Three-City study, found that systolic and diastolic blood pressure values differed significantly across the four seasons of the year and according to the distribution of outdoor temperature. The higher the temperature, the greater the decrease in blood pressure. Systolic blood pressure, for example, decreased with increasing temperature, with an 8.0 mmHg decrease between the lowest (< 7.9°C) and the highest (21.2°C) temperatures. Average systolic blood pressure was 5 mmHg higher in winter than in summer. **High blood pressure, defined as a systolic blood pressure of 160 mmHg or higher, or a diastolic blood pressure of 95 mmHg or higher, was detected in 33.4 per cent of participants during winter and 23.8 percent during summer.** These changes in blood pressure were greater in subjects 80 years or older than in younger participants.

Participants' blood pressure was measured at the beginning of the study (starting in 1999) and again about two years later. Outdoor temperatures on the day of measurement were obtained from local meteorological offices. Participants in the Three-City study were from Bordeaux, Dijon and Montpellier.

"Although our study does not demonstrate a causal link between blood pressure and external temperature, the observed relationship nevertheless has potentially important consequences for blood pressure management in the elderly," the authors state. "Because the risk of stroke or aneurysmal rupture is highest in the elderly, improved protection against these diseases by close monitoring of blood pressure and antihypertensive medication when outdoor temperature is very low could be considered."

Speaking on behalf of the European Society of Cardiology (ESC), Professor Frank Ruschitzka from the University Hospital, Zurich, says that the study reaffirms the place of the elderly as a target group for blood pressure monitoring. "The elderly, especially the increasing number of octogenarians, should not be neglected. They need extra care, and will benefit from monitoring and appropriate treatment. This study emphasises the need for year-round vigilance."

One possible explanation for the study findings, adds Professor Ruschitzka, lies in the emerging link between vitamin D and blood pressure. The elderly, especially those in care homes, are subject to vitamin D deficiency, largely as a result of their limited exposure to sunlight, and vitamin D deficiency can predispose to hypertension via activation of the renin-angiotensin-aldosterone system. "The benefit of sunlight on vitamin D levels in the elderly is under appreciated," says Professor Ruschitzka. "Fifteen minutes exposure to sunlight can produce the equivalent of 2000 international units vitamin D."

A report from the Framingham Heart Study published in 2008 found that moderate vitamin D deficiency nearly doubles the risk of myocardial infarction, stroke and heart failure over a mean of 5.4 years in patients with high blood pressure.(2) The Nurses Health Study, also reporting in 2008, found that lower blood levels of vitamin D are independently associated with an increased risk of hypertension; women with the lowest levels had a 66 per cent higher incidence of hypertension than those with the highest levels.(3)

Ralph's note - Keep in mind that different countries have different ideas of where High Blood Pressure begins.

Public release date: 16-Jan-2009

Progress made in understanding causes and treatment of endometriosis

CHICAGO --- Endometriosis is a poorly understood chronic disease characterized by infertility and chronic pelvic pain during intercourse. It affects between 5 to 10 million women in the U.S.

Serdar Bulun, M.D., George H. Gardner Professor of Clinical Gynecology at Northwestern University's Feinberg School of Medicine, has spent the past 15 years investigating and identifying the causes of this disease. Bulun, and colleagues in his lab, discovered key epigenetic abnormalities in endometriosis and identified existing chemicals that now help treat it.

Bulun describes his lab's findings over the past 10 years in the Jan. 15 issue of the New England Journal of Medicine.

One of the abnormalities he discovered is the presence of the enzyme aromatase -- which produces estrogen -- in endometriosis, the diseased tissue that exists on pelvic organs and mimics the uterine lining. (Normal endometrium, located in the uterine cavity, does not contain aromatase.) As a result, women with endometriosis have excessive estrogen in this abnormal tissue found on surfaces of pelvic organs such as the ovaries. Bulun found the protein SF1 that produces aromatase, which is supposed to be shut down, is active in endometriosis.

"Estrogen is like fuel for fire in endometriosis," Bulun said. "It triggers

the endometriosis and makes it grow fast."

As a result of the aromatase finding, Bulun launched clinical trials in 2004 and 2005 testing aromatase inhibitors -- currently used in breast cancer treatment -- for women with endometriosis. The drug blocks estrogen formation and secondarily improves progesterone responsiveness.

"We came up with a new treatment of choice for post-menopausal women with endometriosis," Bulun said. Moreover, treatment with an aromatase inhibitor is a very good option for premenopausal women with endometriosis not responding to existing treatments, he noted.

Another molecular abnormality Bulun found is that women with endometriosis have a progesterone receptor that is inappropriately turned off. Normal progesterone action would be beneficial because it blocks the growth of endometriosis. In the absence of appropriate progesterone action, endometriosis tissue remains inflamed and continues to grow.

Bulun believes that these abnormalities result from epigenetic defects that occur very early on during embryonic development and may be the result of early exposure to environmental toxins. In fact, other investigators have implicated the environmental pollutant dioxin and the synthetic estrogen DES in the etiology of endometriosis.

"This may be a disease that women are born with," Bulun said. "Perhaps when a baby girl is born, it has already been determined that she is predisposed to have endometriosis. Maybe research can now be directed toward the fetal origins of the disease and raise the awareness of how the disease develops."

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Study links water pollution with declining male fertility

New research strengthens the link between water pollution and rising male fertility problems. The study, by Brunel University, the Universities of Exeter and Reading and the Centre for Ecology & Hydrology, shows for the first time how a group of testosterone-blocking chemicals is finding its way into UK rivers, affecting wildlife and potentially humans. The research was supported by the Natural Environment Research Council and is now published in the journal *Environmental Health Perspectives*.

The study identified a new group of chemicals that act as 'anti-androgens'. This means that they inhibit the function of the male hormone, testosterone, reducing male fertility. Some of these are contained in medicines, including cancer treatments, pharmaceutical treatments, and pesticides used in agriculture. The research suggests that when they get into the water system, these chemicals may play a pivotal role in causing feminising effects in male fish.

Earlier research by Brunel University and the University of Exeter has shown how female sex hormones (estrogens), and chemicals that mimic estrogens, are leading to 'feminisation' of male fish. Found in some industrial chemicals and the contraceptive pill, they enter rivers via sewage treatment works. This causes reproductive problems by reducing fish breeding capability and in some cases can lead to male fish changing sex.

Other studies have also suggested that there may be a link between this phenomenon and the increase in human male fertility problems caused by testicular dysgenesis syndrome. Until now, this link lacked credence because the list of suspects causing effects in fish was limited to estrogenic chemicals whilst testicular dysgenesis is known to be caused by exposure to a range of anti-androgens.

Lead author on the research paper, Dr Susan Jobling at Brunel University's Institute for the Environment, said: "We have been working intensively in this field for over ten years. The new research findings illustrate the complexities in unravelling chemical causation of adverse health effects in wildlife populations and re-open the possibility of a human – wildlife connection in which effects seen in wild fish and in humans are caused by similar combinations of chemicals. We have identified a new group of chemicals in our study on fish, but do not know where they are coming from. A principal aim of our work is now to identify the source of these pollutants and work with regulators and relevant industry to test the effects of a mixture of these chemicals and the already known environmental estrogens and help protect environmental health."

Senior author Professor Charles Tyler of the University of Exeter said: "Our research shows that a much wider range of chemicals than we previously thought is leading to hormone disruption in fish. This means that the pollutants causing these problems are likely to be coming from a wide variety of sources. Our findings also strengthen the argument for the cocktail of chemicals in our water leading to hormone disruption in fish, and contributing to the rise in male reproductive problems. There are likely to be many reasons behind the rise in male fertility problems in humans, but these findings could reveal one, previously unknown, factor."

Bob Burn, Principal Statistician in the Statistical Services Centre at the University of Reading, said: "State-of-the-art statistical hierarchical modelling has allowed us to explore the complex associations between the exposure and potential effects seen in over 1000 fish sampled from 30 rivers in various parts of England."

The research took more than three years to complete and was conducted by the University of Exeter, Brunel University, University of Reading and the Centre for Ecology & Hydrology. Statistical modelling was supported by Beyond the Basics Ltd.

The research team is now focusing on identifying the source of anti-androgenic chemicals, as well as continuing to study their impact on reproductive health in wildlife and humans.

Public release date: 20-Jan-2009

Low-carbohydrate diet burns more excess liver fat than low-calorie diet, UT Southwestern study finds

DALLAS – Jan. 20, 2009 – People on low-carbohydrate diets are more dependent on the oxidation of fat in the liver for energy than those on a low-calorie diet, researchers at UT Southwestern Medical Center have found in a small clinical study.

The findings, published in the journal *Hepatology*, could have implications for treating obesity and related diseases such as diabetes, insulin resistance and nonalcoholic fatty liver disease, said Dr. Jeffrey Browning, assistant professor in the UT Southwestern Advanced Imaging Research Center and of internal medicine at the medical center.

"Instead of looking at drugs to combat obesity and the diseases that stem from it, maybe optimizing diet can not only manage and treat these diseases, but also prevent them," said Dr. Browning, the study's lead author.

Although the study was not designed to determine which diet was more effective for losing weight, the average weight loss for the low-calorie dieters was about 5 pounds after two weeks, while the low-carbohydrate dieters lost about 9½ pounds on average.

Glucose, a form of sugar, and fat are both sources of energy that are metabolized in the liver and used as energy in the body. Glucose can be formed from lactate, amino acids or glycerol.

In order to determine how diet affects glucose production and utilization in the liver, the researchers randomly assigned 14 obese or overweight adults to either a low-carbohydrate or low-calorie diet and monitored seven lean subjects on a regular diet.

After two weeks, researchers used advanced imaging techniques to analyze the different methods, or biochemical pathways, the subjects used to make glucose.

"We saw a dramatic change in where and how the liver was producing glucose, depending on diet," said Dr. Browning.

Researchers found that participants on a low-carbohydrate diet produced more glucose from lactate or amino acids than those on a low-calorie diet.

"Understanding how the liver makes glucose under different dietary conditions may help us better regulate metabolic disorders with diet," Dr. Browning said.

The different diets produced other differences in glucose metabolism. For example, people on a low-calorie diet got about 40 percent of their glucose from glycogen, which is comes from ingested carbohydrates and is stored in the liver until the body needs it.

The low-carbohydrate dieters, however, got only 20 percent of their glucose from

glycogen. Instead of dipping into their reserve of glycogen, these subjects burned liver fat for energy.

The findings are significant because the accumulation of excess fat in the liver – primarily a form of fat called triglycerides – can result in nonalcoholic fatty liver disease, or NAFLD. The condition is the most common form of liver disease in Western countries, and its incidence is growing. Dr. Browning has previously shown that NAFLD may affect as many as one-third of U.S. adults. The disease is associated with metabolic disorders such as insulin resistance, diabetes and obesity, and it can lead to liver inflammation, cirrhosis and liver cancer.

"Energy production is expensive for the liver," Dr. Browning said. "It appears that for the people on a low-carbohydrate diet, in order to meet that expense, their livers have to burn excess fat."

Results indicate that patients on the low-carbohydrate diet increased fat burning throughout the entire body.

Dr. Browning and his colleagues will next study whether the changes that occur in liver metabolism as a result of carbohydrate restriction could help people with nonalcoholic fatty liver disease. Previous research has shown a correlation between carbohydrate intake and NAFLD.

Public release date: 20-Jan-2009

Genes remember sugar hit: Australian research

SYDNEY (AFP) – Human genes remember a sugar hit for two weeks, with prolonged **poor eating habits capable of permanently altering DNA**, Australian research has found.

A team studying the impact of diet on human heart tissue and mice found that cells **showed the effects of a one-off sugar hit for a fortnight, by switching off genetic controls designed to protect the body against diabetes and heart disease.**

"We now know that chocolate bar you had this morning can have very acute effects, and those effects can continue for up to two weeks," said lead researcher Sam El-Osta, from the Baker IDI Heart and Diabetes Institute.

"These changes continue beyond the meal itself and have the ability to alter natural metabolic responses to diet," he told Australian Associated Press Friday.

Regular poor eating would amplify the effect, said El-Osta, with genetic damage lasting months or years, and potentially passing through bloodlines.

The study's findings were reported in the Journal of Experimental Medicine.

**These reports are done with the appreciation of all the Doctors, Scientist, and other Medical Researchers who sacrificed their time and effort. In order to give people the ability to empower themselves. Without the base aspirations for fame, or fortune.
Just honorable people, doing honorable things.**