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Human Technology Research Synopsis

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Editors Top Five:

1. No longer a gray area: Our hair bleaches itself as we grow older
2. Vitamin supplements may protect against noise-induced hearing loss
3. Vitamin B and folic acid may reduce risk of age-related vision loss
4. Broccoli may help protect against respiratory conditions like asthma
5. Cholesterol-reducing drugs may lessen brain function, says ISU researcher

In this issue:

1. Can exercising your brain prevent memory loss?
2. Type of rheumatoid arthritis medication may be associated with increased risk for shingles
3. Research identifies how inflammatory disease causes fatigue
4. Taurine: Key to the visual toxicity of an anti-epileptic drug for children?
5. Vitamin supplements may protect against noise-induced hearing loss
6. Questions of ethics and quality cloud globalization of clinical trials
7. Can breastfeeding reduce multiple sclerosis relapses?
8. When should prostate-specific antigen testing be stopped?
9. Questions of ethics and quality cloud globalization of clinical trials
10. Green, black tea can reduce stroke risk
11. Chili peppers help to unravel the mechanism of pain
12. Vitamin D deficiency may increase risk of colds, flu
13. Vitamin B and folic acid may reduce risk of age-related vision loss
14. No longer a gray area: Our hair bleaches itself as we grow older
15. Cholesterol-reducing drugs may lessen brain function, says ISU researcher

16. Physical fitness improves spatial memory, increases size of brain structure
17. Vegetable-based drug could inhibit melanoma
18. Vitamin A signals offer clues to treating autoimmunity
19. Low levels of vitamin B12 may increase risk for neural tube defects
20. Broccoli may help protect against respiratory conditions like asthma
21. Doctors endorse vegan and vegetarian diets for healthy pregnancies
22. Cleansing toxic waste -- with vinegar
23. Are vitamin supplements effective in celiac disease patients? (Yes They Are)

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Can exercising your brain prevent memory loss?

SEATTLE – Participating in certain mental activities, like reading magazines or crafting in middle age or later in life, may delay or prevent memory loss, according to a study released today that will be presented at the American Academy of Neurology's 61st Annual Meeting in Seattle, April 25 to May 2, 2009.

The study involved 197 people between the ages of 70 and 89 with mild cognitive impairment, or diagnosed memory loss, and 1,124 people that age with no memory problems. Both groups answered questions about their daily activities within the past year and in middle age, when they were between 50 to 65 years old.

The study found that during later years, reading books, playing games, participating in computer activities and doing craft activities such as pottery or quilting led to a 30 to 50 percent decrease in the risk of developing memory loss compared to people who did not do those activities. **People who watched television for less than seven hours a day in later years were 50 percent less likely to develop memory loss than people who watched for more than seven hours a day.**

People who participated in social activities and read magazines during middle age were about 40 percent less likely to develop memory loss than those who did not do those activities.

"This study is exciting because it demonstrates that aging does not need to be a passive process. By simply engaging in cognitive exercise, you can protect against future memory loss," said study author Yonas Geda, MD, MSc, a neuropsychiatrist at Mayo Clinic in Rochester, MN, and a member of the American Academy of Neurology. "Of course, the challenge with this type of research is that we are relying on past memories of the participants, therefore, we need to confirm these findings with additional research."

Ralph's Note - OMG TV does rot the brain..

Public release date: 17-Feb-2009

Type of rheumatoid arthritis medication may be associated with increased risk for shingles

Use of certain medications known as monoclonal anti-tumor necrosis factor α (TNF- α) antibodies for the treatment of rheumatoid arthritis appears to be associated with an increased risk for herpes zoster (shingles), the painful infection characterized by blisters, according to a study in the February 18 issue of JAMA.

There has been evidence from some studies that patients treated with anti-TNF- α agents are at an increased risk of bacterial infections, but little is known about the risk of viral infections, such as herpes zoster, in patients with rheumatoid arthritis receiving these types of medications. Herpes zoster is one of the most common adverse events reported in clinical trials of anti-TNF- α agents, according to background information in the article. Patients with rheumatoid arthritis are at increased risk of herpes zoster compared with the general population.

Anja Strangfeld, M.D., of the German Rheumatism Research Center, Berlin, and colleagues investigated the association of various rheumatoid arthritis treatments, including anti-TNF- α therapy, with the risk of herpes zoster. The researchers analyzed data from patients who began treatment with adalimumab or infliximab (monoclonal anti-TNF- α antibodies), etanercept (a fusion protein), the monotherapeutic agent anakinra, or when patients changed conventional disease-modifying anti-rheumatic drug (DMARD). Treatment, clinical status and adverse events were assessed by rheumatologists at fixed points during follow-up (of up to three years). A total of 5,040 patients were included in the analysis.

There were 86 cases of herpes zoster among 82 patients. Thirty-nine occurrences could be attributed to treatment with anti-TNF- α antibodies (23 to etanercept, 24 to conventional DMARDs). **The researchers found a significant association between herpes zoster and treatment with the monoclonal anti-TNF- α antibodies infliximab and adalimumab, although this risk was lower than the threshold for clinical significance. There was no significant association between herpes zoster and treatment with etanercept, or anti-TNF- α treatment as a class.**

A significantly higher risk of developing herpes zoster was found for patients of older age and for treatment with glucocorticoids (steroid hormones that are widely used as anti-inflammatory medications).

"Based on our data, we recommend careful monitoring of patients treated with monoclonal anti-TNF- α antibodies for early signs and symptoms of herpes zoster," the authors conclude.

Public release date: 17-Feb-2009

Research identifies how inflammatory disease causes fatigue

Findings confirm that immune cells can enter the brain and suggest new communication pathway between diseased organs and nervous system

New animal research in the February 18 issue of The Journal of Neuroscience may indicate how certain diseases make people feel so tired and listless. Although the brain is usually isolated from the immune system, the study suggests that certain behavioral changes suffered by those with chronic inflammatory diseases are caused by the infiltration of immune cells into the brain. The findings suggest possible new treatment avenues to improve patients' quality of life.

Chronic inflammatory diseases like rheumatoid arthritis, inflammatory bowel disease, psoriasis, and liver disease cause "sickness behaviors," including fatigue, malaise, and loss of social interest. However, it has been unclear how inflammation in other organs in the body can impact the brain and behavior.

The researchers found that in mice with inflamed livers, white blood cells called monocytes infiltrated the

brain. These findings support previous research demonstrating the presence of immune cells in the brain following organ inflammation, challenging the long-held belief that the blood-brain barrier prevents immune cells from accessing the brain.

"Using an experimental model of liver inflammation, our group has demonstrated for the first time the existence of a novel communication pathway between the inflamed liver and the brain," said the study's senior author Mark Swain, MD, Professor of Medicine at the University of Calgary.

Swain and his colleagues found that liver inflammation triggered brain cells called microglia to produce CCL2, a chemical that attracts monocytes. When the researchers blocked CCL2 signaling, monocytes did not enter the brain despite ongoing inflammation in the liver.

Liver inflammation also stimulated cells in the blood to make an immune chemical (TNF α). When the researchers blocked the signaling of this immune chemical, microglia produced less CCL2, and monocytes stayed out of the brain.

In the mice with inflamed livers, preventing the entry of monocytes into the brain reduced sickness behaviors; mice showed more mobility and social interaction. These findings suggest that people with chronic inflammatory diseases may benefit from treatments that limit monocyte access to the brain.

"Sickness behavior significantly impacts quality of life. Our findings further our understanding and may generate potential new avenues for treatment of these often crippling symptoms," said Swain.

"The brain is the master coordinator of many of our bodies' defense responses, so it must be able to sense injury and inflammation in distant body organs. This study starts to explain the peripheral communication signals that activate the brain," said Nancy Rothwell, PhD, DSc, at the University of Manchester, an expert on brain inflammation who is unaffiliated with the study.

Public release date: 17-Feb-2009

Taurine: Key to the visual toxicity of an anti-epileptic drug for children?

Vigabatrin (Sabril), first intention molecule for the treatment of epilepsy in children, in many cases produces secondary effects that lead to an irreversible loss of vision. Serge Picaud, head of research at Inserm, and his colleagues of the Institut de la Vision have just discovered the origin of this secondary effect and have proposed strategies for limiting it. **They have shown that vigabatrin provokes a marked decrease in the blood level in an amino acid, taurine, resulting in a degeneration of the retina cells induced by light.** The researchers therefore suggest that exposure to light should be reduced **and a taurine-rich diet introduced in order to curb immediately these secondary effects in children undergoing treatment.** As for the validation of an alternative treatment associating vigabatrin and taurine, this will necessitate several years of development.

This work is published in the review *Annals of Neurology*.

Epilepsy affects 1% of the world's population. With children, its treatment remains extremely restricted, and vigabatrin, (marketed in France under the name of Sabril®), has obtained marketing authorisation for children aged under 2 years. This anticonvulsant, which is also administered to adults in the case of failure of other treatments, is at the same time now being evaluated for the treatment of addiction to heroin, cocaine and methamphetamines.

However, the serious secondary effects of this drug can induce an impairment of the retina and a restriction of the visual field, noted, depending on the studies, in 10% to 40% of patients.

In order to reach an understanding of this drug's modes of actions, and in particular the mechanism of visual function impairment, the Inserm researchers first of all administered vigabatrin to rats over a period of several months and analysed the influence of exposure to light during the treatment. The results show that there is no damage to the retina when the animals are kept in the dark throughout the treatment.

Moreover, since previous work had shown that a deficiency of the organism in taurine (amino acid) triggers the degeneration of the photoreceptors (cells of the retina converting light into nervous signals), the researchers measured, in rodents, the plasma level of 19 amino acids. Whereas the concentration was identical for most of the amino acids in animals under vigabatrin and in non-treated rats, the taurine level turned out to be 67% lower in treated animals

Taurine is essentially contributed by diet. By providing certain of the animals under treatment with a taurine supplementation, the researchers noted that their visual acuity was greater than that of the animals without supplementation. In addition, the amino acid doses administered to six children subject to regular attacks of epilepsy and treated under vigabatrin reveal a taurine level that is far below the normal values reported for children of the same age – and in some cases even undetectable.

On the strength of these various tests, the scientists were able to prove that vigabatrin induces a pronounced reduction of the taurine level in the plasma. This marked fall is responsible for the degeneration of the photoreceptors and thus for the retinal toxicity in the animals exposed to light.

Pending confirmation in the human of the interest of providing patients under vigabatrin with a taurine supplementation, the researchers propose immediate solutions designed to limit the secondary effects in these patients. "In the first instance, care should therefore be taken to ensure that patients under vigabatrin consume a sufficient amount of food containing taurine. It is also important that they should be exposed to as little light as possible (e.g.: no night lights in a baby's bedroom at night) and should be induced to wear sunglasses", says Serge Picaud.

The researchers also emphasise that any taurine supplementation must be subject to medical advice.

Public release date: 17-Feb-2009

Vitamin supplements may protect against noise-induced hearing loss

GAINESVILLE, Fla. — Vitamin supplements can prevent hearing loss in laboratory animals, according to two new studies, bringing investigators one step closer to the development of a pill that could stave off noise-induced and perhaps even age-related hearing loss in humans.

The findings will be reported Wednesday at the Association for Research in Otolaryngology's annual conference in Baltimore by senior author Colleen Le Prell, Ph.D., a researcher at the University of Florida.

The supplements used in the research studies are composed of antioxidants — beta carotene and vitamins C and E — and the mineral magnesium. When administered prior to exposure to loud noise, the supplements prevented both temporary and permanent hearing loss in test animals.

"What is appealing about this vitamin 'cocktail' is that previous studies in humans, including those demonstrating successful use of these supplements in protecting eye health, have shown that supplements of these particular vitamins are safe for long-term use," said Le Prell, an associate professor in the UF College of Public Health and Health Professions' department of communicative disorders.

About 26 million Americans have noise-induced hearing loss, according to the National Institute on Deafness and Other Communication Disorders, the agency that funded the studies.

In the first study, UF, University of Michigan and OtoMedicine scientists gave guinea pigs the vitamin supplements prior to a four-hour exposure to noise at 110 decibels, similar to levels reached at a loud

concert. Researchers assessed the animals' hearing by measuring sound-evoked neural activity and found that the treatment successfully prevented temporary hearing loss in the animals.

In humans, temporary noise-induced hearing loss, often accompanied by ringing in the ears, typically goes away after a few hours or days as the cells in the inner ear heal. Because repeated temporary hearing loss can lead to permanent hearing loss, the scientists speculate that prevention of temporary changes may ultimately prevent permanent changes.

In the second, related study in mice, UF, Washington University in St. Louis and OtoMedicine researchers showed that the supplements prevented permanent noise-induced hearing loss that occurs after a single loud sound exposure. The researchers found that the supplements prevented cell loss in an inner ear structure called the lateral wall, which is linked to age-related hearing loss, leading the scientists to believe these micronutrients may protect the ear against age-related changes in hearing.

"I am very encouraged by these results that we may be able to find a way to diminish permanent threshold shift with noise exposure," said Debara Tucci, M.D., an associate professor of surgery in the otolaryngology division at Duke University Medical Center. "I look forward to hearing Dr. Le Prell's work and reviewing her data."

The research builds on previous studies that demonstrated hearing loss is not just caused by intense vibrations produced by loud noises that tear the delicate structures of the inner ear, as once thought, said Josef Miller, Ph.D., who has studied the mechanisms of hearing impairment for more than 20 years and is a frequent collaborator of Le Prell's. Researchers now know noise-induced hearing loss is largely caused by the production of free radicals, which destroy healthy inner ear cells.

"The free radicals literally punch holes in the membrane of the cells," said Miller, the Townsend professor of communicative disorders at the University of Michigan.

Miller is the co-founder of OtoMedicine, a University of Michigan spinoff company that has patented AuraQuell, the vitamin supplement formula used in the studies.

The antioxidant vitamins prevent hearing damage by "scavenging" the free radicals. Magnesium, which is not a traditional antioxidant, is added to the supplement mix to preserve blood flow to the inner ear and aid in healing.

Antioxidant supplements can also provide "post-noise rescue," Le Prell said. A previous study by Le Prell and Miller showed that antioxidants can protect hearing days after exposure to loud noise.

"We found that the antioxidant combination of vitamin E and salicylate — the active agent in aspirin — effectively prevented cell death and permanent noise-induced hearing loss even when treatments were delayed up to three days after noise insult," she said.

The researchers are collaborating on National Institutes of Health-funded clinical trials of the vitamin supplements in college students at UF who wear MP3 music players, and noise-exposed military troops and factory workers in Sweden and Spain.

If the trials show that the vitamins are as effective in preventing noise-induced hearing loss in humans as they have been in animals, Le Prell and Miller envision an easy-to-use supplement that could come in the form of a pill for people headed to a rock concert, a daily supplement for factory workers or a nutritional bar included in soldiers' rations.

"Ear protection, such as ear plugs, is always the best practice for the prevention of noise-induced hearing loss, but in those populations who don't or can't wear hearing protection, for people in which mechanical devices just aren't enough, and for people who may experience unexpected noise insult, these supplements could provide an opportunity for additional protection," Le Prell said.

Public release date: 18-Feb-2009

Questions of ethics and quality cloud globalization of clinical trials

DURHAM, N.C. – Top-tier U.S.-based pharmaceutical companies are moving their clinical trials overseas at warp speed, raising questions about ethics, quality control, and even the scientific value of their findings for people back in the U.S.

Many of the trials are taking place in developing countries in Eastern Europe and Asia where study participants are often poorer and less educated than are study participants in the U.S., according to researchers at Duke Clinical Research Institute (DCRI).

"The FDA is supposed to provide oversight for such trials, but it simply wasn't designed to handle this kind of situation," says Kevin Schulman, M.D., the senior author of the report appearing in the New England Journal of Medicine. Schulman **says the number of Food and Drug Administration investigators based outside the U.S. has grown by 15 percent every year since 2002, while the number of U.S.-based investigators has fallen just over 5 percent during the same period.**

Schulman and a research team led by Seth Glickman, M.D., a senior scholar at Duke's Fuqua School of Business, used the clinicaltrials.gov registry to examine recruitment patterns in industry-sponsored Phase 3 trials in 2007. Phase 3 trials are typically the largest and most meaningful trials, often involving thousands of patients. They found that about a third of the trials (157 of 509) were being solely conducted outside the U.S. They also discovered that over half the study sites (13,521 of 24,206) lay outside U.S. borders.

Researchers also reviewed 300 articles reporting clinical trial results appearing in the New England Journal of Medicine, the Journal of the American Medical Association and the Lancet in 1995 and 2005 and found that over that decade, the number of clinical trial sites abroad doubled, while the number in the U.S. and Western Europe declined.

"There are powerful forces luring clinical trials overseas, including the lower cost of doing business and access to larger study populations," says Glickman. "The cost per participant in a clinical trial in India, for example, can be only one-tenth of what it is in the U.S."

"But there are equally powerful forces pushing," says Schulman, who adds that a mix of well-intentioned policy efforts is actually creating barriers to conducting research in a timely fashion in the U.S.

The authors say some of the clinical trials abroad are raising concern about aligning research to the health needs of the population under study. "It's pretty clear that companies are testing drugs in countries where they will not be marketed or sold," says Glickman. "This is a major ethical concern." The researchers found plenty of examples where companies were testing drugs for conditions such as allergic rhinitis, fibromyalgia and overactive bladders in emerging markets – rather than treatments for diseases like malaria or tuberculosis that might be more prevalent there.

Glickman says social ecology and genetics may also play a role in trial outcomes, and may limit their applicability to patients who do not share such characteristics. For example, healthcare-rich economies produce patients with one set of characteristics, while participants with little access to healthcare may have quite different profiles. "It is conceivable that use of the same drug in both populations would produce markedly different results." Likewise, genetic polymorphisms, or "signatures" in some populations can affect response to certain drugs, making it inappropriate to apply results from studies in these populations to patients who do not share those characteristics.

"Clearly, there is some benefit for everyone involved in clinical trials overseas," says Glickman.

"Generally, such trials increase local prosperity, education and access to better healthcare. And it is critical that new drugs and devices be tested in diverse populations. In order to for that to properly continue, however, we need a robust research framework that will protect trial participants and ensure that sponsors adhere to the highest ethical standards."

The authors say a body such as the Institute of Medicine or the World Health Organization could create an international commission that would bring industry, academia, regulatory agencies and patient advocacy groups to the same table. "The future of the pharmaceutical and device industries is predicated on addressing these issues," they write.

Public release date: 19-Feb-2009

Can breastfeeding reduce multiple sclerosis relapses?

SEATTLE – Women who have multiple sclerosis may reduce their risk of relapses after pregnancy if they breastfeed their babies, according to a study released today that will be presented at the American Academy of Neurology's 61st Annual Meeting in Seattle, April 25 to May 2, 2009.

For the study, researchers followed 32 pregnant women with MS and 29 pregnant women without MS during each trimester and up to a year after they gave birth. The women were interviewed about their breastfeeding and menstrual period history.

A total of 52 percent of the women with MS did not breastfeed or began supplemental formula feedings within two months of giving birth. **Of those, 87 percent had a relapse after pregnancy compared to 36 percent of women with MS who breastfed exclusively for at least two months after pregnancy.**

Sixty percent of the women reported their main reason for not breastfeeding exclusively was to start taking MS treatments again. Women who began taking MS treatments within the first two months after giving birth had significantly higher risk of suffering a relapse than women with MS who did not start taking medications early, regardless of whether they breastfed. Those who breastfed exclusively got their menstrual periods back later than the women who did not breastfeed or began early supplemental feedings.

"Our findings call into question the benefit of choosing not to breastfeed or stopping breastfeeding early in order to start taking MS therapies," said study author Annette Langer-Gould, MD, PhD, of Stanford University in California, and a member of the American Academy of Neurology. "Larger studies need to be done on whether women should delay taking MS medications in order to breastfeed."

Ralph's Note - Ironic

Public release date: 20-Feb-2009

When should prostate-specific antigen testing be stopped?

New York, NY, February 20, 2009 – Although widespread Prostate-Specific-Antigen (PSA) testing has undoubtedly decreased prostate cancer mortality, is there a point of diminishing returns? In a study published in the April 2009 issue of **The Journal of Urology**, researchers found that in a subgroup of elderly men, among those who were 75 years old or older and had a PSA below 3 ng/ml (nanograms per milliliter), none subsequently died of prostate cancer. The discontinuation of routine PSA screening in these men may not increase the rates of undetected lethal disease, and could avoid potentially

unnecessary treatments and reduce diagnostic costs.

Because PSA screening can find cancers that may become life-threatening in 5 to 25 years, there has been increased usage of the test in 40 to 50-year-olds. But the test can also discover cancers that never become life-threatening, perhaps in up to 30% of the cases. Many men who are older than 75 undergo continued PSA screening, potentially leading to unnecessary treatment since death from other causes is more likely than death from prostate cancer.

The study conducted by investigators from the Baltimore Longitudinal Study of Aging (National Institute on Aging, National Institutes of Health) and the Department of Urology at Johns Hopkins School of Medicine involved 849 men (122 with and 727 without prostate cancer) with serial PSA measurements. Researchers found that for men over 75 with PSA < 3ng/ml, none died of prostate cancer and only one developed high-risk prostate cancer. In contrast, men of all ages with a PSA \geq 3.0 ng/ml had a continually rising probability of death from prostate cancer.

Writing in the article, Edward M. Schaeffer states, "The optimal approach to prostate cancer screening remains controversial. To date, there is limited evidence from which to inform the decision on when to discontinue prostate cancer screening. Our findings suggest that men at an age of 75-80 years who have a PSA level below 3ng/ml are unlikely to be diagnosed with a high risk prostate cancer during life. These men may therefore represent an ideal target group for discontinuation of PSA testing, which could dramatically reduce the costs associated with screening and the potential morbidity of additional evaluations and/or treatment in a population unlikely to gain benefit." Dr. Schaeffer emphasized that these findings need to be confirmed in a much larger study, and that men over the age of 75 years should continue to be monitored for development of clinical signs of prostate cancer.

Public release date: 20-Feb-2009

Green, black tea can reduce stroke risk

Drinking at least three cups of green or black tea a day can significantly reduce the risk of stroke, a new UCLA study has found. And the more you drink, the better your odds of staving off a stroke.

The study results, published in the online edition of Stroke: Journal of the American Heart Association, were presented Feb. 19 at the American Heart Association's annual International Stroke Conference in San Diego, Calif.

The UCLA researchers conducted an evidence-based review of all human observational studies on stroke and tea consumption found in the PubMed and Web of Science archives. They found nine studies describing 4,378 strokes among nearly 195,000 individuals, according to lead author Lenore Arab, a professor of medicine in the division of general internal medicine and health services research at the David Geffen School of Medicine at UCLA.

"What we saw was that there was a consistency of effect of appreciable magnitude," said Arab, who is also a professor of biological chemistry. "By drinking three cups of tea a day, the risk of a stroke was reduced by 21 percent. It didn't matter if it was green or black tea."

And extrapolating from the data, the effect appears to be linear, Arab said. **For instance, if one drinks three cups a day, the risk falls by 21 percent; follow that with another three cups and the risk drops another 21 percent.**

This effect was found in tea made from the plant *Camellia sinensis*, not from herbal teas.

There are very few known ways to reduce the risk of stroke, Arab said. And developing medications for stroke victims is particularly challenging, given that the drug has to get to the stroke-damaged site quickly because damage occurs so fast. Arab said that by the time a stroke victim gets medical care, it's nearly too late to impede the damage.

"That's why these findings are so exciting," she said. "If we can find a way to prevent the stroke, or prevent the damage, that is simple and not toxic, that would be a great advance."

Though no one is certain which compounds in tea are responsible for this effect, researchers have speculated that the antioxidant epigallocatechin gallate (EGCG) or the amino acid theanine may be what helps. Antioxidants are believed to help prevent coronary artery disease.

"And we do know that theanine is nearly 100-percent absorbed," Arab said. "It gets across the blood-brain barrier and it looks a lot like a molecule that's very similar to glutamate, and glutamate release is associated with stroke."

"It could be that theanine and glutamate compete for the glutamate receptor in the brain," she added.

Although a randomized clinical trial is needed to confirm this effect, the findings suggest that drinking three cups of green or black tea a day could help prevent an ischemic stroke.

Public release date: 23-Feb-2009

Chili peppers help to unravel the mechanism of pain

Capsaicin, the active ingredient in chili peppers, is most often experienced as an irritant, but it may also be used to reduce pain. A new work published by Drs. Feng Qin and Jing Yao in this week's PLoS Biology uses capsaicin to uncover novel insight into how pain-receptor systems can adapt to painful stimuli. Sensory systems are well known to adapt to prevailing stimuli. For example, adaptation happens when your eyes adjust from a dark movie theater during a matinee to the bright sunlight outside. Whether pain receptors truly adapt or rescale their responses (versus simply desensitizing) has been an open question.

Capsaicin acts by binding to a receptor in the cell wall of nerve endings and triggering an influx of calcium ions into the neuron. Eventually, the nervous system interprets this cascade of events as pain or heat, depending on which nerves are stimulated. Scientists had previously linked the pain-relieving effects of capsaicin to a lipid called PIP2, found in cell membranes. When capsaicin is applied to the skin it induces a strong depletion of PIP2 in the cell membrane.

"The receptor acts like a gate to the neurons," said Qin. "When stimulated it opens, letting outside calcium enter the cells until the receptor shuts down, a process called desensitization. The analgesic action of capsaicin is believed to involve this desensitization process. However, how the entry of calcium leads to the loss of sensitivity of the neurons was not clear."

Capsaicin creams are commonly sold over the counter as effective treatment for a variety of pain syndromes, from minor muscle or joint aches to those that are very difficult to treat, such as arthritis and neuropathic pain.

By combining electrical and optical measurements, the authors now have been able to link directly the depletion of PIP2 and the desensitization of the receptor. The authors also showed that the receptor is fully functional after desensitization – i.e. although you stop feeling pain – are desensitized – if another event occurs that would normally trigger a 'pain' response – such as an increased concentration of capsaicin - the desensitization does not affect that feeling. "What changed was the responsiveness threshold," said Qin. "In

other words, the receptor had not desensitized per se, but its responsiveness range was shifted. This property, called adaptation, would allow the receptor to continuously respond to varying stimuli over a large capsaicin concentration range."

The findings have implications for pain sensation mechanisms as well as clinical applications. With an adaptive response, the receptors are essentially autoregulated without a fixed threshold, thus the intensity of the pain you experience is dependent on the recent history of pain.

Public release date: 23-Feb-2009

Vitamin D deficiency may increase risk of colds, flu

Large-scale study supports potential role in boosting immune system, more research needed
Vitamin D may be an important way to arm the immune system against disorders like the common cold, report investigators from the University of Colorado Denver (UC Denver) School of Medicine, Massachusetts General Hospital (MGH) and Children's Hospital Boston.

In the largest and most nationally representative study of the association between vitamin D and respiratory infections, people with the lowest blood vitamin D levels reported having significantly more recent colds or cases of the flu. The risks were even higher for those with chronic respiratory disorders, such as asthma and emphysema. The report appears in the February 23 Archives of Internal Medicine.

"The findings of our study support an important role for vitamin D in prevention of common respiratory infections, such as colds and the flu," says Adit Ginde, MD, MPH, UC Denver Division of Emergency Medicine and lead author of the study. "Individuals with common lung diseases, such as asthma or emphysema, may be particularly susceptible to respiratory infections from vitamin D deficiency."

While vitamin C has been used for the prevention of colds and other respiratory disorders for decades, little scientific evidence supports its effectiveness. In contrast, in recent years evidence has accumulated that vitamin D – most commonly associated with the development and maintenance of strong bones – may also play a key role in the immune system. Circumstantial evidence has implicated the wintertime deficiency of vitamin D, which the body produces in response to sunlight, in the seasonal increase in colds and flu; and small studies have suggested an association between low blood levels of vitamin D and a higher risk of respiratory infections.

The current study analyzed data from the Third National Health and Nutrition Examination Survey (NHANES III), conducted by the National Center for Health Statistics. Participants were interviewed in their homes regarding their health and nutrition, and most participants also received a physical examination that included collection of blood and other samples for laboratory analysis. The research team analyzed blood levels of 25-hydroxyvitamin D (25OHD) – the best measure of vitamin D status – from almost 19,000 adult and adolescent NHANES III participants, selected to be representative of the overall U.S. population.

Study participants with the lowest vitamin D blood levels – less than 10 ng per milliliter of blood – were about 40 percent more likely to report having a recent respiratory infection than were those with vitamin D levels of 30 or higher. The association was present in all seasons and even stronger among participants with a history of asthma or chronic obstructive pulmonary disease (COPD), including emphysema. Asthma patients with the lowest vitamin D levels were five times more likely to have had a recent respiratory infection; while among COPD patients, respiratory infections were twice as common among those with vitamin

D deficiency.

"A respiratory infection in someone with otherwise healthy lungs usually causes a few days of relatively mild symptoms," explains Carlos Camargo, MD, DrPH, MGH Department of Emergency Medicine and senior author of the study. "But respiratory infections in individuals with an underlying lung disease can cause serious attacks of asthma or COPD that may require urgent office visits, emergency department visits or hospitalizations. So the impact of preventing infections in these patients could be very large."

The authors stress that the study's results need to be confirmed in clinical trials before vitamin D can be recommended to prevent colds and flu. "We are planning clinical trials to test the effectiveness of vitamin D to boost immunity and fight respiratory infection, with a focus on individuals with asthma and COPD, as well as children and older adults – groups that are at higher risk for more severe illness," Ginde says. "While it's too early to make any definitive recommendations, many Americans also need more vitamin D for its bone and general health benefits. Clinicians and laypeople should stay tuned as this exciting area of research continues to expand."

Public release date: 23-Feb-2009

Vitamin B and folic acid may reduce risk of age-related vision loss

Taking a combination of vitamins B6 and B12 and folic acid appears to decrease the risk of age-related macular degeneration in women, according to a report in the February 23 issue of Archives of Internal Medicine, one of the JAMA/Archives journals.

Age-related macular degeneration (AMD) is a leading cause of vision loss in older Americans, according to background information in the article. Treatment options exist for those with severe cases of the disease, but the only known prevention method is to avoid smoking. Recent studies have drawn a connection between AMD and blood levels of homocysteine, an amino acid. High levels of homocysteine are associated with dysfunction of the blood vessel lining, whereas treatment with vitamin B6, vitamin B12 and folic acid appears to reduce homocysteine levels and may reverse this blood vessel dysfunction.

William G. Christen, Sc.D., of Brigham and Women's Hospital and Harvard Medical School, Boston, and colleagues conducted a randomized, double-blind clinical trial involving 5,442 women age 40 and older who already had heart disease or at least three risk factors. Of these, 5,205 did not have AMD at the beginning of the study. **In April 1998, these women were randomly assigned to take a placebo or a combination of folic acid (2.5 milligrams per day), pyridoxine hydrochloride (vitamin B6, 50 milligrams per day) and cyanocobalamin (vitamin B12, 1 milligram per day). Participants continued the therapy through July 2005 and were tracked for the development of AMD through November 2005.**

Over an average of 7.3 years of treatment and follow-up, 137 new cases of AMD were documented, including 70 cases that were visually significant (resulting in a visual acuity of 20/30 or worse). Of these, 55 AMD cases, 26 visually significant, occurred in the 2,607 women in the active treatment group, whereas 82 of the 2,598 women in the placebo group developed AMD, 44 cases of which were visually significant.

Women taking the supplements had a 34 percent lower risk of any AMD and a 41 percent lower risk of visually significant AMD. "The beneficial effect of treatment began to emerge at approximately two years of follow-up and persisted throughout the trial," the authors write.

"The trial findings reported herein are the strongest evidence to date in support of a

possible beneficial effect of folic acid and B vitamin supplements in AMD

prevention," the authors write. Because they apply to the early stages of disease development, they appear to represent the first identified way—other than not smoking—to reduce the risk of AMD in individuals at an average risk. "From a public health perspective, this is particularly important because persons with early AMD are at increased risk of developing advanced AMD, the leading cause of severe, irreversible vision loss in older Americans."

Beyond lowering homocysteine levels, potential mechanisms for the effectiveness of B vitamins and folic acid in preventing AMD include antioxidant effects and improved function of blood vessels in the eye, they note.

Public release date: 23-Feb-2009

No longer a gray area: Our hair bleaches itself as we grow older

New research report in the FASEB Journal gets to the roots of gray hair

Wash away your gray? Maybe. A team of European scientists have finally solved a mystery that has perplexed humans throughout the ages: why we turn gray. Despite the notion that gray hair is a sign of wisdom, these researchers show in a research report published online in The FASEB Journal (<http://www.fasebj.org>) that wisdom has nothing to do with it. **Going gray is caused by a massive build up of hydrogen peroxide due to wear and tear of our hair follicles. The peroxide winds up blocking the normal synthesis of melanin, our hair's natural pigment.**

"Not only blondes change their hair color with hydrogen peroxide," said Gerald Weissmann, MD, Editor-in-Chief of The FASEB Journal. "All of our hair cells make a tiny bit of hydrogen peroxide, but as we get older, this little bit becomes a lot. We bleach our hair pigment from within, and our hair turns gray and then white. This research, however, is an important first step to get at the root of the problem, so to speak."

The researchers made this discovery by examining cell cultures of human hair follicles. They found that the build up of hydrogen peroxide was caused by a reduction of an enzyme that breaks up hydrogen peroxide into water and oxygen (catalase). They also discovered that hair follicles could not repair the damage caused by the hydrogen peroxide because of low levels of enzymes that normally serve this function (MSR A and B). Further complicating matters, the high levels of hydrogen peroxide and low levels of MSR A and B, disrupt the formation of an enzyme (tyrosinase) that leads to the production of melanin in hair follicles. Melanin is the pigment responsible for hair color, skin color, and eye color. The researchers speculate that a similar breakdown in the skin could be the root cause of vitiligo.

"As any blue-haired lady will attest, sometimes hair dyes don't quite work as anticipated," Weissmann added. "This study is a prime example of how basic research in biology can benefit us in ways never imagined."

Public release date: 23-Feb-2009

Cholesterol-reducing drugs may lessen brain function, says ISU researcher

AMES, Iowa -- Research by an Iowa State University scientist suggests that cholesterol-reducing drugs known as statins may lessen brain function.

Yeon-Kyun Shin, a biophysics professor in the department of biochemistry, biophysics and molecular biology, says the results of his study show that drugs that inhibit the liver from making cholesterol may also keep the brain from making cholesterol, which is vital to efficient brain function.

"If you deprive cholesterol from the brain, then you directly affect the machinery that triggers the release of neurotransmitters," said Shin. "Neurotransmitters affect the data-processing and memory functions. In other words -- how smart you are and how well you remember things."

Shin's findings will be published in this month's edition of the journal Proceedings of the National Academy of Sciences of the United States of America.

Cholesterol is one of the building blocks of cells and is made in the liver. Low-density lipoprotein (LDL) -- often referred to as bad cholesterol -- is cholesterol in the bloodstream from the liver on the way to cells in the body. High-density lipoprotein (HDL) -- so-called good cholesterol -- is cholesterol being removed from cells. Too much LDL going to cells and not enough being removed can lead to cholesterol deposits and hardening of the cells.

"If you have too much cholesterol, your internal machinery is not going to be able to take away enough cholesterol from the cells," said Shin. "Then cells harden and you can get these deposits."

Cholesterol-reducing statin drugs are helpful because they keep the liver from synthesizing cholesterol so less of the substance is carried to the cells. This lowers LDL cholesterol.

It is the function of reducing the synthesis of cholesterol that Shin's study shows may also harm brain function.

"If you try to lower the cholesterol by taking medicine that is attacking the machinery of cholesterol synthesis in the liver, that medicine goes to the brain too. And then it reduces the synthesis of cholesterol which is necessary in the brain," said Shin.

In his experiments, Shin tested the activity of the neurotransmitter-release machinery from brain cells without cholesterol present and measured how well the machinery functioned. He then included cholesterol in the system and again measured the protein function. Cholesterol increased protein function by five times.

"Our study shows there is a direct link between cholesterol and the neurotransmitter release," said Shin. "And we know exactly the molecular mechanics of what happens in the cells. Cholesterol changes the shape of the protein to stimulate thinking and memory."

While reducing the cholesterol in the brain may make you have less memory and cognitive skills, more cholesterol in the blood does not make people smarter. Because cholesterol in the blood cannot get across the blood brain barrier, there is no connection to the amount of cholesterol a person eats and brain function.

Shin says that for many people taking cholesterol-lower statins can be very healthful and they should listen to their doctor when taking medication.

Public release date: 24-Feb-2009

Physical fitness improves spatial memory, increases size of brain structure

When it comes to the hippocampus, a brain structure vital to certain types of memory, size matters. Numerous studies have shown that bigger is usually better. Now researchers have found that elderly adults who are more physically fit tend to have bigger hippocampi and better spatial memory than those who are

less fit.

The study, in the journal *Hippocampus*, shows that hippocampus size in physically fit adults accounts for about 40 percent of their advantage in spatial memory.

The hippocampus, a curved structure deep inside the medial temporal lobe of the brain, is essential to memory formation. Remove it – as was done in the well-known case of surgical patient Henry Gustav Molaison – and a person's ability to store most new experiences in memory is destroyed.

The hippocampus also is a key player in spatial navigation and other types of relational memory.

Certain activities are believed to modify hippocampus size in humans. For example, a study of London taxi drivers found that the posterior portion of the hippocampus was larger in experienced taxi drivers than in other subjects. And a study of German medical students found that the same region of the hippocampus increased in size as they studied for their final exams.

Studies also have found that the hippocampus shrinks with age, a process that coincides with small but significant cognitive declines. The rate at which this occurs, however, differs among individuals.

Earlier studies found that exercise increases hippocampus size and spatial memory in rodents, but the new study is the first to demonstrate that exercise can affect hippocampus size and memory in humans.

The researchers, from the University of Illinois and the University of Pittsburgh, measured the cardiorespiratory fitness of 165 adults (109 of them female) between 59 and 81 years of age. Using magnetic resonance imaging, the researchers conducted a volumetric analysis of the subjects' left and right hippocampi. They also tested the participants' spatial reasoning.

They found a significant association between an individual's fitness and his or her performance on certain spatial memory tests. There was also a strong correlation between fitness and hippocampus size.

"The higher fit people have a bigger hippocampus, and the people that have more tissue in the hippocampus have a better spatial memory," said U. of I. psychology professor Art Kramer, who led the study with Pittsburgh psychology professor Kirk Erickson.

"Even ignoring the hippocampus data, we see there is this significant and substantial relationship between how fit you are and how good your memory is, or at least a certain kind of memory, a certain kind of memory that we need all the time," Kramer said.

"This is really a clinically significant finding because it supports the notion that your lifestyle choices and behaviors may influence brain shrinkage in old age," Erickson said. "Basically, if you stay fit, you retain key regions of your brain involved in learning and memory."

An impairment of spatial memory "is one of a number of reasons why older people end up losing their independence," Kramer said. "Here is yet more evidence that becoming fit has implications for how well you're going to live your life."

Public release date: 1-Mar-2009

Vegetable-based drug could inhibit melanoma

Compounds extracted from green vegetables such as broccoli and cabbage could be a potent drug against melanoma, according to cancer researchers. Tests on mice suggest that these compounds, when combined

with selenium, target tumors more safely and effectively than conventional therapy.

"There are currently no drugs to target the proteins that trigger melanoma," said Gavin Robertson, associate professor of pharmacology, pathology and dermatology, Penn State College of Medicine. **"We have developed drugs from naturally occurring compounds that can inhibit the growth of tumors in mice by 50 to 60 percent with a very low dose."**

Robertson and his colleagues previously showed the therapeutic potential of targeting the Akt3 protein in inhibiting the development of melanoma. The search for a drug to block the protein led them to a class of compounds called isothiocyanates.

These naturally occurring chemicals found in cruciferous vegetables are known to have certain cancer-fighting properties. However, the potency of these compounds is so low that a successful drug would require large impractical amounts of these compounds.

Instead, the Penn State researchers rewired the compounds by replacing their sulfur bonds with selenium. The result, they believe, is a more potent drug that can be delivered intravenously in low doses.

"Selenium deficiency is common in cancer patients, including those diagnosed with metastatic melanoma," explained Robertson, whose findings appear in the March edition of *Clinical Cancer Research*. "Besides, selenium is known to destabilize Akt proteins in prostate cancer cells."

To study the effectiveness of the new drug -- isoselenocyanate -- researchers injected mice with 10 million cancer cells. Six days later, when the animals developed large tumors, they were divided into two groups and treated separately with either the vegetable compounds or the compounds supplemented with selenium.

"We found that the selenium-enhanced compounds significantly reduced the production of Akt3 protein and shut down its signaling network," explained Robertson, who is also associate director of translational research and leader of the experimental therapeutics program at Penn State Hershey Cancer Institute. The modified compounds also reduced the growth of tumors by 60 percent, compared to the vegetable-based compounds alone.

When the researchers exposed three different human melanoma cell lines to the two compounds, the selenium-enhanced drug worked better on some cell lines than others. The efficiency was from 30 to 70 percent depending on the cell line.

The exact mechanism of how selenium inhibits cancer remains unclear. However Robertson, who has a filed provisional patent on the discovery, is convinced that the use of naturally occurring compounds that target cancer-causing proteins could lead to more effective ways of treating melanoma.

"We have harnessed something found in nature to target melanoma," said Robertson. "And since we only need tiny amounts to kill the cancer cells, it means even less toxic side-effects for the patient."

Human trials of the new drug are still some years away, but the Penn State researcher envisions a drug that could be delivered either intravenously to treat melanoma, or added to sunscreen lotion to prevent the disease

Ralph's Note - Sometimes it can become obvious that patents are more valuable than helping people now. If there was a way to reward independent researchers for discovery, as opposed to lucrative patent deals. I feel science would be purer and of a greater service. Than just as a reward to some ones mutual fund.

Public release date: 1-Mar-2009

Vitamin A signals offer clues to treating autoimmunity

Distributed around the body, dendritic cells act as the security alarms of the immune system. After sensing the presence of intruders, dendritic cells can transmit the alarm to white blood cells or tell them to relax, depending on the signals they send out.

Researchers at the Emory Vaccine Center and Yerkes National Primate Research Center have discovered that dendritic cells can respond to the same compound, through two different receptors, by sending out both stimulatory and calming messages at once.

The compound is zymosan, a component of yeast cell walls. However, the finding could guide scientists in designing vaccines against many infectious agents since the calming receptor is known to respond to bacteria and viruses as well as yeast. In addition, silencing the calming receptor's messages might boost the immune system's ability to fight a chronic infection.

The results are published in the March 2009 issue of Nature Medicine.

The calming receptor, known as TLR2 (Toll-like receptor 2), uses vitamin A to transmit its signals, which provides an explanation for the connection between vitamin A deficiency and autoimmune diseases. Vitamin A deficiency has been linked to diseases such as rheumatoid arthritis, lupus and type I diabetes.

This "two signals at once" feature of the immune system can be viewed as the result of an evolutionary tug of war, says senior author Bali Pulendran, PhD, professor of pathology and laboratory medicine at Emory University School of Medicine and Yerkes National Primate Research Center.

"The immune system has to provide a defense against infection, while avoiding the destruction of too much of the body along the way," he says. "At the same time, pathogens have evolved strategies to manipulate the immune system for their own purposes."

Working with Pulendran, postdoctoral fellow Santhakumar Manicassamy, PhD, examined which genes are turned on in dendritic cells by zymosan in cell culture. They were surprised to find that both zymosan and live *Candida albicans*, which causes yeast infections, turned on genes involved in converting vitamin A to its active form, retinoic acid.

"Others have seen that these genes are turned on constitutively in the gut, but seeing how they can be induced elsewhere is new," Pulendran says.

Manicassamy and colleagues found that dendritic cells use retinoic acid along with other chemical messengers to steer white blood cells into a regulatory mode, rather than an attack mode. For dendritic cells to do so, they need TLR2, since zymosan also activates another receptor called dectin-1, which sends out stimulatory signals.

The effects of zymosan and TLR2 can deter white blood cells from attacking nerve tissue in a mouse model of multiple sclerosis, the authors found.

In the model, mice are immunized against myelin, which forms a protective sheath around nerves. Injecting the mice with zymosan at the same time as immunization reduced the damage to their nerves.

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Public release date: 2-Mar-2009

Low levels of vitamin B12 may increase risk for neural tube defects

Children born to women who have low blood levels of vitamin B12 shortly before and after conception may have an increased risk of a neural tube defect, according to an analysis by researchers at the National Institutes of Health, Trinity College Dublin, and the Health Research Board of Ireland.

Women with the lowest B12 levels had 5 times the risk of having a child with a neural tube defect compared to women with the highest B12 levels.

Women who consume little or no meat or animal based foods are the most likely group of women to have low B12 levels, along with women who have intestinal disorders that prevent them from absorbing sufficient amounts of B12.

Neural tube defects are a class of birth defects affecting the brain and spinal cord. One type, spina bifida, can cause partial paralysis. Another type, anencephaly, is a fatal defect in which the brain and skull are severely underdeveloped.

Researchers have known that taking another nutrient, folic acid, during the weeks before and after conception can greatly reduce a woman's chances of having a child with a neural tube defect. Folic acid is the synthetic form of the vitamin folate. In the United States, cereal grains are fortified with folic acid to reduce the occurrence of neural tube defects in the U.S. population.

The study appears in the *March Pediatrics*. The study's first author was Anne M. Molloy, Ph.D., Trinity College Dublin. Scientists from the Health Research Board of Ireland and two NIH institutes, the Eunice Kennedy Shriver National Institute of Child Health and Human Development and the National Human Genome Research Institute, also took part in the study.

"Vitamin B12 is essential for the functioning of the nervous system and for the production of red blood cells," said Duane Alexander, M.D., director of the NICHD. "The results of this study suggest that women with low levels of B12 not only may risk health problems of their own, but also may increase the chance that their children may be born with a serious birth defect."

Ireland has a high rate of neural tube defects, and NIH scientists have frequently collaborated with Irish researchers to gain insight into the causes of this group of disorders.

To conduct the study, the researchers analyzed stored blood samples originally collected during early pregnancy from three groups of Irish women between 1983 and 1990. During that time, pregnant women in Ireland rarely took vitamin supplements. The study authors reasoned that the lack of routine vitamin supplementation would allow them to identify a sufficient number of women with low Vitamin B12 to conduct their analysis.

For their analysis, the researchers classified the women into three groups. The first group consisted of 95 women who were pregnant with a child having a neural tube defect at the time the blood was taken. The second group was composed of 107 women who had previously given birth to a child with a neural tube defect but whose current pregnancy was not affected. Like the first group, women in the third group (a total of 76) were pregnant with a child having a neural tube defect at the time the blood sample was obtained, but were enrolled in a different study than the women in group 1. The researchers measured the Vitamin B12 and folate levels of the women's blood samples, and compared them to those of control groups whose pregnancies were unaffected by a neural tube defect.

Because low folate levels are a known risk factor for neural tube defects, the researchers used statistical techniques to evaluate the role of Vitamin B12 independently of the role of folate. In all three groups, women with low B12 concentrations (estimated at less than 250 ng/L, before pregnancy) had 2.5-3 times the risk of having a child with a neural tube defect compared to those with higher levels. Women with levels in the deficient range (0-149 ng/L) were at the highest risk: 5 times that of women with higher levels.

The study authors wrote that it is not known how B12 and folate might interact to influence the formation of the neural tube, the embryonic structure that gives rise to the spine and brain. They noted that the two vitamins are jointly involved with several key biochemical reactions, as well as with the synthesis of DNA. Lack of either Vitamin B12 or folate in any of these chemical processes theoretically could increase the risk of a neural tube defect.

The authors noted that their results needed to be confirmed by other studies among other populations of women. They suggested, however, that women should have Vitamin B12 levels above 300 ng/L before

becoming pregnant. (Because B12 levels drop sharply during pregnancy, the researchers adjusted the levels measured during pregnancy to provide a target level for women to achieve before they become pregnant.)

Because Vitamin B12 comes from foods of animal origin, women who adhere to a strict vegan diet may be at risk for a B12 deficiency, said an NICHD author of the paper, James L. Mills, M.D., senior investigator in the Division of Epidemiology, Statistics, and Prevention Research. He added it is advisable for women with digestive disorders that interfere with the absorption of foods to consult a physician before getting pregnant, to make sure they are receiving adequate amounts of B12.

Dr. Mills explained that critical events in the formation of the brain and spinal column occur very early in pregnancy—in the first 28 days after conception—before many women even realize they are pregnant.

He added that the U.S. Public Health Service recommends that all women of childbearing age consume 400 micrograms of folic acid each day. This amount assures that a woman will have adequate stores of the vitamin, in the event of an unintended pregnancy.

"If women wait until they realize that they are pregnant before they start taking folic acid, it is usually too late," Dr. Mills said.

Similarly, he said, it would be wise for all women of childbearing age to consume the recommended amount of Vitamin B12, whether they are planning a pregnancy or not. "Half of the women who become pregnant each year in the U.S. were not planning to become pregnant."

"Our results offer evidence that women who have adequate B12 levels before they become pregnant may further reduce the occurrence of this class of birth defects," Dr. Mills said.

Public release date: 2-Mar-2009

Broccoli may help protect against respiratory conditions like asthma

Here's another reason to eat your broccoli: UCLA researchers report that a naturally occurring compound found in broccoli and other cruciferous vegetables may help protect against respiratory inflammation that causes conditions like asthma, allergic rhinitis and chronic obstructive pulmonary disease.

Published in the March edition of the journal *Clinical Immunology*, the research shows that sulforaphane, a chemical in broccoli, triggers an increase of antioxidant enzymes in the human airway that offers protection against the onslaught of free radicals that we breathe in every day in polluted air, pollen, diesel exhaust and tobacco smoke. A supercharged form of oxygen, free radicals can cause oxidative tissue damage, which leads to inflammation and respiratory conditions like asthma.

"This is one of the first studies showing that broccoli sprouts — a readily available food source — offered potent biologic effects in stimulating an antioxidant response in humans," said Dr. Marc Riedl, the study's principal investigator and an assistant professor of clinical immunology and allergy at the David Geffen School of Medicine at UCLA.

"We found a two- to three-fold increase in antioxidant enzymes in the nasal airway cells of study participants who had eaten a preparation of broccoli sprouts," Riedl said. "This strategy may offer protection against inflammatory processes and could lead to potential treatments for a variety of respiratory conditions."

The UCLA team worked with 65 volunteers who were given varying oral doses of either broccoli or alfalfa sprout preparations for three days. Broccoli sprouts are the richest natural source of sulforaphane; the alfalfa sprouts, which do not contain the compound, served as a placebo.

Rinses of nasal passages were collected at the beginning and end of the study to assess the gene expression

of antioxidant enzymes in cells of the upper airways. Researchers found significant increases of antioxidant enzymes at broccoli sprout doses of 100 grams and higher, compared with the placebo group.

The maximum broccoli sprout dosage of 200 grams generated a 101-percent increase of an antioxidant enzyme called GSTP1 and a 199-percent increase of another key enzyme called NQO1.

"A major advantage of sulforaphane is that it appears to increase a broad array of antioxidant enzymes, which may help the compound's effectiveness in blocking the harmful effects of air pollution," Riedl said.

According to the authors, no serious side effects occurred in study participants receiving broccoli sprouts, demonstrating that this may be an effective, safe antioxidant strategy to help reduce the inflammatory impact of free radicals.

Riedl notes that more research needs to be done to examine the benefits of sulforaphane for specific respiratory conditions. It is too early to recommend a particular dosage.

Riedl recommends including broccoli and other cruciferous vegetables as part of a healthy diet.

Public release date: 2-Mar-2009

Doctors endorse vegan and vegetarian diets for healthy pregnancies

WASHINGTON--Well-planned vegetarian and vegan diets are healthful choices for pregnant women and their children, and vitamin B12 needs can be easily met with fortified foods or any common multivitamin, say doctors and dietitians with the Physicians Committee for Responsible Medicine (PCRM). PCRM nutrition experts are available for comment in response to a new Pediatrics study showing that low levels of vitamin B12 may increase the risk for neural tube defects.

The Pediatrics study is based on analysis of stored blood samples originally collected during pregnancy from three groups of Irish women between 1983 and 1990. It's not clear if any of the women were vegan, but the study clearly states that this population was deliberately chosen because vitamin supplementation and food fortification were rare at that time. The women lived in a region of traditionally high neural tube defects prevalence, suggesting a moderately high genetic predisposition.

Experts agree that pregnant women can thrive on vegan diets. The American Dietetic Association, the nation's largest organization of food and nutrition professionals, states that "well-planned vegan and other types of vegetarian diets are appropriate for all stages of the life cycle, including during pregnancy, lactation, infancy, childhood, and adolescence." Vegetarian diets offer a number of nutritional benefits, including lower levels of saturated fat and cholesterol and higher levels of fiber, folate, and cancer-fighting antioxidants and phytochemicals.

"Women who follow vegan diets not only have healthy pregnancies, they are often healthier than moms who consume meat," says Susan Levin, M.S., R.D., staff dietitian with PCRM. "By eating a variety of fruits, vegetables, and other healthful vegetarian foods and including breakfast cereals or other foods fortified with vitamin B12, mothers and their children can obtain all the nutrients they need to thrive."

Choosing a vegetarian or vegan diet can also help women avoid the unhealthy hormones and environmental toxins found in dairy products, meat, and fish. Analyses of vegetarians' breast milk show that the levels of environmental contaminants in milk are much lower than in non-vegetarians.

Vitamin B12 needs can be met easily with fortified breakfast cereals and soymilk, which are low in fat and calories. The most convenient and reliable B12 source is a daily multivitamin.

Public release date: 2-Mar-2009

Cleansing toxic waste -- with vinegar

Engineers and environmental scientists at the University of Leeds are developing methods of helping contaminated water to clean itself by adding simple organic chemicals such as vinegar.

The harmful chromium compounds found in the groundwater at sites receiving waste from former textiles factories, smelters, and tanneries have been linked to cancer, and excessive exposure can lead to problems with the kidneys, liver, lungs and skin.

The research team, led by Dr Doug Stewart from the School of Civil Engineering and Dr Ian Burke from the School of Earth and Environment, has discovered that adding dilute acetic acid (vinegar) to the affected site stimulates the growth of naturally-occurring bacteria by providing an attractive food source. In turn, these bacteria then cleanse the affected area by altering the chemical make-up of the chromium compounds to make them harmless.

"The original industrial processes changed these chemicals to become soluble, which means they can easily leach into the groundwater and make it unsafe, says Dr Burke. "Our treatment method reconverts the oxidised chromate to a non-soluble state, which means it can be left safely in the ground without risk to the environment. As it is no longer 'bio-available' it doesn't present any risk to the surrounding ecosystem."

Chromate chemicals have previously been successfully treated in situ in neutral Ph conditions, but this study is unique in that it concentrates on extremely alkaline conditions, which are potentially much more difficult to treat.

The current favoured method of dealing with such groundwater contaminants is to remove the soil to landfill, which can be costly, both financially and in terms of energy usage. The Leeds methods being developed will allow treatment to take place on site, which is safer, more energy efficient and much cheaper.

Dr Stewart says: "Highly alkaline chromium-related contaminants were placed in inadequate landfill sites in the UK right up until production stopped in the 1970's – and in some countries production of large quantities of these chemicals still continues today. The soluble and toxic by-products from this waste can spread into groundwater, and ultimately into local rivers, and therefore will remain a risk to the environment as long as they are untreated."

Current environmental regulations mean that before the team can test out its research findings in the field, they need water-tight proof that their methods can work, as it is illegal to introduce any substance into groundwater - even where it is contaminated - unless it has been shown to be beneficial.

"From the results we have so far I am certain that we can develop a viable treatment for former industrial sites where chromate compounds are a problem," says Dr Stewart. "Our next step is to further our understanding of the range of alkalinity over which our system can operate. As society becomes more environmentally-aware, new regulations demand that past mistakes are rectified and carbon footprints are reduced. By designing a clean-up method that promotes the growth of naturally occurring bacteria without introducing or engineering new bacteria, we are effectively hitting every environmental target possible."

Public release date: 3-Mar-2009

Are vitamin supplements effective in celiac disease patients? (Yes they are)

Coeliac disease is a typical example of a malabsorption syndrome conferring increased risk for various deficiency states, including folate and vitamin B12. Hyperhomocysteinemia is significantly more frequent in patients with newly diagnosed coeliac disease than healthy controls.

A research team led by Dr. Muhammed Hadithi from Netherlands investigated the effect of vitamin B6, folate, and vitamin B12 daily supplements on homocysteine levels in patients with coeliac disease. Their study will be published on February 28, 2009 in the World Journal of Gastroenterology.

In their study, vitamin B6, folate, vitamin B12, and fasting plasma homocysteine levels were investigated in 51 consecutive adults with coeliac disease and 50 healthy control individuals matched for age and sex.

They found that patients with celiac disease and using vitamin supplements had higher serum vitamin B6 ($P = 0.003$), folate ($P < 0.001$), and vitamin B12 ($P = 0.012$) levels than patients who did not or healthy controls ($P = 0.035$, $P < 0.001$, $P = 0.007$, for vitamin B6, folate, and vitamin B12, respectively). Lower plasma homocysteine levels were found in patients using vitamin supplements than in patients who did not ($P = 0.001$) or healthy controls ($P = 0.003$). However, vitamin B6 and folate, not vitamin B12, were significantly and independently associated with homocysteine levels. Twenty-four (48%) of 50 controls and 23 (50%) of 46 patients with celiac disease carried the MTHFR thermolabile variant T-allele ($P = 0.89$).

They concluded that Homocysteine levels are dependent on Marsh classification and the regular use of B-vitamin supplements is effective in reduction of homocysteine levels in patients with celiac disease.

The study demonstrates in agreement with earlier findings, that both the presence and the severity of coeliac disease were determinants of homocysteine levels. The regular use of B vitamin supplements was associated with higher serum vitamin B6, folate, and vitamin B12 and lower plasma homocysteine levels in patients with coeliac disease. Furthermore, B vitamin supplements seem to have a protective role against the effect of villous atrophy on homocysteine levels, irrespective to the genetic susceptibility status as manifested by carrying the C677T polymorphism of 5,10-methylenetetrahydrofolate reductase

**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.**