Researchers find key genetic trigger of depression

(PhysOrg.com) -- Yale University researchers have found a gene that seems to be a key contributor to the onset of depression and is a promising target for a new class of antidepressants, they report Oct. 17 in the journal Nature Medicine.

"This could be a primary cause, or at least a major contributing factor, to the signaling abnormalities that lead to depression," said Ronald S. Duman, professor of psychiatry and pharmacology at Yale and senior author of the study.

Scientists have had a difficult time pinning down the cause of depression, which afflicts almost 16 percent of Americans in any given year and carries an annual economic burden of $100 billion.

Symptoms of depression vary widely among individuals. Most now believe that multiple physiological processes are involved in major depressive disorder. That explains why people respond differently to most commonly prescribed antidepressants, which work by manipulating the uptake of the neurotransmitter serotonin. However, as many as 40 percent of depressed patients do not respond to currently available medications, which take weeks to months to produce a therapeutic response.

Duman's team did whole genome scans on tissue samples from 21 deceased individuals who had been diagnosed with depression and compared gene expression levels to those of 18 individuals who had not been diagnosed with depression. They found that one gene called MKP-1 was increased more than two-fold in the brain tissues of depressed individuals.

This was particularly exciting, say the researchers, because the gene inactivates a molecular pathway crucial to the survival and function of neurons and its impairment has been implicated in depression as well as other disorders. Duman's team also found that when the MKP-1 gene is knocked out in mice, the mice become resilient to stress. When the gene is activated, mice exhibit symptoms that mimic depression.

The finding that a negative regulator of a key neuronal signaling pathway is increased in depression also identifies MKP-1 as a potential target for a novel class of therapeutic agents, particularly for treatment resistant depression.

Chocolate cholesterol claims spark debate

An ingredient of dark chocolate may help diabetics control dangerously high cholesterol levels, it is claimed.

Chocolate with high levels of cocoa solids is rich in polyphenols, which other studies suggest can reduce the risk of heart disease.

The Hull University study found cholesterol fell in a small number of diabetics given bars rich in this ingredient. But Diabetes UK said the high fat and sugar content would outweigh benefits.

High cholesterol levels are a particular problem for many diabetes, and are linked strongly to an increased risk of heart disease.

The Hull study, published in the journal Diabetic Medicine, tested the theory that chemicals found in cocoa beans could influence this. A total of 12 volunteers with the type II form of the condition were given identical chocolate bars, some enriched with polyphenols, over a 16 week period.

Those given the enriched bars experienced a small improvement in their overall cholesterol "profile", with total cholesterol falling, and levels of so-called "good" cholesterol rising.

Sensible approach

Professor Steve Atkin, who led the study, suggested that it could mean a reduction in heart risk. He said: "Chocolate with a high cocoa content should be included in the diet of individuals with type II diabetes as part of a sensible, balanced approach to diet and lifestyle."

However, there were some concerns from researchers at Diabetes UK that the message would be interpreted as a "green light" to eat more chocolate. They pointed out that even bars with the highest levels of cocoa solids would contain high levels of fat and sugar, and could end up doing more harm than good.

Regular bars of two of the UK's best selling varieties of dark chocolate each contain more than 200 calories and up to 16 grams of fat.

Dr Iain Frame, director of research at leading health charity Diabetes UK, said he was unconvinced by talk of health benefits. "On no account should people take away the message from this study, conducted in only 12 people, that eating even a small amount of dark chocolate is going to help reduce their cholesterol levels.

"The tiny health benefit of this compound found in cocoa-rich chocolate would be hugely outweighed by the fat and sugar content. "The design of the study is also somewhat unrealistic as they asked participants to eat only around half the size of a normal, dark chocolate bar every day for eight weeks.

"It would, however, be interesting to see if further research could find a way of testing whether polyphenols could be added to foods which weren't high in sugar and saturated fat such as chocolate," Dr Frame said.

Aqueous cream 'aggravates eczema'

Cream often prescribed to relieve the symptoms of eczema may be making the condition worse, researchers claim.

Scientists at Bath University found that aqueous cream thinned the skin after a few weeks of use. This, they say, is because it contains a detergent rather than just moisturisers. Another expert said most GPs seemed unaware of official advice not to prescribe the cream as a moisturiser.

Eczema, which affects millions of adults in the UK, happens when the skin gets dry and cracked. One way to reduce the discomfort and keep it under control is to use moisturising creams.

Aqueous cream, sold at every pharmacy, is an emollient cream, and is officially recommended as an alternative to soap when washing. However, it is also frequently recommended by doctors for its moisturising properties - one recent poll suggested nine out of 10 GPs recommended it for childhood eczema.

The University of Bath study, published in the British Journal of Dermatology looked directly at its effects on the skin when used regularly.

Volunteers, none of whom had eczema, rubbed it into their forearms every day over a four-week period. Scientists then compared skin samples taken before and after. They found the thickness of the stratum corneum, the outermost skin layer, was reduced by about 10% in this time.

Professor Richard Guy supervised the research, conducted as part of a PhD course by researcher Manda Tsang. He said the sodium lauryl sulphate detergent in the cream was affecting a thin layer of fats lying on top of the skin. He said: "Our study has found that rubbing aqueous cream containing sodium lauryl sulphate into the skin thins this protective barrier, making the skin more susceptible to irritation by chemicals.

"So to use this cream on eczematous skin, which is already thin and vulnerable to irritation, is likely to make the condition even worse."

'Heavy duty'

The National Eczema Society recommends alternatives such as white soft paraffin or even other types of emollient without such a high sodium laurel sulphate content.

Margaret Cox, chief executive of the National Eczema Society, said: "Aqueous cream contains sodium lauryl sulphate, which is a fairly heavy duty detergent. Sadly it is widely used - one it's cheap and two, it's prescribing habit."

Professor Michael Cork, an academic dermatologist from the University of Sheffield, said despite advice from the National Institute of Health and Clinical Excellence in England and Wales not to prescribe or recommend aqueous cream in this way, it was still widespread practice. He recommended that people with eczema use a formulation without the detergent instead. "This layer of skin will grow back over time, but if you're using aqueous cream on it every day, it simply won't get the chance."

Vitamin B12 link to Alzheimer's backed by study

Vitamin B capsule Experts say it is too early to recommend supplements

Evidence is mounting that levels of vitamin B12 may be connected to the risk of developing Alzheimer's disease. A study of 271 Finns found those with the highest levels were the least likely to be diagnosed with dementia. However, an Alzheimer's charity said despite the findings, published in the journal Neurology, it was "too early" to think about taking supplements. It called for more research into the protective power of vitamins such as B12 - found in meat, fish and eggs. Vitamin B12 can also be found in milk and some fortified cereals.

Alzheimer's has been linked to B vitamins for some years, and scientists know that higher levels of a body chemical called homocysteine can raise the risk of both strokes and dementia. Homocysteine levels can be lowered by increasing the amount of vitamin B12 in the blood.

A recent trial found that "brain shrinkage", which has been associated with Alzheimer's, was slowed in older people taking high doses of vitamins, including B12. The volunteers for the latest study, carried out by scientists from the Karolinska Institute in Sweden, were all aged 65 to 79, and did not have dementia at the start of the study. Over the next seven years, 17 of them were diagnosed with the condition, and researchers were able to work out whether high or low levels of the active component of B12 had made any difference.

Again, those with high levels of homocysteine appeared to be at greater risk, and those with the highest levels of the vitamin appeared to be at lower risk.

Professor Helga Refsum, from the University of Oslo, another B-vitamin researcher, said that the study was "further evidence" that low levels of B12 were linked to Alzheimer's.

"Though relatively small, with few cases of dementia, it should act as another incentive to start a large scale trial with homocysteine-lowering therapy using B vitamins to see whether such a simple treatment may slow the development of Alzheimer's or other dementia."

Rebecca Wood, the chief executive of the Alzheimer's Research Trust, was cautious about the findings. She said: "It might be tempting at this stage to stock up the cupboard with B vitamin in the light of recent findings - it remains too early to do that at this stage. "The strongest evidence we have for reducing dementia risk is to eat a healthy, balanced diet, take moderate exercise, and keep cholesterol and blood pressure in check, particularly in mid-life."

A separate study offered some encouragement to those looking for future treatments for the disease.

A treatment to lower levels of a protein called "STEP" in mice bred to develop a condition similar to Alzheimer's disease appeared to reverse some of their memory and learning problems. The Alzheimer's Research Trust said it was too soon to know whether a similar treatment might be viable in humans.

Study: Religious diversity increases in America, yet perceptions of Christian nation intensify

WEST LAFAYETTE, Ind. - While America continues to become more religiously diverse, the belief that America is a Christian nation is growing more intense, according to research from Purdue University.

"America is still predominantly Christian, but it is more diverse than ever," said Jeremy Brooke Straughn, an assistant professor of sociology who studies national identity. "At the same time, many people feel even more strongly that America is a Christian country than they did before the turn of the century. This is especially true for Americans who say they are Christians and who attend religious services at least once a week."

The fact that these beliefs have intensified since the mid-1990s suggests a connection to events such as the Sept. 11 attacks and the wars in Afghanistan and Iraq, Straughn said.

"We suspect that these events accentuated the connection between Christianity and American identity by reinforcing boundaries against non-Christians and people of foreign origin," he said. "Although we can't be certain of the underlying causes, our data clearly show diverging attitudes between American Christians and their non-Christian counterparts here in the United States. Those who express these views might say the belief is rooted in love of country and religion and is not about hating or discouraging others. But voicing these beliefs may cause others to feel that they do not belong and to withdraw from participating in public life."

Straughn and co-author Scott L. Feld, a professor of sociology, looked at two waves of public opinion data from the General Social Survey, which was collected by the National Opinion Research Center. They found that between 1996 and 2004 the percentage of people who said Christian faith was a very important attribute of being "truly American" rose by more than 11 percentage points, from about 38 percent to 50 percent. The findings are published in the current issue of the journal Sociology of Religion.

This trend coincides with a steady increase in religious diversity, with Protestants suffering the greatest relative losses. Although Christians still account for about 78 percent of the adult population, the share of Protestants has fallen from more than 60 percent in the early 1990s to 50 percent in 2006.

"Religious boundaries can be politically divisive," Straughn said. "And this is important to take note of as we approach the November elections. Religion and national identity continue to be in the news, from questions about President Barack Obama's religion to the recent controversy about an Islamic center near the Sept. 11 site.

"Even when voters seem focused on problems like the economy and unemployment, the issue of religion and national identity could make a difference at the margins. Especially in close races, the advantage may go to candidates seen as committed to the values of American Christians."

Straughn also is working on national identity related to language, race and education. His work is supported by the College of Liberal Arts, Department of Sociology and the Purdue Research Foundation.

Abstract on the research in this release is available at:
http://www.purdue.edu/newsroom/research/2010/101019StraughnReligion.html

Drought may threaten much of globe within decades

Dryness likely to increase substantially across Eurasia, Africa, Australia

The United States and many other heavily populated countries face a growing threat of severe and prolonged drought in coming decades, according to results of a new study by National Center for Atmospheric Research (NCAR) scientist Aiguo Dai. The new findings appear this week as part of a longer review article in Wiley Interdisciplinary Reviews: Climate Change. The study was supported by the National Science Foundation (NSF), NCAR's sponsor.

The detailed analysis concludes that warming temperatures associated with climate change will likely create increasingly dry conditions across much of the globe in the next 30 years. The drought may reach a scale in some regions by the end of the century that has rarely, if ever, been observed in modern times.

Using an ensemble of 22 computer climate models and a comprehensive index of drought conditions, as well as analyses of previously published studies, the paper reports that by the 2030s, dryness is likely to increase substantially across most of the Western Hemisphere, along with large parts of Eurasia, Africa, and Australia.

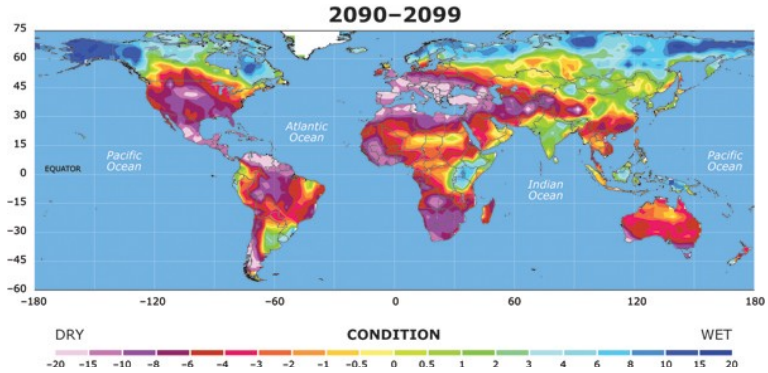
By later this century, many of the world's most densely populated regions will be threatened with severe drought conditions. In contrast, higher-latitude regions from Alaska to Scandinavia are likely to become more moist. Dai cautioned that the findings are based on the best current projections of greenhouse gas emissions.

What happens in coming decades will depend on many factors, including actual future emissions of greenhouse gases as well as natural climate cycles such as El Niño.

"This research does an excellent job of placing future warming-induced drought in the context of the historical drought record," says Eric DeWeaver, program director in NSF's Division of Atmospheric and Geospace Sciences, which funds NCAR.

"The work argues credibly that the worst consequences of global warming may come in the form of reductions in water resources."

While regional climate projections are less certain than those for the globe as a whole, Dai's study indicates that most of the western two-thirds of the United States will be significantly drier by the 2030s. Large parts of the nation may face an increasing risk of extreme drought during the century.



Widespread drought in 2099, based on current projections of greenhouse gas emissions. **Credit: UCAR**

"We are facing the possibility of widespread drought in the coming decades, but this has yet to be fully recognized by both the public and the climate change research community," Dai says. "If the projections in this study come even close to being realized, the consequences for society worldwide will be enormous."

Other countries and continents that could face significant drying include:

* Much of Latin America, including large sections of Mexico and Brazil
* Regions bordering the Mediterranean Sea, which could become especially dry
* Large parts of Southwest Asia
* Most of Africa and Australia, with particularly dry conditions in regions of Africa
* Southeast Asia, including parts of China and neighboring countries
* The study also finds that drought risk can be expected to decrease this century across much of Northern Europe, Russia, Canada, and Alaska, as well as some areas in the Southern Hemisphere.

However, the globe's land areas should be drier overall.

"The increased wetness over the northern, sparsely populated high latitudes can't match the drying over the more densely populated temperate and tropical areas," Dai says.

Previous climate studies have indicated that global warming will probably alter precipitation patterns as the subtropics expand.

The 2007 assessment by the Intergovernmental Panel on Climate Change (IPCC) concluded that subtropical areas will likely have precipitation declines, with high-latitude areas getting more precipitation.

In addition, previous studies by Dai have indicated that climate change may already be having a drying effect on parts of the world. He and colleagues found that the percentage of Earth's land area stricken by serious drought more than doubled from the 1970s to the early 2000s. Last year, he headed up a research team that found that some of the world's major rivers are losing water.

In his new study, Dai turned from rain and snow amounts to drought itself, and posed a basic question: how will climate change affect future droughts?

If rainfall runs short by a given amount, it may or may not produce drought conditions, depending on how warm it is, how quickly the moisture evaporates, and other factors.

Droughts are complex events that can be associated with significantly reduced precipitation, dry soils that fail to sustain crops, and reduced levels in reservoirs and other bodies of water that can imperil drinking supplies.

A common measure called the Palmer Drought Severity Index classifies the strength of a drought by tracking precipitation and evaporation over time and comparing them to the usual variability one would expect at a given location.

Dai turned to results from the 22 computer models used by the IPCC in its 2007 report to gather projections about temperature, precipitation, humidity, wind speed, and Earth's radiative balance, based on current projections of greenhouse gas emissions. He then fed the information into the Palmer model to calculate the

PDSI index. A reading of +0.5 to -0.5 on the index indicates normal conditions, while a reading at or below -4 indicates extreme drought. The index ranges from +10 to -10 for current climate conditions, although readings below -6 are exceedingly rare, even for small areas.

By the 2030s, the results indicated that some regions in the United States and overseas could experience particularly severe conditions, with readings potentially dropping to -4 to -6 in much of the central and western United States as well as several regions overseas, and -8 or lower in parts of the Mediterranean.

By the end of the century, many populated areas, including parts of the United States, could face readings in the range of -8 to -10, and much of the Mediterranean could fall to -15 to -20.

Such readings would be almost unprecedented.

Dai cautions that global climate models remain inconsistent in capturing precipitation changes and other atmospheric factors, especially at the regional scale.

However, the 2007 IPCC models were in stronger agreement on high- and low-latitude precipitation than those used in previous reports, says Dai.

There are also uncertainties in how well the Palmer index captures the range of conditions that future climate may produce. The index could be overestimating drought intensity in the more extreme cases, says Dai.

On the other hand, the index may be underestimating the loss of soil moisture should rain and snow fall in shorter, heavier bursts and run off more quickly.

Such precipitation trends have already been diagnosed in the United States and several other areas over recent years, says Dai. "The fact that the current drought index may not work for the 21st century climate is itself a troubling sign," Dai says.

Study reveals superior sedation method for children

Procedural sedation and analgesia is an essential element of care for children requiring painful procedures in the emergency department. The practice of combining ketamine and propofol, two common medications used in emergency departments, has become more popular. However, until recently, it was unclear whether this practice was superior to the use of either agent alone, especially in children.

Research led by Drs. Amit Shah, Gregory Mosdossy and Michael Rieder of the Schulich School of Medicine & Dentistry at The University of Western Ontario and Lawson Health Research Institute provides evidence that when compared to ketamine alone, patients who receive a combination of ketamine and propofol have a slightly faster recovery time and suffer from less severe side effects.

The study, published online in the Annals of Emergency Medicine, included 136 children treated at London Health Sciences Centre's Children's Hospital.

Ketamine is well established as a safe and effective solitary agent for procedural sedation and analgesia. However, it is known to cause adverse side effects, such as vomiting. Propofol is associated with a dose-dependent risk of respiratory depression, but has less severe side effects.

It has been theorized that by combining the two agents, you can decrease the dose requirement of both agents thereby reducing the negative side-effects, but still have a safe and effective analgesia.

This is the first large well-conducted study exploring the use of ketamine-propofol for children in the Paediatric Emergency Department. Previous studies had hinted at its advantage but up until now results were not conclusive.

"Our study found that ketamine-propofol is an effective combination for pediatric procedural sedation, providing a slightly shorter total sedation time than ketamine alone, with less adverse events and higher satisfaction scores," says Dr. Shah. "We believe this study provides evidence for a safe and effective alternative sedation regimen for children in the Emergency Department and may lead to a change in sedation practices in other hospitals."

Shock tactics: Bioelectrical therapy for cancer and birth defects?

Voltage-sensitive cells can instruct stem cells

Stem cell therapies hold increasing promise as a cure for multiple diseases. But the massive potential of a healthy stem cell has a flip side, as faulty regulation of stem cells leads to a huge range of human diseases. Even before birth, mistakes made by the stem cells of the foetus are a major cause of congenital defects, and cancer is also caused by the body losing control of stem cell function. Guiding stem cells along the correct pathways and, where necessary, reversing their mistakes is the goal of everyone in this field. Now, Michael Levin (http://www.drmichaellevin.org/) and colleagues from Tufts University (http://www.tufts.edu/), Medford, MA, have identified a novel and readily modifiable signal by which an organism can control the behaviour of stem cell offspring. Their work is published in Disease Models & Mechanisms on October 19th, 2010, at http://dmm.biologists.org/.

Levin's laboratory works on an intriguing phenomenon: bioelectrical signalling. There is always a difference in voltage, called the transmembrane potential, between the inside and outside of all cells, and controlling exactly what this difference is turns out to be vitally important. Specialised protein checkpoints sited in a cell's outer membrane regulate ion flow in and out of the cell, producing voltage gradients. These, combined with more conventional protein-based signalling systems, can specify cell destiny.

Levin's team already knew from collaborative work with David Kaplan's lab, also at Tufts, that the properties of human stem cells growing in artificial culture could be drastically altered by changing their transmembrane potential. Now they have taken this work one important step further, by asking whether tampering with the transmembrane potential of one kind of cell can have a domino effect in a whole organism, altering the destiny of other cell types. To do this, they focused on the development of neural crest stem cells, which are responsible for directing development of the face and heart, but which also generate melanocytes, the pigment cells of the skin. Using frog tadpoles and melanocytes as a model system, they showed that tweaking the transmembrane potential of a tiny population of 'instructor' cells sends a signal to developing melanocytes that causes them to overgrow and start to resemble metastatic cancer cells. Most excitingly, they found that the signal can travel over long distances in the tadpole, and that the messenger carrying it is serotonin – an important neurotransmitter involved in mood regulation and many other aspects of nervous system function.

This novel bioelectrical method of changing stem cell behaviour has huge implications. It is very likely that there are similar 'instructor' cells that direct other important cell populations, and changing their voltage gradients would be relatively easy (Levin's lab simply used an anti-parasitic drug already available on prescription). The resulting bioelectrical therapy could potentially be harnessed to improve regenerative repair after injury, repair birth defects and detect and prevent cancer.

REFERENCE: *Douglas Blackiston, Dany S. Adams, Joan M. Lemire, Maria Lobikin and Michael Levin (2010). Transmembrane potential of GlyCl-expressing instructor cells induces a neoplastic-like conversion of melanocytes via a serotonergic pathway. Dis. Model. Mech.[in press]*

Early pregnancy in spring linked to child's susceptibility to food allergies

Season of first trimester of pregnancy predicts sensitisation to food allergens in childhood: A population based cohort study from Finland

A child's likelihood of developing food allergies can be traced back to the season during which s/he completes their first three months of life in the womb, suggests research published online in the Journal of Epidemiology and Community Health.

The Finnish researchers base their findings on just under 6000 children, all of whom were born between 2001 and 2006 and lived in one area of Finland.

Out of the total, just under 1000 were tested for sensitisation to food allergens between the ages of 0 and 4 years, with the likelihood of a positive test result rising sharply during the first year of life.

Up to the age of 4, the incidence of an allergic response to certain foods varied according to season of birth, ranging from 5% for children born in June/July to 9.5% for those born in October/November.

Around one in 10 (11%) children, whose 11th week of development in the womb had occurred during April or May were sensitised to food allergens. This compared with a rate of 6% among children who reached that stage of fetal development in December/January.

Readings of ambient pollen for the years in question showed that levels of birch and alder pollen peaked during April and May. When narrowed down to specific allergens, the results indicated that a child whose first three months of fetal development ended in April or May was three times more likely to be sensitised to milk and eggs than those who reached this stage of development in November or December.

Research already indicates that children born in autumn or winter are more prone to eczema and wheeze, and that they have higher levels of circulating antibodies to allergens than children born in spring or summer, say the authors.

This might be because the fetus begins to produce antibodies to allergens at around the 11th week of development, and antibodies to specific allergens by around 24 weeks, they suggest.

An allergic type response is thought to be necessary for the pregnancy to continue, and in some cases this persists after birth. But the timing of the development of sensitisation has been the subject of heated debate.

Low testosterone linked to heightened risk of early death

Low serum testosterone and increased mortality in men with coronary heart disease

Low testosterone levels seem to be linked to a heightened risk of premature death from heart disease and all causes, suggests research published online in Heart.

The finding refutes received wisdom that the hormone is a risk factor for cardiovascular disease.

The researchers base their findings on 930 men, all of whom had coronary artery heart disease, and had been referred to a specialist heart centre between 2000 and 2002. Their heart health was then tracked for around 7 years. On referral, low testosterone was relatively common. One in four of the men was classified as having low testosterone, using measurements of either bioavailable testosterone (bio-T) - available for tissues to use - of under 2.6 mmol/l or total testosterone (TT) of under 8.1 mmol/l. These measures indicate clinically defined testosterone deficiency, referred to as hypogonadism, as opposed to a tailing off in levels of the hormone as a result of ageing.

During the monitoring period almost twice as many men with low testosterone died as did those with normal levels. One in five (41) of those with low testosterone died, compared with one in eight (12%) of those with normal levels. The only factors that influenced this risk were heart failure (left ventricular dysfunction), treatment with aspirin or a high blood pressure drug (beta blocker) and low bio-T levels.

A low bio-T level was an independent risk factor for premature death from all causes and from heart disease, after taking account of other influential factors, such as age, other underlying health problems, smoking and weight. Borderline levels of low total testosterone (15.1mmol/l) also increased the risk of an early death.

While high doses of testosterone found in anabolic steroids are harmful to health, the evidence suggests that low, rather than high, levels of the hormone, are associated with obesity, risky blood fats, and insulin resistance, all of which are risk factors for diabetes and heart disease, say the authors.

Men at high risk of these diseases may stand most to gain from testosterone replacement, they suggest.

An accompanying editorial points out that there is increasing interest in looking at the impact of testosterone replacement. "There has been a marked increase in prescription of testosterone over recent years. While the long term cardiovascular impact of testosterone supplements in those with low levels remains to be demonstrated, accumulating evidence suggests there is a sound basis for examining this," write the authors.

But the authors warn that it is not without its risks, which include prostate cancer. And high testosterone in women boosts the risk of diabetes and cardiovascular disease.

Research into testosterone has taken a back seat for many years, in favour of looking at the impact of oestrogens on heart disease, they conclude, adding: "Recent data suggest that this important pathway warrants a lot more attention."

Paleolithic Humans Had Bread Along With Their Meat

By REUTERS

LONDON (Reuters) — Starch grains found on 30,000-year-old grinding stones suggest that prehistoric humans may have dined on an early form of flatbread, contrary to their popular image as primarily meat eaters.

The findings, published in The Proceedings of the National Academy of Sciences journal on Monday, indicate that Paleolithic Europeans ground down plant roots similar to potatoes to make flour, which was later whisked into dough.

“It’s like a flatbread, like a pancake with just water and flour,” said Laura Longo, a researcher on the team, from the Italian Institute of Prehistory and Early History. “You make a kind of pita and cook it on the hot stone,” she said, describing how the team replicated the cooking process. The end product was “crispy like a cracker but not very tasty,” she added.

The grinding stones, each of which fits comfortably into an adult’s palm, were discovered at archaeological sites in Italy, Russia and the Czech Republic.

The researchers said their findings throw humankind’s first known use of flour back some 10,000 years, the previously oldest evidence having been found in Israel on 20,000-year-old grinding stones.

The findings may also upset fans of the so-called Paleolithic diet, which follows earlier research that assumes early humans ate a meat-centered diet. Also known as the “cave man diet,” the regime frowns on carbohydrate-laden foods like bread and cereal, and modern-day adherents eat only lean meat, vegetables and fruit.

It was first popularized by the gastroenterologist Walter L. Voegtlin, whose 1975 book lauded the benefits of the hunter-gatherer diet.

Toads evolve into super-invaders

By Victoria Gill Science and nature reporter, BBC News

Scientists have demonstrated a "runaway evolutionary effect" that is speeding up Australia's cane toad invasion.

This explains why the invasive toads have increased their rate of spread so dramatically, the researchers say.

They found that toads living at the very edge of their range were "super-invaders" - able to move beyond the boundaries of this existing habitat. And when toads at the frontiers bred, their offspring inherited this ability to move quickly into new territory.

This phenomenon, which scientists have termed the Olympic Village Effect, has been proposed before, since these same scientists observed that the toads at the edge of the range had bigger front legs and stronger back legs - all the better to jump and to invade new areas.

In this study, the researchers tested the effect, essentially setting up a cane toad race.

Dr Ben Phillips from James Cook University in Queensland, Australia collected cane toads from four different populations. He captured ten toads from the core population in northern Queensland, and ten from each of three populations that were increasingly distant from this point.

Cane toads have increased their rate of spread fivefold in the last 70 years

He took the toads to a facility in the appropriately named Middle Point near Darwin, where he fitted them with radio tags and then released them. The tags enabled the scientists to follow the toads' progress.

As Dr Phillips expected, toads that were collected from the edge of the range were much faster movers.

All in the genes

To confirm that this increased strength and speed had a genetic basis and could be inherited, Dr Phillips studied a generation further. He allowed toads from the same population to breed. Then he set up another radio-tagged toad race, this time between these captive-bred offspring. Toads that had parents from the edge of the range won the dispersal race, revealing that they inherited their speed and strength from their parents.

"It's bad news," Dr Phillips told BBC News. "It means they're getting faster and better at invading new areas."

Even worse, the researchers say, all animal invasions are likely to follow this pattern. He explained that the faster moving toads even reproduced more quickly. But this could point to a chink in their biological armour.

"They have to be trading something off to do that," he said. "And one of the things we suspect is that they're trading off their immune systems."

Since the bigger, faster toads spread and breed so quickly, they are likely to leave any endemic diseases and parasites behind them because toads that move so quickly are likely to be disease-free.

This could mean that they and their offspring have less natural immunity. If this is the case, it could help scientists develop some sort of biological defence against the toads.

"If you re-introduce [these] parasites at the edge of the range, perhaps you could slow down the invasion," said Dr Phillips. He and his colleagues plan to study the creatures in more detail in the hope of pinpointing some of these biological weak spots.

Cane toads were introduced to Australia in 1935, to north tropical Queensland to control sugar cane pests. They failed to do this, but succeeded in becoming one of the International Union for the Conservation of Nature's (IUCN) top 100 invasive species. Their range now extends through most of Queensland and into Australia's Northern Territory.

"They're certainly up there with the worst invasive species," said Dr Phillips. "They're doing well for themselves, you have to give them that." The work was published in the Journal of Evolutionary Biology.

Need Willpower? Clench Up

Analysis by Liz Day

People often clench their muscles as a response to an unfavorable stimulus -- say, clenching your teeth before the climax in a horror movie or tightening your legs before impact in an accident.

But what about tightening your muscles as a proactive measure? Can tensing the muscles ease the short-term experience of unpleasant situations, such as ingesting nasty medicine, for long-term gain?

Indeed, that's what researchers Iris Hung of the National University of Singapore and Aparna Labroo from the University of Chicago found in their study published in the Journal of Consumer Research.

The researchers designed four studies to put participants through a range of self-control dilemmas that involved accepting immediate pain for a longer-term good. One study involved participants submerging their hands in ice buckets, another had participants imbibe a vinegary swill that boosted health but tasted awful and a third exposed the participants to disturbing photos of Haitian earthquake victims in need of help.

The fourth study is tamer yet commonplace. Participants shopped for food choices in a cafeteria under observation. In all four studies, the muscle clenchers exercised self control for their greater benefit.

"Participants who were instructed to tighten their muscles, regardless of which muscles they tightened -- hand, finger, calf or biceps -- while trying to exert self-control demonstrated greater ability to withstand the pain, consume the unpleasant medicine, attend to the immediately disturbing but essential information or overcome tempting foods," the authors write.

However, two restrictions were found. First, muscle tightening will only work if the choice aligns with the participants' goals. They have to want to eat healthier to be affected by the self-control clench. Second, the tightening has to occur at the moment of decision. Pre-clenching only wears one out.

The researchers believe the mind-body connection accounts for the muscle-willpower impact.

"Simply engaging in these bodily actions can serve as a non-conscious source to recruit willpower, facilitate self-control and improve consumer wellbeing," the authors write.

So next time a tempting vice catches your eye, clench your muscles and soldier on.

Marijuana Soda Provides a High Without the Smoke

Analysis by David Teeghman

One Colorado soda company has developed a line of sodas that have an unusual ingredient: marijuana. Dixie Elixirs has made their drinks available to anyone with a prescription for medical marijuana.

The drinks come in eight different flavors, including pink lemonade, root beer and grape. But if the company really wants to get their drinks into the hands of marijuana lovers, they may want to start working on pizza and nachos flavors.

But marijuana is only legal to consume in 14 states with a prescription from a doctor. So, unless you are one of the approximately half-million people who is a medical marijuana patient, this pot-infused soda won't do you much good.

It's an open secret that you can smoke marijuana and still be a valuable part of society. But when you think of smoking weed, you're more likely to think of Cheech and Chong than the people running the United States government. (Even though at least two U.S. presidents have admitted smoking it.)

The drink makers say part of the reason they developed their line of mary-jane drinks was to remove that "reefer madness" stigma associated with marijuana smokers. If California voters decide to make recreational marijuana legal this November, you may start seeing these organic sodas (the drink makers really know their audience) in grocery stores and liquor stores right next to the stuff from Pepsi and Coke.

But if Coca-Cola's history is any sign of what the company might do next, they could return to the heady days of putting mind-altering substances in their sodas.

At a time of sagging soda sales, drink makers are looking for a way to boost sales, and marijuana might be the answer. Medical marijuana has already proven an effective way at boosting newspaper sales, of all things. The New York Times reports that medical marijuana ads in small Colorado newspapers boost revenues enough for it to increase the size of its staff.

'Lubricin' molecule discovered to reduce cartilage wear

Research presented today at AVS Meeting in Albuquerque has implications for osteoarthritis

WASHINGTON, D.C., - A team of researchers in North Carolina has discovered that lubricin, a synovial fluid glycoprotein, reduces wear to bone cartilage. This result, which has implications for the treatment of sufferers of osteoarthritis, will be presented today at the AVS 57th International Symposium & Exhibition, taking place this week at the Albuquerque Convention Center in New Mexico.

Osteoarthritis is the most common form of arthritis, the degenerative joint disease. It mostly affects cartilage, the slippery tissue that covers the ends of bones where they meet to form a joint, and allows bones to glide over one another with limited friction and wear. Osteoarthritis causes cartilage to be broken down through a vicious cycle of mechanical and metabolic factors, and mechanical wear of cartilage is widely believed to contribute to this process. Eventually, the bones under the cartilage rub together, which can cause a tremendous amount of pain, swelling, and loss of motion at the joint.

Many studies have examined cartilage friction and lubrication with the goal of understanding cartilage wear prevention. Very few studies have focused on measuring wear directly, though, and until now no other studies have directly assessed the effects of synovial fluid constituents in mediating wear.

"We measured the effect of the synovial fluid protein lubricin on cartilage wear," explains research team member Stefan Zauscher, an associate professor of mechanical engineering and materials science, as well as biomedical engineering, at Duke University in Durham, N.C.

"Our measurements were performed at the surface level using an atomic force microscope with pressures and sliding speeds comparable to those seen in joints. The measurements show a direct link between lubricin in solution and reduction of cartilage wear," says Zauscher.

This indicates that lubricin is important for cartilage preservation physiologically, which may have important implications for treating or preventing joint disease in the future.

The presentation, "Lubricin Reduces Microscale Cartilage Wear" is at 4:00 p.m. on Wednesday, October 20, 2010.
ABSTRACT: http://www.avssymposium.org/Open/SearchPapers.aspx?PaperNumber=TR+NS+SS-WeA-7

Energy revolution key to complex life

The evolution of complex life is strictly dependent on mitochondria, the tiny power stations found in all complex cells, according to a new study by Dr Nick Lane, from UCL (University College London), and Dr William Martin, from the University of Dusseldorf.

"The underlying principles are universal. Energy is vital, even in the realm of evolutionary inventions," said Dr Lane, UCL Department of Genetics, Evolution and Environment. "Even aliens will need mitochondria."

For 70 years scientists have reasoned that evolution of nucleus was the key to complex life. Now, in work published today in Nature, Lane and Martin reveal that in fact mitochondria were fundamental to the development of complex innovations like the nucleus because of their function as power stations in the cell.

"This overturns the traditional view that the jump to complex 'eukaryotic' cells simply required the right kinds of mutations. It actually required a kind of industrial revolution in terms of energy production," explained Dr Lane.

At the level of our cells, humans have far more in common with mushrooms, magnolias and marigolds than we do with bacteria. The reason is that complex cells like those of plants, animals and fungi have specialized compartments including an information centre, the nucleus, and power stations – mitochondria. These compartmentalised cells are called 'eukaryotic', and they all share a common ancestor that arose just once in four billion years of evolution.

Scientists now know that this common ancestor, 'the first eukaryote', was a lot more sophisticated than any known bacterium. It had thousands more genes and proteins than any bacterium, despite sharing other features, like the genetic code. But what enabled eukaryotes to accumulate all these extra genes and proteins? And why don't bacteria bother?

By focusing on the energy available per gene, Lane and Martin showed that an average eukaryotic cell can support an astonishing 200,000 times more genes than bacteria.

"This gives eukaryotes the genetic raw material that enables them to accumulate new genes, big gene families and regulatory systems on a scale that is totally unaffordable to bacteria," said Dr Lane. "It's the basis of complexity, even if it's not always used."

"Bacteria are at the bottom of a deep chasm in the energy landscape, and they never found a way out," explained Dr Martin. "Mitochondria give eukaryotes four or five orders of magnitude more energy per gene, and that enabled them to tunnel straight through the walls of the chasm."

The authors went on to address a second question: why can't bacteria just compartmentalise themselves to gain all the advantages of having mitochondria? They often made a start but never got very far.

The answer lies in the tiny mitochondrial genome. These genes are needed for cell respiration, and without them eukaryotic cells die. If cells get bigger and more energetic, they need more copies of these mitochondrial genes to stay alive.

Bacteria face exactly the same problem. They can deal with it by making thousands of copies of their entire genome – as many as 600,000 copies in the case of giant bacterial cells like Epulopiscium, an extreme case that lives only in the unusual guts of surgeonfish. But all this DNA has a big energetic cost that cripples even giant bacteria – stopping them from turning into more complex eukaryotes. "The only way out", said Dr Lane, "is if one cell somehow gets inside another one – an endosymbiosis."

Cells compete among themselves. When living inside other cells they tend to cut corners, relying on their host cell wherever possible. Over evolutionary time, they lose unnecessary genes and become streamlined, ultimately leaving them with a tiny fraction of the genes they started out with: only the ones they really need.

The key to complexity is that these few remaining genes weigh almost nothing. Calculate the energy needed to support a normal bacterial genome in thousands of copies and the cost is prohibitive. Do it for the tiny mitochondrial genome and the cost is easily affordable, as shown in the Nature paper. The difference is the amount of DNA that could be supported in the nucleus, not as repetitive copies of the same old genes, but as the raw material for new evolution.

"If evolution works like a tinkerer, evolution with mitochondria works like a corps of engineers," said Dr Martin. The trouble is that, while cells within cells are common in eukaryotes, which often engulf other cells, they're vanishingly rare in more rigid bacteria. And that, Lane and Martin conclude, may well explain why complex life – eukaryotes – only evolved once in all of Earth's history.

'The energetics of genome complexity' is published in the 21 October issue of Nature. Journalists can obtain copies of the paper by contacting UCL Media Relations.

A redeeming role for a common virus

Washington, DC – A common virus that can cause coughing and mild diarrhea appears to have a major redemptive quality: the ability to kill cancer. Harnessing that power, researchers at Georgetown Lombardi Comprehensive Cancer Center, part of Georgetown University Medical Center, are conducting a clinical trial to see if the virus can target and kill certain tumor types.

By the age of five, most people have been exposed to the virus, called reovirus. For some, it can trigger brief episodes of coughing or diarrhea while many other don't develop any symptoms. The body simply overpowers the virus. But what scientists have discovered is that the virus grows like gangbusters inside tumor cells with a specific malfunction that leads to tumor growth. That finding led researchers to ask: Is it possible to use the virus as a treatment?

At Lombardi, researchers are collaborating with other institutions to look for an answer by conducting a phase II clinical trial for people with advanced or recurrent non-small cell lung cancer with a specific tumor profile.

"With reovirus, we're able to accentuate the positive and attenuate the negative," says the study's lead investigator at Lombardi, Deepa Subramaniam, MD, interim-chief of the Thoracic Medical Oncology Program. In other words, researchers have genetically altered the virus so that it won't replicate in a healthy cell (attenuated), which is what makes a person sick. "What's left is a virus in search of a host, and reovirus loves the environment inside a specific kind of cancer cell," explains Subramaniam.

That specific kind of cancer cell is one with malfunctioning machinery called KRAS or EGFR mutation.

"These mutations leave the cancer vulnerable to a viral take-over. Once it's in, the reovirus exploits the cell's machinery to drive its own replication. As a result, the cell is filled with virus particles causing it to literally explode."

Volunteers in the clinical trial will receive reovirus (REOLYSIN®) in addition to paclitaxel and carboplatin. The physicians will watch to see if the cancer shrinks while also seeing if this combination of drugs causes serious side effects.

"This is a subset of cancer where we haven't had many successes in terms of finding drugs that extend life after diagnosis," says Subramaniam. "This trial represents an attempt to seek and destroy cancer by choosing a treatment based on specific tumor characteristics. Preliminary data from the study should come quickly."

Researchers are also studying the effect of reovirus in other cancer types.

Patients interested in learning about this or any other clinical trial should call 202-444-4000.

This study is sponsored by Oncolytics Biotech Inc., maker of REOLYSIN. Subramaniam reports no potential financial interest.

'Unsafe' drug found in herbal tea

Herbal medicines marketed as weight loss aids have been found to include a drug withdrawn in Europe and US on safety grounds. Analysis of Payouji tea and Pai You Guo Slim capsules by the UK medicines watchdog revealed they contained diet drug sibutramine. It was taken off the market in January 2010 over fears it increased the risk of heart attack and stroke. Anyone taking the drug was urged to stop and consult their doctor.

Sibutramine was first approved as an anti-obesity drug in 1997, but data from recent studies suggests a higher rate of heart attacks and strokes among people taking it.

While it was withdrawn in Europe in January, it was only withdrawn in the US earlier this month.

Various herbal products in the US have been recalled because they were found to contain the drug.

The Medicines and Healthcare Regulatory Authority, which oversees medicines in the UK, said that any product containing the drug was considered "harmful to human health". They said that not only could it have side effects including high blood pressure, seizures, heart attacks or strokes, but could interfere with other prescription medicines.

Its head of herbal policy, Richard Woodfield, said: "People need to be aware that Payouji tea and Pai You Guo Slim Capsules are unlicensed herbal medicines and therefore have not met assured standards."

Anyone who experiences side-effects after taking a herbal medicine can report this to the MHRA using its Yellow Card Scheme.

NASA-engineered collision spills new Moon secrets

PROVIDENCE, R.I. [Brown University] - Scientists led by Brown University are offering the first detailed explanation of the crater formed when a NASA rocket slammed into the Moon last fall and information about the composition of the lunar soil at the poles that never has been sampled. The findings are published in a set of papers in Science stemming from the successful NASA mission, called LCROSS for Lunar CRater Observing and Sensing Satellite.

Mission control at NASA Ames sent the emptied upper stage of a rocket crashing into the Cabeus crater near the Moon's south pole last October. A second spacecraft followed to analyze the ejected debris for signs of water and other constituents of the super-chilled lunar landscape.

In one of the papers, Brown planetary geologist Peter Schultz and graduate student Brendan Hermalyn, along with NASA scientists, write that the cloud kicked up by the rocket's impact showed the Moon's soil and subsurface is more complex than believed: Not only did the lunar regolith - the soil - contain water, it also harbored other compounds, such as hydroxyl, carbon monoxide, carbon dioxide, ammonia, free sodium, and, in a surprise, silver. Combined, the assortment of volatiles - the chemical elements weakly attached to regolith grains - gives scientists clues where they came from and how they got to the polar craters, many of which haven't seen sunlight for billions of years and are among the coldest spots in the solar system.

Schultz, lead author on the Science paper detailing the impact crater and the ejecta cloud, thinks many of the volatiles originated with the billions of years-long fusillade of comets, asteroids and meteoroids that have pummeled the Moon. He thinks an assortment of elements and compounds, deposited in the regolith all over the Moon, could have been quickly liberated by later small impacts or could have been heated by the sun, supplying them with energy to escape and move around until they reached the poles, where they became trapped beneath shadows of the frigid craters.

"This place looks like it's a treasure chest of elements, of compounds that have been released all over the Moon," Schultz said, "and they've been put in this bucket in the permanent shadows."

Schultz believes the variety of volatiles found in Cabeus crater's soil implies a kind of tug of war between what is being accumulated and what is being lost to the tenuous lunar atmosphere.

"There's a balance between delivery and removal," explained Schultz, who has been on the Brown faculty since 1984 and has been studying the Moon since the 1960s. "This suggests the delivery is winning. We're collecting material, not simply getting rid of it."

Astronauts sent as part of NASA's Apollo missions found trace amounts of silver, along with gold, on the near-side (Earth-facing side) of the Moon. The discovery of silver at Cabeus crater suggests that silver atoms throughout the moon migrated to the poles. Nevertheless, the concentration detected from Cabeus "doesn't mean we can go mining for it," Schultz said.

The crater formed by the rocket's impact within Cabeus produced a hole 70 to 100 feet in diameter and tossed up six-foot deep lunar material. The plume of debris kicked up by the impact reached more than a half-mile above the floor of Cabeus, high enough to rise into sunlight, where its properties could be measured for almost four minutes by a variety of spectroscopic instruments. The amount of ejecta measured was almost two tons, the scientists report. The scientists also noted there was a slight delay, lasting roughly one-third of a second, in the flash generated after the collision. This indicated to them that the surface struck may be different than the loose, almost crunchy surface trod by the Apollo astronauts.

"If it had been simply lunar dust, then it would have heated up immediately and brightened immediately," Schultz said. "But this didn't happen."

The scientists also noticed a one-half-mile, near-vertical column of ejecta still returning to the surface. Even better, the LCROSS spacecraft was able to observe the plume as it followed on the heels of the crashing rocket. Schultz and Hermalyn had observed such a plume when conducting crater-impact experiments using hollow spheres (that mimicked the rocket that crashed into Cabeus) at the NASA Ames Vertical Gun Range in California before the LCROSS impact. “This was not your ordinary impact," Hermalyn said. "So in order to understand what we were going to see (with LCROSS) and maybe what effects that would have on the results, we had to do all these different experiments."

Even though the mission has been judged a success, Schultz said it posed at least as many questions as it answered. "There's this archive of billions of years (in the Moon's permanently shadowed craters)," Schultz said. "There could be clues there to our Earth's history, our solar system, our galaxy. And it's all just sitting there, this hidden history, just begging us to go back."

Contributing authors on the paper include Anthony Colaprete, Kimberly Ennico, Mark Shirley, and William Marshall, all from NASA Ames Research Center in California. NASA funded the research.

Why complex life probably evolved only once

* 12:52 21 October 2010 **by Michael Le Page**

The universe may be teeming with simple cells like bacteria, but more complex life – including intelligent life – is probably very rare. That is the conclusion of a radical rethink of what it took for complex life to evolve here on Earth. It suggests that complex alien life-forms could only evolve if an event that happened just once in Earth's history was repeated somewhere else.

All animals, plants and fungi evolved from one ancestor, the first ever complex, or "eukaryotic", cell. This common ancestor had itself evolved from simple bacteria, but it has long been a mystery why this seems to have happened only once: bacteria, after all, have been around for billions of years.

The answer, say Nick Lane of University College London and Bill Martin of the University of Düsseldorf in Germany, is that whenever simple cells start to become more complex, they run into problems generating enough energy. "It required a kind of industrial revolution in terms of energy production," says Lane. "[Our hypothesis] overturns the traditional view that the jump to complex eukaryotic cells simply required the right kinds of mutations."

"It is very, very convincing, in my opinion," says biologist John Allen of Queen Mary, University of London, on whose work Lane and Martin have drawn.

Growing costs

To become more complex, cells need more genes and more proteins – and so they need to get bigger. As the volume of any object increases, however, its relative surface area falls: an elephant has less surface area per unit of volume than a mouse, for instance. This is a major problem because simple cells generate the energy they need using the membrane that encloses them.

Lane and Martin calculate that if a bacterium grew to the size of a complex cell, it would run out of juice. It might have space for lots of genes, but it would have barely enough energy to make proteins from them.

Folds don't help

In theory, there is an easy answer to the energy problem: create lots of folds in the cell membrane to increase its surface area, which in turn will increase the amount of energy the membrane can produce. Indeed, many bacteria have such folds. But this leads to another problem as they get larger.

Producing energy by "burning" food is playing with fire. If the energy-producing machinery straddling the membrane is not constantly fine-tuned, it produces highly reactive molecules that can destroy cells. Yet fine-tuning a larger membrane is problematic because detecting and fixing problems takes longer.

These obstacles were overcome when a cell engulfed some bacteria and started using them as power generators – the first mitochondria. By increasing the number of mitochondria, cells could increase their membrane area without creating maintenance problems: each mitochondrion is a self-contained system with built-in control and repair mechanisms.

Birth of complexity

Once freed from energy restraints, genomes could expand dramatically and cells capable of complex functions – such as communicating with each other and having specialised jobs – could evolve. Complex life was born.

So if Lane and Martin are right, the textbook idea that complex cells evolved first and only later gained mitochondria is completely wrong: cells could not become complex until they acquired mitochondria.

Simple cells hardly ever engulf other cells, however – and therein lies the catch. Acquiring mitochondria, it seems, was a one-off event. This leads Lane and Martin to their most striking conclusion: simple cells on other planets might thrive for aeons without complex life ever arising. Or, as Lane puts it: "The underlying principles are universal. Even aliens need mitochondria."

Journal reference: Nature, vol 467, p 929

Sequencing the "Exposome": Researchers Take a Cue from Genomics to Decipher Environmental Exposure's Links to Disease

Technological and analytical advances are helping to take environmental exposures the route of genetic profiles in predicting a person's disease risk with greater precision

By Katherine Harmon Thursday, October 21, 2010 2

Anxious about BPA? Petrified of pesticides? Plenty of scientific literature shows that concerns about certain chemicals' potential to up the risk for chronic disease are justified. And although genetics can predispose a person to many ills, more than half of disease risks—and possibly as much as 90 percent—likely stem from environmental factors, according to recent epidemiological research.

Hard data—of the quality now gleaned from genetic studies—however, has been lacking in the environmental field. And if there is to be any hope of untangling the complex web of risks behind chronic diseases, many scientists argue, researchers need to develop an "exposome," a highly detailed map of environmental exposures that might occur throughout a lifetime, which can be mapped onto the etiology (the study of causes) of major illnesses, including cancer, diabetes and heart disease.

Environmental factors have long been relegated to questionnaires in epidemiological research, often requiring subjects to estimate a lifetime of exposure in a single question. Even for studies that have focused on environmental correlations, researchers "just ask people what their exposures are," Steve Rappaport, a professor of environmental health at the University of California, Berkeley, says. "How can you imagine you're going to get any resolution like that?"

Although broad-picture trends can help researchers draw connections among exposures and disease, with those loose associations, "you can't really tell what's going on" at a biological level, he notes.

Furthermore, chemicals do not just enter the body and persist in an isolated state. Once inside, they can interact with a wide range of cells inside the body, and often themselves undergo changes.

And chemical exposures of interest to researchers such as Rappaport do not only come from the world beyond our skin, but from natural processes occurring within our bodies as well. From biological processes that produce oxidative stress or inflammation, our bodies face a constantly changing internal environment. "We really need to think about the environment as what's going on inside the body and accept things that come from every source," says Rappaport, who lays out evidence in support of moving forward with exposome research in an essay published online October 21 in Science.

"We've never looked at the whole environment we have inside our bodies in a way that allows us to discover these things," Rappaport says.

But how does one profile a seemingly infinite set of external—and internal—factors?

Atul Butte, an assistant professor at Stanford University School of Medicine who has also worked in the field of environmental links to disease, admits that it is no small task. "You're looking at an unbounded set of variables here," he says. "But that doesn't mean we shouldn't try to start measuring them."

The toxins within

Many environmental risk studies have turned their sights on the everyday world people inhabit. Children and mothers-to-be have toted around air quality monitors; researchers have sampled drinking water for a litany of compounds. But Rappaport and his co-author, Martyn Smith, also a professor at Berkeley's School of Public Health, argued in their new essay that exposures should be assessed from within the body, such as via blood samples.

"People do think of chemical exposures are coming in from outside the body," Rappaport says. But, he argues, "if people are always thinking about air pollution and water pollution, we're not going to get very far." In fact, he adds, "there are so many natural processes that produce chemicals that are toxic—and are going on right inside the body."

And when compared with headline-grabbing pollutants, such as bisphenol A, phthalates or benzene, toxic exposures coming from within the body are much more common. "The blood concentrations of these things are really high compared to the concentrations you get from exogenous chemicals," Rappaport notes.

New findings about the human microbiome, for example, have shown that even more that we could have—or might have liked to—imagine. "We're full of bacteria that are generating waste products," Rappaport says. And from an exposure standpoint, "there's just no reason to think that we can ignore something like that."

Counting chemicals

Before researchers can start mapping out patterns among the constellations of environmental exposures, however, they need to assemble a more comprehensive picture of the possible internal and external exposures.

"It's complicated," Rappaport concedes, of embarking on the massive endeavor. "But if you look at it from the perspective of the Human Genome Project that we tackled about 20 years ago, I don't think it's any more daunting."

The dearth of environmental data stems mainly from a preoccupation with the seemingly sexier field of genetic correlates, Rappaport says. "People have been spending all of their time and energy and money looking at the genetic factors," he notes. So in terms of understanding factors at work on the environmental—and arguably more potent—side, "they've hardly scratched the surface."

There has been some progress, however. Thousands of small-molecule metabolites have been profiled in the effort to develop a chemical signature profile, or metabolome. But the few studies that have been done have taken relatively smaller samples of available chemical readings to assess.

One study, published in May in PLoS ONE and co-authored by Butte, scanned blood and urine samples of thousands of people for the presence of different chemical compounds, looking for correlates with type 2 diabetes. "I think that's really a good example of what we should be able to do," Rappaport says.

The study, however, was not quite as strong as a contemporary genome-wide association study (GWAS) would be, Rappaport notes. He explains that a true GWAS surveys hundreds of thousands of genes and the diabetes study looked only at 266 environmental chemicals.

This reduced approach can lead to both false-positive associations and more robust correlations being missed. These smaller chemical sample studies are more "analogous to what they call a candidate gene study," in which researchers assess only a handful of likely genes," than to a GWAS, Rappaport notes. And to take a lesson from the genomics field, he says, after a GWAS follow-up original flagged genes from candidate studies "almost always turn out not to be important."

Joining genetics

Much of the allure of the Human Genome Project as a model for other fields is its allegiance to the data. Its search-and-map mandate allowed for a largely unbiased survey of the genome. Such a clean plan, however, has thus far proved difficult in a field often tainted—and indeed driven—by sweeping chemical- and disease-specific hypotheses.

Rappaport and Smith argued in their essay that the familiar tactics of single-source and single-disease research are premature and should be put on hold in favor of more expansive investigation of all internal and external exposures. "We're at the point now where we don't really know what's important," Rappaport says. So surveying every possible exposure—and combination of exposures—is crucial, he notes.

A comparable environmental data set to the human genome, such as the exposome, however, is still a ways off, and its completion depends on support by major research funders, such as the National Institutes of Health. The NIH is in the midst of a $200-million, five-year program called the Genes, Environment and Health Initiative (GEI). Nevertheless, Rappaport says, such pairings are still challenging, given the lack of data about the environmental exposure side of the equation.

"Even if we keep making progress on the genetics of these diseases, we have to keep studying the environment," Butte says. "Maybe a variant of a gene only leads to a disease when an individual happens to be in an environment that we don't even know about," he says. Getting to the bottom of environmental exposure has the potential to clarify many of the diseases and genetic mutations that currently seem completely random.

The long-standing division between genetics and environment itself might need some blurring. "We set up these artificial constructs going back a hundred years," Butte says. "It makes us think it's either a genetic factor or it's an environmental factor, but in reality most of these are probably hybrid factors."

Improving our understanding of chronic disease risk will thus require more cross-disciplinary partnerships like the NIH's GEI. "Most geneticists think of the environment as a confounder to their genetic studies," Butte says. But he proffers the altered adage: "One researcher's confounder is another researcher's signal."

The work will also require a boost in technology, similar to that which has come to speed along the genomic field with high-throughput genetic sequencing. Fast, affordable and comprehensive chemical analyses could go a long way toward collecting the immense quantity of data needed to start better parsing environment's role in disease risk. Butte hopes that study subjects and even willing hospital patients will start to be screened as a matter of course to help amass the vast quantities of data needed to start making sound disease-exposure connections.

And ultimately that data could start paying dividends. "These are things you could eventually see in a doctor's office," Rappaport says. "It wouldn't take a great stretch of the imagination" to expect physicians to regularly screen patients for 100 or so of the most implicated chemicals to assess disease risk, much the way blood work today can reveal high cholesterol or other red flags. Such a practical application, he notes, would fit in well with the vision of "the future of medicine being more predictive and more personalized."

70-year-olds smarter than they used to be

Today´s 70-year-olds do far better in intelligence tests than their predecessors. It has also become more difficult to detect dementia in its early stages, though forgetfulness is still an early symptom, reveals new research from the University of Gothenburg, Sweden, based on the H70 study.

The H70 study provides data on cognitive symptoms that researchers have used to predict the development of dementia, and also to investigate whether the symptoms have changed in recent generations. The study involves a large proportion of 70-year-olds from Gothenburg, Sweden, who have been extensively examined over the years, including tests that measure memory, speed, language, logic and spatial awareness. New results from the study were published earlier this year in the reputable American journal Neurology.

"Using the test results, we've tried to identify people who are at risk of developing dementia," says Simona Sacuiu, resident in psychiatry at Sahlgrenska University Hospital and medical researcher at the Sahlgrenska Academy's Unit of Neuropsychiatric Epidemiology. "While this worked well for the group of 70-year-olds born in 1901-02, the same tests didn't offer any clues about who will develop dementia in the later generation of 70-year-olds born in 1930."

The 70-year-olds born in 1930 and examined in 2000 performed better in the intelligence tests than their predecessors born in 1901-02 and examined in 1971. There were no differences in test results between 70-year-olds who developed dementia and those who did not over the next five years in the group born in 1930 and examined in 2000, while many of the tests identified early signs of dementia in the group born in 1901-02.

"The improvement can partly be explained by better pre- and neonatal care, better nutrition, higher quality of education, better treatment of high blood pressure and other vascular diseases, and not least the higher

intellectual requirements of today's society, where access to advanced technology, television and the Internet has become part of everyday life," says Dr. Sacuiu.

The study showed that memory problems were the only predictor of which 70-year-olds were at risk of developing dementia. However, far from all of the 70-year-olds with a poor memory went on to develop the illness. "That's why it's important for people with memory problems to receive a thorough examination," explains Dr. Sacuiu. "If we are to identify dementia effectively at an early stage, we need good tools that include psychometric tests. However, these must constantly be adapted to new generations, as older people are performing better and better in standardised psychometric tests."

At the same time, the incidence of dementia remained unchanged – it is just as common between the age of 70 and 75 today as it was 30 years ago. The study included over 800 dementia-free 70-year-olds, 5% of whom went on to develop the illness over the subsequent five years. "Learning more about the early signs of dementia means that patients may get help and support more quickly," says Dr. Sacuiu.

THE H70 STUDY

The study started in 1971 with an examination of 70-year-olds who were then regularly followed over a period of 30 years. A new H70 study started in the year 2000, and is still ongoing. Data from a total of more than 2,000 senior Gothenburg residents are included in these studies. The participants have been examined both physically and psychiatrically and have enabled several research groups to describe different trends in physical and mental health in the aging population.

UMMS biomedical researchers develop more reliable, less expensive synthetic graft material

FlexBone seen as potential replacement for current bone transplantations

WORCESTER, Mass. – With a failure rate as high as 50 percent, bone tissue grafts pose a significant obstacle to orthopedic surgeons attempting to repair complex fractures or large areas of bone loss, such as those often caused by trauma and cancer. Current synthetic substitutes rarely possess the bone-like properties needed for successful grafting and are often difficult for surgeons to manipulate in the operating room. In response to these challenges, researchers at UMass Medical School have developed an easy-to-produce, inexpensive, synthetic bone material called FlexBone.

Building upon previous development of a material that combines a key mineral found in bone (nanocrystalline hydroxyapatite) with a hydrogel similar to that used in contact lenses, Jie Song, PhD, assistant professor of orthopedics & physical rehabilitation and cell biology, and a team of graduate students and orthopedic surgeons, along with their collaborators at the University of Michigan, have created a bone substitute that can be press-fit into a bony lesion.

"Functionally sophisticated synthetic materials don't have to be complicated to manufacture or difficult to reproduce," said Dr. Song. "Our idea was to create an inexpensive, off-the-shelf product that can be easily manipulated in the operating room to fill large bone voids and facilitate the tissue repair." Research published online ahead of print in Tissue Engineering Part A describes the efficacy of the FlexBone as a synthetic bone substitute in repairing large bone defects in animal models.

In large, complex bone voids caused by trauma or tumor removal, stabilization with traditional metal plates and other internal and external fixation devices often isn't enough to facilitate healing. In many cases, surgeons turn to bone tissue grafts to bridge the gap left by the break, transplanting bone from another donor. Complications from infection, immune response or incomplete union between the transplanted and host tissue, however, result in almost 50 percent of these procedures failing. Synthetic substitutes, meanwhile, do not have the necessary bone-like properties to make them an ideal alternative.

David Ayers, MD, the Arthur M. Pappas, MD, Chair in Orthopedics and chair and professor of orthopedics & physical rehabilitation said, "FlexBone has a bone mineral content approaching that of human bone, enabling the elastic FlexBone material to be cut and shaped prior to surgery or intraoperatively and then pressed into a bone gap. When used in conjunction with traditional fixation techniques, the FlexBone material provides ideal scaffolding for new bone growth."

The density of the FlexBone material also allows surgeons to pre-drill channels in it, allowing for bone marrow from adjacent bone to migrate and penetrate. This helps to attract progenitor cells that are critical to new bone formation.

Beyond the benefits of its physical properties, FlexBone has also proved to be an ideal material for speeding recovery. "What makes FlexBone so ideal for healing large bone gaps is that it absorbs and retains the proteins associated with the natural healing process from the surrounding tissue once implanted," said Song. "This helps accelerate healing." Conversely, it can also be loaded with therapeutic agents, such as protein factors and antibiotics that can facilitate faster healing and fight infection through localized and controlled delivery over a sustained period of time.

"Because of this combination of factors, our study shows that FlexBone, combined with a protein growth factor in a dose 100 times less than what currently needed, was able to heal a large, long bone defect that would not heal on its own in a short period of time," said Song. "This material has enormous potential to solve a major problem that orthopedic surgeons face when reconstructing large bone deficits in the skeleton."

"Its ability to deliver growth factors and antibiotics to the patient and the handling characteristics simplifying the surgical procedure combine to make this material very exciting," said Dr. Ayers.

Song and Ayers would like to next test the safety and efficacy of the material in large animals, which they hope will pave the way for future clinical trials.

Scorpion has welcome sting for heart bypass patients

A toxin found in the venom of the Central American bark scorpion (Centruroides margaritatus) could hold the key to reducing heart bypass failures, according to research from the University of Leeds.

The study, published online in Cardiovascular Research, reports that one of the scorpion's toxins, margatoxin, is at least 100 times more potent at preventing neointimal hyperplasia – the most common cause of bypass graft failure - than any other known compound. Neointimal hyperplasia is the blood vessel's response to injury. It triggers the growth of new cells, causing chronic obstruction on the inside of the vessel.

When a vein is grafted onto the heart during a bypass procedure, the injury response kicks in as the vein tries to adapt to the new environment and different circulatory pressures. Whilst the growth of new cells helps to strengthen the vein, the internal cell growth restricts blood flow and ultimately causes the graft to fail.

The potency of the margatoxin in suppressing the injury response mechanism took the team by surprise, says lead author Professor Beech from the University's Faculty of Biological Sciences. "It's staggeringly potent. We're talking about needing very few molecules in order to obtain an effect."

The toxin works by inhibiting the activity of a specific potassium ion channel - a pore in the cell membrane that opens and closes in response to electrical signals and indirectly enhances delivery of a intracellular messenger, the calcium ion.

"We knew from experimental research in immunology that the ion channel Kv1.3 is involved in activating immune system responses and that it's linked with chronic inflammation problems in the immune system, such as those you see with multiple sclerosis," says Professor Beech. "Since our own studies had identified Kv1.3's presence in injured blood vessels, which are also often complicated by chronic inflammation, we wanted to see if the same immune system blockers would inhibit neointimal hyperplasia."

"There were a number of good blockers of this ion channel available to screen. Several compounds are developed from plants, and one comes from scorpion venom," he says, "but margatoxin was the most potent of all these compounds by a significant margin."

Professor Beech says margatoxin would probably be unsuitable as a drug that could be swallowed, inhaled or injected, but it could potentially be taken forward as a spray-on treatment to the vein itself once it's been removed and is waiting to be grafted onto the heart.

The research was funded by the British Heart Foundation, the Wellcome Trust and the Medical Research Council.

Low-dose aspirin slashes colon cancer risk - study

Low doses of aspirin, taken daily and over the long term, cut cases of colorectal cancer by a quarter and the death toll from this disease by a third, according to a study published online on Friday by The Lancet.

Aspirin is already recommended in low, daily doses by many doctors for patients at risk of a heart attack or a stroke. High doses of this cheap, over-the-counter medication have similarly been found to help prevent cancer of the rectum and colon. But, studies have also found, the benefits may well be outweighed by the risks, such as increased bleeding from high aspirin use.

Eager to verify whether low doses can also be protective, researchers followed up four trials in Britain and Sweden, conducted in the 1980s or early 1990s, on the cardiovascular impacts of aspirin.

The study entailed looking over centralised data banks to see whether volunteers in these trials had since died or been diagnosed with colorectal cancer. On average, the trials lasted six years, entailing volunteers who took either aspirin or a dummy lookalike pill called a placebo. The doses ranged up to 1,200 mg.

Out of 14,033 patients whose health could be traced 18 years or so since the trial, 391 had colorectal cancer, the investigators found. Taking aspirin reduced the risk of cancer by 24 percent and the risk of dying from it by 35 percent. The results were consistent across all four trials -- and there was no increase in benefits beyond a dose of 75 mg.

Where the reduction was most remarkable was in cases of proximal colon cancer. These occur in the upper colon and are thus liable to be missed in lower-intestine scans for polyps, the precursor of tumours.

The authors say their study had limits, as the original trials were not designed to look at aspects of colorectal cancer, nor was data available for any deaths from aspirin's side effects.

Also, aspirin's benefit may have been somewhat over-estimated, they said. This was because the original trials took place before colon screening for polyps became a routine practice in those countries.

Even so, the evidence has now swung the scales in favour of low-dosage aspirin for a disease that claims 600,000 deaths worldwide each year, they said.

"Our findings suggest that long-term low-dose aspirin treatment and sigmoidoscopy screening would combine to substantially reduce cancer incidence in all parts of the colon and rectum," said the lead author, Peter Rothwell, a professor at the Department of Clinical Neurology at Oxford University.

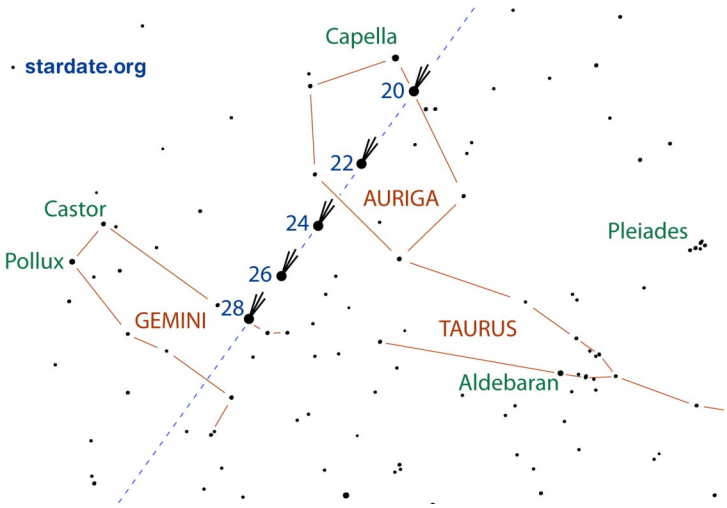
In a commentary, doctors Robert Benamouzig and Bernard Uzzan of the Avicenne Hospital in Bobigny, on the outskirts of Paris, said the study should unleash "the next logical step," of formulating guidelines for people at risk.

Colorectal cancer is the second commonest cancer in developed countries, with a lifetime risk of five percent, according to figures cited in the city. Aspirin is believed to have a preventive effect because it inhibits an enzyme called COX-2, which promomotes cell proliferation in colorectal tumours.

Get a glimpse of Comet Hartley 2 less than two weeks before NASA probe's flyby

By John Matson Oct 22, 2010 12:45 PM 4

Comet Hartley 2 from the EPOXI spacecraftNASA's EPOXI spacecraft is closing in on a comet called Hartley 2 in advance of a November 4 flyby, but stargazers on Earth are already getting a fairly good look at the icy object, which is currently in the midst of an unusually close passage of Earth.



Hartley 2 is now only about 18 million kilometers from Earth, the closest it has been since the comet was discovered nearly 25 years ago, and on October 28, the comet will make its closest approach to the sun. Hartley 2, which orbits the sun in an elongated ellipse every 6.5 years, is named for Malcolm Hartley, an English-born astronomer who first spotted it from an Australian observatory in 1986.

The comet is rather small, just over one kilometer in diameter, but may be visible to the naked eye under clear, dark skies, according to StarDate magazine, which has assembled a finder chart to locate Hartley 2 in the sky. (As always, a good pair of binoculars never hurts when trying to locate a relatively small, faint object.) Viewing the comet may be easier toward the end of the month, when Hartley 2 is closer to the sun and there is less moonlight in the night sky. Sky & Telescope made available a similar guide, which includes the path of the comet through the end of 2010 for more dedicated and better-equipped skywatchers.

EPOXI's impending flyby will bring the spacecraft to within about 700 kilometers of Comet Hartley 2 to investigate its structure and makeup. The planned rendezvous will not be the first comet encounter for the NASA probe. In 2005 the spacecraft, then known as Deep Impact, sent an impactor careening into Comet Tempel 1 to excavate a crater and get a glimpse of its composition.

Warmer Arctic Spells Colder Winters

By Kieran Mulvaney Fri Oct 22, 2010 01:58 PM ET

The Arctic is moving into "a new climate state" and a return to previous Arctic conditions is "unlikely," according to a new assessment from the National Oceanic and Atmospheric Administration (NOAA). One consequence of a warmer Arctic could be colder winters in other parts of the Northern Hemisphere.

The basic facts have been reported widely and often:

The area covered by sea ice hovered near its historic low this summer. In Greenland, record-high temperatures this year have helped accelerate the melting of the country's massive ice sheet. Throughout the Arctic, permafrost is warming and the blanket of snow is shrinking. Those changes appear to be long-lasting, said an international team of climate experts who wrote the National Oceanic and Atmospheric Administration report. [...] "The Arctic is a system, and the system is changing," said Don Perovich, a sea ice expert with the U.S. Army Corps of Engineers who worked on the report. "It's not just that sea ice is being reduced. There's changes in Greenland, the atmosphere, the ecosystem, and these changes are affecting human activity."

What is increasingly apparent, as researchers have warned for years, is that "polar amplification" is causing many of these changes to feed on themselves, amplifying each other year after year. In this regard, what is happening to Arctic sea ice is in many ways key.

As the National Snow and Ice Data Center (NSIDC) pointed out earlier this month, Arctic sea ice is not just diminishing in extent -- the four lowest levels have been in the last four years -- but it is also younger and thinner. That makes ice easier to melt each year, which in turn exposes greater areas of heat-trapping ocean, which causes further melting, making it more and more difficult for sea ice to recover to previous levels. Among other impacts, such changes in sea ice cover could have significant and seemingly contradictory impacts on weather patterns in mid-latitudes. Rising heat from a warmer Arctic may increasingly disrupt the circumpolar winds that normally confine cold air within Arctic realms, allowing blasts of cold to hurtle south, similar to what happened when parts of the United States were buried under thick snow this past winter.

As a consequence:

"While individual weather extreme events cannot be directly linked to larger scale climate changes, recent data analysis and modeling suggest a link between loss of sea ice and a shift to an increased impact from the Arctic on mid-latitude climate," concludes the report. "With future loss of sea ice, such conditions as winter 2009-2010 could happen more often. Thus we have a potential climate change paradox. Rather than a general warming everywhere, the loss of sea ice and a warmer Arctic can increase the impact of the Arctic on lower latitudes, bringing colder weather to southern locations."

Poop Plastic Puts Waste to Work

By Alyssa Danigelis Fri Oct 22, 2010 03:06 PM ET

It's ultimate dream: Take human waste and turn it into one of the most ubiquitous materials around, plastic. The Sacramento-based startup Micromidas has the technology to do it. And no, their polymer doesn't stink.

When Micromidas CEO John Bissell and his colleagues were undergrads at UC Davis, they recognized that plastic and sludge from wastewater treatment facilities were two very large problems. After graduating in 2008, they started their company to develop technology that could offer a solution.

Usually wastewater treatment facilities separate the liquids from the solids in a large settling tank, Bissell told me. He and his colleagues presented their innovation at the PopTech conference currently underway in Camden, Maine. The heaviest nastiness at the bottom is incinerated or sent to a landfill or used to grown non-edible crops that are tilled. Not great options. Micromidas can take that sludge and turn 50 to 70 percent of it into plastic by feeding it to their own special microbes.

"Traditionally in biotech you take a bug, play with its genetic code, and make a Michael Jordan bug for whatever you want to do," Bissell explained. "Instead, we form teams, like a pro sports teams." Their robust bugs eat the sludge, get really fat, and that fat turns into a form of polyester. Then, Bissell says, the bugs are killed and the polyester is extracted. This polyester is part of a family of plastics called polyhydroxyalkanoates or PHA.

PopTechLogo-278x225 Of the plastic, Bissell says that it will biodegrade nontoxically in the body. "Usually things are measured in terms of the lethal limit," he said. "There is no lethal limit to this. You'd have to choke on it for it to hurt you." Definitely wouldn't want to test that, but the properties of the plastic mean that it biodegrades in the environment within a year and a half, depending on the fill thickness. If the final plastic product doesn't have many additives, it could be composted in the yard, although Bissell points out that the requirements for industrial compostability are more stringent than backyard composting.

Micromidas's plastic is an analog to polyethylene, polystyrene, polypropylene, and PET, Bissell says. Between 20 to 60 percent of the applications that use those materials could probably use their polymer, or a blend with the polymer and other chemicals. In terms of cost, the company plans to either be on par with petroleum-based resin or actually beat it.

Currently the polymer is at the pilot stage, with the company operating a several-thousand-liter system that has a maximum capacity of a few kilos daily. They're starting to work with industrial partners on applications, but haven't publicly announced most of those companies yet. When I asked about the byproducts from this process, Bissell said that they actually have a negative water footprint because water is a residual. Sounds like a gold medal bug team to me.

Vaccines could help elephantiasis spread

PARASITIC worms can adjust their survival strategy based on their host's immune response. This means potential vaccines against elephantiasis might make the infection spread more easily through communities.

Elephantiasis infects 120 million people a year in Africa and Asia. Tiny filaria worms carried by mosquitoes block the lymph vessels that normally drain fluid from limbs or genitals, which then swell to grotesque proportions. The only prevention is a yearly dose of worming drugs, but fewer than half the people at risk receive them.

Work is under way on a vaccine, but Simon Babayan at the University of Edinburgh, UK, and colleagues, have discovered that some vaccines may make the worms worse. When filaria worms in mice sense that the

mouse is mounting a strong immune reaction, they change their life cycle, producing more offspring in the blood earlier. This helps the worm ensure that it will be picked up and transmitted by another mosquito despite the immune attack (PLoS Biology, DOI: 10.1371/journal.pbio.1000525).

Unfortunately, experimental vaccines rely on the very immune reactions that warn the worms, Babayan says. People who get such a vaccine may defeat their own infection, but the worms' early response means they will pass on more infections.

Babayan says potential vaccines should be tested for whether their targets adapt to them in this way.

Vancomycin is the drug of choice for treating cellulitis

DETROIT – Patients admitted to the hospital for the common bacterial skin infection cellulitis should be treated as a first line of defense with the potent antibiotic drug vancomycin rather than other antibiotics such as penicillin, according to a Henry Ford Hospital study.

For some time, medical practice guidelines have been ambiguous about whether vancomycin or so-called B-lactam antibiotics like penicillin or cephalosporins was the more appropriate therapy for treating patients admitted for cellulitis. If left untreated and infection spreads, cellulitis could become life threatening.

The Henry Ford study found that 226 patients treated intravenously with vancomycin between December 2005 and October 2008 fared better and were discharged on average one day earlier than 199 patients treated intravenously with the B-lactam antibiotics. The study is being presented Oct. 23 at the 48th annual meeting of the Infectious Diseases Society of America Oct. 21-24 in Vancouver.

"We believe vancomycin is the better treatment option for managing patients hospitalized with cellulitis," says Hiren Pokharna, M.D., an Infectious Diseases fellow at Henry Ford Hospital and the study's lead author.

With MRSA skin and soft tissue infections increasing, researchers sought to compare the two groups of antibiotics commonly used for treating hospitalized patients with cellulitis. The common bacterial skin infection is caused by many types of bacteria including staphylococcus and streptococcus. Symptoms include redness, swelling, tenderness and pain.

MRSA strains have proven resistant to common antibiotics like penicillin and other drugs. However, they have been shown to be susceptible to vancomycin. *The study was funded by Henry Ford Hospital.*

Generosity Might Keep Us Healthy

Christie Nicholson

At the PopTech conference in Camden Maine this weekend I caught up with social psychologist Liz Dunn who studies links between money and happiness. Recently she's found a possible link between generosity and physical health and I asked her about it:

"We did a little experiment where we gave people some money, ten dollars. And we said, "Hey, you can keep all this money for yourself or you can give as much of it as you want away." What we found, consistent with all our past research, was that the more money people gave away, the happier they felt. Conversely though, the more money people kept for themselves the more shame they experienced.

And the more shame people felt the more we saw their cortisol levels rise. Now this is important because cortisol is thought to explain some of the links that we've seen between stress and disease. So we know that over time elevated levels of cortisol cause wear and tear on the body.

So what we think is that we may be seeing just the first hint of this kind of missing link between generosity and health. So we know that a lot of generous behaviors are associated with consequences for health. Engaging in volunteer work is good for longevity. So why is that? Well, we are beginning to see that cortisol may play a role.