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# Coming Soon: A Low-Heartburn Coffee

(HealthDay News) -- For millions of coffee-lovers with delicate stomachs, scientists may have found a way to enjoy an eye-opening cup of java without gastrointestinal discomfort.

European researchers studying stomach-irritating chemicals in coffee have unexpectedly found one that actually inhibits acid production in the stomach.

"The major import of our work is that it provides scientific evidence that you can produce a more stomach-friendly coffee by varying the processing technology," said study author Veronika Somoza, professor and chair of the Research Platform of Molecular Food Science at the University of Vienna, Austria.

The finding offers the promise that coffee makers can produce a blend that will be easier on the tummy, Somoza said.

The scientists looked at coffee's effect on human stomach cells using a variety of preparations, including dark-roast, regular roast, decaffeinated and stomach-friendly. Instead of one single element, they identified a mixture of compounds -- caffeine, catechols and N-alkanoly-5-hydroxytriptamides -- as the chemicals in coffee that promote the production of stomach acid.

But a fourth chemical, N-methylpyridinium, which is more common in dark roasts, such as espresso and French roast blends, was found to inhibit acid.

N-methylpyridinium is a product of the roasting process itself, resulting in dark roasts that are less likely than lighter ones to cause stomach irritation, according to the research.

Whether the findings will translate to human coffee drinkers remains unclear. The authors hope to conduct tests with human coffee drinkers this year.

SOURCES: Veronika Somoza, Ph.D., professor and chair, Research Platform of Molecular Food Science, University of Vienna, Austria; Dr. Joseph Vinson, professor, chemistry, University of Scranton,pa ,March 21, 2010, presentation, American Chemical Society annual meeting, San Francisco .

Pharma Info

Inside This Issue

# FDA MedWatch Safety Alert. Zyprexa (olanzapine): Use in Adolescents.

January 29, 2010. The FDA is recommending that healthcare professionals use caution when considering Zyprexa (olanzapine) for treating adolescents 13 to 17 years old for schizophrenia and bipolar disorder.

Physicians, patients and caregivers should understand that adolescents may experience weight gain and hyperlipidemia with Zyprexa. In fact, they have a greater risk of these effects than adults. They are likely to gain more weight than adults and have greater increases in total cholesterol, LDL cholesterol, triglycerides, prolactin, and hepatic transaminase levels. Adolescents are also more likely to experience sedation than adults.

Clinicians should take these effects into account when deciding on antipsychotic medications for adolescents, and they may want to consider trying other drugs first.


If Zyprexa is prescribed for adolescents, it should be part of a comprehensive treatment program that often includes psychological, educational and social components. It is also important to note that Zyprexa has not been approved for patients under the age of 13.

***Source :***http//:www.fda.gov

# Clopidogrel: Caution or confusion?

Recently, the **Food and Drug Administration** (FDA) announced it is requiring a boxed warning to be added to the anticoagulant **clopidogrel** (Plavix, Bristol-Myers Squibb/Sanofi-Aventis) to caution that poor metabolizers of the drug may not receive its full benefits.

The boxed warning also states that tests are available to determine the genetic profile of a key liver enzyme and predict whether a patient will ineffectively convert clopidogrel to its active form. It advises clinicians to consider other antiplatelet medications or alternative dosing strategies for clopidogrel in poor metabolizers.

***Source:*** http://www.theheart.org/article/1063749.do

# Study Finds That Insulin-Producing Beta Cells Can Be Reborn

April 5, 2010 (HealthDay News) -- Certain cells in the pancreas can regenerate themselves into insulin-producing cells after normal insulin-producing cells have been destroyed, as happens in type 1 diabetes, a new study found.

Swiss researchers discovered that when they destroyed the insulin-producing cells, known as beta cells, in mice to induce an artificial form of type 1 diabetes, other cells in the pancreas called alpha cells then changed into insulin-producing beta cells.

"The adult pancreas can regenerate new beta cells even if they are totally absent -- like in type 1 diabetes," said the study's senior author, Pedro Herrera, a professor in the department of cell physiology and metabolism at the University of Geneva Medical School.

Diabetes experts cautioned, however, that far more research is needed to see if the process could benefit people with type 1 diabetes, an autoimmune disease in which the immune system attacks the beta cells in the pancreas that produce insulin, the hormone that allows people to convert food into energy. People with type 1 diabetes must rely on insulin therapy for the rest of the lives.

And if such a process occurs in humans, or could be induced to occur, one large roadblock remains. In type 1 diabetes, the immune system attack on beta cells appears to go on indefinitely, which is why people who've had transplants of insulin-producing cells eventually must go back on insulin. The immune system destroys the transplanted beta cells, too.

"Any time you're thinking about any type of a cure or really good treatments for type 1 diabetes, you have to consider both the beta cells and the immune side," said Andrew Rakeman,

the scientific program manager in beta cell regeneration at the Juvenile Diabetes Research Foundation (JDRF). The JDRF funded a portion of the new research.

"At this point, it's unknown whether reprogrammed alpha cells would be vulnerable. Alpha cells are normally not destroyed by the immune system, but in regenerating, they're losing traits that make them alpha cells to turn into beta cells. So, it's likely that they'll appear to the immune system as beta cells," Rakeman said.

To induce type 1 diabetes in the mice, the Swiss researchers exposed the rodents to a toxin that destroyed just their beta cells. Alpha cells are normally found in the pancreas alongside beta cells. Alpha cells secrete a hormone called glucagon that counteracts the effects of too much insulin and helps the body maintain normal blood sugar levels.

More than 99 percent of the beta cells were destroyed in the mice. The researchers labeled alpha cells with a fluorescent protein so they could track those cells.

They found that when nearly all of the beta cells had been destroyed, if mice were given insulin therapy to keep them alive, the alpha cells spontaneously changed into functioning beta cells. After enough alpha cells converted into beta cells, insulin therapy was no longer needed.

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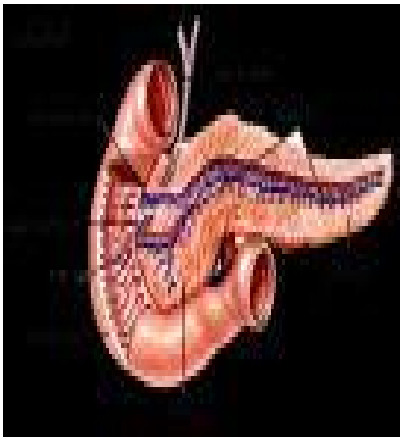
The new research, published April 4 in the online edition of the journal Nature, is the first to show that this change can happen naturally and spontaneously, the study authors said. Previous research has been able to change adult cells into insulin-producing cells, but that change required genetic manipulation, which might make it harder to develop a type of drug to reproduce the effect.

Rakeman said what's exciting in this new study is that "reprogramming is something that can happen naturally. If one can delineate what's causing it to happen in mice, it might be possible to find interventions to induce that to happen in humans."

Herrera said the researchers want to learn more about how the alpha cells undergo this change, and they also want to learn whether other cells can undergo these types of conversions.

Dr. David Kendall, chief scientific and medical officer at the American Diabetes Association, said, "Anything that speaks of a potential source of new insulin producing cells is pretty exciting.

"However," he added, "I always have cautious enthusiasm for such findings. Early promise is not always a guarantee, and a number of mouse findings haven't translated well in human research."



## ***SOURCES:***

Pedro Herrera, Ph.D., professor, department of cell physiology and metabolism, University of Geneva Medical School, Geneva, Switzerland; Andrew Rakeman, Ph.D., scientific program manager in beta cell regeneration, the Juvenile Diabetes Research Foundation, New York City; David Kendall, M.D., chief scientific and medical officer, the American Diabetes Association, Alexandria, Va.; April 4, 2010, Nature, online HealthDay .