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Voglibose can Significantly Lower Postprandial Blood Glucose Levels in Type 2 Diabetes Mellitus

The ability to achieve the best possible glycemic control both in the fasting and postprandial states is the primary goal in the treatment of diabetic patients. A study published in the journal *Diabetes Research and Clinical Practice* reports that voglibose can significantly lower postprandial blood glucose levels in type 2 diabetes mellitus. A randomized crossover open comparative study was conducted to evaluate the efficacy and safety of voglibose and acarbose in 30 patients with type 2 diabetes who were not well controlled with diet therapy. There was no significant reduction of fasting blood sugar (FBG) with either voglibose or acarbose at 4 and 8 weeks after treatment. The 1 h postprandial blood glucose (PPBG) level was significantly decreased from 224.9±42.8 to 204.1±37.6 (P=0.005) and 206.1± 38.9 mg/dl (P=0.038) after voglibose therapy at 4 and 8 weeks, respectively. Significant decrease was also obtained after, acarbose treatment from 228.3±37.4 to 182.7±35.5 (P<0.001) and 186.636.1 mg/dl (P<0.001) at 4 and 8 weeks. The decrease of 1 h PPBG was associated with a significant fall of serum insulin concentration. Hemoglobin A1c (HbA1c) levels were also significantly decreased from 7.07±1.21 to 6.83±1.11 (P=0.017) and 6.79±1.33% (P=0.036) after voglibose and 6.98±0.98 to 6.70±1.04 (P0.001) and 6.591.04% (P<0.001) after acarbose at 4 and 8 weeks. In contrast to voglibose, treatment with acarbose significantly decreased the 2 h PPBG at 4 and 8 weeks and the 2 h postprandial serum insulin concentration at 8 weeks. Adverse drug events were more commonly reported in acarbose-treated patients (P<0.05). Increased flatulence was observed in 56.7 and 90% of the patients taking voglibose and acarbose, respectively, while abdominal distention was noted in 10 and 16.7%. Significantly decreased body weights of 0.9 and 0.8 kg were recorded at 8 weeks after voglibose and acarbose therapy, respectively. The study conclude that both voglibose (0.2 mg) and acarbose (100 mg) thrice daily significantly decreased HbA1c, PPBG and postprandial insulin levels. At these dose levels, voglibose was associated with less gastrointestinal side effects.

ARTICLES INSIDE

Bexinfo-Diabetology is a publication aimed to present the current noticeable happenings of diabetes from most authentic sources. Our effort will be successful only if it can meet some of your needs of knowledge in your daily practice.

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Metformin Slows Aging and Lengthens Lifespan

A study provides new evidence that metformin, the world's most widely used anti-diabetic drug, slows aging and increases lifespan. In experiments reported in the journal *Proceedings of the National Academy of Sciences,* the researchers tried to find out the mechanism behind metformin's age-slowing effects: the drug causes an increase in the number of toxic oxygen molecules released in the cell and this, surprisingly, increases cell robustness and longevity in the long term. Mitochondria generate tiny electric currents to provide the body's cells with energy. Highly reactive oxygen molecules are produced as a by-product of this process. While these molecules are harmful because they can damage proteins and DNA and disrupt normal cell functioning, a small dose can actually do the cell good, say the researchers: "As long as the amount of harmful oxygen molecules released in the cell remains small, it has a positive long-term effect on the cell. Cells use the reactive oxygen particles to their advantage before they can do any damage," explains the researcher. "Metformin causes a slight increase in the number of harmful oxygen molecules. We found that this makes cells stronger and extends their healthy lifespan." It was long thought that harmful reactive oxygen molecules were the very cause of aging. The food and cosmetics industries are quick to emphasize the 'anti-aging' qualities of products containing antioxidants, such as skin creams, fruit and vegetable juices, red wine and dark chocolate. But while antioxidants do in fact neutralize harmful reactive oxygen molecules in the cell, they actually negate metformin's anti-aging effects because the drug relies entirely on these molecules to work. The researchers studied metformin's mechanism in the tiny roundworm *Caenorhabditis elegans*, an ideal species for studying aging because it has a lifespan of only three weeks. "As they age, the worms get smaller, wrinkle up and become less mobile. But worms treated with metformin show very limited size loss and no wrinkling. They not only age slower, but they also stay healthier longer," says the researcher. "While we should be careful not to over-extrapolate our findings to humans, the study is promising as a foundation for future research." Other studies in humans have shown that metformin suppresses some cancers and heart disease. Metformin could even be an effective drug for counteracting the general effects of aging, say the researchers.

Diabetes Drug Shown to Help Body Rebuild After Heart Attack

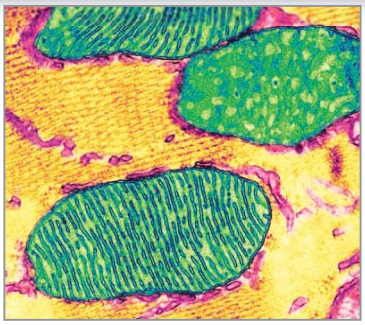
New light has been shed on how a common diabetes drug can be used to aid recovery from a heart attack. Heart disease is the leading cause of illness in diabetic patients. It accounts for more than half of all fatalities and the search for enhanced treatments is of high importance. For the first time, researchers have explored the mechanism behind metformin, a key treatment used by diabetic patients to prevent heart disease. The findings are published in the journal *Cardiovascular Diabetology*. Experts used stem cells from cord blood and cells from umbilical cord to construct a model simulating a heart attack in a lab. They found new blood vessel formation that is essential for heart attack recovery, and they established that metformin enhances the physiological process through which new blood vessels form. The research has shown that lack of oxygen in the presence of high glucose levels -- as occurs during a heart attack in diabetes -- delays blood vessel formation whilst metformin reverses that process. A further discovery is that metformin affects several new genes important in promoting the growth of new blood vessels. The researcher said: "The outcome of heart disease interventions in patients with diabetes is much worse in comparison with non-diabetic individuals. As a result there is a demand for improved treatment approaches to enhance the outcomes of those with diabetes in order to increase heart attack survival rates. "Our research is exciting as it has can instantly make a difference to the treatments we are exploring, offering a new approach to heart disease in diabetes and new therapies may now be developed. "It is believed that our study is the first report describing the effect of the physiological concentration of metformin as seen in patients. Furthermore, our study concentrated on the time period vital during a heart attack when, with new therapy, we can help patients most." Recent reports from the International Diabetes Federation highlight that 8.3% of adults have diabetes, affecting 382 million worldwide. It is estimated that this number will rise to 592 million by the year 2035. Metformin is a cost-efficient drug usually used as a first-line treatment in Type 2 diabetes as it helps to make the body more responsive to insulin. It is hoped that future studies of metformin's ability to aid heart attack recovery will focus on patient clinical trials.

Today I ate two bowls of dog food, a sandwich crust, some spaghetti that fell on the floor, half of your cat food, a wet tea bag, three bugs and the inside of a sneaker.How many grams of fat is that?

What Primary Care Providers Should Know About Diabetic Neuropathy

An estimated 60 to 70 percent of people with diabetes develop some form of diabetic neuropathy, or the chronic nerve damage diabetes causes, according to the National Institute of Diabetes and Digestive and Kidney Diseases. With so many people affected, researchers at Michigan Medicine led a group of internationally recognized endocrinologists and neurologists from both sides of the Atlantic and teamed up with the American Diabetes Association (ADA) to craft a new position statement on the prevention, treatment and management of the condition. The statement provides recommendations for physicians on the overall prevention of diabetic neuropathy, noting that preventing this complication is a key component of diabetes care because treatments to reverse the underlying nerve damage are lacking. "Our goal was to update the document so that it not only had the most up-to-date evidence, but also was easy to understand and relevant for primary care physicians," lead author of the statement. Another goal of the statement was to clarify the multiple forms of diabetic neuropathy that exist. "Many physicians have used different classifications for neuropathies. We came to a consensus to classify them in a more logical pattern, or format, for clinical care." The research team includes a classification system for diabetic neuropathies within the statement, which describes the three main types: diffuse neuropathy, mononeuropathy, and radiculopathy or polyradiculopathy. Diffuse neuropathy can be broken down into two categories, peripheral, which affects the feet and hands, and autonomic, which affects the internal organs. Common examples of diffuse neuropathy are distal symmetric polyneuropathy (DSPN) and cardiovascular autonomic neuropathy (CAN). Mononeuropathy occurs when a single nerve or an isolated nerve group is damaged. Radiculopathy occurs when the root of a nerve is pinched. After establishing the classification system, the research team provides recommendations for overall prevention of diabetic neuropathy, including-in type 1 diabetes, work to effectively control glucose as soon as possible to prevent or delay the development of DSPN and CAN. In type 2 diabetes, work to effectively control glucose to prevent or slow the progression of DSPN. With type 2 diabetes, consider a multifactorial approach with targeting glycemia and other risk factors to prevent CAN. Recommendations for screening and diagnosing, managing and treating the specific forms are also now available. For example, with DSPN, one of the most common forms of diabetic neuropathy encountered in the clinical setting, the researchers say: All patients should be screened for DSPN as soon as they are diagnosed with type 2 diabetes and five years after the diagnosis of type 1 diabetes, with annual screenings afterward. If you have a patient with prediabetes who has symptoms of peripheral neuropathy, consider screening. Assessment should include a careful history check, in addition to either a temperature or pinprick sensation (if the patient has small-fiber function) or a vibration sensation using a 128-hertz tuning fork (if the patient has large-fiber function). All patients should have an annual 10-gram monofilament testing to determine if their feet are at risk for ulceration or amputation. Electrophysiological testing or referring the patient to a neurologist is rarely needed for screening, except if the symptoms presenting are atypical, such as motor greater than sensory neuropathy, rapid onset or asymmetrical presentation. If the diagnosis is unclear or different etiology is suspected the patient can be referred. The researcher mentions pain is often the reason many diabetic neuropathy patients seek help from their providers. The researchers recommend: As the initial approach, consider either pregabalin or duloxetine. Gabapentin can also be considered as an effective initial approach, but the patient's socioeconomic status, comorbidities and potential drug interactions have to be taken into consideration. Tricyclic antidepressants are also effective but are not approved by the U.S. Food and Drug Administration and should be used with caution because of the higher risk of serious side effects. Opioids are not recommended as first- or second-line agents for treating pain associated with DSPN because of the high risks of addiction and other complications."Treatment of neuropathy pain is specifically relevant because, unfortunately, there has been much overprescribing of narcotics for neuropathic pain," says the researcher. "We now provide clear evidence to fellow physicians that other agents are available and are more effective in treating diabetic neuropathy. We also demonstrate that there are ways to stay away from prescribing opioids and avoiding the epidemic of addiction and serious health consequences associated with opioid use in patients with diabetes."She adds, "We hope these guidelines bring together primary care physicians, endocrinology specialists and neurologists to expand the care provided to diabetic patients."

Blocking Neuron Signaling Pathway Could Lead to New Treatments for Peripheral Neuropathy

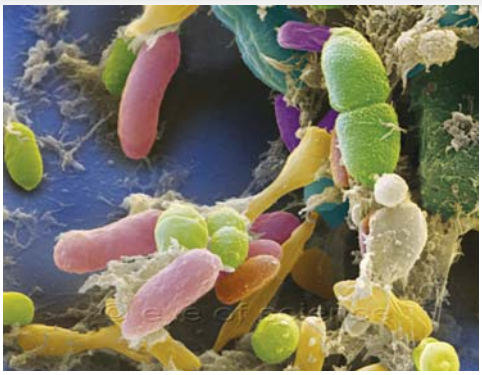


Researchers have identified a molecular signaling pathway that, when blocked, promotes sensory neuron growth and prevents or reverses peripheral neuropathy in cell and rodent models of type 1 and 2 diabetes, chemotherapy-induced neuropathy and neuropathy associated with HIV. The findings are published in the *Journal of Clinical Investigation.* Peripheral neuropathy is a condition resulting from damage to the peripheral nervous system -- the vast communications network that transmits information between the central nervous system and the rest of the body. Symptoms range from numbness, tingling and muscle weakness to severe pain, paralysis and organ dysfunction. "Peripheral neuropathy is a major and largely untreated cause of human suffering," said first author of the study. "It has huge associated health care costs." Previous research has described at least some of the fundamental processes involved in healthy, on-going peripheral nerve growth regeneration, including the critical role of mitochondria that produce adenosine triphosphate (ATP) which is vital to driving nerve recovery after injury. In their journal paper, the researchers looked for key molecules and mechanisms used in sensory neuron growth and regrowth. In particular, they noted that the outgrowth of neuritis was constrained by activation of muscarinic acetylcholine receptors. This was surprising, because acetylocholine is a neurotransmitter usually associated with activation of cells. With identification of this signaling pathway, the scientists suggest it is now possible to investigate the utility of anti-muscarinic drugs already approved for use in other conditions as a new treatment for peripheral neuropathy. "This is encouraging because the safety profile of anti-muscarinic drugs is well-characterized, with more than 20 years of clinical application for a variety of indications in Europe," said senior study author. "The novel therapeutic application of anti-muscarinic antagonists suggested by our studies could potentially translate relatively rapidly to clinical use"-he concluded.

Association between Insufficient Sleep and Gestational Diabetes Mellitus Discovered

A study has found a new health link between short sleep during pregnancy and gestational diabetes mellitus (GDM). This is the first study to examine the relationship between sleep duration and GDM in a multi-ethnic Asian population. Findings, published in the journal *SLEEP*, suggest that addressing sleep concerns during pregnancy could potentially reduce the risk of developing GDM. Diabetes is a global health concern and is an increasingly pressing problem in Asia. Gestational diabetes mellitus is diagnosed by high blood glucose levels, is one of the most common health problems during pregnancy. Unmanaged high glucose levels in pregnancy can result in complications that can affect both mother and child including pre-term labor, obstructed labor, birth trauma, high blood pressure for mothers, and increased risk of mother and fetal deaths. To reduce and manage the burden of diabetes, it is imperative to identify the factors that may contribute to unhealthy blood glucose levels. Sleep has been identified as one of the factors that affects glucose metabolism, and some studies have indicated that short sleep is a risk factor for Type 2 diabetes. Recent work suggests that adults in Singapore are among the most sleep-deprived in the world. This lack of sleep could contribute to GDM in Asian women, who are already at increased risk of GDM compared with Caucasian women. In order to determine if short sleep duration is associated with increased risk of GDM, the researchers analyzed the sleep and glucose levels of participants in the Growing Up in Singapore Towards healthy Outcomes (GUSTO) study. To examine the potential link between short sleep and GDM, 686 women completed a sleep questionnaire and had their glucose levels measured in a standard clinical test (oral glucose tolerance test) at 26 to 28 weeks of gestation. Of the 686 participants who had their glucose levels measured, 131 (19%) were diagnosed with GDM. Statistical analyses were run to assess whether exposure to short sleep, defined as less than 6 hours per night, was associated with greater odds of having GDM. The researchers found that short sleep was associated with increased risk of GDM, after adjusting for factors including age, BMI, and history of GDM. Interestingly, the frequency of GDM was highest (27.3%) in women who reported sleeping less than six hours a night and was lowest (16.8%) in women who reported sleeping between seven to eight hours a night. Results are consistent with findings which show that short sleep is associated with Type 2 diabetes in non-pregnant populations. They are also consistent with smaller studies done in Caucasian and African-American pregnant women. "Our results raise the possibility that good sleep habits could reduce the likelihood of developing hyperglycemia and GDM," said senior author. "With the recently launched 'War on Diabetes' in Singapore, the importance of healthy sleep habits should be emphasized to doctors and patients, in addition to initiatives that are geared toward improving other lifestyle factors, such as diet and exercise." "Our study provides a better understanding of how we may be able to counter a potentially serious condition for pregnant women and her child," added first author.

Type 1 Diabetes Linked To Gut Inflammation, Bacteria Changes



People with type 1 diabetes exhibit inflammation in the digestive tract and gut bacteria- a pattern that differs from individuals who do not have diabetes or those who have celiac disease, according to a new study published in the Endocrine Society's *Journal of Clinical Endocrinology & Metabolism*. Type 1 diabetes occurs when the body produces less to no insulin. It tends to begin at a young age. It typically develops when the body's own immune system attacks the pancreas and prevents the gland from producing insulin. As a result, type 1 diabetes is an autoimmune condition. Among every 1,000 American adults, between one and five people have type 1 diabetes, according to the Society's Endocrine Facts and Figures report. "Our findings indicate the individuals with type 1 diabetes have an inflammatory signature and microbiome that differ from what we see in people who do not have diabetes or even in those with other autoimmune conditions such as celiac disease," said the study's senior author. "Some researchers have theorized that the gut may contribute to the development of type 1 diabetes, so it is important to understand how the disease affects the digestive system and microbiome." The study examined the microbiome of 54 individuals who underwent endoscopies and biopsies of the first part of the duodenum, at San Raffaele Hospital between 2009 and 2015. The individuals were either undergoing a diagnostic procedure to diagnose a gastrointestinal disorder or volunteered to participate in the study. This approach allowed researchers to directly assess the gastrointestinal tract and bacteria, unlike studies that rely on stool samples for analysis. The analysis of tissues sampled from the endoscopy produced high-resolution snapshots of the innermost layer of the gastrointestinal tract. Individuals with type 1 diabetes showed significantly more signs of inflammation of the gut's mucous membrane linked to 10 specific genes than the participants who had celiac disease and control healthy subjects. Participants with type 1 diabetes also displayed a distinct combination of gut bacteria that was different from the other two groups. "We don't know if type 1 diabetes' signature effect on the gut is caused by or the result of the body's own attacks on the pancreas," said the researcher. "By exploring this, we may be able to find new ways to treat the disease by targeting the unique gastrointestinal characteristics of individuals with type 1 diabetes."