

OBESITY BLOG POSTS

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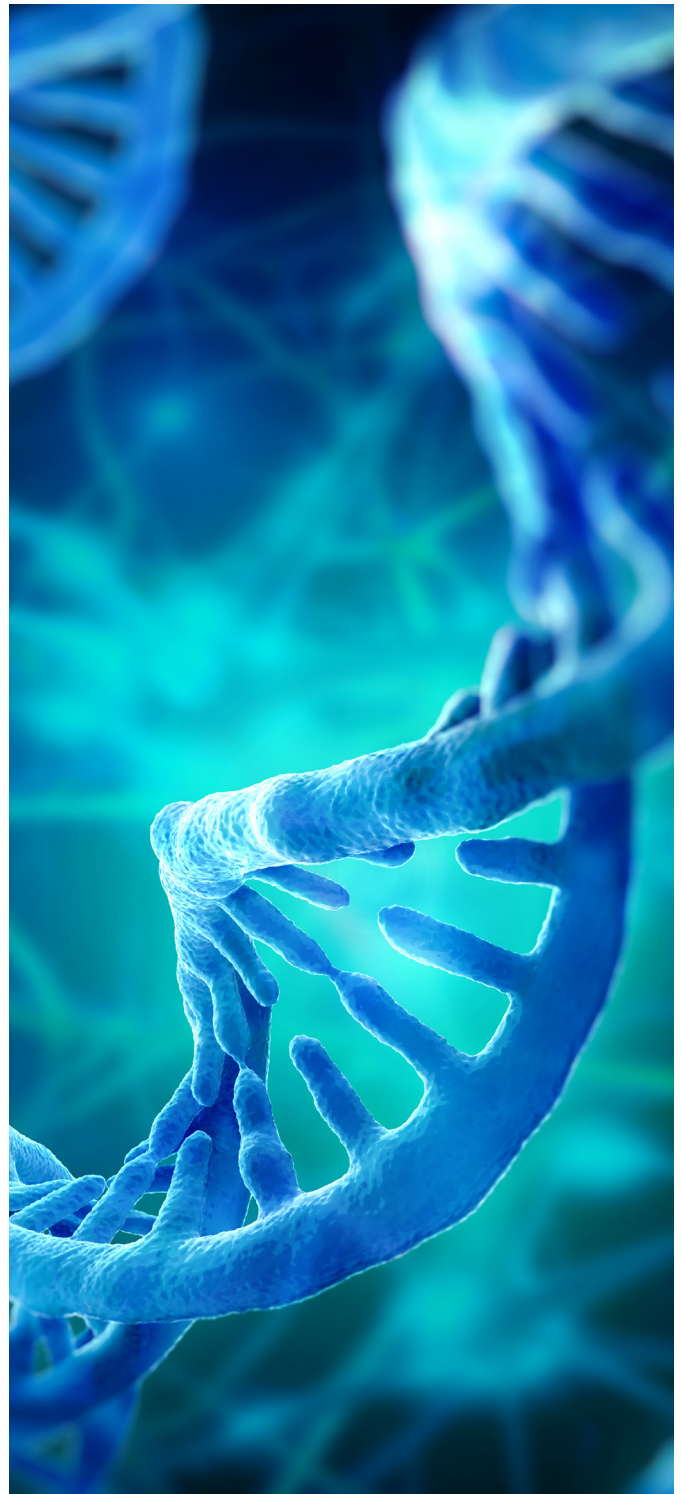
A Genetic Game-Changer in Treating Obesity

An estimated 650 million adults live with obesity today, which puts those individuals at increased risk for some of the leading causes of death worldwide, including diabetes, heart disease, stroke, and some types of cancer. Most prescription weight-loss drugs work by decreasing appetite or providing a feeling of satiety to help people lose weight. The exception is orlistat, which impedes fat absorption. This summer, the U.S. Food and Drug Administration approved the first new obesity drug since 2014 – semaglutide – which helped clinical trial participants with a BMI of 27 or higher lose as much as 12.4% of their body weight when combined with diet and exercise. This new therapy offers a significant increase in body weight loss over the average 3 to 7% associated with older therapies. But according to a new study published in [Science](#), treating obesity may soon rely on a genetic target.

The Promise of a Game-Changer

Scientists from Regeneron Pharmaceuticals sequenced exomes of almost 650,000 people from the United Kingdom, United States and Mexico. The sequencing revealed a protective ‘loss of function’

continued »



A Genetic Game-Changer in Treating Obesity (continued)

GPR75 mutation in one out of every 3,000 people sequenced. Individuals with at least one inactive copy of the GPR75 gene weighed, on average, 12 pounds less than the rest of the population, with a 54% reduction in risk of obesity. There was also evidence of an association with glucose lowering.

Based on these insights, Regeneron plans to target GPR75 using a range of therapeutic approaches including antibodies, small molecules, and gene silencing. The biotech has recently announced a partnership with AstraZeneca to research, develop and commercialize small molecule compounds directed against the GPR75 target with the potential to treat obesity and related comorbidities. With Regeneron's expertise in genetics and AstraZeneca's knowledge of chemistry and small molecules, the collaboration can enable a game-changing therapy in an area in great need of innovation.

A Goal of Treatments Offering Both Efficacy and Safety

With regulatory agencies highly focused on safety profiles, attempts at therapeutics to treat obesity have often resulted in failure. Despite this, Regeneron remains convinced that its genetics center will point the way to the big blockbusters of the future.

“While the behavioral and environmental ties to obesity are well understood, the discovery of GPR75 helps us put the puzzle pieces together to better understand the influence of genetics.”

Dr. Christopher Still, director for the Geisinger Obesity Research Institute at the Geisinger Medical Center.

“The next era of drug development is being fueled by important genetic findings that direct drug developers on how to deploy our toolkit of biologics, small molecules and gene editing technologies,” says Dr. George D. Yancopoulos, Regeneron's President and Chief Scientific Officer. “Regeneron is excited to join forces with AstraZeneca, as we seek to develop new medicines tackling the harmful and costly obesity epidemic.”

Establishing a Clear Genetic Link

“While the behavioral and environmental ties to obesity are well understood, the discovery of GPR75 helps us put the puzzle pieces together to better understand the

Treating Obesity May Soon Rely on a Genetic Target



650 Million

Adults live with obesity today



Risks

Diabetes, Heart disease, Stroke

New Obesity Drug

Semaglutide can help

To lose 12.4% of their body weight

influence of genetics,” says Dr. Christopher Still, director for the Geisinger Obesity Research Institute at the Geisinger Medical Center. “Further studies and evaluation are needed to determine if reducing weight in this manner can also lower the risk of conditions commonly associated with high BMI, such as heart disease, diabetes, high blood pressure and fatty liver disease.”

The conclusion of the study published in *Science* captures the significance of the findings, stating the inhibition of GPR75 “illustrates the power of massive-scale exome sequencing for the identification of large-effect coding variant associations and drug targets for complex traits.” This game-changer in obesity treatment has the potential to reduce the impact of numerous comorbidities, and may be the first of many new options for patients.

Fourteen Obesity Genes Discovered

The [World Health Organization](#) reports that around 13 percent of the world's population is classified as "obese." Today, medical experts know that obesity is much more than just a battle with over-eating. In fact, obesity is a medical condition largely affected by an individual's genetics. As researchers work to develop drugs to treat obesity, exploring the connection between obesity and genetics is more important than ever before. Fortunately, as [Science Daily reports](#), University of Virginia scientists have identified 14 genes that can cause weight gain, as well as three genes that can prevent weight gain. These obesity genes could prove instrumental in treating obesity moving forward.

Exploring Obesity Genes

An individual is defined as "obese" when they exhibit a [Body Mass Index \(BMI\) of 30.0](#) or higher. Treating obesity isn't as simple as advising someone to exercise or eat right; the condition goes far beyond lifestyle choices, involving several genes that regulate fat storage and nutrient viability. For years, scientists have sought to understand these genes that convert excessive food into fat. The idea is that "inactivating" these genes with drugs could prove life-changing for individuals with obesity. The question is: How can

scientists explore those genes? Eyleen O'Rourke of the University of Virginia led a team to find out. Their key test subjects: humble worms known as *C. elegans*.

Can Worms Help Researchers Explore Human Genetics?

Per *Science Daily*, the worms used in the University of Virginia study are [tiny creatures](#) that live in rotting vegetation and subsist on microbes. Surprisingly, these worms share more than 70 percent of our genes. That's not all we have in common: Like people, *C. elegans* worms "become obese if they are fed excessive amounts of sugar," *Science Daily* writes. Given our shared genetics, these worms are highly useful in a research capacity. This is why O'Rourke and her collaborators used the worms for their research, which was recently published in the scientific journal [PLOS Genetics](#).

Creating a Worm Model of Obesity

The University of Virginia study required the research team to assess 293 of the genes associated with obesity in humans. The researchers had one goal: to discover which of the genes were actually causing – or, in some cases, preventing – obesity. To accomplish this, the researchers

developed a worm model of obesity, feeding some of the worms a high-fructose diet. With the help of machine learning-assisted testing, the researchers were then able to monitor the worms' weight gain and evaluate the worms' genes. During that process, the team identified 14 genes that cause obesity, as well as three genes that helped prevent obesity in some worms.

Implications of the Research

Ultimately, the researchers did exactly what they sought out to do. They discovered that enhancing the action of the three genes that prevented obesity also had other benefits: namely, a longer lifespan and improved [neuro-locomotory function](#). Those benefits are two examples of the benefits researchers would hope to deliver via anti-obesity medicines for humans. "Anti-obesity therapies are urgently needed to reduce the burden of obesity in patients and the healthcare system," O'Rourke said. "Our combination of human genomics with causality tests in model animals promises yielding anti-obesity targets more likely to succeed in clinical trials because of their anticipated increased efficacy and reduced side effects."

14 New Obesity Genes



1 Shared cause of obesity: excessive amounts of sugar.

2 Benefits that researchers are hoping for: Longer lifespan and improved neuro-locomotory function.

3 Genes associated with preventing obesity in worms.

13%

Percent of the world's population classified as obese

30

Body Mass Index (BMI) of 30.0 or greater is considered obese.

70%

C. elegans worms surprisingly share more 70% of our genes.

293

The number of genes associated with obesity in humans.

14

Genes identified as causing obesity in humans.

While further research is needed, this study could prove promising as researchers work to reduce obesity in the global population. The use of C. elegans worms to evaluate the human genetic structure is just the latest example of how modeling human disease in other organisms drives the medical community forward.

New Obesity Drug Shows Great Promise in Phase III Trials

Danish pharmaceutical giant [Novo Nordisk](#) changed the game last year, presenting data that suggested one of the company's diabetes drugs was also highly effective at treating obesity as a medical issue. With that, Novo Nordisk sent a message to pharmaceutical companies everywhere: Obesity drugs could be the next frontier of life-changing medical innovations. Now, Eli Lilly [has introduced](#) a new obesity drug that shows great promise in [Phase III trials](#). Read on to find out more about what this development means for the pharmaceutical industry.

What Sets Eli Lilly's Obesity Drug Apart

As mentioned above, Novo Nordisk pioneered a fascinating breakthrough when the company proved that its diabetes drug could also address obesity. Competitor Innovent Biologics also [recently debuted](#) promising Phase II data for a GLP-1 receptor/glucagon receptor called mazdutide, which was shown to be effective in reducing trial participants' body weights. Now, Eli Lilly is in the final stages of testing another obesity drug – this time, in an entirely different drug class than the Novo Nordisk and Innovent products. Eli Lilly's drug is known as tirzepatide. [Per USA Today](#), the drug “works on two naturally occurring hormones that help control blood sugar

and are involved in sending fullness signals from the gut to the brain.”

Study results show that patients taking the drug “achieved superior weight loss compared to placebo at 72 weeks of treatment.” Specifically, 96 percent of people taking higher doses of tirzepatide (10 mg and 15 mg) achieved at least 5-percent body weight reductions. Finally, per the company, some participants lost as much as 22.5 percent of their body weight during the trial. Per [industry resource Endpoints News](#), the study enrolled 2,539 participants and was the “first Phase III global registration trial evaluating the efficacy and safety of tirzepatide in adults with obesity, or overweight with at least one comorbidity, who do not have diabetes.”

When Will the FDA Approve Eli Lilly's Obesity Drug?

The trial data cited above proves extremely promising for tirzepatide, especially given the rapid approval process granted to Novo Nordisk's competitor drug, semaglutide. Originally approved to treat diabetes, semaglutide recently won FDA green light status to treat obesity as well [under the drug name Wegovy](#). Eli Lilly is doubtless hoping for a similarly speedy process, especially since, per *EndPoints*

News, the “overall safety and tolerability profile of tirzepatide was similar to other incretin-based therapies approved for the treatment of obesity.” With these considerations in mind, Eli Lilly may well be en route to rapid approval.

Next Steps for Tirzepatide

Tirzepatide is already gaining international attention, with [USA Today](#) noting that the drug could be a “game-changer.” USA Today also cites Dr. Robert Gabbay, chief scientific and medical officer for the American Diabetes Association, who notes that the drug provides the kind of stunning results previously limited to weight loss surgery. Eli Lilly spokespeople agree. “Tirzepatide is the first investigational medicine to deliver more than 20 percent weight loss on average in a phase III study, reinforcing our confidence in its potential to help people living with obesity,” said Jeff Emmick, MD, Ph.D., Eli Lilly’s vice president of product development. “Obesity is a chronic disease that requires effective treatment options, and Lilly is working relentlessly to support people with obesity and modernize how this disease is approached. We’re proud to research and develop potentially innovative treatments like tirzepatide, which helped nearly two-thirds of participants on the highest dose reduce their body weight by at least 20 percent in SURMOUNT-1.”

New Obesity Drug Shows Great Promise in Phase III Trials

 **40 %**

Of adults in the United States are living with obesity

 **55 %**

Of people with obesity receive a formal diagnosis

 **60**

Health conditions are associated with obesity

 **650**

Million people worldwide are considered obese

 **2,539**

Participants were in the first Phase III global registration trial evaluating the efficacy and safety of tirzepatide in adults with obesity

Ultimately, Eli Lilly’s success proves extremely promising for adults struggling with obesity, as well as medical providers searching for creative solutions. This may be just the beginning of an entirely new approach to obesity.

Do Obesogens Cause Obesity?

For years, obesity and weight gain have been blamed on the “calorie concept”: the idea that obesity is caused by taking in more calories (through diet) than one burns off (through exercise). But as the [obesity epidemic](#) across the globe continues to grow – even as caloric consumption has stayed flat and, in some cases, gone down – some researchers are beginning to challenge the accepted wisdom that obesity is simply caused by a caloric imbalance. And new research has identified one likely culprit for rising obesity numbers: obesogens.

Obesogens and Obesity

Obesogens are a class of environmental chemicals that are thought to promote weight gain by interfering with the body’s

endocrine system and disrupting the normal regulation of lipid metabolism. Obesogens are everywhere: [present in](#) “dust, water, processed foods, food packaging, cosmetics and personal care products, but also furniture and electronics, air pollutant, pesticides, plastics and plasticisers.”

The term “obesogen” was first [coined in 2006](#) by Bruce Blumberg, a cell biologist at the University of California, Irvine. Blumberg’s lab observed that mice who were exposed to [tributyltin chloride](#) (a common toxic compound used as an antifoulant in marine and industrial paints, wood preservatives, and PVC plastics, among other applications) in utero experienced “strikingly elevated lipid accumulation” and fat formation compared to control group mice, despite no difference in their diet or exercise. As [Blumberg explained](#), “Mice we expose to tributyltin don’t eat more, and they don’t exercise less than animals not exposed to it. But they use calories differently – they store more as fat. That’s very relevant to humans.”

Dozens more animal studies that followed continued to confirm that animals exposed to obesogens experienced an increase in fat accumulation. In one study, mice exposed to the estrogen drug diethylstilbestrol while pregnant had offspring who demonstrated increased



body weight as adults. In another study, mice fed bis(2-ethylhexyl) phthalate (DEHP) – a common obesogen found in flooring, wall coverings, food containers, toys, and cosmetics – were observed to eat more, gain more weight, and accumulate more belly fat than control mice.

Obesogens and the Mechanisms of Weight Gain

Obesogens trigger weight gain in a number of ways. Research has found that many common obesogens can easily bind to and activate the peroxisome proliferator-activated receptor gamma (PPAR- γ), triggering the accumulation of unhealthy fat cells. Obesogen chemicals can also target estrogen and androgen receptors, glucocorticoid receptors, and the retinoid X receptor.

Not only do obesogens trigger weight gain by activating various receptors, but they can also affect appetite control, alter basal metabolic rate, influence insulin regulation, and change the microbiome, all of which have a significant impact on a person's weight.

As Leonardo Trasande, an environmental health scientist at New York University, puts it, "Imagine you've had a good workout, you've eaten a good protein meal, and you are thinking you are going to gain some muscle." But instead?



Exposure to chemicals like phthalates can “change how the body processes a meal and ultimately turn it into fat or carbohydrate.”

This is why a small but growing group of researchers in the obesogenic community insist that the calorie concept or energy balance model, which treats obesity only as a problem of eating too much or exercising too little, is insufficient and incomplete. While obesogens are not the only cause of weight gain, exposure to these chemicals is likely a key to unlocking gaps in obesity research. Understanding the way exposure to obesogens affects a person's metabolism, weight gain, and appetite is a critical step in understanding and addressing the obesity epidemic.

Examining the Expanding Landscape of Weight Loss Injectables

Recent headlines have been abuzz with the groundbreaking weight loss results achieved with the injectable drug semaglutide, demonstrating the potential for people with obesity to shed up to 20 percent of their body weight. These findings have sparked considerable interest, particularly given that conventional lifestyle interventions typically yield only 5 to 10 percent weight loss. Eli Lilly is poised to take this innovation further with two potential injectable medications — tirzepatide and retatrutide — currently awaiting approval for the treatment of obesity and type 2 diabetes.

Mimicking Multiple Hormones for Enhanced Efficacy

The new medications work by mimicking multiple hormones involved in regulating appetite and blood sugar. In addition to being a glucagon-like peptide 1 (GLP-1) receptor agonist, like semaglutide, tirzepatide also mimics glucose-dependent insulintropic polypeptide (GIP), a hormone involved in blood glucose regulation and the storage of excess energy in fat cells.

One [study](#) compared tirzepatide in three different weekly doses (5mg, 10mg, and 15mg) with the standard weekly 1mg semaglutide injection. All three doses of

tirzepatide led to more substantial weight loss than the 1mg semaglutide injection. [Research](#) also suggests that the higher doses (10mg and 15 mg) of tirzepatide may outperform a 2mg semaglutide dose, resulting in more weight loss (3.15 kg and 5.15kg, respectively).

Two Phase III clinical trials, SURMOUNT-3 and SURMOUNT-4, evaluated tirzepatide compared to a placebo in people with obesity or weight-related comorbidities. For SURMOUNT-3, all participants first received a 12-week intervention that included a low-calorie diet, exercise, and weekly counseling. The average weight loss during this period was 6.9 percent. Following this intervention, participants were randomized to receive either tirzepatide or a placebo. After the 72-week treatment phase, those receiving tirzepatide lost, on average, an additional 21.1 percent of their body weight, while those in the placebo group regained, on average, 3.3 percent body weight.

All participants in SURMOUNT-4 took tirzepatide for the first 36 weeks, with an average weight loss of 21.1 percent. This was followed by randomization and a 52-week study period. Participants in the treatment group had an average 25.3 percent body weight loss over the 88-week trial, compared to an average weight regain of 14 percent among the placebo group. “The findings from SURMOUNT-3

Examining the Expanding Landscape of Weight Loss Injectables



\$2.1b

In supplements for weight loss alone are sold every year



17.1%

Of adults are on a diet



49.1%

Of US adults try to lose weight in any given 12 months



64m

Americans have gym memberships



95%

Of people want to lose weight for their own wellbeing



150min

Of moderate aerobic activity per week is recommended by the CDC



challenge the notion that patients living with obesity or overweight can achieve their weight loss goals with diet and exercise alone, said Jeff Emmick, senior vice president, product development at Eli Lilly, in a [statement](#). “The findings

from SURMOUNT-4 reinforce that obesity should be regarded like other chronic diseases where chronic therapy may be needed to maintain treatment benefits,” he added.

continued »

Examining the Expanding Landscape of Weight Loss Injectables (continued)

Retatrutide: A Triple-Hormone Receptor Agonist

Retatrutide is a triple-hormone receptor agonist that mimics GLP-1, GIP, and glucagon. In the brain, GLP-1 reduces appetite and food-seeking behaviors. In the gut and pancreas, it helps to control blood sugar levels. GIP also regulates blood sugar and affects fat cells' energy absorption. Glucagon plays a role in preventing low blood sugar levels during fasting, exercise, or low-carb intake. A randomized clinical [trial](#) of retatrutide showed that participants, who received either 1 mg, 4 mg, 8mg, or 12 mg doses per week, achieved substantial weight loss after 48 weeks of treatment. All participants who received at least 4 mg weekly doses lost at least 5 percent of their body weight. Those who received

the highest dose achieved a mean weight reduction of 24.2 percent, translating to an average absolute weight reduction of about 58 pounds over 11 months of the study, the company reported in a [statement](#).

The Future of Weight Loss Medications

The weight loss medication landscape is changing rapidly, as researchers continue to explore new compounds to regulate appetite and blood sugar while enhancing the efficacy and safety of these drugs. Following the attention received by semaglutide, potential options like tirzepatide and retatrutide loom on the horizon, offering hope for additional weight loss solutions in the future.





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Research Challenges Conventional Wisdom on Obesity and Mortality

Research using data from a large U.S. health survey has disrupted conventional thinking about body mass index (BMI) and its role in predicting all-cause mortality. The research suggests that BMI alone does not necessarily have a strong relationship with a person's risk of premature death.

The researchers based their BMI calculations on data from the 1999-2018 National Health Interview Survey. All-cause mortality data was based on the U.S. National Death Index. The authors note that similar studies in the past had relied on data from the 1970s, focusing primarily on non-Hispanic white adults. Changes in the U.S. population since then in BMI distribution, life expectancy, ethnic and racial diversity, and healthcare make the new research relevant to current discussions of weight and health.

BMI and Mortality: A Complex Relationship

Traditionally, BMI has been considered a key indicator of health, but the study, [published](#) in *PLOS One*, reveals a more nuanced picture. The findings show that for people with a BMI of 25.0 to 29.9 (in the overweight category), this measure alone may not necessarily indicate an increased mortality risk independent of other risk factors. However, mortality risk increased by 21% to 108% for adults with

a BMI of 30 or greater, indicating obesity. Among older adults (age 65 and older), the researchers reported no significant rise in mortality at BMI levels between 22.5 and 34.9. In younger adults (age 20 to 64), there was no increased mortality risk at BMIs of 22.5 to 27.4.

The authors suggest that, for older adults, higher muscle mass could be reflected in the higher BMIs, which would explain why mortality risk did not increase for those with BMIs up to 35. "People with higher BMIs may be paradoxically healthier because of sustained muscle mass and bone density," [said](#) study author Aayush Visaria, a postdoctoral research fellow at Rutgers Center for Pharmacoepidemiology and Treatment Science in New Jersey.

Limitations of BMI as a Health Measure

Although BMI is easy to [calculate](#) and widely used, it overlooks important factors such as body composition, fat distribution, and metabolic health. This can lead to misinterpretations. For instance, athletes often have high BMIs because they have increased muscle mass. Fat distribution around the waist, meanwhile, is known to increase the risk of disease but is not reflected in a person's BMI.



“[BMI] does not distinguish between muscle mass and fat mass, and some individuals like bodybuilders may have a high BMI because of more muscle mass,” said Dagfinn Aune, a research associate at the School of Public Health at Imperial College London, who was not involved in the study.

The authors also point out that people in the overweight category may be better able to recover from critical illness or severe infection than leaner individuals. Furthermore, among people who develop hypertension or diabetes, those with lower BMIs may have more aggressive or treatment-resistant disease, while people with higher BMIs may be able to combat disease with weight loss.

Exploring Alternative Predictors

To better predict all-cause mortality, the researchers recommend supplementing BMI with other measures like weight history, waist circumference, and waist-to-hip ratio. By incorporating multiple measures and evaluating cardiometabolic health parameters such as blood pressure and cholesterol levels, physicians can better identify people at risk and tailor interventions accordingly.

“I think the real message of this study is that overweight as defined by BMI is not an appropriate measurement tool, at least for all-cause mortality,” said Visaria. “Overweight as a medical condition is still important but will likely need to be diagnosed taking into account body composition and body fat distribution,” he added.

The Link Between Obesity Drugs and Brain Health

Glucagon-like peptide-1 (GLP-1) receptor agonists are arguably the hottest topic in medical innovation. The drugs semaglutide and tirzepatide, known under brand names like Ozempic, Wegovy, and Mounjaro, were originally used to treat type 2 diabetes; now, these drugs have emerged as highly sought-after medications used for weight loss. But new research suggests that GLP-1 receptor agonists may serve a third purpose: subduing brain inflammation, thus potentially protecting against Alzheimer's and Parkinson's diseases. The study, entitled "[Central glucagon-like peptide 1 receptor activation inhibits Toll-like receptor agonist-induced inflammation](#)," was published in the journal Cell Metabolism. Read on to find out more about the potentially far-reaching effects of the research into obesity drugs.

Understanding Obesity Drugs

GLP-1 agonists like semaglutide and tirzepatide mimic a hormone called glucagon-like peptide-1 (GLP-1), which is produced in the intestine in response to food intake. GLP-1 receptor agonists activate the GLP-1 receptors on the tissues involved in glucose metabolism, enhancing insulin secretion and inhibiting the release of glucagon into the body.

[Previous research](#) has suggested that GLP-1 agonists also have an anti-inflammatory

effect, as the GLP-1 hormone is involved in inflammation processes within the body. For that reason, GLP-1 agonists appear to protect against cardiovascular disease, which is largely linked to inflammation. "We know from animal studies and human studies that GLP-1 seems to reduce inflammation almost everywhere," says endocrinologist Daniel Drucker, co-author of the study published in Cell Metabolism. Now, Drucker's research has shed light on the potential impact of those anti-inflammatory properties on brain health.

Exploring the Link Between Obesity Drugs and Brain Health

Drucker's research team expanded on anti-inflammatory research to explore the relationship between GLP-1 agonists and one organ particularly susceptible to the negative effects of inflammation: the brain.

The team started by assessing mice with pre-induced inflammation. They found that, while GLP-1 agonists reduced the inflammation, the effect only took hold when the brain receptors weren't blocked. That shed light on a stunning possibility: that GLP-1 drugs are capable of interacting with the brain-immune system axis. That could have major implications for

brain conditions like Alzheimer's and Parkinson's diseases, both of which are linked to pathways of inflammation.

Could GLP-1 Drugs Treat Alzheimer's Disease?

Alzheimer's disease and Parkinson's disease both involve pathological proteins which interact with certain receptors that trigger neuroinflammation, or chronic inflammation in the neurological system. If GLP-1 receptor agonists prove effective in further trials, they could provide relief against neuroinflammation, potentially improving symptoms for individuals diagnosed with these degenerative diseases.

As a next step, the team hopes to establish which brain cells are interacting with GLP-1. The team also plans to assess other animal models of inflammation throughout the organ system.

"As the scientific community deservingly celebrates GLP-1 agonists and their impact, there are many unknowns left," noted Anne-Claude Gingras, director of the Lunenfeld-Tanenbaum Research Institute. "Dr. Drucker and his team have remained tenacious in their efforts to unpack how these drugs work, and this study deepens our understanding of metabolism and the complex brain-immune network that regulates it."



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