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AI Overview

The neuroscience of metabolism explores how the brain regulates and interacts with the body's metabolic processes, including energy balance, nutrient utilization, and the control of eating behavior. Brain regions like the hypothalamus and brainstem play a key role in sensing and responding to changes in blood glucose levels, influencing hunger, satiety, and energy expenditure. Neural signals also regulate metabolic processes in other organs like the liver, pancreas, and adipose tissue.

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22 Jan 2025 — Michael W. Schwartz, MD from the University of Washington Medicine Diabetes Institute, explains how the brain can be targeted to treat obesity and diabetes.

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Targeting the brain for the treatment of type 1 diabetes

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TARGETING THE BRAIN FOR THE TREATMENT OF TYPE 1 DIABETES

Michael W. Schwartz, MD, explains the role played by the brain as a cause of elevated blood sugar and related metabolic derangements in type 1 diabetes, and how therapeutic targeting of this brain system can eliminate the need for insulin therapy in animal models

What is type 1 diabetes?

There are two primary forms of diabetes, referred to as type 1 and type 2. While the latter is associated with obesity and is more common in adults, type 1 diabetes (T1D) is an autoimmune disease that typically begins in childhood. The disease is caused by an immune attack on the cells that make the hormone insulin, known as

secretion of insulin into the circulation. In adults, in turn, lowers the blood glucose level in two ways: by reducing glucose entry into the blood primarily from the liver) and promoting its uptake out of the blood into tissues such as muscle, heart, and fat cells (adipocytes). At blood glucose levels below 65 mg/dl, so too does insulin secretion, closing a negative feedback loop.

energy needs of virtually all tissues in the body must still be met, namely by mobilizing fatty acids from within the body. These fatty acids include glucose and ketones, which are released from the liver, and free fatty acids and glycerol, which are mobilized from stored fat. Failure to mobilize these fatty acids when they are unneeded can threaten survival, but excessive or unrestrained fat

What else does insulin do?
Insulin plays another role that, while less widely recognized, is crucial to our understanding of diabetes mellitus: the most likely consequence of untreated T1D. This condition results from unrestrained mobilization of glucose and other fuels that effectively starve the brain of its primary energy source. Because insulin prevents this from happening, even when present at very low levels, it does not occur except in the setting of severe hypoglycemia. Differently, insulin's function to prevent unrestrained fuel mobilization is

How does insulin lower the blood sugar level?
 Pancreatic beta cells are equipped with a specialized system for sensing glucose, the primary sugar in the bloodstream, and a key source of fuel for the body and tissues throughout the body. Detection of a rising blood glucose level by this glucose-sensing system in beta cells triggers the release of insulin. Insulin then binds to insulin receptors on the surface of cells, activating a signaling pathway that promotes the uptake of glucose into the cell. In the absence of insulin, glucose uptake is impaired, and blood glucose levels rise. This can lead to a condition called **diabetes mellitus**, which is characterized by high blood glucose levels. There are two main types of diabetes: **type 1 diabetes**, which is an autoimmune disease where the body's immune system attacks and destroys the insulin-producing beta cells, and **type 2 diabetes**, which is a metabolic disorder where the body's cells become resistant to the effects of insulin. Both types of diabetes can lead to serious complications if not managed properly.

secretion of insulin into the circulation. Insulin, in turn, lowers the blood glucose level in two ways: by reducing glucose entry into the blood (primarily from the liver) and promoting its uptake out of the blood (into tissues such as muscle, heart, and fat cells [adipocytes]). As blood glucose levels begin to fall, so too does insulin secretion, closing a negative feedback loop.

What else does insulin do?

Insulin plays another role that, while less widely recognized, is crucial to our understanding of diabetic ketoacidosis; the most lethal consequence of untreated T1D. This condition results from untreated/poorly managed diabetes, and is characterized by the fact that effectively food the bloodstream (discussed below). Because insulin prevents this from happening, even when present at very low levels, it does not occur except in patients with diabetes. Stated differently, insulin's function to prevent overeating and overdrinking is crucial to survival because diabetic ketoacidosis occurs when insulin is unable to perform this function.

So, how does the body ensure that fuels are mobilized in amounts that meet but do not exceed ongoing body requirements? This balancing act is achieved by a kind of "fabian" strategy in which the hormones that orchestrate fuel mobilization when energy stores are threatened, and the pancreas, which secretes enough insulin into the bloodstream to keep things from spinning out of control (preventing to the food anarchy, the brain and pancreas are the "up" and "down" levers on the diner's footstool).

What does all this have to

How is this fuel mobilization process governed?

In some ways, diabetic ketoacidosis can be viewed as a pathological extension of the normal adaptive response to fasting. When there is no longer any food remaining in the GI tract, the

energy needs of virtually all tissues in the body must still be met, namely, by mobilizing fuels from within the body. These fuels include glucose and ketones, which are released from the liver, and free fatty acids and glycerol, which are mobilized from stored fat. Failure to mobilize these fuels when they are needed can threaten survival, but excessive or unrestrained fuel

So, how does the body ensure that fuels are mobilized in amounts that meet but do not exceed ongoing body requirements? This balancing act is achieved by a kind of Kabuki dance between the brain, which orchestrates fuel mobilization when energy stores are threatened, and the pancreas, which secretes enough insulin into the bloodstream to keep things from spinning out of control (presuming to the flood analogy, the brain and pancreas are the 'up' and 'down' levers on the dam's floodgate).

Go with type 1 diabetes: Because the complete absence of insulin releases the brake on fuel mobilization, the circulation becomes flooded with glucose, ketones (released from the liver as ketoacids), free fatty acids, and glycerol, i.e., ketoacidosis. The underlying process is essentially

identical to what occurs during fasting, except that because the brake normally provided by insulin is missing, there's no way to close the floodgate.

If untreated, diabetic ketoacidosis causes dehydration, electrolyte disturbance, and destabilization of the body's carefully controlled acid-base balance. In its effort to respond to this very stressful constellation of events, the brain mistakenly turns on the

secretion of adrenaline, which has the unfortunate effect of further activating fuel mobilization. This vicious cycle cannot be broken without insulin - or so we thought.

Where does the brain fit in?
As noted above, the brain's ability to respond when fuel stores are threatened is essential for survival. But how is this message delivered to the brain? Here's the crazy part: the brain relies on the detection of insulin and another hormone called leptin (made by fat cells) to gauge the

When these two hormones are present at sufficiently high levels, the brain will not perceive fuel deficiency, regardless of how much stored fuel actually exists. Conversely, the brain perceives the absence of these hormones as an urgent, 'all hands on deck' situation requiring maximal fuel mobilization.

Complicating matters further, leptin production is dependent on insulin. So, in untreated T1D, insulin deficiency rapidly causes leptin deficiency, and the

Targeting the brain to treat T1D

How do we know this scenario is true? Because in animal models of T1D, this vicious cycle can be blocked by silencing neurons that trigger fatty mobilization

Remarkably, related work in animal models has shown that T1D can be rescued by infusing small amounts of

replenish energy into the brain, it is not the brain, under the influence of leptin, says, "No need to mobilize fuel here!" and the powerful cascade of metabolic decompensation described above simply fails to materialize. *Even more impressive is that in these animals, the brain not only lowers the blood sugar level but also restores normal control of blood sugar without the need for*

insulin or frequent blood sugar monitoring - something previously considered impossible.

Implications for the future treatment of type 1 diabetes

The profound implications of these findings have motivated a proposed human study to formally test whether leptin infusion into the brain can normalize blood sugar in T1D without the need for insulin. A positive outcome from this study would open entirely new avenues for the treatment of this disease – without the burden of insulin therapy (and the ever-present and dangerous risk of insulin-induced hypoglycemia) or regular blood sugar monitoring.

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AI Overview

Targeting the brain for type 1 diabetes (T1D) treatment involves several research approaches, including engineering brain cells to produce insulin while protecting them from the immune system, using brain cells to shield pancreatic beta cells from attack, investigating hormones and neurotransmitters to regulate glucose via brain signalling pathways, and developing therapies that restore brain function damaged by the disease. These strategies aim to offer potential cures or improved management by leveraging the brain's central role in glucose control.

Engineering Brain Cells for Therapy

- Combining insulin production and immune evasion: Researchers are exploring ways to combine insulin-producing beta cells with special brain cells, grown

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A target for treating type 1 diabetes mellitus
by R Ramakrishnan · 2019 · Cited by 3 — From early to later stages of Type 1 Diabetes Mellitus (T1DM), signalling molecules including brain indolamines and protein kinases are altered significantly.

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