

How Mitochondrial Function Influences Diabetes Risk

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STORY AT-A-GLANCE

- Research in mice and human pancreatic islet cells has revealed that damaged mitochondria in metabolic tissues don't die off as expected, but instead send distress signals that cause cells to become immature and dysfunctional
- This cellular "immaturity" particularly affects insulin-producing cells in the pancreas, causing them to stop making enough insulin to regulate blood sugar properly
- This same pattern of mitochondrial damage and cellular dysfunction was observed across multiple types of tissue involved in metabolism, including liver and fat cells
- Proper mitochondrial function requires eliminating processed foods and vegetable oils high in linoleic acid, while maintaining adequate healthy carbohydrate intake of 250 grams daily for optimal cellular energy production
- Environmental factors like endocrine-disrupting chemicals and electromagnetic fields also damage mitochondria, creating a cycle that compromises cellular energy production

Diabetes poses a significant health threat, impacting 589 million adults globally, a number projected to climb to 853 million by 2050.¹ Type 2 diabetes, the most common form, often emerges from unhealthy lifestyle habits that diminish your body's ability to respond to insulin.

Early symptoms can be subtle, sneaking up quietly. But as the condition progresses, most people notice persistent thirst, frequent bathroom trips, unexplained weight loss, and a bone-deep exhaustion that lingers. If left unchecked, diabetes steadily wreaks

havoc on organs like your heart, kidneys, eyes, and nerves, paving the way for life-altering complications.

The link between mitochondrial function and diabetes has become a cornerstone of cutting-edge research, and it's a connection I explored deeply in my book, “Your Guide to Cellular Health: Unlocking the Science of Longevity and Joy.” There, I argued that virtually all disease stems from insufficient cellular energy — energy your cells need to repair and restore themselves.

A study has now added weight to this view, spotlighting mitochondria, those tiny cellular powerhouses, as far more than just energy generators. They're intricately tied to your metabolic health. When mitochondria falter, especially in tissues critical to metabolism, emerging research shows your risk of diabetes rises sharply.

Dysfunctional mitochondria set off a cascade of chaos, driving insulin resistance and weakening your body's ability to produce insulin effectively — both defining features of diabetes. This isn't just about energy shortages; damaged mitochondria unleash cellular disruption that fuels the disease's progression.

As I proposed in “Your Guide to Cellular Health,” and as this study reinforces, the secret to dodging diabetes lies in restoring mitochondrial vitality. By ensuring these cellular engines hum efficiently, you can bolster the energy reserves your cells need to fend off metabolic collapse and thrive.

Mitochondrial Damage Provokes Cellular Immaturity in Key Metabolic Tissues

A 2025 study published in *Science* sought to understand what happens when mitochondria become damaged in tissues necessary for metabolism, such as your pancreas, liver, and fat tissue.² In experiments using mice, the researchers intentionally disrupted key aspects of mitochondrial function.

- **The researchers targeted mitochondrial DNA integrity** — This refers to the dynamic processes mitochondria use to maintain themselves and the pathways for mitochondrial turnover — essentially, the cellular mechanisms for quality control in these organelles. Damage to these mitochondrial components triggered a consistent cellular stress response across different metabolic tissues.

This response, known as a retrograde signaling program, represents a distress signal emanating from the damaged mitochondria back to the cell's nucleus, the control center of the cell. Instead of leading to cell death, a common expectation with cellular damage, this mitochondrial distress signal promoted cellular immaturity in key metabolic tissues.

- **Cells become less specialized and mature** — Instead of functioning properly or undergoing programmed cell death, affected cells lose their ability to carry out their intended metabolic roles effectively. In pancreatic beta-cells, the insulin-producing cells necessary for blood sugar regulation, this mitochondrial distress had a particularly detrimental effect. The beta-cells started to falter in their primary duty — insulin production.

They essentially stopped maturing into fully functional insulin factories and, consequently, could not generate sufficient insulin to manage blood sugar effectively. In a news release, study author Emily Walker explained, “In all three cases, the exact same stress response was turned on, which caused beta-cells to become immature, stop making enough insulin and essentially stop being beta-cells.”³

- **Other cell types are affected** — Expanding beyond pancreatic cells, the research team broadened their investigation. Repeating their experiments in liver cells and fat-storing cells in mice, they observed the same stress response activation and subsequent impairment of cell maturation and function.

This led senior study author Dr. Scott Soleimanpour to explain, “Although we haven't tested all possible cell types, we believe that our results could be applicable to all the different tissues that are affected by diabetes.”⁴ The team also confirmed the same findings in human pancreatic islet cells, so the mechanism is not limited to mice.

- **Mitochondrial damage didn't lead to cell death** — This raises the prospect of reversing the damage and restoring normal cell function. The researchers tested this hypothesis using an integrated stress response inhibitor (ISRIB) drug, which blocks the stress response pathway.

After four weeks of ISRIB treatment, the beta-cells in mice regained their capacity to regulate glucose levels.⁵ Targeting mitochondrial health therefore offers a promising direction for diabetes treatment and prevention, suggesting that future therapies could move beyond symptom management to address the fundamental cellular dysfunctions at play. ISRIB is an experimental compound that has not been tested as a diabetes treatment in people.

Note: the findings described here come from research in mice and in isolated human cells, not from clinical trials in people.

Decades of Research Indicate Mitochondrial Problems as a Core Defect in Diabetes

The strong connection between mitochondrial health and diabetes has been known for some time. A review published in the Journal of Diabetes Investigation in 2010 examined the body of evidence pointing to mitochondrial dysfunction as a key player in diabetes development.⁶

- **Mitochondria are crucial energy producers** — The paper emphasized the importance of mitochondria in producing adenosine triphosphate (ATP) for the cells in your body. Beyond general energy production, mitochondria are also involved in the very specific function of insulin secretion in your pancreatic beta-cells.

These cells rely heavily on healthy mitochondria to sense glucose and release insulin appropriately. Therefore, if mitochondria in these beta-cells aren't working correctly, it directly impacts your body's ability to manage blood sugar.

- **Mitochondrial dysfunction could be the “central defect” causing abnormal glucose metabolism in diabetes** — The dysfunction affects not only insulin production but also contributes to insulin resistance, a condition where your body's cells don't respond properly to insulin, further exacerbating blood sugar issues.

The researchers asserted that gaining a “deeper understanding of the role of mitochondria in diabetes will provide us with novel insights in the pathophysiology of diabetes.”⁷ Building on this established base of knowledge, more investigations continue to explore the intricate relationship between mitochondria and diabetes.

Therapies Targeting Mitochondrial Health for Diabetes

A comprehensive review published in the journal *Mitochondrion* focuses on therapeutic strategies for diabetes that target mitochondria.⁸ Like the other studies, it emphasizes that mitochondrial dysfunction isn't just a passive consequence of diabetes; it's an active contributor to insulin resistance, the failure of beta-cells, and the many complications associated with diabetes.

- **The importance of understanding the mechanisms involved behind diabetes** — Examples include the damaging effects of reactive oxygen species (ROS) and mutations in mitochondrial DNA that are crucial in treating diabetes. Importantly, the review highlights that our growing understanding of these mechanisms is paving the way for therapies that go beyond managing blood sugar to actually fixing the mitochondrial problems at the heart of the disease.
- **Mitochondrial dynamics** — This term, which is one of the key areas explored in the study, refers to the continuous processes of mitochondrial fusion, fission, and mitophagy. In simple terms, fusion is when mitochondria merge together, fission is

when they divide and mitophagy is the cell's way of cleaning up damaged mitochondria. Maintaining a healthy balance of these processes is important.

The review explains that in diabetes these dynamics are often disrupted, leading to unhealthy mitochondria. Therefore, therapies aimed at restoring the proper balance of fusion, fission, and mitophagy are emerging as promising strategies. The role of mitochondrial biomarkers for managing diabetes is also discussed. Biomarkers are measurable indicators that signal disease or physiological states. The review's author notes that these approaches remain largely preclinical, and that further comprehensive preclinical and clinical investigation is needed before they reach patients.

Practical Steps to Enhance Your Mitochondrial Function and Boost Cellular Energy

Knowing the key role mitochondria play in diabetes, it's empowering to realize you have the ability to take action and support these essential cellular powerhouses. By making informed lifestyle choices, you directly impact your mitochondrial health, reducing your risk of diabetes and improving your overall metabolic well-being. Here are practical steps to incorporate into your daily life to nurture your mitochondria and enhance their function:

- **Eliminate processed foods and vegetable oils from your diet** — Most processed foods contain seed oils that are loaded with linoleic acid (LA), a mitochondrial poison that compromises your cellular energy production. Avoid nuts and seeds, which also contain LA, and dining out, as most restaurants cook with vegetable oils. It's also wise to avoid chicken and pork, since these meats tend to contain high levels of LA.

Instead, focus on whole foods and healthy fats like grass fed butter, tallow and ghee. Keep your LA intake within 2 to 5 grams daily — the historical average intake, and the range worth returning to. Two grams is a floor, not a target to go under. The

Seed-Oil Sleuth feature in the Pax health platform will track this for you.

- **Optimize your carbohydrate intake for cellular fuel** — Carbohydrates play a key role in supporting your mitochondrial function. Glucose, derived from carbohydrates, serves as your cells' preferred fuel source for energy production. The key is to choose the right types and amounts of carbohydrates.

Most adults need a daily intake of around 250 grams of targeted carbohydrates to support cellular energy. If you lead a more active lifestyle, you likely need even more. It's important to reintroduce carbohydrates into your diet gradually, however, giving your gut microbiome time to adapt.

If you have dysbiosis, avoid fiber until your gut heals. In an unhealthy gut environment, complex carbohydrates like fiber feed harmful bacteria that multiply and eventually die off, releasing a highly virulent endotoxin called lipopolysaccharide (LPS). In addition to causing digestive issues, when LPS enters your bloodstream through a compromised gut barrier, it leads to a severe condition known as endotoxemia.

For those with a severely compromised gut, I recommend dextrose water, sipped slowly throughout the day, as a transitional solution. Unlike complex carbohydrates, dextrose is absorbed in your small intestine and doesn't feed bacteria in your colon, minimizing the production of harmful endotoxins. This strategy allows for gradual gut healing without worsening dysbiosis.

If your gut is generally healthy or you have only minor gut issues, start with easily digestible options like white rice, fruit juices with pulp, and whole fruits. As your gut adjusts, consider adding root vegetables, then non-starchy vegetables, starchy vegetables like sweet potatoes or squash, beans, legumes and, finally, minimally processed whole grains.

- **Minimize your exposure to environmental toxins** — The modern world exposes you to countless environmental toxins that sabotage your cellular energy production. Endocrine-disrupting chemicals (EDCs), including estrogen-mimicking compounds

like xenoestrogens, and pervasive electromagnetic fields (EMFs) interfere with your cells' ability to generate energy efficiently.

This energy deficit has a cascading effect, making it harder to maintain a healthy, oxygen-free gut environment that beneficial bacteria like *Akkermansia* thrive in. Moreover, when your cells are energy-starved, your gut environment shifts to favor endotoxin-producing bacteria, creating a vicious cycle.

To actively reduce your exposure to these toxins, be mindful of plastics, which are common sources of xenoestrogens, and try to minimize your use of plastic containers and wraps.

Seek out products free from EDCs, and take steps to reduce EMF exposure in your home and daily life. By addressing EDCs and EMFs, as well as reducing LA and exposure to other estrogenic compounds, you effectively restore your mitochondrial function, boost cellular energy, and set yourself on a path toward better overall health.

- **Optimize sun exposure and boost your NAD⁺ levels** — Sunlight and NAD⁺ are two powerful, natural allies for your mitochondrial health. Daily sun exposure promotes cellular energy production by stimulating the production of melatonin within your mitochondria, which your body makes in response to the near-infrared light in sunlight.

This mitochondrial melatonin is a potent antioxidant that helps protect your mitochondria from damage. It's important to avoid intense, direct sunlight during peak hours (typically 10 a.m. to 4 p.m. in most regions), however, until you've eliminated seed oils from your diet for at least six months.

This is because stored linoleic acid in your skin increases your risk of sunburn. In addition to sunlight, boosting your NAD⁺ (nicotinamide adenine dinucleotide) levels is also beneficial. Consider taking niacinamide, a form of vitamin B3, at a dosage of 50 milligrams three times daily to increase NAD⁺ production, which helps your mitochondria generate more energy.

- **HOMA-IR – a simple test for insulin resistance** – Recognizing insulin resistance early is essential, as it's a warning sign for your metabolic health – one that often precedes Type 2 diabetes. The HOMA-IR (Homeostatic Model Assessment of Insulin Resistance) test is a valuable diagnostic tool that helps assess insulin resistance through a simple blood test, so you can spot issues early and make necessary lifestyle changes. Talk to your healthcare provider about whether this testing is appropriate for you.

Created in 1985, it calculates the relationship between your fasting glucose and insulin levels to evaluate how effectively your body uses insulin. Unlike other more complex tests, HOMA-IR requires just one fasting blood sample, making it both practical and accessible. The HOMA-IR formula is as follows:

HOMA-IR = (Fasting Glucose x Fasting Insulin) / 405, where

- Fasting glucose is measured in mg/dL
- Fasting insulin is measured in $\mu\text{IU/mL}$ (microinternational units per milliliter)
- 405 is a constant that normalizes the values

If you're using mmol/L for glucose instead of mg/dL, the formula changes slightly:

HOMA-IR = (Fasting Glucose x Fasting Insulin) / 22.5, where

- Fasting glucose is measured in mmol/L
- Fasting insulin is measured in $\mu\text{IU/mL}$
- 22.5 is the normalizing factor for this unit of measurement

Anything below 1.0 is considered a healthy HOMA-IR score. If you're above that, you're considered insulin resistant. The higher your values, the greater your insulin resistance. Conversely the lower your HOMA-IR score, the less insulin resistance you have, assuming you are not a Type 1 diabetic who makes no insulin.

Interestingly, my personal HOMA-IR score stands at a low 0.2. This low score is a testament to my body's enhanced efficiency in burning fuel, a result of increased glucose availability. By incorporating additional carbohydrates into my diet, I provided my cells with the necessary energy to operate more effectively.

This improved cellular function has significantly boosted my metabolic health, demonstrating how strategic dietary adjustments lead to better insulin sensitivity and overall metabolic performance.

Frequently Asked Questions (FAQs) About Mitochondrial Function and Diabetes

Q: What is the link between mitochondrial health and diabetes?

A: Mitochondrial dysfunction is now recognized as a key contributor to the development and progression of diabetes. When mitochondria are damaged, they trigger a stress response that impairs the function and maturity of cells crucial for metabolism, especially insulin-producing beta-cells in the pancreas. This disruption leads to insulin resistance and poor blood sugar control, which are hallmarks of Type 2 diabetes.

Q: How does mitochondrial damage affect insulin production?

A: Damaged mitochondria in beta-cells send a distress signal to the cell's nucleus, halting proper maturation of the cells. As a result, these cells become less specialized and lose their ability to produce insulin effectively. This dysfunction was shown in mice and confirmed in human pancreatic islet cells, and the researchers believe it applies to other metabolic tissues affected by diabetes.

Q: Can mitochondrial dysfunction in diabetes be reversed?

A: In mice, it has been. Research using an experimental drug (ISRIB) showed that blocking the mitochondrial stress response restored insulin-producing beta-cell function and improved glucose regulation. ISRIB has not been tested as a diabetes treatment in people, so this points to a possible path toward addressing diabetes at the cellular level rather than an available treatment.

Q: What lifestyle changes support mitochondrial function and help prevent diabetes?

A: To support mitochondrial health and prevent diabetes, focus on:

- Eliminating processed foods and vegetable oils high in linoleic acid
- Optimizing carbohydrate intake with whole, digestible sources
- Minimizing exposure to environmental toxins, such as plastics and electromagnetic fields
- Getting regular sun exposure and boosting NAD⁺ levels with niacinamide

Q: How can I check for insulin resistance early on?

A: The HOMA-IR test is a simple, effective tool to assess insulin resistance using fasting glucose and insulin levels. A score under 1.0 indicates good insulin sensitivity, while higher scores suggest increasing resistance. Early detection allows for lifestyle interventions that may prevent the onset of Type 2 diabetes.

This article is for informational purposes only and does not constitute medical advice. Consult a qualified healthcare provider before making changes to your health regimen.

Sources and References

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