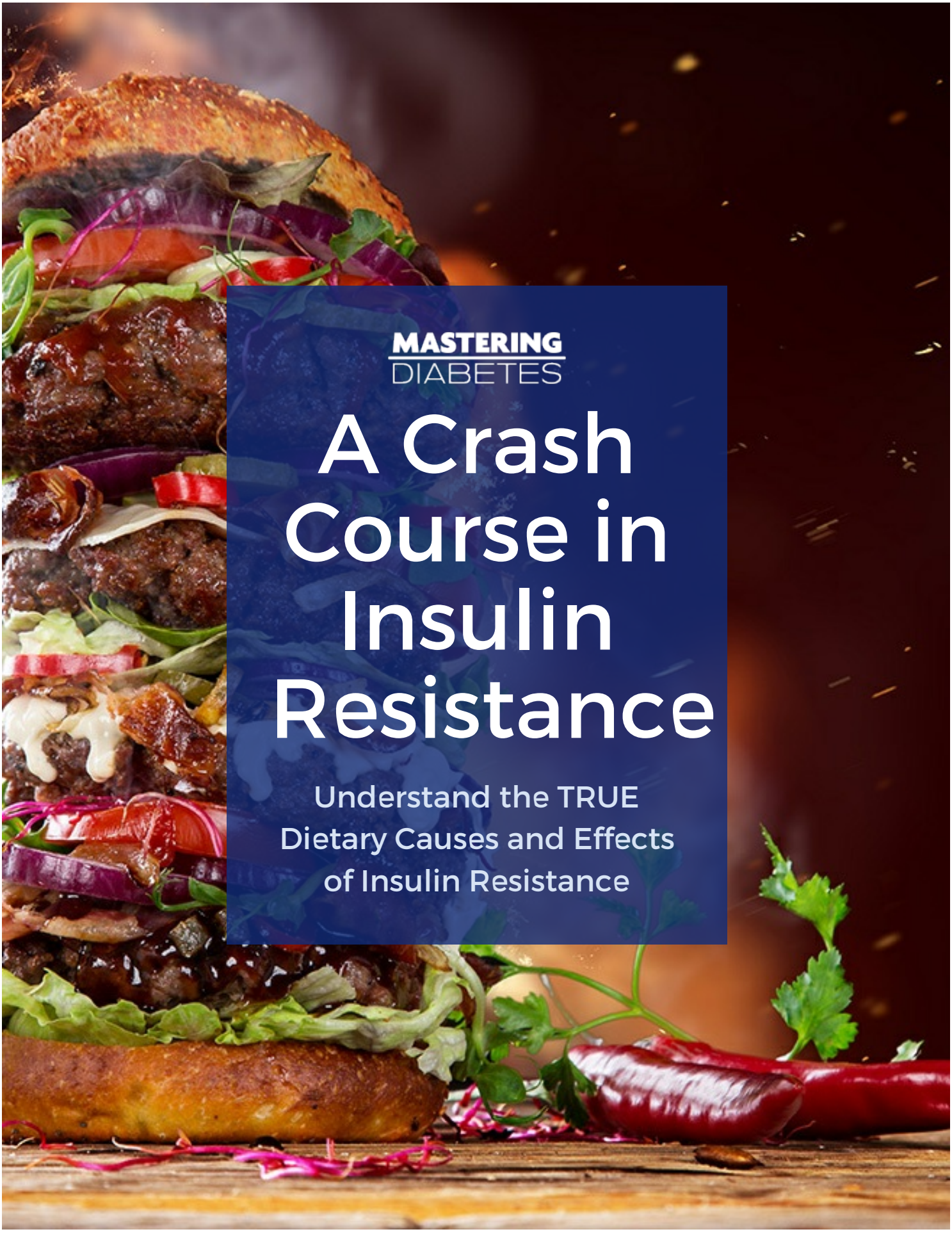

MASTERING
DIABETES
A Crash Course in Insulin Resistance
Understand the TRUE Dietary Causes and Effects of Insulin Resistance

The Causes of Insulin Resistance in Type 1 and Type 2 Diabetes

Insulin resistance is the common thread that underlies all forms of diabetes. In this ebook, you'll find out exactly why.

Most people believe that people with type 1 diabetes are not insulin resistant simply because they are not overweight. This could not be farther from the truth.

While insulin resistance affects many overweight individuals, many people with type 1 diabetes remain skinny their entire lives despite a large degree of insulin resistance (1–3).

Over the past decade, we have helped many people with type 1 diabetes, prediabetes and type 2 diabetes measure, track and reverse insulin resistance. In practice, 100% of all our clients with type 1 diabetes suffer from insulin resistance despite the *assumption* that they were insulin sensitive.

By measuring their baseline insulin resistance, we were able to identify an impaired ability to utilize glucose as a fuel, and through dedicated diet modification and frequent exercise, some of our clients have reduced their insulin usage by as much as 60%.

If you have type 1 diabetes, do not be fooled into thinking that you are insulin sensitive simply because you are skinny. Insulin resistance is a hidden condition, and affects both normal weight and overweight individuals (1–3).

What Causes Insulin Resistance?

Insulin resistance underlies all forms of diabetes, and is a condition which primarily affects your muscles, liver and adipose tissue.

Many people think that diabetes is caused by an excess intake of sugar and candy starting from a young age. While eating artificial sweeteners and drinking soda can certainly increase your risk for the development of insulin resistance and diabetes, in most cases diabetes is caused by excessive FAT intake.

The most important thing you can do as a person with diabetes is understand the following:

Diabetes is caused by a fat metabolism disorder, which results in a glucose metabolism disorder. At the heart of all forms of diabetes is insulin resistance, a condition fueled primarily by the intake and accumulation of excess fat across many tissues.

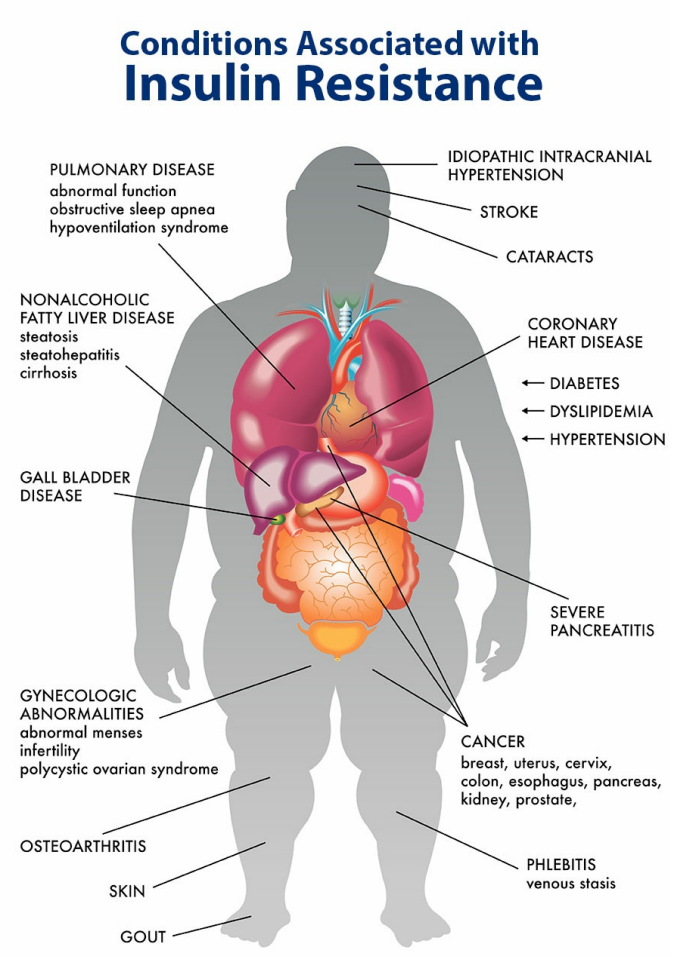
This concept is one of the most fundamental realities that can significantly improve your ability to control blood glucose. While most diabetes education will instruct you to count and minimize your carbohydrate intake, this does nothing to treat the underlying condition of insulin resistance.

In fact, minimizing carbohydrate intake results in an increase in fat intake, which makes you more insulin resistant over time. Minimizing carbohydrate intake only treats the *symptoms* of diabetes (high blood sugar), and often results in increased insulin resistance over time.

Importantly, insulin resistance is a *risk factor* for many chronic health conditions, including heart disease, high blood pressure (hypertension), high cholesterol, cancer, diabetes, Alzheimer's disease, kidney failure, stroke and nerve damage.

Unfortunately, insulin resistance is grossly misunderstood, and as a result there are many fad diets that claim to improve overall health but actually *increase* insulin resistance. As a consumer, it is very challenging to understand exactly what information to ingest and what information to disregard. Food manufacturers take advantage of this, and intentionally deceive people into buying their products.

But you are highly intelligent, highly motivated and are capable of applying these powerful methods to your daily life, and experience the difference that it makes first hand. By directly reducing your level of insulin resistance, you will improve the health of every organ in your body, to prevent against the conditions listed in the following diagram:



Low-Carb Diets are High-Fat Diets by Definition

A wealth of evidence supports the concept that insulin resistance results from the accumulation of excess fatty acids in tissues that are not designed to store fat, such as the liver and muscle. As these tissues accumulate fat over time, they experience cellular distress and mitochondrial dysfunction, which then results in a significantly impaired ability to respond to insulin (4–27).

There are a few fundamental problems with the no-carb or low-carb strategy that is specifically designed to reduce your intake of glucose (the primary breakdown product of carbohydrates):

- Glucose is a molecule which can be used by every tissue in the body for immediate energy
- Glucose is the very molecule that body tissues are designed to use as fuel
- Limiting carbohydrate intake results in the consumption of high amounts of protein and fat, which promote increased fat storage and exacerbate insulin resistance
- Eating large quantities fat directly blocks the action of insulin in the muscle and liver

Let's discuss the 3 causes of insulin resistance in detail.

Insulin Resistance Cause #1: Lipid Overload

There is only one tissue in your body that is designed to store fat, and that tissue is called adipose tissue (fat tissue). Fat tissue is perfectly designed to uptake, store and export fatty acids, and possesses all of the enzymatic machinery to do so.

Most importantly, fat tissue is an elastic tissue which expands and contracts in response to periods of high and low fatty acid availability.

When fatty acids are available, adipose tissue expands.

When fatty acids are limiting, adipose tissue contracts.

This very elastic behavior of adipose tissue is exactly what results in weight gain and weight loss, and can be manipulated to your advantage if weight loss is your long-term goal.

Insulin Resistance Cause #2: Refined Carbohydrates

We've all heard that you should eat less sugar, and eliminate food additives like high fructose corn syrup, because they increase your risk for cancer, heart disease and diabetes. But why is that the case?

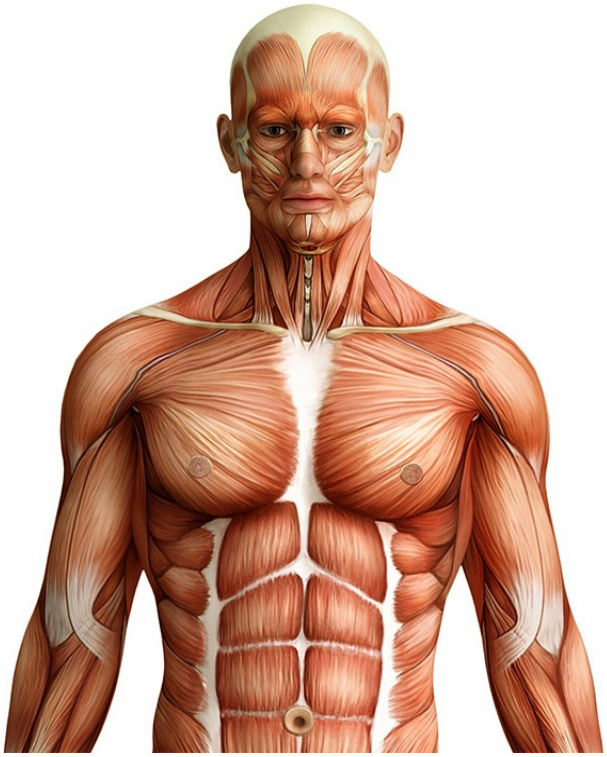
One simple way that refined carbohydrates promote diabetes is specifically by causing insulin resistance in the liver. Think of your liver as a glucose sponge, whose responsibility is to uptake glucose when it first appears in your bloodstream. In this way, your liver is actually *protecting* other tissues against large glucose surges following a carbohydrate-rich meal.

If you haven't done so already, thank your liver for acting as a glucose sponge every time you eat carbohydrates – it is literally sparing other tissues from metabolic damage caused by the rapid appearance of glucose.

When you eat refined carbohydrates - most often disguised as sugarnyms in packaged and processed food products – the breakdown products of these artificial sweeteners (mainly glucose and fructose) flood your liver immediately.

Insulin Resistance Cause #3: Insufficient Movement

Mitochondria are cellular organelles that function as cellular power plants. In the same way that a power plant produces electricity for a city, mitochondria are responsible for the production of energy derived from the breakdown of carbohydrates and fatty acids.



Mitochondria oxidize or “burn” carbohydrates, amino acids and fatty acids for energy, yielding ATP. ATP is the cellular form of energy utilized by cellular processes all throughout the body, providing the energy to pump your heart, power neurons in your brain, contract muscles in your limbs, exchange gases in your lungs, extract nutrients from food and regulate body temperature, to name just a few.Insufficient movement results in low mitochondrial number and a reduced ability to “burn” fuels. As a result, the muscle tissue is prone to accumulating fatty acids as triglycerides, resulting in insulin resistance.

Since muscle tissue occupies more than 40% of the human body by mass, a reduced ability of muscle mitochondria to burn fatty acids and glucose for energy is partly responsible for feelings of low energy and sluggishness that many people with diabetes experience.

Insulin resistance is a metabolic disaster for muscle tissue, and reversing insulin resistance involves the direct “reprogramming” of muscle mitochondria.

More importantly, defective muscle mitochondrial function often induces a mild inflammatory state within the muscle tissue that results in the production of blood borne myokines that signal a state of stress to circulating immune cells.

Take Home Messages

The take home message of this article is simple: insulin resistance has 3 very clear causes, and taking a series of specific actions can result in significantly increased insulin sensitivity in a short period of time.

- If you have type 1 diabetes, do not be mistaken into thinking that you are insulin sensitive simply because you are normal weight or skinny.
- If you have type 2 diabetes, insulin resistance is the underlying cause of high blood sugar.

References

1. Szadkowska A, Pietrzak I, Mianowska B, Markuszewski L, Bodalska-Lipińska J, Bodalski J. . Endokrynol Diabetol Chor Przemiany Materii Wieku Rozw Organ Pol Tow Endokrynol Dziecięcych. 2006;12(2):109–15.
2. Reinehr T, Holl RW, Roth CL, Wiesel T, Stachow R, Wabitsch M, et al. Insulin resistance in children and adolescents with type 1 diabetes mellitus: relation to obesity. Pediatr Diabetes. 2005 Mar;6(1):5–12.
3. Bergman BC, Howard D, Schauer IE, Maahs DM, Snell-Bergeon JK, Eckel RH, et al. Features of hepatic and skeletal muscle insulin resistance unique to type 1 diabetes. J Clin Endocrinol Metab. 2012 May;97(5):1663–72.
4. Roden M. How free fatty acids inhibit glucose utilization in human skeletal muscle. News Physiol Sci Int J Physiol Prod Jointly Int Union Physiol Sci Am Physiol Soc. 2004 Jun;19:92–6.
5. Silveira LR, Fiamoncini J, Hirabara SM, Procópio J, Cambiaghi TD, Pinheiro CHJ, et al. Updating the effects of fatty acids on skeletal muscle. J Cell Physiol. 2008 Oct;217(1):1–12.
6. Brehm A, Krssak M, Schmid AI, Nowotny P, Waldhäusl W, Roden M. Increased lipid availability impairs insulin-stimulated ATP synthesis in human skeletal muscle. Diabetes. 2006 Jan;55(1):136–40.
7. Roden M, Price TB, Perseghin G, Petersen KF, Rothman DL, Cline GW, et al. Mechanism of free fatty acid-induced insulin resistance in humans. J Clin Invest. 1996 Jun 15;97(12):2859–65.
8. Shulman GI. Cellular mechanisms of insulin resistance. J Clin Invest. 2000 Jul;106(2):171–6.
9. Randle PJ, Garland PB, Hales CN, Newsholme EA. The glucose fatty-acid cycle. Its role in insulin sensitivity and the metabolic disturbances of diabetes mellitus. Lancet. 1963 Apr 13;1(7285):785–9.
10. Hirabara SM, Silveira LR, Alberici LC, Leandro CVG, Lambertucci RH, Polimeno GC, et al. Acute effect of fatty acids on metabolism and mitochondrial coupling in skeletal muscle. Biochim Biophys Acta. 2006 Jan;1757(1):57–66.
11. Massao Hirabara S, de Oliveira Carvalho CR, Mendonça JR, Piltcher Haber E, Fernandes LC, Curi R. Palmitate acutely raises glycogen synthesis in rat soleus muscle by a mechanism that requires its metabolization (Randle cycle). FEBS Lett. 2003 Apr 24;541(1-3):109–14.
12. Hirabara SM, Curi R, Maechler P. Saturated fatty acid-induced insulin resistance is associated with mitochondrial dysfunction in skeletal muscle cells. J Cell Physiol. 2010 Jan;222(1):187–94.

13. Hirabara SM, Silveira LR, Abdulkader F, Carvalho CRO, Procopio J, Curi R. Time-dependent effects of fatty acids on skeletal muscle metabolism. J Cell Physiol. 2007 Jan;210(1):7–15.
14. Yuzefovych L, Wilson G, Rachek L. Different effects of oleate vs. palmitate on mitochondrial function, apoptosis, and insulin signaling in L6 skeletal muscle cells: role of oxidative stress. Am J Physiol Endocrinol Metab. 2010 Dec;299(6):E1096–105.
15. Yu C, Chen Y, Cline GW, Zhang D, Zong H, Wang Y, et al. Mechanism by which fatty acids inhibit insulin activation of insulin receptor substrate-1 (IRS-1)-associated phosphatidylinositol 3-kinase activity in muscle. J Biol Chem. 2002 Dec 27;277(52):50230–6.
16. Griffin ME, Marcucci MJ, Cline GW, Bell K, Barucci N, Lee D, et al. Free fatty acid-induced insulin resistance is associated with activation of protein kinase C theta and alterations in the insulin signaling cascade. Diabetes. 1999 Jun;48(6):1270–4.
17. Funai K, Song H, Yin L, Lodhi IJ, Wei X, Yoshino J, et al. Muscle lipogenesis balances insulin sensitivity and strength through calcium signaling. J Clin Invest. 2013 Mar 1;123(3):1229–40.
18. Delarue J, Magnan C. Free fatty acids and insulin resistance. Curr Opin Clin Nutr Metab Care. 2007 Mar;10(2):142–8.
19. Martins AR, Nachbar RT, Gorjao R, Vinolo MA, Festuccia WT, Lambertucci RH, et al. Mechanisms underlying skeletal muscle insulin resistance induced by fatty acids: importance of the mitochondrial function. Lipids Health Dis. 2012;11:30.
20. Wang P-Y, Kaneko T, Wang Y, Tawata M, Sato A. Impairment of Glucose Tolerance in Normal Adults Following a Lowered Carbohydrate Intake. Tohoku J Exp Med. 1999;189(1):59–70.
21. Xiao C, Giacca A, Carpentier A, Lewis GF. Differential effects of monounsaturated, polyunsaturated and saturated fat ingestion on glucose-stimulated insulin secretion, sensitivity and clearance in overweight and obese, non-diabetic humans. Diabetologia. 2006 Apr 5;49(6):1371–9.
22. Wolpert HA, Atakov-Castillo A, Smith SA, Steil GM. Dietary Fat Acutely Increases Glucose Concentrations and Insulin Requirements in Patients With Type 1 Diabetes Implications for carbohydrate-based bolus dose calculation and intensive diabetes management. Diabetes Care. 2013 Apr 1;36(4):810–6.
23. Savage DB, Petersen KF, Shulman GI. Disordered Lipid Metabolism and the Pathogenesis of Insulin Resistance. Physiol Rev. 2007 Apr 1;87(2):507–20.

24. Risérus U, Willett WC, Hu FB. Dietary fats and prevention of type 2 diabetes. Prog Lipid Res. 2009 Jan;48(1):44–51.
25. Pańkowska E, Błazik M, Groele L. Does the fat-protein meal increase postprandial glucose level in type 1 diabetes patients on insulin pump: the conclusion of a randomized study. Diabetes Technol Ther. 2012 Jan;14(1):16–22.
26. Gormsen LC, Nielsen C, Jessen N, Jørgensen JOL, Møller N. Time-course effects of physiological free fatty acid surges on insulin sensitivity in humans. Acta Physiol Oxf Engl. 2011 Mar;201(3):349–56.
27. Boden G. Fatty acid-induced inflammation and insulin resistance in skeletal muscle and liver. Curr Diab Rep. 2006 Jun;6(3):177–81.
28. Hellerstein MK, Schwarz JM, Neese RA. Regulation of hepatic de novo lipogenesis in humans. Annu Rev Nutr. 1996;16:523–57.
29. Hellerstein MK. De novo lipogenesis in humans: metabolic and regulatory aspects. Eur J Clin Nutr. 1999 Apr;53 Suppl 1:S53–65.
30. Stanhope KL, Schwarz JM, Keim NL, Griffen SC, Bremer AA, Graham JL, et al. Consuming fructose-sweetened, not glucose-sweetened, beverages increases visceral adiposity and lipids and decreases insulin sensitivity in overweight/obese humans. J Clin Invest. 2009 May;119(5):1322–34.

MASTERING DIABETES

Looking for a Diabetes Coach to Help You Transition?

If you're looking for education, guidance, and accountability to help you transition to a plant-based diet for the BEST diabetes health you've ever had, sign up for a free strategy session to see if you're a good fit for an online group coaching program (type 1, type 1.5, prediabetes and type 2 diabetes).

BOOK A FREE STRATEGY SESSION