

'Reversing type 2 diabetes starts with ignoring the guidelines': education from Dr Sarah Hallberg's TEDx talk

Sarah Hallberg^{1,2,3}

When I was preparing this talk for TEDx Purdue in 2015, I had no idea it would be viewed over 2.8 million times. I firmly believe the reaction to this talk is an expression of people being fed up. Fed up with the status quo. Fed up with listening to the advice they were given only to get worse. Fed up with yet another prescription bringing another side effect and more costs. Fed up and looking for advice that is not only evidence-based,¹⁻¹¹ but also doable. Fed up and searching for a way to escape the progressive cycle of type 2 diabetes. Here it is. A solution that goes back to the basics and remembers that we can and should use Food as Medicine.

I have the best job in the world. I'm a doctor... believe me that's not why. I am an obesity doctor. I have the honour to work with a group of people subject to the last accepted prejudice...being fat. These patients have suffered a lot by the time they come to see me - shame, guilt, blame and outright discrimination. The attitude most people take, including those in healthcare, is that these people are to blame for their situation. If they could just control themselves they would not be overweight. They are not motivated to change. Please let me tell you; that is not the case. The real blame, if we need to assign some, has been our advice and it's time to change that.

Obesity is a disease, not something created by lack of character. It is a hormonal disease. There are many hormones involved, and one of the main ones is a hormone called insulin. The vast majority of obese individuals are resistant to insulin and that causes a lot of trouble. So, what does being insulin-resistant mean? Insulin resistance is essentially 'pre-pre-type 2 diabetes'. Insulin's job is to drive glucose or blood sugar into

cells where it can be used. In a nutshell, when someone has insulin resistance, they are having trouble getting glucose where it needs to go, into the cells. It can't all hang out in the blood after we eat or we would all have a diabetic crisis after every meal. When there is resistance to insulin, our bodies will just make more of it. The insulin levels rise and rise and for a while, years usually, this will keep up and blood sugar will stay normal. However, eventually it can't keep up, and even elevated insulin levels are not enough to keep blood sugar normal, and blood sugar rises...that is diabetes.

It is probably not surprising that most of my patients have type 2 diabetes or insulin resistance. If you think that is "phew", not you, you might want to think again. Over 50% of adult Americans have diabetes or pre-diabetes.¹² This is almost 120 million Americans.¹³ However, that hardly includes everyone with insulin issues. Most people have had insulin resistance (remember, that is elevated insulin levels) for years, decades even, before they actually get the diagnosis of type 2 diabetes. In addition, it has been shown that up to 25% of normal weight adults have insulin resistance.¹⁴ In case you are keeping track, that is a heck of a lot of us.

So, the issue with elevated insulin is that it leads to diabetes, but also insulin may promote hunger. It increases fat storage and has been shown to be associated with inflammation. Inflammation itself contributes to a host of problems including heart disease and cancer. So, what if we go back to the initial science of the problem with elevated insulin, and just did not have as much glucose around that needed to be dealt with?

Let's look at how that could be...

Everything you eat is either a carbohydrate, protein or fat. They all affect glucose, and therefore insulin levels differently.

Fat does not cause a glucose or insulin response, and that winds up being pretty important.

Now, let's look at how that fact translates into a real-world situation.

Look at an American version of Chinese food. We all know that there are two rules that come with meals like this. Number one, you will overeat. The shut-off signal does not get sent until you are literally busting at the seams. Number two, you will be starving 1 hour later. Why? The rice has caused glucose and then insulin to shoot up and then plummet. This often triggers hunger, fat storage and cravings.¹⁵

So, if you have insulin resistance or diabetes, and therefore your insulin is higher, you really can be just plain hungrier all the time AND you are more likely to store your intake as fat.

Now, here is how the guidelines currently handle diabetes and insulin resistance. The recommendation generally is to eat 40–65 g of carbohydrates per meal plus snacks. Trust me, that is a lot of carbs, and remember what we just saw about what happens to glucose and insulin when we eat them? Yes, essentially it is being recommended that they eat exactly what is causing their problems. Seem crazy? It actually really, really is. At its root, diabetes is a state of carbohydrate toxicity. We can't get the glucose into cells, and its build-up is dangerous in the short term but even worse in the long term. Insulin resistance is a state of carbohydrate intolerance. Why oh why do we want to recommend to patients that they continue to eat them?

The American Diabetes Association (ADA) guidelines state that there is inconclusive evidence to recommend a specific carbohydrate limit. However, the guidelines also go on to state what we know; carbohydrate intake is the biggest predictor of glucose levels and the need for medication. In fact, they also advise that if you are on certain meds you MUST eat carbs so you won't get low blood sugars. Or, I would just recommend restricting the carbs to make all those meds and side effects unnecessary. Seriously, so it is: eat carbs, take meds, then eat more carbs so the meds don't make your blood sugar low. HUH? Please, think about that vicious cycle for a moment. That is really crazy. Even worse is that nowhere in the ADA guidelines is the goal of reversing type 2 diabetes. This needs to be changed. Type 2 diabetes can be reversed in many or most if we start early enough. Not only do we need to let people know this, we have to start giving them practical advice so that they can DO this.

Consider carbs. First, here is a shocker... we don't need them.¹⁶ Seriously, our minimum daily requirement for carbs is zero. We have essential amino acids (proteins) and essential fatty acids, but

¹Clinical Medicine, Virta Health, San Francisco, California, USA

²Medically Supervised Weight Loss, Indiana University Health, Lafayette, Indiana, USA

³Medicine, Indiana University School of Medicine, Indianapolis, Indiana, USA

Correspondence to Dr Sarah Hallberg, Clinical Medicine, Virta Health, San Francisco, CA 94105, USA; hallbers@IUHealth.org

nope, no essential carb. Now of course, that does not mean we don't eat them, we just don't need them. A nutrient is essential if we can't produce it from something else, and it is required by our body to function. We can produce glucose and plenty of it; we do this all the time, it is called gluconeogenesis.

So, we don't actually need them, the overconsumption of them is making us sick, yet it continues to be advised to eat close to half or more of our energy from them. Doesn't make sense. Let's talk about what does.

Cutting carbs, A LOT. Yes, at our clinic, we teach patients how to eat with carbs as the minority of their intake, not the majority.

So, how does that work? Well, when patients cut their carbohydrate intake, their glucose goes down and they don't need as much insulin, so insulin levels drop and fast. This is very significant as an article analysing National Health and Nutrition Examination Survey (better known as NHANES) data showed that insulin resistance is the single most important risk factor for coronary artery disease (CAD), responsible for 42% of heart attacks.¹⁷ More disturbing for the influence of insulin resistance in vascular health is that these were data from 1998 to 2004. Quick secret—WE ARE WAY WORSE NOW!

Low-carb intervention works so fast we have to cut meds right away in patients with diabetes. Patients can get off over 100 units of insulin in weeks.

One of my favourite stories is a very recent one. A young gal with almost a 20-year history of type 2 diabetes came to see us after a physician at another clinic told her she was just 'sick'. She had evidence of coronary artery disease, and her diabetes was very uncontrolled despite multiple diabetic medications including 300 units of insulin being injected into her continuously through the day via a pump. Let me tell you, even in the daily world of diabetes I work in, 300 units is a lot of insulin. Plus, remember, her diabetes was NOT controlled with all of these. She was overweight, but not actually obese. We put her on a low-carb diet. Fast forward 4 months. Sick no longer. She lost weight yes, but much more important is her blood sugar was totally normal all the time on... NO medications. No 300 units of insulin, no more pump, no more pricking her finger multiple times each day. Gone, all of it. No more diabetes.

Probably the best part of my job is to be able to tell a patient that they do not have diabetes any longer and ceremoniously

take it off their problem list. Just since I wrote this talk, we have had yet another person off a pump and multiple more see their diabetes resolved and I don't just see patients with diabetes.

So, are they cured? Is it miraculous? We will leave that grandstanding to Dr Oz. Cured would imply it can't come back; if they start eating excessive carbs again it will, so no, not cured. However, they don't have diabetes any longer. Their blood sugars are normal so it is resolved, and can stay that way if we continue to keep away the cause.

So, what does this look like? How can you possibly eat this way? First, let me tell you what it is not. It is not zero-carb eating. This is a big criticism of low-carb that is so frustrating. Low carb is not zero carb.

Second, if we cut carbs what do we put in? There are only three macronutrients classes for food. So, if one goes down, one has to go up. Well, my patients eat fat, and a lot of it. What? What happens when you eat fat? Well, you are happy for one, because fat tastes great and is incredibly satisfying. Most people don't even realise this because we have been so programmed to be fat-phobic. Also, remember, fat is the macronutrient that keeps our glucose down.

Here are the simple rules for eating:

1. If it says "light", "low fat" or "fat free", it stays in the grocery store. If they took the fat out, they put carbs and chemicals in it.
2. EAT food—I mean real food. This is the single most important component of eating low carb. Real food does not come in boxes. You don't have to be told food is natural; you should just know that by looking at it.
3. Don't eat anything you don't like.
4. Eat when you are hungry and don't eat when you are not.
5. No GPS—no grains, potatoes or sugar. The last one is a biggie—no grains? Correct, no grains. But wait, we need them! Uh, actually we don't, they are a carb. But what about wholegrains they are so good! First, there are actually very few foods out there that are actually wholegrain even when they say they are. Most of the foods that purport themselves to be wholegrain are processed and the fibre benefit is ruined or they come with a lot of highly processed flour in them. Usually it is both of these things. So, someone who is not already insulin-resistant can eat true wholegrain, but in the enormous slice of our population with insulin issues, they are making things worse.

Can you eat low carb if you are one of the lucky insulin-sensitive people? Yep.

I'm a good example. I believe in practice what you preach. I have always been lower carb. I just didn't really realise it when I was younger. Over 1 year ago, I decided I would go as low as I recommend to my patients with diabetes. I am not insulin-resistant. So, would this be bad? No, that's the thing. It might not be necessary like it is for a lot of my patients, but unless you have an exceedingly rare syndrome, then cutting carbs will be good for you even if not necessary. I would never change my food. I love it!

People send me pictures of all the time of their 'radical' food.

This is actually very significant. People who suffer from obesity would usually never take a picture of their food. There is too much shame associated with that. However, when they start losing, feeling great about what they are putting in their body, and are making incredibly fantastic and delicious food, this is gone. That is huge.

So, what about the research on this? Is this just anecdotal evidence from our clinic? No, there are literally dozens of randomised controlled trials on low carb looking at cardiovascular risk factors, diabetes and weight loss, and they are consistent.^{1-11 18-27} It works. There is even a study from *The New England Journal of Medicine* comparing it with the Mediterranean diet, and it was better there as well.⁵ There are also multiple studies that show that low carb decreases markers of inflammation, which is part of the reason it is being looked at for cancer.^{10 27-29}

Our retrospective research pilot comparing patients with diabetes from our low-carb, high-fat-based programme to patients treated with ADA guidelines show not only a metabolic advantage, but (and let's face it, this is big and important) a cost savings. Our analysis showed an annual cost saving of over \$2000 each year per patient just in the diabetic meds they are no longer taking. Just think how fast this adds up given the epidemic of diabetes.

Results JUST for insulin over 6 months show a decrease of 493 units in the low carb group compared to an increase of 349 units in the standard of care group. We can start to see where the cost savings are coming from. Insulin is really expensive, and remember, this is just insulin. Many patients with diabetes are not even on insulin, but the numbers are striking, and really represents two ways of approaching this disease: the first, has the goal of resolving it; the second, very clearly aligns with the ADA guideline statement that diabetes is a progressive disease that over time needs more meds. Again, I point out, it is progressive unless we take away the cause.

This intervention is the direction employee health needs to go. Diabetes' costs to employers are staggering. Not only are healthy patients more productive patients, but we can save a huge amount on their healthcare expenses as well.

So, what is the problem? Why is a low carb diet not widespread? It is actually almost like it is a secret. In fact, a very common scenario in my office is that patients will come in crying. "Why didn't anyone tell me this years ago? Think of what this could have saved me."

Well there are two big reasons it is not in widespread use. First, is status quo. It is hard to break. There are many egos involved. We adopted the idea that low fat is the way to go decades ago, but a recent paper just published showed there was no evidence supporting the recommendation to cut fat from our diet.³⁰ Cutting the fat is what added the extra carbs into diets everywhere. It was essentially a big experiment on hundreds of millions of people and it failed miserably, but let's face it, it sounds good. If you don't want to be fat, you don't want to eat fat. I would argue that a lot of the issue is just in the name. Let's try calling dietary fat rainbows and butterflies instead, and then maybe we can keep from automatically associating it with the problems of being fat. Dietary fat and the fat we carry on us are fundamentally different. However, this association has stuck and has influenced guidelines for years. The second reason we are not seeing low carb everywhere is money. Don't be fooled, there is a lot of profit to be made by keeping you sick. Many of the specialty guideline panels are fraught with conflict of interest. The *British Medical Journal* has covered the conflict of interest recently.³¹

The solution to our diabetes epidemic is very evident in my clinic. For a problem with its root cause being carbohydrates, cut the carbohydrates. And remember what we used to know:

Let food be thy medicine and medicine be thy food.—Hippocrates.

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REFERENCES

- Goday A, Bellido D, Sajoux I, et al. Short-term safety, tolerability and efficacy of a very low-calorie-ketogenic diet interventional weight loss program versus hypocaloric diet in patients with type 2 diabetes mellitus. *Nutr Diabetes* 2016;**6**:e230.
- Daly ME, Paisley R, Paisley R, et al. Short-term effects of severe dietary carbohydrate-restriction advice in Type 2 diabetes – a randomized controlled trial. *Diabet Med* 2006;**23**:15–20.
- Dyson PA, Beatty S, Matthews DR. A low-carbohydrate diet is more effective in reducing body weight than healthy eating in both diabetic and non-diabetic subjects. *Diabet Med* 2007;**24**:1430–5.
- Westman EC, Yancy WS, Mavropoulos JC, et al. The effect of a low-carbohydrate, ketogenic diet versus a low-glycemic index diet on glycemic control in type 2 diabetes mellitus. *Nutr Metab* 2008;**5**:36.
- Shai I, Schwarzfuchs D, Henkin Y, et al. Weight loss with a low-carbohydrate, mediterranean, or low-fat diet. *N Engl J Med* 2008;**359**:229–41.
- Volek JS, Phinney SD, Forsythe CE, et al. Carbohydrate restriction has a more favorable impact on the metabolic syndrome than a low fat diet. *Lipids* 2009;**44**:297–309.
- Guldbrand H, Dizdar B, Bunjaku B, et al. In type 2 diabetes, randomisation to advice to follow a low-carbohydrate diet transiently improves glycaemic control compared with advice to follow a low-fat diet producing a similar weight loss. *Diabetologia* 2012;**55**:2118–27.
- Saslow LR, Kim S, Daubenmier JJ, et al. A randomized pilot trial of a moderate carbohydrate diet compared to a very low carbohydrate diet in overweight or obese individuals with type 2 diabetes mellitus or prediabetes. *PLoS One* 2012;**9**:e91027.
- Davis NJ, Tomuta N, Schechter C, et al. Comparative study of the effects of a 1-year dietary intervention of a low-carbohydrate diet versus a low-fat diet on weight and glycemic control in type 2 diabetes. *Diabetes Care* 2009;**32**:1147–52.
- Jonasson L, Guldbrand H, Lundberg AK, et al. Advice to follow a low-carbohydrate diet has a favourable impact on low-grade inflammation in type 2 diabetes compared with advice to follow a low-fat diet. *Ann Med* 2014;**46**:182–7.
- Tay J, Luscombe-Marsh ND, Thompson CH, et al. A very low-carbohydrate, low-saturated fat diet for type 2 diabetes management: a randomized trial. *Diabetes Care* 2014;**37**:2909–18.
- Menke A, Casagrande S, Geiss L, et al. Prevalence of and trends in diabetes among adults in the United States, 1988–2012. *JAMA* 2015;**314**:1021–9.
- U.S. Census Bureau. *Population Estimates, July 2016*, 2016.
- Wildman RP, Muntner P, Reynolds K, et al. The obese without cardiometabolic risk factor clustering and the normal weight with cardiometabolic risk factor clustering: prevalence and correlates of 2 phenotypes among the US population (NHANES 1999–2004). *Arch Intern Med* 2008;**168**:1617–24.
- Rodin J. Insulin levels, hunger, and food intake: an example of feedback loops in body weight regulation. *Health Psychol* 1985;**4**:1–24.
- Bier DM, Brosnan JT, Flatt JP, et al. Report of the IDECG Working Group on lower and upper limits of carbohydrate and fat intake. International Dietary Energy Consultative Group. *Eur J Clin Nutr* 1999;**53**(Suppl 1):S177–8.
- Eddy D, Schlessinger L, Kahn R, et al. Relationship of insulin resistance and related metabolic variables to coronary artery disease: a mathematical analysis. *Diabetes Care* 2009;**32**:361–6.
- Brehm BJ, Seeley RJ, Daniels SR, et al. A randomized trial comparing a very low carbohydrate diet and a calorie-restricted low fat diet on body weight and cardiovascular risk factors in healthy women. *J Clin Endocrinol Metab* 2003;**88**:1617–23.
- Yancy WS, Olsen MK, Guyton JR, et al. A low-carbohydrate, ketogenic diet versus a low-fat diet to treat obesity and hyperlipidemia: a randomized, controlled trial. *Ann Intern Med* 2004;**140**:769–77.
- Volek J, Sharmar M, Gómez A, et al. Comparison of energy-restricted very low-carbohydrate and low-fat diets on weight loss and body composition in overweight men and women. *Nutr Metab* 2004;**1**:13.
- Meckling KA, O'Sullivan C, Saari D. Comparison of a low-fat diet to a low-carbohydrate diet on weight loss, body composition, and risk factors for diabetes and cardiovascular disease in free-living, overweight men and women. *J Clin Endocrinol Metab* 2004;**89**:2717–23.
- Gardner CD, Kiazand A, Alhassan S, et al. Comparison of the Atkins, Zone, Ornish, and LEARN diets for change in weight and related risk factors among overweight premenopausal women: the A TO Z Weight Loss Study: a randomized trial. *JAMA* 2007;**297**:969–77.
- Foster GD, Wyatt HR, Hill JO, et al. A randomized trial of a low-carbohydrate diet for obesity. *N Engl J Med* 2003;**348**:2082–90.
- Samaha FF, Iqbal N, Seshadri P, et al. A low-carbohydrate as compared with a low-fat diet in severe obesity. *N Engl J Med* 2003;**348**:2074–81.
- Krebs NF, Gao D, Gralla J, et al. Efficacy and safety of a high protein, low carbohydrate diet for weight loss in severely obese adolescents. *J Pediatr* 2010;**157**:252–8.
- Bazzano LA, Hu T, Reynolds K, et al. Effects of low-carbohydrate and low-fat diets: a randomized trial. *Ann Intern Med* 2014;**161**:309–18.
- Stern L, Iqbal N, Seshadri P, et al. The effects of low-carbohydrate versus conventional weight loss diets in severely obese adults: one-year follow-up of a randomized trial. *Ann Intern Med* 2004;**140**:778–85.
- Ruth MR, Port AM, Shah M, et al. Consuming a hypocaloric high fat low carbohydrate diet for 12 weeks lowers C-reactive protein, and raises serum adiponectin and high density lipoprotein-cholesterol in obese subjects. *Metabolism* 2013;**62**:1779–87.
- Forsythe CE, Phinney SD, Fernandez ML, et al. Comparison of low fat and low carbohydrate diets on circulating fatty acid composition and markers of inflammation. *Lipids* 2008;**43**:65–77.
- Harcombe Z, Baker JS, Cooper SM, et al. Evidence from randomised controlled trials did not support the introduction of dietary fat guidelines in 1977 and 1983: a systematic review and meta-analysis. *Open Heart* 2015;**2**:e000196.
- Neuman J, Korenstein D, Ross JS, et al. Prevalence of financial conflicts of interest among panel members producing clinical practice guidelines in Canada and United States: cross sectional study. *BMJ* 2011;**343**:d5621.