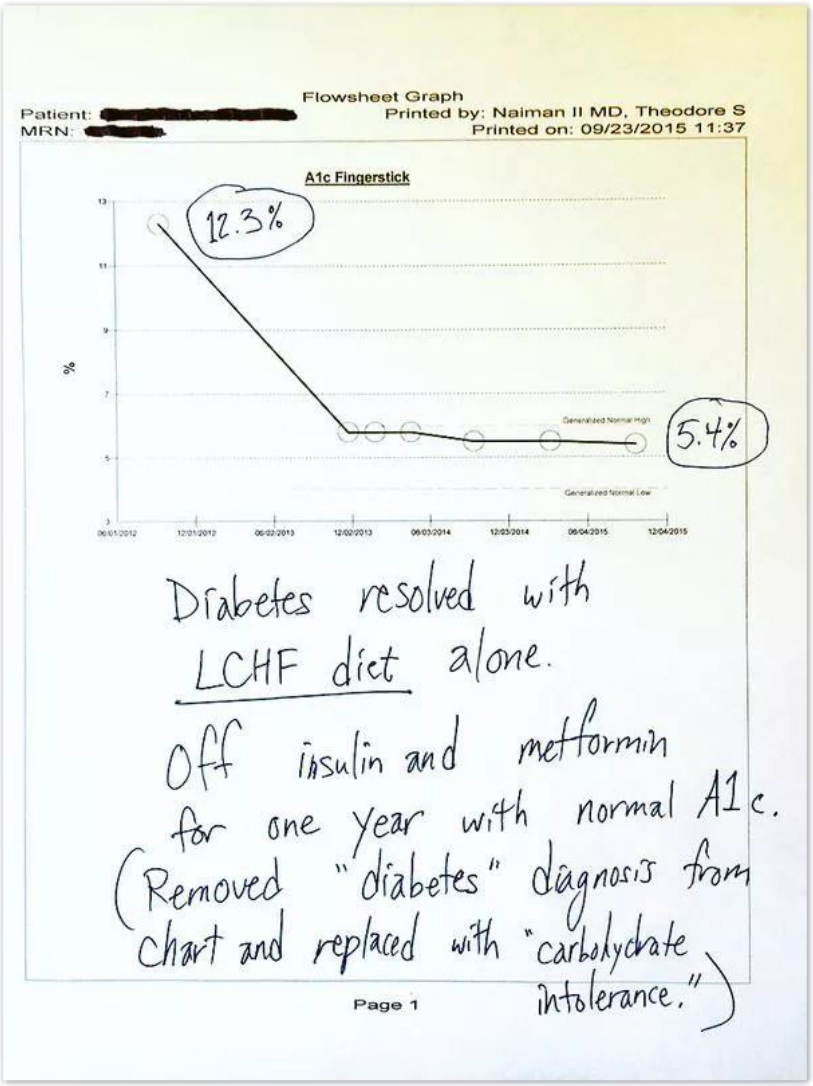
In my last video, I talked about how despite what some authorities may say, Type 2 diabetes does not have to be a chronic and progressive condition as clearly evidenced by the fact that Doctors like Jason Fung, David Unwin, Sarah Hallberg, and Ted Naiman just to name a few have been reversing people's diabetes with fasting or a low carb or ketogenic diet. While some people might be suspicious of a new fad diet supposedly reversing diabetes, low carb isn't actually new at all, nor is the concept of using it to reverse diabetes.



Dr. Richard Thomas Williamson [wrote in his 1898 book "Diabetes Mellitus and Its Treatment"]: that "*Potatoes should be excluded from the diet first,*

then bread, and gradually all carbohydrates should be cut off."

In 1797 military surgeon John Rollo released a book that described how he treated diabetes with a carbohydrate restricted meat diet.

Referring to this, Dr. Williamson wrote "*Ever since Rollo published his book on diabetes in 1797, and pointed out the value of restriction of the carbohydrates in the food, it has been acknowledged that of all forms and methods of treatment this dietetic one is the most important.*"

Interestingly, the concept of "grain avoidance" or *bìgǔ, (*also known as BIGU) has been around for more than 2000 years, going back to the Han dynasty.

Eastern Jin Dynasty scholar Ge Hong claimed that there was not one fat person among those practicing *bìgǔ* for spiritual reasons and Wáng xuán hé wrote in around the year 680AD that "before the times of farming, ancient people did not eat grain, so they were long lived."
(玄古之人所以壽考者造次之閒不食穀也 | 玄古之人所以寿考者，造次之间不食谷也。)

Before I get into discussing recent concerns about low carb diets, we need to look in more depth at how carbohydrate is a factor in the development of diabetes. Based on the work of Dr. Ted Naiman, Dr. Jason Fung and Ivor Cummins, the path to developing diabetes looks something like this:

-First, something is filling up your fat cells, making you fat.
-More specifically, something isn't just filling your fat cells up with fat, it's pinning that fat there and preventing it from being pulled out of the fat cell to be burned up for energy.
-As we discussed last time, this thing filling up your fat cells is insulin. Insulin is an anabolic hormone that has many functions, but one is that it acts as your "fat storage" hormone and by inhibiting three important

enzymes, it prevents you from burning fat.[R2]
-Simply put, the more insulin you have floating around, the harder it is to empty your fat cells and the fat builds up.
[Play Ted Naiman: "You filled up your fat cells, *because you suck at burning fat because you eat too much glucose… you're eating carbs and glucose, you're not burning fat, it accumulates, you fill up your adipose."]*
-What Dr. Ted Naiman said here is easy to follow because glucose which comes from carbohydrates raises insulin more than protein and *much more* than dietary fat. However, in this presentation, Dr. Naiman talks about another phenomenon usually called the Randle cycle. This is where independent of the action of insulin, glucose prevents the burning of fat all by itself.
"Ted Naiman: *Glucose and fat are oxidized reciprocally,, so anytime you're burning more glucose you're burning less fat, and more fat you're burning less glucose, right?*"

And this makes a lot of sense, because you need to be able to quickly sort out incoming glucose *first* since there's not many places to put it. Depending on the person you can store a couple hundred grams of glucose across your muscles and liver as glycogen, but if you don't empty your glycogen by from the liver by not eating carbohydrate long enough and you don't empty muscle glycogen by doing enough exercise, those glycogen stores will fill up. At this point there's nowhere to put incoming carbohydrate, so insulin has to turn it into fat via de novo lipogenesis so it can be stored in the only place available: your fat cells.

Dr. Naiman presents a model for how this works [Roll Naiman clip]

This highlights how dietary fat can also be very fattening, *if* you eat it with a enough carbohydrate because the carbohydrate will prevent you from burning that fat and the insulin release will hold the fat in storage.

So, to put this together: if you ingest too much low fiber carbohydrate *too*

frequently, it will interfere with your ability to burn fat and fat will accumulate. As your fat cells get more and more filled with fat, you'll need more insulin to hold that fat in place, making your fasting insulin very high - kind of like needing more force to cram more stuff in your suitcase. And these higher levels of insulin worsen the whole cycle making you more resistant to insulin, meaning glucose is harder to process because there's basically nowhere to put that glucose - you can only make energy from glucose at a certain rate, and your glycogen stores were filled up a long time ago, so the glucose needs to be turned into fat for storage, but your fat cells are already filled up. When your fat cells have no more room for new fat, where does it go?

Unfortunately it overflows into the abdominal cavity, the liver, the muscle and the pancreas. On that note, global rates of non-alcoholic fatty liver disease are as high as 24%.

The pancreas seems to be the last place that the fat accumulates, and when the pancreas is stuffed with fat, the beta cells stop functioning properly. Whereas your beta cells were pumping out a ton of insulin to make everything I just mentioned happen, now that the beta cells have become dysfunctional, they stop properly producing insulin, your insulin levels drop drastically meaning glucose can't be dealt with, so you get diabetic level blood sugar and then you are prescribed insulin injections to keep the blood sugar down but that makes it even harder to empty the fat cells, so the situation gets worse and worse .

Keep in mind that as Ivor Cummins points out in this talk, there are other unhealthy lifestyle factors that can worsen this insulin resistance and worsen this whole situation - things like insufficient sleep, smoking, and poor omega-3 to omega-6 ratio. Maybe the worst is fructose, a component of table sugar that directly worsens insulin resistance in the liver. So depending on the person, they might be fine and insulin sensitive eating spaghetti carbonara twice a day for many years if they're not drinking too

much fruit juice or sugar sweetened sodas and have these other factors in order.

Soda is only food that's actually directly linked to obesity. Margo Wootan says it's the calories, but I would bet Dr. Robert Lustig would say it's the fructose in the sugar causing insulin resistance by serine phosphorylating insulin receptor IRS-1

Before we move on let me present two pieces of evidence for the overflow phenomenon. Going back to Dr. Ted Naiman's talk, he gives an excellent example of overstuffed fat causing insulin resistance: People with lipodystrophy. this is a disorder where you have hardly any subcutaneous fat - So these people look like athletes, but this means they barely have any space to safely store any fat. So where does new fat go? Naiman: "*The visceral fat is completely maxed out, and almost everyone with lipodystrophy has horrible insulin resistance.*" Because there's much less room in these people to safely store fat, the overflow phenomenon happens much more quickly, making them more insulin resistant.

This could explain the peculiar situation in Japan, where despite having a very low obesity rate of about 3.7%, their rate of diabetes is 7.6%, only 2% lower than America's 9.4% , but America's obesity rate is about 30%. The people of Japan and other asian countries' difficulty getting fat could ironically be making them more susceptible to diabetes and insulin resistance because they have less safe fat storage space.

So what happens if you clear up this roadblock by giving an insulin resistant person a new place to store fat ? There was a study that tried just that -at least, in mice. They got some lipodystrophic mice with diabetes. Surgically implanted some fat tissue under the mouse's skin, connected it to their blood supply and like opening a new lane clears traffic congestion, having a new place to put fat cleared up the road block and magically cured

insulin resistance in these mice.

So, given all this, fixing these other factors Ivor Cummins talked about and fasting or going on a low carbohydrate diet so you get the insulin down and the fat burning up would make the most sense. But, **are Low carb diets bad for long term health?**

You surely saw articles about that study showing that low carb diets are bad for longevity, I saw that everywhere in English websites, and it was even popping up on my notifications from Japanese websites. Though, as Nina Teicholz points out, this study was based on a simple fill in the dot questionnaire, similar to this one, querying the participants on on only 66 foods. What if you don't happen to eat these particular vegetables? The data gets left out. What if you eat a bunch of pizza and energy drinks, it's not on the list so that data is left out.

As Teicholz explains: "*Further, the ARIC participants' eating habits were tracked only twice, from 1987-89 and 1993-95. After 1995 the study's participants were assumed to have continued eating the same diet for the next 15 years."*

Also, the authors threw out any data on carb consumption from subjects who "developed heart disease, diabetes, and stroke" before the second diet visit. I would think that's pretty relevant.

Diet questionnaires are inherently unreliable since most people aren't tracking precisely what they ate in what amount. Especially in this case, you can't hope to be precise with this kind of questionnaire. Can you remember how many times per month you ate bananas, broccoli and chicken *over the past year??*

On the other hand, this study, published last year *in the same journal* found the exact opposite result. High carb intake was linked with a higher risk of

total mortality. Saturated fat intake was not associated with heart disease and was linked to decreased stroke risk.

The conclusion was that "*High carbohydrate intake was associated with higher risk of total mortality, whereas total fat and individual types of fat were related to lower total mortality. Total fat and types of fat were not associated with cardiovascular disease, myocardial infarction, or cardiovascular disease mortality, whereas saturated fat had an inverse association with stroke. Global dietary guidelines should be reconsidered in light of these findings.*"

Next, you may have heard that top US cardiologist Kim Williams was saying just last month that "No one should be doing the keto diet." His argument is based on a 2013 systematic review of 17 studies that found low-carbohydrate diets to be associated with an increased risk of heart disease and death.

So, I went ahead and took a look at these 17 studies which by the way are based on questionnaires. First of all, let me point out that none of these studies even mentioned a ketogenic diet. One study specifically said "*Our results **do not** support a clear, general association between LCHP score and mortality.*"[R4] And four studies actually showed *better* health outcomes with carbohydrate reduction, particularly high glycemic carbohydrates.[R12,R14,R15,R16] Then, five studies weren't even looking at carbohydrate intake in specific, but just healthy eating patterns.

McCullough, 2000* [25]	Healthy eating index-f	Relative risk	Quintile 5	Quintile 1	Age (5-y categories), smoking (never, past, 1–14 cigarettes/d, 15–24 cigarettes/d, or ≥25 cigarettes/d), time period, body mass index (quintiles), alcohol intake (7 categories), physical activity (6 categories of metabolic equivalents), history of hypertension or hypercholesterolemia at baseline, total energy intake (quintiles), postmenopausal status, postmenopausal hormone use, multivitamin and vitamin E supplement use
McCullough, 2000* [26]	Healthy eating index-f	Relative risk	Quintile 5	Quintile 1	Age (5-y categories), body mass index (quintiles), smoking (never, past, 1–14 cigarettes/d, 15–24 cigarettes/d, ≥25 cigarettes/d), alcohol intake (7 categories), physical activity (6 categories), total energy intake (quintiles), time period, multivitamin use, vitamin E use, and diagnosis of hypercholesterolemia and hypertension at baseline
McCullough, 2002* [14]	Recommended Food Score	Relative risk	Quintile 5	Quintile 1	Age (5-y categories), smoking (never, past, 1–14 cigarettes/d, 15–24 cigarettes/d, >25 cigarettes/d), time period, body mass index (quintiles), physical activity (6 categories of metabolic equivalents), total energy intake (quintiles), history of hypertension or hypercholesterolemia at baseline, vitamin E and multivitamin supplement, and for women, postmenopausal hormone use
Fung, 2001* [27]	Prudent pattern/ Western pattern	Relative risk	Quintiles 4,5/1	Quintiles 1/4,5	Age, period, smoking, body mass index, hormone replacement therapy, aspirin use, caloric intake, family history, history of hypertension, multivitamin and vitamin E use, and physical activity
Diehr, 2003* [28]	Diet quality	Years of life in 10 yr, CVD incidence	Healthy diet	Unhealthy diet (high fat, low fiber, low carbohydrate, high protein, high calorie)	Demographics, health, behaviors, and baseline health variables

Then, maybe most importantly, several of these studies did not state the specific carb intakes so for those we have no idea if people are eating an actual low carb diet or just eating “less carb than most people.” This is very important, because many of the benefits of low carb and keto arise only when you sufficiently restrict carbohydrate and get your insulin low enough.[R]

The ~~ten studies~~ nine studies[R3,R4,R5,R12,R13,R14,R15,R16,R17] that *did* clearly state carb intake showed that the diets were not actually low carb or keto at all. Low carb typically allows a maximum of 60g of carbohydrate or around 10% of your daily calories from carbs, and the ketogenic diet only allows 5% of your calories from carbs. Two studies only went as low as 25%[R3,R14] of calories from carbs and the remaining ~~seven~~ six didn’t even get below 30% - this a three times higher carb intake than what is actually considered “low carb,” and 6 times higher than ketogenic.

So this data being used to criticize ketogenic diets is not actually data on ketogenic diets or even low carb diets.

On the other hand here’s a study looking at 83 obese people on a proper

ketogenic diet for 24 weeks. By the way, the diet was administered to the patients, data was not based on questionnaires. It concluded: "*The present study shows the beneficial effects of a long-term ketogenic diet. It significantly reduced the body weight and body mass index of the patients. Furthermore, it decreased the level of triglycerides, LDL cholesterol and blood glucose, and increased the level of HDL cholesterol. Administering a ketogenic diet for a relatively longer period of time did not produce any significant side effects in the patients.*"

And there's plenty more studies that showcase the beneficial effects of a ketogenic diet - I've put links to a couple dozen of them in the description. [See below]

So as we've seen, based on how diabetes develops, a low carb or ketogenic diet would be the most logical approach to treating it, yet people are constantly advised against it. Another approach this video unfortunately didn't get to touch on is fasting- this is an excellent way restore insulin sensitivity as mentioned in Jason Fung's book the diabetes code. This book is a must read for anyone concerned about diabetes.

You may still have several concerns regarding low carb, ketogenic diets and you may just now be thinking about concerns you have about fasting. As I've covered many of these points in previous videos, take a look at the description for links to relevant videos and more information.

1. The effects of the ketogenic diet on behavior and cognition [Neuroprotective]
2. Novel ketone diet enhances physical and cognitive performance
3. Diet-Induced Ketosis Improves Cognitive Performance in Aged Rats
4. Dietary ketosis enhances memory in mild cognitive impairment
5. "*In addition, due to its neuroprotective capacity, the KD may also hold potential benefit for the treatment of other neurological or neurodegenerative disorders.*"
6. Ketogenic diet improves the spatial memory impairment…
7. A ketogenic amino acid rich diet benefits mitochondrial homeostasis…
8. The antidepressant properties of the ketogenic diet
9. "*The adult KD offspring exhibit reduced susceptibility to anxiety and depression…*"

10.*"...ketogenic diets reduce hunger and lower food intake…"*
11. Improvement in age-related cognitive functions and life expectancy by ketogenic diets
12. Effects of Ketogenic Diets on Cardiovascular Risk Factors: Evidence from Animal and Human Studies "*Based on the available literature, KD may be associated with some improvements in some cardiovascular risk factors, such as obesity, type 2 diabetes and HDL cholesterol levels…"*
13. Efficacy of ketogenic diet on body composition during resistance training in trained men: a randomized controlled trial "*Our results suggest that a KD might be an alternative dietary approach to decrease fat mass and visceral adipose tissue without decreasing lean body mass*"
14. Beneficial effects of ketogenic diet in obese diabetic subjects. "*This study shows the beneficial effects of ketogenic diet in obese diabetic subjects following its long-term administration. Furthermore, it demonstrates that in addition to its therapeutic value, low carbohydrate diet is safe to use for a longer period of time in obese diabetic subjects.*"
15. Ketogenic diet in cancer therapy "*Based on the results of rigorous preclinical and clinical studies performed thus far, the KD would appear to be a promising and powerful option for adjuvant therapy for a range of cancers. Cancer-specific recommendations await the findings of randomized controlled clinical trials."*
16. The Ketogenic Diet: Uses in Epilepsy and Other Neurologic Illnesses
17. Ketogenic diet in endocrine disorders: Current perspectives "*There is clinical evidence to support the use of KD in diabetes, obesity, and endocrine disorders. ... Such diets may positively influence hormonal balance and endocrinological disorders, but future studies are required to assess the long-term effects on health and reversing of diabetic complications in humans.*"
18. D-beta-hydroxybutyrate rescues mitochondrial respiration and mitigates features of Parkinson disease.
19. D-beta-hydroxybutyrate protects neurons in models of Alzheimer's and Parkinson's disease.
20. A ketogenic diet reduces amyloid beta 40 and 42 in a mouse model of Alzheimer's disease.
21. Mitochondrial biogenesis in the anticonvulsant mechanism of the ketogenic diet.
22. Ketones inhibit mitochondrial production of reactive oxygen species production following glutamate excitotoxicity by increasing NADH oxidation
23. Ketone bodies are protective against oxidative stress in neocortical neurons.
24. Application of a ketogenic diet in children with autistic behavior: pilot study. "*Significant improvement (> 12 units of the Childhood Autism Rating Scale) was recorded in two patients (pre-Scale: 35.00 +/- 1.41[mean +/- SD]), average improvement (> 8-12 units) in eight patients (pre-Scale: 41.88 +/- 3.14[mean +/- SD]), and minor improvement (2-8 units) in eight patients (pre-Scale: 45.25 +/- 2.76 [mean +/- SD]). Although these data are very preliminary, there is some evidence that the ketogenic diet may be used in autistic behavior as an additional or alternative therapy.*"
25. The anti-depressant properties of the ketogenic diet. Biol Psychiatry. "*The rats on the ketogenic diet spent less time immobile, suggesting that rats on the ketogenic diet, like rats treated with antidepressants, are less likely to exhibit "behavioral despair." ...It is concluded that the ketogenic diet may have antidepressant properties.*"
26. Diet-induced ketosis increases capillary density without altered blood flow in rat brain. "*The increase in extraction fraction and capillary density with increased plasma ketone bodies indicates a significant flux of substrates available for brain energy metabolism.*
27. Growth of human gastric cancer cells in nude mice is delayed by a ketogenic diet supplemented with omega-3 fatty acids and medium-chain triglycerides "*Application of an unrestricted ketogenic*

diet enriched with omega-3 fatty acids and MCT delayed tumour growth in a mouse xenograft model."

28. Stroke outcome in the ketogenic state - a systematic review of the animal data "*To restate the major findings from our analyses, we found beneficial effects on pathologic and functional outcomes of dietary intervention, or exogenous ketone administration, either prior to or following experimental stroke… Neuropathologies, such as a stroke, cause a mismatch between energy demand and supply: blood flow goes awry, oxygen levels fall, and mitochondria malfunction. A period of fasting results in a short-term ketosis, and increased reliance on ketone bodies appears to be a form of cerebral metabolic adaptation (Manzanero et al., 2011). Ketone metabolism is enzymatically simpler and more efficient than glucose or pyruvate metabolism…*"

29. β-hydroxybutyrate: Much more than a metabolite "*βOHB is already known to induce resistance to oxidative stress via HDAC inhibition, and other HDAC inhibitors regulate gluconeogenesis. The unique effects of βOHB may help explain the therapeutic benefit of low-carbohydrate and ketogenic diets.*"

30. Potential Synergies of β-Hydroxybutyrate and Butyrate on the Modulation of Metabolism, Inflammation, Cognition, and General Health "*The studies presented here demonstrate safety and efficacy of the diet including the scientific support and rationale for the administration of exogenous ketone bodies and ketone sources as a complement to the restrictive dietary protocol or as an alternative to the diet."*

31. A very low carbohydrate ketogenic diet improves glucose tolerance in ob/ob mice independently of weight loss

32. Effects of beta-hydroxybutyrate on cognition in memory-impaired adults. "*Higher ketone values were associated with greater improvement in paragraph recall with MCT treatment relative to placebo across all subjects*"

33. The therapeutic implications of ketone bodies: the effects of ketone bodies in pathological conditions: ketosis, ketogenic diet, redox states, insulin resistance, and mitochondrial metabolism.

34. β-Hydroxybutyrate Elicits Favorable Mitochondrial Changes in Skeletal Muscle

35. Fueling Performance: Ketones Enter the Mix.

36. D-β-Hydroxybutyrate rescues mitochondrial respiration and mitigates features of Parkinson disease