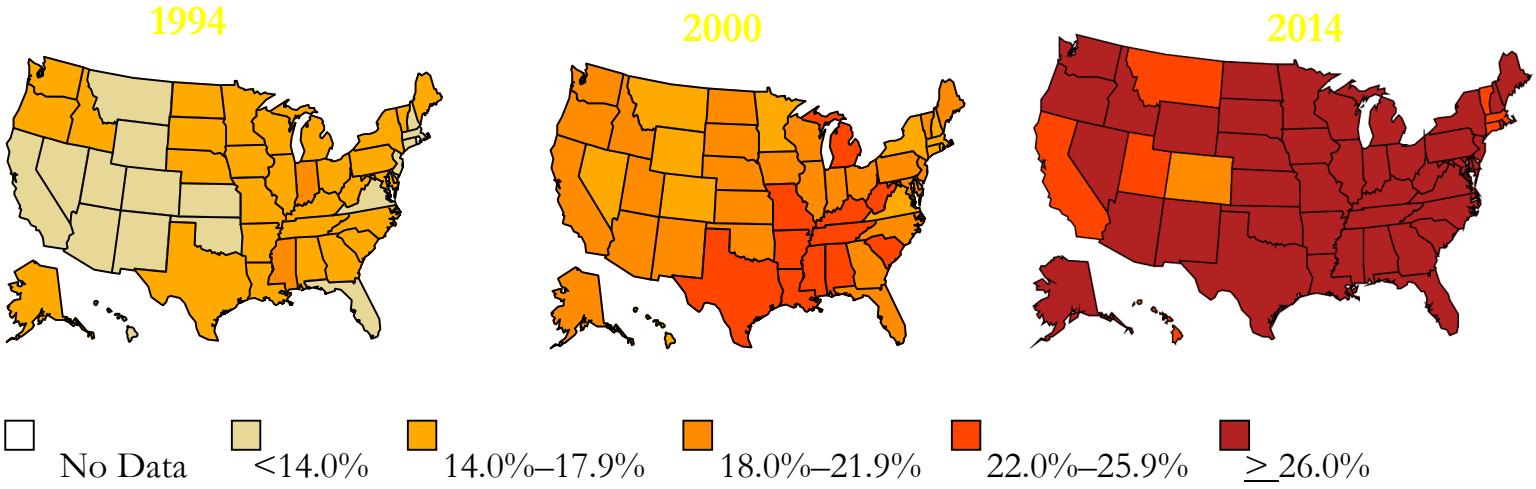
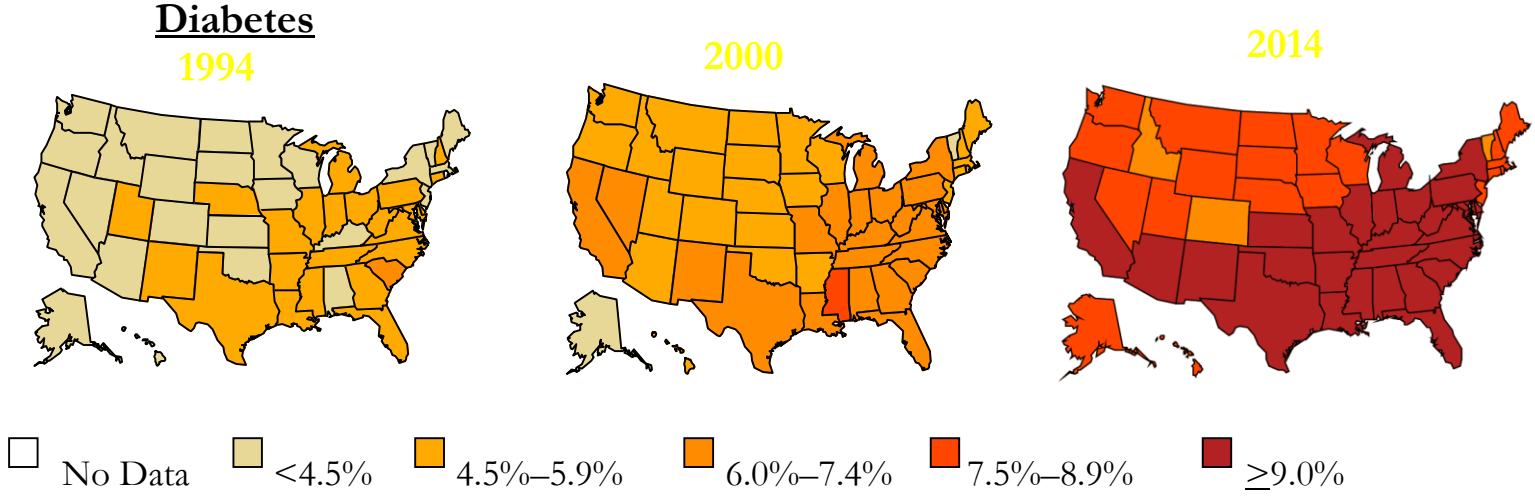
# Age-adjusted Prevalence of Obesity and Diagnosed Diabetes Among US Adults

## Obesity (BMI ≥30 kg/m$^2$)




## Diabetes




CDC's Division of Diabetes Translation. United States Surveillance System available at http://www.cdc.gov/diabetes/data

# What does type II diabetes mellitus mean?

It has been presumed that the symptomology of the disease (type II diabetes mellitus) was first recorded in 1550 BC by the Egyptians (Ahmed, 2002, p. 373). Araetus of Cappodocia (81-138 AD) "used the Greek word diabetes, literally meaning to run through or siphon" when describing the excessive flow of urine within individuals inflicted with the disease (Ahmed, 2002, p. 373). According to Ahmed (2002) Avicenna, an Arab physician (960-1037 AD) "emphasized the idea of sweet taste of urine" when describing his patients with diabetes (p. 373). Diabetes while a disease that has been with us for some time was lost to science and rediscovered in 1675 when Thomas Willis a physicians in London rediscovered the "sweetness of urine in diabetic patient's" with his new discovery he added the Latin term "mellitus, literally meaning honey sweet to the Greek diabetes to describe the disease" (Ahmed, 2002, pg. 374). Type II Diabetes mellitus, or as commonly referred to type II diabetes, literally means sweet urine which is due to the kidneys excretion of sugar from the blood.

# How many people are affected by type II diabetes and pre-diabetes?

According to the Center for Disease Control and Prevention (CDC), (2015a) "29.1 million people have diabetes, that's about 1 out of every 11 people" and those living with prediabetes is estimated to be approximately "86 million American adults-more than 1 out of 3-have prediabetes" (CDC, 2016). According to the UnitedHealth Group "based upon current trends, 52 percent of the U.S. adult population could have prediabetes or diabetes by 2020" resulting in approximately 3.4 trillion dollar spent on diabetes management (p. 4).

# Why is diabetes a problem?

Diabetes increases the risk of cardiovascular disease, which increases the risk of heart attack and stroke and could potentially lead to amputation of legs or feet due to multiple issues such as nerve damage. Diabetes also increases the risk of eye complications like blindness and other vision problems. As well as increasing the risk of kidney disease, thus leading to kidney damage potentially leading to kidney failure and dialysis. (CDC, 2015b).

# How is an individual diagnosed with prediabetes or diabetes?

Currently there are a few different ways diabetes is diagnosed in individuals. According to the American Diabetes Association (ADA) (2014), there are 3 ways to diagnose diabetes.

A1C Levels

One way to be diagnosed with prediabetes or diabetes is by testing A1C levels within the blood. This test can be performed at any time, no fasting or ingestion or drinks is needed prior to administering this test (ADA, 2014). According to Mayo Clinic (2016) "the A1C test goes by many names, including hemoglobin, glycosylated hemoglobin, hemoglobin A1C and HbA1c" and the results of the test "reflect your blood sugar level for the past two to three months". When a doctor reviews your A1C levels with you, they use the chart below to make a clinical diagnosis. A1C levels between 5.7 and 6.4% are deemed pre-diabetic or a diagnosis of prediabetes is made while a level greater than 6.5% means a clinical diagnosis of diabetes is made. Depending upon the results of your initial test and your symptomology a diagnosis may be made. However, in some cases a retest may be needed. The graph below is reconstructed based upon the ADA (2014) graph of A1C levels.

| Results | A1C |
|---|---|
| Normal | Less than 5.7% |
| Prediabetes | 5.7% to 6.4% |
| Diabetes | 6.5% or higher |
| Graph reconstructed based on ADA (2014) graph for diagnosing diabetes in individuals with increased A1C levels | |

Fasting Plasma Glucose

The second way of determining diabetes, according to the ADA (2014), is based upon Fasting Plasma Glucose (FPG) levels within the blood. To ensure the effectiveness and reliability of these test results individuals need to fast 8 hours prior to the test most physicians order the tests to be completed early morning so the patient can eat prior to starting their work day (ADA, 2014). When a doctor reviews your FPG levels with you they use the chart below to make a clinical diagnosis. FPG levels between 100mg/dl and 125mg/dl are deemed pre-diabetic or a diagnosis of prediabetes is made while a measure of FPG greater than 126 mg/dl means a clinical diagnosis of diabetes is made. Depending upon the results of your initial test and your symptomology a diagnosis may be made. However, in some cases a retest may be needed. The graph below is reconstructed based upon the ADA (2014) graph of FPG lab values.

| Results | Fasting Plasma Glucose (FPG) |
|---|---|
| Normal | Less than 100 mg/dl |
| Prediabetes | 100 mg/dl to 125 mg/dl |
| Diabetes | 126 mg/dl or higher |
| Graph reconstructed based on ADA (2014) graph for diagnosing diabetes and prediabetes in individuals using FPG results | |

Oral Glucose Tolerance Test

The third way of determining diabetes, according to the ADA (2014), is based upon an oral glucose tolerance test (OGTT). This is a longer test, lasting 2 hours, in which a patient has blood glucose levels tested prior to and 2 hours after the consumption of a sweetened beverage. The test informs the physician how the patient's body handles glucose over the course of 2 hours' time (ADA, 2014). When a doctor reviews your OGTT results with you they use the chart below to make a clinical diagnosis. OGTT levels between 140mg/dl and 199mg/dl are deemed pre-diabetic or a diagnosis of prediabetes is made

while a measure of OGTT greater than 200 mg/dl means a clinical diagnosis of diabetes. Depending upon the results of your initial test and your symptomology a diagnosis may be made. However, in some cases a retest may be needed. The graph below is reconstructed based upon the ADA (2014) graph of OGTT lab values.

| Results | Oral Glucose Tolerance Test (OGTT) |
|---|---|
| Normal | Less than 140 mg/dl |
| Prediabetes | 140 mg/dl to 199 mg/dl |
| Diabetes | 200 mg/dl or higher |
| Graph reconstructed based on ADA (2014) graph for diagnosing diabetes and prediabetes using the OGTT results. | |

# What is prediabetes?

The CDC (2015c) and the ADA (2014) state that prediabetes is when individuals have elevated glucose levels but are not within the ranges of those with diabetes. Therefore these individuals are classified as having prediabetes. These ranges and cut off values are critical in understanding how diabetes is diagnosed.

Each test has a specific range in which blood sugar levels need to fall between to issue a diagnosis. For prediabetes to be diagnosed based upon an A1C levels the results need to be between 5.7 and 6.4%; Fasting Plasma Glucose 100 mg/dl to 125 mg/dl; and Oral Glucose Tolerance Test 140mg/dl to 199mg/dl.

# What can I do to prevent prediabetes from turning into diabetes?

According to the CDC (2015c) “people can delay and possibly prevent the disease by losing a small amount of weight (5 to 7 percent of total body weight) through 30 minutes of physical activity 5 days a week and healthier eating”. As you will learn, prevention is a necessary component of this disease and while exercise is an important step in controlling diabetes the most critical step in preventing type II diabetes is the types of food we consume on a daily basis.

# What is type II diabetes?

Type II diabetes is a disease characterized by increased glucose levels within the blood which are above normal, called hyperglycemia (CDC, 2015b; ADA, 2016).

> According to the CDC (2015b):
> Most of the food we eat is turned into glucose, or sugar, for our bodies to use for energy. The

pancreas an organ that lies near the stomach, makes a hormone called insulin to help glucose get into the cells of our bodies. When you have diabetes, your body either doesn't make enough insulin or can't use its own insulin as well as it should. This causes sugar to build up in your blood.

It is important to understand that our bodies, our muscles, our brain, our organs all run on glucose.

## Is there a treatment for type II diabetes?

Currently, according to the CDC (2015b) "Healthy eating, physical activity, and blood glucose testing are basic therapies for type 2 Diabetes" and medications may also be required to keep blood glucose levels under control. According to a position paper by the ADA on the standards of care for patients with diabetes mellitus (2002), "the ideal goal of treatment is the normalization of blood glucose and A1C values" (p. 14).

## Isn't type II diabetes called adult-onset diabetes?

Yes, previously diabetes was considered a disease that only middle aged and older adults were diagnosed with. It recent years the prevalence of the disease has been increasing in children and young adults. According to Hannon, Rao, & Arslanian (2005) in recent years "overweight and obesity is the most important risk factor for the development of type 2 diabetes mellitus in youth" (p. 473).

## Can children get type II diabetes?

In 2015, the European Association for the study of Diabetes in Stockholm, Sweden reported on a 3-year-old American Hispanic girl who was diagnosed with Type II Diabetes. The family had a history of obesity, but not diabetes. The case report states that the family had "poor family nutritional habits with uncontrolled counting of calories and fat" (p. 152). With proper lifestyle changes, nutrition education and exercise routine, along with medication the child's condition reversed. Within 6 months of starting this intervention the child's medication was reduced and eventually no medication was needed. Currently, this is the youngest reported case of type II diabetes ever recorded according to Yafi, M., Collins, K. (2015).

## Can being overweight increase my risk of developing diabetes?

Yes, according to the CDC (2015c) "being overweight or obese is a leading risk factor for type II diabetes. Being overweight can keep your body from making and using insulin properly, and can also cause high blood pressure."

## Can diabetes be prevented?

The CDC, (2016) states “A number of studies have shown that regular physical activity can significantly reduce the risk of developing type 2 diabetes”.

## Is diabetes caused by poor diet and lack of exercise?

Yes and no. Diet, as the research has consistently shown, is the main contributing factor to the prevention, progression, and reversal of diabetes. According to the CDC (2015c), individuals at high risk for developing diabetes can delay or prevent the onset of the diseases by losing weight, exercising (approximately 30 minutes a day, 5 days a week) and eating a healthier diet.

Multiple studies have shown that providing lifestyle advice to individuals, such as eating a healthier diet, consisting of lower fat foods, less oil, less meat, less dairy, high fiber, more fruits, more vegetables and daily physical activity along with losing weight reduces the risk of developing type II diabetes (Pan, 1997; Tuomilehto, 2001; Diabetes Prevention Program, 2002; Salas-Salvadó, 2011).

## What causes diabetes?

According to the CDC (2016), in regards to the cause of type II diabetes the “exact mechanism” is unknown. As we will see, some researchers feel they have found the “cause” or “exact mechanism” that causes type II diabetes. However, we need to keep in perspective that there are multiple contributing factors to the disease but researchers consistently reach the same conclusion according to John McDougall, MD (2012), a board certified internist, physician, and low fat plant-based advocate who promotes a starch based diet and has written many books on the topic of diabetes, has been “curing” patients with type II diabetes since 1968, by treating the cause of the disease, which he relates to the rich western diet stating that “Fat paralyzes insulin, making diabetes worse” he believes and as research shows that the fat we eat, especially the fat found in animal products, is the main contributor to the development of diabetes as well as many other chronic western diseases. But can these claims be substantiated and what does the ADA have to say about the consumption of fat within the diet? In 2014 the ADA updated their standards of care for individuals with diabetes and stated “dietary strategies including reduced calories and reduced intake of dietary fat, can reduce the risk for developing diabetes” (p. 529). But does this statement “reduced intake of dietary fat” go far enough? This statement is ambiguous and unfortunately does not provide ample information to those wanting to improve their health.

# The cause of Type II Diabetes?

A consensus statement written by the ADA in 2002 stated that in regards to diabetes "the evolution from normal to impaired glucose tolerance is associated with a worsening of insulin resistance" (p. 384). The paper further explains that "it is well recognized that resistance to insulin-stimulated glucose uptake is a characteristic finding in patients with type 2 diabetes and impaired glucose tolerance" (p. 384). While it has also been noted that the "the failure of the Beta-cell to continue to hypersecrete insulin underlies the transition from insulin resistance (with compensatory hyperinsulinemia and normoglycemia) to clinical diabetes (with overt fasting hyperglycemia and increased hepatic glucose production)" (p.384). According to Hannon, Rao, and Arslanian (2002), "insulin resistance which develops as a result of both genetic and environment factors, is strongly associated with obesity. Moreover, insulin resistance is now widely believed to be the first step in the development of T2DM, cardiovascular disease and other conditions" (p. 473-474).

According to Scrauwen-Hinderling, V. B., Hesselink, M K. C., Schrauwen, P., & Kooi, M. E.,. (2006) "physically inactive humans consuming a high-energy, high-fat diet, a positive energy and fat balance may occur chronically, resulting in fat accumulation in adipose tissue and probably also in skeletal muscle" (p. 362). Kraegen & Cooney (2008) stated the "acute exposure to fatty acids causes insulin resistance in muscle, and excess dietary lipid and obesity are strongly associated with muscle insulin resistance" (abstract, para. 1). In other words, the ingestion of fat impairs the muscles cells ability to effectively uptake insulin. Which leads to excess sugar in the blood stream. Individuals who are overweight or obese (having excess adipose tissue, or fat) also tend to have an increased risk of diabetes. The researchers concluded "whether the initial events leading to muscle insulin resistance are direct effects of fatty acids in muscle or are secondary to lipid accumulation in adipose tissue or liver remains to be clarified" (Kraegen & Cooney, 2008, abstract, para. 3).

According to Scrauwen-Hinderling, et al., (2006), "fat can be stored not only in adipose tissue but in other tissues such as skeletal muscle" (P. 357). Fat droplets contained within the muscle cells, intramyocellular lipids (IMCL), are "valuable energy stores during prolonged exercise, which, however, in the absence of regular physical activity and with overconsumption of fat, can have detrimental effects on muscular insulin sensitivity" or muscle insulin resistance (p. 357). This excess consumption of fat leads to an accumulation of fat droplets in all skeletal muscle within the body and has been given the name intramyocellular lipids. Intra = Inside, myo = muscle, cellular = cell, lipids = fat. Fat within the muscle cell. And according to Scrauwen-Hinderling et al. (2006):

> Elevation of plasma fatty acid levels or dietary fat content also increases IMCL content, suggesting that skeletal muscle also stores fat simply if the availability of fatty acids is high under these conditions, the uptake into skeletal muscle may have negative consequences on insulin sensitivity (p. 357).

Rachek, L, I., (2013), argue that "Lipotoxicity, characterized by the accumulation of ectopic lipids in skeletal muscle, is a major factor in the etiologies of insulin resistance and type 2 diabetes" (p. 269). Lipo = Fat, Toxicity = toxic; Ectopic = in an abnormal place; Etiologies = the cause of a disease. The cause of disease is fat where it doesn't belong, which increases the risk of insulin resistance and can eventual lead to the development of type 2 diabetes. Rachek, L, I., (2013) argues that "the development of skeletal muscle insulin resistance can be independent of a family history of type 2 diabetes" (p. 269).

This means family history/genetics may not be a determining factor in the development of insulin resistance in some individuals. Unger, R. H., Clark, G. O., Scherer, P. E., and Orci, L. (2010) further clarified this statement "although genes determine susceptibility to environmental factors, the epidemic is clearly due to increased consumption of calorie-dense, highly lipogenic foods, coupled with a marked decrease in physical exertion resulting from modern technologies" (Para. 1). Lipo = fat, genic = creation of. What the researcher is saying is that our sedentary lifestyles along with the consumption of high fats foods results in the progression of diabetes.

According to Estadella, D., da Penha Oller do Nascimento, C. M., Oyaa, L. M., Ribeiro, E. B., Damaso, A. R., et al. (2013) in their scientific journal article titled, Lipotixicity: effects of dietary saturated and transfatty acids, "the ingestion of excessive amounts of saturated fatty acids and trans fatty acids is considered to be a risk factor for cardiovascular diseases, insulin resistance, dyslipidemia, and obesity" (P. 1). This corroborates research conducted by Lichenstein & schwab, (2000) who found that "saturated fat, relative to monounsaturated and polyunsaturated fat, appears to be more deleterious with respect to fat-induced insulin sensitivity" (p. 227). While fat is vital macronutrient within the human diet, the types of fat and amount of fat consumed have an a impact on our health but those fats that have the greatest impact, saturated fats and trans fats, are predominantly found in meat and dairy as well as refined oils according to Estadella et al. (2013, p. 1).

In an effort to improve insulin sensitivity in all individuals, especially those suffering from prediabetes and diabetes, it would be advantageous to recommend reducing, or eliminating, the consumption of high fat foods from the diet. The high fat foods include meat and dairy products as well as refined oil and processed foods containing oils. If the consumption of high fat foods can lead to insulin sensitivity removing them from the diet should be a prudent goal in the reversal of insulin sensitivity. A similar goal of reducing the intake of trans fats was recently implemented by the FDA, while not for the reason of diabetes prevention, in 2013 the FDA proposed a ban removing partially hydrogenated oils, which are a synthetic man-made form of trans fat, from the food supply due to the link of increasing the risk of heart disease. Susan Mayne, Ph. D., Director of FDA's Center for Food Safety and Applied Nutrition, stated "Trans fat wouldn't be completely gone… because it also occurs naturally in meat and dairy products" (para. 6). She also went on to say "It is also present at very low levels in other edible oils, where it is unavoidably produced during the manufacturing process" (para. 6).

Keep in mind that saturated and trans fats are synonymous with meat and dairy products, however, it should be noted that high levels of saturated fat and trace amounts of trans fats can be found in oils and especially palm oil, palm kernel oil, coconut and coconut oil. To learn more about where saturated fats and trans fats are found and how they increase your risk of heart disease click here.

While researchers have shown that fat, especially saturated and trans fat, increase muscle insulin sensitivity research has also shown that excessive free fatty acids within the blood have can be detrimental to pancreatic beta-cells and has been shown cause death to the beta-cells through lipoapoptosis (Estadella, 2013, p. 2). Lipo = fat, apoptosis= cell death. Cell death by fat. This coincides with kraegens (2008) research stating that beta-cell failure can occur decades before diabetes complications occur in patients. The high consumption of animal fat, predominantly saturated and trans fat, decrease beta cell function in individuals regardless of complications with diabetes thus the consumption of high fat animal foods have a detrimental effect on our beta cells. Lichtenstein & Schwab (2000), argue that "in humans, high-fat diets, independent of fatty acid profile, have been reported to

result in decreased insulin sensitivity" (p. 227). And Unger et al., (2010) research claims that "fatty acid derivatives can interfere with the function of the cell and ultimately lead to its demise through lipoapoptosis, the consequences of which are gradual organ failure" (para. 1). The affected organs that contribute to metabolic disorders are skeletal muscle, liver, heart and pancreas (Unger, 2010, para. 1).

To improve pancreatic beta-cell function in humans a change in diet should be recommended to those suffering from prediabetes as well as type II diabetes mellitus and all other patients consuming a high fat diet due to its negative effects on both pancreatic beta-cell function and beta-cell production. The only way fat enters our body is through our mouth, unless fat is injected intravenously which is typically only done in clinical research trials. The diet that should be recommended to patients is a low fat diet of whole plant-based foods, which is naturally Low in fat. The foods recommended to all patients would consist of fruits, vegetables, whole grains, beans, legumes, and a few nuts and seeds and a vitamin B-12 supplement; no meat, dairy or oils should be encouraged due to these products containing large amounts of saturated and trans-saturated fats.

According to the CDC (2015b) "most of the food we eat is turned into glucose, or sugar, for our bodies to use for energy". The body's natural fuel source is glucose (sugar) not fat, especially saturated and trans fat. The body does not run on sucrose (table sugar) which is found in processed foods such as soda and candy. With this general understanding we can begin to understand the development of diabetes. Glucose (sugar) cannot enter our muscle cells directly it needs a key (insulin) to get in. After eating food, blood glucose (sugar) levels rise and beta cells signal the release of insulin. Insulin attaches to glucose (sugar) within the blood and acts like a key allowing glucose (sugar) into the muscle cell. We can think of insulin as the key. The cell (door) is locked until insulin (key) opens it allowing glucose (sugar) to be absorbed. But in individuals with increased levels of circulating blood glucose (sugar), fat droplets (lipids) have plugged the cell (door) where glucose (sugar) is supposed to enter via insulin (key).

In the following example, picture in your mind that, you are sugar and as your circle around the house it symbolizes sugar circulating in the blood stream, the key you hold in your hand represents insulin, and the multiple doors to your home represent entry into the cell while the gum in the lock represents fat. The best way to think of this is as if you are looking at the lock to the front door of your home and someone has stuck gum in it. Your key won't fit so you can't get in. You circle around the house to the side garage door and find gum in that lock as well, again, the key won't fit. So you circle around to the backdoor to find gum in that lock as well and again, your key does not fit. But the fix, as you can imagine, is relatively easy, just take the gum out of the lock, then the key will fit in the lock and you can get in the house. The same holds true for the glucose circulating in your body, the sugar can't access the cell until the fat has been removed from cell. Managing blood glucose levels with drugs and insulin does not address the underlying issue, fat in the muscle cell, or as in this example, the gum in the lock. This continues to occur until the fat is removed from the lock. Without addressing the cause, we cannot address the issue. In the case of insulin resistance prediabetes/diabetes there are two ways to make the key fit.

1. As the research has pointed out physical activity can help manage Intramyocellular lipids by removing fat contained with the muscle cells through physical exertion. While this is a valiant attempt at addressing the problem, as soon as the exercise regimen stops, if no dietary modifications are made, the muscle cells revert to their previous state and insulin resistance returns. While we recommend physical activity, light walking or riding a bike, this attempt does nothing to stop further damage from being done to the body's internal organs if the diet continues to remain the same. This can be seen as a temporary

fix. This attempt does nothing to address the cause of the problem, it does however, address the symptoms, which is high blood sugar.

2. The second way to make the key fit, and ensure it fits continuously, is to remove high fat foods from the diet. As shown this is the initial cause of the problem. Excess fat in the diet, primarily saturated and trans fat found predominantly in meat and dairy products as well as oils, but high levels of saturated fat can also be found in plant based-foods such as some nuts and oils like palm oil, palm kernel oil, coconut and coconut oil. Removing oils from the diet and keeping fats to a minimum (preferably less than 10 to 15%, but at least under 20%, of calories from fat) diminishes, stops, slows, or reverses the cascading and damaging effect within the body. If fat consumption is kept to a minimum, fat cannot plug up the cells which lead to insulin resistance. To learn how to determine the % of calories from fat on a label click here.

Below is a great clip from VegSource.com in which Jane Esselstyn explains intramyocellular lipids and the role in the development of type II diabetes in individuals. She provides a visual demonstration of the same concept I referred to above in a different way.

https://www.youtube.com/watch?v=MonwPt4LIN0

Taylor (2013) argues that "people who are relatively insulin resistant in muscle-and who therefore have a raised plasma insulin level-are especially likely to accumulate fat in the liver" and as fat builds up in our organs their ability to function properly diminishes (p. 269). In the case of the liver, according to Medline Plus, "Nonalcoholic fatty liver disease is the buildup of fat in the liver that is NOT caused by drinking too much alcohol" and risk factors include insulin resistance (Medline Plus, 2016). Based upon this information if Nonalcoholic fatty liver disease is not caused by drinking alcohol it must be cause by something in our diets and Almeda-Valdes, Cuevas-Ramos, D., and Aquilar-Salinas, C. A. (2009) state lipotoxicity increases the risk of both nonalcoholic fatty liver disease and metabolic syndrome.

> Estedalla et al. further states that the "composition of the diet exerts an important role in the development of non-alcoholic fatty liver disease and its treatment and that it is essential to consider excessive saturated fatty acid intake as a critical risk factor for development of non-alcoholic fatty liver disease" (p. 6).

The consumption of excess fat within the diet, for a few days or years, leads to fat storage either subcutaneously, viscerally or in the liver. Due to this process excess carbohydrate can no longer enter the muscle cells due to insulin resistance and is therefore turned into fat by a process of de novo lipogenesis (Taylor, 2013 p. 268-269). De novo = to begin new, lipo = fat, genesis = creation. De novo lipogenesis means the creation of new fat. This means glucose (sugar) is only turned into fat by our body, in the presence of excess fat within the diet and on the body. The ingestion of excess fat causes de novo lipogenesis from glucose but "this process only happens in the liver in humans, and triglycerides synthesized in situ is particularly likely to be stored in hepatocytes rather than exported for storage subcutaneously" (Taylor, 2013, p. 268). As fat continues to store within the liver "this will cause relative resistance to insulin suppression of hepatic glucose production" which eventually results in a cycle of hyperinsulinaemia (P. 269).

According to Taylor (2012):

> A vicious cycle of hyperinsulinaemia and increased liver fat will become established. Fatty liver leads to increased export of VLDL triacylglycerol, which will increased fat delivery to the islets, with excess fatty acid availability impairing the acute insulin secretion in response to ingested food. Eventually the fatty acid and glucose inhibitory effects on the islets will reach a trigger level, precipitating clinical diabetes (p. 269).

Taylor (2012) concluded that:

> These self-reinforcing cycles between liver and pancreas eventually cause metabolic inhibition of insulin secretion after meals and onset of hyperglycemia. It is now clear that type 2 diabetes is a reversible condition of intra-organ fat excess to which some people are more susceptible than others (p. 267).

A representative model of "The twin cycles of type 2 diabetes Mellitus"

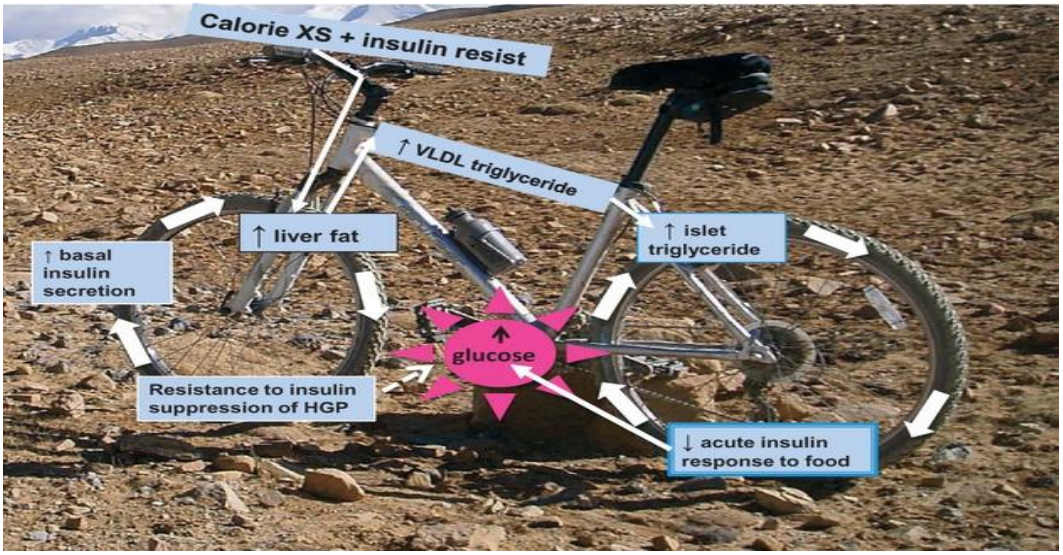



Image and text below retrieved 9/14/2016 from Taylor, R. 2013: http://onlinelibrary.wiley.com/doi/10.1111/dme.12039/epdf

This bicycle is controlled by handlebars representing chronic, excess (XS) calorie intake in the presence of muscle insulin resistance. Raised plasma insulin levels will expedite chronic excess calorie storage from carbohydrate via *de novo* lipogenesis, and hence promote liver fat storage. This will cause the liver to become relatively resistant to insulin and a small increase in plasma glucose will occur. In turn, insulin secretion will increase to control plasma glucose down. The further increased insulin levels will bring about a self-reinforcing cycle. Excess fat will result in increased export of VLDL triglyceride from the liver, uptake by islets and inhibition of meal insulin secretion. At a personal threshold, the pancreas fat becomes too great a load and plasma glucose levels will then rise relatively rapidly. HGP, hepatic glucose production.

# What foods are associated with increases in insulin resistance?

The foods associated with insulin resistance are low carbohydrate, high protein, high fat, low fiber foods. But this means nothing to the general public. These confusing terms are used by physicians,

researcher, and academics and tend to confuse people. These obscure phrases may cause individuals to give up their quest to be healthy because they cannot understand what these foods are. Most researchers and physicians when speaking about food this way are speaking about specific attributes contained within the food: low carbohydrate, high protein, high fat, low fiber. These are all confusing ways of saying meat (red meat, fish, pork, eggs, and poultry), dairy, as well as oils including palm oil, palm kernel oil, coconut and coconut oil.

When you hear low carbohydrate, high protein, think of meat, dairy, nuts, seeds, and oils. As Estadella et al. (2013, p. 1) and Mayne, S. (2013, para. 6) stated saturated fat and trans fats are found in all meat and dairy products as well as oils. This includes all types of meat and to elaborate further this means muscle meats of an animal as well as eggs. Dairy products consist of: milk, yogurt, cheese, cottage cheese, cream cheese, yogurt, sour cream. While these are not exhaustive lists, these foods contain both saturated fats and trans fats which have not only been associated with the progression of diabetes but also heart disease, dyslipidemia and obesity (Estadella, 2013, p. 1).

A low carbohydrate, high protein, high fat, low fiber diet has been popularized in many books and goes by names like Atkins, Paleo, Grain brain, and Wheat belly. These diets are easy to follow and very convenient for most people. These can also be considered fast food diets or window diets, where you pull up to a fast food window, order a burger, through away the bun, scrape of or leave the pickle, onions, and lettuce but scrape off the ketchup and mustard and you have your meal. Simple, easy! Sadly, long term results of low carbohydrate, high protein, high fat, low fiber diets show us that the potential short term benefits of the diet have rather disastrous long term health implications such as: increased risk of heart disease, stroke, and diabetes.

Recently two studies have been published showing the negative effects of high animal protein diets. In 2012, “Low carbohydrate-high protein diet and incidence of cardiovascular disease in Swedish women; prospective cohort study concluded: “Low Carbohydrate-high protein diets, used on a regular basis without consideration of the nature of carbohydrates or the sources of proteins, are associated with increased risk of cardiovascular disease” (Lagiou, P., Sandin, S., Lof, M., Trinchopoulos, D., Adami, H. O., Weiderpass, E. 2012). A similar study was published in 2012, Low Carbohydrate diets and all-cause and cause-specific mortality: Two cohort studies, also came to the same conclusion that “a low-carbohydrate diet based on animal sources was associated with higher all-cause mortality in both men and women, whereas a vegetable –based low-carbohydrate diet was associated with lower all-cause and cardiovascular disease mortality rates” (Fung, T. T., Van Dam, R. M., Hankinson, S. E., Stampfer, M., Willet, W. C., Hu, F. B., 2010).

We propose a High Carbohydrate, high fiber, low fat diet (or a whole foods plant based diet) for the reversal of diabetes and heart disease as well.

# What causes insulin resistance?

The cause of insulin resistance seems to be the presence of excess fat stored within muscle cells as well as intra organ fat stores, which eventually culminates into diabetes (Taylor, 2012, p. 267). According to Lichenstein & schwab, (2000, p. 227) “saturated fat, relative to monounsaturated and polyunsaturated fat, appears to be more deleterious with respect to fat-induced insulin sensitivity”. While Estadella et al. (2013, p. 1) states that the types of fat that leads insulin resistance, saturated fat and trans fats, are found in meat and dairy products as well as oils. Therefore we can conclude that our daily dietary

choices, the consumption of meat and dairy as well as oils, are the only source of high levels of saturated and trans fat that lead to the development of type II diabetes. Our dietary patterns are the number one predictor of developing insulin resistance. We cannot get fat in our diet any other way than through the foods we consume.

## Can diabetes be reversed?

According to the CDC the "cure" for diabetes may be found in "Pancreas transplantations, islet cell transplantation (islet cells produce insulin), artificial pancreas development, and genetic manipulation (fat or muscle cells that normally make insulin have a human insulin gene inserted – then these pseudo islet cells are transplanted into people with type 1 diabetes". While some of these methods apply to type 1 diabetes, which is an autoimmune disease, it should be generally be accepted that if certain foods cause harm, the removal of those foods should bring about good health. Some researcher have been able to show that motivated individuals can also reverse their type II diabetes and "diabetes reversal should be a goal in the management of type II diabetes" (Steven, S., Lim, E. L., & Taylor, R., 2013, p. 135).

As Taylor (2012) noted, diabetes is a disease of excess fat on our body, in our muscles and on our organs, removing the fat from our organs can reverse diabetes (p. 267). Taylor (2012) concluded that "these self-reinforcing cycles between liver and pancreas eventually cause metabolic inhibition of insulin secretion after meals and onset of hyperglycemia. It is now clear that type 2 diabetes is a reversible condition of intra-organ fat excess to which some people are more susceptible than others" (p. 267).

According to Yafi & Collins (2015), in the case of the three year old girl who was diagnosed with diabetes, she was provided a diet and exercise routine, was placed on medication and within 6 months she no longer needed diabetes medication, her conditioned was reversed because her blood glucose numbers became controllable through lifestyle modifications and healthy eating habits (152-153).

## In lieu of changing diet, is intensive insulin therapy good for those with type II diabetes?

In past years, intensive insulin therapy, was recognized a way to reduce circulating blood glucose levels within the blood. The American Diabetes Association felt strongly about the potential therapeutic effect this treatment could have on potentially limiting the risk of cardiovascular events. According to a position paper by the ADA on the standards of care for patients with diabetes "Prospective randomized clinical trials have shown that achieving glycemic control is associated with decreased rates of retinopathy, nephropathy, and neuropathy, and epidemiological studies support the potential of intensive glycemic control in the reduction of cardiovascular disease" (2002, p. 215). While this research is now old, the new guidelines only encourage the intensive treatment in the seriously ill and those with type I diabetes.

Recent research in regards to the intensive therapy of type II diabetes patients has been mounting that this is not an acceptable practice. According to Gustafsson et al., (2000) concluded that "diabetic patients treated with oral hypoglycemic agents or insulin, but not those treated with diet alone, have a

significantly increased mortality following acute myocardial infarction compared with non-diabetic patients"(p. 1937). Other studies have also come to the same conclusion, in fact "the Action to Control Cardiovascular Risk in Diabetes (ACCORD) trial was specifically designed to determine the therapeutic strategy targeting normal glycated hemoglobin levels" however the trial stopped "17 months before the scheduled end of the study" due to "the effects of the intensive intervention on mortality and the primary composite outcome of major cardiovascular events in all patients and in prespecified subgroups" (p. 2546). They researchers concluded "these findings identify a previously unrecognized harm of intensive glucose lowering in high-risk patients with type 2 diabetes" (p. 2545)

A meta-analysis of 13 studies conducted by Boussageon et al. (2011) concluded there is "limited benefit of intensive glucose lowering treatment on all cause mortality and deaths from cardiovascular causes" they further concluded "the benefit:risk ratio of intensive glucose lowering treatment in the prevention of macrovacascular and microvascular evens remains uncertain" (p. 1-2).

# We need to ask our self, what does Cure, Remission, Prolonged Remission, Reversal, Prevention mean?

"It is now clear that type 2 diabetes is a reversible condition of intra-organ fat excess to which some people are more susceptible than others" (Taylor, 2013, 267). Taylor uses the term reversible condition. Otherwise have also used the same term. Does the term used matter or is it the outcome?

According to a position paper written by Buse, et al. (2009) the ADA states that a "cure may be defined as restoration to good health" (p. 2133). However they further elaborate that "remission is defined as abatement or disappearance of the signs and symptoms of a disease. Implicit in the latter is the possibility of recurrence of the disease" (p. 2133). This article goes on to state that "prolonged remission is complete remission that lasts for more than 5 years and might operationally be considered a cure" (p. 2133).

Maybe cure is not the word we should be using to describe those who no longer exhibit symptoms of the disease. Many researchers use the term "reverse". Stevens, S., Lim, E. L., and Taylor, R. (2013) state "these data demonstrate that intentional weight loss achieved at home by health-motivated individuals can reverse Type 2 diabetes" in which they further state that "diabetes reversal should be a goal in the management of Type 2 diabetes" (P. 135). Lim, E. L. Hollingsworth, K. G., Aribisala, B. S., Chen, M. J., Mathers, J. C., and Taylor, R. (2011) have stated that "the abnormalities underlying type 2 diabetes are reversible by reducing dietary energy intake" (P. 2506). Other researchers have concluded that high nutrient dense diets can reverse the progression of the disease "the HND diet was very effective in controlling glycemic levels and cardiovascular risk factors" (Dunaief, D. M., Fuhrman, J., Dunaief, J. L. and Ying, G., 2012, p. 364).

According to Medline Plus (2016) reverse is to initiate recovery from. Thus going back to a state in which diabetes did not occur is similar to remission, but reverse may also mean that the disease may reoccur. While the word prevention means, according to Medline Plus (2016), to stop the occurrence of disease.

Diabetes is not only a reversible disease, but could quite possible be a preventable disease. Excess consumption of saturated and Tran fat, only found in meat and dairy and oils to include, palm oil, palm

kernel oil, and coconut oil as well as processed foods containing oils, leads to insulin resistant muscles, which leads to insulin resistant pancreas, which over time leads to an insulin resistant liver. And at some point the whole system crashes. Fat can only enter our body one way, through our mouth. Thus the disease can be considered a food borne illness. Therefore removing high fat meat, dairy, and oils can aid in the reversal of type II diabetes, in addition exercise is helpful for removing fat from muscles, but removing high amounts of saturated and trans fat from the diet should be the number 1 priority as these foods are the initial cause of the disease. This is not to discredit physical activities role in the prevention or the reversal of the disease as it helps the body remove Intramyocellular lipds (fat contained within the muscles). But with all disease we want to learn the root cause, which is the intake of saturated, trans fats and oils.

This brings us to the question of moderation according to Trapp C. B., and Barnard N. D. (2010),

> Patients diagnosed with hypercholesterolemia are usually advised to lower their intake of fat and cholesterol. Ornish states that these moderate changes in diet usually result in only modest reductions in LDL cholesterol levels. Similarly, asking people with diabetes to make moderate changes in nutritional intake (eg, “eat fewer carbs”; “remove the skin from chicken”; “get more exercise”) often achieves equally moderate results, which is one possible reason why 84% of those with type 2 diabetes require oral medications, insulin, or both (p. 158).

Therefore we do not promote moderate consumption of meat, dairy, and oils. The goal for all patients is to reverse their diabetes by the eliminating all meat, dairy products, and oils as well as processed foods that contribute to the progression of the disease. These foods promote the progression of type II diabetes as well as heart disease dyslipidemia and obesity (Estadella, 2013, p.1). Severely restricting or eliminating these foods is the best thing we can do for our overall health. While exercise is helpful, this does not address the cause, which is the consumption of meat, dairy and oils which contain high amounts of saturated fat and trans fat.

# Are whole foods plant-based diet becoming common in preventing and reversing diabetes?

A whole foods plant based diet may be best for individuals with type II diabetes according to Rinaldi, S., Campbell, E. E., Fournier, J., O’Connor, C., Madill, J. (2016, para. 1) stating that “the Canadian Diabetes Association has included plant based diets among the recommended dietary patterns to be used in medical nutrition therapy for persons with type 2 diabetes”. This new report echo’s a report in the Permanente Journal in 2013 which it was determined that “plant-based diets may offer an advantage over those that are not plant based with respect to prevention and management of diabetes” Tuso, P. J., Ismail, M. H., Ha, B. P., Bartolotto, C. (2013, p. 62). This report even encourages physicians to “consider recommending a plant-based diet to all their patients, especially those with high blood pressure, diabetes, cardiovascular disease, or obesity” (p. 61).

What scientist and researchers know is that the accumulation of fat is the primary cause of the disease. In the case of diabetes it is the type of fat, Saturated, and Trans Fat which cause the most significant problems (Estadella, 2013, p. 1; Lichenstein, 2000, p. 227). The source of these fats in the food supply are found in meat and dairy products as well as oils (Estadella et al. 2013; Mayne, 2013, para. 6). A whole foods plant based diet encourages the consumption of fruits, vegetables, whole grains, beans,

peas, and legumes along with nuts and seeds. The diet does not encourage and in fact discourages the consumption of meat and dairy products as well as oils. The diet, in essence, discourages the consumption of products which are shown to increase the risk of developing diabetes.

If we take a step back and look at a general overview of the problem we see that excess fat in the diet, leads to excess fat in the muscles, which leads to excess fat on the body which leads to excess fat on our internal organs which leads to the development of diabetes. Therefore changing the foods we eat should be the primary focus of diabetes prevention and reversal with a small amount of physical activity like walking or biking. As John McDougall says in regards to our nation's most chronic diseases and as is proudly posted on his website "It's the food" (McDougall, 2016).

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