Eat Food, Mostly Fat, to Satiety

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A PHYSICIAN-SUPERVISED PROGRAM

Disclaimer - The information presented here is to be not to be used without formal evaluation and counselling by a licensed physician or healthcare professional. Embarking on the journey of a low carbohydrate high fat diet should not be undertaken without physical examination, cardiac evaluation/imaging, appropriate laboratory testing and regular follow up under the supervision of a healthcare professional and without their express consent. The follow should include but not be limited to physical examination, laboratory testing and when appropriate imaging studies under the guidance of a healthcare professional.
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Root Cause of Obesity

The root cause of “Obesity” and many other chronic diseases like,

1. High Blood Pressure,
2. Diabetes,
3. Cancers,
4. Dementia,
5. Blockages of blood vessels of heart, and
6. Strokes

is Insulin Resistance.

If we can understand what “Insulin Resistance” is and what causes Insulin Resistance, then we can effectively improve these chronic diseases.

Insulin is a key hormone that not only regulates blood sugar control, but also regulates fat metabolism and modulates hunger and satiety signals in the brain.

Insulin resistance is related to a western diet, that his high is refined carbohydrates and sugar (carbohydrates are linked chains of sugar molecules). Years and decades of consumption of a diet high in grains, sugar and processed food is the root cause of stress on the pancreas (which produces insulin).

The pancreas is producing Insulin in response to consumption of carbohydrates. However, decades of stress on the pancreas with a high carbohydrate diet leads to abnormal glucose handling. The pancreas has to make more and more insulin to handle the carbohydrate load and eventually leads to the Insulin not working well and this condition is called Insulin Resistance.

A person with Insulin resistance is making more and not less Insulin at the expense of burning out the pancreas quicker. A question to ask is, are excess insulin level harmful?

A high insulin level seen with insulin resistance does the following

1. Promotes fat deposition
2. Prevents mobilization of fat for energy use
3. Reduce satiety signals by making the brain not see certain hormones that promote satiety – like Leptin (more on Leptin below)
4. Decreases activity because the brain is asking the body conserve energy since it thinks that the body is starving (since it cannot see Leptin)
Obesity is thus not a behavioral problem but a hormonal problem. The treatment of obesity should not therefore be to eat less and exercise more. This will likely fail almost always in the long run. The treatment of obesity should be to ameliorate Insulin Resistance or promote Insulin sensitivity.

How can we promote Insulin Sensitivity or reduce Insulin Resistance

Answers

1. Eat a low Carbohydrate diet
2. Fasting (Intermittent)
3. Exercise (when combined with the above two modalities)

Leptin is a hormone that is made by the fat cells. When fat cells have an increase in fat storage, they elaborate Leptin which then tells the brain that we have enough energy and to stop eating and promote satiety. It also increases activity level.

However, in the presence of high insulin levels and/or Insulin Resistance, the brain cannot see the Leptin and thus gives the sensation to the body that it is starving and to increase food intake and reduce energy expenditure (reduce activity).

**Pure White and Deadly**

The dysregulation of Appetite and Satiety Signals by a Diet High in Sugar and Carbohydrates
“Nature made Sugar hard to get, Man made it easy” – Dr Robert Lustig and others

“When god made the poison (sugar), he packaged it with the antidote (fiber)” – Dr Robert Lustig and others

Why is sugar bad for us?

Sugar and HFCS (high fructose corn syrup) are almost identical in chemical composition. They are 55% Fructose and 45% Glucose.

Fructose is what gives the sugar its sweetness.

There is only one organ in the body that can metabolize the Fructose and this is the liver.

Our ancestor’s livers were used to exposure of about 15 grams of Fructose per day and this was mostly covered with fiber and thus the absorption of this fructose by
the liver was slow. “When god made the poison (sugar), he packaged it with the antidote (fiber)” – Dr Robert Lustig and others

When fructose is consumed as a part of processed food, (like a can of soda) the liver is almost immediately exposed to about 150 calories of sugar. The fructose in this situation is then converted by the liver to fat and leads to the following other events:

1. Production of fat in the liver
2. Increase in Uric Acid production
3. Uric Acid in turns reduces the activity of NO or nitric oxide (NO regulates blood pressure downward)
4. Increase in Uric Acid and reduction in NO leads to Hypertension or high BP.
5. Fat is exported by the liver as triglycerides and gets deposited in Fat cells as Fat.
6. Fat that cannot be exported by the liver is converted to fat droplets in the liver and leads to NASH (Non-alcoholic steatohepatitis)
7. Increases Hepatic Insulin Resistance and thus Insulin does not work making the Pancreas make more insulin.

8. Increased insulin reduces the effectiveness of Leptin which downregulate appetite. However, in the presence of Insulin Resistance, the brain cannot see Leptin and thus gives the impression to the body that it is starving, increasing food intake (leading to excess calories and Obesity).
Sugar vs Fat Debate of the 1970s

Ancel Keys is credited with the Diet Heart Hypothesis or the Dietary Fat Heart Hypothesis. When we observed an increase in “Heart Disease” in the United States in the 1950s to 1970s, he suggested that the culprit for this increase in the risk of Heart Disease in Americans was the results of an increased consumption of Saturated Fat in the Standard American Diet.

There were many other scientists who questioned the validity of the Diet Heart Hypothesis. One of them was John Yudkin who proposed based on his observations that the increase in “Heart Disease” was attributable to increased consumption of refined sugar which our body was not designed to deal with metabolically. He observed that humans have been eating a high fat diet for millions of years but sugar was a recent invention.
Sugar is sweet mostly because of the five carbon molecule called “Fructose”. Higher the fructose content, sweeter the sugar is and our brains crave sugar. With the advent of “High Fructose Corn Syrup” or HFCS, the consumption of Fructose took a major leap. The HFCS is about 55% Fructose and 45% Glucose.

There is only one organ in the body that can metabolize Fructose. This is the liver. Our body is designed genetically to deal with about 15 grams of Fructose per day that is slowly absorbed by the body since most of the Fructose that is present in nature is covered by fiber. Our digestive systems had to first break down the fiber to access the sugar and thus the rate of exposure to the liver of Fructose was limited.

There has been a steady increase in the consumption of pure and refined “Fructose” in our diets since the 1950s as following.

1. Table Sugar
2. High Fructose Corn Syrup
3. Popularization of Fruit Juices – a Glass of Orange Juice contains more Fructose than a can of Soda
4. Soft Drinks
5. Genetic engineering of fruits - The fruits we consume are sweet because they have been bred to contain higher fructose content compared to the amount of fiber. The fruits that our ancestors consumed were “Tart and Bitter”.
6. Juicing – Separates the fiber from the fructose and thus increases the rate of exposure.
7. Addition of Sugar in processed foods (hidden by 50 different names).
8. Breakfast Cereals - The first or second ingredient of cereals is sugar
9. Low fat foods - Like flavored Yogurts have high “Fructose content”

The consumption of Fructose has gone up from about 15 grams per day as part of fruits and vegetable of our ancestors to about 80-100 grams per day for average American consumer – mostly from Sugar, HFCS in Sodas and hidden sugar in processed foods and cereals.
Why is Fructose Bad for our Health

As mentioned, there is only one target for Fructose in the body and this is the liver. When the liver is exposed to “Fructose” in large amounts, it converts this sugar to fat in the liver cells and promotes “Insulin Resistance”. This leads to..

1. The brain not interpreting satiety signals.
2. Fructose consumption also dis-regulate appetite control and leads to ingestion of excess calories.

What makes us Overweight and Obese?

Years of dietary stress from intake of carbohydrates is the culprit.

Our ancestors were not exposed to such high levels of refined carbohydrate in their diet. Even fruits that they ate were seasonal and naturally bitter and tart unlike the fructose-rich fruits that are sold in the supermarkets in the modern era. Fructose is a sugar (carbohydrate) and thus nature’s candy.

Carbohydrates - both simple and complex are broken down to simple sugars by the body. This increases the level of the hormone Insulin to keep the level of glucose (sugar) in the blood at normal levels.

Insulin is the key hormone that makes the body store fat. In its presence the fat cells of our body gain fat and the fat cells of our body do not release fat into the blood circulation to be used as energy.

Thus carbohydrate restriction is a key factor for an effective weight loss diet especially if obesity is present. Obesity is defined as body mass index (BMI) of 30. A BMI between 25 and 30 is considered overweight. The prevalence of obesity in the U.S. in the 1970s was about 10-15%. In 2015, the prevalence is about 33%. This increase in obesity rate is directly attributable to the change in our diet with an increase in dietary carbohydrates, especially from processed foods.

Restriction of carbohydrates leads to a reduction in Insulin levels. This allows the fat cells in our body to release the fats (fatty acids) in to the blood stream. Over time, the muscle cells adjust to the low carbohydrate high fat (LCHF) diet by using the fatty acid directly for energy. More importantly, the liver takes up these fatty acids and converts them to simpler water soluble small molecules call Ketones.
Thus, ketones are energy-giving by-products of fat metabolism.

Ketones are preferred as fuel by the brain and heart muscles. The skeletal muscle can utilize the fatty acids themselves as fuel after a few weeks of a LCHF diet.

There is a common myth that 100 grams of sugar (glucose) per day is necessary for the brain to function. This is true for a person who is consuming a high carbohydrate diet. However, the fat in a person eating a LCHF diet is mobilized and converted to ketones by the liver. These ketones are a more efficient fuel that the brain prefers over glucose/sugar.

The epidemic of obesity in the US and the world is directly attributed to increase in carbohydrate consumption. The food industry has been able to insert highly processed, simple carbohydrates in packaged foods. In addition, our learned fear of fat has led to the consumption of the alternative macronutrient – carbohydrates. Our bodies are not designed to effectively metabolize these carbohydrates without increasing Insulin levels. This fear that fat is dangerous to our health was instilled and popularized by the Health Organizations and physicians like me for the last 50 years.

To summarize the information above

Carbohydrates ingestion makes the body release Insulin.

Insulin promotes fat storage and prevents fat mobilization.

Low carbohydrate diets are essential to decrease the levels of Insulin in the body.

Low insulin allows the fat cells to mobilize fatty acids to be used for energy.

The liver takes up the fatty acids from the fat cells and converts them to small simple molecules called ketones that can be used by the brain as fuel instead of glucose/sugar.

What is Ketosis and what are Ketone bodies?

Ketones are small molecules that the liver makes from fatty acids. These are water soluble molecules much like sugar.

Ketones are only produced when the intake of carbohydrates is very low, during fasting or after exercise. The body is mobilizing fat from fat cells to run essential metabolism.
If after exercise or during exercise, sugar or carbohydrates are consumed then ketones are not produced or produced at lower rates.

Ketones can be measured in the breath, urine or blood. However, only the blood measurement of Ketones is reliable.

Nutritional Ketosis is a term that refers to the production of ketones with a LCHF diet.

The degree of carbohydrate restriction necessary to produce ketones varies from person to person. Typically, overweight (BMI 25-30) individuals have to reduce carbohydrate consumption to less than 50 grams per day for nutritional Ketosis. On the other hand, obese individuals (BMI >30) have to more seriously reduce carbohydrate consumption to between 20-30 grams per day.

Nutritional ketosis is defined as ketone levels in blood of 0.5 to 3 mMol/L.

The Abbott Precision Xtra meter is a reasonably priced and easy-to-use device with which one can measure ketone levels with a finger stick blood draw.

**Can a high protein diet prevent nutritional Ketosis?** (This is an advanced concept – one should worry about this if you are at the plateau in weight loss or not achieving the results that you desire)

Over the years our fear of fat intake has made us think that more lean meats and less red meat is good for us. This theory is based on the myth that increased fat intake from animal sources increases our blood cholesterol level which leads to heart disease. This theory is unproven and there are numerous studies that refute this hypothesis. This information is now being dispersed to the general public and it is not unusual to find that many are knowledgeable that high fat and cholesterol diets do not correlate with heart disease. In fact, a low fat diet, which by design is a carbohydrate rich diet, leads to Insulin Resistance, Metabolic syndrome and Type 2 Diabetes. These disease states are scientifically proven to increase the likelihood of heart disease, cancer and dementia.

**What is less well known is that consuming lean meats and proteins can prevent nutritional ketosis; this is because the body converts the consumed proteins to glucose in the liver by a process called gluconeogenesis.** Through gluconeogenesis proteins stimulate insulin release and prevent fat mobilization.

Thus proteins should be consumed in moderate amounts, about 1 to 1.5 grams per kilogram body weight.
In summary,

Eat good fat to reverse the years to metabolic stress that we have heaped on our bodies by consuming a diet high in both simple and complex carbohydrates.

Eat protein in moderation.

Measure serum Ketone levels especially if you suffer from type II diabetes or obesity. Weight loss is accelerated once an individual achieves a state of nutritional ketosis. Once in this state, you are using the fat as fuel for the body.

If you are not able to measure serum ketone levels, they can be measured in our office during your office visit.

Do Diabetes medications need to be adjusted with a LCHF diet?

Yes. The blood sugar in diabetics can fall dramatically with a LCHF diet. This can create serious hypoglycemia or low blood sugar levels in people taking diabetic medications. One needs to consult with the primary or diabetic physician to get the doses of these medications adjusted with the LCHF diet.

Does blood pressure drop with the LCHF diet?

Yes. The glycogen that is stored in the body also stores water and salt. With the LCHF diet, the glycogen stores are reduced resulting in a reduction of water and salt within the human body. In addition, as Insulin levels fall, the kidneys dump the salt that is filtered through them in to urine. Thus blood pressure may fall and the person may experience the LCHF diet flu also called the “Atkins Flu”. This can easily be corrected by increased salt intake and/or by reducing or eliminating the blood pressure medications.

Do you need to follow this diet under the supervision of a health care professional?

Yes. Before starting on this diet, get key blood tests of metabolic health done.

These include but not limited to the following:

- Lipid Profile – Especially HDL, Triglycerides and LDL
- HbA1c
• Fasting Insulin Levels
• Lp (a)
• LDL particle size and number – The LDL particle size is a measure of metabolic health. Larger the particle, better the health.
• IDexa measured body fat and abdominal fat percentage
• Cardiac evaluation with stress test in some patients – especially is symptoms of heart disease or risk factors for heart disease are high

These tests may have to be repeated after the diet is started in order to measure the improvements in certain key areas, especially HDL, Triglycerides, HbA1c, Fasting Insulin levels, abdominal fat percentage and LDL particle size.
Sample Menus

Breakfast:

Free Range Egg Khagina

3 eggs – free range fried in 3 table spoons of butter or Ghee (clarified butter) with lemon, pepper, onions, salt and Cilantro.

Add onions to the Ghee and Sauté. Add eggs and then fry them with the seasoning. Can add additional butter or sour cream or grated cheese especially if more calories are required.

Add breakfast meats as desired.

Add ½ cup of Avocado instead of sour cream

Sour Cream with Pistachios/Ground Flaxseed/Blackberries

Take 3 or more tablespoons of organic sour cream

Add half a cup of Pistachios

1 tablespoon of ground flaxseed.

½ cup of blackberries

Mix them in a bowl and enjoy as breakfast cereal

Add half an Avocado for additional calories.

Buttered Coffee

Brew coffee or better still consider making an Americano (expresso with hot water)

Add 2 to three table spoons of butter - about 2 ounces

Place the coffee and butter in a blender and blend on high for about 45 seconds.

Pour in a cup, reheat if required for about 20-30 seconds in a microwave and enjoy.

The butter mixes well with coffee in a blender and gives it a unique taste.
**Black Coffee with French Cheese** – Port Salut, Brie or Double Cream Brie

Make black coffee as per your favorite drip coffee maker or café Americano.

Drink while biting a moderate sized wedge of Port Salut or Double Cream Brie.

The coffee is quite satisfying with cheese. Alternatively you can you use heavy cream in your coffee.

**Lunch**

Large Salad of above the ground vegetables.

Green leafy vegetable – Kale, spinach, spring mix

Brussel sprouts, Celery, Olives, green and red peppers, Cilantro,

¾ cup to 1 cup of nuts – Almonds, Pistachios, Pecans or Walnuts

3-4 tablespoons of olive oil

Salt, Pepper and Lemon seasoning as desired.

½ cup Avocado promotes the flavor of the seasoning also

Sour Cream with Nuts and ½ cup berries as dessert - see above.

**Dinner**

**Meat Casserole**

Ground chuck from free range Bison or free range Kobe Beef – 1 lb.

(Alternatively grass fed lamb ground chuck can be used)

3 eggs – whisked

2 table spoons butter or clarified Butter – Ghee
2 table spoons of Coconut oil

Seasoning – salt, pepper, garlic, crushed red pepper, paprika, turmeric - per taste and tolerance

3 – 4 large Jalapeno peppers - diced in to small pieces.

½ cup of diced onions

½ a stalk of Cilantro

Mix in a baking glass dish.

Pre heat oven to 325 degrees Fahrenheit – cook for about 30 to 35 minutes

Wait 5 minutes after it comes out of the oven to serve.

**Indian Baked Shrimp Curry**

1 lbs. of peeled and deveined Large Gulf Coast Shrimps – wild caught - not farmed

2 table spoons butter or clarified Butter – Ghee

2 table spoons of Coconut oil

Seasoning – salt, pepper, garlic, crushed red pepper, paprika, turmeric - per taste and tolerance

3 – 4 large Jalapeno peppers - diced in to small pieces.

½ cup of diced onions

½ a bunch of Cilantro

Mix in a baking glass dish.

Pre heat oven to 325 degrees Fahrenheit – cook for about 30 to 35 minutes

Wait 5 minutes after it comes out of the oven to serve.
Foods to Buy

**Fats and Oils**

Ghee

Butter

Coconut Oil

Olive Oil

(Avoid corn, sunflower, safflower and soybean oil since it is high in Omega – 6)

**Meats**

Free range chicken

Grass fed beef and lamb

Marbled Steak

High fat content ground Chuck - preferably from grass fed cattle

Lamb chops – free Range

**Dairy**

French Cheese – Port Salut, Brie (double cream), Delice, Gouda

Organic Sour Cream

Organic heavy cream

**Vegetables**

Above ground vegetable

Avocado
Cabbage
Olives

**Nuts** – all except peanuts – they are legumes

Pistachios
Pecans
Walnuts
Almonds

Ground Flaxseed - this is important source of Omega 3 fatty acids for vegetarians /vegans

**Fish**

Wild Caught fish – Salmon, Red Snapper, Trout, Atlantic Grouper

Gulf Shrimps – Wild caught not farmed

**Fruits** – to be eaten in moderation

Blackberries
Raspberries
Blueberries
Strawberries
What not to buy

Sugar
Sweets and cookies
Candy
Ice cream
Bread or grains of any kind
Cereal
Margarine – especially if it has transfat
Flour
Pasta
Tuberous or starchy vegetables – like potatoes, beans
Light Yogurts
Milk - contains lactose
Almond milk – usually contains added sugar or preservatives.

What not to eat

Sugar – of all kinds – comes in different names in processed food.
Sweets
Soft drinks with high fructose corn syrup
Diet soda
Honey
Juices
Heart Physicians simple guideline of the do’s and don’ts of Food Intake

No sugar and sweets

No Grains
This means no whole wheat bread, rice, brown rice, pasta, oatmeal or store bought cereal - The first or second ingredient of store bought cereal is sugar and grains

No Starch
Starch is found in below ground vegetable – Potatoes
Also fruits have starch – like Bananas
Also certain foods are high is Fructose which is natures sugar – like Apples, Oranges, etc
What not to eat

Sugar – of all kinds – comes in different names in processed food.

Sweets

Soft drinks with high fructose corn syrup

Diet soda

Honey

Juices

Avoid all Grains – no wheat, bread, rice, pasta, quinoa, oats – Includes cereal.

Avoid starchy Vegetable and fruits - no bananas, no potatoes, no apples, no oranges

Apples and oranges is small quantities okay after 3 months on diet.

No Juices and no juicing

No Honey.

No beans in the first 3 months – then in moderation.

Okay to Eat –

Major goal - Eat the good fat – Found in Avacodos, Nuts and Olive Oil and saturated fat. Avoid vegetable oils and trans-fat

Important – do not eat a high fat diet and cheat on Carbohydrates – this can be detrimental.

Protein consumption should be in moderation since high protein diet can stress the kidneys. In addition as mentioned above excess protein consumption can increase insulin levels.

Reduce carbohydrate intake to less than 50 grams/day - more if you are severely overweight or obese or a type 2 diabetic.
Any food that you want – you can check in google - write the food name and then nutritional information – you will get the content of carbohydrate and sugars per serving.

Eat high quality carbohydrate which is green leafy vegetables. Starchy vegetable are not high quality vegetables. Grains are not high quality vegetables.

Green Leafy Vegetable as Salads – **no commercial Salad dressing** – most commercial salad dressings have sugar and sugar like substances in it

Meats. – preferably grass fed - if you cannot get this add ground Flaxseed to your meats.

Free Range Poultry

Free range Eggs

Fish – wild Caught

Shrimps - Wild Caught.

Avacados – considered good fat!

Nuts – all except peanuts. (Peanuts are legumes)

Cheese – in moderation – but no milk and yogurt. (Milk and Yogurt have lactose which is a sugar/carbohydrate)

Coconut and coconut oil okay

Olive and olive oils – other vegetable oils are not permitted.

Butter

Focus on Fat – not on protein

Avoid Carbohydrate.

**Fruits** - that are **okay to eat in moderation** - About a cup per day - More fruits will increase Insulin Resistance.

Berries - Blueberries, Raspberries, Blackberries, Strawberries – about less and one cup per day.

Avoid fruits that are high in fructose – like mangoes, bananas, apples, oranges – especially in the first three months on the diet.
“Coating of the bodies proteins with sugar deforms them and increases risk of heart disease and dementia”

Hemoglobin A1C

Hemoglobin A1C is the oxygen carrying protein in our red blood cells that is coated with sugar in the blood. It is a reflection of blood sugar over a 90 day period.

Why is coating of proteins with sugar relevant to disease?

Proteins are molecules in the body that serve important functions of living. They carry out muscular contraction, thinking, digestion, etc. They maintain a structural integrity by folding of the chain of amino-acids (Building Blocks of Proteins) in a certain way. When coated with sugar the folding and hence the structure of the protein is altered. Thus leads to malfunctioning proteins that get clumped and do not get removed and recycled properly.

Why is clumping and “Structural Deformation of Proteins” important to dementia?

Dementia is now considered to be “Type 3 Diabetes”. The clumping of proteins prevents their proper removal from the brain and leads to protein plaques (scars) in the brain leading to dementia.

Are small increases in Blood Sugar or Hgbaic associated with Dementia?

Yes

What is normal Hemoglobin A1C levels?

Normal Hgbaic levels are less than 5.6 percent.
A level between **5.7 to 6.0** is considered to be pre diabetes (some like an upper limit of 6.3)

A level above **6.0** is considered to be “**Diabetes Mellitus**”
“Exercising your body is better at protecting the brain than exercising the brain itself”

Why is Exercise Important and Beneficial?

Human body is genetically programmed to move. In the words of experts who have explored our genetic heritage we have been “Born to Run”. *

If we take a human and compare it to any other mammal (animal) there are two very distinct features (as far as exercise is concerned)

1. **Brain Size** – Our brains are over 3 pounds and much larger compared to our body size
2. **Capacity to regulate body heat or sweat** – Thermoregulation – this is better than any other animal.

Thus we can out run any other animal in terms of distance in a hot/warm climate – Better than horses, dogs and every other animal.

Benefits

**Prevention of Dementia** – Exercise has been shown in studies to reduce the risk of dementia better than brain teaser and crossword puzzles - in other words “exercising your body is better at protecting the brain than exercising the brain itself”.

**Diabetes prevention**

**Weight management**

**Blood pressure Control**

**Improved Cholesterol Metabolism**

**Muscle mass preservation**

**Sense of well being**

*Christopher Mcduggal’s book - Born to Run.*
Why is Fiber important in diet

Fiber that is found in vegetable is both soluble and non-soluble fiber

**Soluble fiber is one that absorbs water and swells up.**

Insoluble fiber is not absorbed.

**Soluble fiber is worked on by the bacteria in our gut and can cause gases**

However, both types of fiber are necessary for our diet.

**Our ancestors ate about 100 grams of fiber per day.**

We are recommended about 25-30 grams per day.

**Fiber prevents the following**

1. Constipation
2. Slow down the absorption of sugar
3. Gives liver the time to process the sugar properly

Natural fiber is better than artificially added fiber.

When fiber is added to processed food it is not helpful since it does not coat the sugar in the food and the sugar is rapid absorbed.

**For fiber to prevent sugar absorption quickly to the liver, it has to coat the sugar completely.**

Thus fruits and vegetable high in fiber are good since the sugar of these vegetables and fruits is coated.

Bread, juices, and other processed food may have fiber but it does not good at preventing the sugar absorption since the fiber does not coat the sugar and the body does not have to break down the fiber first to get at the sugar.

**Good sources of fiber**

Green Leafy vegetable

Avocado – each medium to large size Avocado has about 15 grams of fiber
Fruits - high in fiber fruits should only be consumed after exercise since their sugar content is high – like Blackberries, Blueberries, Raspberries, and Strawberries.
**Why your heart doctor does not like Sulphonylurea medications for Diabetes Control**

**Sulphonylurea Medications**

- Glipizide (Glucotrol)
- Gliburide (Diabeta)
- Glimepiride - (Amaryl)

These medications make the pancreas release insulin. In diabetics the Insulin does not work well and thus the pancreas is already under stress because it is being asked by the body to overproduce the Insulin to keep the blood sugars down. Thus when you ask the pancreas to make more insulin as these sulphonyl urea medications do, the pancreas loses its function over time (in essence burns out quicker).

Diabetes is one of the strongest risk factors for heart disease.

Thus, your heart doctor would like to focus on diet to improve diabetes control rather than focus on medications. A low carbohydrate diet is a very powerful tool to reduce your blood sugars with just as much or more potency than medications. This will have to be done under physician supervision.

However just diet alone may not control your diabetes.

Thus, we prefer medications that improve the effectiveness of Insulin

These are:

- Metformin (Glucophage)
- Januvia (sitagliptin) /Tradjenta (linagliptin)
- Actos (pioglitazone)

If these medications are not effective in controlling your blood sugars then we would even recommend taking Insulin shots rather than the sulphonylurea medications. However, we would like to leave the final decision between you and your primary physician or endocrinologist.
Functions of Cholesterol

1. Reduce the risk of infections
2. Reduces cancer risk
3. Reduces inflammation
4. Provides the precursor molecules for hormones
   a. Vitamin D
   b. Sex Hormones
   c. Steroid Hormones
5. Cholesterol is important for the structure of the brain
   a. Provides integrity to the nerve cell
   b. Aids in conduction of messages across the nerve cells
   c. Important for memory and cognition
   d. 25% of body cholesterol is found in the brain
6. Epidemiologic studies have shown that higher cholesterol levels (when compared to lower levels) are associated with better memory and cognitive function.
7. Important for muscle function and strength
   a. Muscle needs cholesterol to use energy
   b. Co-Q10 is derived from cholesterol
   c. Co-Q10 is need by muscles to burn fuel
   d. Taking Co-Q10 by mouth does not help since it does not get to the level of the muscles
   e. Expecting Co-10 taken by mouth to reach the levels of the mitochondria inside muscle cells is like thinking that we can place something in the glove compartment of the car and thinking that it will get in to the engine.
8. LDL is a carrier molecule in blood that is used to transport Cholesterol to other cells from the liver.
   a. LDL is composed of a fat layer along with special proteins called lipoproteins
   b. Inside this molecule is carried cholesterol and triglycerides (fat) for transport to other cells.
   c. LDL however also carries vitamin E, Co-Q10 and antioxidants.
   d. Reducing LDL levels will thus reduce vitamin E, Co-Q10 and antioxidant supply to other cells.
9. The degree of benefit of cholesterol lowering medications in prevention of death in people without established heart disease is very low to non-existent. Thus, several physicians are making a quiet retreat from the cholesterol hypothesis.
10. The prevention of death by use of cholesterol in women has not been shown conclusively in any clinical study
11. The magnitude of benefit of lowering cholesterol with medication in the prevention of death in middle aged men with established heart disease is very low – at best about 0.5% per year in the best clinical trials. This is known as secondary prevention in which a medication is being used in
a patient with already established coronary arteri disease (CAD) as opposed to primary prevention in which medications are used to prevent the likelihood of CAD.

12. This benefit in the prevention of death in patients with CAD has however not been consistent across all clinical trials. In addition, the earlier studies done in the 1990s like the 4S trial showed about an overall 0.5% absolute risk reduction of death with the use of statins. However, subsequent trials done with greater oversight after the Vioxx scandal relating to pharmaceutical industry’s manipulation of the clinical trial data (for the Vioxx study – VIGOR) have shown overall lower benefit of statins.

13. The side effects of these drugs on muscles, memory, cancer risk, infection, diabetes, libido (erectile dysfunction) has not been well studied on purpose because it does not serve the business interest of pharmaceutical companies making these drugs. It is estimated that the worldwide sale of statins is over 20 billion dollars. The side effects of these medications are seldom discussed by physicians who prescribe statins.

**Side effects of statins**

**Neurocognitive side effects – Memory and Thinking**

Cholesterol is needed by the brain to function. About 25% of the body’s cholesterol is in the brain. The cholesterol is a key factor needed for cognitions and memory. Thus, when we use statins, the cholesterol production in the brain is altered.

There are reports from patients about neurocognitive side effects of statins (see Dr Graveline’s book - Lipitor, thief of memory)

The FDA lists memory and cognitive problems as a potential side effect of statins.

The cholesterol is also needed by some of the neurotransmitters to function well in the brain (these are signaling molecules). In the absence of cholesterol, serotonin does not work and this can lead to depression. There is no strong data that attests to his fact but there is a lack of research in this area. To ask pharmaceutical companies to do research that statins can lead to depression is like asking “Big Tobacco” companies to fund research on the role of smoking in causing lung cancer.

**Myopathy - Muscle Aches and pains**
The muscle utilize a substance that is a product of cholesterol metabolism called Co-Q 10. The Co-Q 10 is needed by the internal machinery of the muscle to obtain energy for muscle use. (The Co-Q 10 is a part of the mitochondria, the energy producing part of the muscle cells)

When statins are used, the production of the Co-Q 10 is reduced inside the muscle cells. In the absence of Co-Q 10, the muscle cells do not get the energy that they need and thus this can lead to some of the symptoms of statins induced myopathy such as:

**Muscle Aches**

**Fatigue and tiredness**

**Lack of energy**

I have often noticed that patients on this drug get “disuse atrophy of major muscle groups” and get progressively weaker over a number of years. Since cholesterol lowering medications are taken for years if not decades, the side effects are slow and insidious and thus hardly noticed by patients. They tend to think that there are tired and fatigued because they are getting older.

**Joint Injury – Muscle as the back bone support for the joints**

Strong muscles are the backbone of a stable joint. If the muscle get weaker the joints are at risk of damage. Many patients on statins complain of joint pains. These could be the results of weak muscle groups that would ordinarily support and keep the joint strong. Over a number of years this can lead to joint injury. Many patient get surgical correction by orthopedic surgeons for these joint ailments, but the possibility that statins may have contributed to joint injury should be considered and the statins stopped to see if the symptoms improve and surgery avoided if the function improves.

**Type II Diabetes**

There is an increased likelihood of type two diabetes in statin users. This has been found in numerous studies such at the Jupiter trial with Crestor and also in the large 50 000 patient Women Health Initiative (WHI).

**Vitamin D**
Vitamin D is derived from cholesterol. When cholesterol like compounds are exposed to the sunlight under our skin, the body converts it to Vitamin D.

**Sex Hormones**

The sex hormones are derived from cholesterol. There is a potential of reduction in libido when statins are used.
Why Fasting Works so Well

The root cause of Western diseases is Insulin Resistance. We will describe what insulin resistance in a bit. At this time we would like to elaborate what Western Diseases are.

1. Coronary Artery disease – Leading to Heart Attacks, stents and bypass surgery
2. Hypertension – or high Blood pressure
3. Cancers
4. Obesity
5. Diabetes
6. NASH – a type of Liver Cirrhosis not related to alcohol
7. Strokes
8. Dementia

The above is a partial list.

Insulin resistance is when the insulin does not work well to regulate blood sugar in the bloodstream and thus the body has to make more insulin to overcome the lack of effectiveness of this hormone.

Insulin Resistance is related to the western diet.

The Western diet consists of large amounts of Carbohydrates (especially refined grains, Sugars, Juices, certain fruits and starchy vegetables). Whole grains also come in this category to some extent. Our bodies are not designed to consume large amounts of carbohydrates which is an invention of civilization. Thus excess carbohydrate consumption leads to insulin resistance.

If we address and treat this biochemical abnormality – that is Insulin Resistance, then we will effectively treat the root cause of Hypertension, Obesity, Diabetes and Heart Disease. On the other hand, you may not want to go down this path and wants medications for all of these like someone wanting Tylenol for fever. With this strategy we are addressing the symptoms but not the underlying hormonal/biochemical abnormality.

We think that there are three possible approaches.

1. Treat with medications
2. Treat with a combination of medications and some measures of life style changes.
3. Focus more on life style changes and use medications temporarily

This is a physician supervised interventions. This process requires you to gain knowledge in the hormonal abnormalities that are leading to medical condition for which you need help.
Let us now look at what we mean by lifestyle changes to address Insulin Resistance.

1. **Low Carbohydrate High Fat diet - LCHF diet - (focus will be on good fats)**
2. **Interruption Fasting**
3. **Exercise**

**Intermittent Fasting**

If you are heavy, your body makes more insulin than normal.

High insulin levels prevent the body for using the stored fat or the fat that you eat for energy. Thus, insulin promotes lipogenesis – which is deposition of fat, and prevents Lipolysis – with is breakdown of fat (for energy use)

An obese and overweight person has high fasting insulin levels and higher than normal Insulin release from pancreas with ingestion of standard American meal consisting of a mixture of carbohydrates, fats and proteins.

Thus for some people, just changing to a LCHF diet which is high is fats and very low in carbohydrates reduces insulin release and improves the biochemical abnormality. However this is not uniformly effective. The reason for this is that about 50% of the insulin release is not related to ingestion of food and is occurring as background insulin secretion. Thus, obese individuals and type 2 diabetics have high background basal insulin levels that prevents fat burning or use of fat for fuel. Thus, LCHF diet may not help some individuals in correcting the biochemical abnormality and hence there will be limited weight loss or a weight plateau.

Normal fasting insulin levels are less than 5 uIU/mL.

**Homeostatic Model of Insulin Resistance and Beta Cell function.**

The glucose circulation in the blood is in feedback loop with the insulin producing beta cells of the pancreas. With progression of impaired glucose tolerance, there is resistance to the action of insulin that initially increases insulin levels (albeit, there is resistance to the action of insulin on glucose transport in to the cells). However over time, there is destruction of beta cell of pancreas as this endocrine gland is asked to pump out more insulin for the regulation of sugar metabolism.
HOMA is a method developed to assess not only Insulin Resistance but also the assess pancreatic beta cell function.

**HOMA-IR** is calculated as follows:

\[
\text{HOMA-IR} = \frac{\text{glucose in mg/dl} \times \text{Insulin ulU/mL}}{405}
\]

\[
\% \text{ Beta Cell function or HOMA-Beta} = \frac{360 \times \text{Insulin ulU/mL}}{\text{Glucose in mg/dl} - 63}
\]

HOMA-IR levels of less than 1.0 is considered to show normal insulin sensitivity.

>1.5 - Insulin Resistance

>2.9 – severe insulin resistance

In order to overcome this high insulin resistance we want to introduce you to the concept of intermittent fasting.

However first lets us show graphically how high the insulin levels are in obesity.
Then, we should graphically look at how high insulin levels go to with ingestion of food in obese and normal weight individuals.

Finally, we should look at what happens to insulin levels with a LCHF diet and fasting. As you can see one of the most powerful treatment tools you have at your disposal is fasting.
As a bonus we should answer two questions,

1. Does Intermittent Fasting lead to loss of muscle mass
2. Does Intermittent Fasting lead to slowing of metabolism

**ALTERNATE-DAY FASTING IN NONOBSESE HUMANS**

**TABLE 2**

Resting metabolic rate (RMR), respiratory quotient (RQ), and fat and carbohydrate oxidation measured at baseline and after a fed day (day 21) and a fast day (day 22)

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Day 21</th>
<th>Day 22</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMR (kJ/d)</td>
<td>6675 ± 283</td>
<td>6292 ± 268</td>
<td>6329 ± 260</td>
</tr>
<tr>
<td>RQ</td>
<td>0.85 ± 0.01</td>
<td>0.86 ± 0.02</td>
<td>0.79 ± 0.01</td>
</tr>
<tr>
<td>Fat oxidation (g/24 h)</td>
<td>64 ± 8</td>
<td>54 ± 10</td>
<td>101 ± 92</td>
</tr>
<tr>
<td>Carbohydrate oxidation (g/24 h)</td>
<td>175 ± 17</td>
<td>184 ± 24</td>
<td>81 ± 16</td>
</tr>
</tbody>
</table>

**Fat Burning up 58%**

**Carb burning down 53%**
How to Fast

One has to train the body to fast.

Start by gradually increasing the interval between meals.

If you are used to eating lunch at 12 noon, delay the first day to 1 pm. Then the next day to 2, subsequent days to 3, 4, 5 pm until you learn to skip a meal altogether. This along with the LCHF diet will have incremental benefits in reducing insulin levels.

You should consider skipping meals at least 2-5 days per week depending on your individual metabolic abnormality.

Other protocols of rigorous fasting, like consuming only 500 to 600 calories per day, should be considered after you are a pro at skipping meals several times per week.

FAT FAST

Another concept that one can consider is what is known as a FAT FAST. With this type of fast, you can consume just pure fat to stave of hunger but not consume any carbohydrates or proteins. Proteins, especially highly processed proteins like found in protein drinks and/or protein bars stimulate insulin secretion almost as much as carbohydrates.

In a FAT FAST, you can consume the following foods.

1. Butter as part of butter coffee or butter drink
2. Coconut Oil
Intermittent Fasting – a synopsis for those who do not want to read why fasting works so well!

Fasting is a powerful method to improve Insulin Sensitivity or reduce Insulin Resistance.

With fasting the carbohydrate stores are depleted within an 8 hour time frame and then body has to resort to mobilizing fat for energy use. For fat to be mobilized and used as fuel, the Insulin levels have to drop. In the presence of high insulin levels or Insulin resistance, the body cannot mobilize fat for energy use.

A popular myth is that with fasting there is a reduction or slowing of metabolism. This is not the case. Studies have shown that with fasting there is an increase in metabolism. The evolutionary reason for this is that our ancestors were exposed to periods of food shortage unlike the modern man who has a steady supply of calories. Thus fasting was not a choice for them but a part of their food supply chain.

For them to survive food shortages they had to mobilize fat to be used as energy so that they could procure food through hunting. This requires an increase in energy expenditure and if fasting reduced energy expenditure they would not have been able to survive.

A second popular myth is that fasting and/or starvation lead to muscle loss and thus the lean body mass will decrease while fat mass is not used. This is also false. It would not make sense for our physiology to store energy as fat when food is available and burn muscle when food is in short supply. If this were the case, our ancestors should have been blobs of fat because they would store energy as fat and when food was not available, burn muscle and thus get fatter as time went by.

Studies show that with fasting while you drop the total fat mass, the lean body mass does not change.

The key benefit of fasting thus is to reduce Insulin levels so that the body can now mobilize fat for energy use.

Fasting is one of the most metabolically beneficial things we can do to our bodies.

Fasting promotes the following:

- The capacity of the body to burn fat for fuel
- Improve Insulin Sensitivity – thus improving diabetes or blood sugar control
- Help promote weight loss
Intermittent fasting is done the following way:

We recommend fasting for two days of the week: Mondays and Thursdays.

We recommend a total of 600 calories per day divided in two meals.

**Breakfast**

Two eggs – with a slice of bacon or eggs done in Olive Oil

**Lunch – Skip lunch**

Can have water, black tea and coffee – without sugar - thus not non-calorie drink

Avoid artificial non-caloric sweeteners.

**Dinner**

Mixed greens salad with above ground vegetable with two table spoons of Olive Oil (salt and pepper for seasoning)
Diabetes – an alternative perspective

The other day I was asked to advice on the management of a young man with Type II diabetes.

Here are the particular of this man.

Age - 32
Height - 5 feet 8 inches
Weight 93 kgs or for us Americans - 204 lbs
Waist Circumference - 38
BMI 33.9
Hgbaic - 6.4
Oral Glucose Tolerance Test - 1 hour glucose - over 300
Triglycerides - about 400 mg/dl
HDL - in the 30s
LDL - about 120 mg/dl

He had been to a local physician and was prescribed
Glimepiride 2 mg/Metformin 1000 mg combo twice daily with breakfast and dinner.
Empaglifozin 10 mg every day

Dietary counselling was not stressed but patient in passing was told cut down on sugar and fats and to start an exercise program.

Now this is quite a common scenario. A middle aged man or woman presenting to a physician with new diagnosis of Type II diabetes. The most common advice is the following.

1. Diabetes is a progressive disease.
2. Need to cut down on fat because diabetes is a strong risk factor for heart disease as is consumption of fat and therefore the two together can be dangerous. Thus, stop eating fat period. The emphasis on cutting down on sugar is less vigorously stated.
3. Take medications to control your sugars because there is no other real option.

There is almost no attempt to discuss the pathophysiology of this metabolic abnormality. It is mentioned in passing that this is a genetic disorder and runs in families and that the victim brought it on himself/herself to some degree by overeating and getting obese like the individual mentioned above.

Such a stance taken by the medical profession is nothing short of insanity. It indicates that the medical profession is ready to abdicate its ethics and intelligence to the opinion leaders who are intent to make the propaganda of the pharmaceutical companies take center stage in the treatment of type II diabetes. There can be no other explanation, otherwise the treatment of Type II diabetes would look very different.

Let us examine why I make such a bold statement. Will it hold up to logic, common sense and what we know about medical science?

Type II diabetes is a disorder of Insulin Excess and not insulin deficiency in the vast majority of patients. The reason for Insulin excess is that our tissues that utilize carbohydrates become resistant to the action of insulin (Insulin Resistance or IR). Our tissues need insulin to be able to transport the sugar from the blood stream in to the cells. We have evolved over millions of years to be able to deal with only small amounts of sugar and carbohydrates in our diet. In addition, the carbohydrates that were available to our ancestors were mostly complex. For example the fruits that they ate were:
1. Confining to a few weeks in summer.
2. Were bitter and tart and thus did not have the amount of fructose that modern fruits have in abundance. This change in the nutrient content of our fruits is most likely through selective breeding.

When I ask people how much sugar we have circulating in the blood, they often overestimate. When I tell them if you drain the entire blood of a person and tally up the total amount of sugar in it, they are totally surprised to find that it is only about one teaspoon (about 5 grams).

Thus when we eat sugar, we have to dispose it immediately because our bodies cannot tolerate high levels of sugar in the blood stream. The typical American consumes about 400-500 grams of carbohydrate per day. Since most of this is simple carbohydrates, it is rapidly absorbed in to the blood stream.

The major organ that has to handle this sugar load is the liver. The muscle can also take up sugar but only if their sugar stores are empty. The liver takes up sugar and convert it to a storage form of carbohydrate called glycogen. We have a limited capacity in our liver to store sugar as glycogen. Only about a 100 grams of glycogen can be stored by the liver. The ingested carbohydrate that cannot be either burned or stored as glycogen then is converted by the liver in to fat which is then transported to the fat cells for storage. The conversion of sugar to fat in the liver is called hepatic lipogenesis and this process increases Insulin Resistance.

When a person eats a majority of their calories as carbohydrates, the metabolism becomes carb adapted. However, the disadvantage of carb adaptation is that one loses the ability to burn fat as fuel. As mentioned above, our liver can store only 100 grams of carbohydrates as glycogen.

We evolved over 2.5 million years as fat and protein eating animals. Thus, our pancreas that makes insulin is suited to function for ingestion of small amounts of carbohydrates. The carbohydrates were available as complex molecules that were covered with fiber and thus the rate at which they were absorbed was slow. Thus, our liver and metabolism did not have to deal with a rapid influx of large amount of sugar and carbs. The advent of agriculture perhaps is responsible for this large scale increase in carb intake. However, agriculture itself pales in comparison to the nutritional and metabolic load imposed on our liver by the food industry. They have succeeded in changing our diet to comprise a larger pie of processed and refined carbohydrates. Both of these so called advances are just a blip in time, in terms of human presence on earth. The short time frame during which the human body has had to adapt to carbs and sugar has not been enough for our genome to change and our physiology to adapt without metabolic consequences of diabetes, heart disease, dementia, cancers and strokes.

**Homeostasis of sugar (Regulation of sugar)**

The human body tries to maintain a normal level of blood sugar, which is only about a teaspoon in the entire blood of a normal human. When carbohydrates are consumed, especially ones that are readily absorbed (simple carbs), the pancreas releases insulin to drive the sugar in to the cells. These are muscle, brain and liver cells that take up the carbs. However, as we have seen we have a limited capacity to store carbohydrates that we do not use immediately. The glycogen tank (storage form of carbohydrates) get full early and the excess carbs that we have eaten are processed by the liver in to fat. Thus, contrary to what we would infer, fatty liver is often related to consumption of excess carbohydrates and not from fat consumption. There is an entity that is being commonly diagnosed called NASH, or non
alcoholic hepatosteatosis. This is fatty liver related to our Standard American Diet (SAD) that is heavy in the content of simple carbs.

A person on a 2000 calorie diet typically consumes about 50-70 percent of their calories as carbohydrates, which would be about 300-400 grams. This is a conservative estimate of carb intake by most accounts. In the typical situation, this level of carb consumptions occurs for decades. Since our physiology is not adapted to handling the load of carbs to this degree, the cells resist the influx this macronutrient, resulting in an increase the sugar levels in the blood. In order to maintain a normal blood sugar, the pancreas is called upon to work extra hard to produce more insulin to drive the sugar in to the cells. The cells in turn show resistance to the action of insulin.

It is important here to understand two aspects of diabetic pathophysiology:

1. The cells are resisting sugar intake because they are already full.
2. The pancreas is called upon to overproduce insulin to maintain blood sugar in normal range. However this is at a cost of higher insulin levels (Insulin Resistance)
3. High Insulin levels lead to obesity and loss of satiety signaling in the brain. This leads to hunger and over-eating despite obesity.
4. Eventually after decades of excess carbohydrate ingestion, the pancreas can no longer produce enough insulin to keep blood sugar in control (Pre-diabetes and diabetes).
5. Eventually the islet cells in the pancreas die and sugar levels go to dangerously high levels requiring exogenous insulin.

Type II diabetes is a disorder to insulin resistance and insulin overproduction as the root cause. The high blood sugar is a late manifestation diabetes. The treatment of this disorder should be to correct insulin resistance. This is best achieved by reducing the consumption of carbs, not by making the body produce more insulin or giving addition exogenous insulin to a person who is already resistance to the action of this hormone.

Yet most diabetologists to this day prescribe a sulphonyl urea (Glipizide is an example of this drug) to newly diagnosed diabetic. This is insane for the following reasons.

1. The pancreas is already in stress by over producing insulin and losing its capacity to keep up with the demand.
2. Excess insulin is already present in the blood stream.
3. High insulin levels are associated with heart disease, strokes and cancers
4. Burdening the pancreas to make more insulin kills the pancreas earlier - much like flogging a dead horse, which will function a bit better for a short while but gives up earlier. The studies have been done that demonstrate destruction of islet cells (Pancreatic cells that make insulin) with the use of Sulphonyl Urea medications.

Another major error by the medical profession is its abject failure to recognize the prevalence of Insulin Resistance. Since diabetes is diagnosed by high blood sugar and not by measuring Insulin Resistance, only the tip of the iceberg is visible. A person suspected of having diabetes is subjected to fasting blood sugar assay or to an oral glucose tolerance test, which is done with ingestion of about 50 to 100 mg of sugar solution and measuring glucose levels over time. However, as Dr Joseph Kraft pointed out, this is a late manifestation of diabetes when one consider that Insulin levels go up many years (perhaps decades) before the pancreas gives up on keeping blood sugar levels in normal range. During this decade of pre diabetes or diabetes in situ, high Insulin levels are raging their metabolic menace on the body, while a blissfully ignorant medical profession reassures the masses that everything is normal.

If a physician body is polled about a person who has normal fasting blood sugars and/ or a normal hgbaic (more on this later), most would not think of this person as having Insulin Resistance. They would not consider doing a glucose tolerance test in which not only sugar levels are measured are different time points after sugar ingestion but also obtain simultaneous insulin levels. This would be perhaps the best and only way in the majority of individuals to diagnose Insulin Resistance and Pre-diabetes.
Hemoglobin a1c is sugar coated hemoglobin. The sugar in the blood coats the proteins of the body and one such protein is hemoglobin, which not only carries oxygen but gives the blood it distinctive red color. Higher the sugar level in blood, more the coating of the hemoglobin. Since the blood cells have a life span of about 90-120 days, the hemoglobin a1c is a measure of a person’s average blood sugar over a 90 day period. A mistake made by physicians as mentioned above is to only diagnose pre diabetes when the hemoglobin a1c is above normal range (>5.6) and to diagnose diabetes when its value is over 6.3. As shown above, pre-diabetes starts with insulin overproduction and insulin resistance which pre dates the onset of high blood sugars by several years if not decades.

The best illustration of how wrong it is to diagnose pre diabetes and diabetes on the basis of blood sugar levels rather than insulin levels was done by Dr. Joseph Kraft. During his career of working as a pathologist at a Chicago Hospital, he had an opportunity to do 14,384 oral glucose tolerance tests. The major difference however was that he not only measured glucose levels but also obtained simultaneous insulin levels.

Of the 14,383 oral glucose tolerance tests that he had performed, about 9,598 had normal glucose levels. Yet as shown in the figure below, more than 75% of these 9598 patients with normal sugar levels who would have ordinarily been diagnosed as non diabetic had high insulin levels, indicating the presence of diabetes in situ or pre-diabetes. Thus, this metabolic abnormality is being vastly underestimated by an ignorant medical profession who is only too happy to continue prescribing diabetic medication without understanding the pathogenesis of the disease.

Figure - Kraft Insulin and Glucose Assay during oral glucose tolerance test (GTT)

Total number of patients – 14384
Number with abnormal GTT and Diabetes – 2011 of 14384
Number with abnormal GTT and Pre-diabetes – 2775 of 14384
Number with normal GTT – 9598 of 14385
Number with normal GTT along with normal Insulin Levels - 2112 of 9598*
Number with normal GTT but abnormal Insulin levels - 7102 of 9598*

*thus only 22% of patients with normal hgbaic has truly normal insulin levels, while the other 78% had high insulin levels consistent with pre-diabetes.

SGLT-2 inhibitors - (Empagliflozin)

As the blood passes through the filtering system of the kidney, not only are unwanted waste products eliminated but sugar is also leached in to the filtrate. However, before it becomes urine, there is a system in the kidneys, the so called sodium glucose transporter that re absorbs the sugar from the urine in exchange for salt which is eliminated. This process is efficient and effective to the degree that in a normal person, there is absence of sugar in urine because it is reabsorbed before it becomes urine. From the standpoint of a biologic system it would be foolish to get rid of an important nutrient like sugar, that the body can use. In a diabetic, the kidney filters more sugar in to the filtrate because the blood sugar is higher. In order not lose this nutrient, the kidney compensates by re-absorbing more sugar than a non-diabetic. This is a protective mechanism of disordered sugar and insulin metabolism, a friend in need and not one to get rid of. However, the pharmaceutical companies see an opportunity to create a drug to prevent the reabsorption of sugar. Such drugs have been available and are called the SGLT-2 inhibitors. They prevent the working of the sodium-glucose transport protein in the kidney tubules. This prevents the sugar from being reabsorbed by the kidney tubules which is then dumped in urine. This reduces blood
sugar levels and leads to an improvement in diabetes. However such a strategy is not without problems, namely:

1. Sugar is dumped in urine which increases the risk of infections - Urinary infections increase since the sugar is a substrate for bacteria.
2. Blood sugar is high for a reason, which is Insulin Resistance and the sugar is not getting in to the cells. Thus, unless this underlying mechanism is corrected, the body is going to try to maintain a higher than normal sugar levels.
3. In order to compensate for the loss of sugar in urine, the body jack up the internal de novo production of sugar by a process called “gluconeogenesis”. This is the term used to describe production of sugar by the body. Since the body cannot make sugar from fat cells, it burns muscle to make this sugar. Thus there is a potential loss of muscle mass with the use of SGLT-2 inhibitors.

The availability of several classes of diabetic medications is an excuse for the medical professions failure to utilize the three most effective strategies for treating and in several cases curing diabetes

1. Low carbohydrate, high fat diet - the emphasis is on high fat, not high protein, since highly processed protein also leads to insulin secretion by the pancreas albeit to a lesser degree.
2. A low carb diet also gives the pancreas a rest, since it is overworked with decades carb excess and its islet cell machinery which makes insulin is dying (so called islet cell destruction of type II diabetes). In some instances, the pancreatic islet cells recover and start producing insulin normally when they are rested with a low carb diet.
3. Fasting - One of the most effective methods of giving the pancreas a rest is to not eat at all. This is a very effective strategy to improve insulin resistance of diabetes. There are studies documenting the reduction in insulin levels and insulin resistance with fasting in the month of Ramadan. The practitioners of this religious fast, do not eat or drink from sunrise to sunset. During summer in North America, this duration is about 18 hours. Not eating for this long has been shown to improve pancreatic function. The medical fast however does not restrict consumption of water and black tea or coffee.
4. Exercise is another method, albeit far less effective than low carb high fat diet and fasting in alleviating insulin resistance.
Essential Fatty Acids 101

A fatty acid is a chain of carbon, oxygen and hydrogen atoms with a carboxyl group on one end. Fatty acids are classified on the basis of how many carbon atoms are in the chain, as well as how many double bonds exist within the molecule.

Fish contain a variety of fatty acids, but the ones that are believed to confer the majority of the benefits are the long-chain omega-3 fats eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). These omega-3 fats are found exclusively in seafood and marine algae.

As you can see from the chart above, it is also possible for the body to synthesize EPA and DHA from the short-chain omega-3 alpha-linolenic acid (ALA). ALA is found in plant foods such as flax, hemp and pumpkin seeds and walnuts.
However, research clearly indicates that the conversion of ALA to EPA and DHA is extremely limited. Less than 5% of ALA gets converted to EPA, and less than 0.5% (one-half of one percent) of ALA is converted to DHA.

A common misconception, especially amongst vegetarians and vegans, is that our need for EPA and DHA can be met by consuming flax oil and other plant sources of ALA. But the conversion numbers above clearly indicate that this isn’t the case.

Studies have shown that ALA supplements (like flax oil) are unable to raise plasma DHA levels in vegans, despite low DHA levels at baseline. So unless they are supplementing with an algae-derived source of DHA, it is likely that most vegetarians and vegans are deficient.

This is significant because researchers now believe that the majority of the health benefits we get from dietary omega-3 fats come from the longer chain derivatives (especially DHA, as I will explain below).

**Is DHA essential?**

In fact, some researchers have proposed that DHA is essential. When scientists label a nutrient as “essential”, they’re not just saying that it’s “very important”. In the context of nutrition essential means that the nutrient cannot be synthesized in the human body, and must be derived from dietary sources.

According to today’s nutrition textbooks, there are only two essential fatty acids, omega-6 linoleic acid (LA) and omega-3 alpha-linolenic acid (ALA). It is believed that as long as these fats are present in the diet, all of the longer-chain omega-3 and omega-6 derivatives can be synthesized in the body.

As I pointed out above, while this is theoretically possible, in reality the conversion doesn’t work well. This is true even for healthy people, but it’s especially true for those with nutrient deficiencies, because the conversion of ALA to DHA depends on zinc, iron and pyridoxine.

The bioavailability of iron in plant sources is poor compared to animal sources, so iron deficiency is common in vegans and vegetarians. This is another reason why they tend to be poor converters of ALA to DHA.

Several other observations support the hypothesis that DHA is essential:

- DHA content in the tissues of all mammals is very similar despite widely varying intakes of omega-3 fatty acids.
- DHA and AA, but not other omega-3 or omega-6 fatty acids, are selectively transferred across the placenta.
- 60% of the dry matter of the brain is lipid, and DHA and AA are the most abundant fatty acids of brain phospholipids.
- DHA status in newborns is much lower in those receiving formula with LA and ALA, than in those receiving milk or formula with pre-formed DHA.
It is possible that the primarily carnivorous diet of our ancestors, which ensured a consistently high dietary intake of DHA and AA, precluded the need to evolve efficient conversion mechanisms.

In other words, since we were eating a lot of meat and fish with pre-formed DHA and AA, our bodies didn’t need to be experts at converting ALA and LA in plants to DHA and AA. It is far easier for the body to assimilate pre-formed DHA and AA than it is to synthesize them from precursors.

**What about EPA? Isn’t it essential too?**

EPA is another long-chain omega-3 fatty acid that is conventionally believed to be responsible for the benefits of fish consumption.

EPA is often referred to as “anti-inflammatory”. However, according to this report on essential fatty acids by Masterjohn, EPA’s effect seems to be more of an interference with the metabolism of omega-6 arachidonic acid (AA) than the performance of any essential role itself.

Take a look at the chart again that I linked to in the beginning of the post. The fatty acids in blue boxes are less inflammatory, and those in pink boxes are more inflammatory. The chart shows that AA is used to synthesize prostaglandins that cause inflammation (indicated by the pink box on the chart). Because it has the same number of carbon atoms, EPA competes with AA for the enzymes that metabolize it. Since the prostaglandins made by EPA are less inflammatory than those made by AA (indicated by the blue box), EPA is often referred to as “anti-inflammatory”.

But while EPA is certainly less inflammatory than AA, it doesn’t make sense that the body would require an essential fatty acid just to block the inflammatory effects of another fatty acid.

By contrast, DHA is used to synthesize compounds that play an active role in resolving inflammation. EPA only makes these compounds in the presence of aspirin (PDF). EPA is thus likely to simply be a byproduct of compromised DHA synthesis.

**What does this mean to you?**

Putting all of this information together yields the following conclusions:

1. DHA is the most important of the omega-3 fatty acids, and is primarily responsible for the benefits we get from consuming them.
2. DHA is likely to be essential, which means that you must consume it in the diet to prevent disease and ensure optimal function.
3. The conversion of plant sources of ALA, such as flax seed oil, to DHA is poor in healthy people and even worse in people deficient in certain nutrients. Vegans and vegetarians are especially prone to be poor converters of ALA to DHA.
4. If you’ve been buying flax oil in the hopes that it will help, you’d be far better off putting that money towards some fish or fish oil capsules.

Dietary changes over the past century have lowered the DHA status to a state of subclinical deficiency in many people. Countless studies show that this deficiency is at least in part to blame for the rising incidence of cardiovascular disease, inflammatory disease, mental and psychiatric disorders and suboptimal neurodevelopment.
DHA is not the only reason to eat fish, which is also rich in selenium and vitamin D. However, DHA is likely to be the primary reason why populations that eat fish on a regular basis have consistently been shown to healthier than those that don’t. We’ll discuss this further in the next article.


**Omega-3 versus Omega-6 Fats**

Omega-3 fats are considered anti-inflammatory and also have anticoagulant properties. They dampen inflammation and thin the blood. Inflammation is considered to a major cause of degenerative diseases (heart disease, stroke, cancers, dementia and diabetes). On the contrary omega-6 fats are considered to be pro-inflammatory and also have pro-coagulant properties. The ratio of omega-6 to omega-3 consumption is not known definitively. Some experts suggest a ratio of 4:1 while others recommend a ratio of 1:1. The standard American diet however is very high in omega-6 and deficient in omega-3. The information below gives you approximate ratio of these two fats in different foods.

**FATS AND OILS (per 100g)**

**Fish Oils (average cod, halibut, mackerel, rockfish and salmon oils)**
- 1.2g LA (n-6)
- 0.9g ALA (n-3)
- 9.9g EPA (n-3)
- 12.8g DHA (n-3)

Fish Liver Oil (Atlantic Cod)
- 1.5g LA (n-6)
- 0.9g ALA (n-3)
- 8g EPA (n-3)
- 14.3 DHA (n-3)

Shellfish Oil (Pacific Oyster)
- 1.2g LA (n-6)
- 1.6g ALA (n-3)
- 21.5g EPA (n-3)
- 20.2g DHA (n-3)

**Nut and Seed Oils**

Cashew Oil
16g LA (n-6)
0.4g ALA (n-3)

Peanut Oil
29g LA (n-6)
1.1g ALA (n-3)

Pumpkin Seed Oil
51g LA (n-6)
0g ALA (n-3)

Sesame Seed Oil
42g LA (n-6)
0.5g ALA (n-3)

Sunflower Oil
53g LA (n-6)
0g ALA (n-3)

Coconut Oil
3g LA (n-6)
0g ALA (n-3)

Flaxseed Oil
15g LA (n-6)
55g ALA (n-3)

Olive Oil
9g LA (n-6)
0.7g ALA (n-3)

Avocado Oil
12.5g LA (n-6)
1g ALA (n-3)

Macadamia Nut Oil
1.5g LA (n-6)
1.5g ALA (n-3)

**Vegetable Oils**

Corn Oil
57g LA (n-6)
0.8g ALA (n-3)

Cottonseed Oil
<table>
<thead>
<tr>
<th>Food</th>
<th>LA (n-6)</th>
<th>ALA (n-3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Canola Oil</td>
<td>22g</td>
<td>11g</td>
</tr>
<tr>
<td>Soybean Oil</td>
<td>53g</td>
<td>7g</td>
</tr>
<tr>
<td>Walnut Oil</td>
<td>62g</td>
<td>4g</td>
</tr>
<tr>
<td>Wheat Germ Oil</td>
<td>54g</td>
<td>7g</td>
</tr>
</tbody>
</table>

**Animal Fats**

<table>
<thead>
<tr>
<th>Food</th>
<th>LA (n-6)</th>
<th>ALA (n-3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beef Tallow (grain-fed beef source)</td>
<td>4g</td>
<td>0.7g</td>
</tr>
<tr>
<td>Chicken Fat</td>
<td>17g</td>
<td>1.1g</td>
</tr>
<tr>
<td>Lard</td>
<td>10g</td>
<td>1.4g</td>
</tr>
<tr>
<td>Mutton Fat</td>
<td>5g</td>
<td>2.9g</td>
</tr>
</tbody>
</table>

**WHOLE FOOD SOURCES (per 100g)**

**Dairy**

<table>
<thead>
<tr>
<th>Food</th>
<th>LA (n-6)</th>
<th>ALA (n-3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cheddar cheese, natural</td>
<td>0.5g</td>
<td>0.4g</td>
</tr>
<tr>
<td>Cream cheese, regular</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
0.8g LA (n-6)
0.5g ALA (n-3)

Gruyere cheese, regular
1.3g LA (n-6)
0.4g ALA (n-3)

American cheese, regular
0.6g LA (n-6)
0.3g ALA (n-3)

Heavy Cream, conventional, grain-fed cows
0.9g LA (n-6)
0.6g ALA (n-3)

Light Cream, conventional, grain-fed cows
0.5g LA (n-6)
0.3g ALA (n-3)

Sour Cream, conventional
0.4g LA (n-6)
0.3g ALA (n-3)

Milk, whole, conventional
0.1g LA (n-6)
0.1g ALA (n-3)

Yogurt, plain, whole milk, conventional
0.1g LA (n-6)
0.1g ALA (n-3)

Egg Yolks, conventional (100g = approximately 4 yolks)
2.6g LA (n-6)
0.05g ALA (n-3)

Egg Yolks, pastured or flaxseed included in diet
4.2g LA (n-6)
2.1g ALA (n-3)

Butter, conventional
2.73g LA (n-6)
0.32g ALA (n-3)

Butter, grass-fed organic
1.8g LA (n-6)
1.2g ALA (n-3)
**Meats & Game**

Elk
0.343g (n-6)
0.056g (n-3)

Bison
0.156g (n-6)
0.026g (n-3)

Beef, grass-fed
0.139g (n-6)
0.052g (n-3)

Beef, grain-fed, conventional
0.275g (n-6)
0.016g (n-3)

**UPDATE - October 5, 2006**

Venison, roast, cooked, braised (approximate levels)
0.13g (n-6)
0.06g (n-3)