I-TREAT

DIABETES

I-TREAT - A Guide To Treatment of the metabolic syndrome, diabetes, obesity, heart disease, hypertension, inflammation, and aging.

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PREVENTION & TREATMENT PROTOCOLS FOR COVID-19

Disclaimer:
The treatment of metabolic syndrome and type II diabetes should always be under the supervision of a health care provider. As all the interventions suggested in this guidance will lower blood glucose levels, those patients taking diabetic medications need to have their medications adjusted (titrated) to avoid life threatening hypoglycemia. Blood glucose monitoring is absolutely critical especially during the induction phase and a continuous glucose monitor is recommended. This guideline should not be used in patients with type I diabetes who have an absolute insulin deficiency.

The metabolic syndrome includes high blood pressure, high blood sugar, excess body fat around the waist, and abnormal triglyceride and cholesterol levels.[1;2] In excess of 30% of adults in the United States meet the diagnostic criteria for the metabolic syndrome.[3] The metabolic syndrome increases a person's risk for type II diabetes, heart attack and stroke, and accelerates the aging process. Insulin resistance is the common factor underlying the metabolic syndrome and type II diabetes.[4;5] Furthermore, it is likely that insulin resistance plays a contributing role in the increasing risk of cancer. Insulin resistance occurs when cells in muscles, fat, and liver don't respond well to insulin (insulin resistance) and can't use glucose from the blood for energy. In an attempt to compensate for this problem (insulin receptor dysfunction), the pancreas makes more insulin resulting in high insulin levels. Over time the blood glucose levels rise with the eventual development of overt TYPE II diabetes. Insulin resistance precedes pre-diabetes and diabetes by many years. The damage from insulin resistance arises due to the combined harms of high blood glucose, high insulin levels and chronic inflammation (see below). High insulin levels and chronic inflammation (and not high cholesterol) likely underly the "pandemic" of coronary and cerebrovascular disease in Western Nations. The causes of insulin resistance are complex and poorly understood and include genetic factors, a high-calorie, high-sugar, high-fructose diet, abdominal (visceral) obesity and increased fat deposition in the liver (fatty liver) and chronic inflammation. Lipid accumulation in liver impairs hepatocyte function leading to further hyperglycemia through increased hepatic glucose production (gluconeogenesis). Furthermore, insulin resistance leads to increased hepatic lipogenesis potentiating hepatic lipid accumulation. Hepatic steatosis (fatty liver) may play a central role in insulin resistance.[4]

Increased fat mass (white adipose tissue) and abdominal obesity may be crucial to the development of insulin resistance as the incidence these two disorders correlate closely (see Figure 1). White adipose tissue (WAT) has previously been considered to be sites of fat storage solely. However, recent studies have established that WAT is an active tissue.[6-8] White fat cells can be located either in the subcutaneous region (subcutaneous fat) or inside the viscera (visceral fat). Subcutaneous fat is the appropriate place where fat 'should be stored' whenever it is needed. Conversely, fat cells located between organs is not an appropriate place for fat storage, and for this reason it is also called ectopic fat or visceral fat. Waist circumference is a reliable marker of visceral fat and hepatic lipid

storage. Critically, WAT produces several inflammatory molecules, called adipokines, which are cytokines produced by fat cells.[6-8] There is an exponential relationship between WAT cells size and amount of adipokines produced. Furthermore, visceral fat produces 4 to 10 times more inflammatory adipokines compared to subcutaneous fat cells of same size. The production of adipokines and chronic inflammation plays a major role in the development of insulin resistance. Visceral fat produces a systemic pro-inflammatory environment (chronic inflammation) which alters the function of every organ system including the brain, heart, pancreas, kidneys, and gastrointestinal tract.

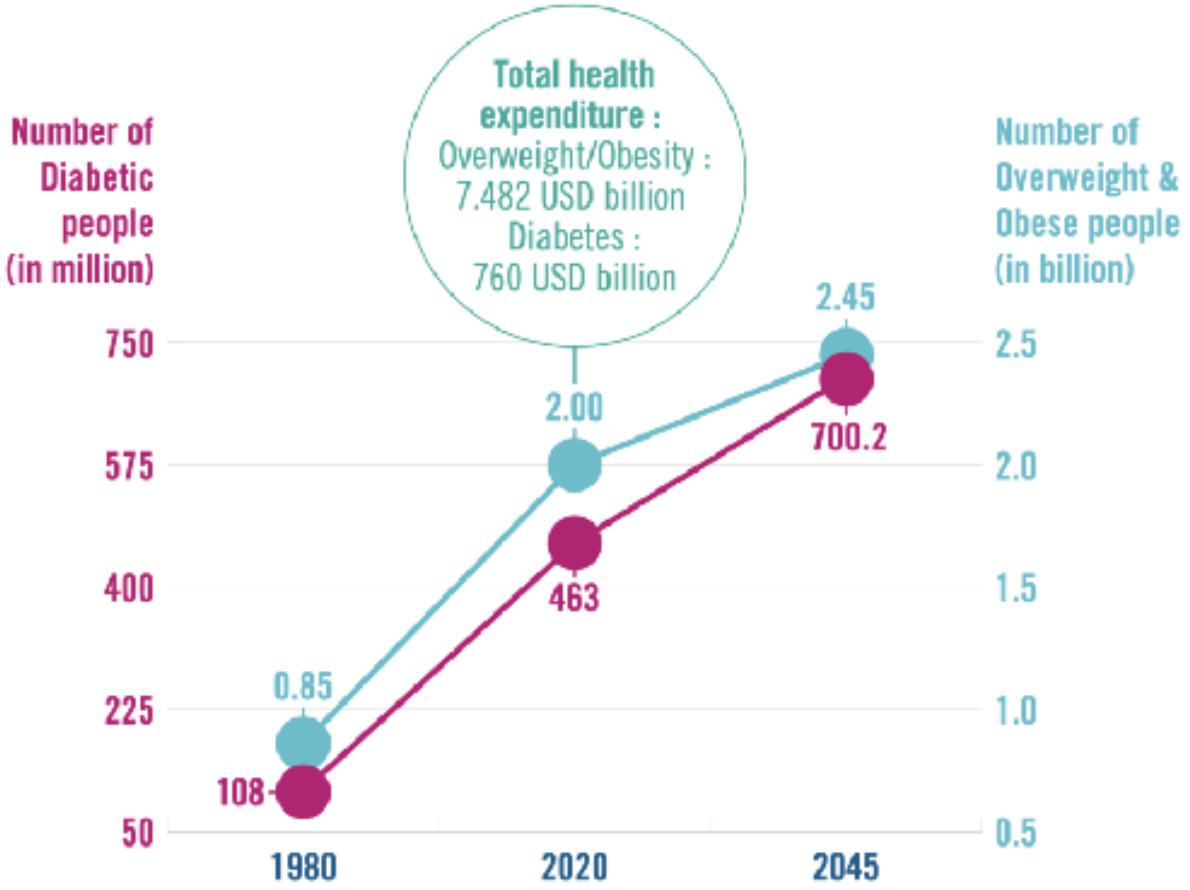
Insulin resistance has emerged in the last 50 years as the commonest disorder on this planet and the single largest cause of loss of life. Currently over 30% of Americans are insulin resistant with that number likely to increase substantially over time (See figure 1).[3] Insulin resistance is a metabolic disorder caused in large part by the "Modern Western lifestyle", namely the excessive consumption of carbohydrates (glucose/fructose), processed foods and polyunsaturated vegetable oils. While there is likely a genetic predisposition to insulin resistance, this is largely a disease of modern lifestyle with poor lifestyle choices and poor eating habits. It is important to note that human genetics evolved over a period of 2.5 million years. Paleolithic-Neolithic man/woman were largely hunters and gathers (had no access to supermarkets and process foods); they usually ate once a day a meal consisting of saturated fats (animal protein), vegetables, and fruits. While the merits of a vegetarian/vegan diet are widely debated, humans have a simple stomach (mono-gastric) with a relatively long small bowel and short caecum and are not designed to ferment an exclusive plant-based diet. Ruminants have either a multi-compartment stomach (fore gut fermenters) or a large complex caecum (hind gut fermenters).[9;10] Furthermore, our forefathers were exposed to sunshine (near infra-red radiation) while artificial light (light emitting diodes) and IR filtering glass did not exist. Lack of exposure to sunlight is associated with an increased all-cause mortality.[11] The modern Western lifestyle has occurred over the last 50-100 years (one or two generations) and our genes have not had enough time to adapt to this new environment.

Big Pharma and the medical establishment have propagated the myth that type two diabetes is a chronic progressive disease that can't be cured and that the primary goal of treatment is lowering blood glucose with a combination of patented medications. Dr Jason Fung considers these to be the two great lies of medicine. [12] As is evident from this guideline insulin resistance and type II diabetes is actually quite easy to reverse largely by adopting a healthy lifestyle.

Diagnostic criteria of the Metabolic Syndrome (and insulin resistance) [1;2]

- A waistline over 40 inches in men and 35 inches in women
- Blood pressure readings of 130/80 mmHg or higher
- A fasting glucose level over 100 mg/dL (5.6 mmol/L)
- A fasting triglyceride level over 150 mg/dL (1.7 mmol/L)
- A HDL cholesterol level under 40 mg/dL (1.0 mmol/L) in men and 50 mg/dL (1.3 mmol/L) in women

Figure 1. Worldwide Incidence of Obesity and Diabetes



Risk Factors for the Metabolic Syndrome/ Insulin Resistance

- Diet high in carbohydrates
- Obesity, especially abdominal (belly) fat
- Inactive lifestyle
- Gestational diabetes (impaired glucose tolerance during pregnancy)
- Polycystic ovarian syndrome (PCOS)
- Health conditions like nonalcoholic fatty liver disease and polycystic ovary syndrome
- A family history of Type II diabetes
- Smoking
- Long COVID syndrome/Covid-19 vaccine injury
- Ethnicity -- it's more likely if your ancestry is African, Latino, or Native American
- Age -- it's more likely after 45 years of age.
- Sleep problems like sleep apnea

Testing for Insulin Resistance

- **Fasting plasma glucose test**.[13] This test measures your blood sugar after you haven't eaten for at least 8 hours. A normal fasting plasma glucose is between 70 mg/dL (3.9 mmol/L) and 100 mg/dL (5.6 mmol/L). A fasting blood glucose of greater than 126 mg/dL (7 mmol/L) on two separate occasions is considered diagnostic of diabetes.
- **Hemoglobin A1c test (A1c)**. [13]This blood test shows your average blood sugar level for the past 2 to 3 months. Doctors use it to diagnose prediabetes or diabetes. If you have diabetes, it helps show whether it's under control.
    - The normal range for the hemoglobin A1c level is between 4% and 5.6%.
    - A1c levels between 5.7% and 6.4% is indicative of prediabetes.
    - An A1c of 6.5% or is diagnostic of diabetes. The target A1c level for people with diabetes is usually less than 7%
- **A serum triglyceride** > 150 mg/dl (based on a fasting lipid profile). A TG between 150 and 199 mg/dl is considered borderline elevated while a TG > 200mg is regarded as high.
- **A low HDL.** A low HDL is considered **l**ess than 40 mg/dl (1.0 mmol/L) in men and less than 50 mg/dl (1.3 mmol/L) in women. A desirable HDL > 60mg/dl (1.6 mmol/L). Saturated fats increase HDL.
- **TG/HDL ratio** The single best predictor of coronary artery disease is the TG/HDL ratio and NOT the total cholesterol level or LDL level. [14-16] Ideally, you want **no more than a 2:1** ratio of triglycerides to HDL cholesterol. So, if your triglycerides are 100 mg/dl, your HDL cholesterol should be 50 mg/dl.

A treatment guide for the management of insulin resistance and diabetes.

The single most important intervention to reverse insulin resistance is adopting a nutrient dense healthy pattern of eating which includes, low carbohydrates (high fat), avoidance of all processed foods, time restricted eating and the avoidance of polyunsaturated vegetable oils.

- Intermittent fasting/time restricted eating (see guidance below). Time restricted feeding has number beneficial metabolic, cellular, and immunologic benefits. Humans did not evolve to eat and snack continuously; this is a highly maladaptive human behavior. Time Restricted Eating is the most efficient and effective way to lower insulin levels and restore insulin sensitivity. In addition, fasting has a profound effect on promoting immune system homeostasis, partly by stimulating the clearing of damaged cells (autophagy), damaged mitochondria (mitophagy), and misfolded and foreign proteins. Fasting also improves mitochondrial health and increases stem cell production. Time restricted feeding is the most effective method to achieve sustained weight loss; one should aim for a healthy weight.
- A low carbohydrate diet (ketogenic diet) high in saturated fat, mono-unsaturated and omega-3 fatty acids. The carbohydrate content of a meal should not exceed 25 grams.
- Berberine 1000-1500 mg/day (500 mg BID/TID or 600 mg BID). Berberine is a quaternary ammonium salt from the protoberberine group of isoquinoline alkaloids found in a number of different plants.[17] Berberine is the main active component of an ancient Chinese herb *Coptis chinensis French*, which has been used to treat diabetes for thousands of years.[17;18] This remarkable herb Berberine has been shown to regulate glucose and lipid metabolism.[17] Berberine lowers blood glucose by both insulin dependent and insulin independent mechanisms (see Figure 2). Berberine increases glucose dependent insulin release from the pancreas, increases insulin receptor expression, increases glycolysis, inhibits hepatic gluconeogenesis and alter hepatic gene expression.[19-24] Furthermore, berberine increases hepatic fatty acid oxidation,[25] and decreases hepatic steatosis, [21] which may be a central pathogenic factor causing insulin resistance.[4] In addition, berberine has potent anti-inflammatory activity and modulates the microbiota thereby reducing insulin resistance.[26-28] Multiple studies have demonstrated that berberine significantly reduces fasting blood glucose, postprandial blood glucose, HbA1c and plasma triglycerides.[18;29;30] In addition, berberine decreases fasting plasma insulin and HOMA-IR as well as total and LDL cholesterol (LDL-C), blood pressure and BMI while increasing HDL cholesterol. In essence this remarkable herb treats/normalizes the entire metabolic syndrome. The metabolic effects of berberine are detectable within a week of initiation of treatment.[18] As an additional bonus, berberine has anticancer effects.[31;32] This herb is remarkable safe; the only adverse events include transient gastrointestinal symptoms (diarrhoea, flatulence). As insulin release is glucose dependent hypoglycemia has not been reported with this herb.[19] Berberine appears to act synergistically with metformin. [18] In summary, berberine is a potent oral hypoglycemic agent with beneficial effects on lipid metabolism. It is a safe and inexpensive treatment that should be included in the treatment approach of all patients with insulin resistance/metabolic syndrome. As berberine lowers blood glucose and lowers blood pressure these parameters should be monitored. Once metabolic stability is achieved it may be possible to reduce the

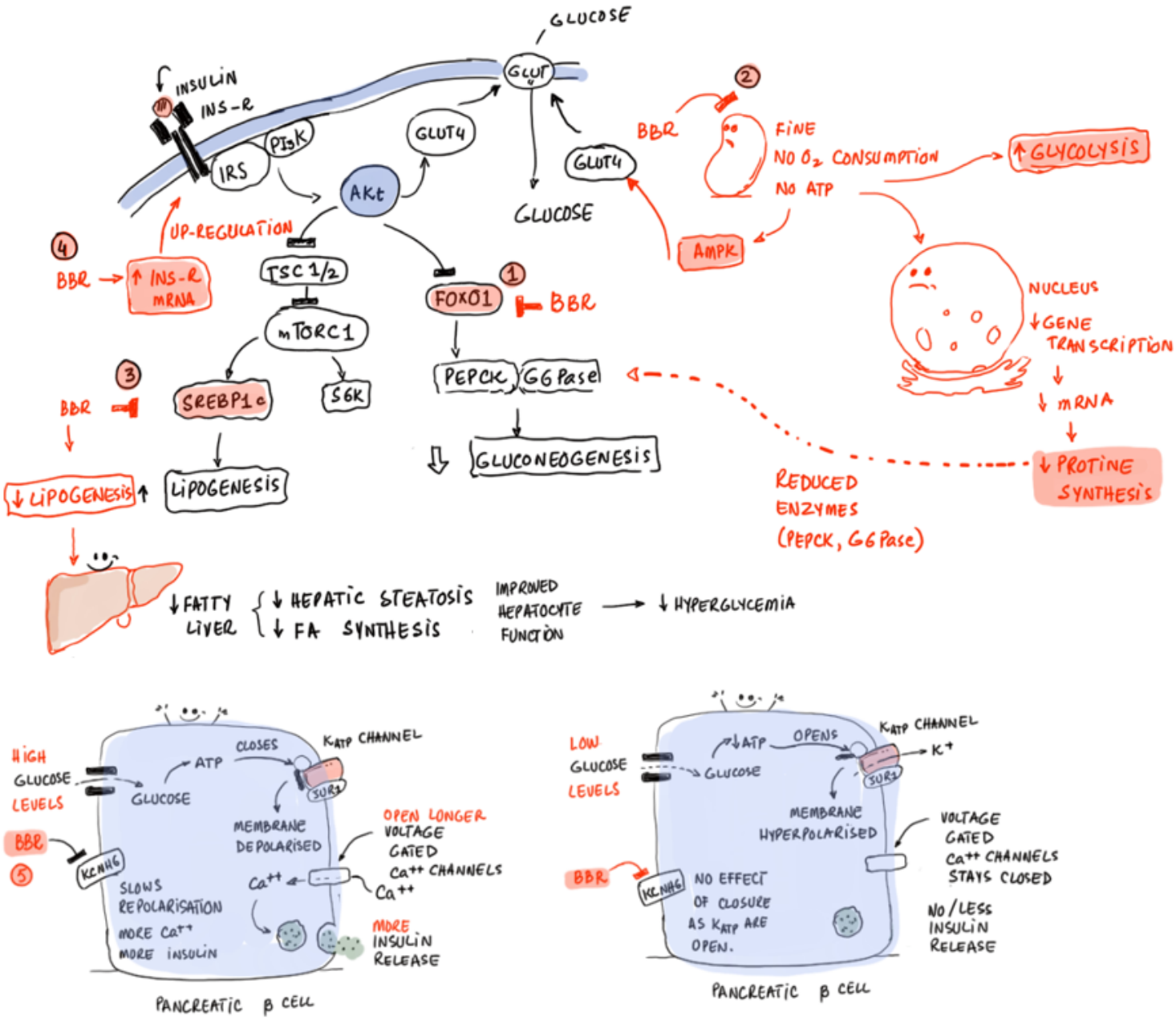
dose of berberine to 500mg once or twice daily. Berberine should not be taken in patients taking cyclospine as this combination will increase cyclosporine levels (absolute contraindication). Berberine may alter the metabolism of the following drugs which should be used with caution (monitor effects): anticoagulants, dextromethorphan, tacrolimus (Prograf), phenobarbitone and sedative drugs (see https://www.webmd.com/vitamins/ai/ingredientmono-1126/berberine). Berberine is contraindicated during pregnancy, breast feeding and in neonates and children.

- Metformin 500 -1000 mg twice daily. Metformin has been used for over 60 years and is the most widely used drug for the treatment of type II due to its efficacy, safety, and low cost. Metformin is considered first-line therapy for patients with type II diabetes according to the American Diabetes Association/European Association for Study of Diabetes.[33] Metformin works by decreasing intestinal glucose absorption, improving peripheral glucose uptake, lowering fasting plasma insulin levels and increasing insulin sensitivity, which result in a reduction of blood glucose concentrations without causing overt hypoglycemia.[34] In recent years, evidence has developed suggesting additional benefits of metformin due to its antitumor effect, antiaging effect, cardiovascular protective effect, neuroprotective effect and for the treatment of polycystic ovary syndrome. [34-36] The dose of metformin will likely need to be reduced in type II diabetics as insulin resistance improves during the induction phase. While metformin has been considered the drug of first choice in patients with type 2 diabetes, berberine appears to be equally (if not more) effective as metformin in the treatment of the metabolic syndrome. It is likely metformin and berberine act synergistically to improve indices of the metabolic syndrome (when either one alone is not sufficient). However, a small study in elderly men demonstrated that metformin attenuated the increase in insulin sensitivity and VO_2 max after aerobic exercise training.[37] However, a similar study demonstrated that circular exercise and berberine has additive effects on cardiovascular risk factors.[38] This limited data suggest that berberine may be preferred over metformin in patients participating in aerobic exercise.
- Magnesium 100-400 mg daily**.** Magnesium has been demonstrated to reduce insulin resistance. [39;40] There are at least 11 different types of magnesium that can be taken in supplement form with varying bioavailability. Generally, organic salts of Mg have a higher solubility than inorganic salts and have greater bioavailability. [41] Magnesium oxide and magnesium citrate compounds, commonly prescribed by physicians, have poor bioavailability. [42] Magnesium Malate, Taurate, Glycinate, and L-threonate have good bioavailability and will readily increase RBC magnesium levels. A starting dose of 100 to 200 mg daily is suggested, increasing the dose as tolerated up to 300 mg (females) to 400 mg daily.
- Melatonin 2 -10 mg slow release/extended release at night (dose as tolerated). Several lines of evidence suggest that melatonin may play a role in glucose metabolism. In vitro, prolonged exposure of β islet cells to melatonin increases β-cell glucose sensitivity.[43] Polymorphisms in the melatonin receptor type 1B gene are associated with increased fasting glucose, HbA1C as well as gestational and type 2 diabetes.[44] Melatonin has been demonstrated to improve hepatic insulin resistance and hepatic steatosis through reduction in ER stress.[45] Higher

nocturnal melatonin secretion has been demonstrated to be inversely associated with insulin levels and insulin resistance.[46] Finally, a meta-analysis of 16 RCT's (dose of 3 -10mg at night) demonstrated that melatonin significantly reduced fasting blood glucose, HbA1C and insulin resistance when compared to placebo.[47]

- Resveratrol 400-500 mg daily. Resveratrol is a plant phytochemical (flavonoid) that has remarkable biological properties. [48-50] Most importantly it activates autophagy. [51;52] Resveratrol may potentiate the effect of time restricted feeding (intermittent fasting) in activating autophagy. Resveratrol should therefore be taken during fasting and not with a meal. In addition, resveratrol may independently improve insulin resistance. [53]
- Cinnamon 1-2g/day. Cinnamon is one of the major herbs used in traditional Chinese medicine. Preparations containing the bark of Cinnamon have been prescribed for more than 2000 years for the treatment of fever, the common cold, inflammation, diarrhoea and pain.[54] In vitro and in vivo studies on cinnamon extracts or its components (mainly cinnamaldehyde) demonstrate that these substances exhibit a wide variety of pharmacological effects, including antifungal, anticancer, antiinflammatory, antidiabetes, antiviral, antihypertensive, antioxidant, as well as being cardioprotective and improving the lipid profile.[54] The glucose lowering effect of cinnamon is postulated to be due to increasing insulin release, enhancing insulin sensitivity, and regulation of protein-tyrosine phosphatase 1B (PTP1B) and insulin receptor kinase. [55] While cinnamon has been demonstrated to reduce fasting blood glucose and serum triglyceride concentration the effects on HbA1C and LDL cholesterol have been less dramatic.[55] Cinnamon may have a role in the management of insulin resistance when combined with berberine.
- Omega-3 fatty acids. Patients with insulin resistance and type II diabetes are at increased risk of cardiovascular disease (CVD). Omega-3 fatty acids have anti-inflammatory and cardioprotective effects. A recent meta-analysis demonstrated that omega-3 fatty acid supplementation lowers the risk for myocardial infarction, coronary heart disease (CHD) death, total CHD, cardiovascular (CVD) death and total CVD.[56] Based on this data we suggest a combination of EPA/DHA with an initial dose of 1 g/day (combined EPA and DHA) and increasing up to 4 g/day of the active omega-3 fatty acids.
- Probiotics with Bifidobacterium. Intestinal dysbiosis is associated with obesity. While animal studies reproducibility demonstrate improved insulin resistance with probiotics, clinical studies have been more heterogeneous.[57] However, a RCT demonstrated that Bifidobacterium reduced anthropometric markers of obesity.[58] Furthermore, a cross-over, double-blind, randomized control trial demonstrated that supplementation of Bifidobacterium reduced blood sugar and improved insulin resistance. [59]
- Avoid excessive stress which increases cortisol and catecholamines which increase blood sugar levels. Consider stress mitigating strategies.
- Exercise. Go for at least 30 minutes a day of moderate activity (like brisk walking) 5 or more days a week. If you're not active now, work up to that. Avoid excessive endurance exercise which increases cortisol levels and worsens insulin resistance.

Figure 2. Insulin dependent and independent mechanism of berberine on lowering blood glucose.



Legend:1. Berberine (BBR) reduces the transcription factors including Forkhead transcription factor O1 (FoxO1) resulting in reduced synthesis of phosphoenolpyruvate carboxykinase (PEPCK) and Glucose 6 Phosphatase (G6Pase) leading to reduced gluconeogenesis. 2. BBR reduces the activity of the complex I of the electron transport chain in mitochondria decreasing production of ATP which leads to more glucose breakdown (glycolysis) and to activation of AMPK. AMPK activation causes more GLUT4 channels to be inserted in the cell membrane leading to more glucose uptake reducing hyperglycemia.3. Reduction SREBP1 results in reduced synthesis of fatty acid synthase with reduced fatty acid production and reduced hepatic steatosis. 4. Berberine upregulates the gene expression for the insulin receptors. 5. Berberine is insulinotropic in high glucose environment and not in the low glucose setting. Hence has no potential to cause hypoglycemia.

Benefits of Intermittent Fasting

- Most efficient method to improve insulin sensitivity and lower blood glucose.
- Induces weight loss and loss of body fat; improves fat oxidation.
- Deceases inflammation
- Increases growth hormone (maintains lean body mass
- Basal metabolic rate remains stable or increases.
- Lowers blood cholesterol.
- Improves memory and mental clarity.
- Reduces the risk of Alzheimers disease and other neurodegenerative diseases.
- Reverses aging and prolongs health-span.

Guide to intermittent fasting/time restricted eating.

- Intermittent fasting/time restricted eating does not mean the patient should starve or severely restrict caloric intake.
- A number of intermittent fasting plans can be adapted and modified to best suit the patient's lifestyle. The excellent book by Dr Jason Fung provides guidance on approaches to intermitte4nt fasting. [12]However, time restricted eating appears to be an effective and practical approach. For timed fasting, begin slowly: start with a 12-hour eating window 5 days a week and reduce weekly to an 8-hour eating window 7 days a week. This eating window can be shortened to 4 hours or less over time. The ideal is a 1-2 hour eating window restricted to one meal a day. Timed fasting can be interspersed with 36- to 48-hour fasts. Premenopausal women appear less tolerant to time restricted eating and should therefore restrict the time-based eating window slowly.
- Don't eat (or snack) within 3-4 hours of going to bed.
- Time restricted eating is best coupled with a low carbohydrate diet.
- Eat real foods and not processed foods.
- Keep meals diverse and include green and coniferous vegetables.
- Avoid fruit juices.
- To prevent large excursions of blood sugar, avoid high glycemic index foods (see below)
- No snacking
- Don't calorie count or obsess in regard to eating and food choices.
- A continuous glucose monitor (CGM) is strongly recommended during the initiation phase of time restricted feeding and until metabolic stability is achieved (e.g., Abbott Freestyle Libre 3). The glucose response to various foods is highly variable; CGM allows the individual determination of the glucose response to a particular food group.

The Glycemic Index

The glycemic index is a value assigned to foods based on how quickly and how high those foods cause increases in blood glucose levels. The glycemic index ranks food on a scale from 0 to 100. Pure glucose is arbitrarily given a value of 100, which represents the relative rise in the blood glucose level after two hours. The glycemic index of a specific food depends primarily on the quantity and type of carbohydrate it contains. Foods low on the glycemic index (GI) scale tend to release glucose slowly and steadily. Foods high on the glycemic index release glucose rapidly. It should be noted that the glycemic index varies between individuals.[60;61] A CGM allows for the individual assessment of the glucose excursion (glycemic index) of various foods.

Figure 3. The blood glucose profile of high and low glycemic index foods.

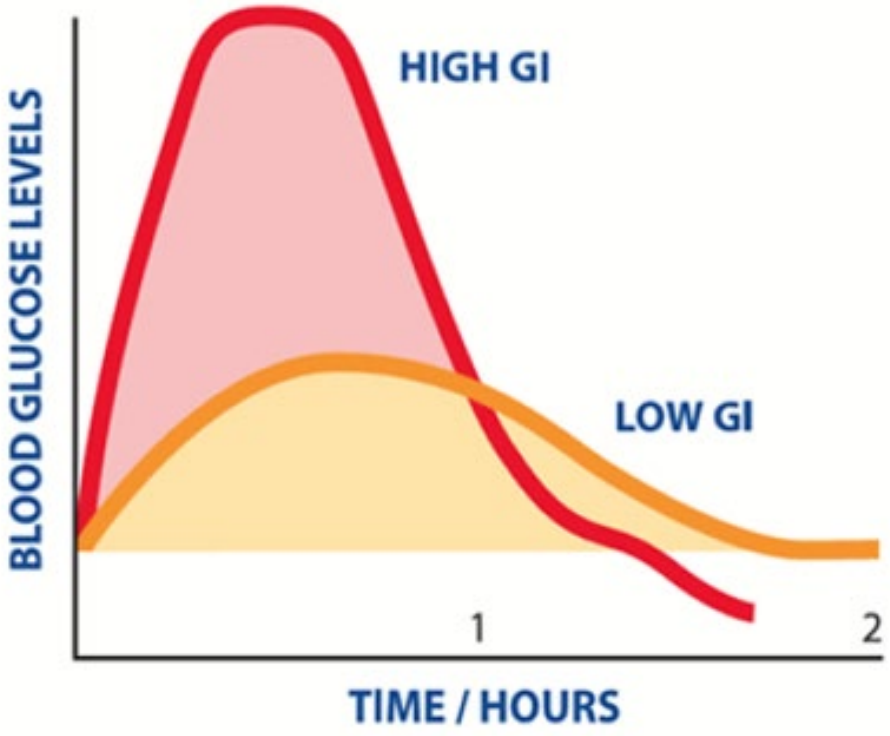


Table 1. Glycemic Index of select foods.

Food Item	Glycemic Index
White Rice	87
White bread	75
Watermelon	76
Orange juice	53
Banana	51
Pineapple	66
Papaya	60
Grapes	46
Oranges	42
Strawberries	40
Apples	34
Grapefruit	25
Fresh berries	25
Most vegetables	< 20
Peanuts	7

Who should not fast?

- Contraindicated
    - Those who are malnourished or underweight (BMI < 20 kg/M^2)
    - Those with anorexia nervosa/bulimia
    - Patients with type 1 diabetes (true insulin deficiency)
    - Children < 18 years of age
    - Pregnant women
    - Breastfeeding women
- Caution- under the supervision of a health care provider
    - Type 2 diabetes (will likely have to adjust diabetic medication)
    - Those with chronic diseases taking multiple medications.
    - Gout

Healthy eating habits

- Eat only at the table.
- No eating at the computer
- No eating in the car
- No eating on the couch
- No eating in bed
- No eating in the lecture hall
- No eating in front of the TV
- No artificial sweeteners and no sodas

The ten best things to eat.

- All vegetables (especially avocado, cruciferous, and leafy vegetables)
- Nuts – almonds, brazil nuts , cashew, and pistachio.
- Peanut butter and chia seeds
- Fish, especially Alaskan salmon, sardines
- Chicken breast (free range, no hormones, no antibiotics)
- Eggs
- Meat (grass fed, no hormones) avoid processed meats.
- Blueberries (limit volume if highly insulin resistant)
- Grapefruit (limit volume if highly insulin resistant)
- Coffee (with heavy cream or coconut oil; Stevia, no sugar or artificial sweetener)

The ten Worst Things to eat.

- Donuts
- Bagels, bread, pretzels, tortillas
- Cookies, muffins, baked products
- Chips
- French fries
- Rice and pasta
- Potatoes
- Canned fruits/fruit juices
- Low fat yogurt (sweetened)
- Watermelon and Bananas

Avoid seed oils high in Linoleic acid (omega-6-PUFA)

Seed Oils

- Soybean oil
- Corn oil
- Cottonseed oil
- Sunflower oil
- Sesame oil
- Grapeseed oil
- Safflower oil
- Rice bran oil
- Margarine

Non-seed oils/ALA seed oil (these are healthy oils; use only high-quality products and check production and expiration dates)

- Olive oil (oleic acid- omega-9 MUFA; do not fry due to low thermal point)
- Avocado oil (oleic acid- omega-9 MUFA)
- Coconut oil (medium chain fatty acid
- Flaxseed oil (alpha-linolenic acid -ALA omega-3)
- Rapeseed/Canola oil (MUFA and ALA)
- Walnut and Pecan oils (but should be refrigerated)
- Butter (saturated fat)

Myths and things to avoid.

- The Cholesterol-Saturated fatty acid hoax. [62-64] Beginning in the 1960's, Dr. Ancel Keys popularized the notion that saturated fats and high cholesterol were the primary causes of atherosclerotic heart disease, the so-called Diet-Heart Hypothesis.[65;66] This concept has been vigorously studied, including many randomized controlled trials and has been convincingly proven to be false. [62;67;68] Indeed, replacing vegetable oils (linoleic acid) for a diet high in saturated fatty acids was associated with higher rates of death, cardiovascular and coronary heart disease as well as a significant increased risk of cancer. [69] Cholesterol in the diet has virtually no effect on total cholesterol levels. Furthermore, the notion that reducing total cholesterol reduces the risk of coronary heart disease (with few exceptions, i.e. recent myocardial infarction, primary hypercholesterolemia) is not supported by the literature.[62;70;71] Despite these findings dieticians, cardiologists and indeed almost all physicians continue to propagate this hoax. Cholesterol does not cause heart disease; the real causes of heart disease are insulin resistance, inflammation and increased oxidative injury.
- Avoid Statins. Big Pharma (supported by the Medical establishment) initiated and propagated the Cholesterol-Statin hoax; with statins amongst the most widely prescribed of all drugs in Western Nations. A Meta-analysis of 11 primary prevention randomized controlled trials Involving 65 229 Participants demonstrated that statins were no better than placebo in reducing all-cause mortality in high risk patients.[72] It should be noted that cholesterol is an important component of the cell membrane and a precursor of many hormones. The brain is particularly rich in cholesterol (about a quarter of total cholesterol) and is essential for brain function. Cognitive dysfunction is a common side effect in patients on statins. In addition, statins interfere with insulin receptor function and glucose transport increasing the risk of developing diabetes.
- Avoid vegetable/seed oils. These process oils are high in omega-6 PUFA (see above). These fatty acids are poorly oxidized to acetyl-CoA and the production of energy. They are stored in the liver and incorporated into cellular and subcellular membranes. These fatty acids autoxidize and create lipid peroxidation products and reactive oxidative species that are linked to an increased risk of cancer. In addition, omega-6 PUFA are pro-inflammatory. As stated above vegetable oils DO NOT reduce cholesterol nor the risk of cardiovascular disease.

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