I-CARE℠

INSULIN RESISTANCE TREATMENT

A guide to managing insulin resistance, metabolic syndrome, and diabetes

February 2023

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Disclaimer

The treatment of metabolic syndrome and type II diabetes should always be done under the supervision of a qualified healthcare provider.

As all the interventions suggested in this guidance will lower blood glucose levels, patients who take diabetic medications need to have their medications adjusted (titrated) to avoid life-threatening hypoglycemia. Blood glucose monitoring is 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.

What is metabolic syndrome?

The National Institutes of Health defines metabolic syndrome as "a group of conditions that together raise your risk of coronary heart disease, diabetes, stroke, and other serious health problems." Metabolic syndrome is also called insulin resistance syndrome.

Metabolic syndrome includes conditions like high blood pressure, high blood sugar, excess body fat around the waist, and abnormal triglyceride and cholesterol levels. [1;2] More than 30% of adults in the United States meet the diagnostic criteria for metabolic syndrome. [3]

This condition increases a person's risk for type II diabetes, heart attack, and stroke, and also accelerates the aging process.

Insulin resistance is the common factor underlying metabolic syndrome and type II diabetes. [4;5] It is also likely that insulin resistance plays a role in increasing a person's risk of cancer. [1-5]

Insulin is a hormone that turns food into energy and controls blood sugar. Insulin moves glucose (sugar) into cells, which use it for energy. Insulin resistance occurs when cells in muscles, fat, and liver don't respond well to insulin and can't use glucose from the blood for energy. In an attempt to compensate for this problem, which is also known as insulin receptor dysfunction, the pancreas makes more insulin. This causes blood sugar levels to rise and can lead to type II diabetes.

Most people who have metabolic syndrome also have insulin resistance. Insulin resistance precedes pre-diabetes and diabetes by many years. Metabolic syndrome may thus signal the development of type II diabetes.

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, rather than high cholesterol, are likely what underlies the "pandemic" of coronary and cerebrovascular disease in many western nations.

What causes insulin resistance?

The causes are complex and poorly understood. They include genetic factors, a diet high in calories, sugar, and fructose, abdominal (visceral) obesity, increased fat deposition in the liver (fatty liver), and chronic inflammation. The accumulation of fat in the liver causes the liver to make more glucose (gluconeogenesis), which further increases blood glucose levels. Furthermore, insulin resistance leads to increased hepatic lipogenesis potentiating hepatic lipid accumulation. 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 of these two disorders correlate closely (see Figure 1). White adipose tissue (WAT) has previously been considered to solely be a site of fat storage. 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, storing fat between organs is not appropriate and for this reason, it is also called ectopic or visceral fat.

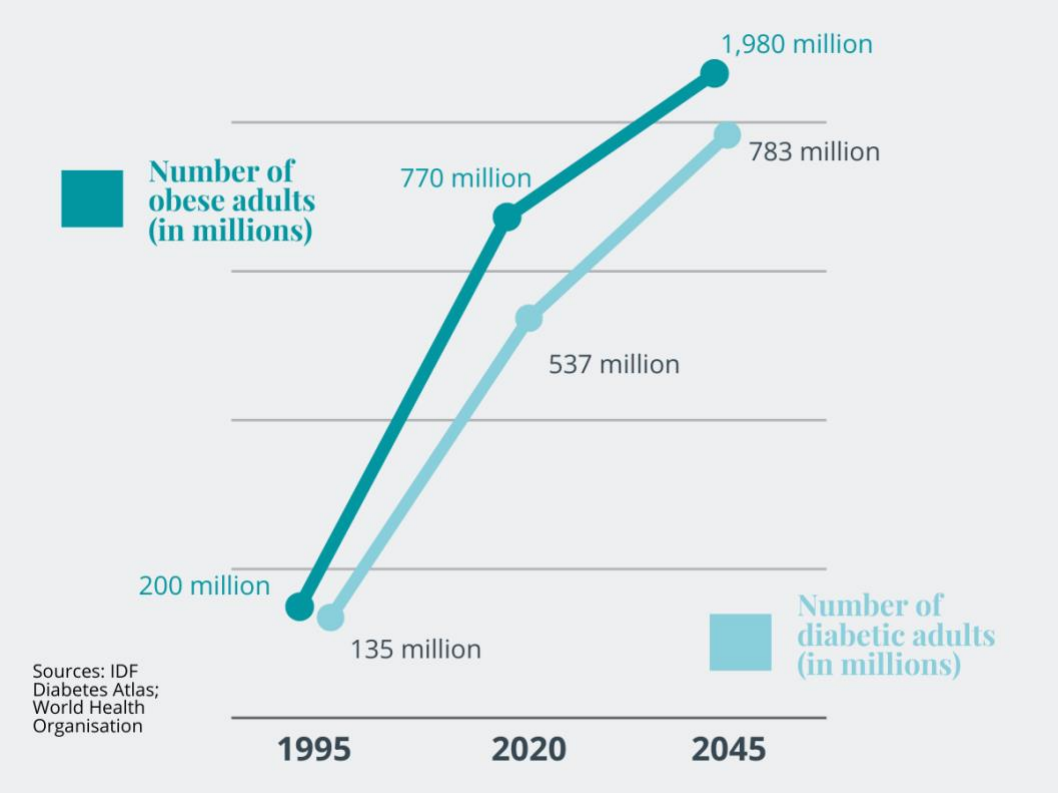
Waist circumference is a reliable marker of visceral fat and fatty liver. Critically, WAT produces several inflammatory molecules, called adipokines, which are cytokines produced by fat cells. [6-8] There is an exponential relationship between WAT cell size and the amount of adipokines produced. Furthermore, visceral fat produces 4 to 10 times more inflammatory adipokines compared to subcutaneous fat cells of the same size.

The production of adipokines and chronic inflammation play 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.

Recent estimates put the global prevalence of metabolic syndrome at about one-quarter of the world's population. [9]

Insulin resistance has emerged in the last 50 years as the most common disorder on this planet and the single largest cause of loss of life. [1-5] Currently, more than 1 in 3 Americans are insulin resistant and that number is likely to increase substantially over time (See figure 1). [3]

Figure 1. Worldwide incidence of obesity and diabetes (1980-2020, projected to 2045



Insulin resistance is largely caused by our modern western lifestyle, namely excessive consumption of carbohydrates (glucose/fructose), processed foods, and polyunsaturated vegetable oils. While there is potentially a genetic predisposition to insulin resistance, this is largely a disease of poor lifestyle choices and poor eating habits.

Human genetics have evolved over a period of 2.5 million years. The modern western lifestyle has occurred only over the last 50-100 years (three to five generations). Our genes have not had enough time to adapt to this new environment.

Our paleolithic-neolithic ancestors were hunters and gatherers who usually ate once a day. Their meal consisted 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 not designed to ferment an exclusively plant-based diet. [10;11]

Furthermore, our forefathers were exposed to sunshine (near-infrared radiation), while our modern lives are filled with artificial light (light-emitting diodes) and infrared-filtering window glass. Lack of exposure to sunlight is associated with increased all-cause mortality. [12]

Big Pharma and the medical establishment have propagated the myth that type II diabetes is a chronic progressive disease that cannot be cured; they say the primary goal of treatment is to lower

blood glucose with a combination of patented medications. Dr. Jason Fung, a nephrologist and functional medicine advocate, considers these to be the two great lies of medicine. [13]

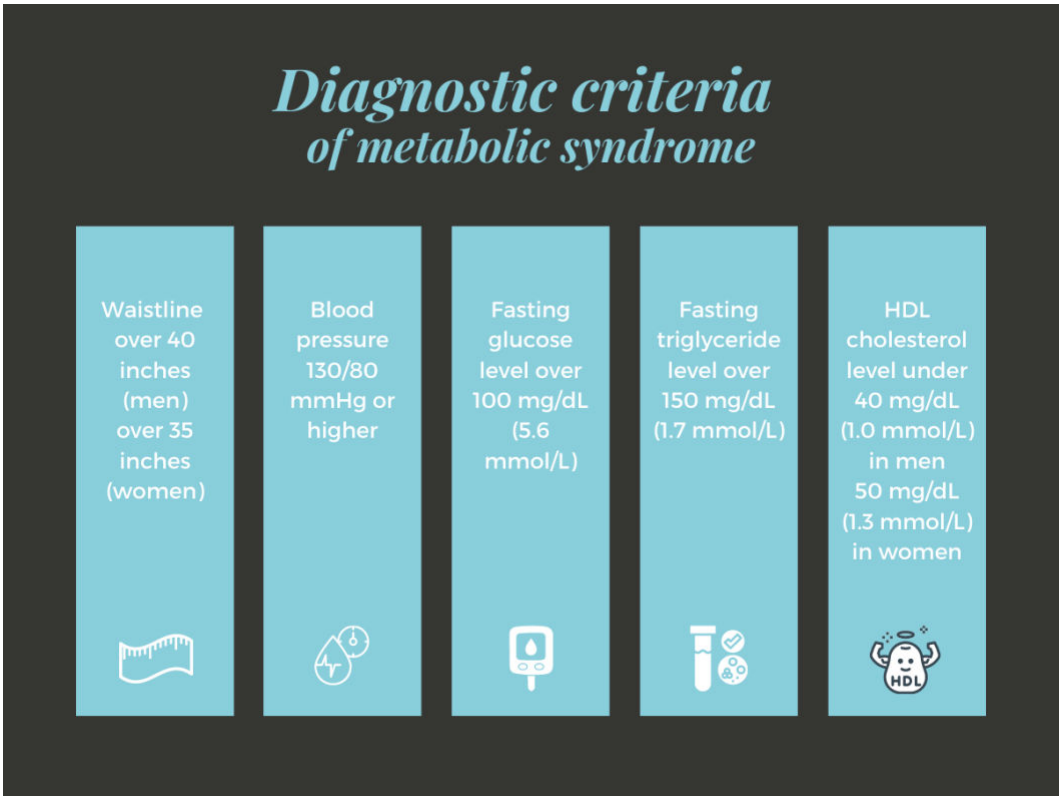
As this document makes clear, insulin resistance and type II diabetes are largely reversible through adopting healthy lifestyles.

Diagnosis and testing

Diagnosis

The following criteria are used to diagnose metabolic syndrome and insulin resistance

Figure 2. Criteria for diagnosing 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)
- An HDL cholesterol level under 40 mg/dL (1.0 mmol/L) in men and 50 mg/dL (1.3 mmol/L) in women

Risk factors

The following may put a person at higher risk for metabolic syndrome/insulin resistance:

- A diet high in carbohydrates
- Obesity, especially excess abdominal (belly) fat
- An inactive lifestyle
- Gestational diabetes (impaired glucose tolerance during pregnancy)
- Health conditions like non-alcoholic fatty liver disease and polycystic ovary syndrome
- Family history of type II diabetes
- Smoking
- Long COVID syndrome/vaccine injury
- Ethnicity (higher risk for people of African, Latino, or Native American descent)
- Age (higher risk after age 45)
- Sleep problems like sleep apnea

Testing

The following can help detect insulin resistance:

- Fasting plasma glucose test. [14] 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). [14] 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 > 200 mg is regarded as high.
- A low HDL.
    - A low HDL is considered less 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 is > 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. [15-17]
    - 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 managing insulin resistance, metabolic syndrome, and diabetes.

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

Intermittent fasting/time-restricted eating

Time-restricted eating has many 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.

Cautions and contraindications:

The following groups should avoid fasting:

- 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

The following groups should be cautious and undertake fasting under the supervision of a healthcare provider:

- Type II diabetics (will likely have to adjust diabetic medication)
- Those with chronic diseases taking multiple medications
- Those with gout

Low-carbohydrate (ketogenic) diet

Aim for a diet high in saturated fat, mono-unsaturated fat, and Omega-3 fatty acids. The carbohydrate content of a meal should not exceed 25 grams.

Berberine

Dose: total daily dose of 1000-1500 mg (take 500 mg two or three times daily or 600 mg twice daily). Once metabolic stability is achieved, it may be possible to reduce the dose of berberine to 500 mg once or twice daily.

Mechanisms: Berberine is a quaternary ammonium salt from the protoberberine group of isoquinoline alkaloids found in a number of different plants. [18] 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. [18;19] This remarkable herb has been shown to regulate glucose and lipid metabolism. [18]

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 alters hepatic gene expression. [20-25] Furthermore, berberine increases hepatic fatty acid oxidation [26] and decreases hepatic steatosis, [22] which may be a central pathogenic factor causing insulin resistance. [4]

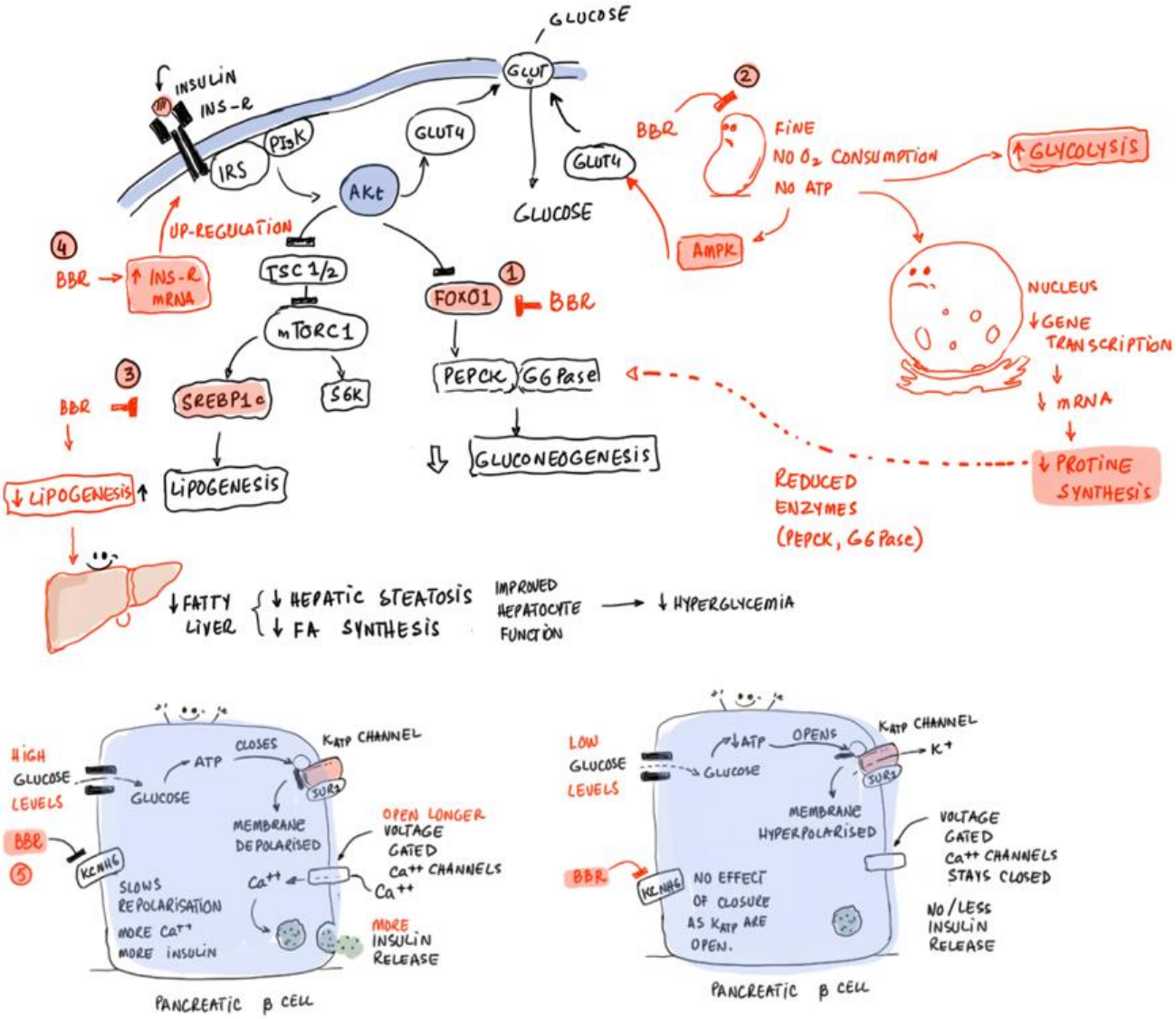
In addition, berberine has potent anti-inflammatory activity and modulates the microbiota, thereby reducing insulin resistance. [27-29] Multiple studies have demonstrated that berberine significantly reduces fasting blood glucose, postprandial blood glucose, HbA1c, and plasma triglycerides. [19;30;31] Berberine also 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. [19] Berberine appears to act synergistically with metformin. [19] As an additional bonus, berberine has anticancer effects. [32;33]

Cautions and contraindications:

- Berberine is remarkably safe; the only adverse events include transient gastrointestinal symptoms (diarrhea, flatulence).
- As insulin release is glucose-dependent hypoglycemia has not been reported with this herb. [20]
- As berberine lowers blood glucose and lowers blood pressure, these parameters should be monitored.
- Berberine should not be taken in patients taking cyclosporine 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, breastfeeding, and in neonates and children.
- If you are scheduled for surgery, please notify your anesthesia team if you are taking Berberine. You may need to stop taking Berberine one week prior to surgery.

Figure 3. Insulin-dependent and independent mechanism of berberine on lowering blood glucose



Drawing by Dr. Mobeen Syed

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.

Metformin

Dose: 500-1000 mg twice daily. The dose of metformin will likely need to be reduced in type II diabetics as insulin resistance improves during the induction phase.

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. [34]

Mechanisms: 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. [35]

In recent years, evidence has developed suggesting additional benefits of metformin due to its antitumor effect, antiaging effect, cardiovascular protective effect, neuroprotective effect, and the treatment of polycystic ovary syndrome. [35-37]

While metformin has been considered the drug of first choice in patients with type II diabetes, berberine appears to be equally (if not more) effective than metformin in the treatment of metabolic syndrome. It is likely metformin and berberine act synergistically to improve indices of metabolic syndrome (when either one alone is not sufficient).

Cautions and contraindications: A small study in elderly men demonstrated that metformin attenuated the increase in insulin sensitivity and VO_2 max after aerobic exercise training. [38] However, a similar study demonstrated that aerobic exercise and berberine have additive effects on cardiovascular risk factors. [39] This limited data suggests that berberine may be preferred over metformin in patients participating in aerobic exercise.

Magnesium

Dose: 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.

Magnesium has been demonstrated to reduce insulin resistance. [40;41] 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. [42] Magnesium oxide and magnesium citrate compounds, commonly prescribed by physicians, have poor bioavailability. [43] Magnesium Malate, Taurate, Glycinate, and L-threonate have good bioavailability and will readily increase RBC magnesium levels.

Melatonin

Dose: 2-10 mg slow release/extended release at night (dose as tolerated).

Mechanisms: 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. [44] Polymorphisms in the melatonin receptor type 1B gene are associated with increased fasting glucose, HbA1C as well as gestational and type 2 diabetes. [45] Melatonin has been demonstrated to improve hepatic insulin resistance and hepatic steatosis through a reduction in ER stress. [46] Higher nocturnal melatonin secretion has been demonstrated to be inversely associated with insulin levels and insulin resistance. [47] Finally, a meta-analysis of 16 RCTs (dose of 3-10mg at night) demonstrated that melatonin significantly reduced fasting blood glucose, HbA1C, and insulin resistance when compared to placebo. [48]

Resveratrol

Dose: 400-500 mg daily. 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.

Mechanisms: Resveratrol is a plant phytochemical (flavonoid) that has remarkable biological properties. [49-51] Most importantly it activates autophagy. [52;53] In addition, resveratrol may independently improve insulin resistance. [54]

Cinnamon

Dose: 1-2 g daily.

Mechanisms: Cinnamon is one of the major herbs used in traditional Chinese medicine. Preparations containing the bark of cinnamon have been prescribed for more than 2,000 years for the treatment of fever, the common cold, inflammation, diarrhea, and pain. [55] Cinnamon may have a role in the management of insulin resistance when combined with berberine.

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, anti-inflammatory, anti-diabetes, antiviral, antihypertensive, antioxidant, as well as being cardioprotective and improving the lipid profile. [55]

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. [56]

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. [56]

Omega-3 fatty acids

Dose: 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.

Mechanisms: 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. [57]

Probiotics with Bifidobacterium

Mechanisms: Intestinal dysbiosis is associated with obesity. While animal studies reproducibly demonstrate improved insulin resistance with probiotics, clinical studies have been more heterogeneous. [58] However, an RCT demonstrated that Bifidobacterium reduced anthropometric markers of obesity. [59] Furthermore, a cross-over, double-blind, randomized control trial demonstrated that supplementation of Bifidobacterium reduced blood sugar and improved insulin resistance. [60]

Cautions and contraindications: Some products can be very high in sugar, which promotes inflammation. Look for brands without added sugar and choose products with more than one strain of lactobacillus and bifidobacteria. Try to choose probiotics that are also gluten-free, casein-free, and soy-free.

Avoid excessive stress

Stress increases cortisol and catecholamines which increase blood sugar levels. Consider stress-mitigating strategies.

Exercise

Aim 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.

A quick guide to intermittent fasting/time-restricted eating

Intermittent fasting or time-restricted eating does not mean starving yourself or severely restricting caloric intake. There are a number of intermittent fasting plans that can be adapted and modified to best suit any lifestyle. The 2016 book by Dr. Jason Fung, *The Complete Guide to Fasting*, provides excellent guidance on approaches to intermittent fasting. [13]

Time-restricted eating appears to be a particularly effective and practical approach. For timed fasting, begin slowly: start with a 12-hour eating window 5 days a week and reduce week-by-week 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.

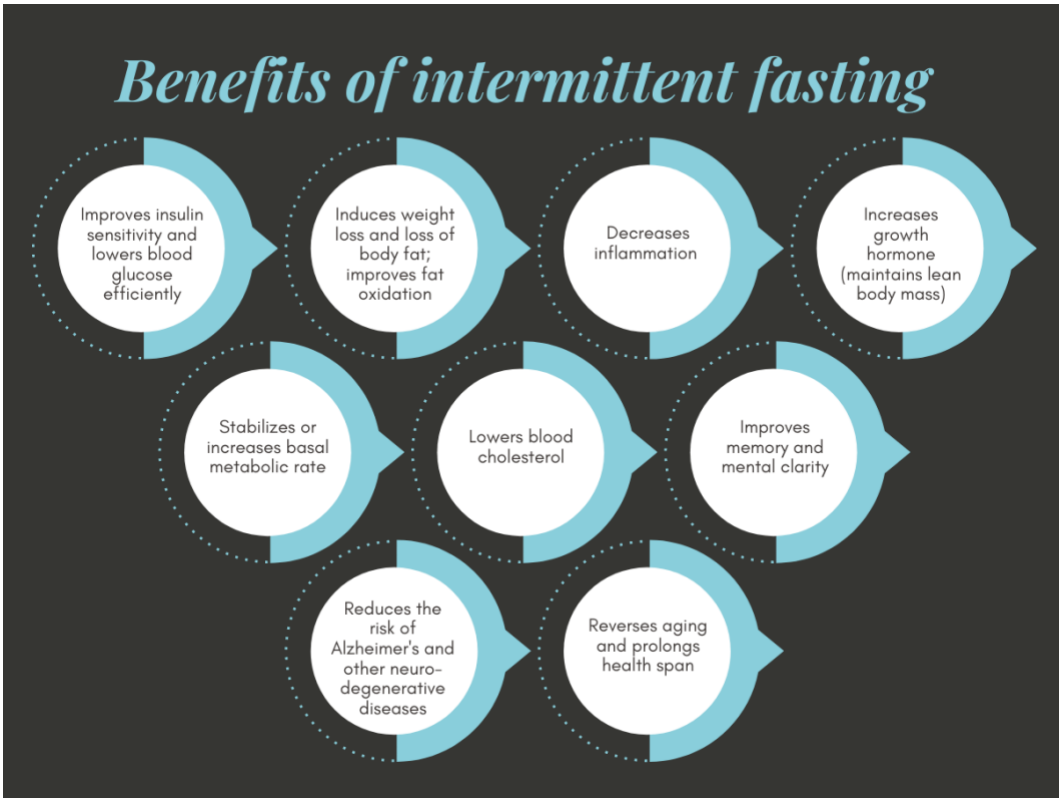
Some things to bear in mind:

- 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 rather than processed foods.
- Keep your meals diverse and include lots of green and cruciferous 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 about eating and food choices.
- No artificial sweeteners and no sodas.

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 an individual to determine their glucose response to a particular food group.

Benefits of Intermittent Fasting

Figure 4. Nine reasons to start intermittent fasting



Intermittent fasting:

- Improves insulin sensitivity and lowers blood glucose efficiently
- Induces weight loss and loss of body fat; improves fat oxidation
- Decreases inflammation
- Increases growth hormone (maintains lean body mass)
- Stabilizes or increases basal metabolic rate
- Lowers blood cholesterol
- Improves memory and mental clarity
- Reduces the risk of Alzheimer's and other neurodegenerative diseases
- Reverses aging and prolongs health span

The Glycemic Index

The glycemic index is a value assigned to foods based on how quickly those foods cause increases in blood glucose levels and how high they spike. 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 that are low on the glycemic index (GI) scale tend to release glucose slowly and steadily. Foods that are high on the glycemic index release glucose rapidly.

It should be noted that the glycemic index varies between individuals. [61;62] A CGM allows for the individual assessment of the glucose excursion (glycemic index) of various foods.

Figure 4. The blood glucose profile of high and low glycemic index foods

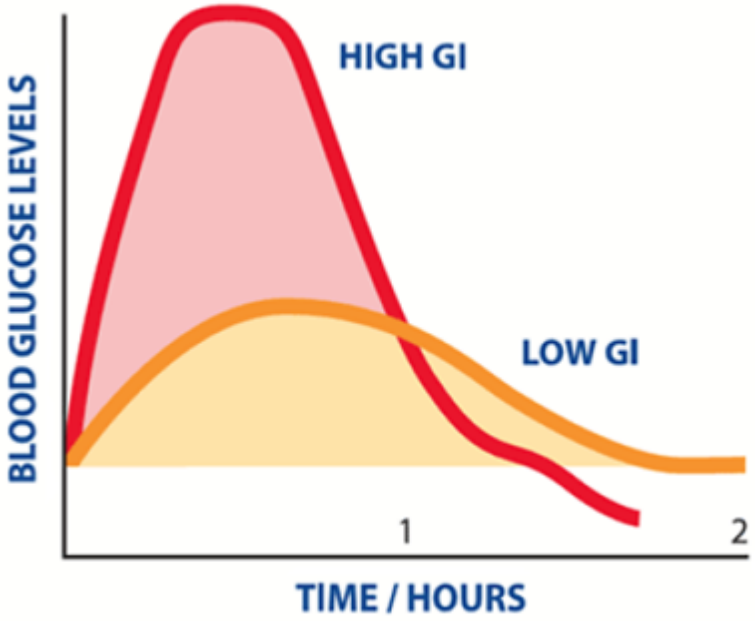


Table 1. Glycemic index of selected foods

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

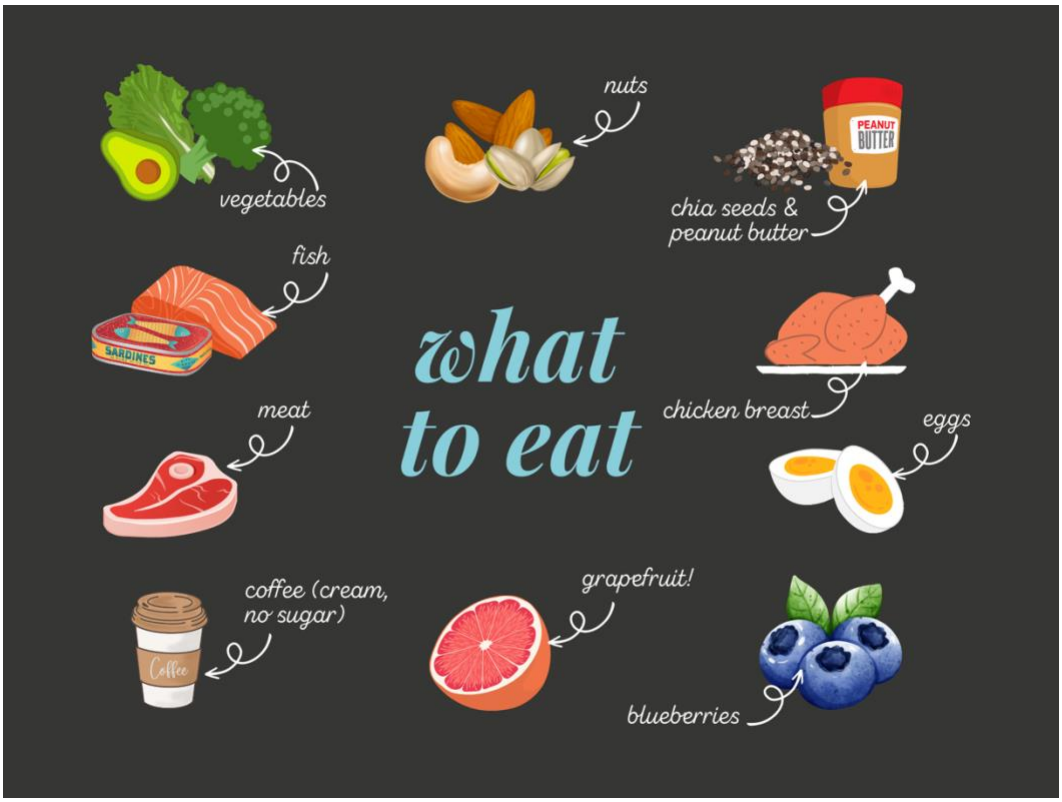
Healthy eating habits

It's not just what you eat, but also where you eat that matters. Studies have shown that eating on the sofa or at a desk can lead to excess weight gain because we are not as aware of how much we have eaten. Researchers distinguish between 'attentive' and 'distracted' eating and have found that attentive eating aids weight loss without the need to count calories. [63]

Figure 5. Not just what you eat, but where you eat

What to eat and what not to eat

Figure 6. The 10 best foods



Healthy foods include:

- All vegetables (especially avocado, cruciferous, and leafy vegetables)
- Nuts (almonds, brazil nuts, cashews, and pistachios)
- Peanut butter (but avoid the white bread and grape jelly!) and chia seeds
- Fish (especially Alaskan salmon and sardines)
- Chicken breast (free range, no hormones, no antibiotics)
- Eggs (they've been given a bad rap!)
- 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; choose Stevia over sugar or artificial sweeteners)

Figure 7. The 10 worst foods

Say goodbye to:

- Donuts
- Bagels, bread, pretzels, tortillas
- Cookies, muffins, baked products
- Chips
- French fries
- Rice and pasta
- Potatoes
- Canned fruits/fruit juices
- Sweetened low-fat yogurt
- Watermelon and bananas

Healthy and unhealthy oils

Avoid seed oils high in linoleic acid. Linoleic acid is an essential Omega-6 fatty acid that our bodies require in small amounts. Unfortunately, many people eat up to ten times the desired amount of linoleic acid, because of excess consumption of foods made with seed oils. Too much linoleic acid is associated with inflammation, obesity, heart disease and other unfavorable conditions. Therefore, avoid:

- Soybean oil
- Corn oil

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

Instead, opt for healthy oils and fats such as the ones listed below. Use only high-quality products and check production and expiration dates.

Figure 8. A guide to healthy cooking oils

- Olive oil (oleic acid, Omega-9 monounsaturated fatty acids); never heat olive oil to the point where it produces smoke
- Avocado oil (oleic acid, Omega-9 monounsaturated fatty acids)
- Coconut oil (medium chain fatty acid)
- Flaxseed oil (alpha-linolenic acid, ALA Omega-3)
- Walnut and Pecan oils; these oils should be refrigerated to avoid spoilage
- Butter (saturated fat)

Debunking three common myths

Myth: Saturated fat and cholesterol are the leading causes of heart disease

The cholesterol-saturated fatty acid hoax [64-66] began to proliferate in the 1960s. 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. [67;68] This concept has been vigorously studied, including many randomized controlled trials, and has been convincingly proven false. [64;69;70] Indeed, replacing saturated fats with a diet high in vegetable oils (linoleic acid) was associated with higher rates of death, cardiovascular and coronary heart disease as well as a significantly increased risk of cancer. [71]

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. [64;72;73]

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.

Myth: People with high cholesterol must take statins to keep it under control

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 helps dispel this myth; it demonstrated that statins were no better than placebo in reducing all-cause mortality in high-risk patients. [74]

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 it 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.

Myth: Vegetable and seed oils are a heart-healthy choice

These processed oils are high in Omega-6 polyunsaturated fatty acids (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. They are linked to an increased risk of cancer and are pro-inflammatory. And, as stated above, vegetable oils DO NOT reduce cholesterol or the risk of cardiovascular disease.

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