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CONTENT

The Effect of Low FODMAP Diet (LFD) on IBS Improvement

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Introduction

Irritable Bowel Syndrome (IBS) is also known as spastic colon, irritable colon, mucous colitis, and spastic colitis. It is a separate condition from inflammatory bowel disease and isn't related to other bowel conditions. IBS is a group of intestinal symptoms that typically occur together (1). Around 10-15 ⊠ of the population worldwide has IBS. IBS is twice as common in women as men and 10.9⊠ of Iranian adults suffer from this disease. About 10-20⊠ of all visits to gastroenterologists are due to IBS symptoms. It should be considered that 70⊠ of people with IBS symptoms never consult to doctor (2).

The IBS symptoms vary in severity and duration from person to person and are usually present for a long time, such as abdominal pain, bloating and gas, constipation, diarrhea, unusual stools, nausea, stress (3).

There's no special test to definitively diagnose IBS. The doctor is likely to start with a complete medical history and physical examination to rule out other conditions, such as celiac disease (4). After other conditions have been ruled out, it is likely to assess Rome IV criteria. The Rome IV criteria for the diagnosis of irritable bowel syndrome require that patients have had recurrent abdominal pain on average at least 1 day per week during the previous 3 months that is associated with two or more of the following: Related to defecation (may be increased or unchanged by defecation), associated with a change in stool frequency, associated with a change in stool form or appearance (4). Colonoscopy, X-ray or CT scan, upper endoscopy, and laboratory tests include lactose intolerance, breath test for bacterial overgrowths, and stool tests are ways to diagnose IBS (4).

Treatment of IBS focuses on relieving symptoms so that patient can live as normally as possible. Mild signs and symptoms can often be controlled by managing stress and by making changes in the diet and lifestyle. People should try to avoid foods that trigger the symptoms, eat high-fiber foods, drink plenty of fluids, exercise regularly, and get enough sleep (4).

FODMAP stands for fermentable oligosaccharides, disaccharides, monosaccharides, and polyols, which are short-chain carbohydrates (sugars) that the small intestine absorbs poorly. Some people experience digestive distress after consumption of them. Symptoms include cramping, diarrhea, constipation, stomach bloating, gas, and flatulence (5). Low FODMAP Diet (LFD) is a three-step elimination diet which in its first stage people stop eating high FODMAP foods. Next, banned foods are given slowly to find out which ones are troublesome. When the foods that cause symptoms are identified, you can avoid or limit their long term while enjoying everything else stressless (5). It is recommended to obey the elimination section of the Low FODMAP Diet for only 2-6 weeks (5).

To relieve IBS and SIBO symptoms, it's important to avoid high FODMAP foods that aggravate the gut, including Dairy-based milk, yogurt and ice cream, Wheat-based products such as cereal, bread, and crackers, beans and lentils, some vegetables, such as artichokes, asparagus, onions and garlic, Some fruits, such as apples, cherries, pears and peaches (5). Instead, meals should include low FODMAP foods such as eggs and meat, certain cheeses such as brie, camembert, cheddar and feta,

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almond milk, grains like rice, quinoa, and oats, vegetables like eggplant, potatoes, tomatoes, cucumbers, and zucchini, fruits such as grapes, oranges, strawberries, blueberries and pineapple (5). This study aims to assess the effect of a low FODMAP diet on IBS.

Literature review

Staudacher et al. conducted a randomized controlled trial in 2017 in the UK. The purpose of this study was to evaluate the effect of a low FODMAP diet on reducing the symptoms of IBS, and the effect of probiotics of Bifidobacterium species on IBS symptoms. The results of this study showed that the low FODMAP diet reduces illness severity and gastrointestinal symptoms and also increases the quality of life in patients with IBS (6).

In a cross-sectional study, Pourmand et al examined adherence to the LFD diet and its association with IBS symptoms in Iranian adults. Finally, no significant relationship was observed between adherence to the LFD diet and reduction of IBS symptoms (2).

To investigate the effect of three diets (low FODMAP, gluten-free and balanced) on the symptoms of irritable bowel syndrome and quality of life, Paduano et al conducted an interventional study on 42 patients with IBS in Italy in 2019. The results of this study showed that the severity of gastrointestinal symptoms and quality of life were the same between all three diet groups and all three diets reduced the symptoms of IBS (7).

In a prospective observational study in 2019, Kortlever et al examined the effect of the low FODMAP diet on the quality of life and improvement in IBS patients. The results showed that following the LFD diet reduced gastrointestinal symptoms, fatigue, anxiety and increased the quality of life and feeling of happiness in patients with IBS (8).

Altobelli et al. conducted a systematic review and meta-analysis of 12 RCT and cohort articles to investigate the effect of the LFD diet on improving IBS in 2017, and finally found that the LFD diet reduced suspicious pain, bloating, and bowel movements in patients with IBS (9).

Discussion

Short-chain fermentable carbohydrates might exacerbate IBS symptoms through various mechanisms, such as increasing small intestinal water volume, colonic gas production, and intestinal motility. Short-chain fermentable carbohydrates increase luminal H2 and CH4 production, resulting in luminal distension and pain in those with visceral hypersensitivity. As a result, consuming high FODMAP foods increases the severity of irritable bowel syndrome (10).

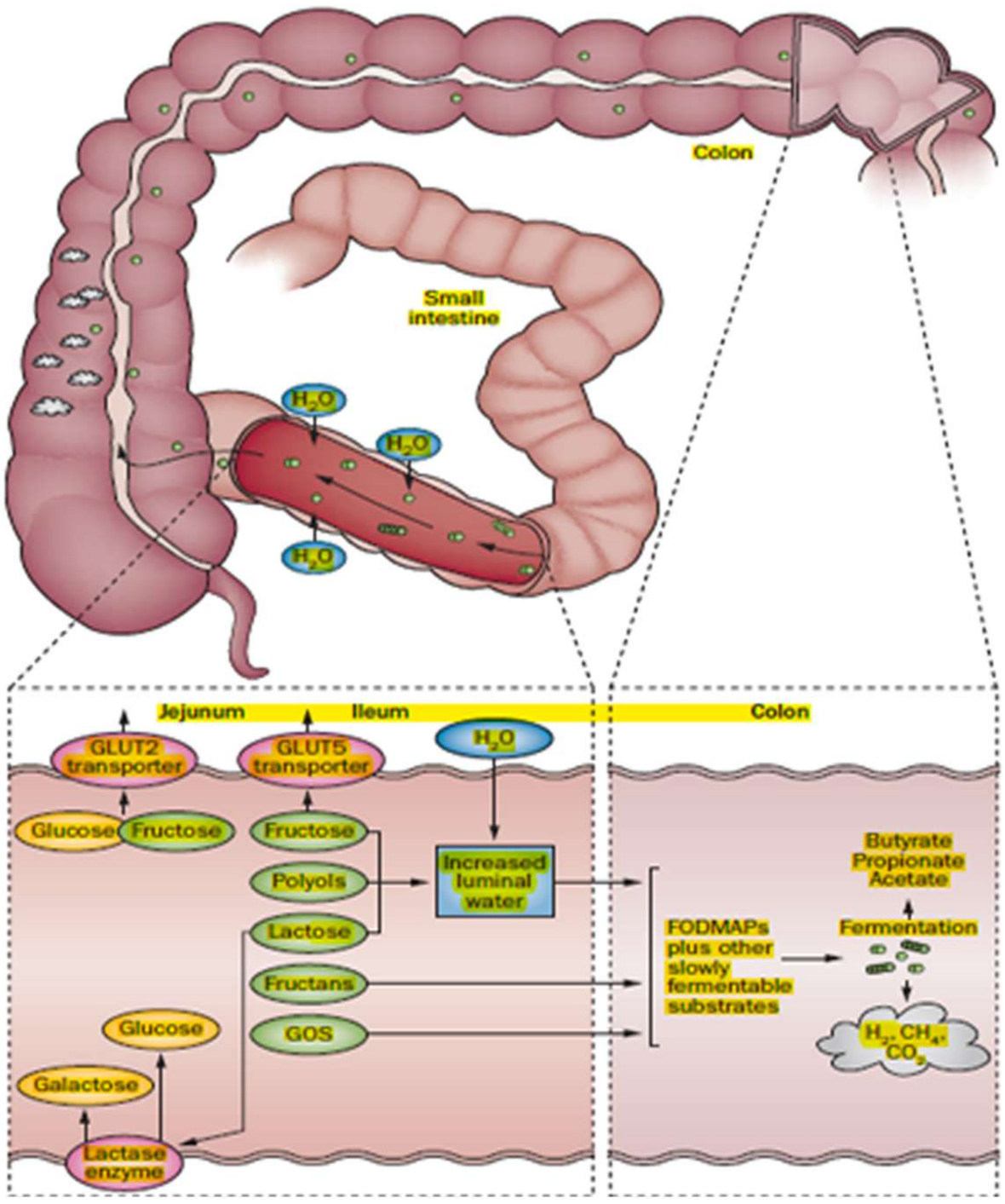

Figure 1. Mechanisms by which short-chain fermentable carbohydrates might induce symptoms in IBS (10)

One of the two most established mechanisms by which FODMAPs are proposed to provoke symptoms in IBS is the augmentation of small intestinal water, which has been demonstrated by both ileostomy recovery and MRI studies (6).

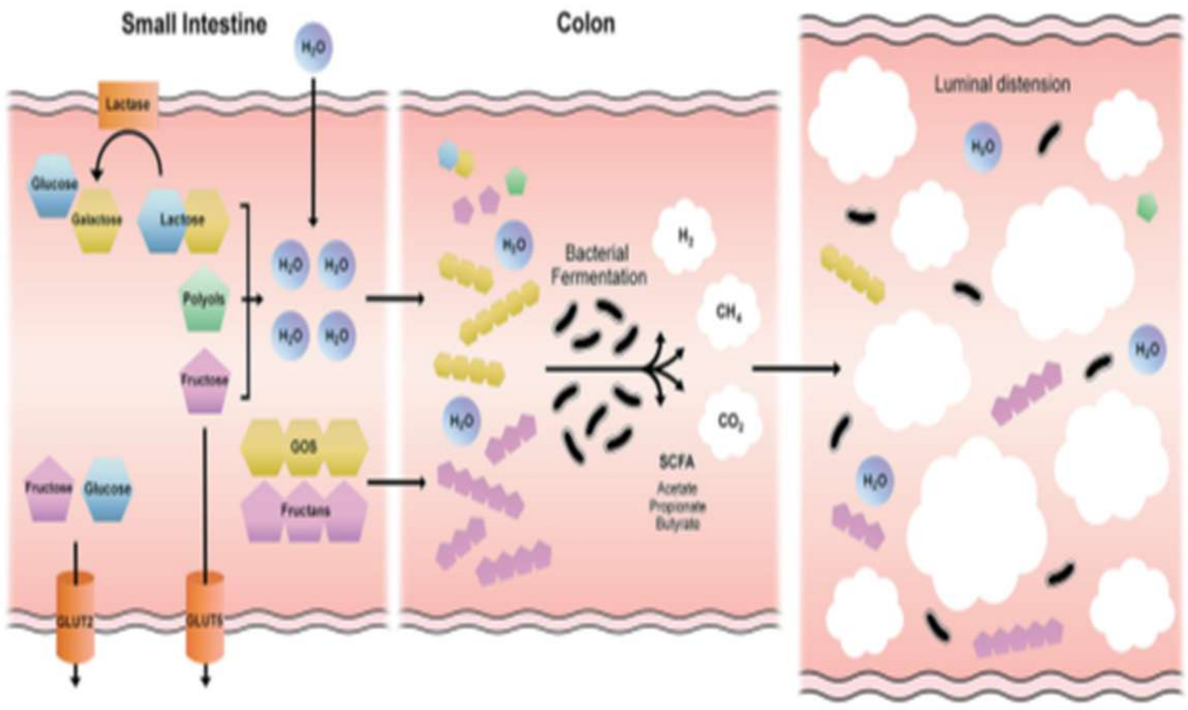

Figure 2. Mechanisms of the effects of fermentable oligosaccharides, disaccharides, monosaccharides, and polyols on GI function (6)

Conclusion

According to the previous studies, following a low FODMAP diet can be effective in reducing and controlling the symptoms of IBS.

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The Effect of High Glycemic Index Diet on Insomnia

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Introduction

Sleep is a naturally recurring state of mind and body. Sleep occurs in repeating periods, in which the body alternates between two distinct modes: REM sleep and non-REM sleep. REM stands for "rapid eye movement" which has many aspects, including virtual paralysis of the body (1). During sleep, most of the body's systems are in an anabolic state, helping to restore the immune, nervous, skeletal, and muscular systems; these are vital processes that maintain mood, memory, and cognitive function, and play a large role in the function of the endocrine and immune systems. The diverse purposes and mechanisms of sleep are the subject of substantial ongoing research (1).

Humans may suffer from various sleep disorders, including dyssomnias such as insomnia, hypersomnia, narcolepsy, and sleep apnea, parasomnias such as sleepwalking and rapid eye movement sleep behavior disorder, bruxism, and circadian rhythm sleep disorders (2).

The sleep cycle of alternate NREM and REM sleep takes an average of 90 minutes, occurring 4–6 times in a good night's sleep. The American Academy of Sleep Medicine (AASM) divides NREM into three stages: N1, N2, and N3, the last of which is also called delta sleep or slow-wave sleep. The whole period normally proceeds in the order:

N1 → N2 → N3 → N2 → REM. There is a greater amount of deep sleep (stage N3) earlier in the night, while the proportion of REM sleep increases in the two cycles just before natural awakening (3).

Insomnia is a general term for difficulty falling asleep and/or staying asleep. Insomnia is the most common sleep problem, with many adults reporting occasional insomnia, and 10–15% reporting a chronic condition (4). Insomnia can have many different causes. Insomnia is often treated through behavioral changes like keeping a regular sleep schedule, avoiding stimulating or stressful activities before bedtime, and cutting down on stimulants such as caffeine. Sleeping medications such as Ambien and Lunesta are an increasingly popular treatment for insomnia but we want to talk about a better solution (5).

When you eat any type of carbohydrates, your digestive system breaks it down into simple sugars that enter the bloodstream. Not all carbs are the same, as different types have unique effects on blood sugar. The glycemic index (GI) is a measurement system that ranks foods according to their effect on blood sugar levels (6). The rates at which different foods raise blood sugar levels are ranked in comparison with the absorption of 50 grams of pure glucose. Pure glucose is used as a reference food and has a GI value of 100. The three GI ratings are Low: 55 or fewer, Medium: 56–69, and High: 70 or more (6). However, the GI is a relative measure that doesn't take into account the amount of food eaten. To solve this, the glycemic load (GL) rating was developed. Like the GI, the GL has three classifications (Low: 10 or fewer, Medium: 11–19, and High: 20 or more) (6). This study aims to evaluate the effect of dietary glycemic index on sleep.

Literature review

According to a study conducted in 2011 by Jalil Qadr et al to investigate the effects of high and low glycemic index drinks on children's sleep patterns, increasing the glycemic index at

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bedtime meals increased the Index of arousal and decreased sleep quality (7). A study by Afaghi et al to investigate the use of dietary carbohydrates and their effects on increasing plasma tryptophan concentrations as a precursor of serotonin and a sleep-inducing factor, showed that a meal with a high glycemic index (HGL) 4 hours before bedtime reduces the effects of insomnia (8). A study conducted in 2018 by Vlahoyiannis et al investigated the effects of glycemic index diet after exercise on the quality and quantity of sleep. It was shown that eating foods with a high glycemic index in the post-exercise meal improves sleep indices (9). A study conducted in 2019 by Daniel et al aimed to investigate the effects of a high glycemic index (HGI) or low (LGI) diet on athletes' sleep. They showed that consuming higher energy during the day, regardless of the GI of dinner, reduces the quality of sleep (10). Gangwisch et al in 2020 investigated the effect of using substances with a high glycemic index on the prevalence of insomnia in women. The results showed that increasing the glycemic index of food increases the risk of outbreaks and insomnia (11).

Discussion

The mechanism by which a high-GI carbohydrate meal shortens the sleep onset latency (SOL) is currently unknown, but the high-GI meal may work through an increased plasma concentration of insulin and Tryptophan (Trp) to a large neutral amino acid ratio (Trp: LNAA) and its ability to compete for entry into the brain with other LNAAs (12). The entry of Trp into the brain is linked to its concentration relative to other LNAAs and the main determinant of brain serotonin concentration is a high plasma Trp: LNAA. It is now known that the plasma Trp: LNAA is affected by both dietary carbohydrates and dietary protein. Once in the brain, Trp is converted to serotonin, which is also spontaneously produced in the raphe system of the brainstem. Serotonin is necessary for the normal sleep of mammals. Ingestion of L-Trp significantly reduces the SOL of insomniacs and that of healthy sleepers. Indeed, several studies and review articles of the effect of Trp on sleep support the notion that Trp reduces SOL without affecting other sleep variables (13).

Conclusion

According to the results obtained from the articles, a diet high in glycemic index can be a risk factor for insomnia. Dietary interventions that promote the consumption of whole carbohydrates (which have a low GI and high fiber) can be considered as a potential treatment for insomnia. following diets with a high glycemic index after exercise can improve the length and efficiency of sleep and reduce sleep delay.

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The Effect of Metformin on Weight Loss

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Introduction

Excessive accumulation of fat in the body is called obesity. The measure by which people measure obesity and thinness is called body mass index (BMI). A BMI over 25 is considered overweight, and when BMI is 30 or greater is called obesity (1). According to the Global Obesity Statistics in 2020, Iran is one of the countries where the prevalence of obesity is almost high (1). The most important complications of obesity include fatty liver, osteoarthritis, obstructive sleep apnea, cardiovascular disease, cancer, and diabetes. Possible treatments for obesity are adequate physical activity, following a weight loss diet, medication, and surgery (2). There are also some weight loss drugs such as carboblockers, phentermine, sibutramine, orlistat, metformin, etc. Metformin is an oral medicine from the category of biguanides. It helps to control blood sugar levels. Also, metformin is the first line of treatment in people with type 2 diabetes and is used in women with Polycystic Ovary Syndrome (PCOS) (3). It is available in tablets in doses of 500, 750, 850, and 1000 mg on the market.

The effects of metformin include lowering blood sugar, improving leptin resistance, suppressing appetite, inhibiting tumors, and delaying aging. Metformin If taken with medications such as insulin can cause hypoglycemia, gastrointestinal side effects, coagulation abnormalities, decreased serum vitamin B12 levels, lactic acidosis, pancreatitis, and unintentional weight loss (3,4). Use of metformin for children Under the age of 10, the elderly, people with hypoglycemia, vitamin B12 deficiency, metabolic stress, diabetic ketoacidosis, liver disease, and kidney failure, and cardiovascular disease are contraindicated (3). Metformin interacts with drugs such as amiloride, cimetidine, vancomycin, digoxin, furosemide, triamterene, ranitidine, morphine, and calcium channel blockers (3). This study aimed to investigate the effect of metformin on weight loss.

Literature review

A study in 2012 by Wen-shan Lv et al in China investigated the effect of metformin on food intake and its role in the regulation of the hypothalamus in obese diabetic rats. The use of metformin played a major role in the function of the nervous system and reduces its genes. Neuropeptide Y(NPY) and Aguti Related Protein (AgRP) were seen in the presence of metformin. Due to the increased phosphorylation of Signal Transducer and Activator of Transcription3 (STAT3) in the presence of metformin, the anorectic effect of metformin can be related to STAT3 (5).

Jensterle et al aimed to investigate the long-term use of metformin in overweight or obese PCOS women. According to the results, long-term treatment with metformin in PCOS subjects with overweight or obesity resulted in weight loss, improved menstrual cycles, and androgen depletion (6).

H-S Ejtahed et al in 2018 conducted a study to investigate the effect of metformin on weight loss in non-diabetic obese women through intestinal microbiome changes. The results showed a decrease in BMI and a decrease in insulin concentration in people taking metformin with a low-calorie diet versus those taking placebo, but the variation in the overall composition of feces and microbiota was not significant (7).

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Shuqin Ji et al in 2019 studied the effect of metformin on short-term High-Fat Diets (HFD), anxiety-like behaviors, and intestinal microbiota. According to the results of this study, a three-week HFD increased body weight and fat cell size, which is reduced by metformin treatment. Anxiety behaviors caused by a high-fat diet were also reduced by concomitant treatment with metformin. Metformin significantly increased phosphor-Adenosine monophosphate-activated kinase (p-AMPK) levels in White Adipose Tissue (WAT) and the brain. HFD and metformin treatments with HFD alter the diversity and composition of the gut microbiota (8).

Also, a meta-analysis was conducted by Solymar, In 2018 in Hungary to examine the effect of metformin on weight loss, total cholesterol, and Low-Density Lipoprotein (LDL) in the elderly. The results showed a slight weight loss and a slight improvement in blood lipid levels in patients over 60 years of age while taking metformin. Due to the risk of unintentional weight loss by metformin, this drug is suitable for the population over 60 years (9).

Discussion

Metformin affects various parts of the body, including the digestive system, central nervous system(CNS), muscles, liver, and intestinal flora (10,11). Metformin in the central nervous system increases the production of incretins and lactate by increasing leptin sensitivity in the hypothalamus and inhibits the activity of orexigenic neurons. Metformin can cross the blood-brain barrier, thereby increasing leptin sensitivity. This hypersensitivity causes more STAT3 activity in the Nucleus of Tractus solitarius (NTS) and thus increases the anorexic effect in the hypothalamus (10). It also inhibits AMPK activity in the hypothalamus and thus inhibits NPY and AgRP gene expression (10). Metformin inhibits the electron transport chain 1 complex and initiates anaerobic respiration and lactate production, thereby suppressing and reducing appetite (11). In the gastrointestinal tract, metformin causes an increase in Glucagon-like peptide-1 (GLP-1) (incretin), impaired taste sensation, nausea and vomiting, diarrhea, abdominal pain, and bloating, all of which reduce a person's appetite and desire to eat (10,11). In the liver, metformin decreases fat and cholesterol synthesis and decreases gluconeo-genesis by increasing AMPK activity (10). In the intestinal tract, metformin increases GLP-1 and alters the intestinal flora by reducing carbohy-drate absorption. In muscle, it also increases fat oxidation and increases glucose uptake by increasing AMPK activity (10).

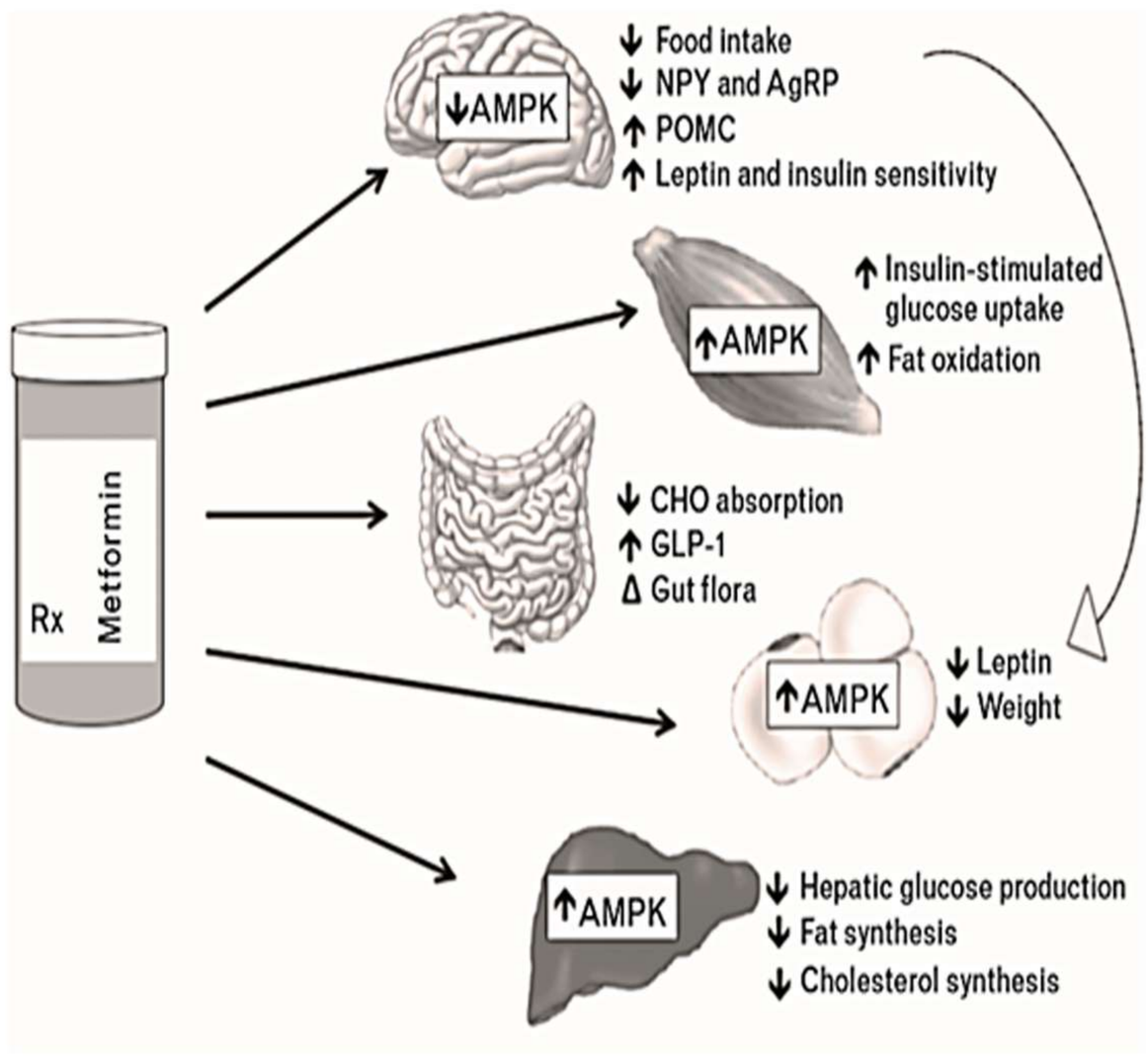

Figure 1. The effect of Metformin on the digestive system, Central Nervous System(CNS), muscles, liver, and fat (10)

Conclusion

Metformin alone should not be recommended as a weight-loss drug. But for people who have insulin resistance and other underlying condi-tions such as diabetes, it can be a good treatment option that also helps them lose weight.

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Serum 25_Hydroxyvitamin D Concentrations With Hashimoto's Thyroiditis

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Introduction

Hashimoto's thyroiditis (HT) is the most common autoimmune disease of the thyroid and the most common cause of hypothyroidism in situations with adequate iodine intake. In Hashimoto, immune cells invade the thyroid and cause fibrosis (1).

About 5% of the world's population has Hashimoto's disease, and the disease is more common in people in their 30s and 50s (2).

Hashimoto's symptoms are similar to those of hypothyroidism and include hair loss, dry skin, depression, unexplained weight gain, fatigue, constipation (3).

Diagnosis is based on high TSH levels, low T3, T4, and high thyroid peroxidase antibodies. Ultrasound and biopsy are also used as complementary tests (4,5).

Hashimoto's treatment includes diet, thyroidectomy, and most importantly, medication. In Hashimoto, like hypothyroidism levothyroxine is used (6,7).

If left untreated or uncontrolled, it can cause problems such as impotence, dementia, marital problems, heart problems, and goiter (1).

Risk factors for Hashimoto's disease include other autoimmune diseases, genes, female gender, age over 30, and vitamin D deficiency (4).

Vitamin D has two precursors: plant precursors (ergocalciferol) and animal precursors (cholecalciferol) which both are biologically inactive and need to be activated. In the first step, it is converted to 25(OH)D, and then it is converted to its most active form, 1,25(OH)2D (calcitriol) (8).

Vitamin D plays role in bone health, reducing inflammation and pain, maintaining calcium and phosphorus balance, and building immunity and its deficiency can lead to autoimmune diseases (9).

The normal range of serum vitamin D is more than 30 ng/ml. (9) Sources of vitamin D include eggs, fish, dairy, liver, and mushrooms (9). This study aims to investigate the possible role of serum 25_hydroxyvitamin D concentrations in Hashimoto's thyroiditis improvements.

Literature review

A case-control study conducted in 2015 by Evliyaoğlu et al, that examined vitamin status in Hashimoto's patients. They concluded that Vitamin D deficiency is associated with Hashimoto's disease in children and adults, and levels below 20 ng/ml can be dangerous (10).

Sonmezgoz et al measured vitamin D levels in children with HT and healthy children. The results showed that vitamin D deficiency is more common in children with HT than children without HT (5).

Yavuzer et al in 2017 conducted a study that the relationship between Hashimoto's thyroiditis and vitamin D and osteoporosis markers was examined. Based on the results, there was no significant difference in vitamin D levels between Hashimoto's patients and healthy individuals. Vitamin D levels were significantly lower in patients with positive antibodies than in patients with negative antibodies (2).

Nalbant et al investigated the relationship between low levels of vitamin D and Hashimoto's in women. The results showed that women with Hashimoto's had lower levels of vitamin D than healthy people and this deficiency could increase the incidence of HT (4).

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A meta-analysis study by stefanic et al investigated the relationship between vitamin D concentration and Hashimoto's thyroiditis. They showed that there was a significant relationship between serum 25-hydroxyvitamin D deficiency and HT (11).

Discussion

Vitamin D reduces the production of inflammatory cytokines such as TNF alpha (these cytokines produce a strong proinflammatory response that increases the accumulation of macrophages and increases the activity of B and T lymphocytes). The binding of vitamin D to B lymphocytes increases its apoptosis. TH1 and TH17 play a role in creating an immune response. Vitamin D reduces the immune response by reducing its expression (12).

Conclusion

A significant relationship between serum 25-hydroxyvitamin D deficiency and Hashimoto's exists. Adequate intake of vitamin D can help to prevent and improve Hashimoto by reducing the activity of immune cells against the native cells.

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The Effect of Almonds Consumption on Blood Pressure

Hale Khasedar*

Introduction

Blood pressure (BP) is the pressure of blood pushing against the walls of your arteries(1). Blood pressure is written as two numbers. Systolic Blood Pressure (SBP) represents the pressure in blood vessels when the heart contracts or beats. Diastolic Blood Pressure (DBP) represents the pressure in the vessels when the heart rests between beats. (2).

High blood pressure (HBP) is one of the important causes of mortality worldwide (3).

Its prevalence is increasing dramatically; there was a 10.5% increase in the death rate attributed to HBP in the United States (US) from 2005 to 2015 (4).

Worldwide, 1.3 billion hypertensive people have been diagnosed in 2010. the number 1 cause of death in 2016 is ischemic heart disease and stroke, the main complications of HBP. Eleven percent of children and young adolescents in the US were prone to this non-communicable disease in 2012 according to the American Heart Association (3).

The prevalence of HBP varies across the world considerably. Its prevalence was 39.1% in Latin America, 26.9% in the Middle East and North Africa, 29.4% in South Asia, 31.5% in European and Central Asia countries, 31.1% in Sub-Saharan Africa, and 35.7% in East China and the pacific(5).

In Iran, two meta-analyses have been conducted related to the prevalence of HBP in 2008 and 2012. According to the first study, the prevalence of HBP in the 30-55 age group and older than 55 years, were 23% and 50%, respectively (6). In 2012, the prevalence of HBP in adults was 22% (7).

Nutrition, in conjunction with pharmacotherapy, plays an important role in either increasing or managing the HBP; high sodium chloride intake increases the incidence, and alcohol consumption may cause an acute elevation in BP. In contrast, a high intake of potassium, polyunsaturated fatty acids, and protein may help in lowering BP (8).

Almonds, a type of tree nut, are rich in vitamins, minerals, mono-saturated and polyunsaturated fatty acids (9).

some studies have shown almonds to have a potential reducing effect on SBP and DBP (10).

This study aimed to investigate the effect of almonds on lowering blood pressure (11).

Literature review

A systematic review and dose-response meta-analysis of randomized control trials study was conducted by Le et al in 2020. This study aimed to determine the effect of almonds consumption on BP in adults. Fifteen studies containing 853 participants, reported SBP as an outcome measure. Pooled results showed a significant reduction in SBP by almond intervention. There is no significant effect from almond consumption on DBP (12).

A randomized controlled 12-wk clinical trial of 86 healthy adults [body mass index (in kg/m2): 25–40] was conducted by Dhillon et al in 2018 in the US, which aimed to investigate the effect of almond consumption when energy restriction on blood pressure in overweight or obese people. A linear mixed-model analysis on primary outcomes such as BP was performed on all participants [intention-to-treat (ITT) analysis] and compliant participants (compiler analysis).

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SBP decreased after 12 weeks of energy restriction in both the ITT and complier analyses (P<0.05) but DBP decreased only in compiler analyses (P=0.029) (13).

Al Tamimi et al was done an Interventional study that examined the effects of Almond Milk on BP in 2016. Thirty volunteers of both sexes participated in the study. The study was conducted over 4 weeks, and results showed that daily replacement of one serving of dairy products with one cup (240 ml) of almond milk did not affect BP (14).

A randomized controlled trial study was conducted by Jamshed et al In 2015 in Pakistan, that aimed to Investigate the relationship between almond supplementation and blood pressure in coronary artery disease patients. The systolic and diastolic blood pressure of the participants remained fairly constant among all the groups (15).

Choudhury et al were done a meta-analysis interventions study that examined the effect of an almond-rich diet on vascular function in 2014. Healthy middle-aged men, healthy young men, and young men with two or more cardiovascular (CV) risk factors consumed 50 g almond/day for 4 weeks. In all groups consuming almonds, SBP decreased significantly after 50 g almonds/day for 4 weeks (P<0.05 but DBP reduced only in healthy men (P=0.002) (11).

Discussion

Nutrients constituent within almonds is associated with beneficial cardiovascular outcomes, such as antioxidant flavonoids and l-arginine, which is key for the synthesis of the vasodilatory molecule, nitric oxide (NO) (16).

Nitric Oxide can stimulate the production of cyclic guanosine monophosphate (cGMP). cGMP can affect cells and activate protein kinases G(PKG). Activation of PKG by cGMP leads to activation of myosin phosphatase which in turn leads to the release of calcium from intracellular stores in smooth muscle cells. This in turn leads to relaxation of the smooth muscle cells (17).

Magnesium is a mineral with important functions in the body such as actions in vascular tone. Increased concentrations of extracellular magnesium cause vasodilation, whereas decreased concentrations cause contraction (18).

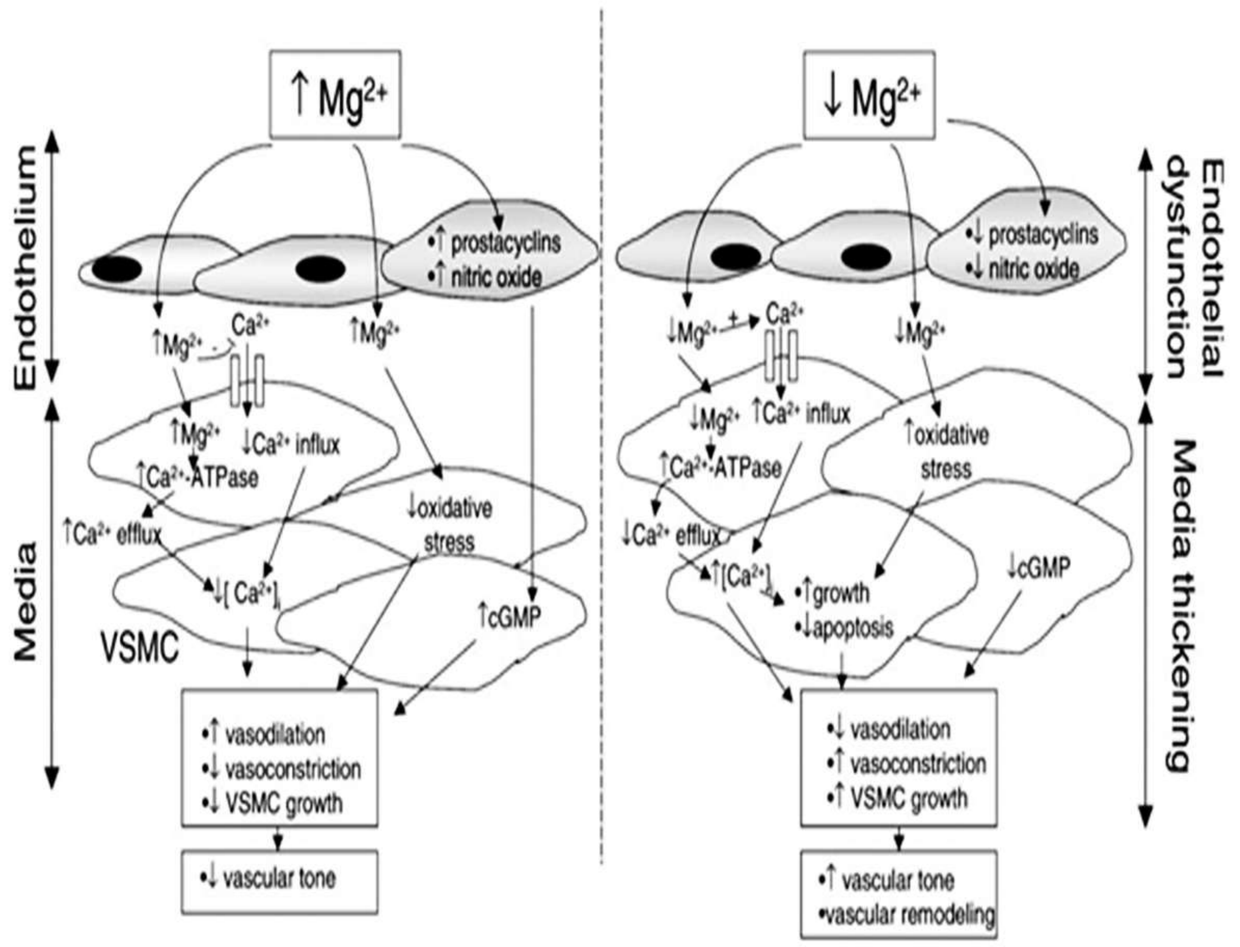

Figure 1. Role of magnesium in hypertension (18)

Potassium can cause endothelium-dependent vasodilation by hyperpolarizing the endothelial cell through stimulation of the sodium-potassium pump and activation of plasma membrane potassium channels. Endothelial hyperpolarization results in decreased cytosolic smooth muscle cell calcium, which in turn induces vasodilation. Furthermore, potassium may soften the cytoskeleton of the vascular endothelium, which in turn may lead to NO release. Potassium may also promote vascular smooth muscle relaxation and increases in blood flow through inhibition of the sympathetic nervous system by increasing the uptake of norepinephrine into the sympathetic nerve terminals (19).

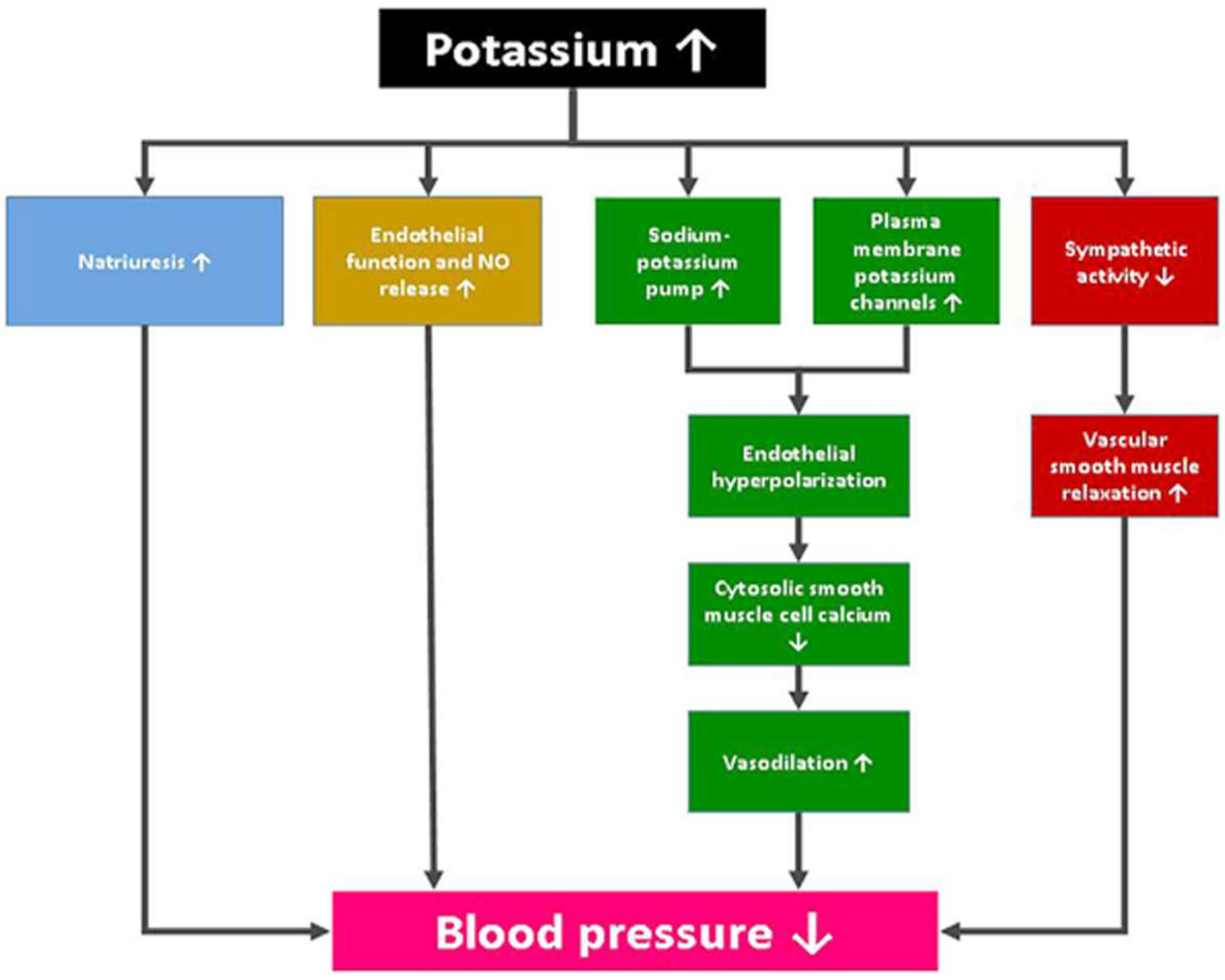

Figure 2.Role of potassium in hypertension(19)

Conclusion

The current body of evidence supports the ingestion of almonds for their beneficial effect on blood pressure. However, both the vascular moderating effects and the safety and acceptability of almond consumption should be further investigated in large, randomized, double-blind, placebo-controlled trials of longer duration, across age groups.

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Evaluation of Nutritional Factors Related to Glycemic Control in Patients with Cystic Fibrosis

Marzie Katanbafnejad*

Introduction

Cystic Fibrosis (CF) is a serious genetic condition that causes severe damage to the respiratory and digestive systems. This damage often results from a buildup of thick, sticky mucus in the organs (1).

CF is more common in whites than in northern Europe, North America, and Australia while Asia has the lowest prevalence rate. Its prevalence is equal in men and women (2).

The CF gene encodes a polypeptide called Cystic fibrosis transmembrane conductance regulator (CFTR) (4). More than 2,000 different mutations have been identified in the CFTR gene. The type of gene mutation is associated with the severity of CF (3).

CFTR is a chloride channel located on the surface of epithelial cells that moves water and salt on either side of the membrane. Mutations in CFTR disrupt the balance of chlorine and water, causing the mucosa to thicken and stick to various organs (4,5).

Symptoms include salty skin taste, wheezing or asthma, cough with sputum, lung infection, nasal polyps, chronic sinus infections, difficulty defecating, weight gain and poor growth despite excessive appetite, infertility in men and women, and prolapse rectum (4,5). CF-related diabetes (CFRD) is the most common complication of this disease (9).

Neonatal screening tests, sweat tests (gold standard), and DNA tests can be used to diagnose the disease (5,6).

At present, there is no definitive cure and we can only manage and reduce the disease and improve the quality of life through medication, airway cleansing techniques (ACTS), treatment regimen, and surgery. The type and severity of CF symptoms vary from person to person, therefore, the treatment plan is tailored to each individual's condition (6,7).

The diet of these patients is a high-calorie, high-salt diet. These patients should consume plenty of fluids and vitamins A, D, K and E daily. (8).

CF-related diabetes mellitus (CFRD) typically develops between ages 18 and 21, with 50% of those with CF developing it by age 30 (10). Thick mucosa in the pancreas causes damage to the endocrine part of the pancreas and not enough insulin is produced. Cases other than mechanical disorders of the islets of Langerhans can cause CF-related diabetes in these patients. The direct effect of CFTR mutation on insulin secretion from pancreatic cells can be mentioned. The recommended management for CFRD is insulin therapy (11).

Given that CFRD increases morbidity and mortality in the CF population, careful control of blood sugar in these individuals is important Therefore, the aim of the seminar is the Evaluation of nutritional factors related to glycemic control in patients with cystic fibrosis.

Literature review

In 2016, Ziaei et al conducted a cross-sectional study on 36 adult patients with CF to examine the relationship between the percentage of macrocephaly in the diet and total energy expenditure (TEE) with glycemic fluctuation indices.Based on the results of this study, patients with CF with normal and impaired glucose tolerance had fewer glucose fluctuations than patients with CFRD ($P<0.05$) and energy, the proportion of carbohydrates, of fat and

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protein, TEE or the number of footsteps walked did not affect glucose fluctuation indexes (P>0.05)(12).

Coriati et al were Conducted a cross-sectional study of 178 adults with CF. This study aimed to investigate the relationship between vitamin D and glucose tolerance and indices of secretion and insulin resistance. Finally, on the analysis of the relationship between 25 (OH) D and glucose metabolism parameters, no correlation was observed(13).

Stonestreet et al. Conducted a cross-sectional study to examine determining the prevalence of CFRD individuals performing carbohydrate counts (CC) and determining barriers to CC use and CC accuracy in CFRD adults. 17 adults with CFRD participated in this study. Of those patients recruited to this study, 59% used CC as a tool to guide insulin dosing, and patients estimated accurate carbohydrate values in only 46% of meals(14).

Armaghanian et al. Conducted a clinical trial study on 18 CF patients. This study aimed to determine the quality of food in the clinical condition of CF patients and compare it with the Australian diet guidelines as well as to investigate the relationships between diet including glycemic index (GI) glycemic load (GL) and glucose response variables using continuous glucose monitoring) CGM(. Adults attending a Sydney hospital were recruited to undergo CGM for five-seven days and 13 records dietary intake using a food record over the CGM period. This study suggests GI and GL may be important dietary factors influencing glucose metabolism in CF (15).

A double-blind randomized clinical trial study was conducted by Gorji et al. In this study, 44 children and adolescents with CF were randomized to receive for three months either high fat, high-calorie diet (n=22) or a low glycemic index/high fat, high-calorie diet (n=22) with similar calorie and macronutrients composition. It seems that adherence to a low glycemic index/high fat, high-calorie diet can improve glycemic indices in children and adolescents with CF compared to the high fat, high-calorie diet (16).

Discussion

Here are three mechanisms:

1. In a diet with a low GI, carbohydrates are digested and absorbed at a slower rate, which results in a gradual increase in blood sugar that reduces postprandial glycemic fluctuations. On the other hand, a gradual increase in blood sugar reduces the secreted dose of insulin and reduces the load on the cells of the pancreas, and delays the progression of CFRD (15,16,17).

2. Vitamin D binds to the vitamin D receptor (VDR) in the nucleus to increase insulin protein gene expression and increase insulin production. Vitamin D, on the other hand, increases the expression of the clobindine protein gene and in this way, calcium ions enter the cells and stimulate the cells to secrete insulin (2)

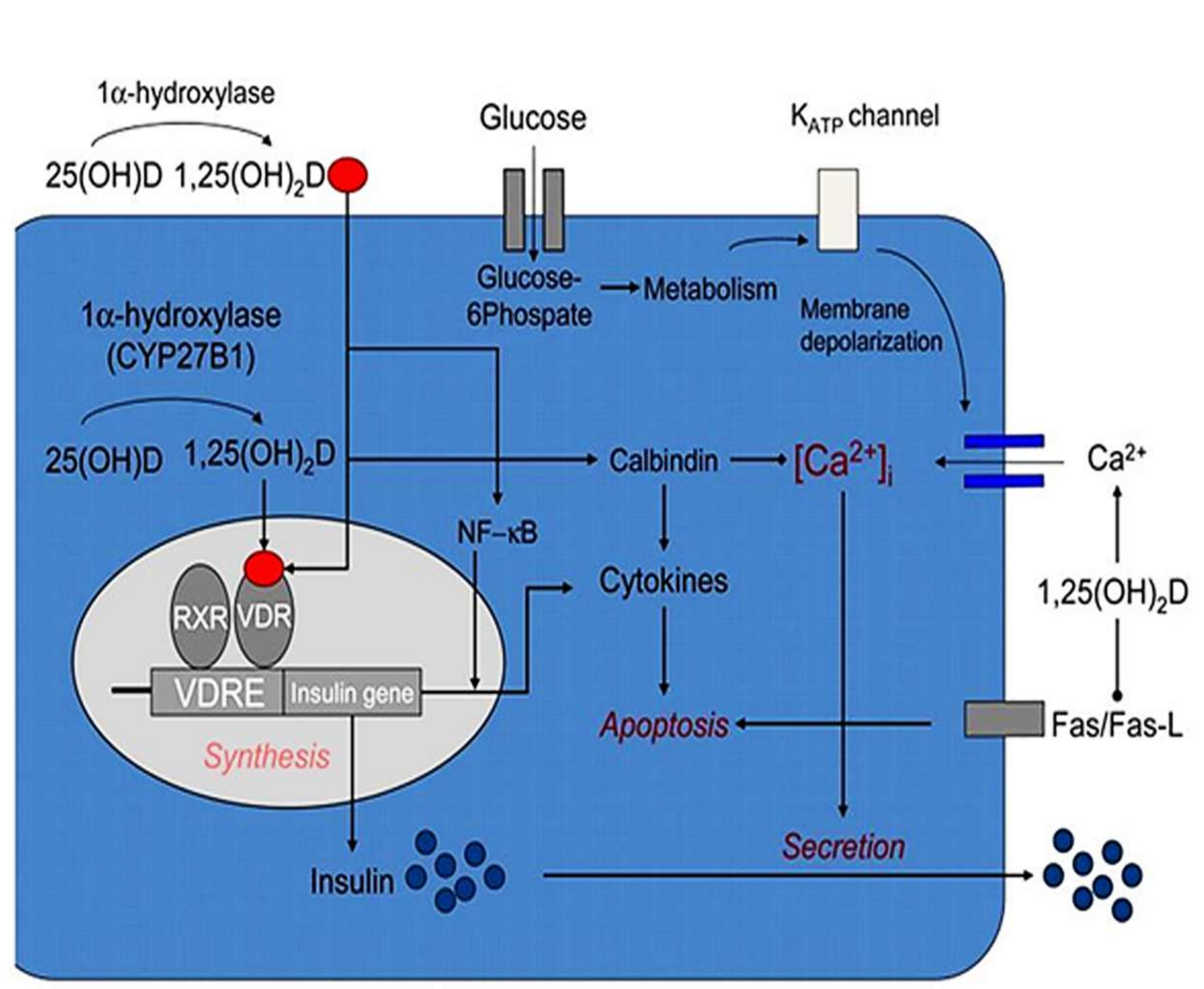

Figure 1. Mechanism of action of vitamin D on insulin secretion and production (2)

3. CC can help determine the right dose of insulin in these people, which helps control glycemia (14).

Conclusion

According to studies, 3 conclusions can be drawn:

1. It seems that a diet with a low GI can help improve the glycemic status of CF individuals and play an important role in the management of CFRD-related complications.

2. Although no correlation was found between vitamin D and glucose metabolism parameters. The idea that vitamin D supplementation may improve glucose tolerance cannot be ignored.

3. Accurate CC can help determine the right dose of insulin and control glycemia.

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Early Childhood Feeding Practices and Dental Caries

Maryam Shojasiahi*

Introduction

Early childhood caries (ECC) is a chronic disease that affects a child's general state of health(1).

ECC is characterized by the presence of one or more primary teeth with caries, in the form of lesions that are cavitary or not, or teeth that are missing or filled because of caries, in preschool children aged from 0 to 60 months. In children younger than 3 years, severe forms of ECC (S-ECC) are defined by signs of caries on the smooth surfaces of teeth(1).

A review of the literature suggests that in most developed countries the prevalence rate of ECC is between 1 and 12%. In less developed countries, the prevalence has been reported to be as high as 70% (2).

The prevalence of caries in deciduous teeth in Iranian children is 62.8%with the highest prevalence in Ahvaz with 87% and the lowest prevalence in Hamedan with 31% (3).

Carious lesions are often painless, but if caries progress from the dentin to the dental pulp, they can lead to pulpitis. This inflammation can be reversible and can be controlled with a dental restoration, but if the inflammation is not reversible, the patient complains of spontaneous pain(4).

There is evidence that ECC has a negative impact on weight gain, growth, and quality of life. In addition, this group of children is at risk of acute infections, which can lead to life-threatening systemic illnesses. They are often prescribed recurrent courses of antibiotics and may be exposed to the risks and side effects of procedures under general anesthetic (4).

Diagnosis of dental caries involves a systematic visual examination, ensuring that the teeth are clean and dry and that there is adequate lighting. A radiographic investigation is often required (5).

Tooth decay is a multifactorial infectious disease such as bacterial factors, social environment, oral health, and diet (1).

This study aims to investigate the effect of food intake on dental caries.

Literature review

Alshehri et al. examined a cross-sectional study to assess the potential association of breastfeeding and some other factors with the risk for ECC among young children in Saudi Arabia in 2015 and the conclusion of this study was children that were breastfed by mixed breastfeeding experienced caries the most and the shorter the duration of breastfeeding the higher the prevalence of caries (6).

Kato et al. examined a longitudinal survey to investigate the association between breastfeeding duration during the first half-year of life and the risk of ECC from the age of 30 to 66 months in Japan in 2015 and it indicates that the duration of breastfeeding for at least 6-7 months increases the risk of dental caries at the age of 30 months (7).

Wong et al. examined a cross-sectional study to determine if there is an association between longer breastfeeding duration and dental caries in healthy urban children in Canada in 2016 and this study showed that the longer the duration of breastfeeding, the higher the risk of dental caries (8).

Dabawala et al. examined a case-control study To study the type of parenting style and oral health practices as risk factors among children

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with ECC in an Indian preschool population in 2016 and the association of parenting style with ECC cannot be confirmed based on the results of the study also lower socioeconomic status and increased birth order of the child significantly increase the risk for ECC. The oral health practices which are significant risk factors for ECC are as follows: lack of knowledge and failure to use fluoridated toothpaste; breast or bottle-feeding duration more than 12 months of age; use of formula milk or cow's milk with sugar in the bottle with which the child falls asleep at night; higher frequency of solid, sticky, and slowly dissolving sugar-containing foods in the diet; visit a dentist only when a problem is perceived rather than for preventive dental checkups (9).

Felden et al. examined a cohort study aimed to investigate the associations between feeding frequency at age 12 months and caries prevalence at age 3 years in Brazil in 2018 this indicated that the higher the number of feedings in late infancy (12 months), the greater the incidence of dental caries in childhood (10).

Discussion

A dental plaque typically forms in four successive stages: attaching a microorganism to a surface and multiplying, forming a colony, secreting extracellular polymeric substance (EPS) and maturing the colony, forming plaque, and finally scattering some of these plaque cells. To eventually form a colony elsewhere in the body and this cycle continues (11).

Streptococcus mutans, in the dental plaque, ferment carbohydrates into organic acid lowering the pH level at the enamel surface. If the pH falls below 5.5 (critical pH), the tooth structure can demineralize. When carbohydrate substrates are no longer available and the acids are neutralized by saliva, remineralization can occur. This is a natural physiological process, however, if the net demineralization outweighs remineralization a carious lesion develops (4).

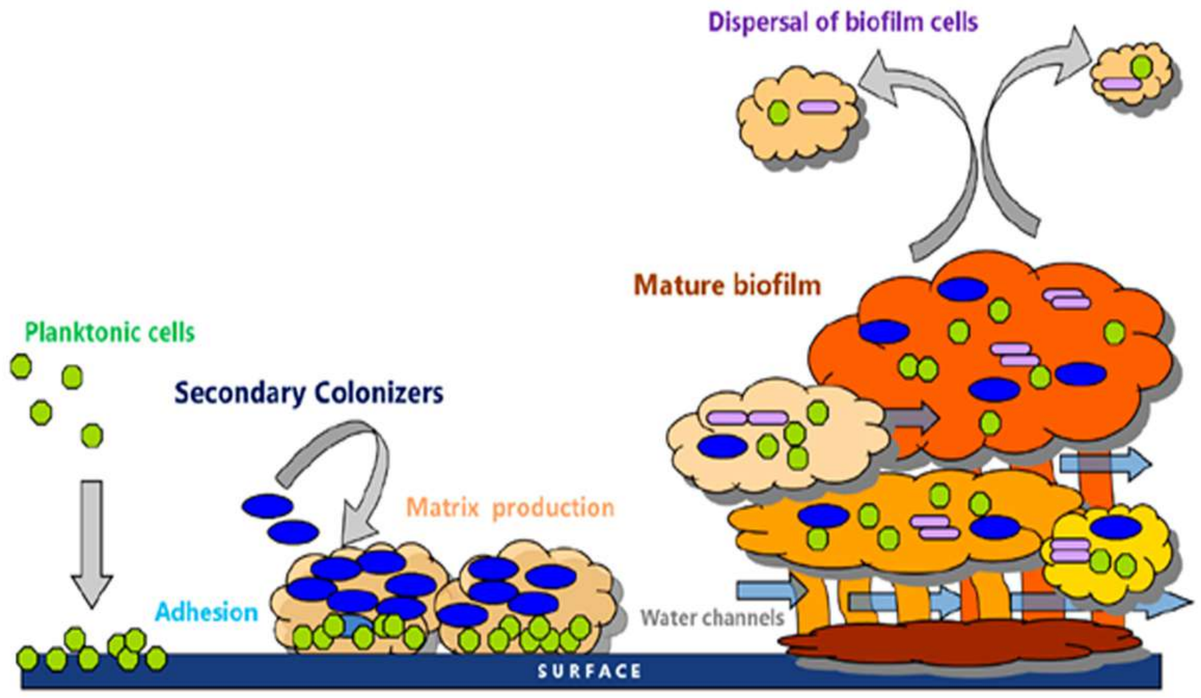

Figure 1. Sequential events taken place during microbial biofilm formation (Reproduced with permission from Seneviratne *et al.*,2008).

Conclusion

Breastfeeding in the first year of life not only does not increase the risk of ECC but also has a protective role, but after 1 year, the longer the duration of breastfeeding, the higher the risk of ECC.

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Association Between Dietary Acid Load And The Risk of Metabolic Syndrome

Haleh Akhtari*

Introduction

Metabolic syndrome (MetS) is a cluster of Cardiovascular Disease (CVD) and Type 2 Diabetes Mellitus (T2DM) risk factors(1). MetS is defined as having three criteria out of five components. These components are elevated blood glucose, blood pressure (BP) and triglyceride levels, and low HDL-C levels, plus central obesity. Hypertriglyceridemia was defined as plasma TG concentration more than 150 mg/dL, elevated blood glucose was considered as fasting blood sugar 100≤ mg/dL, and hypertension was defined as SBP over 130 mmHg or DBP over 85 mmHg. Low HDL-C and abdominal obesity were differently defined in men and women. In women, serum HDL-C<50 mg/dL and a waist circumference greater than 88 cm, and in men serum HDL-C<40 mg/dL and a waist circumference greater than 102 cm are defined as low HDL-C and abdominal adiposity, respectively(2). The prevalence of MetS is more than 30% among apparently healthy Iranian adults(1). The worldwide prevalence of MetS among adults is reported to be 25% (4).

The risk factors of MetS are: age, ethnicity (in the United States, Hispanics — especially Hispanic women — appear to be at the greatest risk of developing metabolic syndrome), obesity (especially Abdominal obesity increases the risk of metabolic syndrome), diabetes (such as gestational diabetes or having a family history of type 2 diabetes), other diseases like nonalcoholic fatty liver disease, polycystic ovarian syndrome, or sleep apnea (3).

To prevent MetS, it is recommended to have a healthy lifestyle including: Getting at least 30 minutes of physical activity most days, Eating plenty of vegetables, fruits, lean protein, and whole grains, Limiting saturated fat and salt in the diet, Maintaining a healthy weight, and Not smoking (4).

Dietary acid load (DAL) includes the potential renal acid load (PRAL) score and the net endogenous acid production (NEAP) score. The PRAL score is based on protein, acidogenic and alkalinogenic ions besides taking into account their intestinal absorption rate(4).

NEAP includes total intakes of protein and organic anions, such as citrate and acetate, which are naturally bound to cations, such as potassium. Both scores have been validated against objective measures of acid-base load determined from 24-h urine (4).High-DAL score may reflect a higher consumption of animal products and processed foods (4).

Dietary acid-base load is a food factor that has recently been considered as a potential risk factor for metabolic disorders and cardiovascular disease (5).

The aim of this study is the effect of the acidic load of food on the rate of metabolic syndrome.

Literature review

In a cross-sectional study in 2020, 1430 people aimed to investigate the relationship between dietary boric acid and the risk of Mets in Iran. The result showed a lack of significant correlation between increased dietary acid load and the risk of metabolic syndrome (1).

In 2019, a cross-sectional study on 371 women aged 20 to 50 years by Mozaffari et al. aimed to evaluate the relationship between dietary acid load and CVD risk factors, and the prevalence of

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metabolic syndrome in women. The study found that women consuming more carbohydrates, fruits, vegetables, potassium, and magnesium had lower acid loads. Despite the increased risk of metabolic syndrome with an increasing acidic load of food, this increase has not been statistically significant (P=0.01) (4).

A cross-sectional study was conducted in 2015 by Iwase et al. in Japan on 149 patients with type 2 diabetes. This study aimed to determine the effect of animal/plant protein intake and dietary acid load on metabolic syndrome risk factors in patients with type 2 diabetes. The result demonstrated that People with type 2 diabetes who had a higher protein-to-energy ratio of plant protein than animal protein had a 78% lower risk of developing metabolic syndrome and a significant positive correlation was observed between dietary acid load (PRAL and NEAP indices) with the prevalence of metabolic syndrome (P=0.0384, P=0.0098) (6).

Bahadoran et al. conducted a cross-sectional study in 2015, in Iran to investigate the relationship between dietary acid load and cardiometabolic risk factors in 5620 adult۔ men and women (Tehran Lipid and Glucose Study). The result of this study is as follows: A significant positive correlation was observed between PRAL and weight, waist circumference, serum triglyceride, diastolic blood pressure, and serum creatinine (P<0.05). In conclusion, an increased dietary acid load can be one of the risk factors for metabolic risks (5).

Discussion

Obesity: As it is shown in Fig. 1, with increasing consumption of foods with a high acid load, the amount of cortisol increases, which causes increased inflammation and leptin, decreased adiponectin, and sensitivity to insulin and lipolysis, all of which increase visceral obesity and it leads to metabolic syndrome (7).

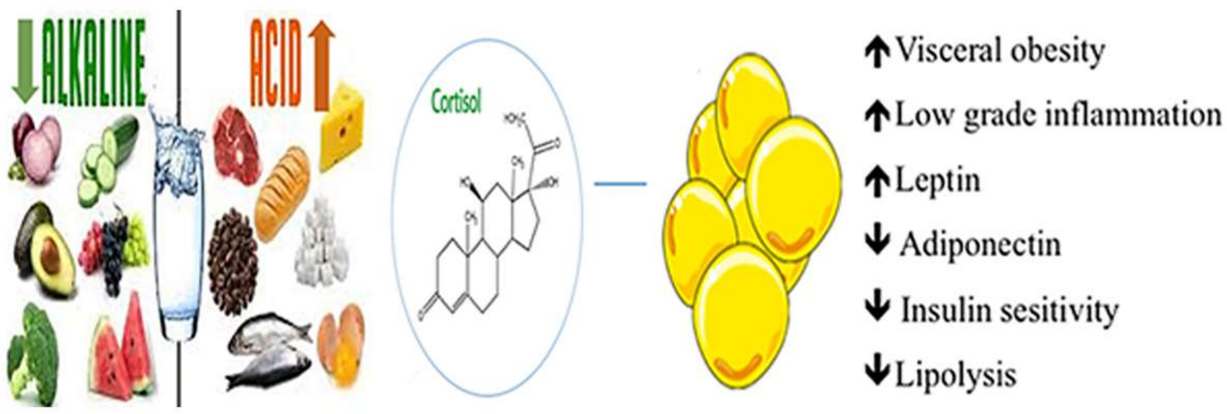

Figure 1. Association between obesity and metabolic syndrome (7)

Diabetes: Increased intake of acidic foods increases cortisol levels and the acid load, which decreases insulin-IGF signaling Glut 4 translocation, Increased insulin and decreases insulin sensitivity, all of which increase the risk of diabetes (10).

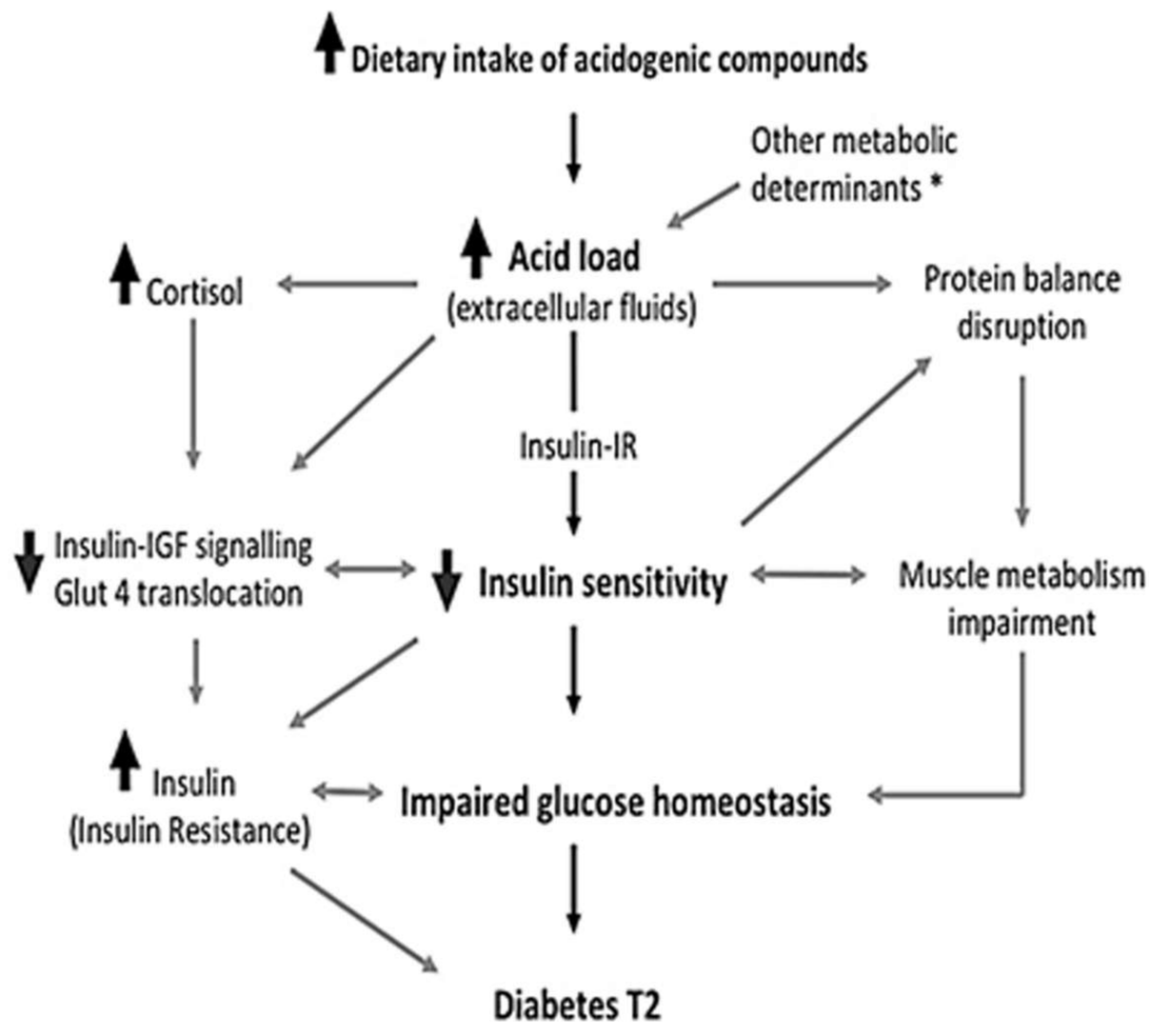

Figure 2. How the effect of dietary intake of an acidogenic compound on the incidence of type2 diabetes (8)

High blood pressure: Metabolic acidosis with Increased insulin resistance, Increased Angiotensin II, Increased Aldosterone Synthesis, decreases Prostaglandins and Increased Renal Sodium retention, Increases blood pressure (9).

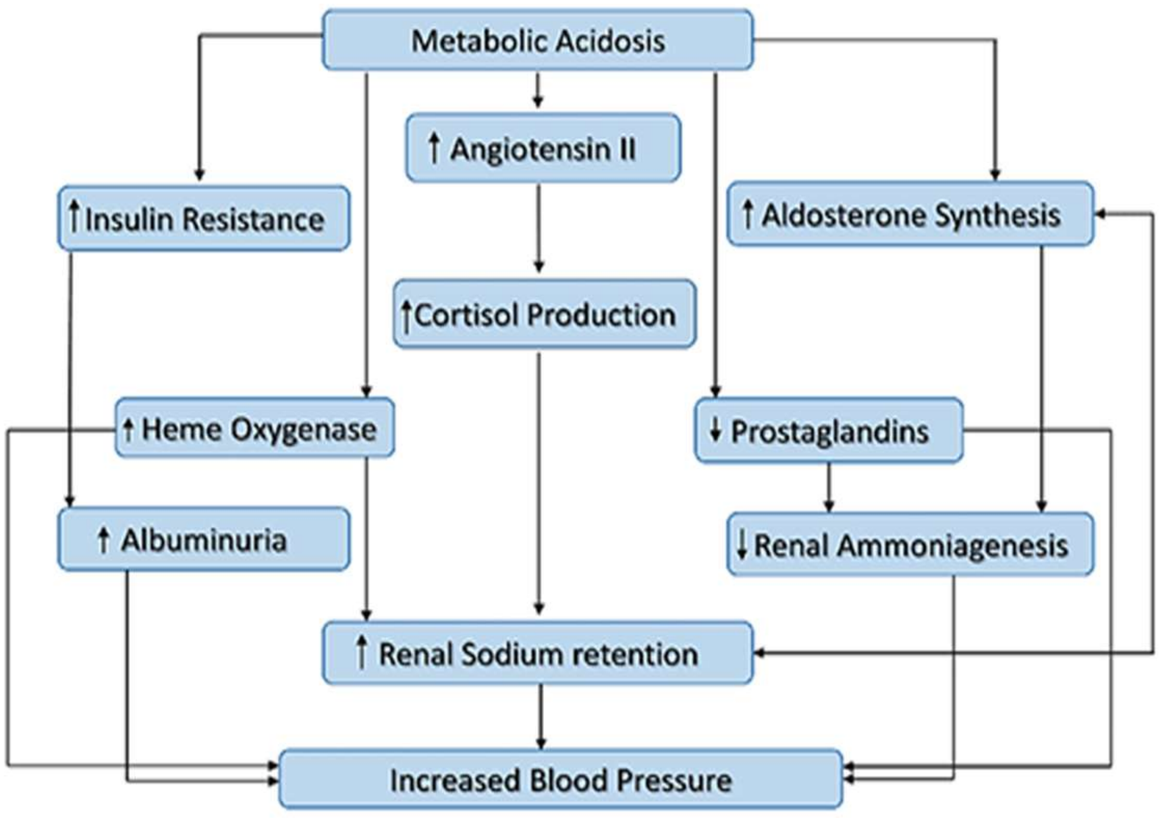

Figure 3. The relationship between metabolic acidosis and high blood pressure (9)

Conclusion

Although some studies have observed no significant relationship between increased acid load and the incidence of metabolic syndrome, others have proposed increased dietary acid load as a risk factor for metabolic syndrome (1,4,5,10).

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Kind Regards
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