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Dietary and Lifestyle Risk Factors and Metabolic Syndrome: Literature Review

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Abstract

Metabolic syndrome (MetS) is considered a threat to public health due to its rapid growing prevalence worldwide. MetS can result from interrelated metabolic abnormalities including insulin resistance (IR), hypertension, dyslipidemia, and abdominal adiposity. Although the pathogenesis of this syndrome is not distinctly understood, it is strongly influenced by multiple genetic variations that interact with many environmental factors such as positive family history of MetS, adherence to unhealthy dietary patterns, low physical activity and smoking and that explain the variations in the prevalence of the MetS within and across populations. All of these factors were found to be associated with IR, obesity, and triglycerides elevation which therefore increase the risk of the MetS Several studies highlighted the effective preventive approach includes lifestyle changes, primarily losing weight, adopting healthy diet, and practicing exercise. All of the mentioned factors can reduce the risk of MetS.

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List of Abbreviations

BMI: Body Mass Index
CVD: Cardio Vascular Diseases
DASH: Dietary Approach to Stop Hypertension
DBP: Diastolic Blood Pressure
DLP: Dyslipidemia
DM II: Type 2 Diabetes Mellitus
FBG: Fasting Blood Glucose
FFA: Free Fatty Acid
FFQ: Food Frequency Questionnaire
GI: Glycemic Index
GL: Glycemic Load
HDL-C: High Density Lipoprotein Cholesterol
HTN: Hypertension
IDF: International Diabetes Federation
IR: Insulin Resistance
LDL: Low Density Lipoprotein
LPL: Lipoprotein Lipase
MetS: Metabolic Syndrome
MUFA: Monounsaturated Fatty Acids
NCEP: ATPIII: National Cholesterol Education Program: Adult Treatment Panel III
OR: Odd Ratio
PA: Physical Activity
PUFA: Polyunsaturated Fatty acids

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Introduction

The metabolic syndrome (MetS) is a precursor of both cardiovascular diseases (CVD) and diabetes. It results from a general imbalance of the body's metabolic processes that increases cardiovascular morbidity, mortality, and all-cause mortality.[1] It has been recently considered as a target topic for research by health care professionals, due to its effect on the quality of life. In addition, its prevalence rate is increasing in a rapid way all over the world.[2] Many factors influence the determination of the global prevalence of MetS, such as region, urban or rural environment, as well as the diagnostic criteria used to define MetS[3]. Also MetS definition is influenced by other parameters such as age- as its prevalence increases with age although it may be present in children and adolescents- and sex (the prevalence of MetS in women is greater than in men).[4] In general, the worldwide prevalence of MetS ranges from <10% to as much as 84%.[4,5] In the United States, it is estimated that 35% of adults, and up to 50% of the population who are over 60 years, were diagnosed to have MetS (30.3% in men and 35.6% in women).[6] Mexican-American women were reported to have the highest MetS prevalence.[7] Depending on The International Diabetes Federation (IDF) diagnostic criteria of MetS, European prevalence has been estimated as 41% in men, and 38% in women.[8] In Mediterranean countries, the prevalence ranges from 0.2% to 45.7% in adults <65 years of age, reaching up to 25% to 55.2% in adults >65 years.[9] In Jordan as one of these Mediterranean countries, cross-sectional studies conducted to determine the prevalence of MetS and its components among Jordanians showed that the estimated prevalence rate in Jordan according to the IDF criteria was alarming at 51%, and it was higher in women (46.4% in men and 55.3% in women).[10,11] Limited studies are presented in the literature that evaluates the relationship between dietary and lifestyle risk factors and the risk of MetS. Therefore, the objective of this review was to evaluate the association between macro- and micronutrients intake, eating patterns, physical activity (PA) and smoking and the risk of developing MetS.

Literature Search

A literature search was conducted using PubMed, Google Scholar, and Sciencedirect for "metabolic syndrome", "lifestyle risk factors", "dietary patterns" and "macro-micronutrients'.

Method and Design

Electronic databases were searched from January 2018 until June 2018 for studies investigating the association between dietary and lifestyle risk factors and metabolic syndrome.

Definition of MetS

The concept of MetS is well-known for many years, but there is a lack of a universally agreed definition of this syndrome. Three definitions of the MetS are currently in use and proposed by different health authorities: World Health Organization (WHO) in 1999, the National Cholesterol Education Program: Adult Treatment Panel III (NCEP: ATP III) in 2001, and the IDF in 2006.[12]

The WHO was the first organization to produce diagnostic criteria for MetS, the criteria included: aside from glucose tolerance status and IR, at least two of any other MetS risk factors should be present such as: central obesity aswaist to hip ratio(WHpR) ≥0.90 for males or ≥0.85 for females and/or body mass index (BMI) ≥30 Kg/m², blood pressure ≥140/90 mmHg, and/or high-density lipoprotein cholesterol (HDL-C)<35 mg/dl in males or <39 mg/dl in females, dyslipidemia as TG ≥150 mg/dl and microalbumenurea ≥20 μg min^{-1} or albumin: creatinine ratio ≥30 mg g^{-1}.

But it was found that insulin resistance (IR) and microalbuminuria measurements are laborious and cannot be practically used in clinics. Thus, ATP III has released its definition of the MetS in 2001.[13] The ATP III focused less on type 2 diabetes mellitus (DM II) and more on CVD risk as compared to the WHO definition.[14]

Therefore, ATPIII recommends the presence of any three of five components to diagnose MetS: abdominal obesity as waist circumference (WC) ≥102 cm for males or ≥88 cm for females, high blood pressure ≥130/85 mmHg, fasting hyperglycemia ≥110 mg/dl, low HDL-C <40 mg/dl in males or <50 mg/dl in females, and raised TG ≥150 mg/dl.[15]

However, a strong demand for a universal practical definition of MetS that can identify people who are

at high risk of CVD and diabetes is still needed. Thus, the IDF has established its own criteria for the definition of MetS due to the persistent need for a single, universally accepted diagnostic tool that is easy to use in clinical practice.[16] They defined patients with metabolic syndrome as suffering from central obesity (defined as WC ≥94 cm in males and ≥80 cm in females) plus any two of the following four factors: elevated triglycerides (TG) levels (≥150 mg/dL) or specific treatment for this lipid abnormality; lowered high-density lipoprotein cholesterol (HDL-C) (< 40 mg/dL in males, <50 mg/dL in females) or specific treatment for this lipid abnormality; elevated blood pressure: (systolic blood pressure (SBP) ≥130 or diastolic blood pressure (DBP) ≥85 mm Hg) or treatment of previously diagnosed hypertension; or elevated fasting blood glucose (FBG ≥100 mg/dL) , or previously diagnosed DM II.[16]

However, the three aforementioned definitions of MetS by the health authorities agreed on the presence of central obesity, hyperglycemia, hypertension (HTN), and dyslipidemia (DLP) as key risk factors for MetS.[17,18,19] Table (1) shows the harmonized definition of MetS for these three authorities.[20]

Pathophysiology of MetS

As aforementioned, many risk factors are related to MetS, thus it is considered to bea subject of debate. The clear mechanism or pathophysiology of this syndrome is still not well-determined, but the main causes are agreed to be IR and central obesity[21,22] These two factors lead to the development of other metabolic risk factors like hyperglycemia, DLP, and HTN which may cause CVD and DM II later on.[21,23]

Insulin Resistance

IR is considered to be the most accepted hypothesis to describe the pathophysiology of MetS.[3] IR is a physiological condition in which beta-cells secrete normal amounts of insulin hormone but are unable to respond normally in the target tissues of liver, skeletal muscle and adipocytes. This reduced responsiveness is a major precursor of DM II.[3]

Table 1: Harmonized Definition of MetS

Risk Factors	Cut- off Points
Elevated WC*	Population-and country-specific definitions
Elevated TG or specific treatment for this lipid abnormality	≥150 mg/dL (1.7 mmol/L)
Reduced HDL-C or specific treatment for this lipid abnormality	<40 mg/dl (1.0 mmol/L) in males; <50 mg/dl (1.3 mmol/L) in females
Elevated blood pressure (treatment of previously diagnosed HTN)	Systolic blood pressure (SBP) ≥ 130 and/or diastolic blood pressure (DBP) ≥85 mm Hg
Elevated FBG (≥ 100 mg/dL), or previously diagnosed DM II	≥100 mg/dL

*It is recommended that the IDF cut off points be used for non-Europeans and either the IDF or AHA/NHLBI cut off points used for people of European origin until more data are available.
Adopted from Alberti *et al.,* 2009.

** Abbreviation: WC: waist circumference; TG: triglycerides; HDL-C: high density lipoprotein cholesterol; HTN: hypertension; DM II: type 2 diabetes mellitus.

IR in skeletal muscle promotes reduction in glycogen synthesis and intracellular glucose transport, while in the liver it impairs insulin signaling pathways; however, discordant to this observation is evidence that hepatic lipogenesis continues.[24]

Ferris & Kahn, (2016) suggested that insulin secretion decreases fat lipolysis, gluconeogenesis, and TG secretion from liver. In addition, insulin acts on the brain, which independently reduces lipolysis through suppression of sympathetic outflow and raises hepatic TG secretion through an unknown mechanism.[24] In diabetes, the decrease in insulin action on the brain and fat leads to an elevation in lipolysis. As a result, this will promote the supply of free fatty acids (FFA) to the liver which in turn will increase TG synthesis. On the other hand, the impairment of insulin action of the liver will result in increased gluconeogenesis.[24] Figure 1 (A, B) illustrates the difference between (A) normal physiology vs. (B) diabetic condition regarding insulin action.

Obesity and Abdominal Obesity

Overweight and obesity are defined as abnormal or excessive fat accumulation that presents a risk to health.[25] Abdominal obesity is a major independent risk factor of insulin sensitivity, impaired glucose tolerance elevated blood pressure and DLP seen in the MetS.[26,27] Overweight and obesity are associated with an increased risk for CVD, HTN, DM II, certain cancers and many other disorders.[28]

It has been assumed that adipose tissue releases an excess of fatty acids and cytokines that induce insulin resistance.[29] The release of these excessive FFA also induces lipotoxicity, as lipids and their metabolites create oxidative stress to the endoplasmic reticulum and mitochondria.[30] This affects adipose as well as non-adipose tissue, accounting for its pathophysiology in many organs, such as the liver and pancreas, and in the MetS.[29] "In addition, these FFA released from excessively stored triacylglycerol deposits contribute to hypertriglyceridemia by the inhibition of lipogenesis, which prevents adequate clearance of serum triacylglycerol levels."[31] Consequently, the secretion of FFA by endothelial lipoprotein lipase (LPL) from elevated blood TG within increased βlipoproteins will lead to lipotoxicity which results in insulin-receptor dysfunction. IR state then will follow and cause hyperglycemia with compensated hepatic gluconeogenesis. The later consequences will increase glucose output from the liver which will enhance the hyperglycemia caused by IR. FFA also will decrease the utilization

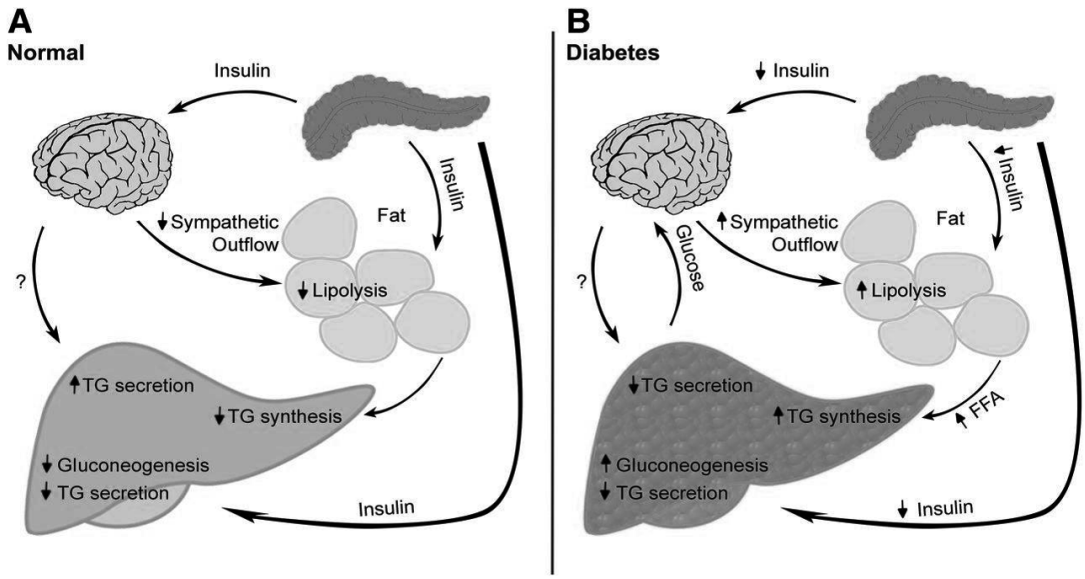


Fig. 1: (A) Normal Physiology. (B) Diabetic Condition Regarding Insulin Action.
*Abbreviation: TG: triglycerides; FFA: free fatty acids. **Adopted from Ferris & Kahn, (2016).

of insulin-stimulated muscle glucose, contributing further to hyperglycemia.[29] As a result, lipotoxicity will decrease the secretion of pancreatic β-cell insulin, which eventually results in β-cell exhaustion.[31] Grundy (2004) has explained the relationship between obesity and IR by which "obesity causes insulin resistance, whereas insulin resistance seemingly exacerbates the adverse effects of obesity".[29] Figure (2) illustrates the role of lipotoxicity and inflammation in obesity.

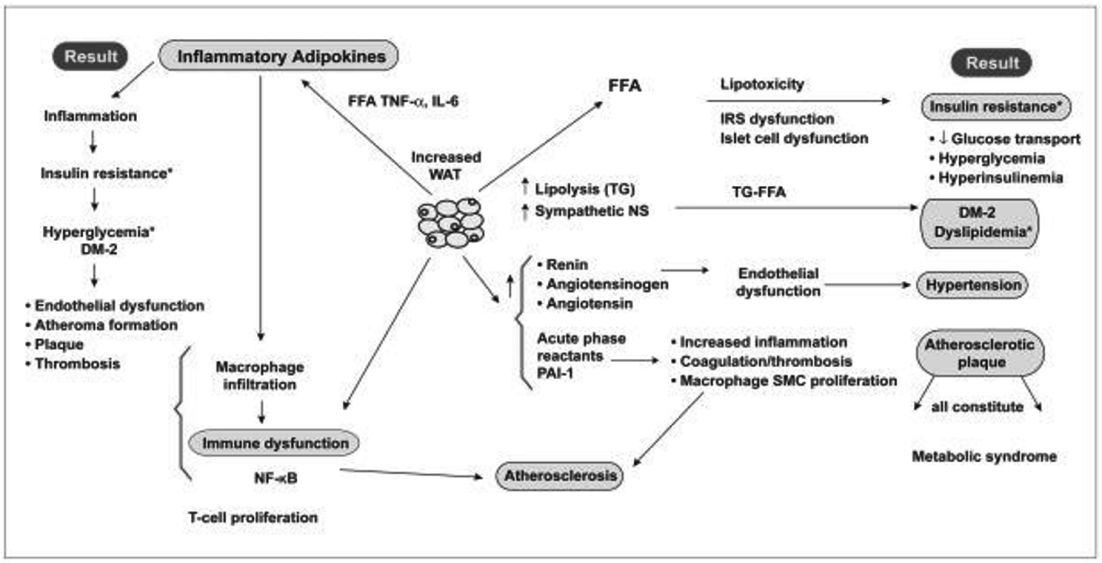
Anthropometric Measurements and MetS

MetS was strongly found to be linked to body weight, height and waist and hip circumferences. It is not only the use of these measurements that is important in this prediction, but also the estimation of the percentage of fat in the body.[32] A study conducted by Khader *et al.,* (2010) to determine cutoff values of anthropometric measurements as indicators of metabolic abnormalities among Jordanians; BMI, WC, and waist to height ratio (WHtR) were found to be associated with CVD risk factors, with WHtR being the better predictor.[10] In addition, Sagun *et al.,* (2014) conducted a study to assess the association between not routinely used body measurements (e.g., mid-upper arm, forearm, and calf circumferences) and MetS.[33] The authors concluded that WC was not associated to MetS in obese and overweight subjects. However, forearm circumference was associated with MetS and visceral fat measured by bioelectric impedance, hip circumference, and WHpR.[33] However, Wahrenberg *et al.,* (2005) suggested that a WC of <100 cm prevents individuals of both sexes from being at risk of IR. Also the authors reported WC is a strong simple measurement for identifying individuals who are at high risk of IR and MetS, so it replaces BMI, WHpR, and other measures of total body fat as a predictor of IR.[34]

Lifestyle Risk Factors

Smoking

Smoking and physical inactivity have been identified as important modifiable risk factors for MetS and



White adipose tissue (WAT) releases pre–fatty acids and adipokines, which are lipotoxic and inflammatory and result in diverse effects, outlined in the left-hand columns. Their correlation to the metabolic syndrome is shown on the right-hand column, whereas all the effects culminate in atherosclerosis on the bottom of the figure.

***** Perturbed glucose and lipid metabolism.

DM-2: diabetes mellitus-2; FFA: free fatty acids; IL: interleukin; IRS: insulin receptor substrate; NF-KB: nuclear factor kappa beta; NS: nervous system; PAI-1: plasminogen activator inhibitor 1; SMC: smooth muscle cell; TNF: tumor necrosis factor

****** Adopted from Unger *et al.,* 1995.

Fig. 2: Role of Lipotoxicity and Inflammation in Obesity

its consequences.[35,36] Several studies have shown that smoking is considered as a major risk factor for CVD and DM II and it is also associated with metabolic abnormalities and increases the risk of MetS.[37,38,39] Tobacco smokers had a 1.07–1.66-fold greater risk of developing MetS than non-smokers as reported by Nakanishi *et al.,* (2005).[38] Weitzman *et al.,* (2005) conducted a study to find the relationship between tobacco smoking and the severity of MetS.[40] They found that both active and passive smoking may increase the risk of MetS among adolescents who are overweight or at risk of overweight.

The mechanism by which cigarette smoking can affect glucose and lipid metabolism may partly attribute to stimulation of sympathetic nervous system.[41] Nicotine released during smoking stimulates the release of several neurotransmitters and hormones such as cortisol, growth hormone and others.[42]

This increased cortisol production in current smokers may lead to having higher WC which results in accumulation of abdominal fat.[43] In addition to the hormone disturbance; it has been shown that endothelial dysfunction and its related arterial compliance reduction were more serious in smokers than nonsmokers. Besides, smokers tend to have IR due to the effects of cotinine (a metabolite of nicotine), carbon monoxide, cortisol, and growth hormone[44], and hence could contribute to development and deterioration of metabolic syndrome.[41]

On the other hand, Harris *et al.,* (2016) demonstrated that smoking cessation may also lead to the development of MetS due to the increased food intake which results in weight gain.[45] It is proposed that nicotine has the ability to suppress appetite, but in the condition of smoking cessation this ability is reversed.[46] In this case, replacing the rewards of food with the rewards of cigarettes will take place.[47] Absence of nicotine raises the rewarding value of food and subsequently increases the intake of food rich in fat and sugar[48] and may lead to excessive intake snacks that are high in carbohydrates and sugar.[49] Additionally, nicotine and/or smoking help control compulsive eating and overeating; during post-cessation these activities are inhibited.[50]

Physical Inactivity

Sedentary PA is one of the major modifiable risk factors for the MetS.[51] The second leading cause of premature morbidity and mortality is excess body weight gain due to poor diet and insufficient PA.[52] PA is associated with many health-related benefits, including a reduced risk of developing several chronic diseases such as obesity,[53] CVD,[54] DM II,[55] MetS.[56,57]

The adverse effect of physical inactivity on MetS components is thought to be due to reduced energy expenditure which results in increased energy intake. Cross-sectional studies reported an inverse association between PA and MetS.[56,58,59]

Most of the guidelines support that at least 150 minutes of moderate-intensity PA per week could be associated with a lower prevalence of MetS.[60]

Dietary and Food Patterns and MetS

Many studies were conducted to examine the dietary patterns that may be associated with MetS and its components.[2,61,62] One of the dietary patterns that are known to be associated with reducing MetS is the Mediterranean Diet.[62] The Mediterranean diet was first defined by Ancel Keys as the diet usually consumed among the populations bordering the Mediterranean Sea.[63] It is characterized by the consumption of fruit, vegetables, nuts, olive oil, fish[64] and low consumption of saturated fat, red meat, processed meat, refined carbohydrates, and whole-fat dairy products.[63] Adherence to the Mediterranean Diet improves both physical and mental health and improves quality of life.[65] Also, it has been revealed that Mediterranean Diet may lower the risk of having elevated low-density lipoprotein cholesterol (LDL-C), blood glucose values,[66] and TG, and improves HDL-C levels.[67] Few cross-sectional studies found a strong effect of the adherence to the Mediterranean Diet on lowering the risk of MetS and all its components (a higher effect of the entire dietary pattern than to individual food components).[62,64] Some components of the Mediterranean Diet, such as olive oil and legumes[67], high fruit and vegetable consumption was generally found to be associated with lower prevalence of MetS.[68] A quantitative meta-analysis of twelve studies (eight observational cross-sectional and four prospective cohort) were pooled together to estimate the association between adherence to

the Mediterranean dietary pattern and MetS risk.[69] The findings of this meta-analysis suggested that high adherence to the Mediterranean diet was significantly associated with reduced risk of MetS. Also the adherence to this dietary pattern had a significant inverse association with some of MetS components such as: WC, blood pressure and low HDL-C levels, but the results were conflicting about blood glucose levels.[69]

Association between consumption of dairy products and the risk of MetS is controversial.[2,70,71] Fumeron *et al.,* (2011) conducted a prospective study which showed that high dairy intake was generally associated with reduced risk of MetS components.[70] A multicenter cohort study of 15105 adults was conducted in order to investigate the association of dairy consumption, types of dairy products and dairy fat content with MetS, dairy consumption was assessed by a food frequency questionnaire (FFQ).[71] The authors found out that full-fat dairy but not low fat intake was inversely and independently associated with MetS in middle-aged and older adults. Another 6-year prospective study that examined the effect of yogurt consumption on the risk of MetS concluded that there is no clear significant association between yogurt consumption and MetS and its components, but only with central adiposity which was inversely associated with high consumption of yogurt.[2] They also found that combination of high consumption of both yogurt and fruits had a significant inverse relationship with MetS risk. Minimally processed cereals appeared to be associated with decreased risk of MetS, while highly processed cereals with high glycemic index (GI) are associated with higher risk.[72]

A study was conducted by Kim *et al.,* (2011) to assess the association between usual dietary patterns and the risk of MetS in adults from South Korean. The authors found out that alcohol and meat dietary pattern was adversely associated with elevated blood pressure and hypertriglyceridemia.[73] On the other hand, after adjusting for possible confounding factors for developing MetS, fish, grains, and vegetables dietary pattern was associated with a reduction in the risk of hypertriglyceridemia and inversely with MetS risk.[73]

Dietary Approach to Stop Hypertension (DASH) diet which is rich in fruits, vegetables, low-fat dairy products and low sodium intake, is also reported to be helpful in preventing and treating MetS.[74,75] This diet is high in certain dietary macro/micronutrients that appear to be beneficial for reducing the risk of MetS and its components such as: fiber, potassium and magnesium.[75] Azadbakht *et al.,* (2005) conducted a randomized controlled outpatient trial on 116 patients with the MetS study to determine the effects of DASH diet on metabolic risks in patients with the MetS.[74] Three diets were planned and followed by participants for 6 months: a control diet, a weight-reducing diet concentrating on healthy food choices, and DASH diet. After adjustment for weight changes, DASH diet was the one which improved all components of the MetS, with improvements in WC and TG.[74]

"Western" dietary pattern has been shown to be associated with increased risk of components of MetS. This type of diet is characterized by high intakes of refined grains, red meat, high fat, sugar sweetened-beverages, desserts, high-fat dairy products, and eggs.[68,76] Additionally, the "Empty-Calorie" pattern which is characterized by high consumption of total and saturated fat, sugars (desserts and sugar-sweetened beverages), low intake of fruits and vegetables was associated with increased prevalence of MetS.[77]

"Vegetarian" dietary patterns are plant-based diets that are characterized by the consumption of vegetables, fruits, grains, legumes, nuts, vegetable oils, dairy products and/or eggs and reduced or eliminated consumption of animal products.[78] "Vegan" diets are more strict than the vegetarian`s since they only contain plant foods.[79] These types of dietary patterns have been suggested to be associated with a lower risk for developing DM II, HTN, specific cancers, and MetS.[78]

Nutrients and MetS

Several studies have highlighted the evidence for the beneficial effect of some macro- and micronutrients on MetS. In general, these nutrients may have a role in energy absorption/production/utilization, pancreatic functions, modulation of systemic inflammation, and oxidative stress.[80,81,82]

Macronutrients and MetS

Carbohydrates

Recent data suggest that increased consumption of refined or rapidly absorbed carbohydrates including syrups, biscuits, and cakes, are closely associated with MetS, DM II, and CVD.[83] This type of carbohydrates is characterized by a high glycemic load (GL)-a measure of both quantity and quality of dietary carbohydrate- which may increase the risk of coronary heart disease by increasing glucose intolerance and DLP.[84] These data are consistent with a cross-sectional study that detected a positive association between GL and MetS.[73] Also it is suggested that individuals who are obese and have IR are particularly prone to the adverse effects of a high dietary GL.[84,85] On the other hand, whole grains contain several nutrients such as fiber and minerals that have been shown to favorably influence components of MetS such as body weight, fasting glucose or IR,[84] blood pressure, HDL-C, and TG.[67,86,87] Limited findings have been reported on the relation between GI and MetS. GI refers to how much a carbohydrate-containing food raises plasma glucose compared with a standard food of either glucose or white bread (50g).[88] Mckeown *et al.,* (2004) demonstrated that there was no effect of total carbohydrate intake on IR or prevalence of the MetS, on the other hand, they found that high GI is positively associated with the prevalence of MetS with a multivariable odds ratio of 1.41 (1.04-1.91) comparing the highest to the lowest quintile of GI.[89]

Sugar

The primary sources of added sugar are sugary beverages that contain nutritive sweeteners such as sugar, fruit juice concentrates, or high-fructose corn syrup.[90] A longitudinal cohort study conducted by Schulze *et al.,* (2004) illustrated that higher consumption of sugar-sweetened beverages may result in unfavorable metabolic changes.[91] The results of this study also showed that the higher consumption of sugar-sweetened beverages is associated with a greater degree of weight gain and an increased risk of developing DM II in women which is attributed to excessive intake of calories and rapidly absorbable sugars.[91]

Protein

Dietary protein intake has been found to reduce cardiometabolic risk factors by lowering fasting TG and reducing fat mass while maintaining lean muscle.[82] Consumption of protein-rich meals has some beneficial effects including increased thermogenesis and metabolic rate and regulation of appetite which improves weight management and thus reduce the risk of MetS components.[82,92]

Fat

A huge body of studies reported the evidence that saturated and trans fatty acids exert deleterious effects on metabolic health.[93,94] In contrast, unsaturated fatty acids including monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA) (omega-3 and omega-6) improve metabolic parameters such as: glycemic control, insulin sensitivity, blood pressure, and lipid profile.[80,94]

Fiber

Dietary fiber intake especially from cereals and whole grains can manage and affects body weight, blood glucose and lipid profile.[95] A prospective cohort study of 9702 men and 15 365 women aged 35 to 65 years was conducted by Schulze *et al.,* (2007) to examine associations between fiber and magnesium intake and risk of DM II. The authors found that increased consumption of cereal dietary fiber significantly reduced the risk of diabetes (relative risk, 0.67).[96] Due to its viscous properties, soluble dietary fiber is found to be associated with glycemic control improvement and insulin sensitivity enhancement in both diabetic and healthy subjects. However, it didn't show any effect on reducing the risk of DM II.[97] Contrary to soluble fiber, insoluble fiber was found to be associated negatively with the risk of diabetes, even though it showed insignificant influence on postprandial glucose levels.[95,94,98]

Alcohol

A prospective cohort study of 3833 male and female Koreans aged between 40 and 69 years was conducted to examine the association between alcohol consumption and incident metabolic syndrome. It was observed that heavy liquor drinking is associated with an increased risk of the MetS components including: WC, TG, blood pressure,

and FBG.[99] Light to moderate alcohol consumption is suggested to increase HDL-C levels, thus exerting a preventive effect on cardiovascular disease.[100]

Micronutrients and MetS

Oxidative stress and systemic inflammation are the central mechanisms that relating energy overload and obesity to IR and consequently metabolic disorders.[101] Antioxidants can reduce the level of oxidative stress and potentially prevent subsequent health complications that are associated with oxidative damage.[102] Bahadoran *et al.,* (2012) reported diets rich in antioxidants (vitamin E, vitamin C, and β-carotene) exert beneficial influences on glucose metabolism and diabetes prevention. They are also associated with a reduction in the risk of developing CVD.[102] Observational evidence further suggested that higher dietary intake or supplementation of antioxidants (such as: vitamin A, C, and E, folic acid, niacin, β-carotene, selenium, and zinc) can reduce mortalities and morbidities related to CVD after a minimum of 2-year intervention. However, natural sources of these antioxidants may be more effective than synthetic forms.[101,103]

Liu *et al.,* (2005) suggested that higher intakes of total, dietary, and supplemental calcium were significantly and inversely associated with the prevalence of MetS.[104] It has been shown that high calcium intake improves BP and diabetes through weight loss and an increase in insulin release and sensitivity.[105]

Magnesium is the fourth most abundant essential mineral in the body and is involved in >300 metabolic reactions, including protein/DNA/RNA synthesis, cellular energy production, and cell growth and reproduction.[106] Observational data indicated an inverse association between magnesium status and the risk of MetS among adults, but this potential benefit needs further investigation.[106,107] Bian *et al.,* (2013) concluded that "vitamin B group" pattern including thiamin, riboflavin and niacin was negatively associated with the risk of MetS.[108] High sodium diet is positively associated with HTN, IR, and DLP as demonstrated by Baudrand *et al.,* (2014).[109]

Management of MetS

A healthy lifestyle including weight loss through calorie restriction, healthy food choices, increased physical activity and smoking cessation has a remarkable role in preventing or delaying the onset of MetS or treating the condition when present.[110] Grundy *et al.,* (2005) suggested that besides lifestyle modification for the management of MetS, clinical therapy is also necessary in many patients for the treatment of this syndrome.[111] For treating IR and delaying DM II, metformin and thiazolidinediones are effective in reducing IR and keeping serum blood glucose in normal ranges, but their effect haven`t been shown to reduce the risk of CVD in those with the MetS, prediabetes, or DM II.[111]

In the case of DLP, Statins are well-known to reduce all apolipoprotein B–containing lipoproteins. Fibrates improve all components of atherogenic DLP and may directly reduce the risk for CVD.[18] Anti-hypertensive agents are recommended for people whose blood pressure exceeds normal ranges.[111]

In addition to the previous strategies; bariatric surgery is now favored to be as an effective approach for the treatment of severe and morbid obesity.[21] It results in a weight loss of 25–30% and improvement of multiple comorbidities associated with obesity, including HTN, DM II, CVD and DLP.[112]

Conclusions

The present review provides evidence supporting the presence of an association between dietary patterns, micro- and macronutrients intake, lifestyle factors and the risk of MetS. Higher WC and positive family history of MetS are associated with developing MetS. Also the finding of this review documented an association of the adherence to some dietary patterns; especially "fast food pattern" with MetS risk.

Compliance with Ethical Standards

Competing Interests

The authors declare that they have no competing interest.

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