

ORIGINAL ARTICLE



The Impact of Lifestyle on Metabolic Syndrome: A Comprehensive Review

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Abstract:

Metabolic syndrome (MetS) is a pathological condition characterized by cardiovascular risk factors, including hypertension, obesity, dyslipidemia, and abnormal glucose metabolism, increasing the risk of developing diabetes and cardiovascular disease (CVD). Despite extensive research and the association of lifestyle variables with the development and progression of MetS, its etiology remains unclear, and research into preventive interventions remains critical. Individuals with MetS can significantly benefit from lifestyle interventions such as regular exercise, healthy eating habits, and other behavioral changes, which can help improve the associated pathophysiological changes. In addition, promoting a healthy lifestyle in the general population can significantly reduce the incidence and prevalence of MetS and associated diseases. Therefore, the main objective of this paper is to provide a comprehensive review of MetS, including its diagnostic criteria, lifestyle, and lifestyle score correlates. This study highlights the importance of lifestyle interventions in preventing and treating MetS as an effective and accessible way to reduce complications and improve the quality of life of patients with MetS.

Keywords: Smoking; sleep duration; alcohol; coffee; tea; physical activity; diet

Introduction

The "metabolic syndrome" is a cluster of metabolic disorders whose diagnosis is based on the presence of several components. These include elevated triglycerides (TG), low high-density lipoprotein cholesterol (HDL-C), high blood pressure (BP), high blood glucose, and central obesity (increased waist circumference). It is a precursor to many diseases, including type 2 diabetes mellitus (T2DM), CVD, and nonalcoholic fatty liver disease (NAFLD) [1,2]. When many risk factors are present, their combined effects are synergistic rather than additive, dramatically increasing the patient's chance of contracting additional diseases compared to someone with only one risk factor

[3].

MetS has become a severe public health problem with wide-ranging effects on people's quality of life and financial stability [4]. The global prevalence of MetS remains unknown, but estimates indicate a wide variation in its occurrence among individuals and across different regions, ranging from 10% to 84% [5,6]. This is because various organizations employ various diagnostic standards. Despite these drawbacks, research indicates that about 25% of the world's population may be affected by MetS [7].

This review aims to provide a comprehensive understanding of the impact of lifestyle on MetS

and effective prevention and management strategies by synthesizing the evidence from epidemiologic studies (including cross-sectional studies, cohort studies, clinical studies, and case-control studies, among others). The relationship between lifestyle factors and the MetS will be examined by addressing pivotal queries: What is the relationship between physical activity (PA) and the MetS? What is the relationship between dietary patterns and MetS in contemporary society due to changing lifestyles? Furthermore, we will investigate how other lifestyle factors such as smoking, drinking, and sleeping affect MetS [8-10]. By amalgamating these factors' impacts on MetS, a deeper grasp of the ailment is attainable. This scholarly assessment elucidates the intricate interplay between lifestyle elements and MetS, aiming to inspire both individuals and communities to adopt beneficial lifestyle adjustments. Through health education and heightened public awareness, our goal is to foster enduring health enhancements and mitigate the risk of MetS.

The following keywords were used throughout the literature search: "metabolic syndrome", "lifestyle-related to metabolic syndrome", "smoking", "coffee", "physical activity", "tea consumption" and "alcohol consumption". There are no time or language restrictions when using Medline as a search source (via PubMed). After screening the titles and abstracts, relevant full-text content was searched, examined, and downloaded into endnotes (clearly numbered). The total number of articles identified was 487 and the total number of articles selected for inclusion in the review was 120. All authors were consulted for clarification of any issues.

2. Metabolic Syndrome

There are no generally accepted diagnostic criteria for MetS. The lack of uniformity hogs the implementation of tailored therapies and the early clinical identification of individuals at risk for MetS. International organizations such as the World Health Organization (WHO), the International Diabetes Federation (IDF), the National Cholesterol Education Program, Adult Treatment Panel III (NCEP-ATPIII), American Heart Association/National Heart, Lung, and

Blood Institute (AHA/NHLBI), and Harmonized 2009 Criteria propose diagnostic criteria for MetS identification [2,11-16]. Most diagnostic criteria for MetS generally focus on four key areas: obesity, hyperlipidemia, hyperglycemia, and hypertension. Despite a consensus on these components, variations exist in entry points and mandatory criteria for diagnosis. Additionally, cut-off values for FPG, TG, HDL-C, and BP differ among different diagnostic guidelines.

Different organizations have varying criteria for diagnosis, with WHO, the European group for the study of insulin resistance (EGIR), IDF, NCEP-ATPIII, AHA/NHLBI, and Harmonized 2009 Criteria highlighting different risk factors and testing procedures. WHO and EGIR [17] require measuring insulin levels, while WHO also includes urinary microalbumin and IDF focuses on waist circumference (WC). Regional or ethnic-specific waist circumference cutoffs are suggested by NCEP-ATPIII, IDF, AHA/NHLBI, and Harmonized 2009 Criteria to enhance accuracy. Despite variations in risk factors and diagnostic thresholds across measures, there is a shared understanding that multiple risk factors are necessary to diagnose MetS.

However, the direct application of these criteria to children and adolescents may be impossible due to the complicated link between MetS, obesity in children and adolescents, and metabolic diseases in adolescents [18,19]. As a result, NCEP-ATP III and IDF provide more suitable criteria than the universally applicable ones. Measure specific to children and adolescents use age- and sex-specific percentile values for WC and BP, considering individual growth and development differences for accurate assessments in these groups [15]. Some research indicates that a thorough clinical assessment, including height, weight, WC, and hip circumference, is crucial for diagnosing MetS in children and adolescents accurately. Additionally, body mass index (BMI) z-scores, abdominal circumference z-scores, A-BMI, and body circumference index are potential diagnostic tools [20]. In summary, when assessing MetS in children and adolescents, it is recommended that multiple measures be used in conjunction with age and sex-specific percentile values.

Table 1 Diagnostic criteria for metabolic syndrome

| Components | Criteria | WHO (1999) | EGIR (1999) | AHA/NHLBI (2005) | Harmonized (2009) | NCEP -ATPIII(2005) | | IDF(2005) | |
|------------------|---------------------------------------|------------------------|--------------------|------------------------------|---|-----------------------|--|-------------------------------|---|
| | | | | | | universal application | Adjusted for children and adolescents | universal application | Adjusted for ages 10-16** |
| Obesity | BMI (kg/m ²) | > 30 | - | - | - | - | - | - | - |
| | WC (cm) | - | ≥ 94(M) ≥ 80(W) | ethnicity-specific | Population and country-specific definitions | ethnicity-specific | ≥ Age and sex-specific 90th percentile | ethnicity-specific | ≥ 90th percentile or adult cut-off if lower |
| | WHR | > 0.90(M) > 0.85(W) | - | - | - | - | - | - | - |
| Dyslipidemia | TG (mg/dL) | > 150 | ≥ 190 | ≥ 150* | ≥ 150* | ≥ 150* | ≥ 110 | > 150* | > 150 |
| | HDL-C (mg/dL) | < 35(M) < 39(W) | < 40 | < 40(M) * < 50(W) * | < 40(M)* < 50(W)* | < 40(M)* < 50(W)* | ≤ 40 | < 40(M)* < 50(W)* | < 40 |
| Hyperglycemia | FPG (mg/dL) | ≥ 100 [#] | ≥ 110 | ≥ 100 /T2DM | ≥ 100 [#] | ≥ 100 [#] | ≥ 110 | ≥ 100 /T2DM ^{#&} | ≥ 100 /T2DM ^{#&} |
| | 2-h PG (mmol/L) | 7.8-11.0 | - | - | - | - | - | - | - |
| Hypertension | SBP (mm Hg) | > 140 | ≥ 140 ⁺ | ≥ 130 ⁺ | ≥ 130 ⁺ | ≥ 130 ⁺ | ≥ 90th percentile for age, sex, and height | > 130 ⁺ | > 130 |
| | DBP (mm Hg) | > 90 | ≥ 90 ⁺ | ≥ 85 ⁺ | ≥ 85 ⁺ | ≥ 85 ⁺ | ≥ 90th percentile for age, sex, and height | > 85 ⁺ | > 85 |
| Microalbuminuria | Urine albumin excretion rate (µg/min) | ≥ 20 | - | - | - | - | - | - | - |
| | Albumin creatinine ratio (mg/g) | ≥ 30 | - | - | - | - | - | - | - |

| | | | | | | | | |
|--------------------|---|--|---------------------------|---------------------------|---------------------------|---------------------------|--------------------------------|--|
| Selection Criteria | IGT/Diabetes/IR plus two or more components | IR/hyperinsulinemia (only non-diabetic subjects) plus two or more components | At least three components | At least three components | At least three components | At least three components | WC and at least two components | Central obesity (WC) plus any two components |
|--------------------|---|--|---------------------------|---------------------------|---------------------------|---------------------------|--------------------------------|--|

* or treatment of such lipid abnormalities; # or receiving conditioning therapy; +or treatment of previously diagnosed hypertension; & Oral glucose tolerance test is highly recommended; ** Under the age of 10, MetS as an entity will not be diagnosed, although a strong message to lose weight will be sent to these children. At age 10 and older, MetS can be diagnosed. It requires the presence of abdominal obesity as well as the presence of two or more other components. The IDF criteria for adults can be used for adolescents ≥16 years of age. DBP: Diastolic Blood Pressure; IGT: Impaired Glucose Tolerance; SBP: Systolic Blood Pressure; 2-h PG: Two-hour postload plasma glucose.

No universally accepted "best" diagnostic criteria exist for MetS, although the Harmonized 2009 Criteria offered one option for adults. These criteria emphasize key components like obesity, hyperlipidemia, hyperglycemia, and hypertension. Adjusting waist circumference limits based on regional or ethnic variations is suggested. The Harmonized 2009 Criteria provide a comprehensive assessment of MetS elements with some flexibility for regional or ethnic considerations. Criteria for children and adolescents are age and gender-specific. It is crucial to remember that more investigation and agreement among international organizations are needed to create a widely used diagnostic criterion for MetS.

3. Health-Related Lifestyle and MetS

MetS is commonly associated with a sedentary lifestyle, poor diet, and other unhealthy lifestyle factors. Several studies have shown a direct relationship between MetS and lifestyle, especially regarding PA and diet. People with a

sedentary lifestyle and an unhealthy diet are at a higher risk of developing MetS [21-23]. In contrast, people who maintain an active lifestyle and a balanced diet are less likely to develop the disorder. In addition, other lifestyle factors, such as smoking, alcohol consumption, and poor sleep, are also associated with an increased risk of MetS. These lifestyle factors can lead to inflammation, IR, and other metabolic imbalances that contribute to the development of the disorder [24]. The relationship between MetS and lifestyle is complex. Still, strong evidence suggests that positive lifestyle adjustments like increasing physical activity, enhancing diet, and cutting harmful habits can lower the risk of developing MetS and its health issues.

3.1 Sleep Duration

Maintaining metabolic stability is vital for well-being, and adequate sleep is key to attaining it. Modern lifestyles and swift economic growth have led to widespread sleep deprivation [25]. The National Sleep Foundation suggests adults should aim for 7 to 9 hours of sleep nightly. Recent research shows that insufficient or excessive sleep is linked to adverse health outcomes such as obesity, MetS and its components, as well as cardiovascular disease [26].

Most studies suggest that individuals who get 7 to 8.5 hours of sleep per night have the lowest risk of developing MetS [27]. A "U-shaped" association between sleep duration and MetS was reported in cross-sectional and cohort studies, where insufficient and excessive sleep duration significantly increases the risk of developing MetS.[26] Moreover, surveys examining individual components of MetS also noted a "U-shaped" connection between sleep duration and TG levels [28].

The "U-shaped" association outlined above by two meta-analyses published in 2014 and 2015, respectively, which conclude that only a short sleep duration (5 h/d or 6 h/d) is substantially associated with an increased risk of MetS [29,30]. Short sleep duration is independent of other lifestyle factors and positively correlates with the number of MetS components. Conversely, research has also established a "J-shaped" relationship between sleep duration and MetS [31,32]. Long sleep duration, specifically over 8 or 9 hours per day, is significantly linked to a higher risk of Metabolic Syndrome (MetS), which is associated with increased chances of hyperglycemia, hyperlipidemia, and low HDL-C levels.

Gender-specific differences in the link between MetS and its components and the impact of short and long sleep were also observed. Specifically, female individuals had a higher likelihood of developing MetS when experiencing short sleep duration (≤ 5 hours/day) or long sleep duration (≥ 9 hours/day). For males, short sleep duration (≤ 5 hours/day) had a higher association with MetS incidence and a higher severity score [33]. On the other hand, prolonged sleep (≥ 7.5 hours/day) affects both genders similarly, with females at higher MetS risk due to extended sleep, and males at risk due to inadequate sleep [27,31]. Weight regulation is adversely affected by inadequate sleep for all. Males experience elevated blood glucose with excessive sleep, while excessive sleep in females is associated with most MetS components except hypertension [32,34].

There are also differences in the association between sleep duration and MetS in different age

groups. For instance, in children (≤ 9 h/d) and adolescents (≤ 8 h/d), short sleep duration has been significantly linked with higher BMI, WC, and TG levels [35,36]. In emerging adults (18-24 years), both long sleep (> 9 h/d) and short sleep (≤ 7 h/d) have been associated with an increased risk of MetS [37]. Middle-aged adults (45-64 years) with either short sleep (< 5 h/d) or long sleep (≥ 9 h/d) have also been observed to have a higher prevalence of MetS, as indicated by data from the EpiHealth cohort study [38]. Similarly, a cohort study revealed that both short sleep (< 7 h/d) and long sleep (≥ 8 h/d) are associated with an increased risk of MetS among older adults (≥ 60 years) [39]. However, it is essential to note that, among older adults (≥ 65 years), only long sleep (≥ 9 h/d) has been associated with a higher prevalence of MetS [38]. By contrast, other research has shown that long sleep (> 8 h/d) is associated with lower odds of MetS when compared to standard sleep duration (7-8 h/d) [40]. [Table 2 near here] In conclusion, there is some correlation between MetS and sleep duration. However, the relationship varies according to the age and gender of the group. This mismatch highlights the need for more research and sophisticated analytical approaches to address current challenges. There is disagreement over the significance of the link between short and long sleep and MetS. The recent study suggests that demographic confounding and, to a lesser extent, differences in study design and analysis may be possible explanations. In addition, the effect of long sleep duration on MetS may be related to confounding factors and comorbidities that need to be considered along with occupation, diet, and PA.

Table 2. Relationship between sleep duration and metabolic syndrome across gender and age groups

| Group | | Sleep duration | Risk of MetS | Risk of MetS components |
|--------|---------------------------------|--|--------------|--------------------------------------|
| Gender | Male | Short sleep duration (≤ 5 h/d) | Rising risks | Increased waist size and weight gain |
| | | Long sleep duration (≥ 9 h/d) | --* | Increased blood sugar levels |
| | Female | Short sleep duration (≤ 5 h/d) | Rising risks | Increased waist size |
| | | Long sleep duration (≥ 9 h/d) | Rising risks | All components (except increased BP) |
| Age | Children and adolescents (6-18) | Short sleep duration (Children ≤ 8 h/d, | --* | Increased BMI, WC, and TG |

| | | adolescents ≤ 9 h/d) | | |
|---|--------------------------------------|---------------------------|-----|--|
| Emerging Adults (18-24) | Short sleep duration (≤ 7 h/d) | Rising risks | --* | |
| | Long sleep duration (> 9 h/d) | Rising risks | --* | |
| Middle-aged (45-64) and elderly (≥ 60) | Short sleep duration (≤ 5 h/d) | Rising risks | --* | |
| | Long sleep duration (≥ 9 h/d) | Rising risks | --* | |
| Elderly (≥ 65) | Short sleep duration (≤ 5 h/d) | --* | --* | |
| | Long sleep duration (≥ 9 h/d) | Rising risks | --* | |

*Not mentioned in the literature.

3.2 Smoking

The prevalence of diseases linked to tobacco use is increasing on a global scale. Research has shown that smoking reduces insulin sensitivity and alters lipid metabolism, leading to increased triglycerides and TC while lowering HDL-C levels. These changes increase the likelihood of MetS development through several pathways [41].

In adults, the prevalence of MetS among current smokers was 42.1%, indicating a higher risk for MetS than nonsmokers [42]. Another study in Korea found a significant association between smoking and MetS in young people (< 40 years) living in urban areas and good health [41]. Smokers were at a 2.38-fold higher risk for MetS than nonsmokers, and an increased risk of both hypertriglyceridemia and low HDL-C was observed. It suggests that smoking-induced changes in blood lipid levels are pivotal in the relationship between smoking and MetS. A Dutch study found a higher prevalence of MetS among smokers, indicating a dose-dependent relationship [43]. The increased risk of MetS was not related to BMI or gender. However, an Iranian study reported no significant association between smoking and MetS in male smokers and a lower prevalence of hypertension, hyperglycemia, and central obesity in female smokers than in nonsmokers [44]. The different results among the studies above may result from regional and ethnic variances that also lead to dissimilar lifestyles. Additionally, the various study designs, widely varying sample sizes, and different ages could have contributed.

Individuals who quit smoking had significantly higher rates of overweight, abnormal BP, impaired FPG, and abdominal fat than nonsmokers and current smokers [45]. The higher risk of MetS, elevated TG levels, and low HDL-C levels disappeared in ex-smokers [45,46]. Furthermore, the prevalence of MetS varied significantly among smoking status groups, with 22.0% among nonsmokers, 34.4% among ex-smokers, and 29.4% among current smokers [45]. In contrast, the OPERA study demonstrated a significant increase in BMI and WC among individuals who quit smoking but no significant increase in the prevalence of obesity-related diseases such as MetS, hypertension, and diabetes [47]. Similarly, other studies have indicated no statistical increase in MetS, high TG, and low HDL-C levels after smoking cessation 1-4 years ago [46]. However, there has been a tendency to reduce the risk of MetS, which is time-dependent. Both active and secondhand smoking carry similar risk factors. Nevertheless, the early process of smoking cessation may increase the rate of MetS by promoting weight gain and abdominal obesity from overeating [48]. Nonetheless, after quitting smoking for 20 years, the risk of MetS was no longer significant in ex-smokers, underscoring the importance of stopping at a younger age to reduce the risk of MetS [49].

Although there is evidence that smoking has a synergistic effect on the development of MetS, it is critical to consider other factors such as gender, age, time to quit smoking, duration of smoking, number of cigarettes, and lifestyle confounders to gain a more detailed and accurate understanding of the relationship between tobacco and MetS.

3.3 Alcohol

Alcohol is commonly consumed, but its relationship with MetS is complex. Moderate intake may lower cardiovascular disease risk and aid glycemic control, potentially reducing MetS risk. However, excessive drinking can cause metabolic abnormalities like obesity, and hypertension, increasing MetS risk despite its potential benefits [50].

Population-based research findings indicate that the prevalence of MetS is significantly higher among frequent drinkers (more than 12 drinks/month) than nondrinkers. Regular drinking was positively associated with some components of MetS, except WC [51]. Another study noted that some MetS components, such as abdominal obesity, hypertension, and FPG, worsened with increasing frequency of alcohol consumption, while the risk of low HDL-C decreased [52]. Nevertheless, evidence that moderate alcohol consumption can reduce MetS risk by increasing HDL-C is also available (< 25 g/d for men, < 15 g/d for women) [42].

It has also been noted that there are different correlations between alcohol consumption and MetS by gender, mainly in women who drink alcohol to reduce their risk of developing MetS. At the same time, men do not show a similar relationship [53]. Paradoxically, current drinkers had higher SBP and, DBP, HDL-C levels in men than nondrinkers. In women, these differences were small or insignificant [54]. There was gender variability in the association between alcohol intake and frequency and MetS, with a cohort study showing that moderate alcohol consumption (15-40 g/day in men and 10-20 g/day in women) was associated with a lower risk of MetS, abdominal obesity, and low HDL-C in men and low HDL-C in women [55]. Heavy drinking (> 40 g/day) in men reduces the risk of hyperglycemia high BP, and low HDL-C. while in women, heavy drinking (> 20 g/day) was associated with a higher risk of abdominal obesity. A Korean study revealed heavy drinkers (> 30 g alcohol/day) faced higher MetS risk than non-binge drinkers, with no gender differences, and all components of MetS were significantly associated with heavy drinking. Some research suggests a linear correlation between alcohol intake and reduced hypertension and dyslipidemia in men, but in

women, alcohol alone may lead to hypertension [54].

While most research indicates that alcohol intake is linked to a higher likelihood of MetS, some studies suggest that moderate alcohol consumption could lower the risk. Red wine, in particular, has been identified as a potential therapeutic option for MetS. Therefore, when examining the link between alcohol and MetS, factors like dietary choices must be taken into account. Further research should explore various aspects of alcohol consumption, including type, amount, and duration, to better understand this relationship. Currently, the prevention of MetS by low alcohol consumption is not recommended, and each component of MetS should be managed individually to ensure cardiorespiratory and metabolic health. Establishing a healthy drinking culture that avoids binge drinking should be prioritized in a public health context.

3.4 Tea

Tea is a popular beverage that is healthy and has positive effects on metabolic syndrome. Tea constituents, which include antioxidants, may improve insulin sensitivity, control blood sugar, and help with weight management, ultimately reducing the risk of metabolic syndrome. Tea has the power to successfully boost overall health when included in a healthy lifestyle [56].

Studies have consistently shown that consuming 3-4 cups or more of tea (Green tea, black tea, pu-erh tea, Tsampa, butter tea, and Qing cha) per day can alleviate MetS [57,58]. Various short-term randomized controlled trials have shown that tea consumption positively affects different components of MetS. Furthermore, meta-analyses have shown that tea consumption (Green tea) is a highly effective approach to weight loss and MetS relief [59]. In obese patients with MetS, supplementation with tea extract (green tea extract, black tea extract, or oolong tea extract) has been found to improve lipid and glucose metabolism, promoting weight loss [60]. Green tea consumption has been shown to significantly reduce body weight, BMI, WC, Total cholesterol (TC), and low-density lipoprotein cholesterol (LDL-C) [61]. The CNHS showed that habitual tea (regardless of the type of tea they consumed) drinkers (those who consumed one or more cups of tea per day at 200 ml per cup) who drank more

than 5 cups per day had a lower risk of MetS, elevated BP, elevated TG, and increased FPG, but a higher risk of central obesity compared to non-habitual tea drinkers [62]. Higher tea consumption among women was also associated with a lower risk of low HDL-C. In conclusion, the studies indicate that tea/tea extract may reduce weight, BMI, WC, blood glucose, TC, TG, and LDL-C levels in MetS patients, ameliorating MetS. However, whether tea consumption affects other MetS components, such as HDL-C and BP, remains unclear. It is worth noting that, in contrast to the above studies, a Japanese cohort study found no association between green tea consumption and the prevalence of MetS. Additionally, one study showed that all MetS components were not negatively associated with green tea consumption [63]. Furthermore, tea consumption was not associated with MetS, high FPG, high TG, central obesity, or hypertension in older Australian adults [64].

In addition to the traditional ways of drinking tea discussed above, some studies suggest that excessive consumption of sugary beverages such as sweet milk tea and fruit tea is associated with MetS and T2DM [65]. Consuming too much sweet tea can be a source of high sugar intake and elevated blood sugar levels. It can cause weight gain and worsen MetS problems if too much sweet tea is consumed over a long period. Furthermore, sugary drinks will be converted into TG, leading to increased blood triglyceride levels, further exacerbating the problem of high triglyceride levels associated with MetS [66]. For this reason, people with MetS and T2DM should limit or avoid excessive consumption of sugary drinks intake to manage blood sugar and weight. Opting for low or no-sugar tea beverages can be a beneficial substitute.

Research on tea consumption and MetS remains inconclusive despite years of study. The ideal amount of tea intake for combating MetS is still uncertain. Drinking traditional tea is thought to offer greater advantages compared to sweetened tea. It's also unclear if age and gender influence MetS risk in habitual tea drinkers. Future cohort studies should focus on accurately measuring tea consumption, types, and potential food interactions. Only then can we definitively determine the role of tea in preventing or alleviating MetS.

3.5 Coffee

Coffee is a trendy beverage in modern society, consisting of active ingredients such as caffeine, chlorogenic acid, fenugreek alkaloids, diterpenoids, and coffee roasting products. MetS, including improved insulin sensitivity, glycemic control, and promotion of weight management [67].

The evidence suggests that adults who consume more than one standard cup (236.59 mL/cup) of coffee daily have a lower incidence of MetS [68]. In Korea, multiple studies have found an inverse relationship between moderate coffee consumption (3-4 times/d) and the prevalence of MetS in Korean adults [69]. In non-obese individuals, there was a marked linear trend towards lower odds of MetS with increased coffee consumption (≥ 5 times/d). In contrast, coffee consumption at this level demonstrated a non-significant positive association with MetS risk in obese individuals [70]. In a similar vein, the Japanese Multi-Institutional Collaborative Cohort study found a negative association between coffee consumption (1.5 to < 3 cups/day or ≥ 3 cups/day) and MetS [71]. Despite these encouraging findings, few studies have reported inconsistencies in the relationship between coffee consumption and MetS prevalence. Expressly, some studies have noted a weak positive association between moderate (3 to < 5 cups/day) and high (≥ 5 cups/day) coffee consumption and increased odds of developing MetS [72]. Some differences were also observed between coffee consumption and MetS components. High coffee consumption was associated with increased BMI, WC, SBP, DBP, TG, and TC, but there are still conflicting relationships between HDL-C and FPG levels [73,74].

In addition, the effect of coffee on MetS is a function of the type of coffee consumption. Instant coffee consumption dramatically increases plasma homocysteine and negatively affects abdominal obesity and low HDL-C, resulting in an increased risk of MetS [75]. However, some studies have negatively associated instant coffee with MetS and its components [76]. The intake of sugar and creamer powder may be the reason for the conflicting effects. Additionally, regular green or roasted coffee blends were associated with positive effects on BP, blood glucose, and TG, negatively associated with MetS [77]. Moreover,

it has been suggested that women may benefit from consuming all three types of coffee (black coffee, coffee with cream, and coffee with milk), while men may only benefit from sipping black coffee [68,78].

Contrary to the above, MetS may not be associated with coffee consumption. For example, the Amsterdam Longitudinal Study of Growth and Health found no significant difference in coffee consumption between subjects with or without MetS or its components over 9 years [79]. A recent meta-analysis further supported this finding. A separate longitudinal study also failed to identify any association between coffee consumption and MetS components in men [80]. Similarly, a Korean study found no significant association between coffee consumption and MetS in men [78].

The link between coffee type and MetS, as well as the dose-response relationship between coffee intake and MetS risk, remains uncertain. The lack of significant association and the varied findings may stem from unaddressed factors like coffee dosage adjusted for energy and polyphenol intake, type, preparation method, density, additives, and composition. Consequently, it's difficult to ascertain coffee's potential effects on MetS. More comprehensive population-based cohort studies are necessary to validate these findings and explore the coffee-MetS relationship further.

3.6 Diet

The global prevalence of obesity has increased in recent years, primarily due to changes in dietary habits. These changes resulted in a concomitant expansion of diet-associated chronic inflammation, ultimately leading to an increased risk of MetS [81].

Diets high in sugar, fat, and calories, ultra-processed foods (UPF), and low in dietary fiber are associated with an increased prevalence of MetS [82,83]. Specific dietary interventions are effective in managing MetS, including low-fat (LF) and low-carbohydrate (LC) diets [84]. The LF diet has also been found to be effective in weight management for patients with MetS, while the LC diet has beneficial effects on metabolic components [85]. A greater risk of MetS is linked to a higher long-term intake of UPF, which is often high in caloric density, salt, added sugars, and trans fats [83]. On the other hand, high

carbohydrate intake has been linked to an increased risk of MetS [86]. According to CHNS, a high intake of red meat (which is high in saturated fatty acids) is associated with an increase in BMI, weight, WC (in men only), and abdominal obesity in Chinese populations [87]. However, the highest intake of red meat among women is associated with a lower risk of BP [88]. Meta-analyses have shown that increased red and processed meat consumption increases the risk of developing MetS [89]. Conversely, white meat intake has been negatively associated with the risk of developing MetS, and recent studies have confirmed that higher information on white meat among participants with MetSSS(T1) is beneficial [90]. Furthermore, a high intake of high-fiber fruits and vegetables was found to be negatively associated with MetS [91]. In Chinese men, an increased intake of fruits and vegetables (100 g/d) was associated with weight loss and decreased BMI [92]. Additionally, increased fruit intake (100 g/d) was associated with a reduced risk of MetS, while increased vegetable intake (100 g/d) was not associated with a reduced risk of MetS [93]. Egg consumption, characterized by ovalbumin, cholesterol, and methionine, has been found to reduce weight and increase HDL-C levels [94,95]. The risk of MetS, central obesity, and BP were negatively associated with egg consumption when the daily intake of eggs was less than 20 g. Elevated TG was negatively associated with egg consumption when the daily intake of eggs was less than 60 g [94,95]. Dairy products (milk and yogurt) containing lactoferrin, lactose, and various minerals are inversely associated with the risk of MetS [96]. In addition, legumes are associated with a lower prevalence of MetS due to their high content of carbohydrates, proteins, and fats, with a better amino acid composition [97]. Finally, an increase in total nut consumption (especially walnuts) characterized by unsaturated fatty acids, protein, and fiber has been associated with significant improvements in metabolic components [91].

In the real world, the risk of developing a disease can be better predicted by examining the total dietary pattern for its role in disease, as individuals tend to get most of their nutrients from mixed foods [98]. The Mediterranean Diet (MD) is a healthy eating pattern rich in monounsaturated fatty acids, dietary fiber, and antioxidants while having a low total and saturated fat [97]. In

children and adolescents, MD improved BMI and lipid profiles [99]. In adults, those with low MetSSS tend to have higher adherence to MD [90,100]. However, studies of older populations have produced mixed results, with some showing no correlation between adherence to MD and a lower incidence of MetS compared to the LF diet [97]. Nevertheless, MD has been associated with a significant rate of MetS reversal, suggesting that it may effectively reduce central obesity and hyperglycemia in patients with this condition [100]. However, compared to Western/unhealthy diets (meat, refined grains, high-fat foods, processed and fried foods) [101], healthy diets did not improve metabolic flexibility and systemic insulin sensitivity. They were not associated with the risk of developing MetS, whereas adherence to unhealthy diets was positively associated with risk [86]. Population studies have shown that greater adherence to Western dietary patterns is associated with a higher risk of MetS and a lower likelihood of increased IR and sensitivity [86,102]. Higher scores on Western dietary patterns were associated with higher odds of developing MetS. Significant associations were found for FPG, HDL-C, and LDL-C [86,102].

Different countries have different dietary characteristics, and their association with MetS varies, in addition to the two dominant dietary patterns mentioned above. Iran's traditional diet (dairy products, animal fat, confectionery, and organ meat) was positively associated with MetS risk and the number of MetS components. However, it had no significant effect on metabolic components (SBP, DBP, HDL-C, and WC), which may be attributed to higher levels of red meat, hydrogenated fat, and fatty dairy products traditionally consumed in Iran [86,103]. Similarly, the traditional Polish diet (potatoes, meat, vegetables, cheese, animal fat, and sugar) is associated with a high risk of abdominal obesity and triglyceridemia [102]. In contrast, the Nordic diet (whole grains, vegetables, berries, rapeseed oil, fish, and low-fat dairy) improves MetS and its components, including DBP, LDL-C, and HDL-C [104]. Moreover, the traditional Korean diet (rice, fermented vegetables, spices, mushrooms, shellfish, seaweed, and fish) benefits glycemic control and body composition. It may improve the risk of MetS, although the main beneficial effects on metabolic components depend on gender [105]. The traditional Chinese diet including rice,

red meat, fish, poultry, offal, and leafy greens, is linked to lower MetS risk. It has a negative relationship with obesity and overweight in males [82].

A thorough investigation reveals that the MD is the most well-known and practical choice for reducing the risk of MetS. Due to several factors, such as environment, ethnicity, and gender, that affect dietary habits and lifestyles across nations, it is difficult to identify an effective and simple-to-follow dietary pattern. The risk of MetS is also substantially influenced by several other variables. Further research is needed, and dietary treatments that are effective, simple to implement, and affordable must be promoted to reduce the risk of MetS and maximize population benefits. To provide more detailed suggestions and recommendations, this calls for cross-cultural and cross-regional research as well as prospective cohort studies and field trials of therapies.

3.7 Physical Activity

PA has become an integral part of clinical treatment protocols for patients with MetS in recent years, as more attention has been paid to understanding the condition. Among physiological systems that respond positively to PA, improved IR is one of the most notable effects of regular exercise [21]. PA has been shown to help reduce body weight, lower BP, and improve dyslipidemia to the extent that MetS criteria are no longer met [106].

SB has been identified as a risk factor for MetS development, independent of PA levels. SB has been found to exhibit a negative association with FPG, TG, and HDL-C [107]. Furthermore, the highest MetSSS(T3) in patients with MetS had prolonged SB [90]. A recent dose-response meta-analysis revealed a significant association between prolonged SB (≥ 4.11 h/d) and a higher risk of MetS [108]. Specifically, a 1-hour increase in daily SB was found to increase the risk of MetS by 16%. Gender differences have also been observed in the association between SB and MetS prevalence. Notably, there is an association between SB and MetS prevalence in men but not women [109]. However, other studies have reported that SB is significantly more strongly associated with MetS prevalence in women than men, especially in women who are sedentary for more than 42 hours per week [34]. Reducing daily

sedentary time slightly reduced fasting insulin but did not affect insulin sensitivity or adiposity [21]. In conclusion, avoiding prolonged SB appears to play a role in reducing the risk of MetS, and the only effective scientific treatment to fully combat sedentary dysfunction is primary prevention through exercise itself.

PA can reduce weight and BP and increase metabolic health in metabolic syndromic patients [110]. Replacing certain daily SB with light and moderate-to-vigorous physical activity (MVPA) may prevent CVD in high-risk populations [111]. 300-420 minutes per week of light physical activity (LPA), 90-300 minutes per week of moderate physical activity (MPA), and more than 150 minutes per week of vigorous physical activity (VPA) were shown to reduce the risk of MetS [112]. MVPA and LPA were associated with high HDL-C, and LPA was associated with low TG [109]. However, there were also gender differences in PA intensity and MetS prevalence, with an association between MPA. MetS observed only in women, and an interaction of gender with MPA, SB, and sleep duration, except for low HDL-C in men, for whom high-intensity PA was significantly associated with a reduced risk of MetS and its components [34]. Recent studies have also shown that more time spent in MVPA (≥ 150 min/w) is associated with improved adiposity and MetS components [113]; MVPA reduces the risk of MetS in Asian populations, with similar results in other folks [114]. Moreover, it is well established that various forms of exercise can elicit distinct effects on the metabolic complex and its components. High-

intensity interval training (HIIT) and moderate-intensity continuous training (MICT) have been reported to induce reductions in insulin sensitivity and lipid levels. In addition, combination exercise regimens have shown superior effectiveness in improving glycemia, lipemia, and sleep quality in MetS patients [115].

In summary, appropriate engagement in PA plays a vital role in preventing and addressing MetS. Reducing sedentary time, diversifying PA forms, and combining interventions have effectively reduced MetS prevalence and its component diseases. PA interventions have positively impacted metabolic disorders and their broader societal burdens. Given the far-reaching benefits of PA, it is imperative to incorporate such interventions into MetS treatment strategies to curb its harmful health effects.

3.8 Lifestyle score

In epidemiological research, it is common to include lifestyle factors as covariates in statistical models when examining individual health behaviors or exposures. However, recognizing the synergistic effects of healthy lifestyle factors and the cumulative impact of these factors, some studies advocate aggregating lifestyle profiles into a score indicator. This results in various healthy lifestyle scores, such as the Healthy Lifestyle Score (HLS) and Mediterranean Lifestyle (MEDLIFE), which differ in factor-score scoring methods and factor weighting [116-120]. Nonetheless, there is a consensus that higher lifestyle scores are associated with a lower risk of MetS.

Table 3. Healthy Lifestyle Score

| Health Lifestyle | Score | Points | Outcome | Reference |
|---|------------------------|--------|---|-----------|
| HLS: ① Never smoking, ② Moderate to high PA (> 20 MET-h/week), ③ MD (≥ 4 adherence points) ④ Moderate alcohol consumption (women, 0.1–5.0 g/day; men, 0.1–10.0 g/day; abstainers excluded), ⑤ Low television exposure (< 2 h/day), ⑥ No binge drinking (≤ 5 alcoholic drinks at any time), ⑦ Taking a short afternoon nap (< 30 min/day), ⑧ Meeting up with friends > 1 h/day, | 0-9 (0-3 4 5 6 7-9) | ⑤/⑦ | HLSes (7 to 9 points) had a significant 34% lower risk of developing MetSyn than lower HLSes (0 to 3) | [116] |

| | | | | |
|---|--|----------|---|--------------|
| <p>⑨ Working at least 40 h/week.</p> | | | | |
| <p>HLI: ① PA* (low/moderate/high), ② Diet⁺ (1st quintile 2nd quintile 3rd quintile 4th quintile 5th quintile), ③ Smoking (current smoker former smoker never smoker), ④ Alcohol use (> 60 g/day 24.0–59.9 g/day 12.0–23.9 g/day 6.0–11.9 g/day < 6.0 g/day) ⑤ BMI (≥ 30 26–29.9 24–25.9 22–23.9 < 22).</p> | <p>0-20 (≤ 10 11-15 ≥ 16)</p> | <p>⑤</p> | <p>A higher HLI score was associated with a lower risk of MetS.</p> | <p>[117]</p> |
| <p>① Moderate alcohol consumption (W ≤ 14 g/day, M ≤ 28 g/day), ② Never/quit smoking, ③ Healthy diet, ④ Sufficient PA (≥ 150 min/week or ≥ 5 d/week ≥ 75 min/week or at least once a week), ⑤ Social support**, ⑥ Less sedentary behaviour (≤4 h/day) ⑦ Good sleep (7–8 h/night)</p> | <p>0-7[#] (Q1 Q2-Q4 Q5)</p> | <p>⑥</p> | <p>A favorable lifestyle is associated with a lower risk of mortality and incidence of cancer among populations with MetS</p> | <p>[118]</p> |
| <p>HLS⁺⁺: ① PA (inactive/ active), ② Diet quality (HEI-2015), ③ Smoking (no/yes), ④ Obesity (no/yes).</p> | <p>0-4 (Q1/Q2/Q3/Q4)</p> | <p>②</p> | <p>Women in the first quartile of HLS had 67% increased odds of MetS compared with those in the bottom quartile</p> | <p>[119]</p> |
| <p>MEDLIFE^{###}: ① Food consumption (15 items), ② Dietary habits (7 items), ③ PA, rest and conviviality (6 items).</p> | <p>0-26 (T₁/T₂/T₃)</p> | <p>②</p> | <p>MetS was 71% less likely among participants with higher MEDLIFE adherence than those with lower MEDLIFE adherence</p> | <p>[120]</p> |

*: Chinese version of the International Physical Activity Questionnaire-Short; +: Beneficial food intake was scored from 0 (never) to 7 (always), while harmful food was scored from 0 (always) to 7 (never). Moreover, a total diet score was the sum of the four food types, ranging from 0 to 28; ++: IPAQ-SF, FFQ, : USDA's Food Composition Table, dietary guidelines for Americans (DGA)-HEI-2015; **: described as having at least two of the following social connections: friend/family visits at least once a week, being able to confide in someone close, having a close relationship with family, or having leisure/social activities; #: weighted healthy lifestyle scores = (β-1 × factor-1 + β-2 × factor-2 + ... + β-7 × factor-7)

÷ (β-1 + β-2 + ... + β-7) × 7; ###: Harvard food-frequency questionnaire and modified Mediterranean diet score.

Individual lifestyle behaviors' consequences are interconnected and differ across diseases. Thus, a tailored scoring system for healthy lifestyles is essential to foster good habits and mitigate MetS prevalence. This system should account for personal habits, preferences, and objectives to generate a holistic lifestyle score indicative of overall health. Regularly monitoring and enhancing this score enables individuals to pinpoint areas for lifestyle refinement, lowering the likelihood of MetS and other ailments.

4. Conclusions

In conclusion, MetS poses a major global public health issue due to its widespread occurrence and substantially adverse effects on health and well-being. While medications exist for treating MetS, lifestyle adjustments are essential for effectively tackling this problem. Various lifestyle factors impact MetS, such as sleep duration, smoking, excessive alcohol consumption, and inadequate diet. Inadequate sleep and excessive sleep can lead to obesity and insulin issues [26]. Smoking has been shown to induce IR, lower HDL-C levels, and increase WC and TG levels [45]. Excessive alcohol consumption leads to abnormal glucose regulation [51]. Poor diet can lead to weight gain, inflammation, and increased MetS risk [23].

However, adopting a healthier lifestyle that includes a healthy diet, proper PA, moderate alcohol consumption, and avoiding smoking has been shown to prevent and improve MetS. The MD, in particular, is an effective dietary intervention. Regular PA can help increase metabolic rate, reduce fat accumulation, improve IR, and regulate glucose levels [110]. Moderate alcohol consumption has increased HDL-C levels [42]. Tea consumption has increased fat metabolism and insulin sensitivity [56,61]. Finally, research has also shown that coffee can decrease weight, blood sugar, and cholesterol levels [67].

Despite extensive research, there are still inconsistent and conflicting findings about the relationship between lifestyle factors and MetS, and lifestyle score development varies. First, there is a lack of consensus on the definition of MetS. Different organizations use different diagnostic criteria, making it difficult to compare study results. This variation makes it challenging to identify specific lifestyle factors that may contribute to MetS consistently. Second, most MetS studies are cross-sectional, which limits our ability to conclude the relationship between lifestyle and MetS. Longitudinal studies with individuals followed over time would provide more meaningful insights into the causal relationship between lifestyle and MetS. Third, there is a lack of standardization in measuring lifestyle factors. Many studies rely on self-reported measures, which are prone to bias. Finally, research on MetS and lifestyle has mainly

focused on high-income populations in developed nations, limiting the generalizability of the findings to diverse populations and environments. Despite offering some understanding, current research has various shortcomings hindering a comprehensive grasp of this issue. Overcoming these limitations by using standardized definitions, longitudinal studies, and objective lifestyle measures can enhance our understanding of MetS about lifestyle choices and facilitate the development of a tailored mechanism for evaluating healthy behaviors.

Overall, the results of these studies will allow early identification and intervention of modifiable risk factors for MetS. Adopting a healthier lifestyle, including a balanced diet, regular PA, moderate alcohol consumption, avoiding smoking, and adequate sleep, remains critical to reducing the incidence and impact of MetS. Healthcare providers and policymakers should promote awareness and education about these lifestyle changes to improve human health.

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Reference

1. Desroches S, Lamarche B. The evolving definitions and increasing prevalence of the metabolic syndrome. *Appl Physiol Nutr Metab*, 2007;32:23-32. <https://doi.org/10.1139/h06-095>
2. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, et

- al. **Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement.** *Circulation*, 2005;112:2735-2752. <https://doi.org/10.1161/CIRCULATIONAHA.105.169404>
3. O'Neill S, O'Driscoll L. **Metabolic syndrome: a closer look at the growing epidemic and its associated pathologies.** *Obes Rev*, 2015;16:1-12. <https://doi.org/10.1111/obr.12229>
 4. Saklayen MG. **The Global Epidemic of the Metabolic Syndrome.** *Curr Hypertens Rep*, 2018;20:12. <https://doi.org/10.1007/s11906-018-0812-z>
 5. Chowdhury MZI, Anik AM, Farhana Z, Bristi PD, Abu Al Mamun BM, Uddin MJ, et al. **Prevalence of metabolic syndrome in Bangladesh: a systematic review and meta-analysis of the studies.** *BMC Public Health*, 2018;18:308. <https://doi.org/10.1186/s12889-018-5209-z>
 6. Huang Y, Zhang L, Wang Z, Wang X, Chen Z, Shao L, et al. **The prevalence and characteristics of metabolic syndrome according to different definitions in China: a nationwide cross-sectional study, 2012-2015.** *BMC Public Health*, 2022;22:1869. <https://doi.org/10.1186/s12889-022-14263-w>
 7. Cornier MA, Dabelea D, Hernandez TL, Lindstrom RC, Steig AJ, Stob NR, et al. **The metabolic syndrome.** *Endocr Rev*, 2008;29:777-822. <https://doi.org/10.1210/er.2008-0024>
 8. Myers J, Kokkinos P, Nyelin E. **Physical Activity, Cardiorespiratory Fitness, and the Metabolic Syndrome.** *Nutrients*, 2019; 11:1652. <https://doi.org/10.3390/nu11071652>
 9. Pucci G, Alcidi R, Tap L, Battista F, Mattace-Raso F, Schillaci G. **Sex- and gender-related prevalence, cardiovascular risk and therapeutic approach in metabolic syndrome: A review of the literature.** *Pharmacol Res*, 2017;120:34-42. <https://doi.org/10.1016/j.phrs.2017.03.008>
 10. Harrison S, Couture P, Lamarche B. **Diet Quality, Saturated Fat and Metabolic Syndrome.** *Nutrients*, 2020;12:3232. <https://doi.org/10.3390/nu12113232>
 11. Alberti KG, Zimmet PZ. **Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation.** *Diabet Med*, 1998;15:539-553. [https://doi.org/10.1002/\(SICI\)1096-9136\(199807\)15:7<539::AID-DIA668>3.0.CO;2-S](https://doi.org/10.1002/(SICI)1096-9136(199807)15:7<539::AID-DIA668>3.0.CO;2-S)
 12. Alberti KG, Zimmet P, Shaw J, Group IDFETFC. **The metabolic syndrome--a new worldwide definition.** *Lancet*, 2005;366: 10 59-1062. [https://doi.org/10.1016/S0140-6736\(05\)67402-8](https://doi.org/10.1016/S0140-6736(05)67402-8)
 13. Grundy SM, Brewer HB, Jr., Cleeman JI, Smith SC, Jr., Lenfant C, National Heart L, et al. **Definition of metabolic syndrome: report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition.** *Arterioscler Thromb Vasc Biol*, 2004;24:e13-18. <https://doi.org/10.1161/01.ATV.0000111245.75752.C6>
 14. Alberti G, Zimmet P, Shaw J, Bloomgarden Z, Kaufman F, Silink M, et al. **Type 2 diabetes in the young: the evolving epidemic: the international diabetes federation consensus workshop.** *Diabetes Care*, 2004;27:1798-1811. <https://doi.org/10.2337/diacare.27.7.1798>
 15. Song P, Yu J, Chang X, Wang M, An L. **Prevalence and Correlates of Metabolic Syndrome in Chinese Children: The China Health and Nutrition Survey.** *Nutrients*, 2017;9:79. <https://doi.org/10.3390/nu9010079>
 16. Cook S, Weitzman M, Auinger P, Nguyen M, Dietz WH. **Prevalence of a metabolic syndrome phenotype in adolescents: findings from the third National Health and Nutrition Examination Survey, 1988-1994.** *Arch Pediatr Adolesc Med*, 2003;157:821-827. <https://doi.org/10.1001/archpedi.157.8.8>

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17. Balkau B, Charles MA. Comment on the provisional report from the WHO consultation. European Group for the Study of Insulin Resistance (EGIR). *Diabet Med*, 1999;16:442-443. <https://doi.org/10.1046/j.1464-5491.1999.00059.x>
18. Christian Flemming GM, Bussler S, Korner A, Kiess W. Definition and early diagnosis of metabolic syndrome in children. *J Pediatr Endocrinol Metab*, 2020;33:821-833. <https://doi.org/10.1515/jpem-2019-0552>
19. Gonzalez-Gil EM, Anguita-Ruiz A, Kalen A, De Las Lamas Perez C, Ruperez AI, Vazquez-Cobela R, et al. Longitudinal associations between cardiovascular biomarkers and metabolic syndrome during puberty: the PUBMEP study. *Eur J Pediatr*, 2023;182:419-429. <https://doi.org/10.1007/s00431-022-04702-6>
20. Choi DH, Hur YI, Kang JH, Kim K, Cho YG, Hong SM, et al. Usefulness of the Waist Circumference-to-Height Ratio in Screening for Obesity and Metabolic Syndrome among Korean Children and Adolescents: Korea National Health and Nutrition Examination Survey, 2010-2014. *Nutrients*, 2017;9:256. <https://doi.org/10.3390/nu9030256>
21. Sjoros T, Laine S, Garthwaite T, Vahaypya H, Loyttyneimi E, Koivumaki M, et al. Reducing Sedentary Time and Whole-Body Insulin Sensitivity in Metabolic Syndrome: A 6-Month Randomized Controlled Trial. *Med Sci Sports Exerc*, 2023;55:342-353. <https://doi.org/10.1249/MSS.00000000000003054>
22. Kastorini CM, Milionis HJ, Esposito K, Giugliano D, Goudevenos JA, Panagiotakos DB. The effect of Mediterranean diet on metabolic syndrome and its components: a meta-analysis of 50 studies and 534,906 individuals. *J Am Coll Cardiol*, 2011;57:1299-1313. <https://doi.org/10.1016/j.jacc.2010.09.073>
23. Sweazea KL. Compounding evidence implicating Western diets in the development of metabolic syndrome. *Acta Physiol (Oxf)*, 2014;211:471-473. <https://doi.org/10.1111/apha.12303>
24. Mascaro CM, Bouzas C, Montemayor S, Casares M, Llompарт I, Ugarriza L, et al. Effect of a Six-Month Lifestyle Intervention on the Physical Activity and Fitness Status of Adults with NAFLD and Metabolic Syndrome. *Nutrients*, 2022;14:1813. <https://doi.org/10.3390/nu14091813>
25. Albqoor MA, Shaheen AM. Sleep quality, sleep latency, and sleep duration: a national comparative study of university students in Jordan. *Sleep Breath*, 2021;25:1147-1154. <https://doi.org/10.1007/s11325-020-02188-w>
26. Che T, Yan C, Tian D, Zhang X, Liu X, Wu Z. The Association Between Sleep and Metabolic Syndrome: A Systematic Review and Meta-Analysis. *Front Endocrinol (Lausanne)*, 2021;12:773646. <https://doi.org/10.3389/fendo.2021.773646>
27. Rafati S, Isheh M, Azarbad A, Ghadiri Soufi F, Rahimi A, Kheirandish M. The association of sleep duration and metabolic syndrome in the Bandare-Kong cohort study, a cross-sectional survey (finding from PERSIAN cohort study). *Diabetol Metab Syndr*, 2021;13:114. <https://doi.org/10.1186/s13098-021-00737-1>
28. Liu X, Huang L, Wu Q, Chen Y, Chen X, Chen H, et al. Sleep characteristic profiles and the correlation with spectrum of metabolic syndrome among older adult: a cross-sectional study. *BMC Geriatr*, 2022; 22:414. <https://doi.org/10.1186/s12877-022-03074-8>
29. Iftikhar IH, Donley MA, Mindel J, Pleister A, Soriano S, Magalang UJ. Sleep Duration and Metabolic Syndrome. An Updated Dose-Risk Metaanalysis. *Ann Am Thorac Soc*, 2015;12:1364-1372. <https://doi.org/10.1513/AnnalsATS.201504-190OC>
30. Xi B, He D, Zhang M, Xue J, Zhou D. Short sleep duration predicts risk of

- metabolic syndrome: a systematic review and meta-analysis. *Sleep Med Rev*, 2014;18:293-297. <https://doi.org/10.1016/j.smr.2013.06.001>
31. Kim CE, Shin S, Lee HW, Lim J, Lee JK, Shin A, et al. Association between sleep duration and metabolic syndrome: a cross-sectional study. *BMC Public Health*, 2018; 18:720. <https://doi.org/10.1186/s12889-018-5557-8>
32. Peila R, Xue X, Feliciano EMC, Allison M, Sturgeon S, Zaslavsky O, et al. Association of sleep duration and insomnia with metabolic syndrome and its components in the Women's Health Initiative. *BMC Endocr Disord*, 2022;22: 228. <https://doi.org/10.1186/s12902-022-01138-9>
33. Smiley A, King D, Bidulescu A. The Association between Sleep Duration and Metabolic Syndrome: The NHANES 2013/2014. *Nutrients*, 2019;11:2582. <https://doi.org/10.3390/nu11112582>
34. Xiao J, Shen C, Chu MJ, Gao YX, Xu GF, Huang JP, et al. Physical Activity and Sedentary Behavior Associated with Components of Metabolic Syndrome among People in Rural China. *PLoS One*, 2016; 11:e0147062. <https://doi.org/10.1371/journal.pone.0147062>
35. Duan Y, Sun J, Wang M, Zhao M, Magnussen CG, Xi B. Association between short sleep duration and metabolic syndrome in Chinese children and adolescents. *Sleep Med*, 2020;74:343-348. <https://doi.org/10.1016/j.sleep.2020.08.018>
36. Sun J, Wang M, Yang L, Zhao M, Bovet P, Xi B. Sleep duration and cardiovascular risk factors in children and adolescents: A systematic review. *Sleep Med Rev*, 2020;53: 101338. <https://doi.org/10.1016/j.smr.2020.101338>
37. Chaudhry BA, Brian MS, Morrell JS. The Relationship between Sleep Duration and Metabolic Syndrome Severity Scores in Emerging Adults. *Nutrients*, 2023;15:1046. <https://doi.org/10.3390/nu15041046>
38. Titova OE, Lindberg E, Elmstahl S, Lind L, Schioth HB, Benedict C. Associations Between the Prevalence of Metabolic Syndrome and Sleep Parameters Vary by Age. *Front Endocrinol (Lausanne)*, 2018;9: 234. <https://doi.org/10.3389/fendo.2018.00234>
39. Wang Y, Qian YX, Liu JH, Miao YM, Ma QH, Pan CW. Longitudinal association between sleep and 5-year incident metabolic syndrome in older Chinese adults: a community-based cohort study. *Sleep Med*, 2021;81:1-7. <https://doi.org/10.1016/j.sleep.2021.02.004>
40. Li W, Kondracki AJ, Sun N, Gautam P, Kalan ME, Jebai R, et al. Nighttime sleep duration, daytime napping, and metabolic syndrome: findings from the China Health and Retirement Longitudinal Study. *Sleep Breath*, 2022;26:1427-1435. <https://doi.org/10.1007/s11325-021-02487-w>
41. Kim SW, Kim HJ, Min K, Lee H, Lee SH, Kim S, et al. The relationship between smoking cigarettes and metabolic syndrome: A cross-sectional study with non-single residents of Seoul under 40 years old. *PLoS One*, 2021;16:e0256257. <https://doi.org/10.1371/journal.pone.0256257>
42. Yao F, Bo Y, Zhao L, Li Y, Ju L, Fang H, et al. Prevalence and Influencing Factors of Metabolic Syndrome among Adults in China from 2015 to 2017. *Nutrients*, 2021; 13:4475. <https://doi.org/10.3390/nu13124475>
43. Slagter SN, van Vliet-Ostaptchouk JV, Vonk JM, Boezen HM, Dullaart RP, Kobold AC, et al. Associations between smoking, components of metabolic syndrome and lipoprotein particle size. *BMC Med*, 2013;11:195. <https://doi.org/10.1186/1741-7015-11-195>
44. Meysamie A, Ghalehtaki R, Ghodsi S, Mohebi M, Ghalehtaki S, Salarvand F, et al. Is there an independent association between metabolic syndrome and smoking in Iranian adults? Results of a large multicenter national survey. *Caspian J Intern Med*, 2021;12:327-335. <https://doi.org/10.22088/cjim.12.3.327>
45. Park S, Han K, Lee S, Kim Y, Lee Y,

- Kang MW, et al. Smoking, development of or recovery from metabolic syndrome, and major adverse cardiovascular events: A nationwide population-based cohort study including 6 million people. *PLoS One*, 2021;16:e0241623. <https://doi.org/10.1371/journal.pone.0241623>
46. Chen CC, Li TC, Chang PC, Liu CS, Lin WY, Wu MT, et al. Association among cigarette smoking, metabolic syndrome, and its individual components: the metabolic syndrome study in Taiwan. *Metabolism*, 2008;57:544-548. <https://doi.org/10.1016/j.metabol.2007.11.018>
47. Suutari-Jaasko A, Ylitalo A, Ronkainen J, Huikuri H, Kesaniemi YA, Ukkola OH. Smoking cessation and obesity-related morbidities and mortality in a 20-year follow-up study. *PLoS One*, 2022;17:e0279443. <https://doi.org/10.1371/journal.pone.0279443>
48. Yoon C, Goh E, Park SM, Cho B. Effects of smoking cessation and weight gain on cardiovascular disease risk factors in Asian male population. *Atherosclerosis*, 2010;208:275-279. <https://doi.org/10.1016/j.atherosclerosis.2009.07.024>
49. Shin HS, Oh JE, Cho YJ. The Association Between Smoking Cessation Period and Metabolic Syndrome in Korean Men. *Asia Pac J Public Health*, 2018;30:415-424. <https://doi.org/10.1177/1010539518786517>
50. Biddinger KJ, Emdin CA, Haas ME, Wang M, Hindy G, Ellinor PT, et al. Association of Habitual Alcohol Intake With Risk of Cardiovascular Disease. *JAMA Netw Open*, 2022;5:e223849. <https://doi.org/10.1001/jamanetworkopen.2022.3849>
51. Lin Y, Ying YY, Li SX, Wang SJ, Gong QH, Li H. Association between alcohol consumption and metabolic syndrome among Chinese adults. *Public Health Nutr*, 2021;24:4582-4590. <https://doi.org/10.1017/S1368980020004449>
52. Lee SW, Jang SI. Association of Alcohol Drinking Patterns with Metabolic Syndrome and Its Components in Korean Adults: The Korea National Health and Nutrition Examination Survey 2016-2018. *Int J Environ Res Public Health*, 2021; 18:6433. <https://doi.org/10.3390/ijerph18126433>
53. Cho KI, Kim BH, Je HG, Jang JS, Park YH. Gender-Specific Associations between Socioeconomic Status and Psychological Factors and Metabolic Syndrome in the Korean Population: Findings from the 2013 Korean National Health and Nutrition Examination Survey. *Biomed Res Int*, 2016;2016:3973197. <https://doi.org/10.1155/2016/3973197>
54. Shimoshikiryo I, Ibusuki R, Shimatani K, Nishimoto D, Takezaki T, Nishida Y, et al. Association between alcohol intake pattern and metabolic syndrome components and simulated change by alcohol intake reduction: A cross-sectional study from the Japan Multi-Institutional Collaborative Cohort Study. *Alcohol*, 2020;89:129-138. <https://doi.org/10.1016/j.alcohol.2020.09.002>
55. Lee K, Giovannucci EL, Kim J. The Effect of Smoking and Sex on the Association Between Long-term Alcohol Consumption and Metabolic Syndrome in a Middle-aged and Older Population. *J Epidemiol*, 2021;31:249-258. <https://doi.org/10.2188/jea.JE20190328>
56. Sirotkin AV, Kolesarova A. The anti-obesity and health-promoting effects of tea and coffee. *Physiol Res*, 2021;70:161-168. <https://doi.org/10.33549/physiolres.934674>
57. Li K, Zhang Q, Cai H, He R, Nima Q, Li Y, et al. Association of Tibetan Habitual Food and Metabolic Syndrome Among Tibetan People in China: A Cross-Sectional Study. *Front Nutr*, 2022;9:888317. <https://doi.org/10.3389/fnut.2022.888317>
58. Yang CS, Zhang J, Zhang L, Huang J, Wang Y. Mechanisms of body weight reduction and metabolic syndrome alleviation by tea. *Mol Nutr Food Res*,

- 20 16;60:160-174.
<https://doi.org/10.1002/mnfr.201500428>
59. Phung OJ, Baker WL, Matthews LJ, Lanosa M, Thorne A, Coleman CI. Effect of green tea catechins with or without caffeine on anthropometric measures: a systematic review and meta-analysis. *Am J Clin Nutr*, 2010;91:73-81.
<https://doi.org/10.3945/ajcn.2009.28157>
60. Li X, Wang W, Hou L, Wu H, Wu Y, Xu R, et al. Does tea extract supplementation benefit metabolic syndrome and obesity? A systematic review and meta-analysis. *Clin Nutr*, 2020;39:1049-1058. <https://doi.org/10.1016/j.clnu.2019.05.019>
61. Liu W, Wan C, Huang Y, Li M. Effects of tea consumption on metabolic syndrome: A systematic review and meta-analysis of randomized clinical trials. *Phytother Res*, 2020;34:2857-2866. <https://doi.org/10.1002/ptr.6731>
62. Yang Y, Yu D, Piao W, Huang K, Zhao L. Association between Habitual Tea Consumption and Metabolic Syndrome and Its Components among Chinese Adults Aged 18~59 Years: Based on China Nutrition and Health Surveillance 2015-2017. *Nutrients*, 2022;14:3502. <https://doi.org/10.3390/nu14173502>
63. Hino A, Adachi H, Enomoto M, Furuki K, Shigetoh Y, Ohtsuka M, et al. Habitual coffee but not green tea consumption is inversely associated with metabolic syndrome: an epidemiological study in a general Japanese population. *Diabetes Res Clin Pract*, 2007;76:383-389. <https://doi.org/10.1016/j.diabres.2006.09.033>
64. Wong THT, Burlutsky G, Gopinath B, Flood VM, Mitchell P, Louie JCY. The longitudinal association between coffee and tea consumption and the risk of metabolic syndrome and its component conditions in an older adult population. *J Nutr Sci*, 2022;11:e79. <https://doi.org/10.1017/jns.2022.78>
65. Nouri M, Shateri Z, Rezaei M, Zangene A, Homayounfar R, Keshani P. Healthy beverage index could decrease odds of metabolic syndrome: A cross-sectional study. *Food Sci Nutr*, 2023;11:4562-4571. <https://doi.org/10.1002/fsn3.3415>
66. He B, Long W, Li X, Yang W, Chen Y, Zhu Y. Sugar-Sweetened Beverages Consumption Positively Associated with the Risks of Obesity and Hypertriglyceridemia Among Children Aged 7-18 Years in South China. *J Atheroscler Thromb*, 2018;25:81-89. <https://doi.org/10.5551/jat.38570>
67. Terentes-Printzios D, Vlachopoulos C. Coffee and cardiovascular health: looking through the steaming cup. *Cardiovasc Res*, 2022;118:e51-e53. <https://doi.org/10.1093/cvr/cvac045>
68. Lu MY, Cheng HY, Lai JC, Chen SJ. The Relationship between Habitual Coffee Drinking and the Prevalence of Metabolic Syndrome in Taiwanese Adults: Evidence from the Taiwan Biobank Database. *Nutrients*, 2022;14:1867. <https://doi.org/10.3390/nu14091867>
69. Shin S, Lim J, Lee HW, Kim CE, Kim SA, Lee JK, et al. Association between the prevalence of metabolic syndrome and coffee consumption among Korean adults: results from the Health Examinees study. *Appl Physiol Nutr Metab*, 2019;44:1371-1378. <https://doi.org/10.1139/apnm-2018-0880>
70. Kim Y, Je Y. Moderate coffee consumption is inversely associated with the metabolic syndrome in the Korean adult population. *Br J Nutr*, 2018;120:1279-1287. <https://doi.org/10.1017/S0007114518002635>
71. Takami H, Nakamoto M, Uemura H, Katsuura S, Yamaguchi M, Hiyoshi M, et al. Inverse correlation between coffee consumption and prevalence of metabolic syndrome: baseline survey of the Japan Multi-Institutional Collaborative Cohort (J-MICC) Study in Tokushima, Japan. *J Epidemiol*, 2013;23:12-20. <https://doi.org/10.2188/jea.je20120053>
72. Stutz B, Ahola AJ, Harjutsalo V, Forsblom C, Groop PH, FinnDiane Study G. Association between habitual coffee consumption and metabolic

- syndrome in type 1 diabetes. *Nutr Metab Cardiovasc Dis*, 2018;28:470-476. <https://doi.org/10.1016/j.numecd.2018.01.011>
73. Senftinger J, Nikorowitsch J, Borof K, Ojeda F, Aarabi G, Beikler T, et al. Coffee consumption and associations with blood pressure, LDL-cholesterol and echocardiographic measures in the general population. *Sci Rep*, 2023;13:4668. <https://doi.org/10.1038/s41598-023-31857-5>
74. Nina R, Lingling H, Qiushuang L, Honglin G, Liyuan S, Yuting Z. Association of coffee consumption pattern and metabolic syndrome among middle-aged and older adults: A cross-sectional study. *Front Public Health*, 2023;11:1022616. <https://doi.org/10.3389/fpubh.2023.1022616>
75. Kim HJ, Cho S, Jacobs DR, Jr., Park K. Instant coffee consumption may be associated with higher risk of metabolic syndrome in Korean adults. *Diabetes Res Clin Pract*, 2014;106:145-153. <https://doi.org/10.1016/j.diabres.2014.07.007>
76. Tan LJ, Jeon HJ, Park S, Kim SA, Lim K, Chung S, et al. Association of Coffee Consumption and Its Types According to Addition of Sugar and Creamer with Metabolic Syndrome Incidence in a Korean Population from the Health Examinees (HEXA) Study. *Nutrients*, 2021;13:920. <https://doi.org/10.3390/nu13030920>
77. Sarria B, Martinez-Lopez S, Sierra-Cinos JL, Garcia-Diz L, Mateos R, Bravo-Clemente L. Regularly consuming a green/roasted coffee blend reduces the risk of metabolic syndrome. *Eur J Nutr*, 2018; 57:269-278. <https://doi.org/10.1007/s00394-016-1316-8>
78. Kim JH, Park YS, Kim H. Association between metabolic syndrome and coffee consumption in the Korean population by gender: a cross-sectional study in Korea. *Asia Pac J Clin Nutr*, 2018;27:1131-1140. <https://doi.org/10.6133/apjcn.022018.04>
79. Driessen MT, Koppes LL, Veldhuis L, Samoocha D, Twisk JW. Coffee consumption is not related to the metabolic syndrome at the age of 36 years: the Amsterdam Growth and Health Longitudinal Study. *Eur J Clin Nutr*, 2009; 63:536-542. <https://doi.org/10.1038/ejcn.2008.6>
80. Balk L, Hoekstra T, Twisk J. Relationship between long-term coffee consumption and components of the metabolic syndrome: the Amsterdam Growth and Health Longitudinal Study. *Eur J Epidemiol*, 2009; 24:203-209. <https://doi.org/10.1007/s10654-009-9323-1>
81. Bovolini A, Garcia J, Andrade MA, Duarte JA. Metabolic Syndrome Pathophysiology and Predisposing Factors. *Int J Sports Med*, 2021;42:199-214. <https://doi.org/10.1055/a-1263-0898>
82. Song P, Zhang X, Li Y, Man Q, Jia S, Zhang J, et al. MetS Prevalence and Its Association with Dietary Patterns among Chinese Middle-Aged and Elderly Population: Results from a National Cross-Sectional Study. *Nutrients*, 2022;14:5301. <https://doi.org/10.3390/nu14245301>
83. Barbosa LB, Vasconcelos NBR, Dos Santos EA, Dos Santos TR, Ataíde-Silva T, Ferreira HDS. Ultra-processed food consumption and metabolic syndrome: a cross-sectional study in Quilombola communities of Alagoas, Brazil. *Int J Equity Health*, 2023;22:14. <https://doi.org/10.1186/s12939-022-01816-z>
84. Willems AEM, Sura-de Jong M, van Beek AP, Nederhof E, van Dijk G. Effects of macronutrient intake in obesity: a meta-analysis of low-carbohydrate and low-fat diets on markers of the metabolic syndrome. *Nutr Rev*, 2021;79:429-444. <https://doi.org/10.1093/nutrit/nuaa044>
85. Shemirani F, Djafarian K, Fotouhi A, Azadbakht L, Rezaei N, Chamari M, et al. Effect of Paleolithic-based low-carbohydrate vs. moderate-carbohydrate diets with portion-control and calorie-counting on CTRP6, asprosin and metabolic markers in

- adults with metabolic syndrome: A randomized clinical trial. *Clin Nutr ESPEN*, 2022;48:87-98. <https://doi.org/10.1016/j.clnesp.2021.11.013>
86. Asadi Z, Shafiee M, Sadabadi F, Saberi-Karimian M, Darroudi S, Tayefi M, et al. Association Between dietary patterns and the risk of metabolic syndrome among Iranian population: A cross-sectional study. *Diabetes Metab Syndr*, 2019;13:858-865. <https://doi.org/10.1016/j.dsx.2018.11.059>
87. Wang Z, Zhang B, Zhai F, Wang H, Zhang J, Du W, et al. Fatty and lean red meat consumption in China: differential association with Chinese abdominal obesity. *Nutr Metab Cardiovasc Dis*, 2014;24:869-876. <https://doi.org/10.1016/j.numecd.2014.03.002>
88. Wang Z, Huang Q, Wang L, Jiang H, Wang Y, Wang H, et al. Moderate Intake of Lean Red Meat was Associated with Lower Risk of Elevated Blood Pressure in Chinese Women: Results from the China Health and Nutrition Survey, 1991-2015. *Nutrients*, 2020;12:1369. <https://doi.org/10.3390/nu12051369>
89. Kim SA, Shin S. Red meat and processed meat consumption and the risk of dyslipidemia in Korean adults: A prospective cohort study based on the Health Examinees (HEXA) study. *Nutr Metab Cardiovasc Dis*, 2021;31:1714-1727. <https://doi.org/10.1016/j.numecd.2021.02.008>
90. Gallardo-Alfaro L, Bibiloni MDM, Mascaró CM, Montemayor S, Ruiz-Canela M, Salas-Salvado J, et al. Leisure-Time Physical Activity, Sedentary Behaviour and Diet Quality are Associated with Metabolic Syndrome Severity: The PREDIMED-Plus Study. *Nutrients*, 2020;12:1013. <https://doi.org/10.3390/nu12041013>
91. Hosseinpour-Niazi S, Bakhshi B, Mirmiran P, Azizi F. Socioeconomic and lifestyle factors modifies the association between nut consumption and metabolic syndrome incidence. *Clin Nutr*, 2021;40:4055-4064. <https://doi.org/10.1016/j.clnu.2021.02.013>
92. Yuan S, Yu HJ, Liu MW, Huang Y, Yang XH, Tang BW, et al. The association of fruit and vegetable consumption with changes in weight and body mass index in Chinese adults: a cohort study. *Public Health*, 2018;157:121-126. <https://doi.org/10.1016/j.puhe.2018.01.027>
93. Shin JY, Kim JY, Kang HT, Han KH, Shim JY. Effect of fruits and vegetables on metabolic syndrome: a systematic review and meta-analysis of randomized controlled trials. *Int J Food Sci Nutr*, 2015;66:416-425. <https://doi.org/10.3109/09637486.2015.1025716>
94. Thomas MS, Huang L, Garcia C, Sakaki JR, Blesso CN, Chun OK, et al. The Effects of Eggs in a Plant-Based Diet on Oxidative Stress and Inflammation in Metabolic Syndrome. *Nutrients*, 2022;14:2548. <https://doi.org/10.3390/nu14122548>
95. Thomas MS, Puglisi M, Malysheva O, Caudill MA, Sholola M, Cooperstone JL, et al. Eggs Improve Plasma Biomarkers in Patients with Metabolic Syndrome Following a Plant-Based Diet-A Randomized Crossover Study. *Nutrients*, 2022;14:2138. <https://doi.org/10.3390/nu14102138>
96. Jin S, Je Y. Dairy Consumption and Risk of Metabolic Syndrome: Results from Korean Population and Meta-Analysis. *Nutrients*, 2021;13:1574. <https://doi.org/10.3390/nu13051574>
97. Babio N, Toledo E, Estruch R, Ros E, Martínez-González MA, Castaner O, et al. Mediterranean diets and metabolic syndrome status in the PREDIMED randomized trial. *CMAJ*, 2014;186:E649-657. <https://doi.org/10.1503/cmaj.140764>
98. Slattery ML. Analysis of dietary patterns in epidemiological research. *Appl Physiol Nutr Metab*, 2010;35:207-210. <https://doi.org/10.1139/H10-006>
99. Velazquez-Lopez L, Santiago-Diaz G, Nava-Hernandez J, Muñoz-Torres AV, Medina-Bravo P, Torres-Tamayo M.

- Mediterranean-style diet reduces metabolic syndrome components in obese children and adolescents with obesity. *BMC Pediatr*, 2014;14:175. <https://doi.org/10.1186/1471-2431-14-175>
100. Montemayor S, Bouzas C, Mascaro CM, Casares M, Llompарт I, Abete I, et al. Effect of Dietary and Lifestyle Interventions on the Amelioration of NAFLD in Patients with Metabolic Syndrome: The FLIPAN Study. *Nutrients*, 2022;14:2223. <https://doi.org/10.3390/nu14112223>
101. Fabiani R, Naldini G, Chiavarini M. Dietary Patterns and Metabolic Syndrome in Adult Subjects: A Systematic Review and Meta-Analysis. *Nutrients*, 2019;11:2056. <https://doi.org/10.3390/nu11092056>
102. Suliga E, Koziel D, Ciesla E, Rebak D, Gluszek S. Dietary Patterns in Relation to Metabolic Syndrome among Adults in Poland: A Cross-Sectional Study. *Nutrients*, 2017;9:1366. <https://doi.org/10.3390/nu9121366>
103. Hassannejad R, Kazemi I, Sadeghi M, Mohammadifard N, Roohafza H, Sarrafzadegan N, et al. Longitudinal association of metabolic syndrome and dietary patterns: A 13-year prospective population-based cohort study. *Nutr Metab Cardiovasc Dis*, 2018;28:352-360. <https://doi.org/10.1016/j.numecd.2017.10.025>
104. Brader L, Uusitupa M, Dragsted LO, Hermansen K. Effects of an isocaloric healthy Nordic diet on ambulatory blood pressure in metabolic syndrome: a randomized SYSDIET sub-study. *Eur J Clin Nutr*, 2014;68:57-63. <https://doi.org/10.1038/ejcn.2013.192>
105. Choi JH, Woo HD, Lee JH, Kim J. Dietary Patterns and Risk for Metabolic Syndrome in Korean Women: A Cross-Sectional Study. *Medicine (Baltimore)*, 2015;94:e1424. <https://doi.org/10.1097/MD.0000000000001424>
106. Freeman AM, Pennings N. in *StatPearls* (2023).
107. Henson J, Yates T, Biddle SJ, Edwardson CL, Khunti K, Wilmot EG, et al. Associations of objectively measured sedentary behaviour and physical activity with markers of cardiometabolic health. *Diabetologia*, 2013;56:1012-1020. <https://doi.org/10.1007/s00125-013-2845-9>
108. Wu J, Zhang H, Yang L, Shao J, Chen D, Cui N, et al. Sedentary time and the risk of metabolic syndrome: A systematic review and dose-response meta-analysis. *Obes Rev*, 2022;23:e13510. <https://doi.org/10.1111/obr.13510>
109. Alkahtani S, Elkilany A, Alhariri M. Association between sedentary and physical activity patterns and risk factors of metabolic syndrome in Saudi men: A cross-sectional study. *BMC Public Health*, 2015;15:1234. <https://doi.org/10.1186/s12889-015-2578-4>
110. Saladini F, Palatini P. Arterial Distensibility, Physical Activity, and the Metabolic Syndrome. *Curr Hypertens Rep*, 2018;20:39. <https://doi.org/10.1007/s11906-018-0837-3>
111. Stewart KJ, Bacher AC, Turner K, Lim JG, Hees PS, Shapiro EP, et al. Exercise and risk factors associated with metabolic syndrome in older adults. *Am J Prev Med*, 2005;28:9-18. <https://doi.org/10.1016/j.amepre.2004.09.006>
112. Yao CH, Zuo HJ, Kong LZ, Yang XG, Zhai FY, Technical Working Group of China National N, et al. [Relationship between physical activity and metabolic syndrome]. *Zhonghua Yi Xue Za Zhi*, 2006;86:2099-2104.
113. Galmes-Panades AM, Konieczna J, Varela-Mato V, Abete I, Babio N, Fiol M, et al. Targeting body composition in an older population: do changes in movement behaviours matter? Longitudinal analyses in the PREDIMED-Plus trial. *BMC Med*, 2021;19:3. <https://doi.org/10.1186/s12916-020-01847-9>
114. Sheng J, Abshire DA, Heiney SP, Wirth MD. Acculturation, Physical Activity, and Metabolic Syndrome in Asian American Adults. *J Transcult Nurs*, 2022;33:675-684. <https://doi.org/10.1177/10436596221114150>
115. Zhou Y, Wu W, Zou Y, Huang W, Lin S, Ye J, et al. Benefits of different

- combinations of aerobic and resistance exercise for improving plasma glucose and lipid metabolism and sleep quality among elderly patients with metabolic syndrome: a randomized controlled trial. *Endocr J*, 2022;69:819-830. <https://doi.org/10.1507/endocrj.EJ21-0589>
116. Garralda-Del-Villar M, Carlos-Chilleron S, Diaz-Gutierrez J, Ruiz-Canela M, Gea A, Martinez-Gonzalez MA, et al. Healthy Lifestyle and Incidence of Metabolic Syndrome in the SUN Cohort. *Nutrients*, 2018;11:65. <https://doi.org/10.3390/nu11010065>
117. Ye Y, Zhou Q, Dai W, Peng H, Zhou S, Tian H, et al. Gender differences in metabolic syndrome and its components in southern china using a healthy lifestyle index: a cross-sectional study. *BMC Public Health*, 2023;23:686. <https://doi.org/10.1186/s12889-023-15584-0>
118. Wu E, Ni JT, Zhu ZH, Xu HQ, Tao L, Xie T. Association of a Healthy Lifestyle with All-Cause, Cause-Specific Mortality and Incident Cancer among Individuals with Metabolic Syndrome: A Prospective Cohort Study in UK Biobank. *Int J Environ Res Public Health*, 2022;19:9936. <https://doi.org/10.3390/ijerph19169936>
119. Vajdi M, Karimi A, Farhangi MA, Ardekani AM. The association between healthy lifestyle score and risk of metabolic syndrome in Iranian adults: a cross-sectional study. *BMC Endocr Disord*, 2023;23:16. <https://doi.org/10.1186/s12902-023-01270-0>
120. Hershey MS, Sotos-Prieto M, Ruiz-Canela M, Christophi CA, Moffatt S, Martinez-Gonzalez MA, et al. The Mediterranean lifestyle (MEDLIFE) index and metabolic syndrome in a non-Mediterranean working population. *Clin Nutr*, 2021;40:2494-2503. <https://doi.org/10.1016/j.clnu.2021.03.026>