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Certain dietary patterns are beneficial for the metabolic syndrome: reviewing the evidence

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ABSTRACT

The metabolic syndrome (MetS) is a global public health issue of increasing magnitude. The Asia-Pacific region is expected to be hardest hit due to large population numbers, rising obesity, and insulin resistance (IR). This review assessed the protective effects of dietary patterns and their components on MetS. A literature search was conducted using prominent electronic databases and search terms that included in combination: diet, dietary components, dietary patterns, and metabolic syndrome. Articles were restricted to prospective studies and high quality randomized controlled trials that were conducted on humans, reported in the English language, and within the time period of 2000 to 2012. Traditional factors such as age, gender, physical activity, and obesity were associated with risk of MetS; however, these potential confounders were not always accounted for in study outcomes. Three dietary patterns emerged from the review; a Mediterranean dietary pattern, dietary approaches to stop hypertension diet, and the Nordic Diet. Potential contributors to their beneficial effects on prevalence of MetS or reduction in MetS components included increases in fruits, vegetables, whole grains, dairy and dairy components, calcium, vitamin D, and whey protein, as well as monounsaturated fatty acids, and omega-3 fatty acids. Additional prospective and high quality randomized controlled trial studies that investigate Mediterranean dietary pattern, the dietary approaches to stop hypertension diet, and the Nordic Diet would cement the protective benefits of these diets against the MetS.

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1. Introduction

The metabolic syndrome (MetS) is a clustering of risk factors for cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM) which include: high blood pressure (BP), low fasting

high-density lipoprotein cholesterol (HDL), high fasting triglycerides (TG), high fasting blood glucose, and abdominal obesity [1]. In Caucasian populations, the presence of the MetS is associated with at least a 2-fold increased risk of CVD [2] and at least a five-fold increased risk for T2DM [3]. In Asian

Abbreviations: ALA, alpha-linoleic acid; BP, blood pressure; CHO, carbohydrate; CVD, cardiovascular disease; DASH, dietary approaches to stop hypertension diet; DHA, docosahexaenoic acid; EPA, eicosapentaenoic acid; GI, glycemic index; GL, glycemic load; HDL, high-density lipoprotein cholesterol; HF, high fiber; HP, high protein; IR, insulin resistance; IS, insulin sensitivity; IL, interleukin; LDL, low-density lipoprotein cholesterol; LFHCn-3, low fat high carbohydrate diet with omega-3; MDP, Mediterranean dietary pattern; MDS, Mediterranean dietary score; Mets, metabolic syndrome; MSDPS, Mediterranean style-dietary pattern score; MUFA, monounsaturated fatty acids; ND, Nordic diet; PA, physical activity; PUFA, polyunsaturated fatty acids; RCT, randomized controlled trial; T2DM, type two diabetes mellitus; TG, triglycerides; UMDS, updated Mediterranean dietary score; VOO, virgin olive oil; WC, waist circumference; WDP, Westernized dietary pattern.

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populations, the MetS is associated with even greater risks of CVD and T2DM [4], thus highlighting the importance and relevance of MetS worldwide. As with other chronic diseases, the prevalence of MetS is increasing [4,5], with current prevalence estimates in the Asia-Pacific region between ~10% and 30% [2,4,6].

Preventing and treating this syndrome is an area of public health urgency from the viewpoint of improving the morbidity and mortality statistics as well as in reducing its economic burden. The precise pathogenesis of the syndrome is still unknown with central adiposity [7], insulin resistance (IR) [8–10], and inflammation [11], all being individually implicated, despite the considerable inter-relationships between them [11,12]. Environmental factors that modulate MetS risk include exercise, climate, socioeconomic status, and diet. In a recent review [12], we highlighted the association between MetS and several metabolic factors. We postulated that the adipocytokine, adiponectin, may have a key role in MetS as many diets and nutrients benefit MetS through increases in adiponectin. The focus of this review was to examine the evidence on dietary patterns and their components that were protective for the syndrome. In doing so, we also aimed to identify priority areas for research.

2. Literature search procedure for diet and MetS

Studies were identified by conducting an electronic search of the following databases: Proquest Central, PubMed Central, Science Direct, and Wiley Online Library. Individual Journals were also searched for relevant studies, these included: *American Journal of Clinical Nutrition*, *Cardiovascular and Metabolic Risk*, *Circulation Journal*, *Diabetologia*, *European Journal of Clinical Nutrition*, and *Obesity Journal*. Studies known to the authors that satisfied the inclusion criteria outlined below were also included. The following key words, in various combinations, were used: diet, dietary patterns, dietary components, and metabolic syndrome. Articles were restricted to human studies in the English language that reported individual MetS components as primary endpoints and were published within the time period of 2000 to 2012. Prospective epidemiological studies which fulfilled the above criteria and high quality randomized controlled trial (RCTs), as determined by a Jadad score ≥ 3 [13] were then identified and included in this analysis, as their design provides high level evidence for causative links between diet and disease. Two reviewers (EKC and PKP) independently assessed the eligibility of all studies for inclusion. Any discrepancy was resolved through discussion.

3. Dietary patterns and MetS

We separated the evidence with prospective cohort studies ($n = 5$) that described diet and MetS relationships in Table 1 and high quality RCTs ($n = 7$), as determined by a Jadad score ≥ 3 [13], that examined the causal relationship between diet and MetS featured in Table 2. Three dietary patterns emerged from our analysis: a Mediterranean dietary pattern (MDP), a dietary approaches to stop hypertension (DASH) diet, and the Nordic Diet (ND). All dietary patterns showed benefits in at

least two of the MetS components, with two studies showing benefits in all five MetS components (Tables 1 and 2).

3.1. MetS and a Westernized dietary pattern

Although some studies have focused on the importance of individual foods for MetS [14], focusing on dietary patterns rather than single nutrients or food groups is preferred due to the expected synergistic effect on disease risk. The Dietary Guidelines for Australian Adults [15] advocates the following dietary pattern to reduce the risk of CVD and T2DM: plant foods including fruits, vegetables, legumes, and whole grain cereals; animal foods including meat, fish, and poultry; low fat dairy; limiting total fat intake to a moderate amount; limiting saturated fat; choosing foods low in salt; consuming only moderate amounts of sugar; and limiting alcohol intake. However, Australians typically consume low amounts of fruits and vegetables and high intakes of meat and processed convenience foods that are high in salt, sugar, and saturated fat [16]. This pattern, otherwise known as a Westernized dietary pattern (WDP), is very similar to current eating patterns in North America [17] and increasingly similar to patterns emerging in Asian-Pacific countries [18]. This may reflect commonality of influence between population groups due to globalization of the food supply [19]. Prospective studies have identified a WDP to be positively associated with MetS incidence, even after adjusting for adiposity among other confounders such as smoking status and PA [20]. Hence, the detrimental effect of the WDP goes beyond its effect on weight status.

3.2. MetS and the MDP

The MDP, as traditionally consumed in Southern Europe, has received significant attention regarding its apparent protective effect against MetS. This pattern is characterized by a diet rich in monounsaturated fatty acids (MUFA) from olives and olive oil; daily intake of whole grain cereals, fruits, vegetables, and dairy; and weekly intakes of fish, poultry, nuts, and legumes. Several studies have found the MDP to be associated with a lower MetS prevalence [21,22]. Moreover, a systematic review and meta-analysis of epidemiological studies and RCTs confirmed that adherence to a MDP was associated with reduced risk of MetS compared to control diets [23], as noted through beneficial effects on all individual criteria for the syndrome. Another meta-analysis also concluded that a MDP reduced hemoglobin A_{1c}, a long term indicator of high blood glucose levels [24].

It is possible that the MDP may exert its benefits on MetS through improvements in insulin sensitivity (IS) and/or inflammation. A well-designed prospective study found improvements in all components of MetS, except for BP, and improvements in IR as well [25]. Evidence that the MDP may act to reduce MetS via decreased inflammation [26] comes from an RCT of good quality [27] which found that, independent of weight loss, the MDP lowered C-reactive protein and interleukins 6, 7, and 8 (IL-6, IL-7, and IL-8) compared to a general healthy diet (50%-60% carbohydrate, 15-20% protein, <30% total fat). The authors indicated that fiber, omega-3 fatty acids, and antioxidants were potential mediators of the

Table 1 – Prospective studies examining the relationship of diet and components of MetS

Authors & year	Study details	Component of MetS					Comments
		↓WC	↓BP	↓Glucose	↑HDL	↓TG	
Lutsey, Steffen & Stevens 2008 [20]	Study: Prospective Subjects: America, 9514, 45-64 y, healthy Duration: 9 y Primary outcome: MetS prevalence	Dairy protective of MetS					No association with a healthy eating pattern Dairy protective against incident MetS Western diet promotes MetS
Tortosa et al. 2008 [22]	Study: Prospective Subjects: Spain, 5360, no MetS or risk factors Duration: 6 y Primary Outcome: MetS risk	Yes	No effect	No effect	Yes	No effect	Highest adherence to MDP had lowest cumulative incidence of MetS.
Rumawas et al. 2009 [25]	Study: Prospective Subjects: America, 1918, no risk marker Duration: 7 y Primary outcome: MetS components, incidence	Yes	No effect	Yes	Yes	Yes	MDP associated with lower HOMA-IR and lower incidence of MetS Very good control for confounders
Kesse-guyot et al. 2012 [30]	Study: Prospective Subjects: France, 3232, non diabetics Duration: 6 y Primary outcome: MetS components, MetS risk	Yes	Yes	-	Yes	Yes	Decreased risk with increased adherence to MetS diet by UMDS, MDS (trend for MSDPS p = 0.06) UMDS associated with reduced WC, BP, TG and higher HDL MDS associated with lower TG and WC MSDPS associated with higher HDL Good adjustment for confounders Adherence to diets low
Fumeron et al. 2011 [44]	Study: Prospective Subjects: France, 1710 M, 1725 F, non diabetics Duration: 9 y Primary outcome: MetS risk	Yes (Only women with high calcium intake)	Yes	-	Yes	Yes	Cheese, dairy other than cheese and dietary calcium associated with reduced risk of MetS High calcium and cheese intake associated with low TG Adjusted for confounders
Forouhi et al. 2008 [55]	Study: Prospective Subjects: England, 214 M, 310 F, 40-69 y, non diabetics Duration: 10 y Primary outcome: MetS risk	No effect	No effect	Yes	No effect	No effect	Decreased IR and MetS risk with higher vitamin D Adjusted for adiposity
Fung et al. 2012 [56]	Study: Prospective Subjects: America, 4727, 18-30 y, free of MetS Duration: 20 y Primary outcome: MetS incidence, MetS components	Yes	Trend p = 0.09	Yes	Yes	No effect	Higher vitamin D intake from diet and supplement associated with lower incidence by 18% Independent of dietary and supplemental calcium Did not adjust for adiposity

HOMA-IR, homeostasis model assessment-insulin resistance; M, male; F, female.
 ↑, increased; ↓, decreased, -, not measured.

Table 2 – Randomized controlled trials examining diet and MetS risk and MetS components

Authors & year	Study details	Quality score ^a	Component of MetS					Comments
			↓WC	↓BP	↓Glucose	↑HDL	↓TG	
Esposito et al. 2004 [27]	Study: Randomized, single-blind trial of Mediterranean or control diet (healthy diet) Subjects: Italy, 99 M, 81 F with MetS Duration: 2 y Primary outcome: MetS, CRP, IL 6-8	3	Yes	Yes	Yes	Yes	Yes	MDP decreased prevalence of MetS. All inflammatory markers decreased independent of weight loss.
Salas-salvado et al. 2008 [33]	Study: Participants randomly assigned to one of 3 diets: MDP and virgin olive oil (VOO), MDP and mixed nuts, or advice about a low fat diet (control) Subjects: Spain, 1264 M and F, M 55-80 y, F 60-80 y, with T2D and/or 3 or more CVD risk factors Duration: 4 y Primary outcome: Primary prevention of CVD		Yes MDP & VOO only	Yes MDP & VOO only	No effect	No effect	Yes MDP & VOO only	The one year prevalence of MetS was reduced by 6.7% in MDP and VOO, 13.7% in MDP and nuts, and 2% in control diet. After adjustment for weight change, and the MDP and nuts diet had a protective effect on MetS prevalence.
Azadbakht et al. 2005 [35]	Study: RCT, one of 3 diets; usual, weight reducing and DASH Subjects: Iran, 116, 34 M, 82 F, overweight or obese with MetS Duration: 6 months Primary outcome: MetS components	3	Yes	Yes	Yes	Yes	Yes	DASH diet decreased MetS, adjusted for weight loss. Weight reducing diet improved WC and TG levels.
Adamsson et al. 2011 [36]	Study: RCT; Nordic diet or control diet (Western diet) Subjects: Sweden, 88 mildly hypercholesterolaemic M and F, otherwise healthy, 25-65 y, Duration: 6 weeks Primary outcome: CVD risk factors	3	-	Yes	No effect	Yes	No effect	Nordic diet reduced plasma cholesterol, and insulin. Minimal but significant decrease in body weight.
Uusitupa et al. 2013 [38]	Study: RCT; Nordic diet or control diet (mean nutrient intake in Nordic countries) Subjects: Sweden, Denmark and Iceland, 166, mean age 55 y Duration: 18-24 weeks Primary outcome: MetS components	3	No effect	No effect	No effect	No effect	No effect	Significant changes between groups in non-HDL cholesterol, HDL to LD ratio, supporting the Nordic diet

Zemel et al. 2004 [43]	Study: Randomized placebo controlled; low calcium diet (500 caloric deficit diet with either 400-500 mg of dietary calcium/d + placebo), high calcium diet (400-500 mg of dietary calcium + 800 mg/d calcium) or high dairy diet (1200-1300 mg/d calcium + placebo) Subjects: America, 32 obese adults Duration: 24 weeks Primary outcome: Body weight and body fat	4	Yes	Yes	No effect	No effect	No effect	High dairy diet had greatest decrease in WC, followed by high calcium diet. Only dairy showed significant improvement in IS. Only high dairy improved systolic BP.
Paniagua et al. 2011 [63]	Study: Participants from eight European centers randomly assigned to one of 4 diets; HSFA, HMUFA, low fat high CHO + 1.24 g/d of LC n-3 PUFA (LFHCn-3), low fat high CHO + placebo (LFHC) Subjects: Europe, 337 with MetS, 35-70 y Duration: 3 months Primary outcome: MetS criteria, MetS prevalence	3	Trend P = 0.09	Yes LFHC, LFHCn-3, LFHCn-3 HSFA	No effect	No effect	Trend P = 0.1 LFHC LFHCn-3	Isoenergetic diets and good power. Decrease in prevalence (20.7%) of MetS following the LFHCn-3 diet unexplained by body weight, IR or change in PA Excellent compliance
Williams et al. 2011 [64]	Study: RCT; low fat high protein (HP) or low fat high fiber (HF) diet Subjects: England, 83 overweight and obese but otherwise healthy F, 18-65 y Duration: 8 weeks Primary outcome: MetS components	3	Yes	Yes in HP only	Yes	-	Yes	Energy intake significantly lower on HF than HP. Data was adjusted for change in weight but not body fat which was also available. Realistic diets Amounts of protein, fat and fiber not defined for HP and HF diets

↑, increased; ↓, decreased, -, not measured.

^a 0-2 = low quality, 3-5 = high quality from Jadad et al. (1996 21; M, male; F, female; y, years; CRP, C reactive protein; IL 6-8, interleukins 6-8; T2D, type 2 diabetes; HSFA, high fat high saturated fat; HMUFA, LC n-3 PUFA, long-chain omega-3 polyunsaturated fatty acids; LFHC, low fat high CHO + placebo.

effects. While critics of the MDP have been concerned with the relatively high percentage of fat in this dietary pattern, there is no evidence of significantly higher body weight or body fat following a MDP in a free-living situation [28]. It may also be argued that the MDP is not a single dietary pattern, since there are many diets in the Mediterranean region, each with its own emphasis on types of foods [29]. This apparent limitation was potentially overcome by assessing the risk of MetS via a number of MDP assessment tools.

A cohort study examined adherence to the MDP via three validated MDP scoring tools [30]. These tools included the traditional Mediterranean diet score (MDS), an updated Mediterranean dietary score (UMDS), and the Mediterranean style-dietary pattern score (MSDPS). The three tools differed in their assessment of grains as whole grain or refined, the inclusion of sweetened beverages as a negative component, and adjustment for energy intake. After adjustment for covariates including age, gender, education level, tobacco smoking status, and PA, the UMDS tool found the MDP to be inversely associated with waist circumference (WC), systolic BP, and TG and positively associated with increased HDL [30]. The MDS tool found the MDP was associated with lower WC and TG, and the MSDPS found this dietary pattern to be positively associated with HDL. Further adjustment for body mass index did not alter these findings, except that the relationship between the MDS and TG became non-significant [30]. Such data emphasizes that excess body weight/adiposity only partially drives the beneficial relationship between MetS and the MDP.

3.2.1. Nuts and olive oil as components of the MDP

A regular intake of nuts has beneficial outcomes on metabolic disturbances linked with MetS [31,32]. The PREDIMED study investigated the impact of two traditional MDPs (MDP 1 was enriched with virgin olive oil (VOO) and MDP 2 was enriched with nuts) compared to a low-fat control diet [33]. Specifically, the nut diet was enriched with walnuts (15 g per day), hazelnuts (7.5 g per day), and almonds (7.5 g per day), and the VOO diet consumed 15 L of VOO over three months. After one year, it was found that the group consuming the VOO diet had less abdominal obesity compared to the control diet, and the group consuming nuts had decreased TG, BP, and abdominal obesity compared to the control diet and the VOO diet. The prevalence of MetS was also reduced in subjects on both the diet enriched with nuts and VOO, although this was only significant in the diet enriched with nuts [33].

3.2.2. Low red meat intake as a component of the MDP

A further analysis of the PREDIMED data investigated the association between red meat and processed meat consumption and the development of MetS [34]. Evidence indicated that moderate to high consumption of red meat (average of 103 g per day) and high consumption of processed red meat (~50 g per day) increased the risk of developing MetS three-fold [34]. However, it was unclear from the study how red meat and processed meat were defined. Adjustment for saturated fat and other dietary variables did not remove this significant association [34]. Thus, the mechanism behind the association between red meat intake and MetS does not rest with its saturated fat composition.

3.3. The DASH diet and MetS

The DASH diet is characterized by a high intake of fruits, vegetables, whole grains, and dairy. A RCT was conducted in which a control diet, a weight reducing diet with energy deficit, and a DASH diet with the same energy deficit were compared. The control diet consisted of 50–60% carbohydrate, 15–20% protein, 30% total fat with a high saturated fat intake, 2–3 servings of fruits, 3 servings of vegetables, and 1 serving of dairy. The macronutrient composition of the weight reducing diet with an energy deficit was similar to the control diet, but with higher amounts of red meat, fat, and saturated fat than the DASH diet; and calcium intake, dairy, nut, and legume intakes were lower than the DASH diet. After adjustment for weight change, the DASH diet was found to improve all components of the MetS [35], with improvements in WC and TG beyond that of the weight reducing diet.

3.4. The ND and MetS

The ND diet is based on traditional foods consumed in the Nordic region (Northern Europe), and includes fruits, vegetables, legumes, low fat dairy, fatty fish (salmon, mackerel, herring) oats, barley, and almonds [36,37]. A recent RCT investigated the impact of the ND compared to a control diet (habitual Western diet lower in cholesterol, higher in fiber, and lower in saturated fat than the ND) on MetS parameters, including changes in cholesterol and IS [36]. Following 6 weeks on the ND, a significant decrease in daily energy consumption was seen, whereas no change occurred in the control diet. This was not a reporting error, as a significant decrease in body weight was also observed after consumption of the ND. After adjustment for weight loss, participants consuming a ND showed significant improvements in total cholesterol (-0.98 ± 0.75 mmol/l) and low-density lipoprotein cholesterol (LDL) (-0.83 ± 0.67 mmol/l); however, HDL (-0.08 ± 0.23 mmol/l) also decreased. The results are likely due to the significantly lower dietary cholesterol consumption, lower saturated fat, and higher dietary fiber intake in the ND. Although BP and IS improved, these changes became non-significant after adjustment for weight loss [36]. Other studies have similarly investigated the impact of the ND compared to a control diet [38]; cholesterol levels showed improvement and there was a significant difference in IL1Ra between the diet groups. The lack of significant findings in the later study may be due to the control diet also being a ND.

3.5. Fruits, vegetables, and whole grains as components of MDP, DASH, and ND

It is possible that the higher intake of fruits, vegetables, and whole grains may have mediated the positive effect of the three dietary patterns discussed above on MetS, and there was a significant difference in IL1Ra between the diet groups. Fruits and vegetables have been found to lower the risk of MetS and inflammation [21,39], as well as decrease BP [21]. Significant inverse associations between whole grain intake and MetS have been shown, independent of confounders including other dietary factors. A higher whole grain intake is associated with lower fasting blood glucose and body mass

index [40]. More quality studies focusing on this is important since current dietary guidelines around the world recommend a high intake of these foods. The low glycemic index (GI) of fruits, vegetables, and whole grains may be the mechanism for the observed benefits of the MDP, DASH diet, and ND on MetS characteristics. However, few studies have examined the association between GI or glycemic load (GL) of diet and MetS [21]. As rice and refined wheat products which characterize Asian diets are high GI/GL [41], and the average GI/GL value of diets in the United States of America have increased [42], this is an important area for future research.

3.6. Dairy as a component of MDP, DASH, and ND

Dairy has been described as protective against the development of MetS. Studies have shown a reduced visceral adipose tissue mass following dairy consumption [43]. Furthermore, RCTs of high quality show the benefit of dairy on at least two MetS components [43], as do several prospective studies (Table 2) [20,44]. However, a recent systematic review indicated that the evidence is supportive, but not yet conclusive of a beneficial effect on MetS [45]. The conflicting results may be, in part, due to the study designs available (cross sectional/observational), the inability to control for all potential confounders, and the many systems of classification of MetS used [45].

There are many components in dairy that could have a beneficial effect on risk markers that comprise the MetS, such as whey protein. Milk proteins are classified as casein (80%) and whey (20%). Whey leads to increased energy expenditure and reduces energy intake by its contribution to short and long term feelings of satiety [46,47]. Whey also provides branched chain amino acids that may have a positive effect on muscle mass and bone, due to an insulin-like growth factor 1 [48]. Such changes favor a negative energy balance through increased energy expenditure of muscle mass and would account for the greater fat loss but preservation of muscle mass that is seen in some studies following whey intake [49]. Consumption of whey protein has also been shown to reduce fasting lipids and could improve IS [50].

Calcium and vitamin D are two other components of dairy which may account for the beneficial effects of dairy on the MetS. An RCT indicated that there was an increase in fat loss in the abdominal region, with an increased intake of calcium through consumption of yogurt [51]. Calcium promotes fat oxidation and increases fecal fat excretion [52,53], which represent potential mechanisms for the reduction in body fat [54], WC [51], and the lowering of circulating TG [44] that is seen with additional calcium intake. Multiple studies have found a positive association between low vitamin D status and MetS risk [55,56]; however, results do not always take adiposity changes into account [22,56] (Table 1). It has been suggested that vitamin D exerts its positive influence on MetS via IS effects [55,57,58]. Furthermore, in a blinded randomized cross-over trial, dairy consumption was shown to increase circulating adiponectin levels [59], and adiponectin is well known to have IS effects [7] and promote improvements in all risk markers of MetS [60]. As both adiponectin [61,62] and vitamin D decrease [62] with increasing adiposity, these two variables may play a key role in the pathogenesis of the syndrome. Intervention studies to examine the effect of vitamin D on adiponectin levels, IR, adiposity, and MetS are now needed.

3.7. Carbohydrate, protein, and fat intake and MetS

Comparisons of dietary patterns low in fat (28% of energy) [63,64], high in carbohydrate (CHO) (50%) [63], and high in protein (amount not specified) [64] show benefits on MetS components (Table 2). However, not all studies define the amounts that constitute high fat, high carbohydrate, and high protein. In an 8-week RCT [64], both a low-fat, high-protein diet (HP) (frequent intakes of non-starchy vegetables and red meat, eggs, and cheese) and a low-fat, high-fiber diet (HF) (high intakes of fruit, salad, and root vegetables; low intakes of fried foods and processed meats), improved WC, glucose, and TG levels, as well as total cholesterol and LDL. However, the HP diet had further beneficial effects on BP and a greater body fat loss, despite greater energy intakes.

The effects of four iso-energetic dietary treatments, which differed in quantity and quality of fat, were studied for 12 weeks in subjects with MetS [63]. After 12 weeks of dietary intervention, the diet containing the low-fat high carbohydrate (LFHCn-3) with 1.24 g per day of omega-3 fatty acid supplementation (Marinol C-38) with an eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) ratio of 1.4:1 [65] produced the greatest benefits, with improvements in BP, abdominal obesity, TG, and prevalence of MetS [63]. The decrease in prevalence (20.7%) of MetS following the LFHCn-3 diet was unexplained by body weight, IR, or change in PA. Although this study provided evidence for the protective effect of a low-fat, high carbohydrate diet supplemented with omega-3 fatty acids on MetS, it is limited by its short duration. Cross-over designs of a long-term duration are now needed.

The association between fatty acids and MetS is now recognized [6] and has recently been reviewed [21]. Saturated fatty acids and trans fatty acids were found to be adversely associated with MetS components. Intake of unsaturated fatty acids, which includes MUFA, omega-3 polyunsaturated fatty acids (PUFA), EPA, DHA, and alpha-linolenic acid (ALA) and omega-6 polyunsaturated fatty acids, have been found to reduce the risk factors associated with MetS [66,67]. Specifically, MUFA were shown to be associated with decreases in BP and TG, increases in HDL, and improved glycemic control [21,68]. It was previously suggested that the protective associations of MUFA with MetS may be due to increased beta cell secretion, thereby improving glucose stimulated insulin secretion [21]. It is important to acknowledge that fatty acids are not consumed in isolation, and a greater proportion of saturated fatty acids are found in animal food products than in plant products. For example, although many Western countries have a high dietary intake of MUFA, this is often associated with a high saturated fat intake through higher consumption of animal products, including dairy products and meat products [69].

Omeegas-3 PUFAs, EPA, and DHA, are associated with improved blood lipids. A review article concluded that EPA and DHA reduced TG and BP [21]. A double blind RCT study showed TG levels improved with DHA supplementation (4-6 g per day), although LDL cholesterol worsened (on the 4 g per day dose only) [70]. While there is other support for DHA supplementation on lowering plasma TG [71] and increasing LDL cholesterol levels [71–73], one other study found no change in LDL cholesterol levels [74]. A recent review identified the positive effects of EPA and DHA was likely to be due to inhibiting very-low-density lipoprotein secretion and enhancing clearance of TG from plasma. However, these authors also indicated a need

for studies to use purified EPA and DHA in order to isolate their effects [75].

Omega-3 fatty acids, EPA, DHA, and ALA are associated with improved inflammatory profiles, resulting in less potent inflammatory mediators compared to omega-6 fatty acids [76]. However, it is not as simple as reducing intakes of omega-6 fatty acids, as studies show beneficial effects of omega-6 fatty acid intakes on MetS components and risk [67]. While it is recognized that EPA, DHA, and ALA have different metabolic effects, attempts to differentiate the separate effects of ALA, EPA, and DHA are often confounded by a diet rich in omega-3 fatty acid sources [76]. It is possible that adiponectin mediates the well-documented relationship between omega-3 fatty acids and inflammation. Adiponectin levels have been found to increase following the consumption of walnuts, a natural source of omega-3 fatty acids [77]. Future studies should investigate whether adiponectin is a mediating factor in the relationship between omega-3 fatty acids and inflammation.

4. Future research

Despite a comprehensive search strategy, a limitation of our narrative review is the possibility of missing some of the evidence in this area. However, based on our criteria, we did not uncover any compelling data from the Asia-Pacific region that emphasized their traditional cuisine and putative protection against MetS. Our analysis also suggests that additional prospective and high quality RCT studies that investigate MDP, the DASH diet, and the ND would cement their benefits. In particular, both the MDP and ND work really well in their regions of origin, but there is a lack of research examining their effect in other parts of the world. One potential research direction may be to conduct multicenter studies that represent different ethnic groups of interest, and thereby extend the applicability of such dietary patterns in the battle against MetS.

5. Conclusion

This review highlights those dietary patterns that are associated with the lowering of MetS characteristics, along with potential dietary components responsible for such outcomes. Viewed collectively, these results are in line with the current dietary patterns that are recommended through the Dietary Guidelines of many countries. From the current evidence based on prospective studies and RCTs, we conclude that the MDP, DASH, and ND all have benefits for MetS. This would stem in part from their emphasis on fruits, vegetables, whole grains, fish, nuts, and dairy. To extend their benefits, future studies could examine the applicability of these diets for the prevention of MetS outside of the regions where they are traditionally consumed.

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REFERENCES

- [1] Corona G, Monami M, Rastrellia G, Aversa A, Tishova Y, Saad F, et al. Testosterone and metabolic syndrome: a meta-analysis study. *J Sex Med* 2011;8:272–83.
- [2] Cameron AJ, Magliano DJ, Zimmet PZ, Welborn T, Shaw JE. The metabolic syndrome in Australia: prevalence using four definitions. *Diabetes Res Clin Pract* 2007;77:471–8.
- [3] Ford ES. Risks for all-cause mortality, cardiovascular disease, and diabetes associated with the metabolic syndrome: a summary of the evidence. *Diabetes Care* 2005;28:1769–78.
- [4] Nestel P, Lyu R, Low LP, Sheu WH, Nitiyanant W, Saito I, et al. Metabolic syndrome: recent prevalence in East and Southeast Asian populations. *Asia Pac J Clin Nutr* 2007;16:362–7.
- [5] Lorenzo C, Williams K, Hunt K, Haffner SM. Trend in the prevalence of the metabolic syndrome and its impact on cardiovascular disease incidence: The San Antonio Heart Study. *Diabetes Care* 2006;29:625–30.
- [6] Shab-Bidar S, Hosseini-Esfahani F, Mirmiran P, Hosseinpour-Niazi S, Azizi F. Metabolic syndrome profiles, obesity measures and intake of dietary fatty acids in adults: Tehran Lipid and Glucose Study. *J Hum Nutr Diet* 2014;Suppl 2:98–108.
- [7] Ryo M, Nakamura T, Kihara S, Kumada M, Shibazaki S, Takahashi M, et al. Adiponectin as a biomarker of the metabolic syndrome. *Circ J* 2004;68:975–81.
- [8] Whitehead JP, Richards AA, Hickman IJ, Macdonald GA, Prins JB. Adiponectin—a key adipokine in the metabolic syndrome. *Diabetes Obes Metab* 2006;8:264–80.
- [9] Koh KK, Han SH, Quon MJ, Yeal AJ, Shin EK. Beneficial effects of fenofibrate to improve endothelial dysfunction and raise adiponectin levels in patients with primary hypertriglyceridemia. *Diabetes Care* 2005;28:1419–24.
- [10] Furuhashi M, Ura N, Higashiura K, Murakami H, Tanaka M, Moniwa N, et al. Blockade of the renin-angiotensin system increases adiponectin concentrations in patients with essential hypertension. *Hypertension* 2003;42:76–81.
- [11] Forsythe LK, Wallace JM, Livingstone MB. Obesity and inflammation: the effects of weight loss. *Nutr Res Rev* 2008;21:117–33.
- [12] Calton EK, Miller VS, Soares MJ. Factors determining the risk of the metabolic syndrome: is there a central role for adiponectin? *Eur J Clin Nutr* 2013;6:485–91.
- [13] Jadad A, Moore R, Carroll D, Jenkinson C, Reynolds DJ, Gavaghan DJ, et al. Assessing the quality of reports of randomized clinical trials: is blinding necessary? *Control Clin Trials* 1996;17:1–12.
- [14] Wang X, Li Z, Lui Y, Lv X, Yang W. Effects of pistachios on body weight in Chinese subjects with metabolic syndrome. *Nutr J* 2012;11:20.
- [15] National Health and Medical Research Council & Department of Health and Ageing Australian dietary guidelines. Canberra: Australian Government; 2013.
- [16] Australian Bureau of Statistics. 4804.0 National Nutrition Survey. Canberra: ABS; 1999.
- [17] Chiuve SE, Willett WC. The 2005 Food Guide Pyramid: an opportunity lost? *Nat Clin Pract Cardiovasc Med* 2007;4:610–20.
- [18] Lim S, Shin H, Song JH, Kang SM, Won Yoon J, Choi SH. Increasing prevalence of metabolic syndrome in Korea: The Korean National Health and Nutrition Examination Survey for 1998–2007. *Diabetes Care* 2011;34:1323–8.
- [19] Food and Agricultural Organization of the United Nations. The state of food and agriculture in Asia and the Pacific region. Bangkok: FAO; 2008.
- [20] Lutsey PL, Steffen LM, Stevens J. Dietary intake and the development of the metabolic syndrome. *Circulation* 2008;117:754–61.

- [21] Djousse L, Padilla H, Nelson TL, Gaziano JM, Mukamal KJ. Diet and metabolic syndrome. *Endocr Metab Immune Disord Drug Targets* 2010;10:124–37.
- [22] Tortosa A, Bes-Rastrollo M, Sanchez-Villegas A, Basterra-Gortari FJ, Nuñez-Cordoba JM, Martinez-Gonzalez MA. Mediterranean diet inversely associated with the incidence of metabolic syndrome: the SUN prospective cohort. *Diabetes Care* 2007;30:2957–9.
- [23] 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;57:1299–313.
- [24] Carter P, Achana F, Troughton J, Gray LJ, Khunti K, Davies MJ. A Mediterranean diet improves HbA_{1c} but not fasting blood glucose compared to alternative dietary strategies: a network meta-analysis. *J Hum Nutr Diet* 2014;27:280–97.
- [25] Rumawas ME, Meigs JB, Dwyer JT, McKeown NM, Jacques PF. Mediterranean-style dietary pattern, reduced risk of metabolic syndrome traits, and incidence in the Framingham Offspring Cohort. *Am J Clin Nutr* 2009;90:1608–14.
- [26] Babio N, Bulló M, Salas-Salvadó J. Mediterranean diet and metabolic syndrome: the evidence. *Public Health Nutr* 2009;12:1607–17.
- [27] Esposito K, Marfella R, Ciotola M, Di Palo C, Giugliano F, Giugliano G, et al. Effect of a Mediterranean-style diet on endothelial dysfunction and markers of vascular inflammation in the metabolic syndrome: a randomized trial. *JAMA* 2004;292:1440–6.
- [28] Soares MJ. The effect of olive oil on postprandial thermogenesis, fat oxidation and satiety: potential implications for weight control. In: Preedy VR, Watson RR, editors. *Olive oil in health and disease prevention*. Oxford: Academic Press; 2010. p. 863–70.
- [29] Noah A, Truswell AS. There are many Mediterranean diets. *Asia Pac J Clin Nutr* 2001;10(1):2–9.
- [30] Kesse-Guyot E, Ahluwalia N, Lassale C, Hercberg S, Fezeu L, Lairon D. Adherence to Mediterranean diet reduces the risk of metabolic syndrome: a 6-year prospective study. *Nutr Metab Cardiovasc Dis* 2012;23:677–83.
- [31] Jiang R, Manson JE, Stampfer MJ, Liu S, Willett WC, Hu FB. Susceptibility of LDL to oxidative modification in healthy volunteers supplemented with low doses of n-3 polyunsaturated fatty acids. *JAMA* 2002;288(20):2554–60.
- [32] Rajaram S, Sabate J. Nuts, body weight and insulin resistance. *Br J Nutr* 2006;96(2):S79–86.
- [33] Salas-Salvado J, Fernandez-Ballart J, Ros E, Martinez-Gonzalez M-A, Fito M, Estruch E, et al. Effect of a Mediterranean diet supplemented with nuts on metabolic syndrome status: one-year results of the PREDIMED randomized trial. *Arch Intern Med* 2008;168(22):2449–58.
- [34] Babio N, Sorli M, Bullo M, Basora J, Ibarra-Jurado N, Fernandez-Ballart J, et al. Association between red meat consumption and metabolic syndrome in a Mediterranean population at high cardiovascular risk: cross-sectional and 1-year follow-up assessment. *Nutr Metab Cardiovasc Dis* 2012;22:200–7.
- [35] Azadbakht L, Mirmiran P, Esmailzadeh A, Azizi T, Azizi F. Beneficial effects of a dietary approaches to stop hypertension eating plan on features of the metabolic syndrome. *Diabetes Care* 2005;28:2823–31.
- [36] Adamsson V, Reumark A, Fredriksson I-B, Hammarstrom E, Vessby B, Johansson G, et al. Effects of a healthy Nordic diet on cardiovascular risk factors in hypercholesterolaemic subjects: a randomized controlled trial (NORDIET). *J Intern Med* 2011;269:150–9.
- [37] Adamsson V, Reumark A, Cederholm T, Vessby B, Riserus U, Johansson G. What is a healthy Nordic diet? Foods and nutrients in the NORDIET study. *Food Nutr Res* 2012;56:181–9.
- [38] Uusitupa M, Hermansen K, Savolainen MJ, Schwab U, Kolehmainen M, Brader L, et al. Effects of an isocaloric healthy Nordic diet on insulin sensitivity, lipid profile and inflammation markers in metabolic syndrome—a randomized study (SYSDIET). *J Intern Med* 2013;274:52–66.
- [39] Esmailzadeh A, Kimiagar M, Mehrabi Y, Azadbakht L, Hu FB, Willett WC. Fruit and vegetable intakes, C-reactive protein, and the metabolic syndrome. *Am J Clin Nutr* 2006;84(6):1489–97.
- [40] Sahyoun NR, Jacques PF, Zhang XL, Juan W, McKeown NM. Whole-grain intake is inversely associated with the metabolic syndrome and mortality in older adults. *Am J Clin Nutr* 2006;83(1):124–31.
- [41] Aronis KN, Vamvini MT, Chamberland JP, Sweeney LL, Brennan AM, Magkos F, et al. Short-term walnut consumption increases circulating total adiponectin and apolipoprotein A concentrations, but does not affect markers of inflammation or vascular injury in obese humans with the metabolic syndrome: data from a double-blinded, randomized, placebo-controlled study. *Metabolism* 2012;61:577–82.
- [42] Ludwig DS. The glycemic index: physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. *JAMA* 2002;287:2414–23.
- [43] Zemel M, Thompson W, Milstead A, Morris K, Campbell P. Calcium and dairy acceleration of weight and fat loss during energy restriction in obese adults. *Obes Res* 2004;12:582–90.
- [44] Fumeron F, Lamri A, Abi Khalil C, Jaziri R, Porchay-Baldérelli I, Lantieri O, et al. Dairy consumption and the incidence of hyperglycemia and the metabolic syndrome: results from a French prospective study, Data from the Epidemiological Study on the Insulin Resistance Syndrome [DESIR]. *Diabetes Care* 2011;34:813–7.
- [45] Crichton GE, Bryan J, Buckley J, Murphy KG. Dairy consumption and metabolic syndrome: a systematic review of findings and methodological issues. *Obes Rev* 2011;12:e190–201.
- [46] Veldhorst M, Nieuwenhuizen AG, Hochstenbach-Waelen A, van Vught AJ, Westerterp KR, Engelen MP, et al. Dose-dependent satiating effect of whey relative to casein or soy. *Physiol Behav* 2009;96:675–82.
- [47] Solah VA, Kerr DA, Adikara CD, Meng X, Binns CW, Zhu K, et al. Differences in satiety effects of alginate- and whey protein-based foods. *Appetite* 2010;54:485–91.
- [48] Zhu K, Meng X, Kerr D, Devine A, Solah V, Binns CW, et al. The effects of a two-year randomized, controlled trial of whey protein supplementation on bone structure, IGF-1, and urinary calcium excretion in older postmenopausal women. *J Bone Miner Res* 2011;26:2298–306.
- [49] Frestedt JL, Zenk JL, Kuskowski M, Ward LS, Bastian ED. A whey-protein supplement increases fat loss and spares lean muscle in obese subjects: a randomized human clinical study. *Nutr Metab (Lond)* 2008;5:8.
- [50] Pal S, Ellis V, Dhaliwal S. Effects of whey protein isolate on body composition, lipids, insulin and glucose in overweight and obese individuals. *Br J Nutr* 2010;104:716–23.
- [51] Zemel M, Teegarden D, Van Loan M, Schoeller A, Matkovic V, Lyle RM, et al. Dairy-rich diets augment fat loss on an energy-restricted diet: a multicenter trial. *Nutrients* 2009;1:83–100.
- [52] Dougkas A, Reynolds CK, Givens ID, Elwood PC, Minihane AM. Associations between dairy consumption and body weight: a review of the evidence and underlying mechanisms. *Nutr Res Rev* 2011;15:1–24.
- [53] Soares MJ, Muhandi L, Kurpad AV, Chan She Ping-Delfos WL, Piers LS. Mechanistic roles for calcium and vitamin D in the regulation of body weight. *Obes Rev* 2012;13:592–605.
- [54] Onakpoya IJ, Perry R, Zhang J, Ernst E. Efficacy of calcium supplementation for management of overweight and obesity: systematic review of randomized clinical trials. *Nutr Rev* 2011;69:335–43.
- [55] Frouhi NG, Luan J, Cooper A, Boucher BJ, Wareham NJ. Baseline serum 25-hydroxy vitamin d is predictive of future

- glycemic status and insulin resistance: The Medical Research Council Ely Prospective Study 1990–2000. *Diabetes* 2008;57:2619–25.
- [56] Fung GJ, Steffen LM, Zhou X, Harnack L, Tang W, Lutsey PL, et al. Vitamin D intake is inversely related to risk of developing metabolic syndrome in African American and white men and women over 20y: the Coronary Artery Risk Development in Young Adults study. *Am J Clin Nutr* 2012;96:24–9.
- [57] Boucher BJ. Is vitamin D status relevant to metabolic syndrome? *Dermatoendocrinology* 2012;4:212–24.
- [58] Soares MJ, Chan She Ping-Delfos W, Sherriff JL, Nezhad DH, Cummings NK, Zhao Y. Vitamin D and parathyroid hormone in insulin resistance of abdominal obesity: cause or effect. *Eur J Clin Nutr* 2011;65:1348–52.
- [59] Zemel MB, Sun X, Sobhani T, Wilson B. Effects of dairy compared with soy on oxidative and inflammatory stress in overweight and obese subjects. *Am J Clin Nutr* 2010;91:16–22.
- [60] Lara-Castro C, Fu Y, Chung BH, Garvey WT. Adiponectin and the metabolic syndrome: mechanisms mediating risk for metabolic and cardiovascular disease. *Curr Opin Lipidol* 2007;18:263–70.
- [61] Ruige JB, Ballaux DB, Funahashi T, Mertens IL, Matsuzawa Y, Van Gaal LF. Resting metabolic rate is an important predictor of serum adiponectin concentrations: potential implications for obesity-related disorders. *Am J Clin Nutr* 2005;82:21–5.
- [62] Vaidya A, Forman JP, Underwood PC, Hopkins PN, Williams GH, Pojoga LH, et al. The influence of body mass index and renin-angiotensin-aldosterone system activity on the relationship between 25-hydroxyvitamin D and adiponectin in Caucasian men. *Eur J Endocrinol* 2011;164:995–1002.
- [63] Paniagua JA, Perez-Martinez P, Gjelstad IM, Tierney AC, Delgado-Lista J, Defoort C, et al. A low-fat high-carbohydrate diet supplemented with long-chain n-3 PUFA reduces the risk of the metabolic syndrome. *Atherosclerosis* 2011;218:443–50.
- [64] Te Morenga LA, Levers MT, Williams SM, Brown RC, Mann J. Comparison of high protein and higher fiber weight-loss diets in women with risk factors for the metabolic syndrome: a randomized trial. *Nutr J* 2011;10:40. <http://dx.doi.org/10.1186/1475-2891-10-40>.
- [65] Shaw DI, Tierney AC, McCarthy S, Upritchard J, Vermunt S, Gulseth HL, et al. LIPGENE food-exchange model for alteration of dietary fat quantity and quality in free-living participants from eight European countries. *Br J Nutr* 2008;101:750–9.
- [66] Baik I, Abbott RD, Curb D, Shin C. Intake of fish and n-3 fatty acids and future risk of metabolic syndrome. *J Am Diet Assoc* 2010;110:1018–26.
- [67] Vanhala M, Saltevo J, Soininen P, Kautiainen H, Kangas AJ, Ala-Korpela M, et al. Serum omega-6 polyunsaturated fatty acids and the metabolic syndrome: a longitudinal population-based cohort study. *Am J Epidemiol* 2012;176(3):253–60.
- [68] Gillingham LG, Harris-Janz S, Jones PJH. Dietary monounsaturated fatty acids are protective against metabolic syndrome and cardiovascular disease risk factors. *Lipids* 2011;46:209–28.
- [69] Sundstrom J, Lind L, Vessby B, Andren B, Aro A, Lithell HO. Dyslipidemia and an unfavorable fatty acid profile predict left ventricular hypertrophy 20 years later. *Circulation* 2001;103:836–41.
- [70] Milte CM, Coates AM, Buckley JD, Hill AM, Howe PRC. Dose-dependent effects of docosahexaenoic acid-rich fish oil on erythrocyte docosahexaenoic acid and blood lipid levels. *Br J Nutr* 2008;99:1083–8.
- [71] Mori TA, Watts GF, Burke V, Hilme E, Puddey IB, Lawrence J. Differential effects of eicosapentaenoic acid and docosahexaenoic acid on vascular reactivity of the forearm microcirculation in hyperlipidemic, overweight men. *J Am Heart Assoc* 2000;102:1264–9.
- [72] Sanders TAB, Gleason K, Griffin B, Miller GJ. Influence of an algal triacylglycerol containing docosahexaenoic acid (22:6n-3) and docosapentaenoic acid (22:5n-6) on cardiovascular risk factors in healthy men and women. *Br J Nutr* 2006;95:525–31.
- [73] Theobald HE, Chowienczyk PJ, Whittall R, Humphries SE, Sanders TAB. LDL cholesterol-raising effect of low-dose docosahexaenoic acid in middle-aged men and women. *Am J Clin Nutr* 2004;79:558–63.
- [74] Higgins S, Carroll YL, McCarthy SN, Corridan BM, Roche HM, Wallace JMW, et al. Susceptibility of LDL to oxidative modification in healthy volunteers supplemented with low doses of n-3 polyunsaturated fatty acids. *Br J Nutr* 2001;85:23–31.
- [75] Poudyal H, Panchal SK, Diwan V, Brown L. Omega-3 fatty acids and metabolic syndrome: effects and emerging mechanisms of action. *Prog Lipid Res* 2011;50:372–87.
- [76] Anderson BM, Ma DWL. Are all n-3 polyunsaturated fatty acids created equal? *Lipids Health Dis* 2009;8:33–53.
- [77] Cornier MA, Dabelea D, Hernandez TL, Lindstrom RC, Steig AJ, Stob NR, et al. The metabolic syndrome. *Endocr Rev* 2008;29:777–822.