

Mediterranean meal favorably effects postprandial oxidative stress response compared with a western meal in healthy women

A randomized crossover trial

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Abstract: Oxidative stress and inflammation are underlying factors in the pathogenesis of chronic diseases. The postprandial state is characterized by low-grade oxidative and inflammatory responses, but the impact of different dietary patterns on these responses is unclear. The objective of this study was to investigate postprandial oxidative and inflammatory responses to Mediterranean diet (MED) and Western diet (WD) meals. In a randomised crossover design, eleven healthy women, aged between 19–45 years with a body mass index of 20.0–24.9 kg/m², consumed two different isocaloric meals: MED and WD. Blood samples were collected at fasting and 2, 3, 4 h postprandially and analyzed for oxidative [total antioxidant status (TAS), total oxidant status (TOS), total thiol, native thiol, malondialdehyde (MDA)] and inflammatory [high sensitivity C-reactive protein (hs-CRP), interleukin (IL)-6, IL-17, IL-23, tumor necrosis factor alpha (TNF- α) and nuclear factor kappa B (NF- κ B)] markers. MED meal intake resulted in increases in TAS (0.05 ± 0.02 mmol/L; $p=0.017$), total thiol (23.00 ± 7.69 μ mol/L; $p=0.013$) and native thiol (12.82 ± 4.94 μ mol/L; $p=0.027$), while a decrease in MDA (-0.17 ± 0.06 nmol/L; $p=0.022$) at 2 h. On the other hand, TAS reduced significantly overall ($p=0.005$) after WD meal intake. There was a significant increase after WD meal intake for IL-6 (1.39 ± 0.49 pg/mL; $p=0.017$), IL-17 (4.30 ± 1.50 pg/mL; $p=0.017$), IL-23 (8.38 ± 3.51 pg/mL; $p=0.038$) at 4 h. However, serum hs-CRP, TNF- α and NF- κ B levels were not changed significantly by meal intake. The results indicate that MED meal induces favorable effects on oxidative stress, while WD meal partially increases inflammation in daily life.

Keywords: Postprandial state, Mediterranean diet, Western diet, postmeal, inflammation, oxidative stress

Introduction

Hippocrates (400 BC), centuries ago, for the first time put forward the view “Let food be thy medicine and medicine be thy food” [1]. Since then, the relationship between diet and chronic diseases has been studied [2]. While the studies have long focused on the role of a single nutrient or food intake, evaluating the effects of dietary patterns approach have an increasing interest recently [3, 4]. “Health promoting” dietary patterns, including Mediterranean diet (MED), can significantly reduce the risk of general mortality, cardiovascular diseases and other metabolic diseases [5, 6, 7]. MED is characterized by high in olive oil (extra virgin), vegetables, fruits, whole grains, legumes and seeds; moderate in fish, red meat, dairy products and red wine, low in eggs and sugar consumption. The dietary pattern is low in saturated fat and animal protein and high in monounsaturated fatty acids (MUFA), fiber, also rich in antioxidant

compounds, bioactive elements and vitamins and has a low glycemic index [8]. Compliance with the MED in the preservation of health is among the nutrition policies of many countries, but globalization in the world affects dietary habits [9, 10]. “Westernization” is observed in the dietary habits of societies due to widespread western type of economy, the globalization of food production and consumption as well as the urban and technology-oriented culture and the homogenization of dietary habits [11]. Characteristics of the Western diet (WD) is high in meat, processed meat products, dairy products, pastry, sugar, processed foods, soft drinks and alcoholic beverages, and low in vegetables and fruits [1]. Accordingly, this diet type contains high energy, carbohydrates, saturated fat, trans fat, polyunsaturated fat (PUFA) (especially n-6 fatty acids) and sodium, insufficient vitamins, minerals and fiber [12]. The rapidly increasing incidence of chronic diseases such as obesity, cardiovascular diseases, metabolic diseases

(metabolic syndrome, type II diabetes, non-alcoholic fatty liver) and some types of cancer are strongly associated with the WD pattern [13].

Oxidative stress and inflammation, directly related to nutrient, food and dietary patterns, are important underlying factors in the development of chronic diseases [14, 15]. Oxidative and inflammatory responses may occur in the long term or may begin immediately after a meal consumption. These responses that starts within minutes with the meal consumption can last for hours depending on the components of the meal [16, 17, 18]. Therefore, it is suggested that postprandial status studies can be considered as a starting point for assessing the long-term effects of dietary patterns [19]. Nowadays, especially in modern societies, the majority of the day is spent in the postprandial state [16, 20]. Accordingly, exposure to prolonged postprandial oxidative and inflammatory responses may be a triggering factor for chronic diseases in long term [21, 22, 23].

Earlier postprandial studies have mostly examined a single nutrient or food intake or “unrealistic” meal models [24, 25, 26]. These are incomplete approaches in which the importance of synergistic interactions between nutrient and non-nutrient compounds of a diet is overlooked. Therefore, in this study, it was aimed to compare the effects of MED and WD meals on postprandial oxidative and inflammatory response using the “real-life” meal model approach in healthy individuals. It was hypothesized that several oxidative and inflammatory markers would induce an increase post-WD meal, while not expecting all markers to result in an increase. According to best of our knowledge, this is the first study to compare postprandial effects of MED and WD meals in healthy adults.

Method

Participants

Eleven healthy women who met the following inclusion criteria: aged 19–45 years, nonsmokers, body mass index (BMI) of 20.0–24.9 kg/m², no use of medications and vitamin, mineral, antioxidant or probiotics supplements last three months, non-menopausal and with normal dietary habits (no special diets, meals etc.), were included in the present study. The exclusion criteria included present or past medical history of inflammatory diseases, diabetes, cardiovascular disease, cancer or other chronic diseases, and also food allergies, pregnancy or lactation, being physically active as defined by three days or more of at least 30 minutes of moderate to vigorous activity weekly, participation in a weight loss program and substance or alcohol abuse. The participants with normal serum glucose, total cholesterol, high-density lipoprotein (HDL) cholesterol,

low-density lipoprotein (LDL) cholesterol, triglycerides and normal thyroid functions were accepted for the study.

The participants were informed about the protocol in detail and provided with written informed consent form at the beginning of the study. The study was approved by the Clinical Research Ethics Committee of Ankara University, Turkey (Ref. No: 24074710-06) and conducted in accordance with the ethical guidelines of the Declaration of Helsinki.

Study design

This study was a randomized crossover trial and was conducted at the Department of Nutrition and Dietetics, Yüksek İhtisas University, Ankara, Turkey. The participants reported to the department on 3 separate sessions. The first session consisted of an initial assessment, participants' body weight and height were measured using the combined digital scale and an integrated stadiometer (SECA 704s) and body composition was measured via bioelectrical impedance analyses (Tanita DC 360) in a fasted state in light clothing. Participants completed a questionnaire which included inquiries about medical history, physical activity and dietary habits. The participants were instructed not to take antioxidant, vitamin-mineral, omega-3 supplements, to refrain from vigorous physical activity and to maintain their usual diets, lifestyle and physical activities during the study period. They were also asked to avoid consuming foods high in antioxidants, including berries, tea (black, green, white and herbal), coffee, chocolate, red wine etc., for 3 days prior to each study session. In addition, all participants were instructed to consume the same meal on the previous evening and to record their dietary intake for 3-days prior to each study session. Also they were asked not to take any medications and to abstain from alcohol 24 hours prior to the study sessions. The compliance was assessed from the dietary record.

In the following two study sessions, the participants were required to consume 1 of 2 test meals that are MED and WD in randomized order. The participants were grouped into two different test meals by block randomization procedure. A washout period of 2 weeks between the test meal sessions was determined to eliminate the possibility of a carry-over effect.

On each meal session day, the participants reported to the department at approximately 8:00 AM after 12-hour overnight fasting. The participants were allowed to consume only water *ad libitum* and maintained their resting state in sitting or lying down throughout the 4-hour testing period. The same protocol was followed for both sessions. Baseline blood samples were collected and then the participants consumed the test meal within 15–20 minutes. Venous blood samples were collected again at 2, 3 and 4 h after consuming

the meal (after the last bite). All the blood samples were collected from the forearm vein via blood collection set (BD Vacutainer® Safety-Lok™) and collected in serum tubes which are coated with microscopic silica particles to activate the coagulation process (Greiner Bio-One Z Serum Sep Clot Activator Tubes Vacuette®). All the samples were centrifuged (2500 g, 10 minutes, 4 °C) immediately. For each sample, the serum was pipetted into eppendorf tubes and stored –80 °C until analysis. All the sample analysis was conducted in a blinded manner.

Meal composition

Two different isocaloric test meals with different dietary patterns were used in the present study: MED and WD meals. The MED meal was comprised of whole-grain bread, legumes (chickpea), salmon, extra virgin olive oil, nuts, red grapes, vegetables, balsamic vinegar and a Turkish traditional fermented grape-based drink “hardaliye”. The WD meal consisted of beef hamburger (meatball, ketchup, mayonnaise, bread), french fries (with sunflower oil) and soft drink with added sugar. The energy and nutrient composition of the meals are presented in Table 1. The meals were calculated using the computer-based nutrient analysis program Ebispro, Turkish Version (BeBiS 8.2) based on the Bundeslebensmittelschlüssel; German Food Code and Nutrient Data Base; Version 3.01B (Stuttgart, Germany).

Biochemical analysis

The serum concentrations of interleukin (IL)-6, IL-17, IL-23 and tumor necrosis factor alpha (TNF-α) and nuclear factor kappa B-p105 (NF-κB) were determined using commercially available enzyme-linked immunosorbent assay (ELISA) kit (Elabscience Biotechnology Co., Ltd, China) in accordance with the manufacturer's instructions. High sensitivity C-reactive protein (hs-CRP) was analyzed in chemiluminescent immunometric assay (Siemens Healthcare Diagnostics Products GmbH, USA).

Total antioxidant status (TAS) and total oxidant status (TOS), total thiol, native thiol, malondialdehyde (MDA) levels were measured using commercially available assay kits (Rel Assay Diagnostics, Turkey) and Mindray BS300 Auto Biochemistry Analyzer™ [27]. Half of the difference of the result obtained by the subtraction of native thiol amount from total thiol content indicated the disulphide level [28].

Statistical analyses

Statistical analyses were performed using the SPSS version 21.0 statistical software package (IBM, Armonk, NY, USA). To identify the potential differences in total energy and

Table 1. Energy and nutrient contents of the meals

	MED meal	WD meal
Energy (kcal)	1122	1116
Carbohydrates (g)	140.9	132.1
Carbohydrates (%E)	51	49
Protein (g)	54.3	29.9
Protein (%E)	20	11
Total fat (g)	36.9	50.4
Total fat (%E)	29	40
SFA (g)	6.9	11.7
SFA (%E)	5.5	9.5
MUFA (g)	19.4	15.7
MUFA (%E)	15.5	12.6
PUFA (g)	9.1	17.6
PUFA (%E)	7.3	14.1
n-3 PUFA (g)	4.1	0.5
n-6 PUFA (g)	5	17.1
Linoleic acid (g)	4.3	17
Palmitic acid (g)	4.5	6.3
Arginine	3578	1569
Methionine	1310.5	590
Cysteine	730.8	468
Dietary fiber (g)	28.3	6.5
Vitamin A (μg)	344.8	81.6
Vitamin C (mg)	64.4	30.1
Vitamin E (mg)	11.6	20.9

MED: Mediterranean diet; WD: Western diet; SFA: saturated fatty acids; MUFA: monounsaturated fatty acids; PUFA: polyunsaturated fatty acids; %E: percentage of energy.

nutrient intake (as recorded for 3-days) on the days prior to test meal sessions, the data were compared using the Paired-t test if whether it follows normal distribution, and the Wilcoxon Sign test if not. The data were checked for normality by using the Shapiro-Wilk formal normality test. To test the differences in inflammatory and oxidative stress markers between time points, data were assessed for statistical significance using repeated-measures analysis of variance (ANOVA). Statistical significance level was taken as $p < 0.05$. Graphs were obtained using GraphPad Prism version 9 (GraphPad Software Inc., La Jolla, CA, USA).

Results

Participant characteristics

Eleven healthy women who met the inclusion criteria were included in the study. The characteristics of the participants are summarized in Table 2. Participants' mean age was 27.5 ± 6.55 years and the mean BMI was 21.8 ± 1.56 kg/m². As compared to standard reference values, all participants

Table 2. Characteristics and blood parameters of the participants (n=11)

Characteristic	Mean±SD
Age (years)	27.5±6.55
Body weight (kg)	59.1±5.86
Height (cm)	164±7.43
BMI (kg/m ²)	21.8±1.56
Body fat (%)	26.7±1.97
Fat-free mass (kg)	42.9±4.01
Serum glucose (mg/dL)	91.5±4.95
Serum total cholesterol (mg/dL)	153±18.3
Serum HDL cholesterol (mg/dL)	54.8±3.98
Serum LDL cholesterol (mg/dL)	85.2±11.1
Serum triglycerides (mg/dL)	67.8±18.9
Serum thyroid stimulating hormone (TSH) (mU/L)	2.4±0.83

All blood parameters were measured in fasting samples. Values are shown as mean±SD. BMI: body mass index; HDL: high-density lipoprotein; LDL: low-density lipoprotein.

had normal serum glucose, total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides and thyroid stimulating hormone levels.

Self-reported dietary intake of energy and nutrients

All participants maintained their usual dietary habits throughout the study. There were no significant differences in terms of the energy and nutrient intakes prior to both test meals ($p>0.05$) (data not shown).

Markers of oxidative stress

Postprandial metabolic responses of oxidative stress markers are shown in Figure 1. The baseline serum TAS level was 1.14 ± 0.10 mmol/L for the MED meal. A small but statistically significant increase of 0.05 ± 0.02 mmol/L in serum TAS level from baseline to 2 h was observed ($p=0.017$). In addition, the change after the MED meal intake was statistically significant overall ($p=0.033$). However, after the WD meal was consumed, the TAS level tended to decrease from the baseline to 4 h postmeal (mean difference: 0.11 ± 0.09 mmol/L). Although there was no significant change in certain time points, the overall change was found significant ($p=0.005$). Postprandial serum TOS levels did not change significantly for none of the meals ($p>0.05$).

The mean serum total thiol level increased significantly for the MED meal at 2 h (mean difference 23.0 ± 7.69 mmol/L; $p=0.013$) and 4 h (mean difference 37.6 ± 14.1 ; $p=0.023$) postmeal, this level peaked at 4 h. The mean level of the baseline was 306 ± 26.4 mmol/L and it increased to 344 ± 38.7 mmol/L at 4 h after the MED meal intake.

Although a significant increase was observed after the WD meal intake at 2 h ($p=0.018$), there was no significant increase in the following hours. Compared to baseline level, native thiol concentrations were significantly higher after the MED meal intake at 2 and 3 h ($p=0.027$, $p=0.025$, respectively). After the WD meal, although the native thiol level decreased below the baseline at 2 h, the difference was not significant ($p>0.05$). Disulphide level increased significantly after the WD meal at 3 h ($p=0.045$), while there was not a significant change in the MED meal ($p>0.05$).

The serum MDA levels showed a significant decrease compared to baseline (0.57 ± 0.52 mmol/L) at 2 h (0.40 ± 0.45 mmol/L, $p=0.022$) post-consumption of the MED meal and continued to decrease insignificantly throughout the following 4 h. After the WD meal intake, MDA response revealed insignificant but higher levels compared to the MED meal ($p>0.05$).

Markers of inflammation

The postprandial changes in IL-6, IL-17, IL-23, hs-CRP, TNF- α and NF- κ B levels are presented in Figure 2. IL-6 and IL-17 showed no significant time interaction for the MED meal. A significant response of the WD meal was seen on postprandial serum IL-6 levels. The baseline IL-6 level was 10.4 ± 0.43 pg/mL and increased to 11.3 ± 1.17 pg/mL at 3 h ($p=0.018$) and 11.8 ± 1.93 at 4 h ($p=0.017$). The mean serum IL-17 level increased significantly for the WD meal at 4 h after ingestion ($p<0.05$), this level peaked at 3 h post-meal. A significant influence of the meals was also detected for IL-23. Serum IL-23 significantly increased for the MED meal ($p=0.031$) and for the WD meal at 3 h ($p<0.001$) and 4 h ($p=0.038$). Serum IL-23 levels rose to maximum at 4 h for both meals.

Although the hs-CRP responded with a decreasing trend after the MED meal and an increasing trend after the WD meal, these changes were not found statistically significant ($p>0.05$). Neither MED nor WD meal affected the postprandial serum TNF- α and NF- κ B levels ($p>0.05$).

Discussion

The postprandial state is characterized by low-grade oxidative and inflammatory responses in the organism depending on the meal components [29]. This randomized crossover postprandial study was designed to compare the oxidative and inflammatory responses to the MED and the WD meals in healthy women. The present study has two main findings: firstly, the MED meal intake induced favorable changes in oxidative stress markers as total thiol, native thiol, disulphide and MDA. Secondly, the WD meal

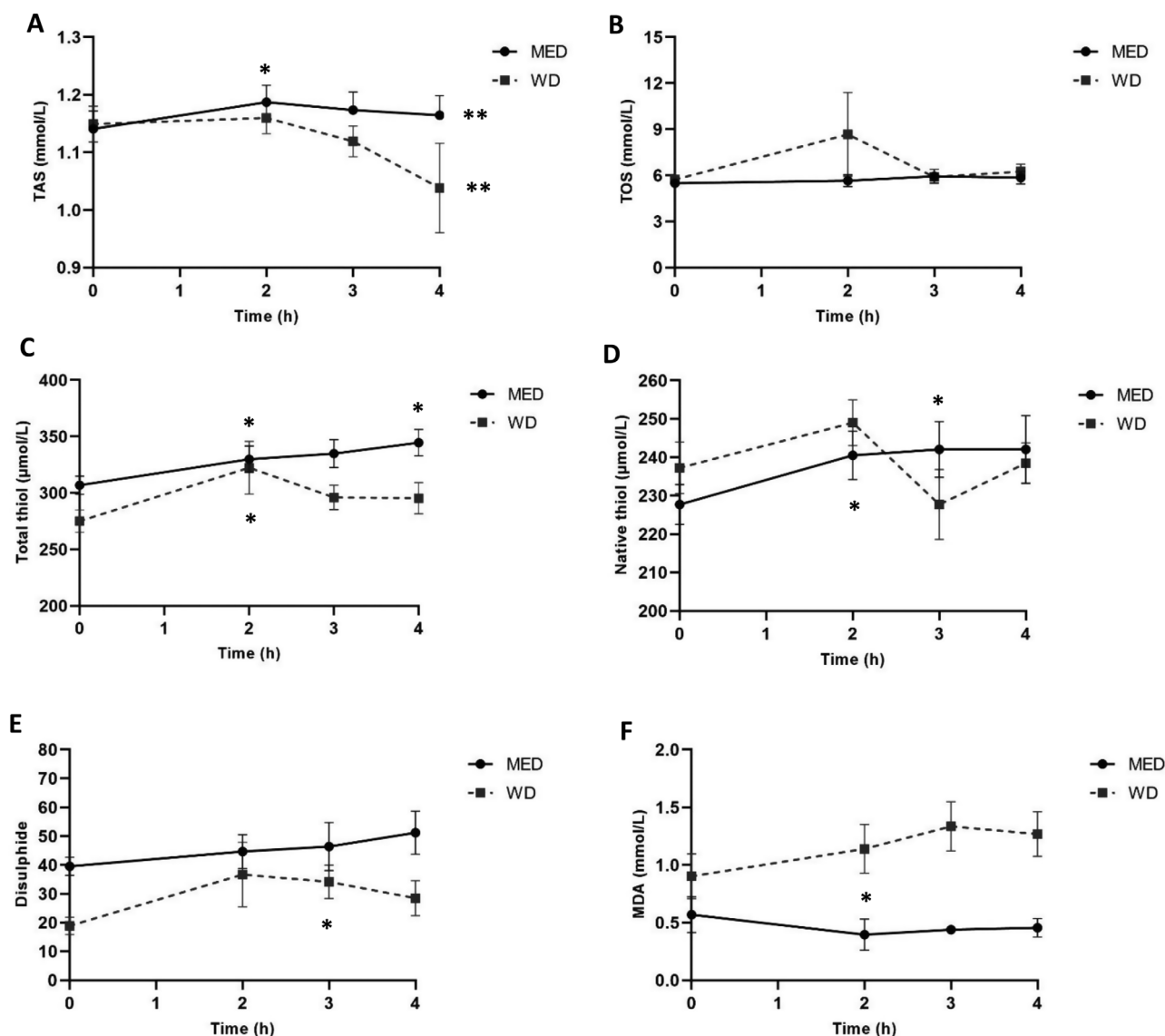


Figure 1. Baseline/fasting and postprandial concentrations of (A) serum TAS, (B) serum TOS, (C) serum total thiol, (D) serum native thiol, (E) serum disulphide (F) serum MDA in healthy women. Data were analyzed with repeated-measures ANOVA with post hoc pairwise comparison. Data are mean±SEM (n=11). *Significantly different from baseline (p<0.05), **statistically significant overall (p<0.05). TAS: total antioxidant status; TOS: total oxidant status; MDA: malondialdehyde; MED: Mediterranean diet; WD: Western diet.

intake induced the onset of a postprandial inflammatory response characterized by high levels of IL-6, IL-17 and IL-23, when compared with the fasted state. These metabolic imbalances are known to play critical roles in the development of chronic diseases and therefore could represent important targets to contribute dietary strategies for disease prevention in healthy individuals.

Markers of oxidative stress

In accordance with the hypothesis of this study, the MED meal intake showed favorable effects on suppression of oxidative stress while the WD meal intake caused an oxidative response. The indicators of these results are,

significantly increased serum levels of TAS at 2 h, total thiol at 2, 4 h, native thiol at 2, 3 h and decreased serum MDA level at 2 h compared with baseline after the MED meal intake (Figure 1). These findings may be explained by the higher content of antioxidant compounds in the MED meal than the WD meal. In this study, the MED meal included black grapes, balsamic vinegar, vegetables, extra virgin olive oil all containing high phenolic components and a Turkish drink named hardaliye with a high content of polyphenol, flavonoid and resveratrol [30]. In previous crossover studies like this, it has been shown that a meal, containing a food or drink with a high antioxidant components, increases the postprandial serum TAS level [31, 32, 33] and that the high polyphenol content is protective

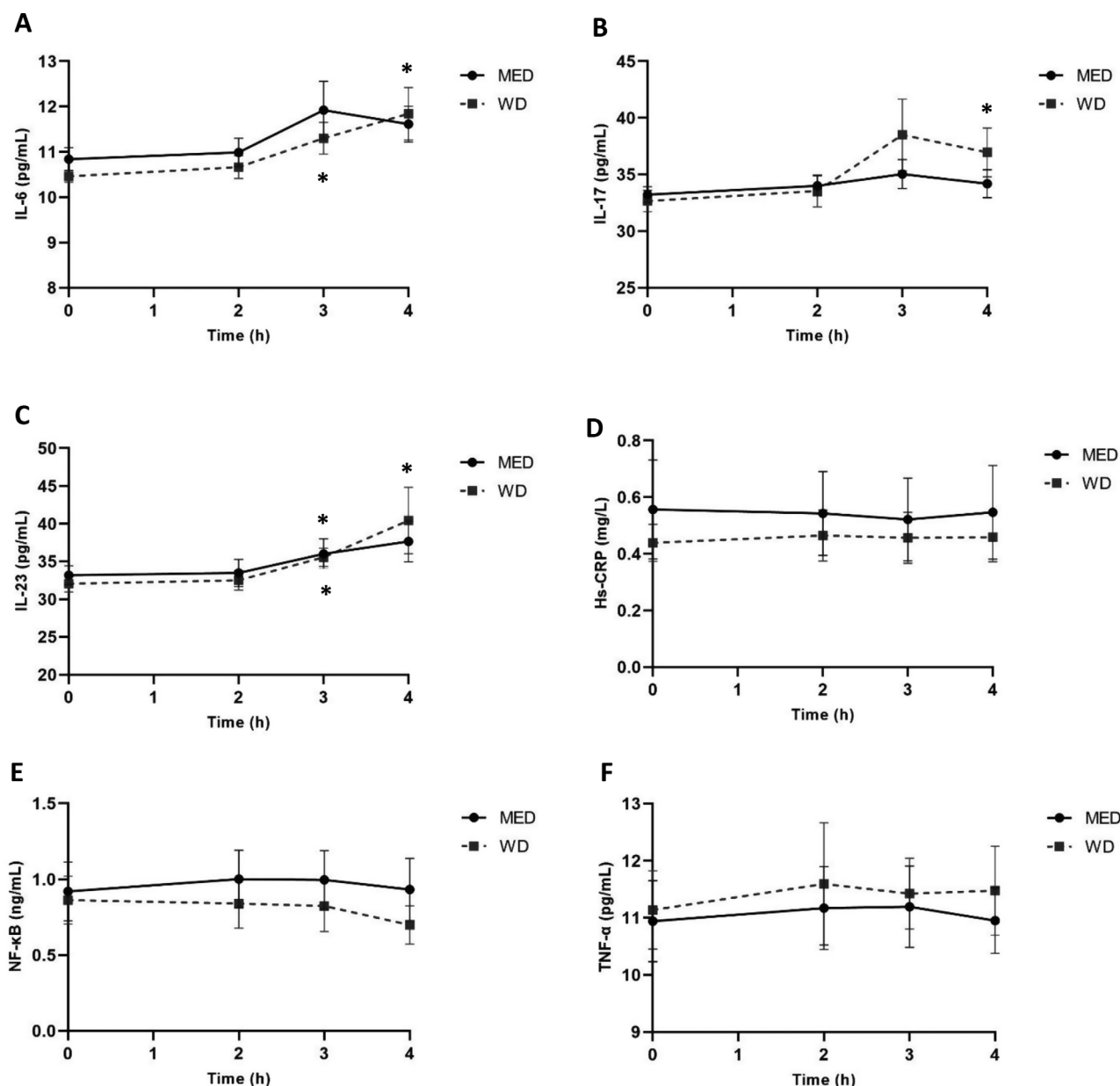


Figure 2. Baseline/fasting and postprandial concentrations of (A) serum IL-6, (B) serum IL-17, (C) serum IL-23, (D) serum Hs-CRP, (E) serum NF-κB (F) serum TNF-α in healthy women. Data were analyzed with repeated-measures ANOVA with post-hoc pairwise comparison. Data are mean ± SEM (n=11). *Significantly different from baseline (p<0.05). IL-6: interleukin-6; IL-17: interleukin-17; IL-23: interleukin-23; Hs-CRP: high-sensitivity C-reactive protein; NF-κB: nuclear factor kappa B; TNF-α: tumor necrosis factor alpha; MED: Mediterranean diet; WD: Western diet.

against postprandial oxidative stress [34]. These observations are consistent with our findings.

MDA level, an indicator of lipid peroxidation, was assessed as an oxidative marker in this study and significantly decreased at 2 h after the MED meal intake. A common conclusion in the studies is that; the antioxidants, especially polyphenol content of the meal directly reduces the MDA level and significantly increases the antioxidant capacity [31, 35, 36]. A study also has shown that daily consumption of hardaliye provides a significant reduction in serum MDA level [30]. Additionally, Masuda et al. [37]

suggested that thiols may have a potential protective role against lipid oxidation. In this study, increased serum thiol levels after the MED meal consumption may have shown synergic effects with polyphenols and contributed to the decrease of MDA level.

Thiols, containing sulfhydryl (-SH) groups, are the first antioxidants depleted in the presence of oxidative stress in cells. Oxidation of -SH groups of thiol-containing molecules by free radicals causes the formation of disulfide bonds in the organism. The disulfide bonds can be reduced back to thiols by antioxidant systems. Thus, a

dynamic thiol/disulphide homeostasis is maintained. A decrease in total thiol and native thiol levels along with an increase in disulphide levels, that is impaired dynamic balance, are indicators of oxidative stress in the organism [38]. Impaired thiol/disulphide homeostasis plays an important role in the pathogenesis of various chronic diseases [28]. To our knowledge, this is the first study investigating the associations between different meal patterns and thiol/disulphide homeostasis. The clarification of dietary pattern-thiol/disulphide interaction is believed to be important because of its potential association with chronic diseases.

In this study, a significant increase was seen in serum total thiol level at 2 h and 4 h postmeal for the MED meal. In accordance with total thiol, serum native thiol level was found significantly increased at 2 h and 3 h postprandially. Meanwhile, disulphide level showed a slight change at all time points. In a previous postprandial study, the observed increase in serum thiol level was attributed to meals' sulphurous compounds [31]. In this context, the significant increases in postprandial serum thiol levels in this current study may be explained with sulfur compounds such as high methionine (1310 mg) and cysteine (730 mg) that the foods in the MED meal contained.

Contrary to the results seen upon consumption of the MED meal, the WD meal intake induced postprandial oxidative stress in this study. After the WD meal intake, while the serum TOS level increased, a decrease in TAS level was observed during the postprandial state (Figure 1). This indicates that the mechanisms to compensate for oxidative stress are insufficient [39]. Moreover, serum native thiol level dropped below baseline while serum disulphide level increased significantly. Although the serum total thiol level increased significantly from the baseline to the 2 h postmeal, this increase could not be maintained after. In addition, the increase in MDA level observed is another supporting factor for oxidative response. A previous postprandial study showed that the meals rich in fat lead to an increase in MDA level due to peroxidation of PUFA. Linoleic acid is a kind of PUFA that can easily oxidize and cause an increasing MDA response [34]. In this context, the most important factors contributing to this effect is assumed to be the WD meal's high fat [40% energy (E)] and PUFA (14%E) together with linoleic acid (17 g) components in this study. Additionally, red meat component of the meal may also have contributed to increase the postprandial MDA level. In a study, the consumption of red meat was shown to increase the postprandial MDA levels threefold [40].

Markers of inflammation

Persistent low-grade inflammation is an important underlying factor in many high mortality chronic diseases, thus

investigating the inflammatory responses to meal components is important [23, 41].

In the present study of healthy individuals, it was hypothesized that the WD meal intake would cause a greater inflammatory response than the MED meal. However, this prediction was partially supported because significant changes were only observed in cytokine levels. In this study, pro-inflammatory markers of IL-6, IL-17 and IL-23 increased significantly compared to baseline in WD meal intake, while MED meal intake did not make a significant difference in any cytokines but IL-23 (Figure 2). On the other hand, it was determined that the consumption of both meals did not indicate a significant difference at any time in hs-CRP, TNF- α and NF- κ B levels compared to fasted state.

Several previous postprandial studies have reported that meals that are rich in fat, especially saturated fatty acids, lead to inflammatory response [29, 42, 43, 44]. In harmony with previous studies, fat content of the meals (total fat: MED: 36.9 g; 29%E; WD: 50.4 g; 40%E) can explain the different inflammatory responses in this study. A review by Emerson et al. [43] demonstrated that, serum levels of IL-6 increased after consumption of high-fat meals in more than 70% of previous studies. Since many studies were not designed with a realistic approach [45, 46] and the fat content of the meals are much higher than this study, it was challenging to compare the results of the current study with the literature. In a similar design with this study, the same amount of fat (50 g) containing meal intake resulted in a higher IL-6 response in healthy individuals [47] in line with this study results. Also, high *n*-6 PUFA (e.g., from sunflower oil) content of the WD meal may also have contributed to the increase in serum levels of inflammatory markers, consistent with Vries et al. [48].

In recent years, IL-17, a proinflammatory cytokine, has attracted the attention of researchers and clinicians with its proven effects in inflammation and autoimmune diseases [49]. In a postprandial crossover trial, it was shown that a high-energy and high-fat meal (1344 kcal, 55%E) increased IL-17 production, while to add high-antioxidant juice to the same meal significantly inhibited IL-17 production in healthy overweight individuals [50]. The result of this study is that, the significant increase in IL-17 level after the WD meal intake is compatible with the literature. Since, the IL-17 level has been started to be evaluated in postprandial studies recently, there are not many studies in the literature yet. It is thought that the studies of the next researchers will contribute to the literature on this subject.

A recently published review reported that foods high in polyphenols reduce postprandial markers of oxidative damage and inflammation [51]. It has been suggested that reactive oxygen species-mediated postprandial inflammation can be prevented in the presence of antioxidants [47]. In a study evaluating the postprandial outcomes of different

oil types, extra virgin olive oil was shown to significantly reduce inflammatory markers [52]. Also dietary fiber, which is almost 4 times higher in the MED meal compared to the WD meal in this study, is known as an important anti-inflammatory component of dietary patterns [53]. In this context, the high polyphenol and antioxidant components in red grapes, hardaliye drink, extra virgin olive oil, green leafy vegetables and nuts, as well as the high fiber of the MED meal, appear to be factors in preventing the activation of inflammatory markers in this study.

The only significant difference reported with the MED meal intake compared with the baseline was seen at the IL-23 level at 3 h. High calorie intake may be a reason for inflammatory response regardless of the meal components [54]. According to the results of this study, the energy load may be the main determinant for acute IL-23 response. However, it was noteworthy that there was a higher mean difference by the WD meal intake, although there was a significant increase after both meal consumptions in this isocaloric meal design. To our knowledge, this is the first study investigating the postprandial responses of IL-23 level.

Chan et al. [23] compared meal models from four different regions (American-West, South China, North China, India) in their study, in which they used a realistic meal model similar to this study design. As a result of the study, test meals did not make a significant difference in serum hs-CRP level. The results in this study are also in line with the study of Chan et al.

Conclusion

The oxidative and inflammatory responses to different dietary patterns in order to better understand the metabolic challenges that individuals experience in daily life was explained in this study. As a result, considering that even a single realistic WD meal consumption induced a greater response in oxidative and inflammatory markers compared to MED meal intake, the impact of chronic exposure could be a reason for impaired metabolism and chronic diseases. Therefore, it is suggested that the meals met nutrient and food characteristics of MED pattern, have beneficial effects in maintaining postprandial homeostasis and thus preventing diseases in long term.

The strengths and limitations

The strengths of this study were the randomized crossover design with healthy and young participants to assess the potential effects of the MED and the WD patterns with the biomarkers which are not usually analyzed like thiols,

IL-17 and IL-23. One of the limitations of this study is that the participants were only women. Therefore it is possible that the results may differ in men. Also, since some of the markers might have been a long half-life, extending the postprandial time from 4 h to longer could help to determine the biomarker exposure period. In addition, follow-up studies may require a larger participant number.

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History

Received June 6, 2021

Accepted September 25, 2021

Published online October 12, 2021

Acknowledgement

Each author, contributed to the development of the overall research plan and study protocol, data management, study oversight, statistical analyses and preparation of the manuscript. The authors approved the final version of the manuscript.

Conflict of interest

The authors declare that there are no conflicts of interest.

Funding

This work was funded by Yüksek İhtisas University Scientific Research Projects under grant number 45825805/13.

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