Disease occurrence clearly impacts eating behavior: A crosssectional study among Jordanians

Buthaina Alkhatib¹, Lana M. Agraib², Islam Al-Shami¹

¹ Department of Clinical Nutrition and Dietetics, Faculty of Applied Medical Sciences, The Hashemite University, Zarqa, Jordan. <u>bkhatib@hu.edu.jo</u>; <u>islamk@hu.edu.jo</u>

² Department of Nutrition and Food Science, Faculty of Allied Medical Sciences, Al-Balqa Applied University, Al-Salt, Jordan. <u>Lanamg2007@yahoo.com</u> ORCID: 0000-0003-0938-7961

Abstract

Background: Many factors have affected eating behaviors, including disease occurrence. The aim is to evaluate eating behaviors and how disease incidence affects them in Jordan's healthy population.

Methods: A cross-sectional study was conducted on 1170 males and 1512 females (>8 years) between March and May 2022. Participants were categorized into two groups: those with diseases (diabetes, cardiovascular diseases (CVD), and comorbidities) and those with no diseases. Eating behaviors were evaluated, including meal timing and frequency, late-night, and fast food consumption.

Results: Most participants tended not to skip their meals, consume two to three meals daily, consume one to three snacks, have lunch between 1:00–and 6:00 p.m., and do not intake food late. The most consumed food group is cereals (Males: 65.1% for healthy and 60.8% for diseased; Females: 64.9% for healthy and 60.4% for diseased). The prevalence of most frequent fats-food consumption (1-5 times/week) was recorded in healthy participants (67.9%) compared to diseased participants (50.4%) (p<0.001). Diseased participants reported significantly higher consumption of vegetables group (10.2%) compared to healthy participants (4.7%, p<0.001). The most missed

or unconsumed food group was dairy products (46.7% for healthy and 40.6% for diseased). The prevalence of morning and night eating was 43.4% and 26.1% of the diseased participants, in contrast to healthy participants (37.7% and 31.0%, respectively).

Conclusion: Even if Jordanians' eating behaviors are not dissimilar to healthy behaviors, the presence of diseases was a beneficial factor in increasing healthy eating behaviors.

Keywords: Eating behaviors, diabetes, cardiovascular diseases, meal timing and frequency, latenight consumption, and fast food intake.

Introduction

Eating behavior is "normal behavior involving eating habits, food selection, and preparation techniques, as well as the amount of food consumed" [1]. Moreover, LaCaille defines it as a broad concept that involves motivations for choosing certain foods, food preferences, eating practices, weight loss efforts, and issues with eating, such as obesity and eating and feeding disorders [2]. Unfortunately, how we eat, and the type and quantity of food we consume considerably affect our health. Obesity and related health complications and diseases were the most significant concerns when considering eating behaviors [3]. Therefore, eating behavior research is concentrated on the causes, prevention, and treatment of obesity, as well as the promotion of healthy eating habits that aid in managing and preventing obesity-related diseases, including diabetes (T2DM), hypertension, and some malignancies [2].

For decades, researchers have focused on the role of diet and its components on obesity and its related disease development, management, and treatment. However, the efforts have recently focused on eating behaviors and dietary patterns. Healthier eating behaviors such as consuming high-quality food and maintaining a balanced diet can help people keep their physical health and mental stability [4]; consumption of fruits, vegetables, and whole grains is inversely related to chronic diseases [5]. Controlling and regulating meal timing may be more important than the macronutrient composition of the diet in managing changes in body weight [6, 7]. As well as these healthy eating habits have been associated with a lower risk of metabolic syndrome (MetS) [8].

Uncooperatively, unhealthy eating behaviors, including eating foods deficient in nutrients, skipping meals, and failing to maintain a regular diet, are linked to several health issues and nutritional deficiencies [9, 10]. Moreover, many researchers have shown that various chronic

diseases have been linked to meal timing and frequency over the past five years [11]. In addition, epidemiological studies have shown that eating late at night is associated with an increased risk for obesity [12], cardiovascular disease (CVD), and T2DM [13, 14]. Furthermore, Madjd et al. documented that weight loss plans should include recommendations about energy intake and the timing of food intake [7].

The Jordanian population is experiencing an alarmingly high rate of obesity [15]. In 2020, Ajlouni et al. reported that three-quarters of men and women in Jordan were overweight or obese [16]. Also, the Jordanian population has been engaged in unhealthy eating behaviors, verified by numerous research. Alomari et al. observed that age, sex, obesity, education, and income appeared to contribute to changes in dietary behaviors. They also found that a higher percentage of Jordanian adults reported increased consumption of high-calorie food and late-night eating [17]. Dalky et al. reported that most Jordanian adolescents did not eat breakfast [18]. Moreover, among Jordanian university students, researchers indicated that most had a high prevalence of fast-food consumption [19] and did not meet the fruit and vegetable intake recommendations [20].

Despite the high prevalence of excess weight among the Jordanian population and its related chronic diseases, including T2DM and CVD, no study has highlighted the general population's eating behaviors and the shifting of eating behaviors due to disease. Therefore, the current study's objectives were to assess eating behaviors among Jordan's healthy population and the impact of disease occurrence on the Jordanians' eating behaviors.

Materials and methodology

Study population and Ethical Approval

A cross-sectional study in which outpatients from three governmental hospitals in the central regions of Jordan were recruited. Between March and May of the year 2022, participants were randomly selected for this population-based study. Participants from both genders who regularly visited these hospitals for follow-up care and/or their caregivers and who were > 8 years of age were invited to participate in the study. Exclusion criteria included subjects younger than eight years, female subjects who were pregnant or lactating, subjects with mental disorders, and any terminally ill subjects. People with a medical condition requiring significant dietary changes in type, frequency, or quantity or a food allergy that significantly affected their intake or changed their eating behaviors were also excluded.

All willing and eligible subjects gave oral informed consent for inclusion in the survey before participation. For children and adolescents, their parents gave verbal consent to participate. The study followed the Declaration of Helsinki, and the Institutional Review Board approved the protocol.

Data Collection and Anthropometric Assessment

Following the participant's agreement, a face-to-face interview was conducted to extract data using a pre-designed set of questions. The data consisted of general demographic information, including age and gender, general health, and questions on the existence of chronic diseases or other health issues. A list of questions on food consumption type, amount, and time was also collected. These data were obtained by a team of well-trained and highly qualified nutritionists. Participants were categorized into two groups: those with diseases (diabetes, cardiovascular diseases (CVD), and comorbidities) and those with no diseases. Weight, height, waist, and hip circumferences were recorded during these interviews. The research team checked the collected anthropometric data to ensure its accuracy and to use it in further analysis. Researchers used the participant's height and weight to calculate their body mass index (BMI), which was then computed using Quetelet's formula: BMI = weight (kg)/height (m²) [21]. BMI values lower than 18.5 kg/m² for adults and older persons indicate underweight, between 18.5 and 24.9 kg/m² indicates normal weight, between 25 and 29.9 kg/m² indicates overweight, and above 30 kg/m² indicates obesity [22]. The Body Mass Index-for-Age Z-Score (BAZ) was calculated for children and adolescents. According to WHO reference curves (2007), values >+1 SD represent overweight, and values >+2 SD represent obesity for the connection of BMI-for-age with certain conditions. Values >-2 were considered thin or underweight, while values between +1 and -2 were considered normal [23].

Eating behaviors and meal timing assessment

Eating behaviors, including meal timing and frequency, and late-night and fast-food consumption were evaluated. Meal timing was categorized as eating in the morning time (breakfast and snacks), evening time (lunch and snacks), and nighttime (dinner and snacks) [24]. Eating times included: morning eating (eating between 5:00–9:00 a.m.), non-morning eating (eating after 9:00 a.m.), evening eating (eating between 6:00–9:00 p.m.), and night eating (eating after 9:00 p.m.) [24, 25]. Regarding meal frequency, participants were asked to describe their regular eating meals, food groups (most and least food groups consumed, type of consumed snacks, and most skipped meals), and eating frequency (number of meals and snacks/day).

Statistical Analysis

Data were analyzed using IBM SPSS Statistics (IBM Corp. Released 2012. IBM SPSS Statistics for Windows, Version 23.0. Armonk, NY: IBM Corp). Kolmogorov-Smirnov test and Kurtosis

and Skewness values were used to verify the normality of variables. Continuous variables were expressed as means with standard deviations. Categorical variables were presented as frequencies and percentages (%). A chi-square (χ 2) test was performed to test participant differences. An independent-sample t-test was performed to analyze the differences between the means of continuous variables. A *p*-value of less than 0.05 was used as the level of significance.

Results

Table 1 shows the general characteristics of the study population based on gender. Of the study population, there were 1170 males and 1512 females. The male participants had a mean of 44.7 \pm 19.4 y for age, 72.1 \pm 16.5 kg for weight, 168.8 \pm 5.0 cm for height, 83.4 \pm 15.4 cm for waist circumference, 96.2 \pm 17.5 cm for hip circumference, and 25.2 \pm 6.4 kg/m² for BMI. The female participant had a mean of 43.2 ± 19.7 y for age, 67.6 ± 15.9 kg for weight, 160.0 ± 10.6 cm for height, 77.2 \pm 21.5cm for waist circumference, 89.0 \pm 23.8cm for hip circumference, and 26.3 \pm 5.9kg/m² for BMI. Adult participants accounted for 71.1% and 71.7% of male and female participants. More than half the male and female participants had a high school or lower education level, and female participants had a higher prevalence of university level or higher education (30.7%) than male participants (22.8%). Regarding the employment state, even though both genders had a high prevalence of unemployment state, the male participant had a higher prevalence of being employed (46.9%) when compared to females (12.5%) (p < 0001). Almost two-thirds of the male and female participants were married. Of males, 74.7% were smokers, while 35.7% of females were smokers. Females were more negative smokers (33.9%) and non-smokers (17.0%) compared to males (7.0%) and 4.2% respectively). Most males and females were not physically active daily. However, the number of males who engage in daily physical activity was higher (25.2% vs. 14.3% for females). Based on BMI classification, males had higher normal weight (50.9%) and overweight (37.6%)

prevalence than females (41.8% and 36.5%, respectively), but females had a higher prevalence of obesity (21.7% vs. 11.4% for males).

Studying the study population's meal frequency and eating behavior (Table 2) revealed that twothirds of healthy or diseased people tend not to skip their meals. If they skip meals, breakfast is the most skipped (18.6% and 17.0%, respectively), followed by dinner (13.0% and 15.3%, respectively). This was supported by the fact that 62.5% of healthy and 64.2% of diseased participants reported consuming three meals daily. Healthy participants reported a higher prevalence of consuming two meals (31.1%) and lower consumption of one meal (0.9%) than diseased participants (28.3% and 2.3%, p=0.009, respectively). Meal frequency and eating behavior based on gender, as illustrated in Table 3, reported similar results of the total population regarding skipping meals, except for females, where healthy females reported a higher prevalence of skipping breakfast (20.1% vs. 16.4% for diseased), and diseased females reported a higher prevalence of skipping dinner (18.5% vs. 13% for healthy). Based on gender (Table 3), both males and females, healthy or diseased, had a high prevalence of consumption of two to three meals daily. Among males, the diseased participant had a higher prevalence of three meals (71.7% vs. 66.1% for healthy; p=0.010), while the healthy had a higher majority of two meals (28.5% vs. 21.5% for diseased; p=0.010). Regarding the number of snacks among the total population (Table 2) or based on gender (Table 3), most study participants, either healthy or diseased, reported that they consumed one to three snacks and did not intake late food.

Fast food eating was more frequently consumed 1-5 times/week among participants, with a higher prevalence of its consumption among healthy participants (67.9%) compared to diseased participants (50.4%) (p<0.001). Also, diseased participants reported a higher prevalence of non-consuming fast food (30.2% vs. 6.0% for healthy). Similar results based on gender were reported

(Table 3); males and females, regardless of whether they had the disease or not, reported a high prevalence of 1-5 times/week frequent consumption of fast food (males: 68.7% for healthy and 60.1% for diseased, p<0.001; females: 67.0% for healthy and 43.6% for diseased, p<0.001). These results indicate that having diseases shifted the population's eating habits toward less fast-food consumption.

From the food group based on MyPlate, the most consumed group by both healthy and diseased participants was cereals (65.1% for healthy and 60.7% for diseased), followed by the protein group (22.3% for healthy and 22.6% for diseased). In addition, disease participants reported a high consumption of vegetables group (10.2%), which was significantly higher than healthy participants consumption (4.7%, p < 0.001) (Table 2). Male and female participants reported similar results for the total study population, with the most consumed group being cereal (Males: 65.1% for healthy and 60.8% for diseased; Females: 64.9% for healthy and 60.4% for diseased) followed by protein (Males: 25.4% for healthy and 22.9% for diseased; Females: 19.8% for healthy and 22.6% for diseased) and higher consumption of vegetables by diseased participants than the healthy participants in both genders (Table 3). The most missed or unconsumed food group based on MyPlate for the total study population (Table 2) was dairy products (46.7% for healthy and 40.6% for diseased), followed by legumes (23.2% for healthy and 25.3% for diseased). Even though the protein group was one of the most consumed groups by participants, it was still one of the food groups with a high prevalence of being unconsumed by diseased participants (16.4%). The same was seen in male and female participants, who reported a high prevalence of missing dairy products, followed by the legumes group (23.9% for healthy and 22.7 for diseased; Females: 22.9% for healthy and 27.3% for diseased). The missing dairy product consumption was reported to be higher among healthy males and females (49.2% for males and 44.5% for females) compared to diseased (40.7% for males and 40.3% for females). Also, diseased males reported a higher prevalence of missing the consumption of protein group (16.6% vs. 6.4% for healthy). At the same time, diseased females reported a higher prevalence of missing the consumption of legumes (27.3%) and protein group (16.4%) compared to healthy females (2.9% and 12.0%, p=0.002, respectively).

The primary meal timing prevalence among the study population is presented in Table 4. Regarding the breakfast meal, 49.3% of healthy and 46.4% of diseased participants were nonmorning eaters who had breakfast after 9:00 a.m. However, 43.4% of the diseased participants reported being morning eaters who had their breakfast before 9:00 a.m., in contrast to healthy participants who had a 37.7% prevalence of being morning eaters, which may indicate that having the diseases may affect breakfast meal timing to become earlier. Almost all the healthy and diseased participants reported having lunch between 1:00-6:00 p.m. About dinner, 57.4% of healthy and 60.7% of diseased participants had dinner between 6:00–9:00 p.m. Nevertheless, 31.0% prevalence of healthy participants had night eating after 9:00 p.m., which is higher than the prevalence of diseased participants (26.1%, p=0.022). Regarding males, 49.3% of healthy and 45.6% of diseased male participants reported being non-morning eaters. However, at the same time, 45.1% of diseased males and 39.2% of healthy males were reported being morning eaters too. Nearly all healthy males (91.9%) or diseased (94.2%) had lunch between 1:00–6:00 p.m. Evening eaters were more prevalent among healthy males (3.8%) than diseased males (0.5%). Having dinner between 6:00-9:00 p.m. was more commonplace among healthy (54.9%) and diseased males (57.0%) compared to night eating after 9:00 p.m. (34.2% and 33.0%, respectively). For females, also, either healthy or diseased, they had a prevalence of being non-morning eaters (49.3% and 46.8%, respectively). However, diseased females had a higher percentage of morning

eating (42.4%) than healthy females (36.5%, p=0.031). Both healthy and diseased females had a prevalence of having lunch between 1:00–6:00 p.m. (92.8% and 94.4%, respectively) and dinner between 6:00–9:00 p.m. (59.3% and 63.3%, respectively). However, healthy females still reported a higher percentage of night eating (28.4%) than diseased females (21.4%, p=0.006).

Discussion:

Most chronic illnesses, such as T2DM and CVD, require significant dietary changes as part of their treatment. These therapeutic dietary changes typically involve making healthier, more nutrient-dense food choices and, most of the time, additional modifications to the patient's behaviors regarding eating time to align their dietary intake with their medications. In the current study, 64.2% of diseased and 62.5% of healthy participants consumed three meals daily. Also, most study participants, whether healthy or suffering from illness, stated that they consumed one to three snacks daily. Food frequency, which refers to the number of eaten, is linked to chronic diseases [11]. Many studies documented this relationship and suggested modifying behavior to minimize the risk or prevent chronic diseases. At the same time, eating less than three meals daily was reported to be related to being overweight or obese [26]. Furthermore, Alkhulaifi and Darkoh supposed that eating frequent meals protects MetS [11]. In another study, Titan et al. discovered that eating more than six meals per day was associated with lower levels of total and low-density lipoprotein (LDL) cholesterol, with a difference of 0.25 mmol/L [27].

Additionally, Tąpolska et al. reported that consuming > 4 meals/day effectively increases highdensity lipoprotein (HDL) cholesterol and lowers fasting triglycerides (TG); thus, higher levels of HDL and lower TG are associated with a reduced risk of heart diseases [28]. On the other hand, compared to people who ate three meals a day, people who only consumed one meal had higher fasting plasma glucose levels and reduced morning glucose tolerance [29]. Most of the published papers gather meals and snacks in one term as meals, while in the present study, we split it into two categories. Regarding the current study results, we do not know if the participants' frequent meal consumption behavior was taught as part of their therapy or if it developed due to their chronic illness diagnosis. Ultimately, people act healthily; thus, either of these explanations is true. Fortunately, whether healthy or not, two-thirds of the participants in the current study did not skip their meals. Nonetheless, among the meal skippers, breakfast is the most skipped meal. Participants with chronic illnesses tend to skip breakfast less than individuals in good health, demonstrating that they engage in healthier behaviors. Meal skipping has generated much discussion in recent years. Although irregular eating of meals is a common practice recently, its effect on health is of concern, especially breakfast skipping. In contrast to our results, only 5.2% of Indonesian adults, according to Khusun et al., skipped breakfast [30]. A systematic literature review by Pendergast et al. found that meal skipping (any meal) was reported in 12 of 35 studies, with prevalence ranging between 5 and 83%, and breakfast was the most frequently ignored meal 14-88%, compared to lunch and dinner [31]. Additionally, Zeballos and Todd documented that rather than skipping dinner, skipping breakfast or lunch has a more significant adverse effect on diet [32]. Furthermore, skipping breakfast (hazard ratio (HR): 1.13, 95% confidence interval (CI): 1.03-1.23) was associated with a 3-point major adverse cardiovascular event that led to the conclusion that diet behavior is a potential risk factor for incident CVD in Japanese people [33]. Earlier, Ofori-Asenso and colleagues had systematically reviewed multiple studies and stated that people who frequently skip breakfast are about 21% more likely than those who regularly eat it to get incident CVD or pass away from it (HR:1.21, 95% CI:1.08-1.35; $I^2 = 17.3\%$, P = 0.34). Additionally, the risk of allcause death was 32% greater in those who routinely skipped breakfast compared to those who consistently ate it (HR:1.32, 95% CI:1.17-1.48; $I^2 = 7.6\%$, P = 0.339) [34]. According to Ballon et

al.'s comprehensive review and meta-analysis, which included 96,175 individuals and 4935 cases, the summary relative risk (RR) for T2DM comparing skipping breakfast occasionally with never skipping was 1.33 (95% CI: 1.22, 1.46, n = 6 studies). Additionally, the nonlinear dose-response meta-analysis revealed that skipping breakfast increased the risk of T2DM with each additional day, reaching a peak risk of 55% at day 5 (summary RR: 1.55; 95% CI: 1.41, 1.71), supporting the findings of Ballon et al. that breakfast skipping increases the risk of T2DM [35]. Surprisingly, Ogata et al. discovered that when breakfast was skipped, plasma-free fatty acids (FFA) levels were significantly greater after lunch and that this relationship between FFA levels and the postprandial glycaemic response was positive (r=0.631, P < 0.01). In summary, skipping breakfast just once causes postprandial hyperglycemia and an altered insulin response after lunch [36]. Regarding gender-specific MetS and its components, Park et al. documented that men who ate two meals a day had a higher risk of MetS than men who ate three meals a day (odds ratio (OR):1.16, 95% CI:1.01–1.33), while women who ate two meals a day and skipped breakfast had a higher risk of having high fasting blood glucose levels (OR:1.18, 95% CI:1.02–1.35), and elevated triglycerides (OR:1.19, 95% CI:1.02–1.39). They hypothesized that meal frequency and the kind of significant meal skipped might be related to MetS and addressed the significance of eating breakfast to prevent MetS [37]. They thankfully noted that our study's diseased participant's behaviors regarding meal skipping in general and breakfast skipping specifically are future promising, hopefully continuing to stop or prevent their illness consequences.

Notably, in the current study, most of the healthy or diseased participants reported that they did not intake late food. Diseased participants preferred to eat in the morning and consumed fewer meals at night than healthier participants. These findings supported the hypothesis that people with diseases may alter the timing of their breakfast meals and shift their bedtimes earlier to match how frequently they take medications. It has been approved that appetite-regulating hormones and energy metabolism in the body are regulated by circadian rhythms that, when disordered, would potentially have unfavorable metabolic consequences [38]. There has been a connection between this circadian misalignment and obesity [38-40], CVD [7, 41], MetS [11, 42], high blood pressure [41, 43], and T2DM [41]. Studies have shown that eating later in the day and closer to bedtime is linked to more excellent weight status and an elevated risk of excess weight consequences [39, 41], bringing the timing of food intake to the fore of studies efforts. In line with our results, Mirghani reported that 20% of his study's participants ate late at night (compared to 21.5% of our diseased participants), and also found a direct relationship between eating dinner late, BMI, and HbA1c (Wald, 4.210, 95% CI, 0.743-0.993, P-value, 0.04 for HbA1c, and Wald, 6.777, 95% CI, 1.0221-1.165, P-value, 0.009 for BMI) [44]. According to Zhang et al.'s hypothesis, the velocity change rate between the group that ate at night most days and the group that never or rarely ate at night was 14.1 (95% CI, 0.6-27.5) cm/s per year in an adult population free of major chronic diseases. Arterial stiffness is a sign of arteriosclerosis and biological aging [45]. Furthermore, as compared to early eaters, late eaters had a lower average weekly rate of weight loss (505 (467) g/wk vs. 585 (667) g/wk; P = 0.008), higher odds of having weight-loss barriers (OR (95% CI): 1.22 (1.03, 1.46; P = 0.025), and lower odds of being motivated to lose weight (0.81 (0.66, 0.99); P=0.044). Claiming that eating late is linked to cardiometabolic risk factors and decreased weightloss intervention effectiveness [41]. Indeed, several researchers have demonstrated that people who eat their meals earlier in the day have more significant weight loss than people who eat the same meal later in the day [3, 7, 39].

Meal timing studies, while there is universal agreement that a meal consists of a certain amount of food consumed at a particular time [11], many cultures worldwide have different mealtime

traditions. The lack of a uniform method for defining meal timing has been one of the critical obstacles [39]. However, clock time has been utilized in numerous research to describe the timing of food consumption. According to Eom et al., eating breakfast, lunch, and dinner simultaneously every day may boost the success of weight loss. A significant determinant of the amount of weight loss was meal time regularity at breakfast (model 1: $\beta = -2,576.526$, P < 0.001), lunch (model 2: $\beta = -1,511.447$, P < 0.05), and dinner (model 3: $\beta = -1,721.428$, P < 0.05). They also noted that eating regularly is frequently more successful than substantially reducing calorie intake [46]. Surprisingly, most of the present study participants who had a chronic illness ate regular meals at established times and continued to practice healthier behaviors.

Although participants in this study consumed fast food more frequently (1–5 times per week), healthy participants consumed it more regularly than those with diseases. Additionally, those with diseases reported a higher prevalence of not eating fast food. These findings suggest that the diseased participants' eating behaviors regarding reduced fast-food consumption have been modified. Considering the abovementioned research on meal timing, it is essential to carefully assess the amount and type of food taken at each meal. A growing body of research indicated that fast food consumption is a vital factor for nutrition and may be a significant factor in obesity and its related comorbidities. Furthermore, fast food is often nutritionally poor and high in calories [47].

Even in previously metabolically fit individuals, Mirmiran et al. hypothesized that eating fast food could be linked to unhealthy phenotypes that included more than one component of MetS [48]. Moreover, during the last decades, numerous studies have assessed the effect of fast food consumption on health and found that individuals who consume it regularly were more likely at risk for developing obesity [49, 50], insulin resistance with or without incidence of T2DM [51], CVD [52], elevated blood pressure [52, 53], and MetS [54, 55]. Odegaard et al.'s study, which included follow-up data on Singaporean women, found that eating fast food ≥ 2 times per week increased the risk of developing type 2 diabetes (HR= 1.27, 95% CI= 1.03-1.54) and dying from coronary heart disease (HR = 1.56, 95% CI= 1.18-2.06)[51]. Furthermore, Bahadoran et al.'s study, which included a three-year follow-up of men and women who took part in the Tehran Lipid and Glucose Study, found that eating more fast food raised the risk of MetS by 85% (OR=1.85, 95% CI=1.17-2.95) [56]. According to Hosseini et al., eating a "fast-food" diet was linked to a 26% increased risk of having MetS (OR = 1.26; 95% CI: 1.04 to 1.54; p=0.0195) [57]. On the other hand, Mohammadbeigi et al. showed that fast food intake was linked to abdominal obesity as measured by the waist-to-hip ratio (WHR) (OR: 1.46, 95% CI: 1.11, 2.26) and that obesity and overweight are common among Iranian students [49].

Undeniably, weight and its associated comorbidities are influenced by meal composition in addition to timing. Cereals were the food category that healthy and diseased participants consumed the most in the present research sample, followed by protein. The amount of vegetables consumed by disease participants was much higher than that of healthy people. Despite being one of the food groups that participants consumed the most, protein was one of the food groups that participants with diseases were least likely to consume. Regarding cereals, Belobrajdic and Bird documented that diets high in whole grains are associated with a 20-30% reduction in the risk of developing T2DM [58]. Surprisingly, Ma et al. found that consumption of sugar and simple carbohydrates significantly decreased in response to T2DM diagnosis [59], which is somewhat consistent with our findings that diseased participants consumed cereals more frequently than healthy participants despite the fact we didn't study the types of cereals.

According to Amiot-Carlin, eating up to 800 g of fruit and vegetables per day and up to 600 g daily will lower your chance of developing CVD and cancer, respectively. Interestingly, increasing the amount of fruits and vegetables by one serving daily decreases the risk of CVD [60].

A meta-analysis of 11 prospective cohort studies with a total of 64,306 deaths among 350,452 participants confirmed the findings for total and animal protein intake: higher total protein intake was linked to higher all-cause mortality (pooled RR for highest versus lowest quantile: 1.05 (1.01, 1.10); and an association between animal protein and CVD mortality (RR: 1.09 (1.01, 1.18)]. A higher intake of plant protein was also linked to lower all-cause and CVD mortality in the meta-analysis [RR: 0.93 (0.87, 0.99) and for CVD mortality [RR: 0.86 (0.73, 1.00) [61]. However, Pfeiffer and colleagues noticed that other research comparing various animal proteins or contrasting animals with plant proteins in diabetic patients found that plant protein resulted in a more significant reduction of blood cholesterol [62]. Unfortunately, we did not distinguish between the source of the eaten protein in this investigation. Therefore, we proposed that this would explain why this food group was the most and least consumed among people with diseases. Dietary changes for chronic diseases typically promote consuming fewer servings of animal protein to reduce the concentration of saturated fat that could be taken alongside it while emphasizing plant-based proteins that are deficient in saturated fat.

Indeed, the results of this study confirmed that dairy products were the most neglected or underutilized food group. According to scientific evidence, milk and dairy products help people meet nutrient guidelines and may prevent most chronic diseases [63, 64]. Additionally, Quann et al. hypothesized that boosting the population's consumption of dairy products to recommended levels is one realistic dietary modification that might significantly enhance the population's sufficiency of some nutrients that are now under-consumed and have beneficial effects on health [65]. A higher total dairy intake-related metabolite profile score was associated with a lower T2DM risk [HR per 1 SD; Spanish: 0.76 (95% CI, 0.63-0.90); US: 0.88 (95% CI, 0.78-0.99)] according to Drouin-Chartier et al. based on 38 identified metabolites [66]. Feng et al. found a linear link between total dairy, milk, and yogurt and overweight or obesity. For each 200 g/d increase in total dairy, high-fat dairy, and milk, the risk fell by 25%, 7%, and 12%, respectively. For yogurt, the risk decreased by 13% for every 50 g/d increase. Also, a nonlinear relationship between total dairy consumption and hypertension was established, although significant inverse associations were reported for low-fat dairy (RR: 0.94; 95% CI: 0.90, 0.98) and milk (RR: 0.94; 95% CI: 0.92, 0.97) per increase in intake of 200 g/d [67]. However, despite saturated fat, a body of observational and clinical evidence suggests that consuming whole-milk dairy products does not increase the risk for cardiovascular disease [68]. So, we assume consuming dairy products lower than the recommendations is harmful behavior among our participants.

Strengths and Limitations

The strength of the present study is that it was carried out in a free-living population, making it simple for people with chronic illnesses who want to get healthier to apply the findings. Our analysis is constrained by the fact that the dietary evaluation in this study only examined the assumed daily nutritional intake in the previous twelve months using FFQ, not the dietary composition at each meal. It is necessary concerning the type, quantity, and timing of food intake. Also, in this study, using clock time was the key to characterizing the timing of food intake. However, this method falls short of accurately describing meal timing in the context of the internal circadian timing system. It should also be highlighted that the study's data on eating behaviors were self-reported and should be assessed with greater objectivity. Future cohort studies examining the relationships between eating behaviors and disease incidence are necessary to capture accurate

information on the temporal timing of meals. There is encouraging evidence that adjusting the time of day when meals are eaten can help those with chronic diseases manage it. At the same time, further study is required to see whether this is also a preventative dietary strategy in healthy people.

Conclusion

In conclusion, the overall study analysis suggests that the Jordanian population has undergone a move toward adopting healthier dietary behaviors—Jordanian adults suffering from chronic diseases adhere to more healthful eating behaviors than their disease-free counterparts. Our findings thus confirm our original theory that having persistent medical conditions causes patients to become more aware of and concerned about their health, leading them to modify all their eating behaviors. Nevertheless, a sizeable portion of the study's participants did not consume dairy products, which is thought to be negative behavior and should be considered.

Declarations

Informed consent "Informed consent was obtained from all participants included in the study."

Conflict of Interest: "The authors declare no competing interests".

Ethics Approval: All procedures performed in studies involving human participants were by the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. The Institutional Review Board of The Hashemite University (No.19/1/2022/2023) and the Jordanian Ministry of Health (MBA/20219) approved the study protocol.

References:

^{1.} Hernandez J, Bamwesigye D, Horák M. Eating behaviors of university students. Am J Public Health. 2016;100:216-22.

2. Collins S, Kirouac M, Gellman M, et al. Encyclopedia of behavioral medicine. NY Springer, New York. 2013:61-5.

3. Garaulet M, Gómez-Abellán P, Alburquerque-Béjar JJ, et al. Timing of food intake predicts weight loss effectiveness. International journal of obesity. 2013;37(4):604-11.

4. Zhu L, Zee PC. Circadian rhythm sleep disorders. Neurologic clinics. 2012;30(4):1167-91.

5. Esmaillzadeh A, Kimiagar M, Mehrabi Y, et al. Fruit and vegetable intakes, C-reactive protein, and the metabolic syndrome. The American journal of clinical nutrition. 2006;84(6):1489-97.

6. Shaw E, Leung GK, Jong J, et al. The impact of time of day on energy expenditure: implications for long-term energy balance. Nutrients. 2019;11(10):2383.

7. Madjd A, Taylor MA, Delavari A, et al. Effects of consuming later evening meal v. earlier evening meal on weight loss during a weight loss diet: a randomised clinical trial. British Journal of Nutrition. 2021;126(4):632-40.

8. Rodríguez-Monforte M, Sánchez E, Barrio F, et al. Metabolic syndrome and dietary patterns: a systematic review and meta-analysis of observational studies. European journal of nutrition. 2017;56:925-47.

9. Kelly T, Yang W, Chen C-S, et al. Global burden of obesity in 2005 and projections to 2030. International journal of obesity. 2008;32(9):1431-7.

10. Schibler U, Gotic I, Saini C, et al., editors. Clock-talk: interactions between central and peripheral circadian oscillators in mammals. Cold Spring Harbor symposia on quantitative biology; 2015: Cold Spring Harbor Laboratory Press.

11. Alkhulaifi F, Darkoh C. Meal timing, meal frequency and metabolic syndrome. Nutrients. 2022;14(9):1719.

12. Yoshida J, Eguchi E, Nagaoka K, et al. Association of night eating habits with metabolic syndrome and its components: a longitudinal study. BMC Public Health. 2018;18(1):1-12.

13. Kutsuma A, Nakajima K, Suwa K. Potential association between breakfast skipping and concomitant late-night-dinner eating with metabolic syndrome and proteinuria in the Japanese population. Scientifica. 2014;2014.

14. Jakubowicz D, Wainstein J, Tsameret S, et al. Role of high energy breakfast "big breakfast diet" in clock gene regulation of postprandial hyperglycemia and weight loss in type 2 diabetes. Nutrients. 2021;13(5):1558.

15. Rahim HFA, Sibai A, Khader Y, et al. Non-communicable diseases in the Arab world. The Lancet. 2014;383(9914):356-67.

16. Ajlouni K, Khader Y, Batieha A, et al. An alarmingly high and increasing prevalence of obesity in Jordan. Epidemiology and Health. 2020;42.

17. Alomari MA, Khabour OF, Alzoubi KH. Changes in dietary habits and eating behaviors during COVID-19 induced confinement: A study from Jordan. Human Nutrition & Metabolism. 2022;30:200169.

18. Dalky HF, Al Momani MH, Al-Drabaah TK, et al. Eating habits and associated factors among adolescent students in Jordan. Clinical nursing research. 2017;26(4):538-52.

19. Amr RA, Hammouh FG, Al-Smadi AM, et al. Eating behaviors, sociodemographics, self-perceived health, and weight status among Jordanian university students. Topics in Clinical Nutrition. 2018;33(4):302-10.

20. Alkhalidy H, Orabi A, Alzboun T, et al. Health-Risk Behaviors and Dietary Patterns Among Jordanian College Students: A Pilot Study. Frontiers in Nutrition. 2021;8:632035.

21. Nieman DC, Lee R. Nutritional assessment: McGraw-Hill Education United States of America; 2019.

22. WHO. World Health Organizatio, Obesity: preventing and managing the global epidemic. Report of a WHO consultation 2000 [Available from: <u>https://apps.who.int/iris/handle/10665/42330</u>

23. Onis Md, Onyango AW, Borghi E, et al. Development of a WHO growth reference for schoolaged children and adolescents. Bulletin of the World health Organization. 2007;85(9):660-7.

24. Ha K, Song Y. Associations of meal timing and frequency with obesity and metabolic syndrome among Korean adults. Nutrients. 2019;11(10):2437.

25. Khraiwesh H, Alkhatib B, Hasan H, et al. The impact of sleep quality, meal timing, and frequency on diet quality among remote learning university students during the COVID-19 pandemic. 2023.

26. Holmbäck I, Ericson U, Gullberg B, et al. A high eating frequency is associated with an overall healthy lifestyle in middle-aged men and women and reduced likelihood of general and central obesity in men. British journal of nutrition. 2010;104(7):1065-73.

27. Titan SM, Bingham S, Welch A, et al. Frequency of eating and concentrations of serum cholesterol in the Norfolk population of the European prospective investigation into cancer (EPIC-Norfolk): cross sectional study. Bmj. 2001;323(7324):1286.

28. Tąpolska M, Spałek M, Skrypnik D, et al. The influence of meal frequency on lipid profile in the Polish population. Neuroendocrinol Lett. 2019;40:325-8.

29. Carlson O, Martin B, Stote KS, et al. Impact of reduced meal frequency without caloric restriction on glucose regulation in healthy, normal-weight middle-aged men and women. Metabolism. 2007;56(12):1729-34.

30. Khusun H, Anggraini R, Februhartanty J, et al. Breakfast Consumption and Quality of Macro-and Micronutrient Intake in Indonesia: A Study from the Indonesian Food Barometer. Nutrients. 2023;15(17):3792.

31. Pendergast FJ, Livingstone KM, Worsley A, et al. Examining the correlates of meal skipping in Australian young adults. Nutrition journal. 2019;18(1):1-10.

32. Zeballos E, Todd JE. Skipping breakfast or lunch has a larger impact on diet quality than skipping dinner. Public Health Nutr. 2020:1-10.

33. Sakai K, Okada H, Hamaguchi M, et al. Eating behaviors and incident cardiovascular disease in Japanese people: The population-based Panasonic cohort study 14. Current Problems in Cardiology. 2023:101818.

34. Ofori-Asenso R, Owen AJ, Liew D. Skipping breakfast and the risk of cardiovascular disease and death: a systematic review of prospective cohort studies in primary prevention settings. Journal of Cardiovascular Development and Disease. 2019;6(3):30.

35. Ballon A, Neuenschwander M, Schlesinger S. Breakfast skipping is associated with increased risk of type 2 diabetes among adults: a systematic review and meta-analysis of prospective cohort studies. The Journal of nutrition. 2019;149(1):106-13.

36. Ogata H, Hatamoto Y, Goto Y, et al. Association between breakfast skipping and postprandial hyperglycaemia after lunch in healthy young individuals. British Journal of Nutrition. 2019;122(4):431-40.

37. Park H, Shin D, Lee KW. Association of main meal frequency and skipping with metabolic syndrome in Korean adults: a cross-sectional study. Nutrition Journal. 2023;22(1):1-11.

38. Basolo A, Bechi Genzano S, Piaggi P, et al. Energy balance and control of body weight: Possible effects of meal timing and circadian rhythm dysregulation. Nutrients. 2021;13(9):3276.

39. Xiao Q, Garaulet M, Scheer FA. Meal timing and obesity: interactions with macronutrient intake and chronotype. International journal of obesity. 2019;43(9):1701-11.

40. Thomas EA, Zaman A, Cornier M-A, et al. Later meal and sleep timing predicts higher percent body fat. Nutrients. 2020;13(1):73.

41. Dashti HS, Gómez-Abellán P, Qian J, et al. Late eating is associated with cardiometabolic risk traits, obesogenic behaviors, and impaired weight loss. The American journal of clinical nutrition. 2021;113(1):154-61.

42. Jakubowicz D, Barnea M, Wainstein J, et al. High caloric intake at breakfast vs. dinner differentially influences weight loss of overweight and obese women. Obesity. 2013;21(12):2504-12.

43. Imamura M, Sasaki H, Shinto T, et al. Association between Na, K, and lipid intake in each meal and blood pressure. Frontiers in nutrition. 2022;9:853118.

44. Mirghani H. The effect of breakfast skipping and late night eating on body mass index and glycemic control among patients with type 2 diabetes mellitus. Cureus. 2021;13(6).

45. Zhang X, Wu Y, Na M, et al. Habitual night eating was positively associated with progress of arterial stiffness in Chinese adults. Journal of the American Heart Association. 2020;9(19):e016455.

46. Eom H, Lee D, Cho Y, et al. The association between meal regularity and weight loss among women in commercial weight loss programs. Nutrition Research and Practice. 2022;16(2):205-16.

47. Ashakiran, Deepthi R. Fast foods and their impact on health. Journal of Krishna Institute of Medical Sciences University. 2012;1(2):7-15.

48. Mirmiran P, Moslehi N, Hosseinpanah F, et al. Dietary determinants of unhealthy metabolic phenotype in normal weight and overweight/obese adults: Results of a prospective study. International Journal of Food Sciences and Nutrition. 2020;71(7):891-901.

49. Mohammadbeigi A, Asgarian A, Moshir E, et al. Fast food consumption and overweight/obesity prevalence in students and its association with general and abdominal obesity. Journal of preventive medicine and hygiene. 2018;59(3):E236.

50. AlTamimi JZ, Alshwaiyat NM, Alkhalidy H, et al. Prevalence of Fast Food Intake among a Multi-Ethnic Population of Young Men and Its Connection with Sociodemographic Determinants and Obesity. International Journal of Environmental Research and Public Health. 2022;19(22):14933.

51. Odegaard AO, Koh WP, Yuan J-M, et al. Western-style fast food intake and cardiometabolic risk in an Eastern country. Circulation. 2012;126(2):182-8.

52. Popa AR, Vesa CM, Uivarosan D, et al. Cross-sectional study regarding the association between sweetened beverages intake, fast-food products, body mass index, fasting blood glucose and blood pressure in the young adults from North-western Romania. Rev Chim. 2019;70(1):156-60.

53. Alsabieh M, Alqahtani M, Altamimi A, et al. Fast food consumption and its associations with heart rate, blood pressure, cognitive function and quality of life. Pilot study. Heliyon. 2019;5(5).

54. Fong TCT, Ho RTH, Yip PSF. Effects of urbanization on metabolic syndrome via dietary intake and physical activity in Chinese adults: Multilevel mediation analysis with latent centering. Social Science & Medicine. 2019;234:112372.

55. Asghari G, Yuzbashian E, Mirmiran P, et al. Fast food intake increases the incidence of metabolic syndrome in children and adolescents: Tehran lipid and glucose study. PloS one. 2015;10(10):e0139641.

56. Bahadoran Z, Mirmiran P, Hosseini-Esfahani F, et al. Fast food consumption and the risk of metabolic syndrome after 3-years of follow-up: Tehran Lipid and Glucose Study. European journal of clinical nutrition. 2013;67(12):1303-9.

57. Hosseini Z, Rostami M, Whiting SJ, et al. Fast-Food Dietary Pattern Is Linked to Higher Prevalence of Metabolic Syndrome in Older Canadian Adults. Journal of Nutrition and Metabolism. 2021;2021.

58. Belobrajdic DP, Bird AR. The potential role of phytochemicals in wholegrain cereals for the prevention of type-2 diabetes. Nutrition journal. 2013;12(1):62.

59. Ma Y, Ailawadi KL, Grewal D. Soda versus cereal and sugar versus fat: drivers of healthful food intake and the impact of diabetes diagnosis. Journal of Marketing. 2013;77(3):101-20.

60. Amiot-Carlin M-J. Consommation des fruits et légumes: quels avantages, quels risques? Revue du Praticien (La). 2019;69(2):139-43.

61. Chen Z, Glisic M, Song M, et al. Dietary protein intake and all-cause and cause-specific mortality: results from the Rotterdam Study and a meta-analysis of prospective cohort studies. Springer; 2020. p. 411-29.

62. Pfeiffer AF, Pedersen E, Schwab U, et al. The effects of different quantities and qualities of protein intake in people with diabetes mellitus. Nutrients. 2020;12(2):365.

63. Rice BH, Quann EE, Miller GD. Meeting and exceeding dairy recommendations: effects of dairy consumption on nutrient intakes and risk of chronic disease. Nutrition reviews. 2013;71(4):209-23.

64. Thorning TK, Raben A, Tholstrup T, et al. Milk and dairy products: good or bad for human health? An assessment of the totality of scientific evidence. Food & nutrition research. 2016;60(1):32527.

65. Quann EE, Fulgoni VL, Auestad N. Consuming the daily recommended amounts of dairy products would reduce the prevalence of inadequate micronutrient intakes in the United States: diet modeling study based on NHANES 2007–2010. Nutrition journal. 2015;14:1-11.

66. Drouin-Chartier J-P, Hernández-Alonso P, Guasch-Ferré M, et al. Dairy consumption, plasma metabolites, and risk of type 2 diabetes. The American journal of clinical nutrition. 2021;114(1):163-74.

67. Feng Y, Zhao Y, Liu J, et al. Consumption of Dairy Products and the Risk of Overweight or Obesity, Hypertension, and Type 2 Diabetes Mellitus: A Dose–Response Meta-Analysis and Systematic Review of Cohort Studies. Advances in Nutrition. 2022;13(6):2165-79.

68. Torres-Gonzalez M, Bradley BHR. Whole-milk dairy foods: biological mechanisms underlying beneficial effects on risk markers for cardiometabolic health. Advances in Nutrition. 2023.

	Mea	1			
Variables	Male (n=1170)	Female (n=1512)	<i>p</i> -value*		
Age (year)	44.7 ± 19.4	43.2 ± 19.7	0.045		
Weight (Kg)	72.1 ± 16.5	67.6 ± 15.9	< 0.001		
Height (cm)	168.8 ± 15.0	160.0 ± 10.6	< 0.001		
Waist circumference (cm)	83.4 ± 15.4	77.2 ± 21.5	< 0.001		
Hip circumference (cm)	96.2 ± 17.5	89.0 ± 23.8	< 0.001		
Body Mass Index (kg/m ²)	25.2 ± 6.4	26.3 ± 5.9	< 0.001		
	n (%)				
Age groups					
Children (8-12)	58 (5.0)	68 (4.5)			
Adolescent (13-19)	84 (7.2)	129 (8.5)	0.420		
Adults (20-64)	832 (71.1)	1086 (71.7)	0.430		
Older adults (>65)	196 (16.8)	231 (15.3)			
Educational level					
High-School or less	565 (51.7)	869 (62.0)			
Diploma	192 (17.6)	213 (15.2)	< 0.001		
University level or higher	335 (30.7)	320 (22.8)			
Job					
No work	611 (53.1)	1290 (78.5)	<0.001		
Work	539 (46.9)	184 (12.5)	<0.001		
Marital Status					
Married	793 (68.2)	981 (65.1)			
Single	300 (25.8)	325 (21.6)	-0.001		
Widowed	51 (4.4)	169 (11.2)	<0.001		
Divorced	19 (1.6)	33 (2.2)			
Smoking Status					
Smoker	213 (74.7)	164 (35.7)			
Non-Smoker	12 (4.2)	78 (17.0)	<0.001		
Negative Smoker	20 (7.0)	156 (33.9)	<0.001		
Ex-Smoker	40 (14.0)	62 (13.5)			
Daily Physical Activity					
No	827 (70.9)	1247 (82.6)			
Yes	294 (25.2)	216 (14.3)	< 0.001		
None	45 (3.9)	46 (3.0)			
BMI categories					
normal weight	570 (50.9)	605 (41.8)	<0.001		
overweight	421 (37.6)	529 (36.5)			
obese	128 (11.4)	314 (21.7)			

Table.1 General characteristics (n=2682)

**p*<0.05 is considered significant. BMI: body mass index.

	Having	·····*		
	No	Yes	<i>p</i> -value*	
Skipped Meal				
Dinner	208 (13.0)	156 (15.3)		
Lunch	21 (1.3)	9 (0.9)	0.021	
Breakfast	299 (18.6)	174 (17.0)	0.231	
I don't Skip meals	1078 (67.1)	683 (66.8)		
Number of Meals	• • •	i		
one meal	14 (0.9)	24 (2.3)		
two meals	504 (31.1)	289 (28.3)	0.000	
three meals	1012 (62.5)	657 (64.2)	0.009	
> three meals	88 (5.4)	53 (5.2)		
Number of Snacks				
one snack	340 (21.0)	234 (22.8)		
two snacks	671 (41.4)	397 (38.7)		
three snacks	336 (20.8)	220 (21.5)	0.165	
four snacks	44 (2.7)	15 (1.5)	0.165	
> four snacks	18 (1.1)	15 (1.5)		
No snacks	210 (13.0)	144 (14.0)		
Late Food Intake				
Yes	359 (23.0)	213 (21.5)	0.260	
No	1204 (77.0)	780 (78.5)	0.309	
Fast Food Eating				
none	257 (16.0)	305 (30.2)		
Less than once /week	244 (15.2)	195 (19.3)		
1-5 times/week	1089 (67.9)	509 (50.4)	< 0.001	
once daily	13 (0.8)	1 (0.1)		
twice or more daily	2 (0.1)	0 (0.0)		
Most Consumed Food Grou	p			
legumes	42 (2.6)	23 (2.2)		
Cereals	1047 (65.1)	622 (60.7)		
Dairy products	47 (2.9)	17 (1.7)	<0.001	
Vegetables	76 (4.7)	105 (10.2)	<0.001	
Fruits	38 (2.4)	26 (2.5)		
Protein	358 (22.3)	232 (22.6)		
Most Missed Food Group				
legumes	373 (23.2)	258 (25.3)		
Cereals	74 (4.6)	49 (4.8)		
Dairy products	751 (46.7)	413 (40.6)	<0.001	
Vegetables	140 (8.7)	77 (7.6)		
Fruits	117 (7.3)	54 (5.3)		
Protein	152 (9.5)	167 (16.4)		

Table 2. Meal frequency and eating behavior among the study populationbased on having diseases.

*p < 0.05 is considered significant.

	Males		Females			
Variables	Having	Disease	<i>p</i> -	Having Disease		
	No	Yes	value*	No	Yes	<i>p</i> -value*
Skipped Meal				•	•	
Dinner	94 (12.9)	43 (10.5)		114 (13.0)	113 (18.5)	
Lunch	11 (1.5)	2 (0.5)	0.262	10(1.1)	7 (1.1)	0.019
Breakfast	122 (16.7)	73 (17.8)	0.263	176 (20.1)	100 (16.4)	
I don't Skip meals	503 (68.9)	291 (71.1)		574 (65.7)	390 (63.9)	
Number of Meals	· · · ·	• <u>·</u> ···		•	• • • •	•
one meal	3 (0.4)	7 (1.7)		11 (1.3)	17 (2.8)	
two meals	210 (28.5)	88 (21.5)	0.010	293 (33.3)	201 (33.0)	0.191
three meals	487 (66.1)	294 (71.7)	0.010	524 (59.6)	360 (59.0)	
> three meals	37 (5.0)	21 (5.1)		51 (5.8)	32 (5.2)	
Number of Snacks						
one snack	150 (20.3)	78 (18.9)		189 (21.5)	156 (25.6)	
two snacks	299 (40.5)	157 (38.1)		371 (42.2)	239 (39.2)	
three snacks	182 (24.7)	117 (28.4)	0.602	154 (17.5)	102 (16.7)	0.110
four snacks	17 (2.3)	7 (1.7)	0.005	27 (3.1)	8 (1.3)	0.119
> four snacks	10 (1.4)	9 (2.2)		8 (0.9)	6 (1.0)	
No snacks	80 (10.8)	44 (10.7)		130 (14.8)	99 (16.2)	
Late Food Intake						
Yes	168 (23.7)	83 (21.0)	0.308	191 (22.4)	128 (21.5)	0.671
No	541 (76.3)	312 (79.0)	0.308	661 (77.6)	468 (78.5)	
Fast Food Eating						
none	115 (15.8)	106 (26.1)		142 (16.2)	199 (33.1)	
Less than once /week	101 (13.9)	55 (13.5)		143 (16.4)	140 (23.3)	
1-5 times/week	501 (68.7)	244 (60.1)	< 0.001	586 (67.0)	262 (43.6)	< 0.001
once daily	11 (1.5)	1 (0.2)		2 (0.2)	0 (0.0)	
twice or more daily	1 (0.1)	0 (0.0)		1 (0.1)	0 (0.0)	
Most Consumed Food	Group					
legumes	13 (1.8)	8 (1.9)		29 (3.3)	15 (2.5)	
Cereals	479 (65.1)	250 (60.8)		567 (64.9)	369 (60.4)	0.014
Dairy products	24 (3.3)	8 (1.9)	<0.001	24 (2.7)	9 (1.5)	
Vegetables	26 (3.5)	45 (10.9)	<0.001	50 (5.7)	60 (9.8)	
Fruits	7 (1.0)	6 (1.5)		31 (3.5)	20 (3.3)	
Protein	187 (25.4)	94 (22.9)		173 (19.8)	138 (22.6)	
Most Missed Food Group						
legumes	175 (23.9)	93 (22.7)		201 (22.9)	165 (27.3)	
Cereals	35 (4.8)	15 (3.7)		39 (4.4)	34 (5.6)	
Dairy products	360 (49.2)	167 (40.7)	<0.001	390 (44.5)	244 (40.3)	0.002
Vegetables	53 (7.2)	40 (9.8)	~0.001	86 (9.8)	37 (6.1)	0.002
Fruits	62 (8.5)	27 (6.6)		56 (6.4)	26 (4.3)	ļ
Protein	47 (6.4)	68 (16.6)		105 (12.0)	99 (16.4)	

Table 3. Meal frequency and eating behavior among the study population based on having diseases based on gender.

**p*<0.05 is considered significant.

Moola	Having	n voluo*			
Ivieais	No	Yes	<i>p</i> -value		
	Total				
Time of breakfast intake					
Morning eating	611 (37.7)	445 (43.4)			
Non-morning eating	799 (49.3)	476 (46.4)	0.006		
Skipping breakfast	211 (13.0)	105 (10.2)	(10.2)		
What time do you have lunch?	• · · · ·	• • • •			
Between 1:00–6:00 p.m.	1498 (92.4)	967 (94.2)			
Evening eating (After 6:00 p.m.)	54 (3.3)	14 (1.4) 0.008			
Skipping lunch	69 (4.3)	45 (4.4)	-		
What time do you have dinner?					
Between 6:00–9:00 p.m.	930 (57.4)	623 (60.7)			
Night eating (After 9:00 p.m.)	503 (31.0)	268 (26.1)	0.022		
Skipping Dinner	188 (11.6)	135 (13.2)			
	Males	• • • • •			
Time of breakfast intake					
Morning eating	290 (39.2)	186 (45.1)			
Non-morning eating	364 (49.3)	188 (45.6)	0.120		
Skipping breakfast	85 (11.5)	38 (9.2)			
What time do you have lunch?					
Between 1:00–6:00 p.m.	679 (91.9)	388 (94.2)			
Evening eating (After 6:00 p.m.)	28 (3.8)	2 (0.5)	0.003		
Skipping lunch	32 (4.3)	22 (5.3)			
What time do you have dinner?					
Between 6:00–9:00 p.m.	406 (54.9)	235 (57.0)			
Night eating (After 9:00 p.m.)	253 (34.2)	136 (33.0)	0.771		
Skipping Dinner	80 (10.8)	41 (10.0)			
F	`emales				
Time of breakfast intake					
Morning eating	321 (36.5)	259 (42.4)			
Non-morning eating	434 (49.3)	286 (46.8)	0.031		
Skipping breakfast	125 (14.2)	66 (10.8)			
What time do you have lunch?					
Between 1:00–6:00 p.m.	817 (92.8)	577 (94.4)			
Evening eating (After 6:00 p.m.)	26 (3.0)	12 (2.0)	0.403		
Skipping lunch	37 (4.2)	22 (3.6)			
What time do you have dinner?					
Between 6:00–9:00 p.m.	522 (59.3)	387 (63.3)			
Night eating (After 9:00 p.m.)	250 (28.4)	131 (21.4)	0.006		
Skipping Dinner	108 (12.3)	93 (15.2)			

 Table 4. Main meal timing prevalence among the study population.

*p<0.05 is considered significant.