Original Article

Subcutaneous and visceral fat volumes measured by MRI and their relationships with nutrient intakes among adults

Reema F Tayyem PhD[1], Ali M Al-Radaideh PhD[2], Shatha S Hammad PhD[1], Sabal Al-Hajaj BSc[3], Sabika S Allehdan MSc[1], Lana M Agraib MSc[1], Kholoud I Al-Fayomi BSc[2], Amer A Malkawi BSc[4], Nawal S Hijjawi PhD[5]

[1]*Department of Nutrition and Food Technology, Faculty of Agriculture, The University of Jordan, Amman, Jordan*
[2]*Department of Medical Imaging, Faculty of Allied Health Sciences, The Hashemite University, Zarqa, Jordan*
[3]*Department of Nutrition, King Hussein Medical Center, Jordanian Royal Medical Services, Amman-Jordan*
[4]*Department of Radiology, King Hussein Medical Center, Jordanian Royal Medical Services, Amman-Jordan*
[5]*Department of Medical Laboratory Sciences, Faculty of Allied Health Sciences, The Hashemite University, Zarqa, Jordan*

Background and Objectives: Types and amounts of nutrients may influence the volume of subcutaneous adipose tissue (SAT) and visceral adipose tissue (VAT). This study targeted to investigate the relationship between SAT and VAT volumes and macro- and micronutrients intake among adults. **Methods and Study Design:** Data were collected via a private face-to-face interview, in which diet history was obtained using validated quantitative food frequency questionnaire. The different fat volumes were assessed using magnetic resonance imaging (MRI) scanning. **Results:** Participants with the lowest VAT volume had the highest intake of saturated fats, monounsaturated fatty acids and omega-3 and omega-6 fatty acids ($p<0.05$). VAT volume was significantly associated with the highest level of total energy and energy from carbohydrate consumption among participants while significantly associated with the lowest energy intake from fat among participants ($p=0.013$). There was a significant relationship with the highest consumption of total carbohydrate, soluble fiber, and insoluble fiber and VAT volume ($p<0.05$). Participants in the highest VAT volume had significantly the highest intake of vitamin A, β-carotene, and copper. **Conclusions:** This study underscores the importance of quantifying depot-specific body fat and highlights the unique responsiveness of various fat depots to dietary intake.

Key Words: macronutrients, micronutrients, subcutaneous fat, visceral fat, magnetic resonance imaging

INTRODUCTION

Obesity is an accelerating health problem worldwide. It is defined as an abnormal or excessive fat accumulation that may impair health. In 2014, more than 1.9 billion adults, 18 years and older, were overweight and over 600 million were obese.[1] In Jordan, prevalence of overweight and obesity among adults was estimated to be 30.5 and 35.9 %, respectively.[2] Obesity is associated with an increased risk of a number of co-morbidities such as hypertension, dyslipidemia, cardiovascular disease,[3] type 2 diabetes[4] and cancer.[5]

  Body mass index and waist circumference are the two main measurements commonly used to assess body fat, but these anthropometric measures cannot provide information regarding the anatomical location and distribution of stored excess fat.[6,7] However, the use of imaging techniques such as computed tomography and magnetic resonance imaging (MRI) has been characterized as an in-

credible advance in our ability to accurately and reliably quantify individual differences in body fat distribution and to selectively distinguish specific abdominal fat depots.[8,9] Two types of fat depots have been recognized in the android region, subcutaneous adipose tissue (SAT) and visceral adipose tissue (VAT). SAT is the adipocyte that lies just beneath the skin, while adipocyte that accumulates around the vital organs in the abdominal cavity is known as VAT. Not all adipocytes in android region induce the same degree of pathogenesis; the association

between the adverse metabolic risk profile and VAT was found to be about 2-folds stronger than that for SAT.[10] Thus, quantifying total and regional fat mass as well as differentiating between the types of fat depots provide a clear picture regarding the pathogenesis of obesity and therefore, better insight to disease risk.

SAT and VAT appear to be differentially influenced by dietary factors.[9,11-13] It has been proposed that diet can explain more of the variation in VAT than in SAT,[13] and lifestyle modification has the ability to reduce VAT more than SAT.[14] For instance, several studies have revealed associations between dietary fatty acids composition and visceral fat accumulation.[15,16] A 5% increase in energy intake from animal protein substituted for carbohydrate, monounsaturated fatty acids, and polyunsaturated fatty acids was reported to be associated with higher visceral adiposity index changes after three years of follow-up among Iranian adults.[9] The consumption of high dietary protein, adjusted for total caloric intake was associated with VAT among a group of young adult Saudi females independent of body weight.[17] Chaput et al (2014) revealed that low calcium and micronutrient intake could be considered as predictor of excess body weight and gains in weight and adiposity over time.[18]

Jordan is considered one of the Middle East countries that witnesses a dramatic transition from its Mediterranean dietary and lifestyle pattern towards Western pattern. This transition is accompanied by an increase in the prevalence of obesity and many other related diseases. Motivated by the current evidence that supports the associations between diet and body fat deposition and distribution, this study aimed at investigating the relationship between subcutaneous and visceral fat volumes (measured using MRI) and the intake of nutrients among Jordanian adults.

METHODS
Study design and participants
In this cross-sectional study, a total of 167 apparently healthy persons agreed to participate in the study. Participants were recruited from Royal Medical Services (RMS) personnel (including the security manpower, hospital cleaners, and employees in administrative positions). Eighty three males and 84 females (age range 20–51) were enrolled in the study during the period of October 2014 to July 2015. Eligibility criteria to participate in the study were: being Jordanian and above 18 years old and free from any chronic disease (self-reported). Pregnant and lactating women and persons suffering from eating disorders or having any disease were excluded. Response rate for dietary assessment and anthropometric measurements were about 90.0%. All participants were asked to sign a formal consent according to the medical center ethics approval. The study was approved by the Institutional Review Board Ethics Committee of RMS; the ethical approval number of the study was 10205/2014. All participants completed the MRI safety questionnaire before participating in this study.

Magnetic resonance imaging (MRI) measurement
Abdominal MRI was performed on a 3T Siemens Trio MR system (Siemens Medical Systems, Erlangen, Germany), equipped with 4-channel phase-array body coil. The acquired axial slices covered the region between the level above of the diaphragm and the head of the femur in the supine position and full expiration. The imaging protocol included in-phase (IP) and out-phase (OP), non-enhanced T1 weighted sequence with the following imaging parameters; repetition time (TR=5 ms), echo time (TEOP=1.225 ms) and (TEIP=2.45 ms), flip angle (FA=10o), slice thickness (ST=5 ms), 80 slices, 256 x 192 matrix size, 380 x 285 mm^2 field of view, number of signal averages (NSA=1), acceleration factor of 2, and scan time=18 seconds. The MRI scanner calculated the "fat-only" and "water-only" images from this sequence as follows:
"Fat" image = [IP – OP] /2; "Water" image = [IP + OP]/2

Image post processing and analysis
"Fat" MR images were imported into image analysis software (SliceOmatic, Tomovision Inc., Montreal, Canada) in their standard formats (Digital Imaging and Communications in Medicine; DICOM) to segment the subcutaneous and visceral fat tissues. Slices covering the region between the top of the diaphragm and the top of the first sacral vertebra (S1) were used to segment and analyze the subcutaneous and visceral fat tissues by an expert in image analysis. Each segmented tissue was saved as a separate "tag" and the total volumes of SAT and VAT were calculated from all analyzed slices and saved into a separate file for further analysis. The model and method employed to segment the various tissues is fully described and illustrated elsewhere.[19] "Fat" images were also used to measure the waist circumference at a level just below the lower costal margin.

Dietary assessment
A validated Arabic quantitative Food Frequency Questionnaire (FFQ) was used for dietary assessment.20 The FFQ questions tracked the information on the dietary history of study participants. The FFQ and 24-hour recalls produced similar agreement percentages ranging between 25.5% and 43.6%. Mean energy-adjusted reliability coefficients ranged from 0.695 to 0.943. A Cronbach's α for the total FFQ items of .857 was recorded. Many studies have used FFQ along with MRI to assess body fat volumes and their relationship with the type of nutrient consumed.21,22 Trained dietitian asked participants, during face-to-face interviews, how frequently, on average, during the past year they had consumed one standard serving of specific food items in nine categories (<1/month, 2–3/month, 1–2/week, 3–4/week, 5–6/week, 1/day, 2–3/day, 4–5/day, or 6/day). Food lists in the modified FFQ questions were classified based on types of foods: 21 items of fruits and juices; 21 items of vegetables; eight items of cereals; nine items of milk and dairy products; four items of beans; 16 items of meat such as red meat (lamb and beef), chicken, fish, cold meat, and others; four items of soups and sauces; five items of drinks; nine items of snacks and sweets; and 14 items of herbs and spices.20 For better portion size estimation food models and standard measuring tools were used. To estimate macro- and micronutrients intake, the intakes of foods collected by FFQ were analyzed using dietary analysis software

(ESHA Food Processor SQL version 10.1.1; ESHA, Salem, OR, USA) with additional data on foods consumed in Jordan.[23]

Physical activity level

Physical activity level was determined using 7-day Physical Activity Recall (PAR).[24] The 7-Day PAR is a structured interview that depends on participant's recall of time spent engaging in physical activity over a seven day period of their usual days.[24] The number of hours spent in different activity levels were collected and transformed to metabolic equivalents (METs). The total physical activity MET minutes per week was calculated by summing the METs.[24]

Anthropometric measurements

All anthropometric measurements were carried out by trained dietitian at the morning between 8-10am while they were fasting. Waist circumference (WC) was measured by tape (WC_tape) at the narrowest level between the lowest rib and the iliac crest at the end of normal expiration in standing position.[25] In addition, waist circumference was measured from the MR images (WC_MRI) at a level just below the lower costal margin using an image analysis software (SliceOmatic, Tomovision Inc., Montreal, Canada) in a semi-automated approach. Body weight was measured to the nearest 0.1 kg, with minimal clothing and without shoes, using a calibrated scale (Tan-

ita, Model SC-331S, Japan).[25] Height was measured to the nearest 1 cm with participants in standing position without shoes using a calibrated portable measuring rod.[25] Body mass index (BMI) was calculated as the ratio of weight in kilograms to the square of height in meters.[25]

Statistical analysis

The data were analyzed using SPSS statistical package version 20. Energy, macronutrients and micronutrients were presented as mean ± standard error of mean (SEM). SAT, VAT and total fat were grouped into tertiles. Data presented in 3 tertiles to have a representative number of subjects in each tertile. Analysis of Covariance (ANCOVA) was used to assess the impact of energy, macronutrients and micronutrients intake on SAT, VAT, and total fat. Data was adjusted for energy, age, gender, physical activity and smoking. The Benjamini-Hochberg correction with critical value for a false discovery rate of 0.25 for multiple comparisons was used. Value of $p \leq 0.05$ was considered statistically significant.

RESULTS

Participants' anthropometric measurements, SAT, VAT and total abdominal fat were published previously in another publication.[26] The authors reported significant statistical differences ($p<0.05$) between males and females in height, weight, and waist circumference, with males showing higher values than females (Table 1). Regarding

Table 1. Anthropometric and socioeconomic characteristics of the study sample based on gender.[26]

Parameter	Male			Female			p-value	Total		
	N	Mean	SEM	N	Mean	SEM		N	Mean	SEM
Height (cm)	77	172	0.81	81	159	0.66	0.001	157	165	0.75
Weight (kg)	77	79.1	1.7	81	68.7	1.4	0.001	157	73.8	1.5
BMI (kg/m²)	77	26.7	0.59	81	27.5	0.63	0.255	157	27.1	0.43
Waist circumference (cm) by										
Tape	65	93.8	1.4	75	86.8	1.3	0.017	140	90.0	0.99
MRI	83	90.5	1.3	84	83.8	1.4	0.001	167	87.1	0.97
Subcutaneous fat (cm³)	83	3779	220.4	84	5223	274	0.001	167	4505	184
Visceral fat (cm3)	83	2967	171.3	84	1882	100	0.001	167	2421	107
Total abdominal fat (cm³)	83	6746	356.4	84	7104	358	0.591	167	6926	252
Physical activity (MET)	52	23081	2449.2	63	20464	1477	0.344	115	21647	1370
BMI categories, N (%)										
Normal	35 (45.5)			35(43.8)				70 (44.6)		
Overweight	23 (29.9)			20 (25.0)			0.458	43 (27.4)		
Obese	19 (24.6)			25 (31.2)				44 (28.0)		
Marital status, N (%)										
Married	31(40.8)			24 (29.6)				55 (35.0)		
Single	45 (59.2)			53 (65.4)			0.053	98 (62.4)		
Divorced	-			4 (4.9)				4 (2.5)		
Education, N (%)										
Illiterate	1 (1.5)			-			0.761	1 (0.6)		
Primary and secondary education	14 (20.6)			22 (28.2)				36 (21.4)		
Diploma	12 (17.6)			13 (16.7)				25 (14.9)		
Bachelor	39 (57.4)			42 (53.8)				101 (61.3)		
Master and PhD	2 (2.9)			1 (1.3)				3 (1.8)		
Occupation, N (%)										
Yes	43 (64.2)			45 (57.7)			0.063	88 (60.7)		
No	24 (35.8)			33 (42.3)				57 (39.3)		
Current smoking, N (%)										
Yes	34 (50.9)			10 (13)			0.004	44 (30.6)		
No	33 (49.1)			67 (87)				100 (69.4)		

SEM: standard error of the mean; BMI: body mass index.

the physical activity level, the results of this study did not show any significant difference between male and female participants. While the SAT tissue volume in males was found to be significantly (p-value=0.001) lower (3779.4 cm^3) than in females (5222.7 cm^3), VAT tissue volume was significantly (p-value=0.001) higher (2967.1 cm^3) in males than in females (1881.7 cm^3). However, the total of participants showed insignificant differences in SAT, VAT and total abdominal fat between males and females due to the equal participation of both genders.[26]

Table 2 shows the relationship between participants' daily total energy intake and macronutrients' contribution to energy intake in relation to their body fat fractions. Participants in the highest tertile of visceral fat had the highest consumption of energy from carbohydrate (p<0.05). In contrast, visceral fat was significantly associated with the highest energy intake from fat among participants in the lowest tertile (p=0.013). There was no significant relationship between protein consumption across different tertiles of visceral fat. We also found no statistically significant relationship with total energy and macronutrient distribution consumptions across all tertiles of subcutaneous fat and total fat. After using Benjamini-Hochberg with a false discovery rate of 25%, the participants in the lowest tertile of total fat was also found to have a significantly higher percent of energy from fats compared to the highest tertile.

Table 3 presents the participants' daily intake of the different macronutrients through different tertiles of body fat fractions. The consumption of carbohydrate, soluble fiber, and insoluble fiber was significantly higher among participants in the highest tertile of visceral fat (p<0.05). Conversely, visceral fat was significantly associated with the highest intake of total fats, saturated fats, monounsaturated fats, omega-6 fatty acids and omega-3 fatty acids among participants in the lowest tertile.

Participants in the highest tertile of visceral fat had significantly the highest intake of copper. There was no statistically significant relationship with the studied fat soluble and water soluble vitamins as well as calcium, iron and zinc intake through all tertiles of body fat fractions (Table 4).

DISCUSSION

The present study was conducted to find the relationship between the main nutrients intake, and SAT and VAT in a convenient sample of Jordanian adults. Body fat tends to accumulate in two main regions, abdominal and peripheral, the former is considered a stronger predictor of disease risks. An increase in cardiovascular risk factors from no to at least 3 factors corresponding to an increase of less than 1 kg of abdominal fat.[27] The two fat depots in the abdominal region differ in their anatomical and physiological properties, consequently, induce different degrees of pathogenicity.[28,29] Although, VAT constitutes only a small proportion of total body fat, its bad reputation persuades by its clinical impact.[28,29] VAT is more cellular, vascular, innervated, and contains a larger number of inflammatory and immune cells as compared to SAT.[28] There are more glucocorticoid and androgen receptors in VAT than SAT, also VAT is more sensitive to lipolysis and lipogenesis and more insulin-resistant compared to

other fat depots.[30] Therefore, VAT can be identified as metabolically active and pathogenic fat depot that drains several adipokines directly into the portal vein.[28] The current evidence supports a strong and independent association of VAT depot, rather than SAT, with chronic diseases and metabolic abnormalities. In addition, VAT has been influenced by genetics, gender, ethnicity, age, and modifiable factors including physical activity and diet.[31,32]

This study successfully highlighted a few significant associations between food consumption and visceral fat accumulation. The results show a significantly greater consumption of carbohydrate among subjects with the highest visceral fat deposition, which might be explained by two possible scenarios. First, is the activation of de novo lipogenesis; the process of lipid synthesis from dietary lipid or non-lipid precursors that takes place primarily in the liver. Despite the fact that de novo lipogenesis lightly contributes to fatty acid pool,[34] this process has been implicated in modulating disease risks and obesity development particularly in a situation of overfeeding.[35,36] De novo lipogenesis has been reported to be upgraded to a higher extent following a high carbohydrate diet, compared to a high-fat diet.[37] Similar pattern of consumption was found among participants in the highest tertile of VAT, where they were found to consume higher amounts of carbohydrate and energy as compared to participants in the lowest tertile. Also, adipocyte has been reported to contribute to de novo lipogenesis process,[38] therefore, even in a case of insulin sensitivity, visceral adipose tissue as being more insulin resistant than SAT, would be less likely to respond to the insulin-induced deactivation of de novo lipogenesis. Additionally, the accumulation of visceral fat has been implicated in insulin resistance,[39] which, in turn, would further stimulate the levels of hepatic lipogenesis, thereby, fat deposition in various fat depots.[40] The second suggested mechanism for the increased total and visceral fat masses associated with high carbohydrate consumption is that the higher carbohydrate consumption, mainly if based on high glycemic index food, would augment postprandial excursions of glucose and insulin. Greater levels of glucose would modulate fuel selection, thereby, increasing carbohydrate, rather than fat, oxidation and shutting down the lipolysis.[41,42] In such case, the spared fat will be further accumulated in adipocyte.

Higher consumption of insoluble fiber was found to be associated with higher visceral and total body fat. The fermentation of insoluble fiber produces short chain fatty acids (SCFA), which contribute to total energy and have been proposed to affect energy homeostasis, and therefore, body weight.[43] Such situation is most likely to happen when high fiber consumption is accompanied with high energy intake. Short chain fatty acids have been also suggested to inhibit lipolysis which might also contribute to obesity.[44] The significant relationship between high soluble fiber consumption and body fat deposition was unexpected and it is hard to give a good explanation, however, the amount or duration of consumption might have not been sufficient to induce the favorable effect of soluble fiber on weight management. It has been previously reported that short-term fiber supplementation of an

Table 2. Total energy intake and the percentages of energy from macronutrients intakes across different tertiles of body fat fractions

Nutrients		Subcutaneous fat			Visceral fat			Total fat		
		Mean±SEM	p-value[*]	Benjamini-Hochberg p-value	Mean±SEM	p-value[*]	Benjamini-Hochberg p-value	Mean±SEM	p-value[*]	Benjamini-Hochberg p-value
Total energy (kcal)	T1	2695±133	0.206	0.409[‡]	2682±153	0.115	0.085[†]	2744±139	0.541	0.375[‡]
	T2	3046±121			2947±173			3036±199		
	T3	3188±167			3299±177			3149±167		
Percent energy from proteins	T1	12.2 ±0.32	0.390	0.409[‡]	11.9±0.33	0.873	0.968[‡]	12.3±0.34	0.407	0.386[‡]
	T2	12.5±0.41			12.1±0.41			12.2±0.4		
	T3	11.4±0.32			12.0±0.33			11.6±0.4		
Percent energy from carbohydrates	T1	61.8±1.14	0.358	0.418[‡]	61.8±1.2	0.013	0.046[†]	60.7±1.1	0.108	0.137[†]
	T2	62.2 ±12.6			61.2±1.0			62.9±1.3		
	T3	65.1±1.5			66.1±1.6			65.4±1.5		
Percent energy from fats	T1	29.5±0.99	0.679	0.537[‡]	29.4±1.0	0.013	0.043[†]	30.2±0.98	0.270	0.205[†]
	T2	28.6±1.0			29.9±0.9			28.3±0.99		
	T3	27.3±1.2			25.9±1.2			26.9±1.2		

[†]Significant based on Benjamini-Hochberg p-value at false discovery rate ≤0.25.
[‡]Not significant based on Benjamini-Hochberg p-value at false discovery rate >0.25.
[*]Statistical significant difference (p<0.05). All data were adjusted for energy, age, gender, physical activity and smoking.

Table 3. Energy-adjusted intakes of macronutrients across different tertiles of body fat fractions

Nutrients		Subcutaneous fat			Visceral Fat			Total Fat		
		Mean±SEM	p-value[*]	Benjamini-Hochberg p-value	Mean±SEM	p-value[*]	Benjamini-Hochberg p-value	Mean±SEM	p-value[*]	Benjamini-Hochberg p-value
Proteins (g)	T1	98.8±2.7	0.376	0.418[‡]	95.9±2.3	0.985	0.848[‡]	99.1±2.7	0.317	0.386[‡]
	T2	100±2.8			97.7±2.9			99.1±2.7		
	T3	93.4 ±2.5			98.8±2.8			94.2±2.6		
Total carbohydrates (g)	T1	506±8.7	0.196	0.366[‡]	511±11.8	0.008	0.041[†]	499±8.9	0.071	0.137[†]
	T2	521±13.4			507±8.9			526±13.1		
	T3	550.3±16.7			559±17.4			551±16.7		
Fiber (g)	T1	57.8±1.8	0.529	0.556[‡]	56.4±1.9	0.053	0.431[‡]	56.2±1.9	0.261	0.362[‡]
	T2	56.5±2.5			57.3±2.3			57.4±2.3		
	T3	60.6±1.9			61.2±2.1			61.3±2.0		
Soluble fiber (g)	T1	6.7±0.60	0.135	0.409[‡]	6.4±0.60	0.001	0.025[†]	6.3±0.60	0.024	0.137[†]
	T2	7.0±0.64			6.8±0.60			7.4±0.63		
	T3	8.5±0.62			9.0±0.62			8.6±0.61		

[†]Significant based on Benjamini-Hochberg p-value at false discovery rate ≤0.25.
[‡]Not significant based on Benjamini-Hochberg p-value at false discovery rate >0.25.
[*]Statistical significant difference (p<0.05). All data were adjusted for energy, age, gender, physical activity and smoking.

Table 3. Energy-adjusted intakes of macronutrients across different tertiles of body fat fractions (cont.)

Nutrients		Subcutaneous fat			Visceral Fat			Total Fat		
		Mean±SEM	*p*-value[*]	Benjamini-Hochberg *p*-value	Mean±SEM	*p*-value[*]	Benjamini-Hochberg *p*-value	Mean±SEM	*p*-value[*]	Benjamini-Hochberg *p*-value
Insoluble fiber (g)	T1	11.1±0.84	0.095	0.366[‡]	10.7±0.91	0.001	0.024[†]	10.6±0.87	0.006	0.137[†]
	T2	12.7±1.1			12.2±0.93			12.9±1.0		
	T3	14.6±0.90			15.4±1.0			14.9±1.0		
Total fats (g)	T1	118±3.71	0.352	0.418[‡]	118±4.6	0.006	0.024[†]	120±3.8	0.139	0.150[†]
	T2	112±4.9			119±3.3			111±4.8		
	T3	104±5.6			98.9±5.8			104±5.6		
Saturated fats (g)	T1	33.9±1.4	0.192	0.556[‡]	33.9±1.5	0.012	0.459[‡]	34.7±1.5	0.104	0.375[‡]
	T2	34.1±2.2			34.4±1.4			33.7±2.2		
	T3	30.7±2.1			30.4±2.7			30.3±2.0		
Monounsaturated fat (g)	T1	29.4±1.4	0.338	0.366[‡]	29.1±1.4	0.024	0.024[†]	29.4±1.5	0.174	0.137[†]
	T2	26.3±1.7			28.3±1.5			26.4±1.7		
	T3	23.6±1.7			21.9±1.8			23.5±1.7		
Trans fats (g)	T1	3.2±0.45	0.885	0.857[‡]	3.7±0.46	0.845	0.579[‡]	3.3±0.46	0.876	0.542[‡]
	T2	3.5±0.37			2.9±0.34			3.6±0.36		
	T3	3.2±0.35			3.2±0.37			2.9±0.35		
Cholesterol (mg)	T1	230±23.8	0.280	0.537[‡]	219±15.7	0.404	0.671[‡]	239 ±24.4	0.085	0.240[†]
	T2	217±19.2			225±28.6			216±19.2		
	T3	188±17.5			191±13.4			180±15.9		
Omega 6-fatty acids (g)	T1	21.9±1.5	0.275	0.366[‡]	21.7±1.7	0.040	0.024[†]	21.6±1.6	0.304	0.205[†]
	T2	17.5±1.9			19.8±1.6			17.4±1.9		
	T3	15.9±1.9			13.7±1.9			16.1±1.9		
Omega 3-fatty acids (g)	T1	1.7±0.12	0.135	0.366[‡]	1.7±0.13	0.044	0.024[†]	1.6±0.12	0.254	0.205[†]
	T2	1.3±0.17			1.4±0.14			1.3±0.16		
	T3	1.3±0.17			1.0±0.16			1.2±0.15		

[†]Significant based on Benjamini-Hochberg *p*-value at false discovery rate ≤0.25.
[‡]Not significant based on Benjamini-Hochberg *p*-value at false discovery rate >0.25.
[*]Statistical significant difference ($p < 0.05$). All data were adjusted for energy, age, gender, physical activity and smoking.

Table 4. Energy-adjusted intakes of micronutrients across different tertiles of body fat fractions

Nutrients		Subcutaneous fat			Visceral fat			Total fat		
		Mean±SEM	p-value	Benjamini-Hochberg p-value	Mean±SEM	p-value	Benjamini-Hochberg p-value	Mean±SEM	p-value	Benjamini-Hochberg p-value
Vitamin A (IU)	T1	18239±1815	0.869	0.654[‡]	17567±1700	0.103	0.053[†]	15837±0.02	0.122	0.137[†]
	T2	21285±2415			17605±2085			24487±2638		
	T3	20660±2339			25012±2583			19859±2256		
β-carotene (µg)	T1	7513±954	0.803	0.569[‡]	7009±834	0.066	0.041[†]	6219±707	0.115	0.137[†]
	T2	9521±1334			7732±1173			11003±1427		
	T3	9454±1327			11747±1467			9267±1310		
Vitamin D (µg)	T1	0.95±0.23	0.257	0.584[‡]	0.87±0.28	0.408	0.968[‡]	0.98±0.23	0.273	0.587[‡]
	T2	0.95±0.30			0.86±0.22			0.90±0.29		
	T3	0.63±0.11			0.80±0.16			0.65±0.12		
Vitamin E (mg)	T1	17.45±2.92	0.288	0.528[‡]	17.62±4.02	0.284	0.848[‡]	17.35±2.93	0.307	0.386[‡]
	T2	17.78±4.17			15.25±2.55			17.85±4.15		
	T3	11.75±1.53			14.10±2.45			11.77±1.58		
Vitamin K (µg)	T1	157±20.7	0.197	0.494[‡]	136±13.9	0.453	0.334[‡]	137±17.2	0.102	0.137[†]
	T2	269±70.4			241±68.3			300±70.3		
	T3	200±34.5			248±40.9			189±34.2		
Calcium (mg)	T1	1486±214	0.247	0.523[‡]	1535±321	0.406	0.914[‡]	1491±215	0.319	0.375[‡]
	T2	1669±347			1343±193			1675±350		
	T3	1121±122			1398±210			1110±112		
Copper (mg)	T1	1.30±0.8	0.094	0.418[‡]	1.23±0.08	0.018	0.046[†]	1.29±0.08	0.105	0.272[‡]
	T2	1.46±0.11			1.49±0.10			1.50±0.12		
	T3	1.56±0.09			1.60±0.10			1.53±0.09		
Iron (mg)	T1	33.32±3.69	0.502	0.569[‡]	33.48±5.58	0.456	0.848[‡]	33.10±3.75	0.465	0.507[‡]
	T2	33.22±5.78			28.91±3.22			33.32±5.70		
	T3	26.8±1.7			30.9±2.9			26.9±1.9		
Zinc (mg)	T1	21.2±3.2	0.313	0.494[‡]	21.6±4.4	0.246	0.737[‡]	21.1±3.2	0.314	0.375[‡]
	T2	20.6±4.6			17.5±2.8			20.4±4.6		
	T3	14.0±1.5			16.7±2.5			14.2±1.5		

[†]Significant based on Benjamini-Hochberg p-value at false discovery rate ≤0.25.
[‡]Not significant based on Benjamini-Hochberg p-value at false discovery rate >0.25.
[*]Statistical significant difference ($p<0.05$). All data were adjusted for energy, age, gender, physical activity and smoking.

ad-libitum, self-selected diet has not induced weight loss or changed the response of individuals to satiety and hunger perceptions.[45,46] A mechanism on how soluble fiber could enhance weight gain has been proposed through the gut microbiota.[47] Gut microbiota could contribute to obesity through improved energy extraction from diet by the conversion of dietary fiber to SCFA. The human intestinal microbiome is enriched in genes involved in the degradation of indigestible polysaccharides. Some of the formed SCFAs, mainly acetate and propionate, play an important role in lipid and glucose metabolism.[47] Acetate acts as a substrate for de novo lipogenesis in liver, whereas propionate can be utilized for gluconeogenesis. The conversion of fermentable dietary fiber to SCFA provides additional energy to the host which could promote obesity particularly visceral fats. Yet, some epidemiologic studies show that diets high in fiber rather prevent than promote obesity development. This may be due to the fact that SCFA are also ligands of free fatty acid receptors (FFAR).[47] Activation of FFAR leads to an increased expression and secretion of enteroendocrine hormones such as glucagon-like-peptide 1 or peptide YY which cause satiety. Indeed, further studies could be warranted to confirm the effect of fiber on body fat volumes.

The quality and quantity of dietary fat can affect obesity and chronic disease risks,[48] however, the quality of dietary fat has been suggested to have a stronger effect on body fat accumulation.[49,50] While an accumulating evidence shows a negative impact of saturated fatty acid intake on fat deposition,[51-53] monounsaturated- and polyunsaturated- fatty acids consumption was proven to have desirable effects on body weight and fat mass.[51,53,54] The overeating of saturated fatty acids stimulated hepatic and visceral fat storage, where excess energy from polyunsaturated fatty acids may promote lean tissue in healthy humans.[17] Likewise, including monounsaturated fat to the diet prevented visceral fat gain within an isocaloric design including other types of dietary fats.[55] The consumption of a monounsaturated fatty acid-rich high-fat diet (40% Kcal total fat and ~29% Kcal monounsaturated fatty acid) for 3 weeks resulted in a reduced android adiposity as compared to a high palmitic high-fat diet (40% Kcal total fat and ~16% Kcal palmitic acid).[50] The findings of this study support the importance of the quality of dietary fat. Subjects in the lowest tertile of visceral fat were found to consume a significantly higher amount of dietary fat, compared to subjects in the highest tertile of visceral fat, nevertheless, the unsaturated fat has contributed to the majority of the consumed fat. The favorable effects of unsaturated fat could be induced by directing the metabolism toward a pathway of oxidation rather than storage.[32,51] Similarly, the inverse relationship (significant or trend toward significance) between monounsaturated-, omega-3-, and omega-6- fatty acids consumption and subcutaneous and total body fat would further support the effectiveness of unsaturated fat consumption on modifying energy homeostasis and body fatness. Compared to saturated fatty acids, greater oxidation rates of polyunsaturated- and monounsaturated fatty acids augment energy expenditure levels, and therefore, reduce the propensity of fat deposition and might promote weight management.[51] Monounsaturated fatty acid (MUFA) has been

also suggested to regulate food intake by one of its metabolite, oleoylethanolamine, which influences metabolic and reward system and controls appetite sensation, and therefore, reduces energy intake.[51] Even though we didn't measure the level of oleoylethanolamine in the participants' food intake, reduced energy consumption accompanied by a high MUFA intake among participants in the lowest tertile of visceral fat could propose the influence of the oleoylethanolamine intake from MUFA on food intake. The present study found (data not displayed) significant inverse correlation ($r=0.498$, $p<0.0001$) between energy intake and MUFA consumption which is in agreement with the previously mentioned studies.

The detectable significant higher consumption of copper among participants in the highest tertile of VAT might reflect the higher consumption of carbohydrate and fiber among this group. In addition to being rich in carbohydrate and fiber, many fruits and vegetables are considered as good sources of copper e.g. dark leafy vegetables, dried fruits, and sweet potato. Up to our best knowledge, no single study has been found that investigating in-depth the relationship between SAT and VAT volumes and micronutrients intake among adults, which imparts our study to be the pioneer in this topic.

One of the main limitations in this study is the one year dietary recall made by FFQ, which may be affected by behavioral and dietary changes, memory and bias. However, we believe that because food selection and taste are mostly based on availability and habits that influence deliberate choices, including endemic cultural biases, we accept that the recall period of one year is very likely reflective of the previous years. Another limitation of the FFQ is that it doesn't consider the bioavailability of the consumed nutrients as in other dietary assessment methods. In addition, our sample size is small due to the limited time available to use the MRI scanner at the medical center. Therefore, we recommend conducting additional studies on a large-scale to generalize these findings and to evaluate the differences between male and female. We also should indicate that this study had no major selection bias in the participants' weight status. The prevalence of overweight and obesity among Jordanians has been found to be above 70% of both males and females adults aged above 25 years.[56]

In conclusion, the study found that consumption of carbohydrate (soluble and insoluble fibers) and fat (monounsaturated, omega-3 and omega-6 fatty acids) from the macronutrient and copper from the micronutrients can influence the amount of fat volumes. This study underscores the importance of quantifying depot-specific body fat and highlights the unique responsiveness of various fat depots to dietary intake. A large scale study might be needed to generalize the obtained results.

AUTHOR DISCLOSURES
The authors declare that they have no conflict of interests.

REFERENCES

1. WHO World Health Organization. Obesity and overweight. [2018/02/16]; Available from http://www.who.int/mediacentre/factsheets/fs311/en/.

2. Al-Nsour M, Zindah M, Belbeisi A, Hadaddin R, Brown DW, Walke H. Prevalence of selected chronic, noncommunicable disease risk factors in Jordan: results of the 2007 Jordan Behavioral Risk Factor Surveillance Survey. Prev Chronic Dis. 2012;9:E25.

3. Poirier P, Giles TD, Bray GA, Hong Y, Stern JS, Pi-Sunyer X, Eckel RH. Obesity and cardiovascular disease: pathophysiology, evaluation, and effect of weight loss: an update of the 1997 American Heart Association Scientific Statement on obesity and heart disease from the Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism. Circulation. 2006;113:898-918.. doi: 10.1161/CIRCULATIONAHA.106.171016

4. Wannamethee SG, Shaper AG. Weight change and duration of overweight and obesity in the incidence of type 2 diabetes. Diabetes Care. 1999;22:1266-72.

5. Ceschi M, Gutzwiller F, Moch H, Eichholzer M, Probst-Hensch NM. Epidemiology and pathophysiology of obesity as a cause of cancer. Swiss Med Wkly. 2007;137:50-6.

6. Green DJ. Is body mass index really the best measure of obesity in individuals? J Am Coll Cardiol. 2009;53:526. doi: 10.1016/j.jacc.2008.08.078.

7. Kaess BM, Pedley A, Massaro JM, Murabito J, Hoffmann U, and Fox CS. The ratio of visceral to subcutaneous fat, a metric of body fat distribution, is a unique correlate of cardiometabolic risk. Diabetologia. 2012;55:2622-30. doi: 10.1007/s00125-012-2639-5.

8. Despres JP. Body fat distribution and risk of cardiovascular disease an update. Circulation. 2012;126:1301-13. doi: 10.1161/CIRCULATIONAHA.111.067264.

9. Moslehi N, Ehsani B, Mirmiran P, Hojjat P, Azizi F. Association of dietary proportions of macronutrients with visceral adiposity index: non-substitution and iso-energetic substitution models in a prospective study. Nutrients. 2015;7:8859-70. doi: 10.3390/nu7105436.

10. Fox CS, Massaro JM, Hoffmann U, Pou KM, Maurovich-Horvat P, LiuCY et al. Abdominal visceral and subcutaneous adipose tissue compartments: association with metabolic risk factors in the Framingham Heart Study. Circulation. 2007;116:39-48. doi: 10.1161/CIRCULATIONAHA.106.675355.

11. McKeown NM., Troy LM, Jacques PF, Hoffmann U, O'Donnell CJ, Fox CS. Whole- and refined-grain intakes are differentially associated with abdominal visceral and subcutaneous adiposity in healthy adults: The Framingham Heart Study. Am J Clin Nutr. 2010;92:1165-71. doi: 10.3945/ajcn.2009.29106.

12. Kondoh T, Takase H, Yamaguchi TF, Ochiaia R, Katashima M, Katsuragi Y, Sakane N. Association of dietary factors with abdominal subcutaneous and visceral adiposity in Japanese men. Obes Res Clin Pract. 2014;8:e16-e25. doi: 10.1016/j.orcp.2012.07.005.

13. Fischer K, Pick JA, Moewes D, Nöthlings U. Qualitative aspects of diet affecting visceral and subcutaneous abdominal adipose tissue: a systematic review of observational and controlled intervention studies. Nutr Rev. 2015;73:191-215. doi: 10.1093/nutrit/nuu006.

14. Borel AL, Nazare, JA, Smith J, Almeras N, Tremblay A, Bergeron J, Poirier P, Després JP. Visceral and not subcutaneous abdominal adiposity reduction drives the benefits of a 1-year lifestyle modification program. Obesity. 2012;20:1223-33. doi: 10.1038/oby.2011.396.

15. Petersson H, Arnlov J, Zethelius B, Riserus U. Serum fatty acid composition and insulin resistance are independently associated with liver fat markers in elderly men. Diabetes Res Clin Pract. 2010;87:379-84. doi: 10.1016/j.diabres.2009.11.019.

16. Rosqvist F, Iggman, D, Kullberg J, Cedernaes, J, Johansson HE, Larsson A. Overfeeding polyunsaturated and saturated fat causes distinct effects on liver and visceral fat accumulation in humans. Diabetes. 2014;63:2356-68. doi: 10.2337/db13-1622.

17. Alissa, EM AlKadi HA. Visceral adiposity in Saudi females and its relationship to diet and serum adipocytokine levels. Int J Nutr Meta. 2011;39:114-22.

18. Chaput JP, Pérusse L, Després JP, Tremblay A, Bouchard C. Findings from the Quebec Family Study on the Etiology of Obesity: Genetics and environmental highlights. Curr Obes Rep. 2014;3:54-66. doi: 10.1007/s13679-013-0086-3.

19. Shen W, Chen J. Application of imaging and other noninvasive techniques in determining adipose tissue mass. Methods Mol Biol. 2008;456:39-54. doi: 10.1007/978-1-59745-245-8_3.

20. Tayyem RF, Abu-Mweis SS, Bawadi HA, Agraib L, Bani-Hani K. Validation of a food frequency questionnaire to assess macronutrient and micronutrient intake among Jordanians. J Acad Nutr Diet. 2014;114:1046-52. doi: 10.1016/j.jand.2013.08.019.

21. di Giuseppe R, Plachta-Danielzik S, Koch M, Nöthlings U, Schlesinger S, Borggrefe J, Both M, Müller HP, Kassubek J, Jacobs G, Lieb W. Dietary pattern associated with selenoprotein P and MRI-derived body fat volumes, liver signal intensity, and metabolic disorders. Eur J Nutr. 2019;58:1067-79. doi: 10.1007/s00394-018-1624-2

22. Wang Y, Davies-Tuck ML, Wluka AE, Forbes A, English DR, Giles GG, O'Sullivan R, Cicuttini FM. Dietary fatty acid intake affects the risk of developing bone marrow lesions in healthy middle-aged adults without clinical knee aged adult without clinical knee osteoarthritis: a prospective cohort study. Arthritis Res Ther. 2009;11:R63. doi: 10.1007/s00394-018-1624-2

23. Pellet P, Shadarevian S. The food composition tables for use in the Middle East. 3rd, Heidelberg: American University of Beirut, Lebanon; 2013.

24. Sallis JF, Haskell WL, Wood PD. Physical activity assessment methodology in the Five-City Project. Am J Epidemiol. 1985;121:91-106. doi: 10.1093/oxfordjournals.aje.a113987.

25. Lee RD, Nieman DC. Nutritional assessment, 6th ed. New York: McGraw Hill; 2012.

26. Al-Radaideh A, Tayyem R, Al-Fayomi K, Nimer N, Malkawi A, Al-Zu'bi R, AgraibL, Athamneh I, Hijjawi N. Assessment of abdominal fat using high-field magnetic resonance imaging and anthropometric and biochemical parameters. Am J Med Sci. 2016;352:593-602. doi: 10.1016/j.amjms.2016.09.009.

27. Wiklund P, Toss F, Weinehall L, Hallmans G, Franks PW, Nordstrom A, Nordstrom P. Abdominal and gynoid fat mass are associated with cardiovascular risk factors in men and women. J Clin Endocrinol Metab. 2008;93:4360-6. doi: 10.1210/jc.2008-0804.

28. Ibrahim MM. Subcutaneous and visceral adipose tissue: structural and functional differences. Obes Rev. 2010; 11:11-8. doi: 10.1111/j.1467-789X.2009.00623.x.

29. McLaughlin T, Lamendola C, LiuA, Abbasi F. Preferential fat deposition in subcutaneous versus visceral depots is associated with insulin sensitivity. J Clin Endocrinol Metab. 2011;96:E1756-60. doi: 10.1210/jc.2011-0615.

30. Matsuzawa Y, Shimomur, I, Nakamura T, KenoY, Kotani K, Tokunaga K. Pathophysiology and pathogenesis of visceral fat obesity. Obes Res. 1995;32:187S-94S. doi: 10.1002/j. 1550-8528.1995.tb00462.x.

31. Tchernof A, Despres JP. Pathophysiology of human visceral obesity: An update. Physiol Rev. 2013;93:359-404. doi: 10.1152/physrev.00033.2011.

32. Fischer K, Moewes D, Koch M, Muller HP, Jacobs G, Kassubek J, Lieb W, Nothlings U. MRI-determined total volumes of visceral and subcutaneous abdominal and trunk adipose tissue are differentially and sex-dependently associated with patterns of estimated usual nutrient intake in a northern German population. Am J Clin Nutr. 2015;101: 794-807. doi: 10.3945/ajcn.114.101626.

33. Pang G, Xie J, Chen Q, Hu Z. Energy intake, metabolic homeostasis, and human health. Food Science and Human Wellness. 2014;3:89-103. doi: 10.1016/j.fshw.2015.01.001.

34. Lambert JE, Ryan EA, Thomson AB, Clandinin MT. De novo lipogenesis and cholesterol synthesis in humans with long-standing type 1 diabetes are comparable to non-diabetic individuals. PLoS One. 2013;8:e82530. doi: 10. 1371/journal.pone.0082530.

35. Jones PJ, Jew S, AbuMweis S. The effect of dietary oleic, linoleic, and linolenic acids on fat oxidation and energy expenditure in healthy men. Metabolism. 2008;57:1198-203. doi: 10.1016/j.metabol.2008.04.012.

36. Schutz Y. Concept of fat balance in human obesity revisited with particular reference to de novo lipogenesis. Int J Obes Relat Metab Disord. 2004;28(Suppl 4):S3-S11. doi: 10. 1038/sj.ijo.0802852.

37. Chong MF, Hodson L, Bickerton AS, Roberts R, Neville M, Karpe F, Frayn KN, Fielding BA. Parallel activation of de novo lipogenesis and stearoyl-CoA desaturase activity after 3 d of high-carbohydrate feeding. Am J Clin Nutr. 2008;87: 817-23. doi: 10.1093/ajcn/87.4.817.

38. Guo ZK, Cella LK, Baum C, Ravussin E, Schoeller DA. De novo lipogenesis in adipose tissue of lean and obese women: application of deuterated water and isotope ratio mass spectrometry. Int J Obes Relat Metab Disord. 2000; 24:932-7. doi: 10.1038/sj.ijo.0801256.

39. Miazgowski T, Krzyzanowska-Swiniarska B, Dziwura-Ogonowska J, Widecka K. The associations between cardiometabolic risk factors and visceral fat measured by a new dual-energy X-ray absorptiometry-derived method in lean healthy Caucasian women. Endocrine. 2014;47:500-5. doi: 10.1007/s12020-014-0180-7.

40. Parks EJ. Dietary carbohydrate's effects on lipogenesis and the relationship of lipogenesis to blood insulin and glucose concentrations. Br J Nutr. 2002;87:S247-53. doi: 10.1079/ BJN/2002544.

41. Bano G. Glucose homeostasis, obesity and diabetes. Best Pract Res Clin Obstet Gynaecol. 2013;27:715-26. doi: 10. 1016/j.bpobgyn.2013.02.007.

42. Brand-Miller JC, Holt SH, Pawlak DB, McMillan J. Glycemic index and obesity. Am J Clin Nutr. 2002;76: 281S-5. doi: 10.1093/ajcn/76/1.281S.

43. Isken F, KlausS, Osterhoff M, Pfeiffer AF, Weickert MO. Effects of long-term soluble vs. insoluble dietary fiber intake on high-fat diet-induced obesity in C57BL/6J mice. J Nutr Biochem. 2010;21:278-84. doi: 10.1016/j.jnutbio.2008. 12.012.

44. den Besten G, van Eunen K, Groen, AK, Venema K, Reijngoud DJ, Bakker BM. The role of short-chain fatty acids in the interplay between diet, gut microbiota, and host energy metabolism. J Lipid Res. 2013;54:2325-40. doi: 10. 1194/jlr.R036012.

45. Howarth NC, Saltzman E, McCrory MA, Greenberg AS, Dwyer J, Ausman, L, Kramer, DG, Roberts SB. Fermentable and nonfermentable fiber supplements did not alter hunger, satiety or body weight in a pilot study of men and women consuming self-selected diets. J Nutr. 2003;133:3141-4. doi: 10.1093/jn/133.10.3141.

46. Slavin JL. Dietary fiber and body weight. Nutrition. 2005; 21:411-8. doi:10.1016/j.nut.2004.08.018.

47. Blaut, M. Gut microbiota and energy balance: role in obesity. Proc Nutr Soc. 2015;74:227-34. doi: 10.1017/S002966511 4001700.

48. Hammad S, Pu S, Jones PJ. Current evidence supporting the link between dietary fatty acids and cardiovascular disease. Lipids. 2016;51:507-17. doi: 10.1007/s11745-015-4113-x.

49. Field AE, Willett WC, Lissner L, Colditz GA. Dietary fat and weight gain among women in the Nurses' Health Study. Obesity. 2007;15:967-76. doi: 10.1038/oby.2007.616.

50. Kien CL, Bunn JY, Poynter, ME, StevensR, Bain J, Ikayeva O et al. A lipidomics analysis of the relationship between dietary fatty acid composition and insulin sensitivity in young adults. Diabetes. 2013;62:1054-63. doi: 10.2337/ db12-0363.

51. Krishnan S, Cooper JA. Effect of dietary fatty acid composition on substrate utilization and body weight maintenance in humans. Eur J Nutr. 2014;53:691-710. doi: 10.1007/s00394-013-0638-z.

52. Nimptsch K, Berg-Beckhoff G, Linseisen J. Effect of dietary fatty acid intake on prospective weight change in the Heidelberg cohort of the European Prospective Investigation into Cancer and Nutrition. Public Health Nutr. 2010;13: 1636-46. doi:10.1017/S1368980009993041.

53. Itariu BK, Zeyda M, Hochbrugger EE, Neuhofer A, Prager G, Schindler K et al. Long-chain n-3 PUFAs reduce adipose tissue and systemic inflammation in severely obese nondiabetic patients: a randomized controlled trial. Am J Clin Nutr. 2012;96:1137-49. doi:10.3945/ajcn.112.037432.

54. Piers LS, Walker KZ, Stoney RM, Soares, MJ, O'Dea K. Substitution of saturated with monounsaturated fat in a 4-week diet affects body weight and composition of overweight and obese men. Br J Nutr. 2003;90:717-27. doi: 10.1079/BJN2003948.

55. Paniagua JA, Gallego de la Sacristana A, Romero I, Vidal-Puig A, Latre JM, Sanchez E et al. Monounsaturated fat-rich diet prevents central body fat distribution and decreases postprandial adiponectin expression induced by a carbohydrate-rich diet in insulin-resistant subjects. Diabetes Care. 2007;30:1717-23. doi: 10.2337/dc06-2220.

56. Khader Y, Batieha A, Ajlouni H, El-Khateeb M, Ajlouni K. Obesity in Jordan: prevalence, associated factors, comorbidities, and change in prevalence over ten years. Metab Syndr Relat Disord. 2008;6:113-20. doi: 10.1089/ met.2007.0030.