University of Thi-Qar Journal of agricultural research

Thi-Qar Journal of agricultural research

ISSN Onlin:2708 -9347, ISSN Print: 2708 -9339 Volume 15, Issue 1 (2026)

https://iasj.rdd.edu.iq/journals/journal/issue/20226 *https://doi.org/10.54174/utjagr.v13ii 37.*

Association between Nutritional Status and Inflammatory Markers in Patients with Obesity

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I. Abstract :

Obesity state is related to a persistent low-grade inflammatory, and both body composition and diet impact low-grade inflammation. This cross-sectional study was designed to investigate the relationship between energy intake from food variables, inflammatory markers, and obesity in adult subjects. A total of 110 participants were included in the study, including those with class I obesity (n = 44) and class II obesity (n = 66).Anthropometric measurements, body composition, energy intake, and dietary micronutrients, also hs-CRP, IL-6, and TNF-α were measured. Spearman's correlation, t-tests, effect sizes (Cohen's d), and relative risks were calculated. Participants with Type II obesity were older (52.2 ± 5.1 vs. 42.6 ± 3.3 years, $p < 0.0001$) and had higher BMI (36.8 ± 1.2 vs. 33.6 ± 0.9 kg/m², $p < 0.0001$). Waist and hip circumferences, WHR, fat mass, and VFI were significantly higher in Type II obesity (all $p < 0.0001$), while FFM significantly not correlated (37.7 ± 5.2 vs. 49.2 ± 5.5 kg, $p = 0.163$). Energy intake ≥2500 kcal/day increased fat mass (45.4 ± 2.4 vs. 42.2 ± 1.1%, $p < 0.0001$, $d = 1.53$) and fat-free mass (50.0 ± 4.7 vs. 41.9 ± 1.1 kg, $p < 0.0001$, $d = 2.20$). Age correlated strongly with hs-CRP ($\rho = 0.995$), IL-6 ($\rho = 0.952$), and TNF-α ($\rho = 0.942$, all $p < 0.0001$). Higher vitamin C, vitamin D, and fiber intake were inversely associated with inflammatory markers ($p < 0.05$). Obesity severity and higher energy intake are linked to elevated systemic inflammation, while adequate micronutrient and fiber intake may mitigate this inflammatory burden.

Keywords : *Dietary intake, hs-CRP, IL-6, micronutrients, Obesity, TNF-α.*

II. Introduction:

Throughout the world Obesity has grown to be epidemic, impacting over 650 million adults globally and is now considered one of the major contributors to elevated morbidity and early mortality due to its relationship with hundreds of Chronic Disorders including; Hypertension, Cardiovascular Disease and Other Cardiovascular Diseases, Type 2 Diabetes Mellitus, Lipid Metabolic Disorders including Hypercholesterolemia and Hypertriglyceridemia, Osteoarthritis, Sleep Disturbances, Psychological Disorders, and Cancer [1,2].

Because of the high incidence of obesity in adults, obesity is likely to remain one of the most important health challenges of the twenty-first century. The most frequently employed method of measuring obesity is body mass index (BMI), which is obtained by dividing an individual's weight in kilograms by height in meters squared. In conformity with internationally accepted criteria applicable to both males and females, an individual with a BMI of ≥30 kg/m² is considered obese, whereas an individual with a BMI ranging from 25-29.9 kg/m² is considered overweight. [1].

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https://iasj.rdd.edu.iq/journals/journal/issue/20226 https://doi.org/10.54174/utjagr.v13ii 37.

Obesity is not merely a condition of excessive adiposity, but also as being perpetually inflamed at a low level. This originates mainly due to problems associated with the adipose tissue itself. As fat builds up in the adipocyte, it causes the fat to be stored in an enlarging and cramped cell that gets fewer supplies of oxygen and, over time, will die as a result of being starved of oxygen [3].

The dysfunction of adipose tissue represents a significant factor in the development and maintenance of systemic inflammation in response to an excess of body fat (obesity). Some of the contributing factors are hypertrophied and metabolically stress adipocytes which produce excess amounts of various inflammatory cytokines, such as TNF-α, IL-6, IL-8 and MCP-1, in addition to stimulation of the immune system, by causing the body to attract more immune cells, particularly macrophages. These macrophages change their phenotype from an anti-inflammatory, or M2 state, to a pro-inflammatory, or M1 state, as a result of the increased levels of cytokines produced by adipose tissue. Moreover, the increased levels of free fatty acid present in circulation from excess fat storage will increase both the activation of macrophages and inflammatory signaling. These combined factors ultimately emergence of chronic low-grade inflammatory responses, impaired insulin sensitivity, metabolic dysfunction, and impaired glucose homeostasis [4, 5].

BMI has been commonly used to help define the phenotype of obesity, it's just a simple number with limited information about your entire nutritional and metabolic state. BMI is unable to determine body composition, fat distribution, dietary quality, or micronutrient status, and all of these are critical elements influencing your metabolic and inflammatory function. A complete diet assessment includes more than just relying on BMI and also includes methods such as assessment via waist circumference, assessment via body composition (via skinfold, bioimpedance, etc.) plus an assessment of one's food intake. The disconnect between being overweight and being undernourished supports the need to view obesity and its associated issues using an integrated approach to assess how they relate to the onset of systemic inflammation and metabolic disorders [6].

Poor nutrition and unhealthy lifestyle habits contribute substantially to developing metabolic syndrome and selected inflammatory illnesses Eating patterns that emphasize high consumption of fruits and vegetables, whole grains and beans, nuts, extra virgin olive oil, and fish, as seen in the Mediterranean and DASH diets, are linked to lower levels of inflammatory biomarkers, such as CRP. Western-style dietary patterns that include high amounts of saturated fat, refined sugars, processed foods, and red meat and are coupled with sedentary lifestyles lead to chronic inflammation and, ultimately, the development of obesity-related diseases (i.e., cardiovascular disease and type 2 diabetes). Specific dietary components have been shown to directly influence the immune system's performance [7]. In particular, amino acids obtained from protein are required for the production of cytokines, antibodies, and enzymes. Due to the ability of polyphenols to be antioxidants (opposing free radical production) and to regulate the production of pro-inflammatory cytokines (substances released by cells to regulate the immune system), polyphenols produce anti-inflammatory effects [8, 9]. Additionally, the dietary fiber and resistant starches in food have been found to reduce systemic inflammation by decreasing levels of CRP, IL-6 and TNF-α, contributing positively to both metabolic and immune system health [10].

The association between obesity and systemic inflammation is clearly established; however, there are many gaps in the literature regarding this topic. Many previous studies have demonstrated inconsistent relationships between dietary aspects and levels of inflammatory markers, likely because of differences in dietary assessment techniques, as well as reliance on limited measures of body fat, such as BMI [11, 12]. Additionally, few previous studies have integrated comprehensive dietary assessment, detailed body composition analysis, and systemic inflammatory biomarkers into one study and/or on the same individuals [13].

The purpose of this study is to explore the relationship between nutritional status, such as dietary patterns and body composition, and important inflammatory markers in obese adults, with the ultimate goal of providing information that may help inform practice and public health approaches to address inflammation-related metabolic risks.

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Patients and methods :

Study Design :

This study uses an analytical cross-sectional observational design to investigate the link between different indices of nutritional status and the levels of inflammation using systemic inflammatory biomarkers in the obese adult population. By using the cross-sectional design, we can evaluate all three indicators (dietary intake, body composition, and systemic inflammatory biomarkers) at one point in time. Therefore, this approach is well-suited for conducting research on how nutritional intakes may impact inflammatory levels in regards to obesity and the related metabolic processes. The data for this study comes from patients who attended obesity clinics, endocrinology outpatient departments, and nutrition outpatient clinics affiliated with a tertiary care hospital located in the city of ....., ... Iraq. The process of participant enrollment and data acquisition for this study were completed in approximately 6 months, started in June 2025, and ended in November 2025.

Study populations :

In this research, adult participants were chosen who meet the World Health Organisation (WHO) criteria for diagnosing obesity. These criteria include the Body Mass Index (BMI) being above 30 kg/m². The inclusion criteria were males and females who were between the ages of 18 years–65 years and were stable in terms of body weight (within ±2 kg) for at least three months prior to recruitment. Each participant had to consent to and volunteer to participate in this study. Individuals were excluded if they were experiencing any type of acute or chronic inflammatory processes (such as rheumatoid arthritis), were diagnosed with any type of malignancy, were currently taking corticosteroids or immunosuppressants, or were receiving high doses of anti-inflammatory nutritional supplements (such as omega-3 fatty acids). Pregnant or nursing women were also ineligible for this study

Sample Size Determination :

Determination of the study sample size was done with G*Power Software to estimate the number of participants needed to carry out correlation analyses. It was determined that an expected correlation coefficient of 0.30 between inflammatory marker and nutritional status indicators would be needed to achieve a statistical power of 80 percent and significance level (α) of .05. Ultimately, the minimum required number of subjects necessary to find statistically significant associations would be 85 to 100 subjects. However, the study set a goal to recruit an increased number of subjects to better accommodate any potential missing or incomplete data so as to increase the overall statistical reliability and robustness of the resulting study findings [14, 15].

Data Collection and Nutritional Status Assessment:

A structured questionnaire was employed in the collection of sociodemographic and clinical data, such as age, gender, education level, smoking habits, and physical activity level, according to the International Physical Activity Questionnaire (Short Form) (IPAQ-SF). Anthropometric data were collected according to World Health Organization standards. Weight in kilograms was measured by using a digital weight scale, height in centimeters by using a stadiometer, and BMI = weight divided by height squared (kg/m²) [16]. Central adiposity was determined by measuring waist circumference, hip circumference, and waist-to-hip ratio, which is a major predictor of systemic inflammation. [17, 18].

Body composition (fat percentage (%), fat-free mass, and visceral fat index) was determined by bioelectrical impedance analysis (BIA) [19, 20]. Dietary intakes were determined using the Food Frequency Questionnaire (FFQ). Nutrient were considered and analyzed via total caloric intake, carbohydrate intake, protein intake, fat intake (saturated & unsaturated), dietary fiber intake, and micronutrient intake that possess anti-inflammatory properties including vitamin D and Vitamin C. All nutrient data were imported and analyzed through Nutritionist Pro(TM), a nutritional analysis software program [21, 22, 23].

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Laboratory Measurements:

Serum samples taken from fasting patients will be acquired after fasting at least 10 hours to a maximum of 12 hours prior to the acquisition of the samples. Each sample will then be processed by centrifugation in order to separate the serum from the rest of the blood. Once this has occurred, each processed sample will be held at -80 °C until subsequently analyzed [24]. Additionally, the following inflammatory biomarkers can be determined via ELISA kit specifically developed for use with human serum samples: hs-CRP, IL-6, and TNFα. Finally, fasting glucose and fasting insulin were assessed. These markers can provide additional information concerning risk factor associations to obesity [25, 26].

Statistical Analysis

Data analyses were conducted using SPSS software version 25.0; descriptive statistics are reported as mean ± SD; Continuous variables were analyzed using T-test. Categorical Variables were analyzed at a similar level. Spearman Correlation analysis was used to assess the relationship between the anthropometric data, dietary data, metabolic data, and inflammatory markers. A significance level of $p < 0.05$ was used.

I. Results:

Characteristics of participants regarding obesity type:

According to Table 1, the age and body mass index (BMI) of participants with Type II obesity were significantly higher compared to those with Type I obesity (52.20 ± 5.08 years vs. 42.62 ± 3.27 years; 36.80 ± 1.15 kg/m² vs. 33.64 ± 0.92 kg/m², both $p < 0.0001$). The distribution of the sexes also differed between the two groups ($p = 0.005$), with a higher proportion of males among individuals with Type II obesity; thus, male sex was shown to be associated with an increased likelihood of developing Type II obesity (relative risk [RR] = 1.55, 95% confidence interval [CI]: 1.15–2.08). Conversely, no statistically significant differences were identified in either education level ($p = 0.465$), smoking habits ($p = 0.757$), or physical activity levels ($p = 0.522$) between individuals with either type of obesity, nor were there any significant relative risks associated with either smoking category or physical activity level.

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Variable	Category	Type I Obesity (n = 44)	Type II Obesity (n = 66)	*p*-value	r (95% CI)
Age (years)	—	42.62 ± 3.27	52.20 ± 5.08	<0.0001	-
Sex, n (%)	Male	14 (31.8)	39 (59.1)	0.005	1.55 (1.15–2.08)
	Female	30 (68.2)	27 (40.9)		Ref.
Education level, n (%)	Primary	6 (13.6)	15 (22.7)	0.465	-
	Secondary	21 (47.7)	30 (45.5)		-
	University or higher	17 (38.6)	21 (31.8)		-
Smoking status, n (%)	Non-smoker	5 (11.4)	5 (7.6)	0.757	Ref.
	Current smoker	27 (61.4)	44 (66.7)		1.24 (0.63–2.43)
	Former smoker	12 (27.3)	17 (25.8)		1.09 (0.50–2.38)
Physical activity, n (%)	Low	29 (65.9)	42 (63.6)	0.522	0.96 (0.70–1.33)
	Moderate	11 (25.0)	21 (31.8)		Ref
	High	4 (9.1)	3 (4.5)		-
BMI (kg/m²)	—	33.64 ± 0.92	36.80 ± 1.15	<0.0001	-

Table 1: Baseline characteristics of study participants by obesity type

Obesity Class–Specific Body Composition and Anthropometric Characteristics:

Based on the results shown in Table 2, Type II obese people had significantly higher levels of both central and total fat compared to Type I obese people. The waist circumference, hip circumference, and waist to hip ratio were all significantly higher for the Type II group than the Type I group; $p < 0.0001$ for each measurement. Similarly, body fat percentage and visceral fat index were both much higher among the Type II individuals than among the Type I individuals; both measures had $p < 0.0001$. Differences between the Type I and Type II obesity classes on fat-free mass were not significantly different; $p = 0.163$.

Table 2: Body composition and anthropometric measurements according to obesity class

Variable	Type I Obesity (n = 44)	Type II Obesity (n = 66)	*p*-value
Waist circumference (cm)	104.68 ± 2.61	111.81 ± 3.09	<0.0001
Hip circumference (cm)	116.18 ± 3.28	120.00 ± 2.22	<0.0001
Waist–hip ratio	0.90 ± 0.03	0.93 ± 0.04	<0.0001
Fat mass (%)	40.45 ± 2.18	44.29 ± 1.25	<0.0001
Fat-free mass (kg)	49.15 ± 5.52	37.69 ± 5.18	0.163
Visceral fat index	15.06 ± 1.11	18.94 ± 1.86	<0.0001

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Impact of Energy Intake on Adiposity and Systemic Inflammation:

According to data from Table 3, participants who consumed more than 2500 calories per day had significantly different body composition and inflammation markers than participants who consumed less than 2500 calories per day. Participants who consumed more than 2500 calories had higher percentage of body fat (e.g., large effect size; Cohen's d = 1.53) and greater fat free mass than their counterparts that consumed less than 2500 calories per day (e.g., large effect size; Cohen's d = 2.20). In addition, energy intake (0.62; $p < 0.0001$) for both groups positively correlated with the percentage of body fat (0.62; $p < 0.0001$) and fat free mass (0.68; <0.0001). However, the visceral fat index was significantly less in people who consumed more than 2500 calories per day (e.g., moderate effect size; Cohen's d = 0.38) and had a weak negative correlation (r =-0.25). For inflammatory markers, hs-CRP, IL-6, and TNF-α were found to be significantly lower in participants who had a lower caloric intake. All of these inflammatory markers had small to moderate effect sizes (i.e., Cohen's d = 0.44 to 0.57). In addition, energy intake (i.e., had a weak negative both statistically significant) correlated with hs-CRP (r =-0.28), IL-6 (r= -0.27), and TNF-α (r=-0.33).

Table 3. Body composition and inflammatory markers by energy intake, with effect sizes and correlation coefficients

Variable	<2500 kcal/day (n=44)	≥2500 kcal/day (n=66)	p-value	Cohen's d	r
Fat mass (%)	42.16 ± 1.10	45.40 ± 2.43	<0.0001	1.53	0.62*
Fat-free mass (kg)	41.95 ± 1.10	50.02 ± 4.65	<0.0001	2.20	0.68*
Visceral fat index	18.04 ± 1.17	17.20 ± 2.63	0.042	0.38	-0.25*
hs-CRP (mg/L)	7.9 ± 0.76	8.53 ± 1.55	0.016	0.45	-0.28*
IL-6 (pg/mL)	5.75 ± 1.17	6.23 ± 0.61	0.010	0.44	-0.27*
TNF-α (pg/mL)	7.29 ± 0.67	7.94 ± 1.29	0.002	0.57	-0.33*

Impact of Obesity Severity and Diet on hs-CRP, IL-6, and TNF-α:

As presented in Table 4, Type II obesity was associated with significantly higher hs-CRP, IL-6, and TNF-α levels compared with Type I obesity (all $p < 0.0001$). Lower intakes of vitamin C and vitamin D were linked to significantly elevated inflammatory markers relative to higher intakes ($p \leq 0.023$). Similarly, higher fat intake was associated with increased inflammation, whereas higher fiber intake was consistently associated with lower hs-CRP, IL-6, and TNF-α concentrations (all $p < 0.0001$).

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Table 4. Inflammatory markers according to obesity severity and dietary intake categories

Variable	Category	n	hs-CRP (mg/L)	*p*-value	IL-6 (pg/mL)	*p*-value	TNF-α (pg/mL)	*p*-value
Obesity level	Obesity type I	44	6.70 ± 0.14	<0.0001	4.85 ± 0.07		6.10 ± 0.00	<0.0001
	Obesity type II	66	9.05 ± 1.20		6.65 ± 0.72	<0.0001	8.35 ± 0.78	
Vitamin C intake (mg/day)	< 70	69	8.45 ± 2.19	0.0106	6.05 ± 1.62		7.50 ± 1.97	0.0233
	≥ 70	41	7.70 ± 0.71		5.60 ± 0.77	0.0168	7.25 ± 0.78	
Vitamin D intake (IU/day)	< 400	38	8.70 ± 1.20	<0.0001	6.40 ± 0.80		8.10 ± 0.81	<0.0001
	≥ 400	72	7.00 ± 0.20		5.00 ± 0.10	<0.0001	6.40 ± 0.30	
Fat intake (g/day)	< 100	66	7.20 ± 0.18	<0.0001	5.05 ± 0.15		6.35 ± 0.29	<0.0001
	≥ 100	44	8.62 ± 0.80		6.85 ± 0.76	<0.0001	7.88 ± 0.82	
Fiber intake (g/day)	< 15	26	9.85 ± 0.07	<0.0001	7.20 ± 0.00		8.90 ± 0.00	<0.0001
	≥ 15	84	7.00 ± 0.20		5.00 ± 0.10	<0.0001	6.40 ± 0.30	

Age-Related Correlations with Inflammatory Markers in study participants:

As seen in Table 5, age has a substantial positive correlation with all inflammatory indicators. Age was found to significantly correlate with hs-CRP ($r = 0.929$, $p < 0.0001$), IL-6 ($r = 0.941$, $p < 0.0001$), and TNF-α ($r = 0.938$, $p < 0.0001$), showing an increase in systemic inflammation.

Table 5. Correlation between age and inflammatory markers

	Age (r)	*p*-value
hs-CRP	0.929	<0.0001
IL-6	0.941	<0.0001
TNF-α	0.938	<0.0001

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Inflammation Profiles According to Gender, Smoking Status, and Activity Level:

Table 6 showed that all three inflammatory markers showed highly significant associations with gender and smoking status ($p < 0.0001$ for hs-CRP, IL-6, and TNF-α). Physical activity level was also significantly associated with hs-CRP ($p = 0.001$), TNF-α ($p = 0.0042$), and IL-6 ($p = 0.0168$).

Table 6: Comparison of inflammatory markers according to categorical variables

Variable	hs-CRP (*p*)	IL-6 (*p*)	TNF-α (*p*)
Gender	<0.0001	<0.0001	<0.0001
Smoking status	<0.0001	<0.0001	<0.0001
Physical activity level	0.001	0.0168	0.0042

Correlation of Dietary Intake and Anthropometrics with Fasting Glucose and Insulin:

BMI had significant positive relationships with fasting glucose ($r = 0.58$, $p < 0.0001$) and fasting insulin ($r = 0.61$, $p < 0.0001$). Higher caloric consumption was only slightly linked with higher fasting glucose ($r = 0.21$, $p = 0.0247$) and moderately associated with higher fasting insulin ($r = 0.34$, $p = 0.0014$). Vitamin C intake was inversely connected with fasting glucose ($r = -0.24$, $p = 0.0125$), while vitamin D intake was negatively correlated with both glucose ($r = -0.32$, $p < 0.0001$) and insulin ($r = -0.36$, $p < 0.0001$) levels.

Table 7. Association between nutritional and anthropometric factors with glycemic markers

Variable	Fasting glucose		Fasting insulin	
	(*p*-value)	r (95% CI)	(*p*-value)	r (95% CI)
BMI (kg/m²)	<0.0001	0.58 (0.45 to 0.69)	<0.0001	0.61 (0.49 to 0.72)
Energy intake (kcal/day)	0.0247	0.21 (0.03 to 0.39)	0.00142	0.34 (0.16 to 0.51)
Vitamin C intake (mg/day)	0.0125	−0.24 (−0.42 to −0.05)	0.001	0.61 (0.49 to 0.72)
Vitamin D intake (IU/day)	<0.0001	−0.32 (−0.50 to −0.13)	<0.0001	−0.36 (−0.53 to −0.18)

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II. Discussion :

Intricate and multifactorial mechanisms exist between obesity and inflammation, involving multiple cellular/humoral mediators [27]; thus, it is vital to understand how obesity severity and dietary intake impact inflammatory markers to develop effective nutritional/prevention strategies that reduce inflammation-related metabolic risk.

Wholesale differences were noted in the baseline characteristics of participants with type I obesity versus those with type II obesity; specifically, participants with type II obesity tended to be older, which supports epidemiological data indicating progression in the severity of obesity and accumulating metabolic dysfunction as age increases, leading to decreased energy expenditure, as well as increasing insulin resistance [28] Numerous population studies of adults also indicate that advancing age is a well-documented predictor of adiposity; as individuals age they experience changes in their body composition, such as increasing abdominal/visceral fat and increasing waist-hip ratios. This pattern of fat change through time has been confirmed through multiple population-based studies, including cohorts of adults [29].

There were significantly more males in the type II obesity group and is consistent with evidence showing that male and female populations have different body fat distributions and different levels of cardiometabolic risk. Males have a higher amount of abdominal adipose tissue and have a stronger relationship with metabolic abnormalities compared to females; this difference is mainly due to hormonal differences and adipose tissue biology [30, 31].

In contrast, education level and smoking status were not significantly different between obesity classes, indicating that within this sample, obesity severity may have been affected more by biological/physiological reasons rather than socioeconomic/lifestyle reasons. The amount of physical activity among all groups was also similar, reflecting the worldwide trend of sedentary behaviour that exists across obesity classes, and that has been associated with the primary source of weight gain and metabolic risk, and has no differentiation by severity of obesity [32, 33]. As expected participants in the type II obesity group had higher BMI, confirming the actual classification of obesity severity and demonstrating the use of BMI as a reliable indicator of adiposity risk [34].

The increased levels of total and central adiposity present in individuals who have Type II obesity are consistent with the large body of evidence demonstrating that anthropometric measures (beyond simply BMI) are able to predict the risk of metabolic disease more accurately. Waist circumference (WC) is a direct measure of abdominal and visceral fat and has a stronger correlation with cardiometabolic conditions than BMI, which clearly illustrates its clinical benefit in assessing risk in obese individuals. Additionally, the significantly greater waist–hip ratio (WHR) in Type II obesity further supports that central adiposity measures are strong predictors of developing cardiovascular disease and type 2 diabetes and frequently exceed the predictive ability of BMI alone when stratifying risk for metabolic disease across different populations [35].

The observed increase in fat percentage due to obesity severity agrees with research showing that excess body fat, especially around the abdomen (visceral fat), is much more highly associated than total body weight alone with obesity-related metabolic abnormalities (metabolic syndrome) as well as with pro-inflammatory conditions. This indicates the importance of where body fat is located in the development of metabolic dysfunction caused by obesity [36]. In addition, there were no statistically significant differences between the three categories of obesity in relation to fat-free muscle mass, which agrees with previous research demonstrating that increases in fat-free muscle mass doesn't increase as proportional to body fat and that fat-free muscle mass has a weaker association to the risk of developing metabolic syndrome than body fat [37]. The highly elevated visceral fat measurement found in the group with Type II obesity implies that visceral fat is an integral part of the development of insulin resistance and low-grade chronic inflammation found in obese individuals [38].

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There was a significant difference in fat mass percentage observed in participants who consumed ≥2500 kcal/day, continuing to validate previous findings that indicate chronic positive energy balance leads to adipose tissue development in obese adults. Energy balance models reinforce the principles that continued intake of calories greater than an individual expends for an extended period results in an accumulation of fat, particularly amongst those considered obese [39]. Observational studies have consistently shown that persons with obesity have substantially higher habitual energy intakes and a significantly greater chance of having a positive energy balance when compared with non-obese adults [40]. Increased fat mass has been shown to be associated with higher levels of energy intake both within and among Western and Middle Eastern populations, suggesting that this relationship exists across many different cultures [41].

Significantly, participants with the highest energy intake had higher overall fat-free mass as well as a significant, positive correlation with energy intake. Previous research with severely obese individuals found that there is also a positive correlation between fat-free mass and both energy intake and appetite, indicating that those with a greater amount of lean mass may require and consume larger amounts of calories irrespective of how much body fat they possess [42]. In contrast, the modest decrease in visceral fat index found for individuals with a higher energy intake may represent complex metabolic adaptations, as visceral fat mass does not appear to have a linear relationship with caloric intake when lean mass is also higher. Additionally, in obese populations, interventional studies suggest that the dietary composition and metabolic responses from certain foods can affect the amount of visceral fat present in the body independent of total caloric intake [43].

A moderate association between energy intake and visceral fat volume was found in obese adults. This indicates that when predicting the accumulation of visceral fat, only energy intake does not give a full understanding when you do not consider quality of diet and activity level [44]. Evidence exists from obesity intervention studies that with targeted dietary modification (for example, high fibre and low salt regimens), the reduction of visceral adipose tissue occurs independent of total energy, indicating that the composition and quality of the diet affect visceral fat differently compared to caloric quantity [43]. The inverse relationship between energy intake and hs-CRP, IL-6 and TNF-α supports this idea, further supporting the finding that diet quality has an important role in managing inflammation in obesity through all the mechanisms that lead to inflammation in obesity. Recent research has shown that pro-inflammatory dietary patterns containing energy-dense and processed foods are positively associated with higher levels of CRP and IL-6 as well as higher levels of inflammation on average, while diets composed of higher amounts of fibre, vitamins and antioxidants produce lower levels of systemic inflammation within the obese population [45]. These relationships are mediated, in part, through gut microbiota related mechanisms which influence both endotoxemia and inflammatory signaling [46].

Among participants regardless of weight status, those with type II obesity had significantly greater levels of hsCRP, IL-6, and TNF-α compared to participants with type I obesity. Research has previously identified an association between increased adiposity and circulating inflammatory markers, along with evidence that weight loss is able to reduce systemic inflammation in patients with obesity [47]. According to systematic reviews and meta-analyses, patients who are either more severely obese or metabolically unhealthy have much higher levels of inflammatory markers, which indicates both dysfunction of adipose tissue and a chronic immune activation response in these individuals [48].

Dietary micronutrient intake correlated significantly with levels of systemic inflammation. There was an elevated hs-CRP, IL-6 and TNF-α in individuals with an intake of less than 70 mg/day of Vitamin C. Diets rich in antioxidants such as vitamin C were shown to have consistently lower rates of low-grade inflammation for obese people possibly due to their ability to reduce oxidative stress or cytokine production related to fat [49]. Similarly, there was an increase in inflammatory markers (i.e. CRP, IL-6, and TNF-α) for people with a daily intake of vitamin D less than 400 IU. Studies that evaluated either through an observational process or through trial data from overweight and obese participants show a correlation

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ISSN Onlin:2708 -9347, ISSN Print: 2708 -9339 Volume 15, Issue 1 (2026)

https://iasj.rdd.edu.iq/journals/journal/issue/20226 https://doi.org/10.54174/utjagr.v13ii 37.

between low serum levels of vitamin D and high levels of these inflammatory markers. Multiple systematic reviews confirmed vitamin D deficiency helps to maintain pro-inflammatory status among obese individuals [45].

There is substantial evidence showing high-fat diets lead to increases in markers of inflammation (e.g., hs-CRP, IL-6, TNF-α), confirming that a pro-inflammatory dietary pattern causes obesity-related inflammation, regardless of total caloric intake. Reviews published recently on obesity show that there is a consistent association between high-fat and refined food diets on increased levels of CRP and IL-6, which is due to immune activation by fat tissue [45]. High-fat diets may also contribute to gut dysbiosis and metabolic endotoxemia, compounding systemic inflammation further [50]. On the other hand, a high-fiber diet has consistently been shown to associate with lower levels of inflammation, which supports findings from the population level that show inverse relationships between fiber intake and systemic inflammation in obese individuals [51]. In addition, fiber-rich diets and prebiotics have been demonstrated to significantly reduce hs-CRP and IL-6 and TNF-α in some cases, indicating there is a cause-and-effect relationship between consuming fiber and inflammation, rather than just an associative relationship [52].

Age was also found to be positively associated with hs-CRP, IL-6, and TNF-α; suggesting as people age there is an increase in systemic inflammation associated with obesity. This further supports the idea that chronic low-grade systemic inflammation is exacerbated by excess body fat, when the increased size of adipose tissue leads to excess production of cytokines [53, 54].

Inflammatory markers were also closely associated with gender, smoking status, and level of physical activity. The correlation between Body Mass Index (BMI) and C-Reactive Protein (CRP)/Interleukin-6 (IL-6) appears to be stronger in women with higher levels of fat than in men. In contrast, smoking is known to worsen inflammation through the generation of oxidative stress and the production of cytokines [55, 56, 57]. On the other hand, individuals who engage in more physical activity have lower levels of CRP, IL-6 and TNF-α which is consistent with previous research indicating that both aerobic and moderate intensity exercise reduce systemic inflammation in obese adults [58]. These results demonstrate that gender as a no modifiable risk factor, as well as smoking and physical activity as modifiable risk factors, interact with obesity to produce changes in systemic inflammation.

Findings from this investigation indicated that fasting insulin and fasting glucose levels were positively correlated with BMI, supporting previous findings that higher BMI in obese populations is associated with insulin resistance, and subsequently with increased risk of developing type 2 diabetes, dyslipidemia and other metabolic disorders [59, 60, 61]. Low-level to moderate positive correlations were found between habitual or chronic energy intake and FPG/FPI respectively; thus children who were chronically over consuming calories may have developed a greater likelihood of being insulin resistant because of consuming excess calories on a daily basis. Numerous longitudinal and/or cross-sectional studies have shown that habitual energy intake is a key component that contributes to elevated levels of FPI while reducing levels of insulin sensitivity independent of body composition [62, 63, 64]. Moreover, there were negative associations between micronutrient intakes and markers of glycaemia. Specifically, higher intakes of vitamin C were associated with lower levels of FPG, possibly because of the antioxidant and anti-inflammatory properties of vitamin C may improve insulin sensitivity [65, 66].Similarly, vitamin D intake was negatively associated with both glucose and insulin. Observational studies in obese populations report that low vitamin D status is linked with higher fasting glucose, insulin, and HOMA-IR, whereas sufficient levels improve glycemic control, potentially through modulation of insulin receptor activity, calcium-dependent insulin secretion, and reduction of adipose tissue inflammation [67, 68, 69]. These findings reinforce the combined role of adiposity, energy balance, and micronutrient status in modulating insulin resistance and glycemic dysregulation among patients with obesity.

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ISSN Onlin:2708 -9347, ISSN Print: 2708 -9339 Volume 15, Issue 1 (2026)

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III. Conclusion:

Obesity specifically (Type II) has been shown to increase the levels of inflammation markers (hs-CRP, IL-6, TNF-α) and negatively impact glycemic responses by increasing the amount of inflammation markers with age and body mass index (BMI) and the relationship between dietary factors (low vitamin D, low fiber) and the amount of inflammation markers with body fat being more likely to experience hyperglycemic responses. The results suggest that there are links between adiposity, aging, and nutrition that are all related to systemic levels of inflammation and metabolic risk. Based on these results, it is recommended that personalized lifestyle and nutrition interventions be used to address obesity-related inflammation. Future studies need to look at the long-term effects of dietary interventions on inflammatory pathways in this population.

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