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Original Research

Meal Frequency, Visceral Adiposity Index, and Atherogenic Index of Plasma in Adults with Overweight and Obesity

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ABSTRACT

Objective: This study aimed to evaluate whether daily main meal count and snack frequency are independently associated with the Visceral adiposity index (VAI) and atherogenic index of plasma (AIP) in overweight and obese adults without known chronic disease or pharmacotherapy.

Methods: This cross-sectional study included 347 adults aged 18–65 years with BMI ≥25 kg/m² who presented for a first cardiological assessment. Participants with diagnosed chronic disease or regular medication use were excluded. Meal frequency was recorded by structured interview as daily main meal count and snack frequency. VAI was calculated using sex-specific formulas incorporating waist circumference, BMI, triglycerides, and HDL-cholesterol. AIP was calculated as $\log_{10}$ (triglycerides/HDL-cholesterol), using molar concentrations.

Results: The mean age of participants was 40.8 ± 11.7 years, and 53.3% were female. Participants with obesity had significantly higher AIP and VAI values than overweight participants. In unadjusted analyses, main meal count was positively correlated with AIP, whereas snack frequency was inversely correlated with both AIP and VAI. Participants reporting one snack per day had lower AIP and VAI values than those reporting no snacks. However, neither main meal count nor snack frequency remained independently associated with AIP or VAI in the multivariable models. The AIP model was adjusted for age, sex, BMI, smoking status, and regular exercise, whereas the primary VAI model was constructed without BMI because BMI is a component of the VAI formula. Higher age, male sex, BMI, and lack of regular exercise were the main independent correlates of AIP. In the primary BMI-free VAI model, higher age and lack of regular exercise were independently associated with VAI, while meal-frequency variables remained non-significant. A BMI-adjusted sensitivity model yielded the same conclusion regarding meal-frequency variables.

Conclusion: In medication-free overweight and obese adults without known chronic disease, meal frequency alone was not independently associated with AIP or VAI after adjustment for demographic, anthropometric, and lifestyle-related factors. These findings suggest that simple eating-occasion counts may be less informative than overall dietary quality, meal composition, timing, and physical activity in cardiometabolic risk assessment.

Keywords: atherogenic index of plasma, cardiometabolic risk, meal frequency, obesity, overweight, visceral adiposity index

INTRODUCTION

Excess adiposity rarely travels alone. In most clinical settings, visceral fat accumulation, dyslipidemia, insulin resistance, and elevated cardiovascular risk cluster so predictably [1–3] that disentangling their individual dietary drivers becomes a genuine methodological challenge. Meal frequency has attracted attention as one potentially modifiable behavior. However, the literature remains inconsistent, partly because study samples almost invariably include participants already carrying diagnoses of type 2 diabetes, hypertension, or established dyslipidemia [4,5].

Visceral adiposity index (VAI) and atherogenic index of plasma (AIP) were developed to give clinicians compact surrogates for visceral fat function and atherogenic dyslipidemia, respectively, both derivable from routine laboratory values. VAI combines waist circumference, BMI, and fasting lipids into a sex-specific score that tracks more closely with visceral fat area assessed by computed tomography than BMI alone [6,7,8]. AIP, expressed as $\log_{10}$ of the triglyceride-to-HDL molar ratio, encodes the small dense LDL-predominant lipid phenotype associated with premature atherosclerosis [9]. Neither index requires specialized imaging, making them attractive for outpatient screening [10].

Main Points

- In overweight and obese adults without known chronic disease or pharmacotherapy, daily main meal count and snack frequency were not independently associated with the atherogenic index of plasma or the visceral adiposity index after adjustment for demographic, anthropometric, and lifestyle-related factors.
- Unadjusted analyses suggested a positive association between main meal count and atherogenic index of plasma and inverse associations between snack frequency and both cardiometabolic indices, but these relationships did not persist in multivariable models.
- Higher age, male sex, higher body mass index, and absence of regular exercise were the main independent correlates of a higher atherogenic index of plasma, whereas higher age, higher body mass index, and absence of regular exercise were independently associated with a higher visceral adiposity index.
- These findings suggest that simple eating-occasion counts alone provide limited cardiometabolic risk information, and that future studies should incorporate meal composition, dietary quality, energy intake, snack type, and circadian timing.

What is far less clear is whether eating-occasion frequency shapes these indices in individuals who have not yet received any cardiometabolic diagnosis and are on no medications. In adults with excess weight but without diagnosed cardiometabolic disease or medication use, any observed association between eating-occasion frequency and metabolic risk may be less affected by confounding from chronic disease and pharmacological treatment. The cardiology outpatient setting offers a clinically meaningful frame: individuals presenting for an initial assessment, before therapeutic decisions are made, represent a window in which modifiable lifestyle factors, including dietary patterns, can still be evaluated before treatment-related influences are introduced. The present study examined whether daily main meal count and snack consumption frequency were independently associated with VAI and AIP after adjustment for demographic, anthropometric, and lifestyle-related covariates.

MATERIALS AND METHODS

Study Design and Participants

This observational, cross-sectional study was conducted in the cardiology outpatient clinic of Gaziantep University Şahinbey Research and Practice Hospital between January and April 2026. This study was approved by the Gaziantep University Non-Interventional Clinical Research Ethics Committee (Decision No. 2026/67, January 21, 2026). Consecutive adults aged 18–65 years presenting for a first cardiological assessment were screened. Those with a documented chronic disease diagnosis (including type 2 diabetes, hypertension, dyslipidemia, thyroid disorders, and cardiovascular events), any regular prescription or over-the-counter medication use, pregnancy, active infectious illness, or a cognitive barrier to completing a structured interview were excluded.

The BMI inclusion threshold was set at ≥ 25 kg/m² to ensure the sample reflected clinically meaningful excess weight.

Power Analysis and Sample Size

For the planned multiple linear regression analyses, assuming six independent variables, a small-to-moderate effect size (Cohen's $f^2 = 0.05$), $\alpha = 0.05$, and 80% power, the minimum required sample size was 279 participants. After allowing for potential data loss, the target sample size was set at 310–320, and the final analytic sample included 347 participants.

RESULTS

Pearson correlation analyses showed that the main meal count was positively correlated with AIP but not with VAI in the total sample. Snack meal count was inversely correlated with both AIP and VAI, whereas total meal count was not associated with either index. In sex-stratified analyses for VAI, no significant correlations were observed among women. Among men, snack meal count and total meal count were weakly and inversely correlated with VAI, while main meal count was not associated with VAI in either sex (Table 2).

No participant reported consuming one main meal per day; therefore, the main-meal comparison was performed between participants consuming two and three main meals per day. AIP differed significantly according to the main meal count. Participants consuming three main meals per day had higher AIP values than those consuming two main meals per day (0.139 ± 0.253 vs. 0.057 ± 0.315; t = −2,665, $p = 0.008$). In contrast, VAI did not differ significantly between the two main meal groups (2,770 ± 1,571 vs. 2,565 ± 1,745; t = −1,154, $p = 0.249$). When participants were grouped according to snack frequency, both AIP and VAI differed significantly across groups. 1-way ANOVA demonstrated significant overall differences for AIP (F (2,344) = 15,176, $p < 0.001$) and VAI (F (2,344) = 8,350, $p < 0.001$). Games-Howell post-hoc comparisons showed that participants reporting one snack per day had significantly lower AIP and VAI values than those reporting no snacks. The 2-snacks-per-day group did not differ significantly from either the no-snack or 1-snack groups (Table 3).

Table 1. Baseline characteristics of the study population stratified by BMI group.

Variable	Total (n = 347)	Overweight (n = 169)	Obese (n = 178)	p
Age (years)	40.8 ± 11.7	37.3 ± 10.7	44.1 ± 11.6	<0.001*
Female, n (%)	185 (53.3)	82 (48.5)	103 (57.9)	0.081
BMI (kg/m²)	30.93 ± 4.62	27.29 ± 1.40	34.39 ± 3.89	<0.001*
Waist circumference (cm)	108.9 ± 11.3	101.9 ± 8.8	115.5 ± 9.2	<0.001*
Neck circumference (cm)	40.9 ± 2.0	40.7 ± 2.0	41.2 ± 1.9	0.063
Fasting TG (mg/dL)	149.0 (91.0–203.0)	118.0 (81.5–183.0)	165.0 (103.8–225.3)	<0.001*
Fasting HDL (mg/dL)	48.0 (43.0–57.0)	49.0 (44.0–58.0)	47.0 (42.0–55.0)	0.047*
Fasting LDL (mg/dL)	105.0 ± 28.1	99.6 ± 28.7	110.0 ± 26.6	<0.001*
Fasting glucose (mg/dL)	98.0 (92.0–107.0)	95.0 (90.5–105.5)	101.0 (94.8–109.0)	<0.001*
HbA1c (%)	5.32 ± 0.42	5.21 ± 0.37	5.43 ± 0.44	<0.001*
AIP	0.100 ± 0.287	0.040 ± 0.290	0.157 ± 0.272	<0.001*
VAI	2.673 ± 1.656	2.324 ± 1.558	3.005 ± 1.682	<0.001*
Main meal count	2.52 ± 0.51	2.47 ± 0.50	2.58 ± 0.51	0.034*
Snack meal count	0.52 ± 0.59	0.49 ± 0.57	0.56 ± 0.60	0.291
Regular exercise, n (%)	70 (20.2)	20 (11.8)	50 (28.1)	<0.001*
Smoking, n (%)	196 (56.5)	92 (54.4)	104 (58.4)	0.454
Alcohol use, n (%)	28 (8.1)	9 (5.3)	19 (10.7)	0.067

Data are presented as mean ± standard deviation, median (Q1–Q3), or n (%), as appropriate. Normally distributed continuous variables were compared using the independent-samples t-test, whereas fasting triglyceride, fasting HDL-cholesterol, and fasting glucose were compared using the Mann–Whitney *U* test because of non-normal distribution. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. *$p < 0.05$. BMI: body mass index; TG: triglyceride; HDL: high-density lipoprotein; LDL: low-density lipoprotein; AIP: atherogenic index of plasma; VAI: visceral adiposity index.

Table 2. Pearson correlation coefficients between meal-frequency variables and cardiometabolic indices.

Meal variable	AIP r Total (n=347)	AIP p	VAI r Total (n=347)	VAI p	VAI r Female (n=185)	VAI p	VAI r Male (n=162)	VAI p
Main meal count	0.139*	0.010	0.058	0.281	0.114	0.121	−0.024	0.760
Snack meal count	−0.221*	<0.001	−0.158*	0.003	−0.036	0.622	−0.248*	0.001
Total meal count	−0.081	0.133	−0.086	0.110	0.045	0.545	−0.203*	0.010

Values are Pearson correlation coefficients. Significant correlations are marked with an asterisk. AIP: atherogenic index of plasma; VAI: visceral adiposity index.

Table 3. AIP and VAI according to main meal count and snack frequency.

Group	n	AIP, mean ± SD	VAI, mean ± SD
Main meal count	-	**t = −2.665; *p* = 0.008***	**t = −1.154; *p* = 0.249**
2 meals/day	164	0.0568 ± 0.3149	2.5646 ± 1.7449
3 meals/day	183	0.1392 ± 0.2534	2.7700 ± 1.5714
Snack frequency	-	**F(2,344) = 15.176; *p* < 0.001***	**F(2,344) = 8.350; *p* < 0.001***
0 snacks/day	182	0.174 ± 0.285[a]	2.987 ± 1.740[a]
1 snack/day	149	0.007 ± 0.262[b]	2.263 ± 1.478[b]
2 snacks/day	16	0.134 ± 0.265[ab]	2.923 ± 1.345[ab]

Data are presented as mean ± standard deviation. AIP and VAI were compared between main meal groups using independent-samples t-test. For the main-meal comparison, Welch's correction was applied for AIP because Levene's test indicated unequal variances, whereas equal variances were assumed for VAI. Snack-frequency groups were compared using one-way ANOVA followed by Games–Howell post-hoc tests. Statistically significant overall tests are marked with an asterisk. Within each outcome, different superscript letters indicate statistically significant between-group differences in post-hoc comparisons. Groups sharing the same superscript letter are not significantly different. No participant reported consuming one main meal per day; therefore, the main-meal comparison included only the two observed categories, namely two and three main meals per day. AIP: atherogenic index of plasma; VAI: visceral adiposity index.

Multiple linear regression analyses were performed separately for AIP and VAI (Table 4). The AIP model included main meal count, snack meal count, age, sex, BMI, smoking status, and regular exercise as independent variables and was statistically significant, explaining 23.8% of the variance in AIP ($R^2 = 0.238$, adjusted $R^2 = 0.223$; F (7,339) = 15,157, $p < 0.001$). In this model, main meal count (B = 0.029, $p = 0.308$) and snack meal count (B = −0.034, $p = 0.172$) were not independently associated with AIP. Higher age (B = 0.005, $p < 0.001$), male sex (B = 0.201, $p < 0.001$), and higher BMI (B = 0.010, $p = 0.002$) were independently associated with higher AIP, whereas regular exercise was independently associated with lower AIP (B = −0.076, $p = 0.030$). Smoking status was not significantly associated with AIP.

For VAI, the primary regression model was constructed without BMI because BMI is a component of the VAI formula. This BMI-free model was statistically significant and explained 11.2% of the variance in VAI ($R^2 = 0.112$, adjusted $R^2 = 0.097$; F (6,340) = 7,180, $p < 0.001$). In this primary VAI model, neither main meal count (B = 0.058, $p = 0.741$) nor snack meal count (B = −0.274, $p = 0.080$) was independently associated with VAI. Higher age was independently associated with higher VAI (B = 0.037, $p < 0.001$), whereas regular exercise was independently associated with lower VAI (B = −0.492, $p = 0.023$). Sex (B = 0.247, $p = 0.229$) and smoking status (B = −0.333, $p = 0.082$) were not independently associated with VAI.

As a sensitivity analysis, a BMI-adjusted VAI model was also performed. This model remained statistically significant and explained 12.7% of the variance in VAI ($R^2 = 0.127$, adjusted $R^2 = 0.109$; F (7,339) = 7,024, $p < 0.001$). In this sensitivity model, neither main meal count (B = 0.012, $p = 0.947$) nor snack meal count (B = −0.268, $p = 0.084$) was independently associated with VAI, indicating that the primary conclusion regarding meal-frequency variables was unchanged after BMI adjustment.

Table 4. Multiple linear regression coefficients for AIP and VAI.

Variable	AIP			VAI		
	B	p	95% CI	B	p	95% CI
Main meal count	0.029	0.308	−0.026; 0.083	0.058	0.741	−0.288; 0.404
Snack meal count	−0.034	0.172	−0.083; 0.015	−0.274	0.080	−0.580; 0.033
Age	0.005	**<0.001***	0.002; 0.007	0.037	**<0.001***	0.022; 0.052
Male sex	0.201	**<0.001***	0.135; 0.267	0.247	0.229	−0.156; 0.650
BMI	0.010	**0.002***	0.004; 0.017	-	-	-
Smoking	−0.046	0.136	−0.106; 0.014	−0.333	0.082	−0.708; 0.042
Regular exercise	−0.076	**0.030***	−0.144; −0.008	−0.492	**0.023***	−0.917; −0.068

AIP model: $R^2 = 0.238$, adjusted $R^2 = 0.223$, $F(7,339) = 15.157$, $p < 0.001$.

VAI model: $R^2 = 0.112$, adjusted $R^2 = 0.097$, $F(6,340) = 7.180$, $p < 0.001$.

BMI-adjusted VAI sensitivity model: $R^2 = 0.127$, adjusted $R^2 = 0.109$, $F(7,339) = 7.024$, $p < 0.001$.

For VAI, the primary model was constructed without BMI because BMI is a component of the VAI formula. A BMI-adjusted VAI model was additionally performed as a sensitivity analysis and did not alter the non-significant associations of main meal count and snack meal count with VAI.

B: unstandardized regression coefficient; CI: confidence interval; BMI: body mass index; AIP: atherogenic index of plasma; VAI: visceral adiposity index. Sex was coded as 0 = female and 1 = male; smoking status and regular exercise were coded as 0 = no and 1 = yes. Significant predictors are marked with an asterisk.

Overall, although meal-frequency variables showed significant associations with AIP and/or VAI in unadjusted analyses, these associations did not persist after adjustment for demographic, anthropometric, and lifestyle-related covariates.

DISCUSSION

In this single-center cross-sectional study of 347 medication-free overweight and obese adults, neither daily main meal count nor snack frequency was independently associated with AIP or VAI in multivariable analyses. The AIP model was adjusted for age, sex, BMI, smoking status, and regular exercise, whereas the primary VAI model was constructed without BMI to avoid mathematical coupling because BMI is a component of the VAI formula. The crude associations observed in unadjusted analyses—a positive association between main meal count and AIP, and inverse associations between snack frequency and both indices—did not persist after multivariable adjustment, and the BMI-adjusted VAI sensitivity model yielded the same conclusion regarding meal-frequency variables. Considered together, these findings suggest that eating-occasion frequency, when assessed without information on dietary composition, energy intake, and meal timing, may have limited independent explanatory value for AIP or VAI in medication-free adults with excess weight.

The absence of an independent meal-frequency signal is consistent with the most recent meta-analytic evidence in adults. The systematic review by Blazey et al. [16], which pooled randomized trials comparing restricted (≤3 meals/day) with unrestricted (≥4 meals/day) eating frequency, reported no consistent advantage of either pattern on body weight or biochemical cardiometabolic outcomes, and rated the majority of included trials at high risk of bias. Abdollahi et al. [4] observed small reductions in total and LDL-cholesterol with higher meal frequency in randomized trials, but no consistent effect on triglycerides, HDL-cholesterol, or insulin homeostasis—the very components from which AIP and VAI are derived. More recently, Liu et al. [17] found that lower meal frequency, when combined with time-restricted eating or earlier caloric distribution, was associated with modestly greater weight loss; effect sizes, however, were small and heterogeneity was substantial. Our null-adjusted findings extend these intervention-based data to a clinically uncharacterized, drug-naïve outpatient population assessed before treatment-related influences could distort the diet-index relationship.

The crude inverse association between snack frequency and both indices warrants careful interpretation. Although participants reporting one snack per day had lower AIP and VAI values than those reporting no snacks, this gradient did not persist after adjustment, indicating confounding rather than a direct protective effect. Several non-causal mechanisms remain plausible. First, snack frequency may behave as a behavioral marker for

broader dietary structure—a more even distribution of energy, fiber, and micronutrient intake across the day—rather than as an independent metabolic exposure [5]. Second, the absence of inter-meal snacks may reflect prolonged post-absorptive intervals followed by larger discrete meals. This pattern can adversely affect postprandial triglyceride-rich lipoprotein handling, particularly in individuals with greater visceral adiposity [18]. Third, snack composition was not characterized; the association between snacking and cardiometabolic risk depends heavily on the food matrix consumed, with consistent evidence linking ultra-processed snack intake to adverse lipid and adiposity profiles [19]. Because we did not capture total energy intake, macronutrient distribution, snack composition, or circadian timing, these explanations remain hypothesis-generating. Another possible explanation is pre-assessment lifestyle modification. Because the study population consisted of individuals presenting for a first cardiological assessment, participants with higher perceived cardiometabolic risk may already have modified their eating habits before admission, including reducing or avoiding snacks. Therefore, lower snack frequency should not be interpreted as a stable long-term dietary exposure. In a cross-sectional design, such behavior change before clinical evaluation may produce reverse causality and may partly explain why crude associations between snack frequency and AIP/VAI disappeared after multivariable adjustment.

A further issue is that meal frequency should not be interpreted as a comprehensive nutritional exposure. Although the present study focused on eating-occasion frequency, we did not standardize or quantify participants' overall dietary intake. Diet type, total energy intake, macronutrient distribution, dietary quality, and snack composition were not assessed using a validated food frequency questionnaire, dietary recall, or dietary record. This is important because AIP and VAI are partly driven by triglyceride and HDL-cholesterol concentrations, which may be influenced not only by the number of eating occasions but also by energy balance, carbohydrate quality, fat quality, alcohol intake, and the degree of food processing. Therefore, the null adjusted association between meal frequency and AIP/VAI should be interpreted as evidence that eating-occasion count alone was not independently informative in this cohort, not as evidence that dietary composition or dietary quality is unrelated to these indices.

The exploratory sex-stratified VAI analyses suggested that the crude inverse correlation between snack frequency and VAI was confined to men, with no significant correlations among women. This pattern is biologically coherent rather than confirmatory. Men accumulate disproportionately greater intra-abdominal adipose tissue than premenopausal women at any given BMI, and this depot is the principal driver of fasting hypertriglyceridemia and of exaggerated postprandial triglyceride excursions [18,20]. Behavioral patterns that influence triglyceride-rich lipoprotein metabolism—including meal spacing—may therefore produce a more detectable signal on a triglyceride- and HDL-driven index such as VAI in men than in women, in whom subcutaneous gluteofemoral storage buffers postprandial lipid flux [18]. Sex stratification reduced statistical power, the analyses were correlation-based rather than multivariable, and the design cannot identify mediation; we therefore present this observation as hypothesis-generating only.

Regular exercise was the sole lifestyle variable independently and inversely associated with both AIP and VAI. Although exercise was recorded as a binary self-reported variable—precluding inference about modality, intensity, or volume—the direction and magnitude of the association are consistent with the well-established lipid-modifying effects of habitual aerobic and combined-modality exercise on triglyceride and HDL-cholesterol metabolism [21,22]. Because triglycerides and HDL-cholesterol are constitutive components of both indices, an exercise effect on lipid metabolism would be expected to propagate into both AIP and VAI. From a clinical standpoint, regular exercise emerged as the only modifiable lifestyle correlate to retain independent informativeness in this cohort, reinforcing the emphasis placed on physical activity in primary cardiovascular prevention guidance. The higher prevalence of reported regular exercise among participants with obesity requires cautious interpretation. Rather than indicating a lower-risk lifestyle profile, this unexpected distribution may reflect reverse causality or recent behavioral modification, whereby individuals with obesity may have initiated exercise in response to weight gain, perceived cardiometabolic risk, or prior lifestyle advice before their first cardiological assessment. Because exercise was recorded as a binary self-reported variable, the duration of this behavior, intensity, adherence, and whether it preceded or followed weight gain could not be determined. Therefore, although regular exercise was independently associated with lower AIP and VAI, this association should be interpreted as observational rather than causal.

The differential explanatory power of the two outcome models also merits comment. The AIP regression model accounted for more variance than the primary BMI-free VAI model (R^2 = 0.238 vs. 0.112). This is mechanistically plausible because AIP is a direct mathematical function of triglyceride and HDL-cholesterol values, both of which are closely related to age, sex, BMI, and exercise. VAI, in contrast, is a composite sex-specific index incorporating waist circumference, BMI, triglycerides, and HDL-cholesterol; therefore, its regression modeling requires particular caution when anthropometric variables are considered [9,15]. The lower R^2 of the primary VAI model should not be interpreted as evidence that VAI is biologically weaker, but rather as a reflection of the different mathematical structure of the index and the deliberate exclusion of BMI from the primary VAI model to avoid mathematical coupling.

An important methodological issue concerns the use of BMI in regression models in which VAI is the dependent variable. Because BMI is an explicit component of the VAI formula, entering BMI as an independent predictor may introduce mathematical coupling and complicate coefficient interpretation. For this reason, we considered the BMI-free VAI model as the primary model, while the BMI-adjusted model was retained only as a sensitivity analysis. Importantly, the lack of independent associations between meal-frequency variables and VAI was unchanged in both approaches. This consistency supports the robustness of the main conclusion regarding meal frequency, while avoiding overinterpretation of BMI coefficients in a BMI-derived outcome.

The significant age difference between overweight and obese participants should also be considered when interpreting the unadjusted group comparisons. Participants with obesity were older than those with overweight, and age is known to influence lipid metabolism, visceral adiposity, and cardiometabolic risk accumulation. Therefore, the higher AIP and VAI values observed in the obese group should not be interpreted as reflecting BMI category alone. To reduce this potential confounding, age was included as an adjustment variable in the multivariable regression models. In these adjusted analyses, age remained independently associated with both AIP and VAI, supporting its relevance as an important covariate in the interpretation of cardiometabolic indices.

Several methodological features strengthen the present analysis. The cohort was deliberately confined to medication-naïve adults without any documented chronic disease, allowing a cleaner assessment of the diet-index relationship than is possible in samples that include individuals receiving lipid-lowering, glucose-lowering, or antihypertensive therapy—conditions that have repeatedly been shown to bias dietary association estimates [4,17]. All anthropometric measurements were taken in triplicate by a single trained investigator, reducing inter-observer variability. The analytical strategy was also revised to address the concern of mathematical coupling by treating the BMI-free VAI model as the primary analysis and retaining the BMI-adjusted VAI model only as a sensitivity analysis. Both AIP and VAI are validated lipid- and adiposity-derived indices that perform robustly as cardiometabolic risk markers in observational cohorts and have been associated with incident cardiovascular disease and mortality [9,10,23.24].

Limitations

Several limitations should be considered. First, the cross-sectional design precludes any inference about temporal direction or causality; individuals with less favorable metabolic profiles or higher perceived cardiometabolic risk may have modified their eating behavior before their first cardiological assessment, including reducing or avoiding snacks. In addition, the significant age difference between BMI groups may have influenced unadjusted comparisons of cardiometabolic indices, although age was included as a covariate in the multivariable regression models. Second, meal frequency was captured using a simple ordinal interview variable that recorded eating occasions but did not capture portion size, total energy intake, macronutrient distribution, dietary pattern, dietary quality, snack composition, food processing level, eating speed, or meal timing. No validated food frequency questionnaire, 24-hour dietary recall, dietary record, or caloric assessment was used. Therefore, dietary standardization and caloric adjustment could not be performed, and residual confounding by overall nutritional status and dietary composition cannot be excluded. Third, physical activity, smoking, and alcohol use were self-reported and may be subject to recall or social desirability bias. In addition, alcohol consumption was assessed only as a binary self-reported variable and was not incorporated into the primary regression models because of its low prevalence and the absence of quantitative information on alcohol dose, frequency, beverage type, or drinking pattern. Therefore, residual confounding related to alcohol intake cannot be fully excluded. Moreover, regular exercise was recorded only as a binary variable, without information on duration, intensity, modality, adherence, or

whether exercise behavior had been recently initiated in response to weight gain or perceived cardiometabolic risk. Fourth, because BMI is incorporated into the VAI formula, including BMI as an independent predictor in VAI regression models may introduce partial mathematical coupling. To address this issue, the BMI-free VAI model was considered the primary model, whereas the BMI-adjusted model was retained only as a sensitivity analysis. The lack of independent associations between meal-frequency variables and VAI was unchanged across both approaches. Fifth, 27 participants had fasting glucose values at or above 126 mg/dL, the diagnostic threshold for diabetes; although none had a formal diabetes diagnosis and none had HbA1c values above 6.5%, their inclusion introduces some heterogeneity into the intended cardiometabolic-disease-free framing. A sensitivity analysis restricted to participants with fasting glucose below 126 mg/dL produced results qualitatively similar to those of the full sample. Finally, the single-center cardiology outpatient setting may limit generalisability to primary-care or community-based populations.

CONCLUSION

In drug-naïve overweight and obese adults without a prior cardiometabolic diagnosis, daily eating-occasion count alone did not provide independent information for AIP- or VAI-based risk stratification after adjustment for demographic, anthropometric, and lifestyle-related covariates. In this cross-sectional cohort, age, sex, BMI, and habitual exercise appeared to be the most consistently associated factors with AIP and/or VAI, whereas reported eating frequency did not provide independent information after adjustment for these covariates. Therefore, eating frequency should be interpreted cautiously and considered mainly as a crude marker of unmeasured dimensions of dietary structure, including energy intake, food matrix, and circadian timing. Future studies should integrate detailed dietary assessment, separate eating-occasion frequency from meal size and dietary quality, characterize snack composition, and ideally use prospective designs to clarify temporal relationships.

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Conflict of Interest: The authors declare that they have no conflict of interest.

Informed Consent: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: The data supporting the findings of this study are not publicly available due to institutional and ethical restrictions. The data were obtained from hospital electronic medical records and analyzed in an anonymized form. Access to the data may be considered upon reasonable request to the corresponding author, subject to approval by the hospital administration.

Ethical Approval: This study was conducted in accordance with the Declaration of Helsinki and approved by the Gaziantep University Non-Interventional Clinical Research Ethics Committee (Decision No. 2026/67, January 21, 2026). Written informed consent was obtained from all participants prior to their inclusion in the study. The study followed the STROBE reporting guidelines.

Author Contributions: Conception: NOL; OO - Design: NOL; OO - Supervision: NOL; OO; MDS - Materials: NOL; OO; MDS - Data Collection and/or Processing: NOL; OO; MDS - Analysis and/or Interpretation: NOL - Literature: NOL; OO - Review: NOL; OO; MDS - Writing: NOL; OO - Critical Review: NOL; OO; MDS

Author Statement Regarding the Use of Artificial Intelligence-Enhanced Tools: Artificial intelligence-based tools were used exclusively for linguistic refinement, including improvements in grammar, clarity, and overall readability. No artificial intelligence systems were involved in the study design, data analysis, interpretation of results, or the generation of scientific content.

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