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Association Between Self-Reported Diabetic Diet and LDL Cholesterol Levels in U.S. Overweight and Obese Adults: A Cross-Sectional Analysis of NHANES 2017–2018

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Abstract

Background: *High levels of low-density lipoprotein cholesterol (LDL-C) and diabetes are major cardiovascular risk factors. Dietary modifications are commonly recommended as part of diabetes management. However, in real-world populations, the association between self-reported adherence to a diabetes-focused diet and LDL-C levels remains unclear.*

Objective: *To examine the cross-sectional association between a self-reported diabetic diet (SRDD) and LDL-C levels in U.S. overweight and obese adults, and to explore potential mediators and effect modifiers of this relationship using nationally representative data.*

Results: *Of the 2,150 adults, 52 participants reported following a diabetic diet. In unadjusted analyses, SRDD was associated with significantly lower LDL-C levels (mean difference = -18.6 mg/dL; 95% CI -28.4 to -8.8; $p < 0.001$). After adjustment for metabolic and treatment-related variables, including diabetes status, statin use, and HbA1c, the association was attenuated and no longer statistically significant ($\beta = -8.4$; $p = 0.3$). Statin use showed the strongest association with lower LDL-C levels ($\beta = -26.6$ mg/dL; 95% CI -30.7 to -22.4; $p < 0.001$). Effect modification analysis indicated that diabetes status significantly modified the association between diet and LDL-C (p for interaction $= 0.024$), with an inverse association observed primarily among participants with diabetes. Mediation analyses indicated that body mass index (BMI) significantly mediated the association between diet and LDL-C ($p = 0.007$), whereas HbA1c did not demonstrate evidence of mediation.*

Conclusions: *While adherence to a self-reported diabetic diet was associated with lower LDL-C levels in unadjusted analyses, this association was attenuated after accounting for metabolic and treatment-related factors. Mediation analyses suggest that body mass index may partially explain this relationship, indicating that the association between diet and LDL-C could operate through broader metabolic mechanisms. In contrast, HbA1c did not appear to mediate the association. Overall, these findings highlight the complex interplay between dietary behaviors, metabolic status, and clinical management in cross-sectional populations and underscore the need for longitudinal studies to clarify causal pathways.*

Introduction

Dyslipidemia is a common condition in patients with type 2 diabetes, with a prevalence as high as 60% in some studies (Addis et al., 2024). It is thought to significantly contribute to the cardiovascular risk associated with diabetes. High low-density lipoprotein cholesterol (LDL-C) levels, a key feature of dyslipidemia, are also frequently observed in patients with obesity and cardiovascular disease (CVD) (Bays et al., 2024). This cluster of conditions is critical, as CVD is a leading cause of morbidity and mortality worldwide (The GBD 2015 Obesity Collaborators, 2017). In the United States, approximately 94 million adults have high cholesterol levels, highlighting the magnitude of the public health burden (Tsao et al., 2023). Elevated LDL-C levels are a major modifiable risk factor for atherosclerotic CVD (Abera et al., 2024). Therefore, LDL-C reduction remains a cornerstone of CVD prevention, and while pharmacologic therapy is often necessary, lifestyle modification including diet remains the first-line recommendation and broadly applicable strategy in both clinical practice and public health policy (Ference et al., 2017).

Current guidelines do not suggest a specific diet but recommend nutritional evaluation and healthy lifestyle modifications (American Diabetes Association Professional Practice Committee, 2025). As a cornerstone of treatment, nutritional intervention often involves calorie reduction and avoiding carbohydrates that stimulate excessive insulin demand (Freeman et al., 2023). Consequently, personalized diet is the treatment backbone for individuals with diabetes, obesity, overweight, and metabolic disorders (Delahanty et al., 2009). In concern of diabetic patients, the diet is usually characterized by controlled carbohydrate intake, high fiber consumption, reduced saturated fats, and the promotion of complex carbohydrates. Understanding its potential impact on lipid profiles, particularly LDL-C, is therefore of significant clinical and scientific importance. Previous studies were often unable to distinguish the independent effect of diet, as they did not utilize stratified or sensitivity analyses to separate it from other factors like medication use, body mass index (BMI), or glycemic control (Liu & Li, 2015; Okumura et al., n.d.). Nevertheless, there is a gap in literature about the impact of a diabetic diet in LDL-C levels. This points to a clear need for research that utilizes comprehensive, large-scale data to separate the possible contribution of diet from other metabolic and treatment-related influences. The aim of this study is to investigate the relationship between a Self-Reported Diabetic Diet (SRDD) and LDL-C level.

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Materials and Methods

This study employed a cross-sectional design using data from the National Health and Nutrition Examination Survey (NHANES), conducted by the National Center for Health Statistics (NCHS). Across multiple U.S locations, 2017-2018 wave was chosen for analysis. NHANES uses a complex, multistage probability sampling strategy to produce data representative of the non-institutionalized U.S. population. Information is collected through structured household interviews, standardized physical examinations, and laboratory testing. The survey was carried out with IRB-approved data. All study protocols are approved by the NCHS Research Ethics Review Board, and informed consent is obtained from all participants.

Demographic, questionnaire, physical examination, and laboratory data were used to analyze the association between consumption of diabetic diet and LDL-C levels among adults with overweight or obesity.

Adults aged 18 years and older were categorized by gender, BMI, medication use, dyslipidemia, personal history of diabetes mellitus (DM) and glycated hemoglobin (HbA1c). LDL-C levels were calculated through the Friedewald equation.

The analytic sample included participants with complete data on LDL-C, SRDD, and BMI. Exclusion criteria were: (i) missing or incomplete data on exposure or outcome variables; (ii) participants that reported following more than one diet.

All analyses were performed using Stata version 19.5 (StataCorp LLC, College Station, TX, USA). Continuous variables were presented as mean and standard deviation (SD), for normally distributed data, and median and interquartile range (IQR) for non-normally distributed data. Univariate analysis was performed using the t-test for categorical variables and chi-square for non-categorical variables for normally distributed data. NHANES sampling weights were applied to account for the complex survey design. Sensitivity and subgroup analyses were also conducted.

A linear regression model was used to examine the association between consumption to a SRDD (exposure) and LDL-C levels (outcome). Models were adjusted for potential confounders, including age, BMI, presence of diabetes, HbA1c, and statin use. We conducted multivariable analyses to adjust for

potential confounders, employing multiple linear regression. Tests for assumptions were made for linearity, homoscedasticity and normality of residuals. Missing data were handled through listwise deletion, and statistical significance was set at $p < 0.05$.

Subgroup analyses were conducted to explore potential effect modification by age ($\geq$50 years) and presence of diabetes. Mediation analyses were performed to assess whether BMI, HbA1c, or statin use mediated the relationship between consumption to a diabetic diet and LDL-C levels.

The primary exposure was SRDD derived from NHANES dietary questionnaire files. Responses were recoded into a binary variable: following a diabetic diet versus not following a diabetic diet. The primary outcome was LDL-C, measured in mg/dL using standardized enzymatic methods at certified laboratories.

Results

Study population characteristics

A total of 2,150 participants were included. 52 reported following a diabetic diet and 2,098 did not. After applying NHANES survey weights, this sample was representative of the U.S. adult population from 2017–2018. Baseline characteristics are shown in Table 1. The mean age of participants was 50.1±18.5 years, with those following a diabetic diet being significantly older (62.5 ± 12.9) than non-followers (50 ± 18.7, $p < 0.001$). Overall, gender distribution was balanced (51.4% female) and similar across diet groups. The mean BMI was 29.2±7.2 kg/m², where 48.1% of the patients following a diabetic diet were obese, and in the group without the diet only 39% were considered obese, without significant differences between groups (p=0.2). LDL-C levels were 109.8 ± 35.6 mg/dL. Participants who followed a diabetic diet had significantly lower LDL-C levels (91.7 ± 32 mg/dL) compared to non-followers (110.3 ± 35.6 mg/dL; $p < 0.001$). Statin use was reported by 17.5%, among those following a diabetic diet (38.5%) than among those not (17%). A total of 15.5% had a known diagnosis of diabetes, and among diabetic diet followers, 92.3% had diabetes. The mean HbA1c was 5.8 ± 1.1%, which is higher in the diet group (7.7 ± 1.6).

Relationship between SRDD and LDL-C levels

In unadjusted analyses, participants who reported adherence to a self-reported diabetic diet (SRDD) had significantly lower LDL-C levels compared to non-followers (mean difference = –18.6 mg/dL; 95% CI –28.4 to –8.8; $p < 0.001$). However, the unadjusted model explained only a small proportion of the variability in LDL-C levels ($R^2 = 0.006$), indicating that SRDD alone accounted for approximately 0.6% of the variation in LDL-C.

In the adjusted multivariable analysis (Table 2), after accounting for age, statin use, diabetes diagnosis, and HbA1c, the model was globally significant ($F = 50.40$; $p < 0.001$) and explained approximately 11% of the variability in LDL-C levels. Statin use showed the strongest association with LDL-C, with an average reduction of 27 mg/dL ($p < 0.001$). A known diagnosis of diabetes was also associated with lower LDL-C levels ($\beta = –15.8$; $p < 0.001$), likely reflecting treatment-related factors. In contrast, age and HbA1c were significantly associated with higher LDL-C levels ($p < 0.001$). Although SRDD was associated with lower LDL-C levels ($\beta = –8.4$), this association did not reach statistical significance after adjustment ($p = 0.3$), suggesting that the unadjusted association was influenced by metabolic and treatment-related factors.

The final regression models included a smaller sample size than the descriptive analyses due to exclusion of participants with missing data on covariates or survey design variables, consistent with automatic casewise deletion in STATA.

Covariates were included based on biological plausibility and prior evidence linking dietary patterns and lipid metabolism. Age was included due to its established association with lipid levels and cardiovascular risk. BMI was evaluated as a potential mediator, given its relationship with both dietary behaviors and lipid metabolism. Statin use and diabetes-related variables, including diabetes diagnosis and HbA1c, were included due to their close association with LDL-C levels and diabetes management.

Secondary/subgroup analysis

We conducted additional analyses to explore potential effect modification and mediation. Interpretation of these effects is based on prior evidence supporting the metabolic and pharmacological pathways.

Effect modification

We tested whether age $\geq$50 years and presence of diabetes modified the association between SRDD and LDL-C levels. Age $\geq$50 years did not significantly modify the effect ($p > 0.05$), indicating that the association was similar across age groups. In contrast, diabetes status significantly modified the association between SRDD and LDL-C (β for

	Total (N = 2150)	Self-Reported Diabetic Diet (n = 52)	No Diet (n = 2098)	p-value
Age (years), Mean (SD)	50.1 (18.5)	62.5 (12.9)	50 (18.7)	<0.001*
Female gender, n (%)	1105 (51.4)	27 (52.9)	1078 (51.4)	0.9ǂ
BMI (kg/m²), Mean (SD)	29.2 (7.2)	31.0 (6.3)	29.1 (7.2)	0.06*
Obese patients (BMI >30%) n(%)	848 (39.4)	25 (48.1%)	823 (39.2%)	0.9ǂ
HbA1c, Mean (SD)	5.8 (1.0)	7.7 (1.6)	5.8 (1.0)	<0.001*
Statin use (yes), n(%)	376 (17.5)	20 (38.5)	356 (17)	<0.001ǂ
Known diagnosis of diabetes (yes), n (%)	323 (15.5)	47 (92.2)	276 (13.6)	<0.001ǂ
LDL (mg/dL), Mean (SD)	109.8 (35.6)	91.7 (32)	110.3 (35.6)	<0.001*

SD: standard deviation, DM: diabetes mellitus, HbA1c: glycated hemoglobin. LDL: low-density lipoprotein. BMI: body mass index. * t-test ǂ chi-square test.

Table 1: *Demographics and baseline characteristics of study participants.*

Covariates	β (95% CI)	P-Value
Age (per decade)	2.9(2.0,3.7)	< .001
Self-reported diabetic diet (yes)	--8.4 (−18.3 1.4)	0.3
HbA1c (%)	5.4 (3.7, 7.1)	< .001
Statin use (yes)	−26.6 (−30.7., −22.4)	< .001
Known diagnosis of diabetes (yes)	−23.7 (−29.0, −18.4)	< .001

Model statistics: N = 2,148; Adjusted R^2 = .11.
BMI: body mass index, HbA1c: glycated hemoglobin.

Table 2: *Multivariable linear regression of LDL-C (mg/dL) on diabetic diet and covariates (NHANES 2017–2018).*

interaction = –33.86, 95% CI –66.56 to –1.17; p = 0.024), indicating that the inverse association between diabetic diet and LDL-C was present primarily among participants with diabetes.

Mediation analyses

We evaluated potential mediators of the association between SRDD and LDL-C, including BMI, and HbA1c. BMI significantly mediated this relationship (p = 0.007), indicating that SRDD was associated with lower BMI, which in turn was associated with lower LDL-C levels. In contrast, HbA1c did not demonstrate evidence of mediation.

Sensitivity analyses

Stratified by diabetes status, the association of SRDD and LDL-C showed opposite directions. Among non-diabetic participants, the diet was associated with a non-significant increase in LDL-C levels (β = 23.2, 95% CI: -9.4 to 55.8, p = 0.16), whereas among diabetics, the association was in the opposite direction (β = -10.7, 95% CI: -21.7 to 0.3, p = 0.057). These results suggest a potential effect modification by diabetes status, although neither association reached statistical significance.

Discussion

This study evaluated the association between SRDD and LDL-C levels in U.S. adults with overweight or obesity, using data from the 2017–2018 NHANES cycle. Even though the initial unadjusted analysis suggested that individuals who reported following a diabetic diet had significantly lower LDL-C levels (18.6 mg/dL) compared to non-followers (p < 0.001), the adjusted analysis accounting for key metabolic and treatment-related factors attenuated this association. The association between SRDD and LDL-C was no longer statistically significant (β = -5.8; p = 0.25), suggesting that the observed relationship is closely related with metabolic status and clinical management factors. Notably, statin use and known diagnosis of diabetes showed strong independent associations with LDL-C levels, likely reflecting the impact of medical treatment. Also, we conducted subgroup and mediation analyses, which revealed that diabetes presence significantly modified the association between SRDD and LDL-C levels, while age ≥50 did not. BMI significantly mediated the relationship, suggesting that metabolic factors related to body composition may partially explain the association, whereas HbA1c did not demonstrate evidence of mediation. In our study, older participants more frequently reported following a diabetic diet. A plausible explanation is that physicians and

nutritionists more often prescribe structured dietary plans to older adults with metabolic comorbidities, increasing the likelihood that they self-identify as following a diabetic diet.

These findings are consistent with prior evidence showing that structured dietary patterns can reduce LDL-C and improve cardiometabolic outcomes. Filippou et al. (2024) demonstrated that both diets - Dietary Approaches to Stop Hypertension (DASH) and the Mediterranean diet (MedDiet) - significantly reduced LDL-C and HbA1c among hypertensive adults. Nevertheless, these results were obtained in controlled settings, which could explain the difficulties of finding positive results in real-world conditions, probably associated with other variables. In our study, the association between SRDD and LDL-C was modest and lost statistical significance after adjusting for confounders, highlighting the influence of pharmacologic treatment and metabolic factors (Bonekamp et al., 2024).

Prior NHANES analyses have shown that statin use remains suboptimal among adults who meet treatment criteria, which may influence observed LDL-C levels and their relationship with lifestyle factors (Thompson-Paul et al., 2023). Additionally, evidence suggests that LDL-C rises with BMI primarily in lean individuals, whereas metabolic impairment may attenuate the expected lipid response to dietary changes (Laclaustra et al., 2018). Taken together, these results suggest that the impact of dietary interventions on LDL-C is context-dependent, influenced by clinical status, pharmacologic therapy, and body composition.

Our study focused on SRDD in both diabetic and non-diabetic individuals, which may explain the modest effect size observed but this is clinically relevant as cholesterol metabolism differs substantially between these groups. Diabetic patients, for instance, demonstrate lower cholesterol absorption efficiency and higher synthesis rates, which may mitigate the lipid-lowering effect of dietary interventions alone (Simonen et al., 2002). However, a network meta-analysis of dietary interventions in patients with established CVD found no significant long-term effect on LDL-C, suggesting that dietary effects may be attenuated in medically treated populations (Bonekamp et al., 2024).

This study provides important insights into the relationship between consumption to a diabetic diet and LDL-C levels in a diverse adult population, including non-diabetic individuals, a distinction that matters given metabolic differences in cholesterol absorption and synthesis (Simonen et al., 2002). Strengths include the use of nationally representative NHANES data, objective LDL-C measurements, and robust statistical adjustments, including mediation and effect modification analyses.

However, the cross-sectional design limits causal inference, and self-reported dietary data may introduce recall bias and misclassification. The small number of participants who reported following the diet (n = 52) reduces statistical power. Collinearity between diabetes status and HbA1c limits the interpretation of their independent effects. Given the close biological and clinical relationship between these variables, separating their individual contributions within regression models may be challenging. Even though HbA1c could have collinearity with diabetes diagnosis, there is a possibility that patients may have elevated HbA1c levels with unknown diabetes diagnosis, as well as diabetic patients may be adequately controlled, and consequently, have HbA1c within normal range.

NHANES is a descriptive survey that relies heavily on self-reported information. In our analysis, "diabetic diet" was based on participants' self-report rather than on standardized dietary assessment or independent verification. Consequently, we could not confirm actual calorie intake, carbohydrate content or the degree of adherence to dietary recommendations at the individual level. This limitation should be considered when interpreting our findings.

It was not possible to determine whether participants with familial hypercholesterolemia (FH) were included in our analysis, as NHANES does not provide a specific variable or diagnostic code identifying this condition. Therefore, the presence of individuals with FH could not be confirmed or excluded, even though it is an important genetic cause of elevated LDL-C levels and it may influence the observed associations. Moreover, current guidelines recommend statins for primary prevention in individuals with diabetes without established CVD; the recommendation of moderate-intensity therapy may bias LDL-C measurements in the diabetic group.

Conclusion

While the SRDD showed a trend toward lower LDL-C and remains as an important component of obesity and cardiovascular risk management, the lack of statistical significance after adjustment suggests that diet alone may not be sufficient for lipid management in populations with metabolic alterations or high statin use. This highlights for clinicians the need to integrate dietary strategies with pharmacologic therapy rather than considering them as alternatives. Findings are broadly generalizable to U.S. adults with overweight or obesity, given NHANES

sampling design. However, the small number of SRDD followers and reliance on self-reported data may limit applicability to populations with different dietary behaviors or healthcare access.

Future research should incorporate objective measures to assess diabetic diet adherence such as detailed food diaries, digital tracking tools, or nutritional biomarkers, to minimize biases inherent to self-reported data. Longitudinal study designs are also recommended to determine whether changes in diet lead to changes in LDL-C, allowing for causal inference. Broader cardiometabolic outcomes such as triglycerides, high-density-lipoprotein cholesterol, glycemic control, and inflammation should be considered to capture the full impact of dietary interventions. Additionally, investigating gene–diet interactions, cholesterol metabolism pathways, and stratification by pharmacologic treatment could help explain individual differences in lipid response and clarify the mechanisms underlying the observed associations.

Supplementary Materials

Supplementary Figure

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Conflicts of Interest

The authors declare no conflict of interest.

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