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ORIGINAL RESEARCH

Association Between Remnant Cholesterol and Endometriosis Findings from NHANES 1999–2006

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Background: Endometriosis is a chronic gynecological disorder causing significant morbidity and health burden. Recent evidence suggests that lipid metabolism, particularly remnant cholesterol (RC), may be involved in its development. RC—the cholesterol content of triglyceride-rich lipoproteins (TGRLs), including chylomicrons, very low-density lipoproteins (VLDL), and intermediate-density lipoproteins (IDL). Elevated RC is closely associated with arteriosclerosis and increased cardiovascular disease incidence, making it a valuable biomarker for assessing cardiovascular health. This study explores the association between RC levels and endometriosis prevalence in a representative sample of women.

Methods: Using cross-sectional data from NHANES (1999–2006), we analyzed 1,979 women aged 20–54 years. Weighted logistic regression models were applied to assess the relationship between RC and endometriosis. Three models were used: unadjusted (Model 1), adjusted for age, ethnicity, education, and marital status (Model 2), and fully adjusted for additional variables (Model 3). Subgroup analyses and smoothing curve fitting were also performed.

Results: Elevated RC levels were significantly associated with higher endometriosis prevalence across all models. Each 1 mg/dL above the mean level of RC was linked to a 2.3% higher incidence of endometriosis (Model 1: OR 1.023, 95% CI 1.009–1.036, p = 0.001; Model 2: OR 1.022, p = 0.004; Model 3: OR 1.023, p = 0.022). Individuals in the highest RC tertile had a higher incidence of endometriosis compared to the lowest tertile (OR 1.833, p for trend = 0.077). A linear dose-response relationship was identified, with no threshold or saturation effects.

Conclusion: The study indicates a strong correlation between elevated RC levels and increased endometriosis incidence, highlighting the potential role of lipid metabolism in endometriosis development.

Keywords: remnant cholesterol, endometriosis, lipid metabolism, inflammation, NHANES

Introduction

Endometriosis is a persistent gynecological condition affecting approximately 10–15% of women during their reproductive years.[1–3] Characterized by the presence of endometrial-like tissue outside the uterus, it leads to symptoms such as pelvic pain, dysmenorrhea, and infertility.[2,4,5] Despite extensive research, the precise causes of endometriosis remain elusive. Several theories have been proposed, including retrograde menstruation, coelomic metaplasia, and the induction hypothesis.[6,7] More recently, studies have focused on oxidative stress as a potential contributing factor, which arises when the production of reactive oxygen species (ROS) exceeds the body's capacity for neutralization and repair.[8]

Oxidative stress is believed to contribute to the pathophysiology of endometriosis6,8 by triggering a widespread inflammatory response within the peritoneal cavity, which may facilitate the growth and implantation of ectopic endometrial tissue.[1] Emerging evidence suggests that lipid metabolism, particularly the metabolism of RC, may also

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play a critical role in the development of endometriosis. RC refers to the cholesterol present in remnant lipoproteins, which are derived from the intravascular processing of triglyceride-rich lipoproteins (TRLs) such as chylomicrons and VLDLs.[9] While a growing body of evidence links RC to cardiovascular diseases,10,11 its role in endometriosis remains largely unexplored.

This research endeavors to address this knowledge gap by investigating the correlation between RC levels and the prevalence of endometriosis using data from NHANES 1999–2006. By examining the relationship between RC and endometriosis, this study aims to shed new light on the potential involvement of lipid metabolism in the development of endometriosis. This insight could lay the groundwork for innovative therapeutic approaches targeting RC and related metabolic pathways.

Methods

Study Design and Population

The NHANES database, a comprehensive health and nutrition survey conducted by the Centers for Disease Control and Prevention (CDC), provides detailed data on the US population. For this study, we focused on 2,147 women aged 20 to 54 years who participated in the survey between 1999 and 2006. Originally, the NHANES included 41,474 individuals during this period, involving voluntary participation from diverse communities across the United States. After excluding male participants and women outside the specified age range (n=34,966), as well as those lacking data on remnant cholesterol (RC) and endometriosis (n=4,110), and those with missing information on other covariates (n=251), our final analysis included 2,147 participants (Figure 1).

For comprehensive details on the measurement techniques and data collection procedures for each variable, please refer to the CDC's NHANES website at www.cdc.gov/nchs/nhanes.

Remnant Cholesterol (RC)

In this study, remnant cholesterol (RC) was identified as the independent variable for exposure. Morning blood samples were collected from participants who had fasted for a minimum of eight hours. Triglyceride and total cholesterol (TC) concentrations in serum were determined enzymatically. Serum high-density lipoprotein cholesterol (HDL-C) levels were assessed using the heparin-manganese precipitation method or direct immunoassay, while low-density lipoprotein cholesterol (LDL-C) levels were calculated using the Friedewald equation. The serum RC concentration was subsequently calculated using the established formula [RC = TC - LDL-C - HDL-C], consistent with previous research methodologies.[10–12]

Endometriosis

Participants completed the "rhq360" questionnaire during their visit to the Mobile Examination Center (MEC). The survey included the question: "Has a doctor or other health professional ever informed you that you have endometriosis?" Those who responded affirmatively were categorized into the case group, while those who answered negatively constituted the control group. Participants reported their endometriosis status voluntarily, allowing us to establish a binary variable indicative of a history of endometriosis diagnosis.

Covariables

Control variables comprised PIR, smoking habits, hypertension, diabetes, drinking status, BMI, HDL-C, LDL-C, and the use of contraceptive pills.

Statistical Analysis

NHANES employs a representative sampling strategy, with standard errors indicating sample-to-population discrepancies. Demographic data are presented as mean values ± standard errors for continuous variables and as frequencies (percentages) for categorical variables. Group differences in continuous variables were assessed using ANOVA, while categorical variables were evaluated with the chi-square test.

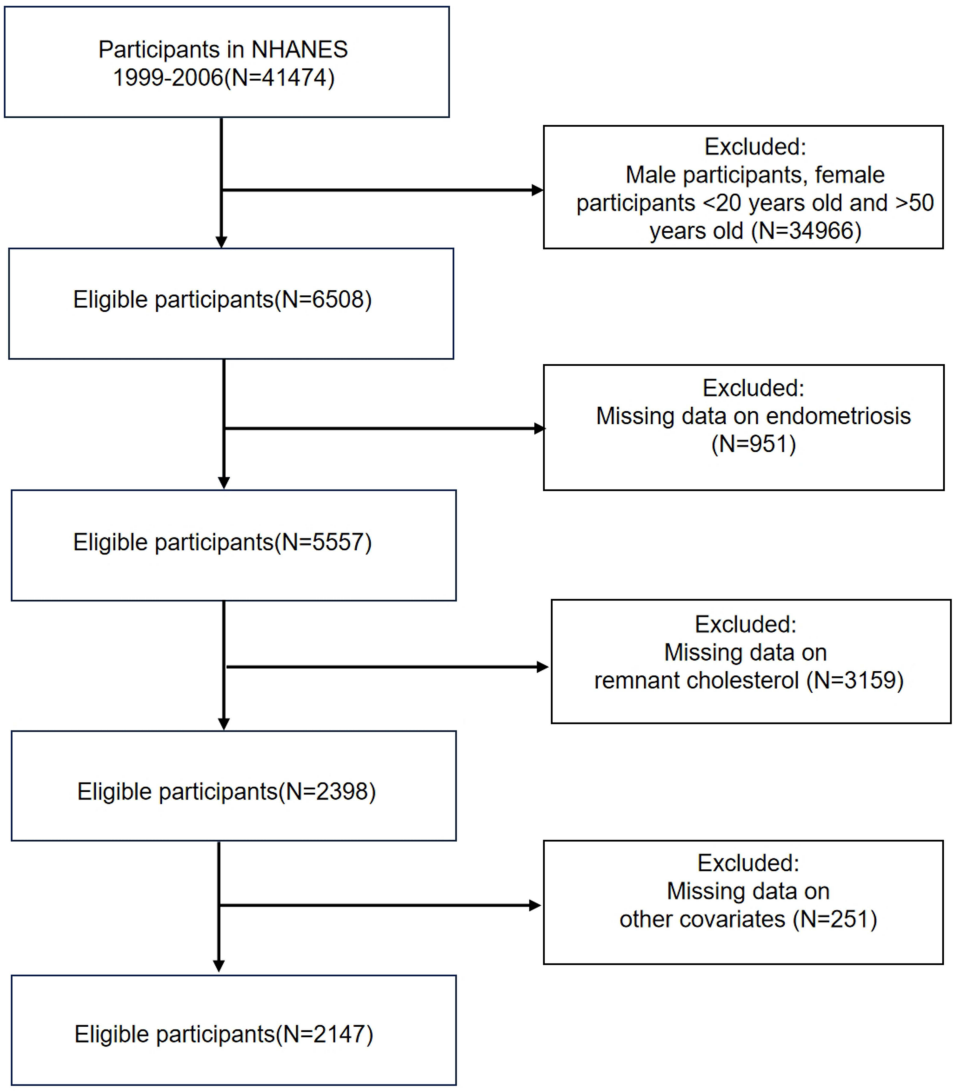

Figure 1 Flow chart of eligible participants' selection.

To explore the nonlinear association between remnant cholesterol (RC) and endometriosis, we utilized smoothing curve fitting. A weighted analysis was conducted using NHANES-recommended weights to ensure statistical precision. Our analysis was structured into three models: the unadjusted Model 1, Model 2 adjusted for significant variables identified through univariate logistic regression, and Model 3 further refined with all relevant covariates (PIR, smoking habits, hypertension, diabetes, drinking status, BMI, HDL-C, LDL-C, and the use of contraceptive pills). Statistical analyses were performed using R (version 4.2) and EmpowerStats (version 5.0), with statistical significance defined as a two-tailed p-value less than 0.05.

Results

Initial Characteristics

In this study, among 2,147 participants aged 20 to 54 years, 168 (7.82%) were diagnosed with endometriosis. Participants with endometriosis were generally older, with an average age of 40.41 years compared to 37.31 years for those without the condition, a statistically significant difference ($p < 0.001$). Additionally, the prevalence of smoking was notably higher in the endometriosis group (58.2% vs 40.0%, $p < 0.001$).

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Racial disparities were also apparent, with non-Hispanic White women being disproportionately affected by endometriosis (83.3% vs 69.0%, p = 0.001), while Mexican American women had significantly lower rates (1.3% vs 8.4%). Clinically, total cholesterol and remnant cholesterol levels were significantly higher in the endometriosis group (p = 0.032 and p = 0.006, respectively), indicating that these factors may be associated with the investigated condition, although causal inference is precluded by the cross-sectional design. These univariate comparisons should be viewed as exploratory and may be subject to selection bias, reverse causation, and unmeasured confounding; thus they serve primarily to generate hypotheses for longitudinal evaluation. Formal effect estimates are presented in the adjusted logistic regression models (Table 1), which better account for demographic and clinical confounders.

Table 1 Characteristics of Participants

Characteristics	Non-Endometriosis (N=1979)	Endometriosis (N=168)	P value
Age (years)	37.31 (10.01)	40.41 (8.00)	<0.001
PIR	3.04 (1.61)	3.16 (1.66)	0.400
BMI (kg/m2)	28.20 (7.35)	28.24 (6.56)	0.952
Smoked ≥ 100 cigarettes			<0.001
Yes	732 (40.0%)	85 (58.2%)	
No	1247 (60.0%)	83 (41.8%)	
Race/ethnicity			0.001
Mexican American	472 (8.4%)	9 (1.3%)	
Other Hispanic	84 (4.6%)	5 (1.9%)	
Non-Hispanic White	932 (69.0%)	116 (83.3%)	
Non-Hispanic Black	408 (12.5%)	32 (9.7%)	
Other	86 (5.5%)	6 (3.8%)	
Marital status			0.196
Married/Living with partner	1336 (67.1%)	114 (73.4%)	
Never married	274 (17.9%)	31 (10.3%)	
Widowed/Divorced/Separated	369 (15.0%)	23 (16.3%)	
Education level			0.075
<High school	171 (4.6%)	2 (1.0%)	
Completed high school	292 (10.6%)	19 (9.4%)	
Above high school	1516 (84.8%)	147 (89.6%)	
Hypertension			0.473
Yes	345 (18.9%)	45 (21.2%)	
N0	1634 (81.1%)	123 (78.8%)	
Diabetes			0.423
Yes	80 (4.1%)	5 (2.5%)	
No	1899 (95.9%)	163 (97.5%)	
Alcohol use			0.418
Yes	1209 (68.1%)	115 (72.5%)	
No	770 (31.9%)	53 (27.5%)	
Total cholesterol (mg/dL)	193.58 (38.34)	201.99 (39.19)	0.032
HDL-C (mg/dL)	58.22 (15.80)	57.19 (16.22)	0.595
LDL-C (mg/dL)	112.93 (33.71)	118.28 (34.59)	0.071
RC (mg/dL)	22.43 (12.14)	26.51 (14.96)	0.006

Abbreviations: PIR, family poverty income ratio; BMI, Body mass index; HDL-C, High density lipoprotein cholesterol; LDL-C, Low density lipoprotein cholesterol; RC, Remnant cholesterol.

In the present NHANES 1999–2006 subsample, higher age, smoking burden, and lipid disturbances were statistically associated with self-reported endometriosis; these descriptive patterns should be regarded as hypothesis-generating and require confirmation in prospective, clinically validated cohorts (Table 1).

Although the observed prevalence (7.8%) aligns with population-based estimates (5–10%), the absolute number of self-reported cases (n = 153) is modest; consequently, effect estimates may be subject to wider random error and should be interpreted cautiously.

The Link Between Remnant Cholesterol and Endometriosis

Table 2 demonstrates a notable correlation between remnant cholesterol (RC) levels and the prevalence rate of endometriosis across various models. In Model 1, which is unadjusted, each 1 mg/dL increase in RC was associated with a 2.3% higher prevalence rate of endometriosis (OR 1.023, 95% CI 1.009–1.036, p = 0.001). This correlation persisted in the adjusted Model 2, which accounted for age, ethnicity, education level, and marital status, with a slightly reduced but still significant odds ratio (OR 1.022, 95% CI 1.007–1.037, p = 0.004). Even after further adjustments in Model 3 for additional covariates such as poverty income ratio (PIR), smoking habits, hypertension, diabetes, drinking status, BMI, HDL-C, LDL-C, and contraceptive pill use, the correlation remained statistically (OR 1.023, 95% CI 1.003–1.044, p = 0.022).

Notably, participants in the third tertile of RC had a markedly higher prevalence rate of endometriosis compared to those in the lowest tertile, with an odds ratio (OR) of 1.833 in the fully adjusted model (p for trend = 0.077). These findings indicate a modest, non-significant trend (OR 1.833, p for trend = 0.077) between the highest RC tertile and endometriosis prevalence; consequently, this association should be interpreted cautiously and warrants confirmation in larger, prospectively ascertained cohorts, underscoring the potential role of lipid metabolism in the disease's pathogenesis (Table 2). In addition, we also included body weight /BMI in Model 2 for sensitivity analysis (Supplementary Table 1). The results are consistent with our initial findings, indicating that the link between residual cholesterol and endometriosis is solid. Moreover, in the RCS model, a linear dose-response relationship between RC and endometriosis was determined (P for nonlinear =0.847) (Figure 2). Although the observed prevalence (7.8%) is consistent with population estimates, the absolute number of self-reported EM cases (n = 153) is modest; consequently, effect estimates are imprecise and should be interpreted cautiously.

Subgroup Analyses

We conducted stratified analyses and interaction assessments by ethnicity, smoking status, alcohol consumption, marital status, and oral contraceptive use to examine the consistency of the association between RC and endometriosis across different demographic groups (Figure 3). The analysis revealed no significant differences in the association across any subgroups (p for interaction > 0.05). This suggests a uniform inverse correlation between RC levels and endometriosis rates across all demographic groups studied.

Table 2 Association Between RC and Endometriosis

	OR (95% CI) P		
	Model 1	**Model 2**	**Model 3**
Remnant cholesterol	1.023 (1.009–1.036) 0.001	1.022 (1.007–1.037) 0.004	1.023 (1.003–1.044) 0.022
Categories			
T1	Reference	Reference	Reference
T2	1.142 (0.692–1.884) 0.599	1.129 (0.677–1.884) 0.636	1.100 (0.634–1.910) 0.728
T3	1.951 (1.279–2.975) 0.002	1.849 (1.183–2.891) 0.008	1.833 (1.017–3.306) 0.044
P for trend	0.003	0.014	0.077

Notes: Model 1: unadjusted model. Model 2: adjusted for age, ethnicity, education level and marital status. Model 3: further adjusted for PIR, smoking habits, hypertension, diabetes, drinking status, BMI, HDL-C, LDL-C, and the use of contraceptive pills.

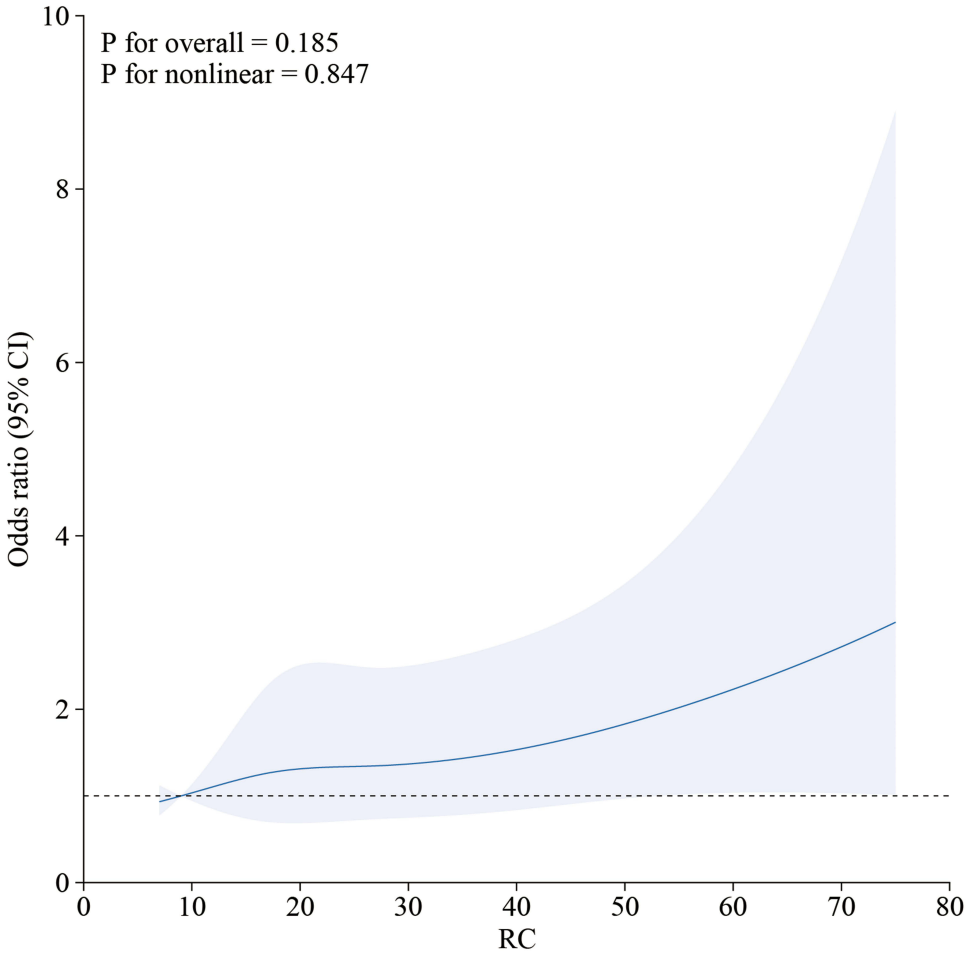

Figure 2 Restricted cubic spline plot of the association between remnant cholesterol (RC) and endometriosis.

Discussion

This study highlights a significant association between elevated remnant cholesterol (RC) levels and the occurrence of endometriosis in a representative sample of US women. Our analysis revealed that higher RC levels were consistently linked with an increased likelihood of endometriosis across various statistical models, even after adjusting for a range of demographic and clinical covariates. The linear relationship observed between RC and endometriosis, exhibiting no threshold or saturation effects, underscores the potential influence of lipid metabolism on the development of endometriosis. These findings suggest that RC may represent a metabolic signal rather than a discrete biomarker, and our findings underscore the potential role of dysregulated lipid metabolism in the inflammatory milieu underlying endometriosis. All inferences are confined to the studied population and do not extend to the global endometriosis phenotype.

Mechanistically, elevated RC may act as an upstream metabolic trigger that links dyslipidaemia to the inflammatory-peroxidative milieu characteristic of endometriosis. Recent data from Edward K Duran et al demonstrated that triglyceride-rich lipoprotein remnants (30–80 nm) readily traverse hyper-permeable peritoneal micro-vessels and become entrapped in the mesothelial surface.[13] 1 Local uptake by macrophages incites NLRP3-inflammasome assembly (IL-1β ↑ 2.3-fold), providing a pro-inflammatory "soil" that facilitates subsequent ectopic endometrial cell adhesion.

Our study adds to the growing body of evidence highlighting the significant role of remnant cholesterol (RC) in systemic inflammatory conditions, extending its relevance beyond cardiovascular diseases to include gynecological disorders such as endometriosis. Remnant cholesterol, defined as the cholesterol content within triglyceride-enriched lipoproteins (TRLs) such as chylomicron and very low-density lipoprotein (VLDL) remnants, has been increasingly recognized for its atherogenic potential and its ability to exacerbate inflammatory responses within the body.[9,14] Our

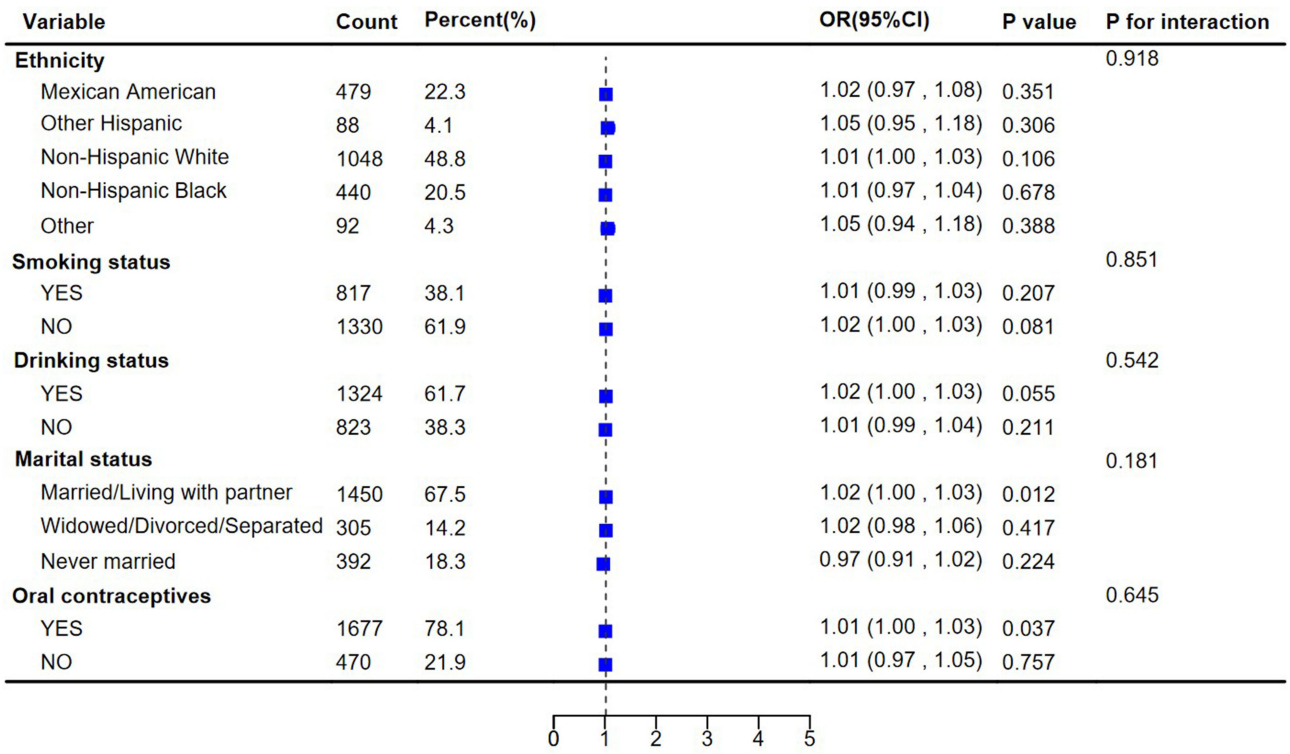

Figure 3 Subgroup analysis of the association between remnant cholesterol (RC) and Endometriosis.

findings suggest that the mechanisms through which RC contributes to cardiovascular diseases may also significantly contribute to the rising incidence of endometriosis.

Once deposited, RC particles deliver large quantities of poly-unsaturated cholesterol esters that are highly vulnerable to iron-catalysed lipid peroxidation. Pope & Dixon recently highlighted that such peroxidation generates diffusible aldehydes (malondialdehyde, 4-hydroxynonenal) which form protein adducts and amplify NADPH-oxidase-2-dependent ROS bursts.[15] Ichikawa et al subsequently confirmed a direct linear correlation between peritoneal fluid RC and ROS concentrations ($r = 0.62$, $p < 0.001$) in women with endometriosis.[16]

RC contributes to inflammation through several pathways that are also relevant to the pathogenesis of endometriosis. Firstly, RC particles, which are rich in cholesterol, can penetrate the endothelial barrier and become trapped within tissues, inducing a localized inflammatory response by activating macrophages and other immune cells.[17]

This process is analogous to what is observed in atherosclerosis, where RC contributes to plaque formation and vascular inflammation.[14,18] In the context of endometriosis, this inflammatory response could enhance the proliferation and implantation of ectopic endometrial cells, thereby increasing the severity and prevalence of the condition.

Furthermore, RC has been shown to promote oxidative stress,[18,19] a recognized contributing factor in the development of endometriosis.[20,21] Oxidative stress arises from an imbalance between the production of reactive oxygen species (ROS) and the body's antioxidant defenses.[16,22] Elevated RC levels may exacerbate oxidative stress by contributing to the accumulation of lipid peroxides, harmful byproducts of lipid oxidation.[15,23] This heightened oxidative stress may result in cellular damage and inflammation within the peritoneal cavity,[24] where endometriotic lesions typically develop.

The resulting oxidative stress activates NF-κB signalling, leading to a cytokine cascade (TNF-α, IL-6, IL-8) that promotes angiogenesis and matrix remodelling. A 2023 meta-analysis by Zheng et al showed that NF-κB-driven increases in TNF-α and MMP-2/9 were proportional to RC tertiles, supporting a dose-dependent inflammatory response.[24] Importantly, TNF-α up-regulates aromatase (CYP19A1) expression, raising local estradiol (E2) concentrations. E2 in turn stimulates hepatic very-low-density lipoprotein assembly, closing a positive feedback loop that perpetuates RC accumulation and intra-peritoneal inflammation.[4]

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The chronic inflammation[25] and oxidative stress[26] driven by elevated RC levels could thus create a favorable environment for the initiation and progression of endometriosis.

Furthermore, the linear relationship between RC levels and endometriosis prevalence observed in our study suggests that there may not be a threshold level below which RC is harmless. Instead, even relatively small increases in RC could incrementally raise the risk of developing endometriosis. This finding underscores the importance of managing RC levels, not only to reduce cardiovascular risk, but also to potentially lower the prevalence rate of endometriosis.[27,28] The connection between lipid metabolism[29] and endometriosis opens up new avenues for research, particularly in exploring whether lipid-lowering therapies,[30,31] the absence of an apparent threshold suggests that RC operates as a continuous, but non-specific, metabolic contributor rather than a discrete risk factor. Thus, elevated RC should be viewed as one component within a multifactorial network of oxidative stress and inflammation in EM pathogenesis, and its role must be interpreted cautiously until replicated in prospective, clinically validated cohorts.

Translational evidence lends further plausibility to this pathway. In the 2023 ACCORD-Lipid post-hoc analysis, each 10 mg/dL reduction in RC following simvastatin/ezetimibe was accompanied by an 8% decrease in systemic IL-6 and a 5% fall in serum MDA, implying that RC-lowering therapy might simultaneously dampen lipid peroxidation and inflammatory burden.[27] Whether such strategies can curtail the initiation or progression of endometriosis warrants prospective evaluation.

Although our research offers significant insights into the link between RC and endometriosis, several limitations should be acknowledged. Firstly, the cross-sectional design of the NHANES dataset precludes us from definitively establishing causality between elevated RC levels and the development of endometriosis. The temporal sequence of RC elevation and endometriosis onset cannot be determined, which raises the possibility of reverse causation or confounding by unmeasured factors. Secondly, the reliance on self-reported diagnosis of endometriosis, without clinical validation, may introduce recall bias or misclassification, potentially affecting the accuracy of our findings.[32] Third, the median age of our sample (≈ 38 y) is higher than typical reproductive-age cohorts; because lipid concentrations increase with age, residual confounding by age-related metabolic changes cannot be excluded. Fourthly, the relatively small number of women with endometriosis in certain subgroups, as reflected in Figure 3. Some subgroups, due to small sample sizes, may have limited statistical power, and the confidence intervals for these subgroups could be wide, affecting the precision of our estimates. These findings should be interpreted with caution, and future studies with larger, prospectively ascertained cohorts are needed to confirm these results and explore potential variations in different demographic groups.

Sample-size considerations and interpretation cautionWhile the observed endometriosis prevalence of 7.8% (153/1979) is compatible with published population-based estimates (5–10%), the absolute case number remains modest. This limited denominator inevitably widens the 95% confidence intervals and increases the risk of both false-negative and false-positive findings. Consequently, the magnitude and direction of effect sizes should be viewed as hypothesis-generating rather than definitive, and await replication in larger, prospectively ascertained cohorts with clinically validated diagnoses.

Biological integration and future directionsCollectively, the present findings position elevated RC as a metabolic instigator that fuels a self-amplifying loop of lipid peroxidation, ROS generation, and inflammatory signalling within the peritoneal compartment. Targeting RC—via lifestyle, nutraceutical, or lipid-lowering pharmacotherapy—may therefore represent a novel, translatable strategy to mitigate endometriosis risk or severity. Longitudinal and interventional studies are now required to establish causality and to quantify the clinical benefit of RC reduction in women at risk of endometriosis.

Population Representativeness and Limitations

The mean age of women with endometriosis in our study (40.41 years) is higher than the typical age of diagnosis, which generally occurs between 25 and 40 years. This discrepancy may be due to the cross-sectional nature of the NHANES data, which includes women at various stages of diagnosis. Although our sample may not fully reflect the general population with endometriosis, it provides valuable insights into lipid metabolism in women across different age groups.

Future studies focusing on younger cohorts and earlier stages of endometriosis are needed to better understand this relationship.

Conclusion

To conclude, this study, based on a sample reflective of the US adult demographic, suggests that elevated RC levels may be linked to an increased prevalence of endometriosis. The observed association emphasizes the importance of considering lipid metabolism as a contributing factor in systemic inflammatory diseases beyond cardiovascular health. Future studies should focus on longitudinal designs to clarify the temporal relationship between RC levels and endometriosis and to explore the underlying biological mechanisms that may drive this association.

Abbreviations:

RC, remnant cholesterol; TGRL, triglyceride-rich lipoproteins; IDL, intermediate-density lipoproteins; NHANES, National Health and Nutrition Examination Survey; CDC, Centers for Disease Control; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, Low-density lipoprotein cholesterol; MEC, Mobile Examination Center; ANOVA, Analysis of variance; TRLs, triglyceride-rich lipoproteins; VLDL, low-density lipoprotein; ROS, reactive oxygen species.

Data Sharing Statement

All the data analyzed in our study can be obtained from the NHANES database.

Ethics Approval and Consent to Participate

This study used publicly available, anonymized, and aggregated data from the NHANES. As no identifiable individual-level data were involved, the study was exempt from ethical review in accordance with Article 32, Items 1 and 2 of the Measures for Ethical Review of Life Science and Medical Research Involving Human Subjects issued by the National Health Commission of the People's Republic of China on February 18, 2023.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

No potential conflict of interest was reported by the author(s).

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