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ORIGINAL ARTICLE

Association between a C-Reactive Protein-Triglyceride Glucose Index and Chronic Kidney Disease in US Adults

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ABSTRACT

Background: **Chronic kidney disease (CKD) is a global health challenge. Inflammation and insulin resistance are established contributors to its pathogenesis. We sought to investigate the association between a novel C-reactive protein-triglyceride glucose index (CTI)), which integrates markers of inflammation and metabolic dysfunction, and the prevalence of CKD in a nationally representative sample.**

Methods: **This cross-sectional analysis included adults aged 20 years or older from the National Health and Nutrition Examination Survey (NHANES) cycles 1999 - 2004. A comprehensive review of the literature indicates that the diagnosis of chronic kidney disease (CKD) relies on meeting either of the following criteria: A reduced glomerular filtration rate (GFR), specifically an estimated GFR (eGFR) below 60 mL/minute/1.73 m²; the presence of albuminuria, defined as an albumin-to-creatinine ratio (ACR) exceeding 30 mg/g. The CTI was calculated using the formula: 0.412 x Ln (C-reactive protein) (mg/dL) + Ln [triglyceride (mg/dL) x fasting glucose (mg/dL)/2]. Logistic regression, curve fitting, and interaction effects were utilized to investigate the association between CTI and CKD.**

Results: **Among the 12,396 participants included, the prevalence of CKD was 11.0%. A strong positive dose-response relationship was observed between CTI and CKD prevalence. Logistic regression analysis revealed a positive association between CTI and CKD, even after adjusting for the confounding variables (odds ratio: (OR: 1.32, 95% CI: 1.25 - 1.38, $p < 0.001$). In addition, curve fitting after adjusting for all the confounding variables showed that there was a linear relationship between CTI and CKD (p for non-linearity = 0.254). CTI was positively correlated with CKD ($p < 0.001$).**

Conclusions: **The novel C-reactive protein-triglyceride glucose index, a composite measure of inflammation and metabolic dysregulation, is strongly and independently associated with a higher prevalence of CKD among US adults. This index may serve as a valuable and simple tool for early identification of individuals at high risk for CKD in clinical and public health settings.**

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KEYWORDS

chronic kidney disease, coronary heart disease, diabetes mellitus, C-reactive protein-triglyceride glucose index

INTRODUCTION

Chronic kidney disease (CKD) constitutes a formidable global public health challenge, affecting approximately 10 - 15% of the adult population worldwide. In the United States, the prevalence of CKD remains high and

is associated with substantial morbidity, mortality, and healthcare costs [1]. CKD is a well-established independent risk factor for cardiovascular disease and end-stage renal disease, necessitating dialysis or kidney transplantation. Despite its significant burden, CKD is often underdiagnosed in its early stages due to the asymptomatic nature of initial disease progression [2]. Therefore, identifying novel and modifiable risk factors, as well as developing simple tools for early risk stratification, is paramount for implementing effective preventive strategies and alleviating the disease burden.

The pathogenesis of CKD is multifactorial, involving a complex interplay of hemodynamic and metabolic pathways [3]. Beyond traditional risk factors such as hypertension and diabetes, accumulating evidence underscores the pivotal roles of chronic inflammation and metabolic dysregulation in the initiation and progression of renal damage [4]. C-reactive protein (CRP), a classical systemic inflammatory marker, has been consistently linked to declined renal function and an increased incidence of CKD. Concurrently, metabolic disturbances, particularly insulin resistance (IR) and dyslipidemia, contribute to renal injury through various mechanisms, including endothelial dysfunction and oxidative stress [5,6]. The triglyceride-glucose (TyG) index, a simple surrogate marker of IR, has recently emerged as a significant predictor of CKD risk [7,8].

Notably, inflammation and metabolic dysregulation are not isolated pathways but are deeply intertwined in a vicious cycle that can amplify renal injury. For instance, insulin resistance can promote a pro-inflammatory state, and conversely, inflammation can exacerbate metabolic abnormalities [9,10]. However, most previous studies have evaluated these risk factors in isolation. An integrated index that simultaneously captures both inflammatory and metabolic status may provide a more holistic and powerful tool for assessing CKD risk than any single marker alone. To our knowledge, no such composite index has been extensively validated in a large, nationally representative population for its association with CKD.

Therefore, we developed a novel C-reactive protein-triglyceride glucose (CRP-TG) index, calculated as 0.412 x Ln (C-reactive protein) (mg/dL) + Ln [triglyceride (mg/dL) x fasting glucose (mg/dL)/2], to synthesize information on both inflammation and metabolism into a single metric [11-13]. By utilizing data from the National Health and Nutrition Examination Survey (NHANES) 1999 - 2004, a large-scale survey representative of the non-institutionalized US population, this population-based cross-sectional study aimed to investigate the independent association between this novel CTI and the prevalence of CKD among US adults. We hypothesized that a higher CTI would be significantly associated with increased odds of CKD after adjustment for potential confounding factors.

MATERIALS AND METHODS

Study design and data source

This cross-sectional study utilized data from the National Health and Nutrition Examination Survey (NHANES) cycles 1999 - 2004. NHANES is a nationally representative survey conducted by the National Center for Health Statistics (NCHS) that employs a complex, multistage, stratified probability sampling design to assess the health and nutritional status of the non-institutionalized civilian population in the United States. The survey protocols were approved by the NCHS Research Ethics Review Board, and all participants provided written informed consent. The NHANES database is publicly available at: https://wwwn.cdc.gov/nchs/nhanes [14].

Study population

The initial study population consisted of adult participants (aged ≥ 20 years) from the continuous NHANES 1999 - 2004 cycles. We excluded the following individuals: pregnant individuals and those with missing CKD status or CTI measurements. A total of 12,396 participants were included in the final analysis (Figure 1): 1,369 with CKD and 11,027 without CKD.

Definition of variables

The primary exposure was the novel CRP-TG index (CTI, a composite marker integrating inflammation and metabolic dysregulation). The CTI was defined as 0.412 x Ln (C-reactive protein) (mg/dL) + Ln [triglyceride (mg/dL) x fasting glucose (mg/dL)/2]. C-reactive protein (CRP) was measured using latex-enhanced nephelometry. Fasting triglycerides and fasting glucose were measured using standard enzymatic methods. The coefficient 0.412 was derived from prior statistical standardization to balance the contributions of the inflammatory (LnCRP) and metabolic (TyG index) components [15].

Chronic kidney disease (CKD) was defined according to the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines. Participants meeting either one or both criteria were classified as having CKD: reduced estimated glomerular filtration rate (eGFR): eGFR < 60 mL/minute/1.73 m^2, calculated using the 2021 CKD-EPI creatinine equation; albuminuria: urinary albumin-to-creatinine ratio (ACR) ≥ 30 mg/g [16].

Based on previous literature, the following potential confounders were adjusted for in multivariate models: demographic factors [age (years), gender (male/female), race (non-Hispanic White, non-Hispanic Black, Mexican American, other race)], lifestyle factors [body mass index (BMI, kg/m^2), smoking status (never, former, current), alcohol consumption (yes/no)], laboratory data including alanine aminotransferase (ALT), aspartate aminotransferase (AST), cholesterol (CHO), total protein (TP), and albumin (ALB), and the status of hypertension, coronary heart disease, and diabetes among the study participants. Individuals were classified as non-

drinkers if their lifetime alcohol consumption was less than 12 standard drinks, or as drinkers if it equaled or exceeded 12 standard drinks. Smoking status was stratified into three groups: never smokers (lifetime consumption of < 100 cigarettes), current smokers (presently smoking), and former smokers (those that had quit following a lifetime consumption of > 100 cigarettes). A diagnosis of hypertension and coronary heart disease was made based on a self-reported physician diagnosis obtained from the questionnaire, and BMI was computed using a standardized technique based on weight and height. The operational definition of diabetes incorporated both subjective and objective measures, including self-reported history of a physician's diagnosis, or objective hyperglycemia defined as either fasting glucose ≥ 7.0 mmol/L or an HbA1c reading ≥ 6.5%.

Statistical analysis

The selection of covariates was informed by fundamental demographic variables, factors with clinical relevance as determined by medical experts, previously established risk factors for CKD documented in existing literature, and variables that, when included, modified the coefficients of the base regression model by more than 10%, suggesting substantive confounding effects. All statistical computations were conducted in R (version 4.2.3; R Foundation for Statistical Computing), utilizing the gtsummary, survey, rms, ggplot2, and forestplot packages. Descriptive statistics summarized the full cohort, with statistical significance determined a priori by two-sided tests at the $p < 0.05$ level.

RESULTS

Baseline demographic characteristics

This study analyzed data from 31,126 individuals within the NHANES database, collected over three survey cycles spanning a six-year period. Following the application of strict inclusion and exclusion criteria, a total of 12,396 subjects were included in the final analysis. The cohort had an average age of 50.9 years (standard deviation ± 18.7). Within this population, 1,369 participants were identified with chronic kidney disease (CKD), while 11,027 had no CKD.

As detailed in Table 1, which summarizes the baseline clinical and laboratory parameters, statistically significant differences were found between the CKD and non-CKD groups concerning the following variables: sex, age, race, hypertensive status, presence of coronary heart disease (CHD), diabetic status (DM), smoking history, alcohol consumption patterns, albumin (ALB), total protein (TP), blood urea nitrogen (BUN), creatinine (Cr), cholesterol levels, uric acid (UA), alanine aminotransferase (ALT), and CTI. Conversely, body mass index (BMI) and aspartate aminotransferase (AST) levels showed no significant differences between the two groups.

Univariate analysis of factors associated with CKD

Univariate logistic regression analysis showed that sex, age, race, hypertensive status, presence of coronary heart disease (CHD), diabetic status (DM), smoking history, alcohol consumption patterns, albumin (ALB), total protein (TP), blood urea nitrogen (BUN), creatinine (Cr), cholesterol levels, uric acid (UA), alanine aminotransferase (ALT), and CTI were significantly associated with CKD. Among these, age, hypertension, CHD, DM, BUN, UA, Cr, cholesterol, alcohol drinking status, and CTI exhibited a positive association with CKD risk, whereas ALB, TP, and ALT showed an inverse relationship. Notably, compared to non-Hispanic Whites, other racial groups have a lower risk of developing chronic kidney disease (CKD). CKD is more prevalent among females than among males. Compared to never smokers, former smokers have an increased risk of CKD, while current smokers have a decreased risk (Table 2).

Multivariate analysis of factors associated with CTI and CKD

Following multiple imputations of missing covariates, we employed logistic regression to assess the relationship between CTI ratio tertiles and chronic kidney disease (CKD). A positive association was observed between the C-reactive protein-triglyceride glucose index (CTI) and the risk of chronic kidney disease (CKD) in all models. The odds ratio (OR) attenuated with sequential adjustments but remained statistically significant. In the unadjusted model (Model 1), the OR was 1.74 (95% CI: 1.68 - 1.80, $p < 0.001$). After adjustment for age, gender, and race (Model 2), the OR was 1.59 (95% CI: 1.52 - 1.65, $p < 0.001$). Further adjustment for BMI, hypertension, CHD, smoking, alcohol use, and DM (Model 3) yielded an OR of 1.52 (95% CI: 1.43 - 1.63, $p < 0.001$). In the fully adjusted model (Model 4, including ALT, AST, TP, ALB, cholesterol), the association persisted (OR = 1.32, 95% CI: 1.25 - 1.38, $p < 0.001$). A consistent positive relationship was observed between CTI and CKD risk in every model, with highly significant p-values (< 0.001). Participants were divided into three groups based on CTI levels (low, medium, high). A positive dose-response relationship was observed between CTI and the risk of CKD, where higher CTI categories were associated with progressively increased effect estimates. The p-value for trend was less than 0.001, indicating a statistically significant linear trend across the groups (Table 3).

Subgroup analysis and curve fitting

We explored whether there were differences in gender, race, hypertension, coronary heart disease (CHD), diabetes mellitus (DM), smoking, and alcohol drinking status between groups with different CTI levels in relation to chronic kidney disease (CKD). The relationship between CTI and CKD remained consistent and stable across all stratified subgroups ($p > 0.05$ for interaction) (Figure 2). These results further indicated a positive linear correlation between CTI and the risk of CKD, sug-

Table 1. Comparison of baseline metrics between participants grouped according to CKD.

Variables	Total	CKD (no)	CKD (yes)	p
	n = 12,396	n = 11,027	n = 1,369	
Gender, n (%)				
Male	6,246 (50.4)	5,598 (50.8)	648 (47.3)	0.017
Female	6,150 (49.6)	5,429 (49.2)	721 (52.7)	
Age (years)	50.9 ± 18.7	48.3 ± 17.7	71.7 ± 13.2	< 0.001
BMI (kg/m^2)	28.3 ± 6.2	28.3 ± 6.2	28.6 ± 6.2	0.066
Race, n (%)				
Non-Hispanic white	6,290 (50.7)	5,397 (48.9)	893 (65.2)	< 0.001
Non-Hispanic black	2,326 (18.8)	2,116 (19.2)	210 (15.3)	
Mexican American	2,804 (22.6)	2,615 (23.7)	189 (13.8)	
Other	976 (7.9)	899 (8.2)	77 (5.6)	
Hypertension, n (%)	3,328 (27.2)	2,525 (23.2)	803 (59.2)	< 0.001
CHD, n (%)	577 (4.7)	378 (3.4)	199 (14.8)	< 0.001
Smoking, n (%)				
Never	6,235 (50.4)	5,570 (50.6)	665 (48.7)	< 0.001
Former	3,353 (27.1)	2,815 (25.6)	538 (39.4)	
Current	2,790 (22.5)	2,627 (23.9)	163 (11.9)	
ALB, (g/L)	43.0 ± 3.4	43.3 ± 3.2	41.2 ± 3.7	< 0.001
BUN, (mmol/L)	5.1 ± 2.2	4.7 ± 1.5	8.1 ± 3.9	< 0.001
Cholesterol, (mmol/L)	5.2 ± 1.1	5.2 ± 1.1	5.3 ± 1.2	0.004
TP (g/L)	73.9 ± 4.9	74.1 ± 4.8	72.7 ± 5.7	< 0.001
CTI	8.0 ± 1.0	7.9 ± 1.0	8.5 ± 0.9	< 0.001
Alcohol drinking				
No	10,447 (84.3)	9,363 (84.9)	1,084 (79.2)	< 0.001
Yes	1,949 (15.7)	1,664 (15.1)	285 (20.8)	
DM, n (%)				
No	10,682 (86.3)	9,742 (88.4)	940 (68.7)	< 0.001
Yes	1,702 (13.7)	1,274 (11.6)	428 (31.3)	
ALT, (U/L)	21.0 (16.0, 29.0)	21.0 (17.0, 29.0)	19.0 (15.0, 24.0)	< 0.001
AST, (U/L)	22.0 (19.0, 27.0)	22.0 (19.0, 27.0)	22.0 (19.0, 27.0)	0.188
UA, (μmol/L)	315.2 (261.7, 380.7)	309.3 (255.8, 368.8)	374.7 (315.2, 446.1)	< 0.001
Cr, (μmol/L)	78.1 ± 45.0	71.5 ± 17.1	132.0 ± 112.8	< 0.001

Data presented are mean ± SD, median (IQR), or n (%).
BMI: body mass index, ALB: albumin, TP: total protein, Hypertension: high blood pressure, DM: diabetes mellitus, CHD: coronary heart disease, ALT: alanine aminotransferase, AST: aspartate aminotransferase, BUN: blood urea nitrogen, Cr: creatinine, UA: uric acid, CTI: CRP-TG index.

gesting that CTI could function as a reliable biomarker for identifying at-risk individuals, independent of the aforementioned factors. To characterize the association more precisely, we performed a multivariable-adjusted analysis (Model 4) and generated a curve, which demonstrated a linear dose-response relationship (p for non-linearity = 0.254) (Figure 3).

DISCUSSION

This population-based study demonstrated a significant positive association between the novel CRP-TG index and the prevalence of chronic kidney disease among US adults, independent of traditional cardiometabolic risk factors. Our findings suggest that this integrated biomarker, which simultaneously captures both inflammatory and metabolic pathways, may serve as a valuable

Table 2. Single-variable analysis examining the relationship between interrelated variables and CKD.

Variable	CKD	p-value
	OR (95% CI)	
Male	1	
Female	1.15 (1.03 - 1.28)	0.017
Age (years)	1.09 (1.09 - 1.1)	< 0.001
BMI (kg/m^2)	1.01 (1 - 1.02)	0.066
Non-Hispanic white	1	
Non-Hispanic black	0.6 (0.51 - 0.7)	< 0.001
Mexican American	0.44 (0.37 - 0.51)	< 0.001
Other	0.52 (0.41 - 0.66)	< 0.001
Hypertension, n (%)	4.8 (4.27 - 5.4)	< 0.001
CHD, n (%)	4.86 (4.05 - 5.83)	< 0.001
Smoking, n (%)		
Never	1	
Former	1.6 (1.42 - 1.81)	< 0.001
Current	0.52 (0.44 - 0.62)	< 0.001
ALB, (g/L)	0.83 (0.82 - 0.84)	< 0.001
ALT, (U/L)	0.98 (0.98 - 0.99)	< 0.001
AST, (U/L)	1 (0.99 - 1)	0.188
BUN, (mmol/L)	2.03 (1.96 - 2.11)	< 0.001
Cholesterol, (mmol/L)	1.08 (1.02 - 1.13)	0.004
TP (g/L)	0.95 (0.93 - 0.96)	< 0.001
UA, (μmol/L)	1.01 (1.01 - 1.01)	< 0.001
Cr, (μmol/L)	1.1 (1.09 - 1.1)	< 0.001
Alcohol use (%)	1.48 (1.29 - 1.7)	< 0.001
DM	3.48 (3.06 - 3.96)	< 0.001
CTI	1.74 (1.64 - 1.84)	< 0.001

Data presented are mean ± SD, median (IQR), or n (%).
BMI: body mass index, ALB: albumin, TP: total protein, Hypertension: high blood pressure, DM: diabetes mellitus, CHD: coronary heart disease, ALT: alanine aminotransferase, AST: aspartate aminotransferase, BUN: blood urea nitrogen, Cr: creatinine, UA: uric acid, CTI: CRP-TG index.

Table 3. Multivariate analysis of the association between CTI and CKD in individuals.

Variable	Model 1		Model 2		Model 3		Model 4	
	OR (95% CI)	p-value	OR (95% CI)	p-value	OR (95% CI)	p-value	OR (95% CI)	p-value
CTI	1.74 (1.68 - 1.8)	< 0.001	1.59 (1.52 - 1.65)	< 0.001	1.52 (1.43 - 1.63)	< 0.001	1.32 (1.25 - 1.38)	< 0.001
CTI group								
T1 (5.11 - 7.60)	1 (Ref)		1 (Ref)		1 (Ref)		1 (Ref)	
T2 (7.60 - 8.41)	2.27 (2.06 - 2.5)	< 0.001	1.47 (1.32 - 1.63)	< 0.001	1.3 (1.17 - 1.45)	< 0.001	1.25 (1.12 - 1.39)	< 0.001
T3 (8.41 - 13.14)	3.54 (3.23 - 3.88)	< 0.001	2.29 (2.07 - 2.54)	< 0.001	1.7 (1.52 - 1.9)	< 0.001	1.53 (1.36 - 1.72)	< 0.001
p for trend		< 0.001		< 0.001		< 0.001		< 0.001

Model 1: non-adjusted, Model 2: adjusted for age, gender, and race, Model 3: Model 2 + adjustment for BMI, hypertension, CHD, smoking, alcohol use, and DM, and Model 4: Model 3 + adjustment for ALT, AST, TP, ALB, and cholesterol.

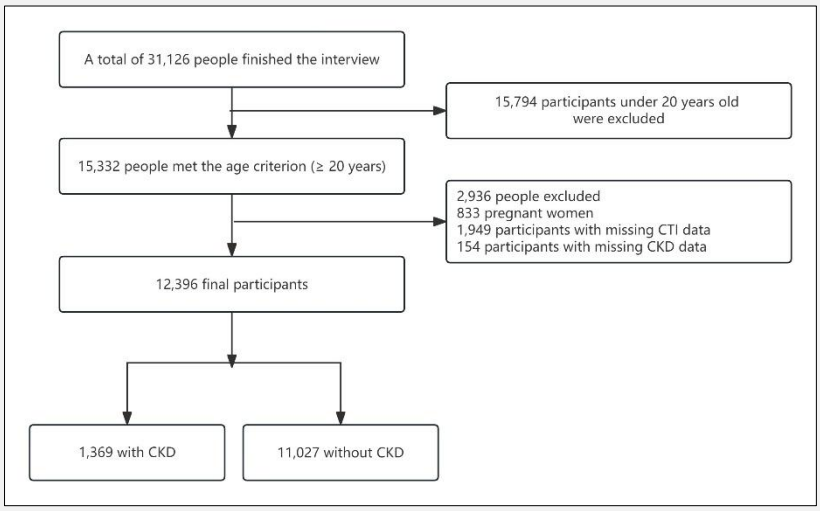


Figure 1. Participant screening and selection diagram.

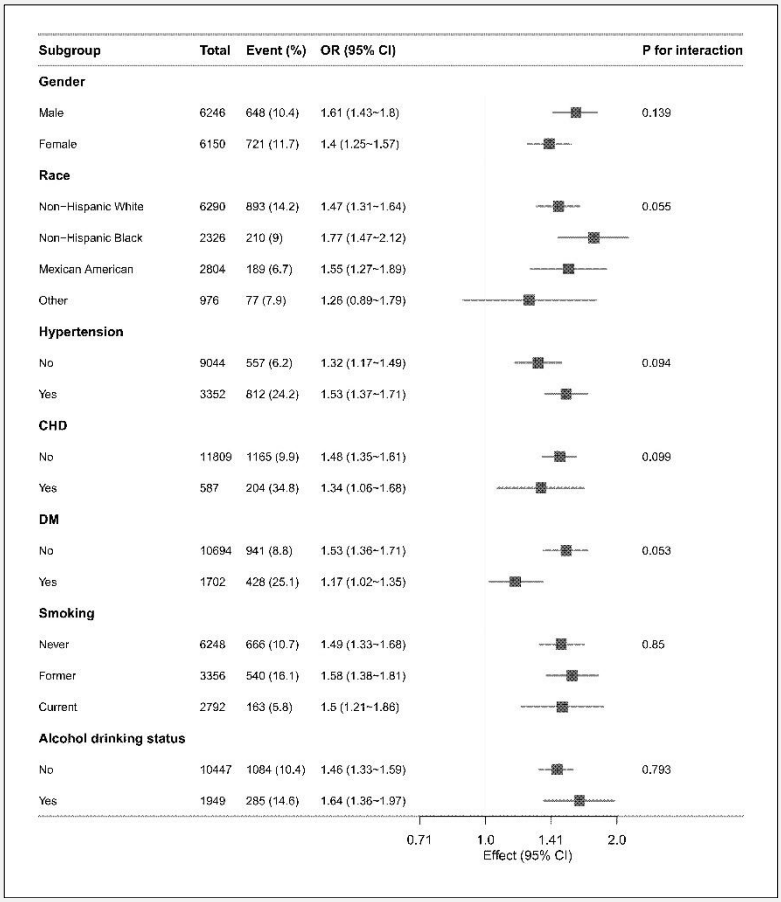


Figure 2. Forest plot of CTI and CKD.

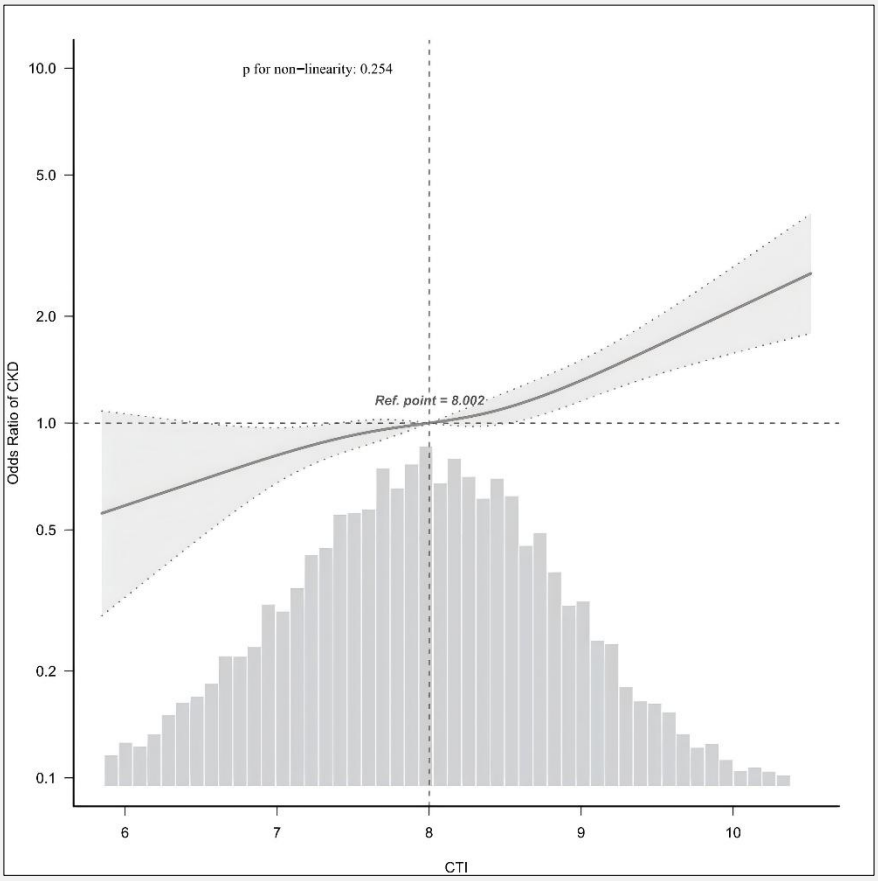


Figure 3. Fitting curve of CTI and CKD.

tool for identifying individuals at high risk for CKD. Our investigation yielded several noteworthy observations. First, we observed a robust dose-response relationship between the CRP-TG index and CKD prevalence, with participants in the highest tertiles exhibiting significantly increased odds of CKD compared to those in the lowest. These results further indicated a positive linear correlation between CTI and the risk of CKD, suggesting that CTI could function as a reliable biomarker for identifying at-risk individuals, independent of the aforementioned factors. To characterize the association more precisely, we performed a multivariable-adjusted analysis (Model 4) and generated a curve, which demonstrated a linear dose-response relationship (p for non-linearity = 0.254) (Figure 3). Second, this association remained significant after comprehensive adjustment for potential confounders, including age, sex, race, relevant laboratory indicators, lifestyle factors, and comorbidities such as hypertension, CHD, and DM. Third, the CRP-TG index demonstrated superior predictive performance compared to its individual components (CRP or TyG index alone), suggesting synergistic effects between inflammation and metabolism in CKD pathogenesis.

The observed association between the CTI and CKD can be explained through several interconnected biological mechanisms. Chronic inflammation, reflected by elevated CRP levels, promotes renal injury through multiple pathways, including activation of pro-fibrotic cytokines, increased oxidative stress, and endothelial dysfunction. Simultaneously, insulin resistance, captured by the TyG index, contributes to glomerular hypertension, podocyte injury, and altered lipid metabolism - all of which accelerate renal function decline [17]. The crosstalk between metabolic and inflammatory pathways forms a damaging feedback cycle. Insulin resistance stimulates cytokine production in adipose tissue, which subsequently interferes with insulin receptor signaling and promotes systemic insulin resistance, thereby creating a self-perpetuating cycle of metabolic and inflammatory dysregulation [18]. This vicious cycle creates a metabolic-inflammatory milieu that promotes

progressive renal damage. Our composite index effectively captures this interplay, potentially explaining its strong association with CKD prevalence.

Our findings align with and extend the existing literature on inflammation, metabolism, and renal disease. Several previous studies have reported independent associations between inflammatory markers (particularly CRP) and CKD risk. Similarly, multiple investigations have demonstrated that insulin resistance, as measured by the TyG index or other methods, predicts incident CKD [19,20]. However, few studies have examined the joint effects of these pathways using a composite measure. Notably, our results regarding the synergistic effect are consistent with emerging research highlighting the cross-talk between inflammation and metabolism in cardiorenal disease. The specific formulation of our index, which weighs the inflammatory and metabolic components based on their relative contributions, may represent a methodological advancement over previous approaches that examined these pathways separately or through simple additive models.

The findings from our study have several important implications. First, the CTI, derived from routinely available laboratory parameters (CRP, triglycerides, and glucose), could serve as a practical tool for improving CKD risk stratification in primary care settings. This composite biomarker might help identify high-risk individuals who would benefit from more intensive monitoring or early intervention.

Second, from a public health perspective, our findings reinforce the importance of integrated approaches to CKD prevention that address both inflammatory and metabolic risk factors simultaneously. Health policies promoting healthy lifestyles (e.g., anti-inflammatory diets, regular physical activity) may confer dual benefits for both metabolic and inflammatory pathways. Third, our study validates the development of integrated biomarkers to capture complex pathophysiological interplay, an approach applicable to other diseases characterized by concurrent dysregulation of multiple biological pathways.

Despite these noteworthy findings, our study has several limitations that should be acknowledged. The temporal relationship between the CTI and CKD cannot be definitively established due to the cross-sectional nature of NHANES data. While our findings are biologically plausible, reverse causality remains a possibility. In addition, although we adjusted for a comprehensive set of potential confounders, the possibility of residual confounding by unmeasured factors (e.g., dietary patterns, genetic predisposition, or environmental exposures) cannot be entirely excluded. Finally, although NHANES measurements are standardized, single-time-point assessments are subject to intra-individual variation. This could cause exposure misclassification and attenuate true associations. We have addressed these limitations thoughtfully and transparently, which adds credibility to our study. Future prospective cohort studies with repeated measurements, complemented by targeted intervention studies and in-depth mechanistic investigations, would help address these limitations by elucidating temporal dynamics, establishing causality, and uncovering underlying biological pathways.

CONCLUSION

In conclusion, our study demonstrates that a novel CRP-TG index, integrating inflammatory and metabolic pathways, is significantly associated with CKD prevalence among US adults. This composite biomarker may reflect the synergistic detrimental effects of chronic inflammation and insulin resistance on renal health. While further validation in prospective studies is warranted, our findings suggest that CTI could serve as a practical tool for improving CKD risk stratification and highlights the potential value of dual-pathway approaches to CKD prevention and management.

Declaration of Generative AI in Scientific Writing:
No generative AI technologies (such as ChatGPT, Copilot, etc.) were used in the creation of this manuscript. The authors take full responsibility for the content of the publication.

Declaration of Interest:
The authors declare no competing interests.

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