BMC Musculoskeletal Disorders

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Associations between estimated glucose disposal rate and osteoarthritis risk in US adults: a cross-sectional study

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Abstract

Background Estimated glucose disposal rate (eGDR) is a novel insulin resistance (IR) assessment surrogate. Although it has shown promising potential in other metabolic disease studies, no research has yet explored its relationship with osteoarthritis (OA). Therefore, this study aims to investigate the association between eGDR and OA in a cross-sectional observational cohort.

Method Data utilized in this cross-sectional study were drawn from the National Health and Nutrition Examination Survey (NHANES). Logistic regression models were used to evaluate the association between eGDR and OA, stratified analysis was applied to assess the stability of the results.

Result A total of 19,040 participants were included in the study, including 2,001 OA patients and 17,039 non-OA participants with an age distribution ranging from 20 to 85 years. The fully adjusted logistic regression model shows that eGDR were less likely associated with OA compared to those with non-OA (OR = 0.879, 95% CI = 0.846–0.914, $P < 0.001$). By dispersing the eGDR into quartiles, the correlation between eGDR and OA remained significant (P for trend < 0.0001).

Conclusion This study suggests that eGDR is independently associated with OA, with lower eGDR values being linked to a higher risk of OA.

Clinical trial number Not applicable.

Keywords Osteoarthritis, Estimated glucose disposal rate, National health and nutrition examination survey, Cross-sectional observational study

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Background

Osteoarthritis (OA), the most prevalent form of arthritis, is a major contributor to disability in the global aging population [1]. According to the World Health Organization (WHO), approximately 595 million people suffer from OA globally, and this number is expected to rise by 2050, making OA one of the most significant chronic diseases in the elderly population [2]. OA is a whole joint disease characterized by the degeneration of articular cartilage, osteophyte formation, subchondral bone changes, and inflammation of the synovial membrane. These changes result in chronic pain, joint stiffness, and functional impairment, which severely impact the quality of life of individuals with OA [3]. OA not only causes physical suffering for patients but also imposes a significant social and economic burden, increasing the demand for healthcare resources and the costs of care for disabled individuals. Traditional risk factors for OA include aging, obesity, joint trauma, and mechanical overload [4]. However, emerging evidence [5] suggests that metabolic factors, particularly insulin resistance (IR), may play a significant role in the onset and progression of OA.

IR is clinically defined as a reduced ability of endogenous or exogenous insulin to enhance glucose uptake and utilization compared to normal population levels [6]. IR is considered a central mechanism in type 2 diabetes mellitus (T2DM) and other metabolic diseases [7–9]. Growing evidence indicates a significant positive association between IR and OA [10–12]. While hyperinsulinemic-euglycemic clamp (HIEC) is regarded as the gold standard for measuring IR, its complexity in clinical practice has led to the use of surrogates such as homeostatic model assessment of IR (HOMA-IR), triglyceride-glucose index (TyG), and their derivatives, including triglyceride glucose with body mass index(TyG-BMI), triglyceride-glucose with waist circumference (TyG-WC), and triglyceride glucose with the ratio of waist circumference divided by height (TyG-WtHR). These indices have been shown to have a significant positive correlation with the occurrence of OA [5], which is one of the key findings of our previous research. These indices provide effective alternatives for assessing the association between IR and OA in clinical practice.

Estimated glucose disposal rate (eGDR) is a novel IR assessment indicator [13, 14]. It is derived from a simple formula based on clinical variables including waist circumference (WC), hypertension, and glycated hemoglobin (HbA1c). It provides an estimate of the ability of the body to clear glucose from the bloodstream, with lower values indicating higher levels of IR. Unlike other IR markers such as HOMA-IR or TyG, eGDR is considered to offer a more practical and accessible measure for evaluating the efficiency of glucose disposal in the body. eGDR has similar accuracy to the HIEC in assessing IR status and is therefore considered a reliable surrogate marker of IR [15, 16]. Its simplicity and ability to reflect both central obesity and metabolic dysfunction make it a promising tool for understanding the relationship between metabolic diseases, such as diabetes. eGDR has shown promising prognosis potential in the studies of some metabolism-related diseases, including stroke, coronary artery disease, diabetic kidney disease, all-cause mortality and so on [15, 17–19]. There is a significant positive correlation between IR and OA, and eGDR serves as one of the alternative indicators of IR, from which we can speculate that there may be a significant negative correlation with OA. However no research has yet explored this association between eGDR and OA directly until now.

Therefore, this study aims to validate the hypothesis that lower eGDR is significantly associated with an increased risk of OA in a cross-sectional observational cohort. By investigating this relationship, the study seeks to explore the potential application of eGDR as a more accurate surrogate for assessing IR in OA patients.

Method

Data source

This research utilized data from the National Health and Nutrition Examination Survey (NHANES) (https://www.cdc.gov/nchs/nhanes/), a program conducted by the Centers for Disease Control and Prevention (CDC). NHANES is a nationally representative survey aimed at evaluating the health and nutritional status of adults and children across the United States. It employs a complex, multistage stratified probability sampling method to gather extensive data, including health interviews, physical examinations, and laboratory analyses. Data from NHANES cycles between 1999 and 2018 were selected for this study. The study protocol was approved by the Institutional Review Board of the National Center for Health Statistics (NCHS). All participants provided written informed consent. This study followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.

Ascertainment of osteoarthritis

OA was ascertained based on self-reported data collected through the NHANES. Participants were asked the question, "Have you ever been told by a doctor or other health professional that you have arthritis?". Those who responded affirmatively were further asked, "Which type of arthritis?". Individuals who answered "osteoarthritis" to this question were considered to have self-reported OA [20].

Ascertainment of eGDR

The eGDR, as a surrogate of IR, was calculated based on WC, hypertension status, and HbA1c. The eGDR was calculated using the following formula: $\mathbf{eGDR = 21.158 - 0.09 \times WC - 3.407 \times Hypertension - 0.551 \times HbA1c}$ [18, 21]. Where WC is measured in meters, hypertension is a binary variable (0 indicating no hypertension and 1 indicating the presence of hypertension), and HbA1c is expressed as a percentage. Participants who actively respond to the questionnaire with hypertension and who are taking medication for hypertension or systolic blood pressure ≥ 140 mmHg, or diastolic blood pressure ≥ 90 mmHg will be considered hypertensive patients [18]. Lower eGDR values indicate higher levels of IR.

Covariates

Several covariates were selected based on their potential to influence OA. The following covariates were included in the analysis: age, sex, ethnicity, marital status, education level, poverty income ratio (PIR), fasting total cholesterol (mg/dL), fasting HDL cholesterol (mg/dL), fasting LDL cholesterol (mg/dL), and physical activity (MET/week).

Age, sex, ethnicity, and marital status were self-reported by participants. Education level was categorized into three groups: "College Graduate or above", "High School Grad or Equivalent" and "Less Than 9th Grade" [22]. The PIR was used as an indicator of socioeconomic status and was divided into three groups ("0-1.3 PIR", "> 1.3–3.5 PIR", and "> 3.5 PIR") [23]. Ethnicity was classified as "Non-Hispanic White", "Non-Hispanic Black", "Mexican American", and "Other" [22]. The marital status was classified as "Married or Living with partner", "Never married", and "Divorced or Widowed or Separated". Smoking was classified as "Never, smoked less than 100 cigarettes in life", "Former, smoked more than 100 cigarettes in life and smoke not at all now", and "Current, smoked more than 100 cigarettes in life and smoke some days or every day" [22]. Alcohol consumption was divided as: "Never", "Former", "Mild", "Moderate", and "Heavy" [24]. Fasting total cholesterol, HDL cholesterol, and LDL cholesterol levels were measured through standardized laboratory procedures and were included as continuous variables in the analysis. Each participant completed a physical activity questionnaire including questions related to all physical activity performed over a past period of time. Metabolic equivalent (MET) scores for a specific activity were calculated based on activity type and intensity [25]. Due to the difference in the data content provided by NHANES, the calculation of physical activity was performed in two parts [26, 27]. During the 1999–2006 cycles, physical activity consisted of "walk or bicycle", "task around home or yard" and "muscle strength". During the 2007–2018 cycles, physical activity consisted of "walk or bicycle", "work activity" and "recreational activity". Detailed recommended MET scores are available in the NHANES's website (PAQ_D; PAQ_E). The final results are presented as the weekly total MET.

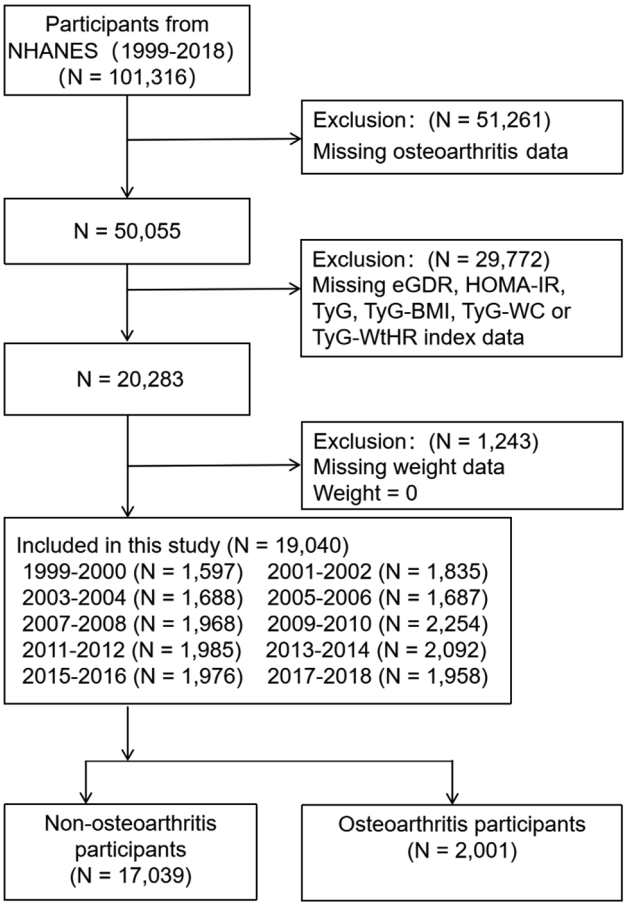

Fig. 1 The flowchart of participants selection

Study participants

Participants for this study were drawn from the NHANES (1999–2018), with an initial sample size of 101,316 individuals. Exclusion criteria: missing OA data; missing data on eGDR, HOMA-IR, TyG, TyG-BMI, TyG-WC, or TyG-WtHR indices; missing weight data or having a weight value of zero. Detailed participant selection procedures are provided in Fig. 1.

Statistical analysis

To ensure the representativeness of the sample, we used the sample weights provided by NHANES (wtsaf2 year. glu and wtsaf4 year.glu). Continuous variables are presented as means with standard deviations (SD), while categorical variables are expressed as counts (n) and percentages (%). The study population was categorized into

two groups based on the presence or absence of OA. Chi-square tests were employed to assess differences between these groups. To evaluate the relationship between eGDR and OA, we used logistic regression models. Three models were constructed in this analysis: the crude model, which did not adjust for any covariates; Model 1, which adjusted for age and sex; and Model 2, which additionally adjusted for ethnicity, marital status, education level, poverty income ratio (PIR), fasting total cholesterol, HDL cholesterol, LDL cholesterol, and physical activity. Furthermore, we employed restricted cubic spline (RCS) curves based on model 2 to examine the existence of a nonlinear association between eGDR and OA. Stratified analyses were performed for factors including age, ethnicity, PIR, marital status, education level, smoking status, and alcohol consumption. These stratified analyses aimed to explore potential variations in the association between eGDR and OA across different subgroups. All statistical analyses were conducted using R software (version 4.3.2). A *P*-value below 0.05 was regarded as indicative of statistical significance.

Result

Baseline characteristics of study participants

Originally 101,316 individuals were included in the study, 51,261 were excluded for missing OA data, 29,772 for the missing data on eGDR, HOMA-IR, TyG, TyG-BMI, TyG-WC, or TyG-WtHR indices data were excluded, 1,243 individuals were excluded due to missing weight data or having a weight value of zero. Finally, a total of 19,040 participants were included in the final analysis, comprising 2,001 individuals with OA and 17,039 without OA.

The baseline characteristics of the participants are summarized in Table 1. People with OA were approximately 17 years older than those non-OA participants, most females (64.7%), non-Hispanic whites (83.3%), married (67,6%), college graduate or above (61.9%), and > 3.5 PIR (45.5%). Smoking, and alcohol use also differed significantly between the groups ($P < 0.0001$). Biochemical indicators showed that eGDR values were significantly lower in the OA group than non-OA group. HbA1c level was higher in the OA group. Total cholesterol and HDL cholesterol levels were significantly higher in the OA group, while LDL cholesterol did not differ significantly between the groups.

Association between eGDR and osteoarthritis

Weighted logistic regression models were used to evaluate the association between eGDR and OA. As shown in Table 2, the crude logistic regression model shows that eGDR was less likely associated with OA compared to those with non-OA (OR = 0.803, 95% CI = 0.785–0.821, $P < 0.001$). After adjusting for age and sex in model 1, the association remained robust, with an OR of 0.882 (95%CI [0.857,0.908], $P < 0.0001$). Model 2, which further adjusted for ethnicity, marital status, education level, PIR, smoking, alcohol use, fasting total cholesterol, HDL cholesterol, LDL cholesterol and physical activity, still shows a significant association (OR = 0.879, 95%CI [0.846,0.914], $P < 0.0001$). By dispersing the eGDR into quartiles, the association between eGDR and OA remained significant (P for trend < 0.0001). In addition, the RCS also suggests a stable negative association between eGDR and OA (Fig. 2).

Stratified analysis

Stratified analyses were conducted to examine the robustness of the association between eGDR and OA across various subgroups (Fig. 3). The results show that the association remained significant across different age groups, with stronger associations observed in younger participants. Sex did not significantly affect the relationship, as both males and females showed similar associations. Ethnicity, marital status, and smoking status also influenced the relationship. The PIR, education level, and alcohol use did not significantly modify the relationship. These findings confirm that the association between eGDR and OA is robust across various subgroups, with age, ethnicity, marital and smoking status playing key roles in modifying the strength of this association.

The efficacy of eGDR in predicting osteoarthritis

This study compared eGDR with IR surrogates (HOMA-IR, TyG, TyG-BMI, Ty G-WC, and TyG-WtHR) in our previous study, where the eGDR for predicted OA alone was significantly stronger than the previous five, with an AUC of 0.68. Detailed results are shown in Fig. 4.

Discussion

This study investigated the association between eGDR and OA risk in US adults. The results revealed that eGDR significantly associated with a 12.1% lower likelihood of OA risk compared to those with non-OA. This association persisted across various demographic subgroups, including age, sex, ethnicity, PIR, marital status, education level, smoking status, and alcohol consumption.

The association between IR and OA has been demonstrated in many studies. For example, Tchetina et al.'s study [28] showed that the molecular and cellular metabolic disorders associated with OA are linked to an IR state similar to T2DM. Courties et al. [29] found that the pro-inflammatory and pro-degradation effects in the IR state made the joints more sensitive to overproduced TNF-α, increasing the possibility of OA. In addition, a study based on NHANES (1999–2018) demonstrated a significant association between IR as measured by the TyG index and an elevated risk of OA in individuals with sarcopenic obesity [30]. Besides, in our previous study

Table 1 Baseline characteristics of study participants

Variable	Non-OA (*N* = 17,039)	OA (*N* = 2,001)	*P* value
Age (year)	43.83(0.22)	60.95(0.32)	< 0.0001
Age (year)			< 0.0001
<40	6869(44.60)	111(6.17)	
40–59	5688(37.29)	569(37.46)	
>=60	4482(18.11)	1321(56.37)	
Sex			< 0.0001
Female	8490(49.42)	1285(64.71)	
Male	8549(50.58)	716(35.29)	
Ethnicity			< 0.0001
Non-Hispanic White	7142(66.12)	1298(83.26)	
Non-Hispanic Black	3326(11.42)	277(6.25)	
Mexican American	3344(9.22)	180(3.01)	
Other	3227(13.24)	246(7.48)	
Marital status			< 0.0001
Married or Living with partner	10,578(64.83)	1234(67.62)	
Never married	3186(19.60)	116(5.36)	
Divorced or Widowed or Separated	3117(15.57)	636(27.01)	
Education level			0.07
College Graduate or above	8757(59.48)	1121(61.94)	
High School Grad or Equivalent	6353(34.86)	709(33.57)	
Less Than 9th Grade	1914(5.65)	171(4.49)	
Poverty income ratio			0.01
0-1.3 PIR	4714(20.94)	445(16.80)	
>1.3–3.5 PIR	6006(36.72)	742(37.70)	
>3.5 PIR	4954(42.33)	651(45.50)	
Smoking			< 0.0001
Never	9566(55.09)	948(46.45)	
Former	3967(23.53)	697(34.80)	
Current	3491(21.39)	355(18.75)	
Alcohol use			< 0.0001
Never	2176(10.89)	255(10.60)	
Former	2485(13.09)	412(18.56)	
Mild	5219(35.73)	771(45.55)	
Moderate	2382(17.30)	242(15.26)	
Heavy	3432(22.98)	179(10.03)	
eGDR	8.30(0.04)	6.59(0.09)	< 0.0001
HbA1c (%)	5.51(0.01)	5.78(0.03)	< 0.0001
Fast total cholesterol (mg/dl)	194.41(0.48)	200.71(1.18)	< 0.0001
HDL cholesterol (mg/dl)	53.40(0.21)	56.64(0.57)	< 0.0001
LDL cholesterol (mg/dl)	115.75(0.39)	116.46(0.91)	0.47
Physical activity (MET/week)	3494.39(76.73)	3003.42(147.30)	0.002

PIR, Poverty income ratio; eGDR, estimated glucose disposal rate; HDL, High Density Lipoprotein; LDL, Low Density Lipoprotein; Alcohol use, never (had < 12 drinks in lifetime), former (had ≥ 12 drinks in 1 year and did not drink last year, or did not drink last year but drank ≥ 12 drinks in lifetime), mild = c(1,2), 1 is for female and 2 is for male, moderate = c(2,3), 2 is for female and 3 is for male; or binge > = 2 and binge < 5, heavy = c(3,4), 3 is for female and 4 is for male; or binge > = 5; Continuous variables are presented as means with standard deviations (SD), categorical variables are expressed as counts (n) and percentages (%)

[5], we comprehensively evaluated five indicators of IR (HOMA-IR, TyG, TyG-BMI, TyG-WC, and TyG-WtHR) and found significant and stable positive correlations with them and OA, with TyG-WtHR showing the strongest predictive efficacy. In conclusion, the association between IR and OA is indisputable, and IR plays a non-negligible role in the pathogenesis of OA.

IR promotes the development of OA through multiple mechanisms. Firstly, IR causes hyperglycemia and hyperinsulinemia. The inhibition of the glycolytic pathway in chondrocytes under pathological IR conditions, the accumulation of glucose and glucose-6-phosphate can lead to the formation of advanced glycation end products (AGEs), which can lead to cartilage damage

Table 2 The results of logistic regression analysis on the association between eGDR and OA risk

Character	Crude model		Model 1		Model 2	
	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*
eGDR	0.803(0.785,0.821)	< 0.0001	0.882(0.857,0.908)	< 0.0001	0.879(0.846,0.914)	< 0.0001
Quartile 1 [-4.232,5.688]	ref		ref		ref	
Quartile 2 (5.688,8.388]	0.555(0.473,0.652)	< 0.0001	0.590(0.491,0.709)	< 0.0001	0.555(0.457,0.675)	< 0.0001
Quartile 3 (8.388,10.008]	0.321(0.270,0.383)	< 0.0001	0.595(0.490,0.724)	< 0.0001	0.617(0.488,0.780)	< 0.0001
Quartile 4 (10.080,13.185]	0.193(0.158,0.235)	< 0.0001	0.433(0.348,0.537)	< 0.0001	0.403(0.302,0.539)	< 0.0001
P for trend		< 0.0001		< 0.0001		< 0.0001

Crudel model: no adjustment was made for any covariate

Model 1: adjusted by age and sex

Model 2: adjusted by age, sex, ethnicity, marital status, education level, poverty income ratio, smoking, alcohol use, fast total cholesterol (mg/dl), HDL cholesterol (mg/dl), LDL cholesterol (mg/dl), and physical activity (MET/week)

OR, odds ratio; 95% CI, 95% confidence interval; eGDR, estimated glucose disposal rate

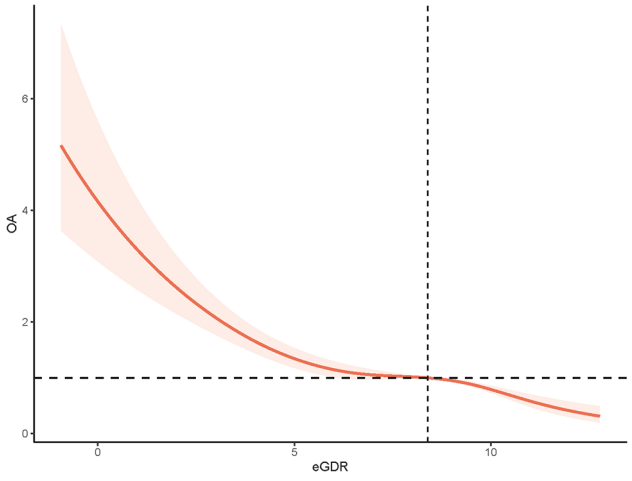

Fig. 2 Restricted cubic spline fitting for the association between eGDR with osteoarthritis. Knot = 4, the position of the nodes is located at 5%, 35%, 65%, and 95% of the percentiles of the predicted variables distribution

[31]. Long-term high insulin state may aggravate joint degeneration and cartilage damage by inducing chronic low-grade inflammatory response as well as activating other metabolic pathways. Research [32] has shown that chronic low-grade inflammation is one of the important mechanisms of OA, and IR further aggravates the degeneration of articular cartilage by increasing the secretion of proinflammatory cytokines (such as TNF-α, IL-6, etc.). Moreover, hyperinsulin can also act directly on cartilage leading to the degradation of cartilage [33]. Secondly, in addition to disordered glucose metabolism, IR may also lead to abnormalities in various lipid metabolism in the body. Before histopathological signs of OA appear, significant lipid accumulation, including total free fatty acids (FFA) and saturated fatty acids (SFA), has been observed in the articular cartilage of OA patients [34]. The accumulation of lipids can disrupt the metabolism of cartilage and contribute to cartilage degradation [35].

In this study, a significant and stable association between eGDR and OA was found, and its predictive efficacy was significantly higher than the five measures of IR as mentioned in our previous study [5]. HOMA-IR and TyG reflect IR through fasting blood glucose, fasting insulin, and fasting triglyceride levels. However, IR is a central feature of the metabolic syndrome, and individuals with IR are more likely to develop some other diseases strongly associated with the metabolic syndrome, such as obesity and hypertension. Obesity and hypertension are also independent risk factors for OA. Obesity can not only accelerate joint wear and tear by increasing joint weight bearing but also aggravate joint load and promote joint degeneration through pro-inflammatory factors [36]. Hypertension can accelerate the progression of OA by increasing the blood vessel burden, affecting the blood supply of the joints and the supply of nutrients [37]. HOMA-IR and TyG only focus on some aspects of IR when reflecting metabolic health and fail to comprehensively together other metabolic risk factors such as obesity and hypertension. TyG-BMI, TyG-WC, and TyG-WTHR, as derivative indicators of TyG, show closer association with OA than HOMA-IR and TyG alone, one of the important reasons is the combination of weight indicators (BMI, WC or WtHR). Although HOMA-IR and TyG already can assess IR, they may be limited due to the lack of comprehensive consideration of obesity and hypertension. In contrast, eGDR can provide a more comprehensive model of metabolic health assessment by taking into account WC and hypertension. Therefore, eGDR outperforms indicators such as HOMA-IR, TyG, and TyG-derived indicators in the predictive power of OA. Similar conclusions have also been found in several studies. A study by He et al. [38] including 17,787 individuals showed that the efficacy of eGDR to assess IR all-cause mortality and cardiovascular mortality was significantly higher than HOMA-IR, TyG, and TyG-derived indicators (TyG-BMI, TyG-WC, and TyG-WtHR). In addition, similar conclusions were found in the Xu et al. [39] study where eGDR was significantly associated with the presence and severity of diabetic retinopathy in T2DM patients and its predictive efficacy was stronger than other IR substitution measures, including TyG-BMI

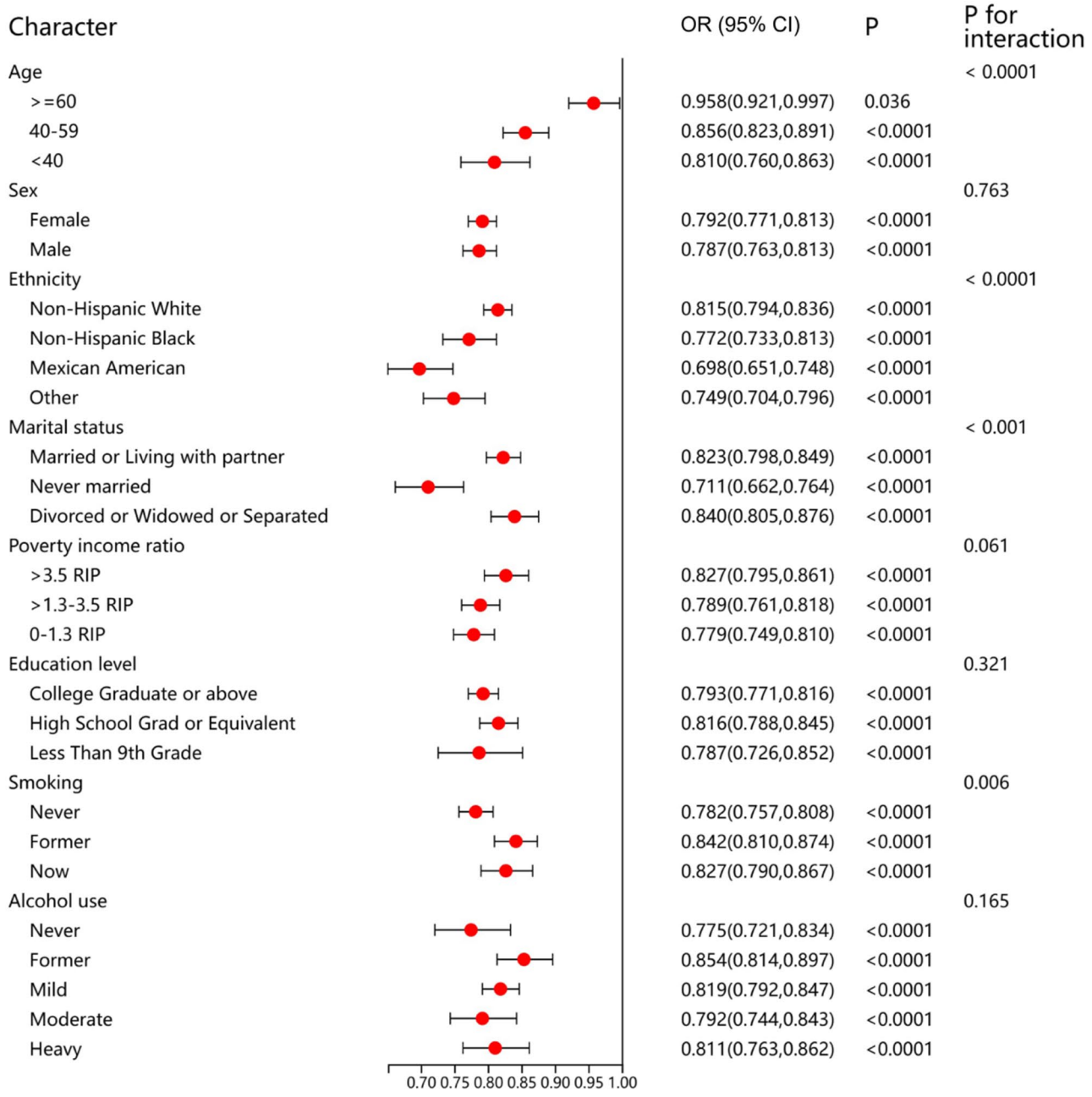

Fig. 3 The results of logistic regression analysis on the stratified association between eGDR and OA risk according to participants characteristics. The round red circles represent the point estimate of the OR, while the bar lines represent the 95% CI around the OR. OR, odds ratio; 95% CI, 95% confidence interval; eGDR, estimated glucose disposal rate

and TyG-WtHR. In conclusion, using eGDR as an IR surrogate to assess the risk of OA is a reliable option.

The strengths of this study lie in its use of a large, representative dataset from NHANES, which enhances the robustness and generalizability of the findings across a broad population. Rigorous statistical analyses, including covariate adjustments and sensitivity analyses, further bolster the reliability of the results. However, this study also has several limitations. Its cross-sectional design limits the ability to establish causal associations. The reliance on self-reported OA diagnoses may introduce reporting bias, and unmeasured confounders may have influenced the observed associations. Additionally, the applicability of these findings to populations outside the U.S. remains uncertain due to the dataset's specific demographic composition. Future studies addressing these gaps could further validate and expand upon these findings.

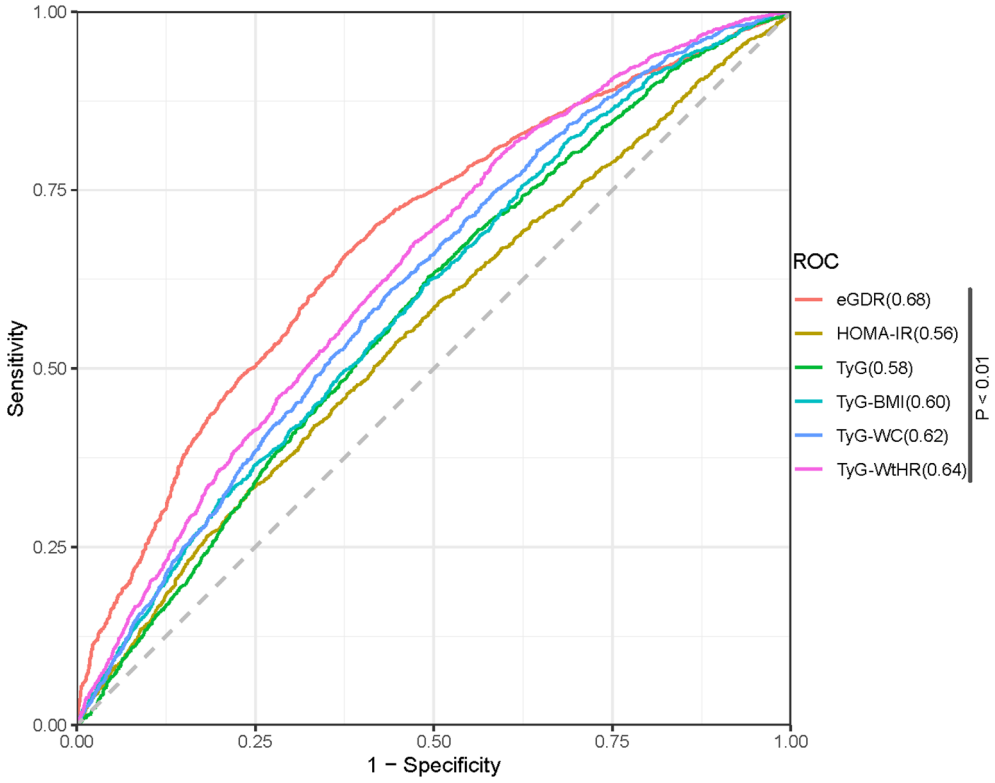

Fig. 4 ROC curves for different surrogates to predict osteoarthritis. All the five indices used to predict OA differ significantly from each other($P < 0.01$)

Conclusion

In conclusion, this study suggests that eGDR is independently associated with OA, with lower eGDR values being linked to a higher risk of OA. These findings support the previous research linking IR to OA and underscore the possibility of using eGDR as a novel IR surrogate indicator to manage OA.

Abbreviations

AGEs	Advanced glycation end products
CDC	Centers for Disease Control and Prevention
CI	Confidence interval
eGDR	Estimated glucose disposal rate
HbA1c	Glycated hemoglobin
HOMA-IR	Homeostatic model assessment of insulin resistance
IR	Insulin resistance
MET	Metabolic equivalent
NHANES	National Health and Nutrition Examination Survey
OR	Odds ratio
OA	Osteoarthritis
PIR	Poverty income ratio
SD	Standard deviation
T2DM	Type 2 diabetes mellitus
TyG	Triglyceride-glucose index
TyG-BMI	Triglyceride glucose with body mass index
TyG-WC	Triglyceride glucose with waist circumference
TyG-WtHR	Triglyceride glucose with the ratio of waist circumference divided by height

Acknowledgements

Thanks to Zhang Jing (Second Department of Infectious Disease, Shanghai Fifth People's Hospital, Fudan University) for his work on the NHANES database. His outstanding work, nhanesR package and webpage, makes it easier for us to explore NHANES database.

Author contributions

Conceptualization: Z.Q. and D.C.; Methodology: Z.Q., and H.C.; Software: Z.Q., H.C., and D.C.; Validation: Y.H.; Formal analysis: Z.Q. and H.C.; Investigation: D.C.; Resources: Z.Q.; Data curation: Z.Q. and D.C.; Writing-original draft preparation: Z.Q., D.C. and H.C.; Writing-review and editing: Z.Q., D.C. and H.C.; Visualization: Y.H.; Supervision: G.R., Y.H. and W.L.; Project administration: G.R.,Y.H. and W.L.; Funding acquisition: G.R. All authors reviewed the manuscript.

Funding

This research was funded by the Xiamen Science and Technology Plan Project (3502Z20224ZD1003).

Data availability

All NHANES data included in this study were publicly available at http://www.cdc.gov/nchs/nhanes/.

Declarations

Ethics approval and consent to participate

The study protocol of NHANES received approval from the Ethics Review Board of the National Center for Health Statistics. All participants provided written informed consent. This study followed the Reporting on Strengthening Observational Studies in Epidemiology reporting guidelines.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Received: 3 December 2024 / Accepted: 20 March 2025
Published online: 26 March 2025

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