

Prognostic Value of Prediabetes for Major Adverse Cardiovascular Events in Patients with Coronary Heart Disease: A Systematic Review and Meta-Analysis

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Abstract

Background

Coronary Heart Disease (CHD) is a leading cause of death among adults, incurring high socio-economic burdens. Prediabetes is highly prevalent in patients with CHD and may be associated with an increased risk of major adverse cardiovascular events (MACE). This study aims to systematically evaluate the effects of prediabetes on the risk of MACE in patients with established CHD and to explore variations in this association across different prediabetes diagnostic criteria.

Methods

Six bibliographic databases (Embase, PubMed, Web of Science, CNKI, Wanfang Data, and VIP Chinese Journals Database) were systematically searched to 1 January 2026 for prospective and retrospective cohort studies investigating the association between prediabetes (defined by the ADA, WHO or IEC criteria) and MACE in adult patients with CHD. Pooled hazard ratios (HRs) with 95% confidence intervals (CIs) were calculated. The risk of bias was assessed by ROBINS-E tool. Subgroup and sensitivity analyses were conducted to explore potential sources of statistical heterogeneity.

Results

Twenty cohort studies involving a total of 63,425 patients with a median of 3 years follow-up were included. Compared with normoglycemic group, coexisting prediabetes was significantly associated with higher risk of MACE in patients with CHD (pooled HR = 1.22, 95% CI = 1.12–1.33, $I^2 = 44.0\%$). Consistently, elevated risks were observed for all-cause death: HR = 1.30 (95% CI: 1.12–1.50, $I^2 = 0.0\%$) and cardiac death: HR = 1.24 (95% CI: 1.08–1.42, $I^2 = 1.4\%$). Subgroup analysis by prediabetes diagnostic criteria showed consistent risk elevation across ADA, WHO, and IEC criteria, and suggested that these criteria contributed to the observed statistical heterogeneity. The pooled HRs of glycated hemoglobin (HbA1c) were: ADA-HbA1c: HR = 1.28 (95% CI: 1.17–1.41, $I^2 = 0.0\%$); IEC-HbA1c: HR = 1.41 (95% CI: 1.21–1.65, $I^2 = 0.0\%$). The ADA composite criteria (HbA1c + FPG): HR = 1.19 (95% CI: 1.07–1.33, $I^2 = 51.0\%$); WHO and IEC composite criteria (WHO-FPG, IEC-HbA1c): HR = 1.53 (95% CI: 1.26–1.85, $I^2 = 0.0\%$). No statistical differences ($P > 0.05$) were found across a range of population and study characteristics.

Conclusions

This meta-analysis of cohort evidence indicates that prediabetes is associated with an increased risk of MACE in patients with coronary heart disease, and the magnitude of risk differs by prediabetes diagnostic criteria. However, pooled estimates for 2h-OGTT were unavailable due to insufficient data.

Future studies with more diverse CHD populations and complete outcome reporting are needed to confirm these findings.

INTRODUCTION

Coronary Heart Disease (CHD) is a leading cause of death among adults globally, affecting approximately 254 million people and accounting for an estimated 8.99 million deaths in 2021¹. CHD affects all age groups, with incidence increasing approximately threefold per decade of life², and a shift towards a younger demographic has emerged in recent years³. The direct annual cost of CHD per patient has reached 21.7% of GDP per capita⁴, causing large socio-economic burden worldwide. Preventing major adverse cardiovascular events (MACE)⁵ is a core goal in CHD management, reflecting both healthcare quality and long-term prognosis.

Prediabetes (PreDM) is a high-risk metabolic state for type 2 diabetes mellitus (T2DM)⁶. It represents a major global public health challenge, with prevalence of approximately 38% among US adults⁷ and 35.2% in China⁸. By 2045, an estimated of 1 billion individuals are expected to be affected globally⁷. Prediabetes can be identified through routine screening with haemoglobin A1c (HbA1c), fasting plasma glucose (FPG), and oral glucose tolerance test (OGTT)⁹. Importantly, early diagnosis enables more effective intervention¹⁰.

Prediabetes is a common comorbidity in patients with CHD. Pathophysiological defects typical of T2DM—such as increased lipolysis, glucagon secretion, and incretin deficiency—are also present in prediabetes⁶, leading to endothelial dysfunction, impaired fibrinolysis, and vascular injury¹¹. A 2020 global meta-analysis¹² reported that prediabetes was associated with an increased risk of CHD (RR = 1.16) compared with normoglycemia. Among patients with established CHD, those with prediabetes may represent a high-risk subgroup requiring targeted prognostic attention.

However, several challenges persist. First is the uncertainty of risk quantification. While the association between prediabetes and cardiovascular risk is well explored in general populations, its specific prognostic value for MACE in established CHD patients remains inconsistently reported¹³. Furthermore, there is currently no consensus on the optimal diagnostic criteria or thresholds for prediabetes¹⁴. Several definitions are adopted across clinical practice, including the American Diabetes Association (ADA)⁹, World Health Organisation (WHO)¹⁵, and International Expert Committee (IEC)¹⁶ (thresholds see Appendix S1). Whether the predictive power of listed criteria for MACE in CHD patients is equivalent, and whether these associations vary across study-level, population-level or risk of bias remains unclear.

This systematic review and meta-analysis of cohort studies aims to provide updated and comprehensive evidence on the prognostic value of prediabetes for MACE in patients with CHD and to compare the associations across different prediabetes diagnostic criteria.

METHODS

This review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Review and Meta-Analyses (PRISMA) guidance¹⁷. The PROSPERO registration number: CRD420261304753.

Search Strategy

The following bibliographic databases were searched: PubMed, Embase, Web of Science, China National Knowledge Infrastructure database (CNKI), Wanfang Data Knowledge Service Platform, and VIP Chinese Journals Database over the period 1 January 2020 to 1 January 2026. Other potentially additional studies were identified through manual screening of reference lists. Inclusion was limited to English and Chinese language studies (details see Appendix S1).

Eligibility Criteria

Prospective or retrospective cohorts were included if they met the following criteria: (1) Population: adults (≥ 18 years) with CHD (using any recognised diagnostic criteria); (2) Exposure: prediabetes defined by the ADA, WHO, and IEC criteria, including impaired fasting glucose (IFG), impaired glucose tolerance (IGT), or elevated HbA1c (see Appendix S1). Prediabetes should be clearly defined and evaluated at baseline; (3) Comparator: normoglycemic patients with CHD as the non-exposed reference group; (4) Outcomes: reporting MACE outcome or similar composite cardiovascular outcome with minimum 6 months follow-up. MACE was defined as a composite endpoint consisting of at least three components¹⁸, include: death endpoints (e.g., all-cause death and cardiac death) and vascular event endpoint (myocardial infarction, stroke, revascularisation, etc.). Secondary outcomes included all-cause death, cardiac death, myocardial infarction, and stroke.

Studies were excluded by the following criteria: (1) Did not report MACE outcome; (2) Measured glycemic parameters but did not classify participants using predefined prediabetes diagnostic thresholds; (3) Reported only unadjusted risk estimates; (4) Did not provide pooled risk estimates; (5) Were not available as full-text publications; (6) Were animal experiments, cross-sectional studies, randomized controlled studies, conference proceedings, editorials, letters, and review.

Literature search results were imported into EndNote 21 for deduplication. Titles and abstracts of the remaining records were screened, and full-texts of potentially eligible studies were reviewed for final inclusion. Studies were screened independently by two reviewers (C.D. and Y.G.). Where inclusion decisions could not be resolved, a third reviewer (R. S. T.) was involved.

Data Extraction

Data extraction included study characteristics (i.e., year, country, study design, predefined prediabetes criteria, follow-up duration, and sample size), patient characteristics (i.e., age, gender, BMI, and comorbidities), and MACE components with corresponding HRs. Core data were extracted from both

full-texts and supplementary materials. Data extraction was undertaken by one reviewer (C. D.) and checked by a second reviewer (Y. G.).

Bias Assessment

Risk of bias for each study was assessed using the Risk Of Bias In Non-randomized Studies - of Exposure (ROBINS-E)¹⁹. We evaluated the following seven domains: (1) Bias due to confounding; (2) Exposure measurements; (3) Selection of participants; (4) Bias due to post-exposure interventions; (5) Missing outcome data; (6) Outcome measurements; (7) Selection of reported results. Each domain was rated as 'low risk', 'some concerns', 'high risk' or 'very high risk'. Two reviewers (C.D. and Y.G.) independently conducted assessments, and inconsistencies were resolved through group discussion. Meta funnel plots and the Egger's test²⁰ were performed to assess publication bias when over 10 included studies. Trim-and-fill method was conducted to assess the potential effects of publication bias.

Data Synthesis and Analysis

Categorical variables and risk estimate sizes were expressed as pooled HRs and corresponding 95% confidence Intervals (95% CIs). The extracted HRs and 95% CIs were converted to natural logarithms and corresponding standard errors, and pooled using the inverse variance approach. For a study²¹ reported only adjusted odds ratios, an approximate HRs were estimated based on the baseline risk in the control group²². Statistical heterogeneity was assessed using Cochran's Q Test and I^2 statistic. Thresholds for the I^2 statistic interpretation were 0% to 40% (low risk of heterogeneity), 30% to 60% (moderate heterogeneity), 50% to 90% (substantial heterogeneity), and 75% to 100% (considerable heterogeneity)²³. A fixed-effects model was employed when $I^2 < 40\%$; otherwise, a random-effects model was adopted.

Subgroup analyses and sensitivity analyses were performed to explore statistical heterogeneity when 10 or more studies reported MACE. Subgroups were predefined by: (1) patients characteristics [e.g., prediabetes diagnostic criteria (ADA vs. WHO vs. IEC), geographic region (Europe vs. East Asia), CHD clinical presentation (acute coronary symptom vs. stable CHD)]; and (2) study characteristics [study design (prospective vs. retrospective), follow-up duration (< median vs. >median), and sample sizes (<median vs. >median)]. Sensitivity analyses were conducted by: (1) comparing pooled estimates between studies rated 'low risk' vs. 'some concerns or higher risk' based on each domain of the ROBINS-E; and (2) leave-one-out analyses, sequentially removing included studies to test the robustness of overall results. All statistical analyses were conducted using Stata MP18.

RESULTS

Study Selection

A total of 1959 papers were identified through database searching. Following de-duplication, 1483 papers were screened by titles and abstracts, of which 95 full-text papers were independently assessed for eligibility. Seventy-three studies were excluded as they did not meet the predefined PICOS inclusion

criteria. Three additional studies were excluded due to insufficient or unreported follow-up data: two in-hospital studies^{24,25}, and one study²⁶ with no median follow-up. One study²⁷ was excluded because the full text was unavailable. Ultimately, 20 relevant studies were included in meta-analysis^{21,28-46}.

[Insert Figure 1 approximately here]

Study and Population Characteristics

The 20 included cohort studies involved a total of 63,425 CHD patients. The population and study characteristics for each included study are summarized in Table 1 and 2. Eleven studies were conducted in China, five in Korea, three in Europe, and one in Iran. One study³³ was originally published in Chinese. Eleven studies were retrospective and nine were prospective. The median sample size was 2118, and the median follow-up was 3 years. Five studies^{30,39,42,43,46} had more than 5000 participants, six studies^{30,35,37,38,41,45} had follow-up > 5 years.

A range of prediabetes diagnostic criteria were reported, including ADA, WHO, and IEC. ADA criteria were most commonly applied in various forms: FPG alone (4 studies), HbA1c alone (9 studies), composite of FPG and HbA1c (12 studies), and a composite of FPG, 2h PG and HbA1c (1 study²⁸). WHO criteria were used as FPG alone (4 studies). IEC criteria were reported based on HbA1c alone (4 studies) and WHO-IEC composite criteria (WHO-FPG + IEC-HbA1c, 3 studies). See Table 2.

[Insert Table 1 approximately here]

[Insert Table 2 approximately here]

Risk of Bias Assessment

Insufficient reporting resulted in a risk of at least ‘some concerns’ risk across several domains, including selection of participants (unclear selection criteria, non-random sampling design), post-exposure interventions (unclear whether interventions were introduced after exposure or whether they affected outcomes), missing outcome data (lost to follow-up >5%, but with no explanation for its reason or assessment of its potential impact), and selection of reported results (no available protocol, incomplete outcome reporting, or unclear rationale for outcome grouping). Studies were judged to have a low risk for the ‘Exposure measurement’ and ‘Outcome measurement’ domain (see Figure 2 and Appendix S1).

[Insert Figure 2 approximately here]

MACE

Twenty studies reporting the association between prediabetes and MACE in patients with CHD were included in this study. The overall meta-analysis result showed that compared to normoglycemic population, the MACE risk of CHD patients complicated with prediabetes was estimated at 1.22 times

(95% CI: 1.12-1.33, $I^2=44.0\%$). Among patients with CHD, the prediabetic state was significantly associated with an increased risk of MACE (See *Figure 3*).

[Insert Figure 3 approximately here]

MACE Results by Different Prediabetes Diagnostic Criteria

Subgroup analyses by prediabetes diagnostic criteria were performed to assess variation in the association between prediabetes and MACE. Table 3 and Figure 4 provide a summary of pooled results across studies.

ADA Criteria

Compared with normoglycaemia, ADA-based diagnostic criteria consistently showed an increasing trend of MACE. Increases were observed in ADA composite criteria (HbA1c+FPG): HR= 1.19 (95% CI: 1.07-1.33, $I^2=51.0\%$), and ADA-HbA1c: HR= 1.28 (95% CI: 1.17-1.41, $I^2=0.0\%$). The association based on ADA-FPG indicated a non-significant trend toward increased risk: HR= 1.16 (95% CI: 0.98-1.38, $I^2=0.0\%$; See *Figure 4A to 4C*). The ADA-2h-PG result was reported only by Karayiannides et al.³⁵ (HR=0.94, 95% CI: 0.69-1.26). Ferrannini et al.⁴³ provided a composite ADA-2h PG+FPG result (HR= 1.11, 95% CI: 0.94-1.32).

WHO Criteria

The association based on WHO-FPG did not reach statistical significance but also showed a trend toward increased risk (HR= 1.13, 95% CI: 0.90-1.42, $I^2=0.0\%$; See *Figure 4D*). Pooled analysis was not possible because no 2h-OGTT data were reported.

IEC Criteria

The IEC-HbA1c criteria showed a significantly increased risk of MACE in patients with CHD and coexisting prediabetes (HR= 1.41, 95% CI: 1.21-1.65, $I^2=0.0\%$; See *Figure 4E*).

WHO and IEC Composite Criteria

Increases of MACE risk were seen in WHO and IEC Composite Criteria (WHO-FPG + IEC-HbA1c): HR= 1.53 (95% CI: 1.26-1.85, $I^2=0.0\%$; See *Figure 4F*).

All-cause Death, Cardiac Death, MI and Stroke

Due to the heterogeneity across prediabetes diagnostic criteria and the limited number of studies reporting secondary outcomes under alternative definitions, analyses were restricted to the ADA composite criteria (HbA1c+FPG). Both all-cause death and cardiac death showed an increased risk associated with prediabetes in patients with CHD. For all-cause death, seven studies involving 32,029 patients were included in the meta-analysis, with a pooled HR of 1.30 (95% CI: 1.12-1.50, $I^2=0.0\%$) (See

Appendix S1). Eight studies (30,741 patients) showed a pooled HR of 1.24 (95% CI: 1.08-1.42, $I^2=1.4\%$) for cardiac death. No statistically significant increased risk was observed for myocardial infarction or stroke (see Appendix S1).

[Insert Table 3 approximately here]

[Insert Figure 4 approximately here]

Subgroup Analyses

Population and study-based subgroup analyses were conducted to explore potential sources of heterogeneity. All subgroups indicated an increased risk of MACE. No significant differences (all P for interaction > 0.05) were observed across subgroups defined by geographic region (Europe vs. East Asia), study design (Prospective vs. Retrospective), median follow-up duration (> 3 years vs. ≤ 3 years), and sample size ($>$ median vs. $<$ median). The pooled HR for MACE was 1.29 (95% CI: 1.15-1.45, $I^2= 42.7\%$) in patients with acute coronary syndrome (ACS) and 1.13 (95% CI: 0.94-1.37, $I^2= 0.0\%$) in those with stable CHD. However, the difference was not statistically significant (P for interaction= 0.363) (See Figure 5).

[Insert Figure 5 approximately here]

Sensitivity Analyses

Sensitivity analyses found no evidence that bias domains of confounding, missing outcome data, and selection of reported results were sources of statistical heterogeneity, as heterogeneity tests between all groups were not statistically significant ($P> 0.05$).

The leave-one-out sensitivity analysis for MACE showed that sequentially excluding each of the 20 included studies yielded pooled HRs ranging from 1.212 to 1.249, and all corresponding 95% CIs excluding 1, suggesting that the overall finding was robust and not driven by a single study (See Figure 6 and Appendix S1).

[Insert Figure 6 approximately here]

Publication Bias

Visual inspection of the funnel plot for MACE showed asymmetry, which was supported by Egger's test ($P = 0.023$), indicating potential publication bias (See Appendix S1). The trim-and-fill analysis imputed 8 additional theoretical studies on the negative side of the funnel plot. After incorporating these imputed studies, the pooled HR shifted to 1.13 (95% CI: 1.05-1.22) under the random effects model, suggesting that the main conclusion was robust despite the potential publication bias (See Figure 7).

[Insert Figure 7 approximately here]

Discussion

This systematic review and meta-analysis of cohort studies presented evidence for the association between prediabetes and adverse cardiovascular outcomes in patients with CHD. The pooled analysis showed that, in patients with CHD, prediabetes was associated with a 22% increased risk of MACE (HR = 1.22, 95% CI: 1.12–1.33), as well as elevated risks of all-cause death (HR = 1.30, 95% CI: 1.12–1.50) and cardiac death (HR = 1.24, 95% CI: 1.08–1.42).

These findings align with recent international studies. A previous meta-analysis¹² demonstrated that, compared with healthy individuals, prediabetes was associated with all-cause death (RR = 1.36) and composite cardiovascular disease (RR = 1.37) in patients with atherosclerotic cardiovascular disease. Our study extends to the subset of patients with established CHD.

Importantly, this observed increasing trend was consistent across all prediabetes diagnostic criteria (all pooled HRs > 1.0), and the differences among these criteria were sources of statistical heterogeneity. HbA1c was a stable predictor, showing a significant and highly consistent risk increase that escalated with the stringency of the diagnostic criteria. Using the lower ADA cut-off point of 5.7% (39 mmol/mol), the pooled HR for MACE was 1.28 (95% CI: 1.17–1.41). When applying the stricter ICE cut-off of 6.0% (42 mmol/mol), the risk increased further to 41% (pooled HR = 1.41, 95% CI: 1.21–1.65). For prediabetes defined by FPG, the observed trend of increased risk was not statistically significant, as consistently reported across multiple studies^{35,40,41,44}. The most frequently used (12/20 studies) ADA composite criteria (FPG+HbA1c) yielded an intermediate pooled HR of 1.19, falling between the isolated HbA1c-based and FPG-based estimates. This heterogeneity may be attributed to multiple factors. FPG primarily reflects hepatic insulin resistance and basal insulin secretion⁴⁷, whereas HbA1c captures the average blood glucose level over a period of time and is directly associated with the core pathophysiological process of atherosclerosis⁴⁸. These distinct physiological meanings may contribute to their different prognostic performance for MACE in patients with CHD. Imaging evidence has shown that elevated HbA1c exacerbates the vulnerability of atherosclerotic plaques in patients with CHD⁴⁹. Mechanistically, HbA1c promotes the formation of advanced glycation end products (AGEs), thereby driving the progression of atherosclerosis⁵⁰.

Subgroup analyses were performed to explore potential sources of heterogeneity. Differences in patient population and study characteristics across included studies were unlikely to contribute to statistical heterogeneity. We found no clear evidence that the pooled results were affected by risk of bias or any individual study. Sensitivity analyses support the robustness of reported results. These findings suggest that prediabetes is associated with a persistently increased risk of MACE in patients with CHD, emphasizing the necessity of incorporating prediabetes management into the lifelong follow-up. The value of integrated management of diabetes and CHD has been widely recognized⁵¹. Our study further suggests lowering the threshold for glycemic screening and implementing routine prediabetes screening. Furthermore, the healthcare management for CHD patients with coexisting prediabetes should not be viewed as a short-term effort. Sustained glucose monitoring and lifestyle interventions are

required to delay or even halt the escalation of cardiovascular risk over several years, thereby improving the health-related quality of life in this potentially large subgroup of patients.

Strengths and limitations

Our study provides a comprehensive analysis of the association between prediabetes and MACE in patients with established CHD. We systematically compared the prognostic value of MACE across different prediabetes diagnostic criteria. However, several limitations were recognised in our study. First, pooled analysis based on OGTT was not feasible due to insufficient reports of the IGT definition. Only one study³⁵ reported an isolated ADA 2h-PG result, and only a few studies^{28,43,46} provided evidence on composite criteria that incorporated 2h PG alongside other glycemic measures. The reasons for the absence of OGTT test were: unavailability (3 studies^{36,38,44}), practical difficulties (5 studies^{30,37,40,44,45}), and lack of IGT classification (4 studies^{31,32,34,39}). These observations reflect the real-world challenges of implementing 2h-OGTT in large clinical cohorts, including time constraints, procedural complexity, and patient acceptability, which were also reported by WHO⁵². Second, all included studies recruited patients from hospital admission records or patient registries rather than random sampling from the general population, which may lead to selection bias and reduce external validity. Third, blinding was not feasible due to the observational design. Adjusting for drugs started after the prediabetes diagnosis may cause mediator bias, whereas not adjusting for pre-diagnosis drugs may confound the association⁵³. However, most studies lacked information on the timing of medication initiation related to prediabetes diagnosis, making the direction of potential bias uncertain. Thus, we cannot exclude residual confounding or overadjustment. Moreover, only three studies^{37,40,46} focused on the stable CHD subgroup population, which may have limited statistical power.

Conclusions

This systematic review and meta-analysis indicates that prediabetes is significantly associated with an increased risk of MACE, all-cause death, and cardiac death in patients with established CHD. The magnitude of this association varies across PreDM diagnostic criteria and was source of statistical heterogeneity. Early detection and prompt management of prediabetes may help reduce the risk of adverse cardiovascular events and premature death. Our findings support the routine HbA1c screening for prediabetes in CHD patients to improve risk stratification and guide early intervention. Future studies with more diverse populations, standardized OGTT study design, and complete reporting are needed to further confirm and extend the prognostic value of prediabetes for MACE in patients with CHD, particularly across different diagnostic criteria.

Declarations

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None.

Author contributions

All authors conceived and designed the study.

C.D: study conceptualization, methodology, investigation (literature search, data collection), data curation, statistical analyses, data visualization, and writing – original draft.

Y.G: study conceptualization, methodology, investigation (literature search, data collection), data curation, statistical analyses, and writing – preliminary drafting.

R.S.T: study conceptualization, methodology, investigation (literature search strategy), statistical analyses, and writing – review & editing.

All authors read and approved the final manuscript.

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Availability of data and materials

Not applicable.

Conflict of interest statement

None.

Ethics approval and consent to participate

Does not involve ethical approval and consent.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing financial interests.

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Tables

Tables 1 to 3 are available in the Supplementary Files section.

Figures

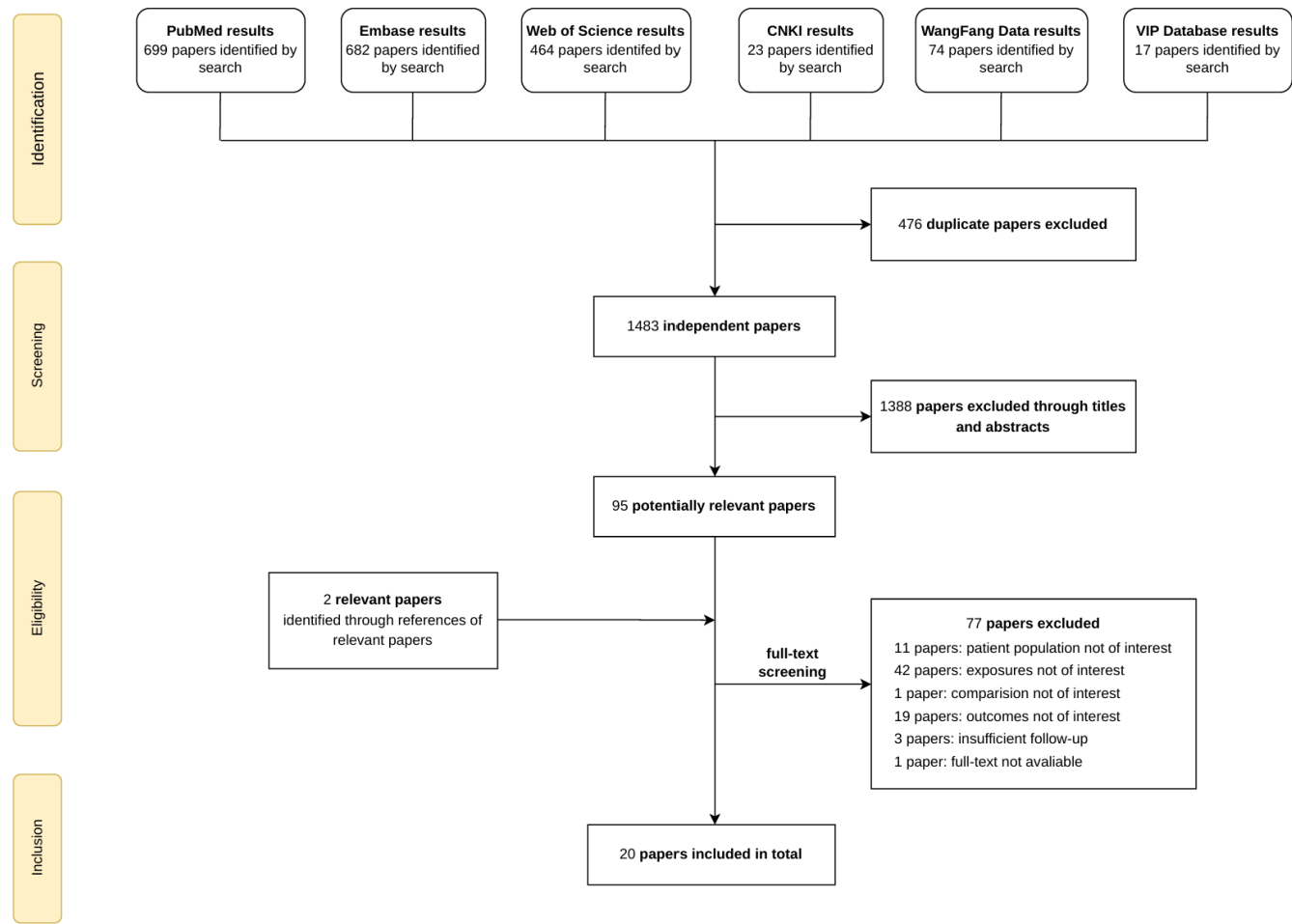


Figure 1

PRISMA flow diagram of study selection process

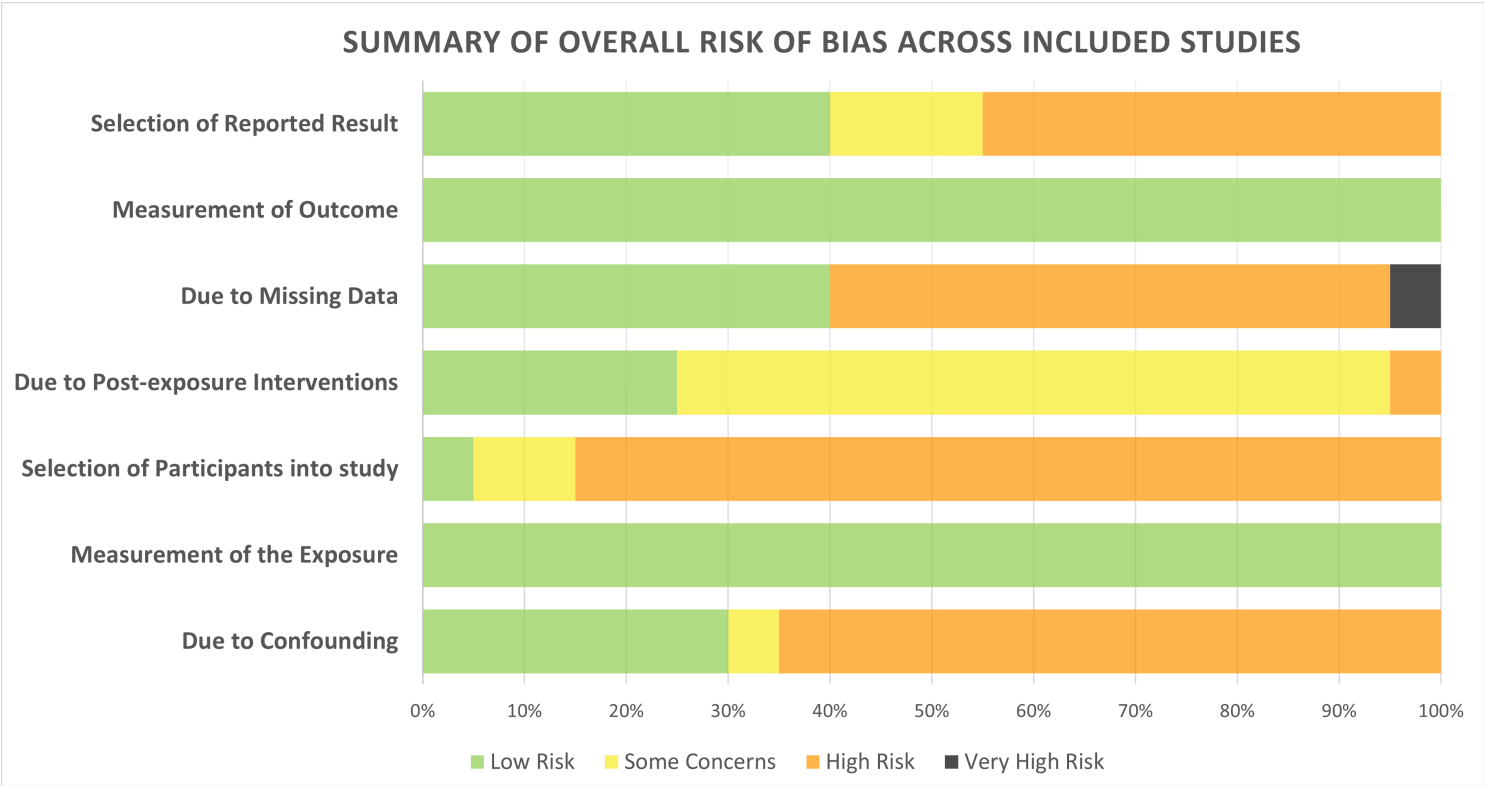


Figure 2

Summary of overall risk of bias across included studies

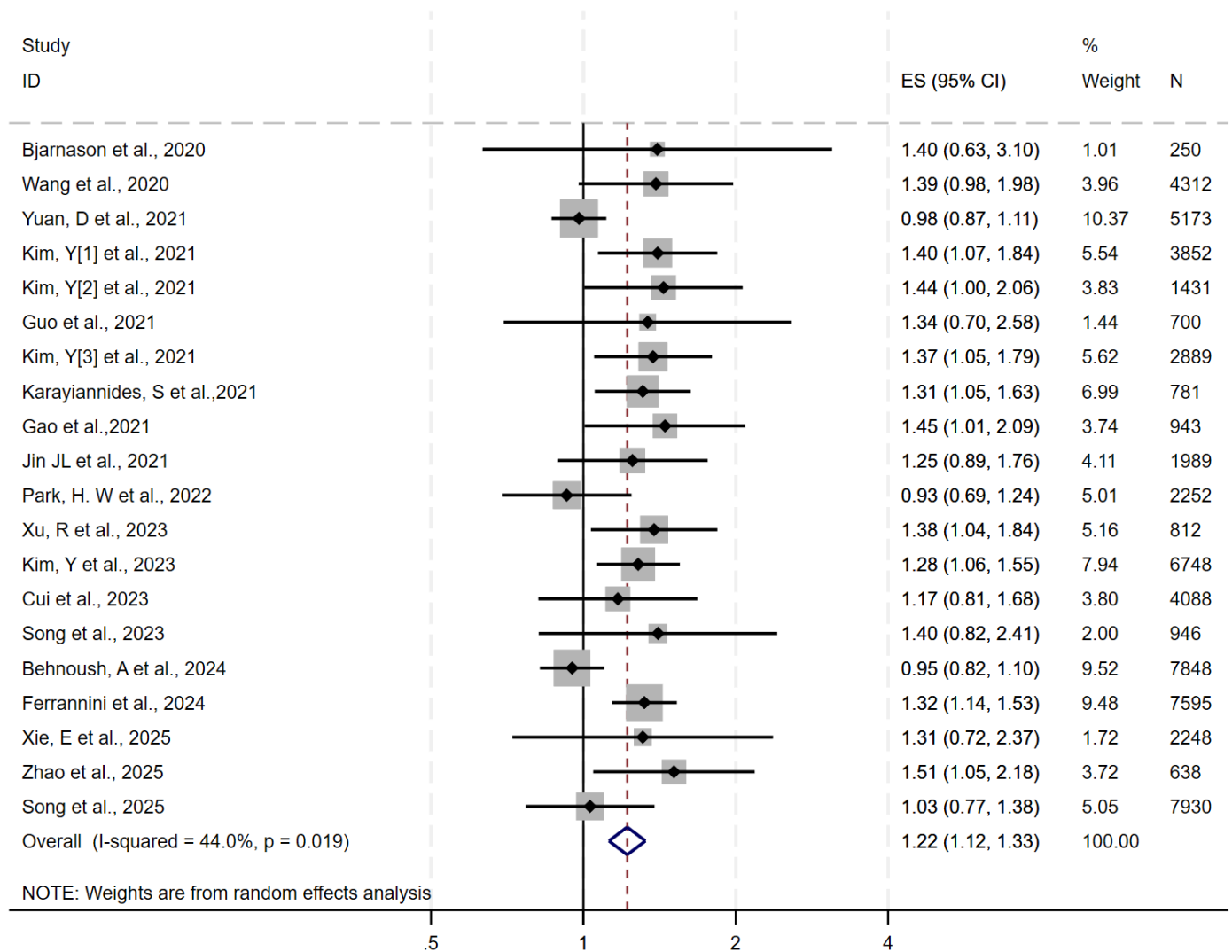


Figure 3

Forest plot of prediabetes vs. normoglycemia for MACE in CHD patients

Abbreviations: ES, effect size (pooled hazard ratio); 95% CI, 95% confidence interval; N, sample size.

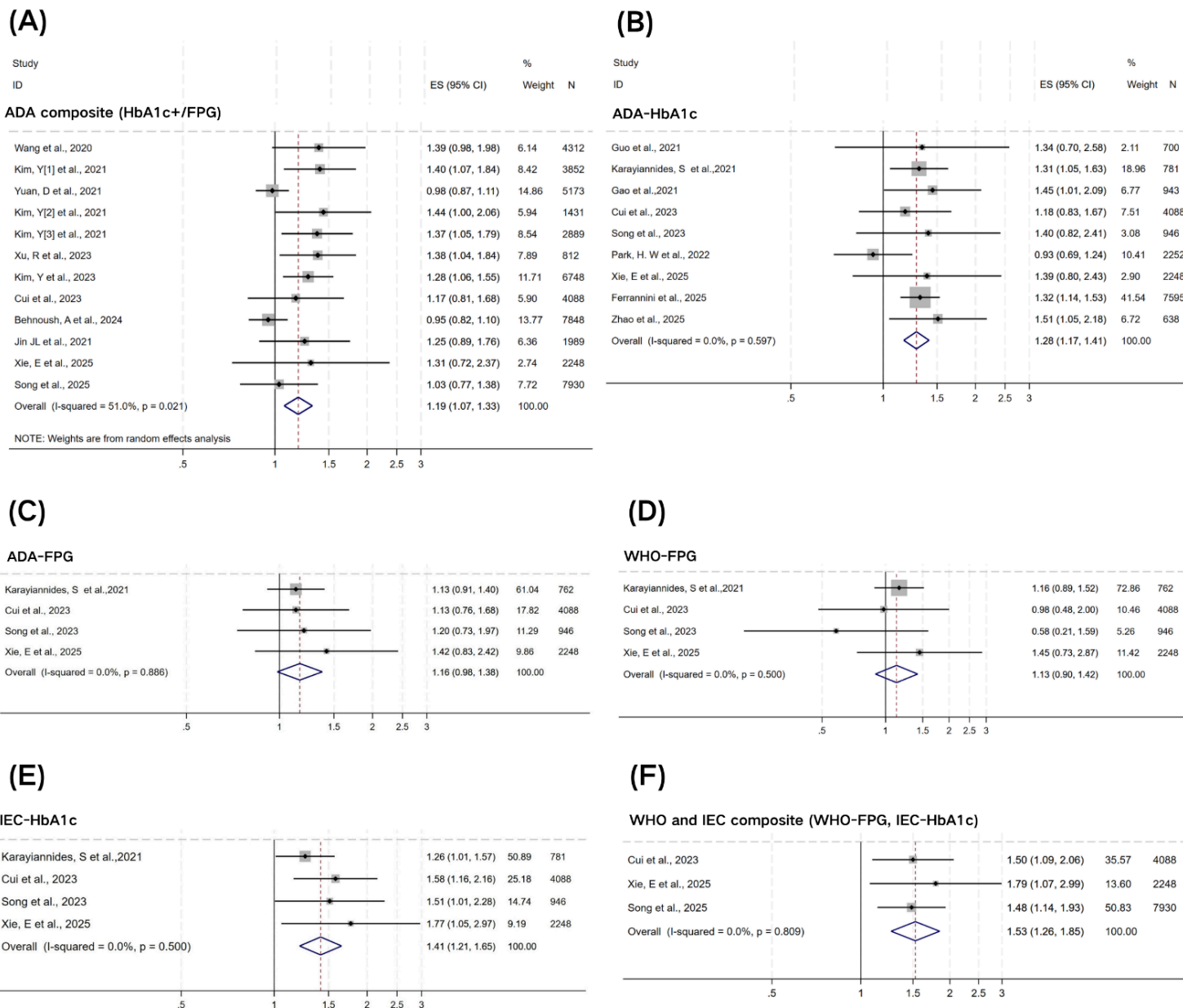


Figure 4

Forest plots of prediabetes vs. normoglycemia for MACE in CHD patients across different PreDM diagnostic criteria

Abbreviations: ADA, American Diabetes Association; WHO, World Health Organization; IEC, International Expert Committee; FPG, Fasting Plasma Glucose; 2h PG, 2-hour Plasma Glucose; HbA1c, Glycated Hemoglobin; ES, effect size (pooled hazard ratio); 95% CI, 95% confidence interval; N, sample size.

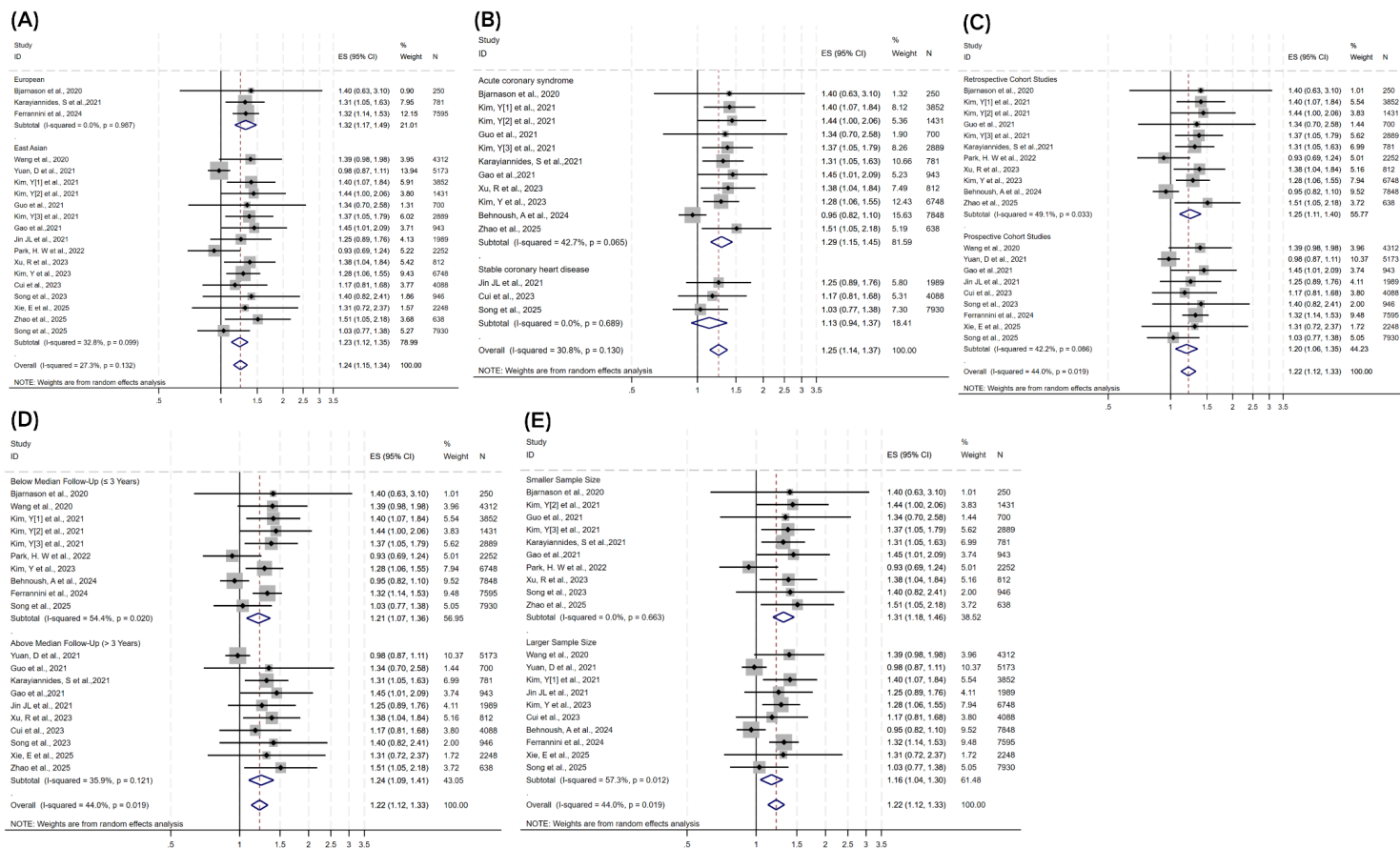


Figure 5

Forest plots of subgroup analysis of MACE

Abbreviations: (A) geographic region, (B) clinical presentation of coronary heart disease, (C) study design, (D) follow-up duration, (E) sample size. ES, effect size (pooled hazard ratio); 95% CI, 95% confidence interval; N, sample size.

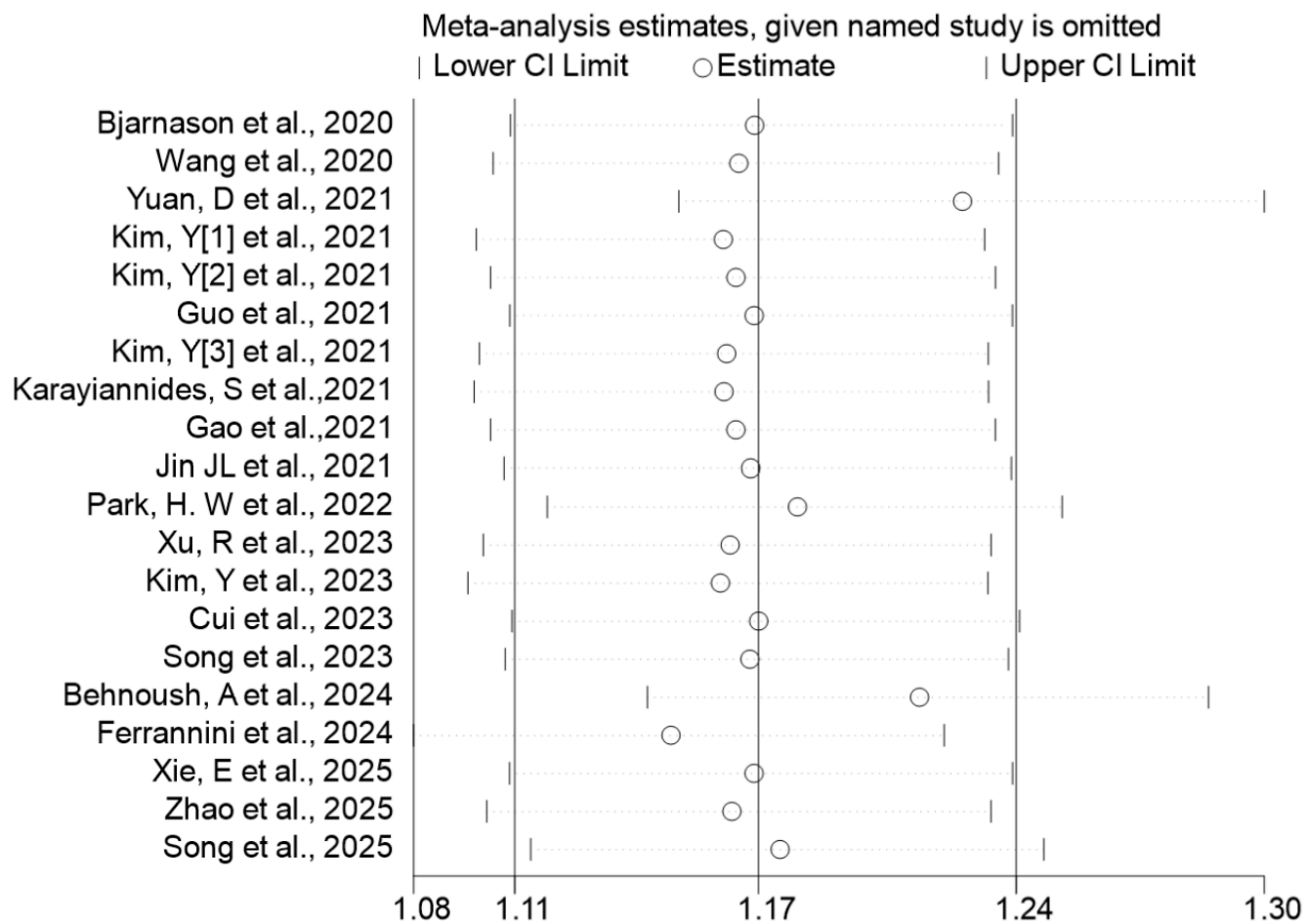


Figure 6

Leave-one-out sensitivity analysis for MACE

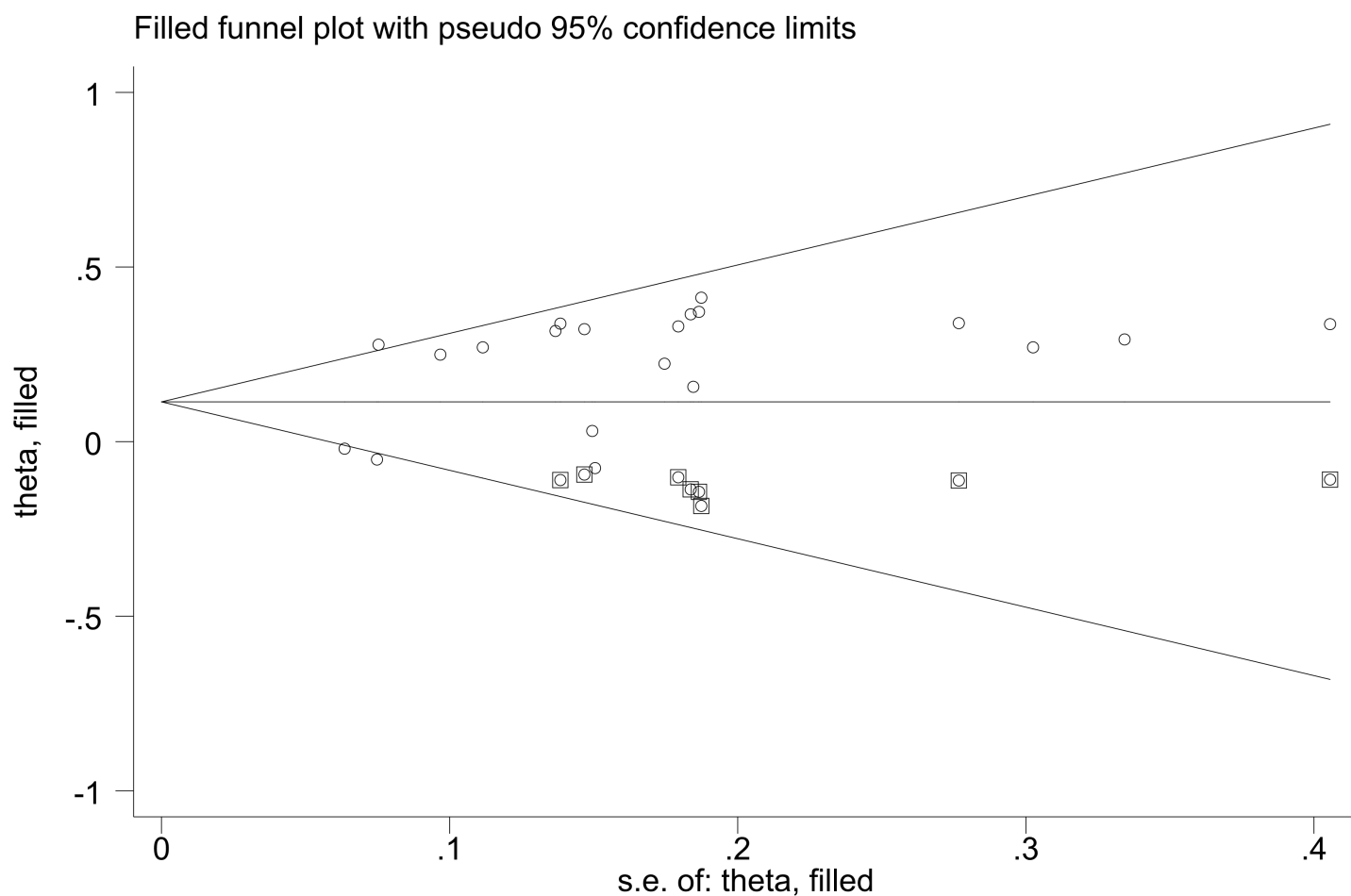


Figure 7

Trim-and-fill funnel plot for publication bias assessment of MACE

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [AppendixTable1Prediabetesdiagnosticcriteria.docx](#)
- [AppendixTable2Riskofbiasassessmentresultsofeachdomain.docx](#)
- [SupplementarymaterialsS1.docx](#)
- [Table1Populationcharacteristicsofincludedstudies.docx](#)
- [Table2Studycharacteristicsofincludedstudies.docx](#)
- [Table3SummaryofMACEmetaanalysesPrediabetesbydifferentcriteriavs.Normoglycemia.docx](#)