

Impact of Socioeconomic Inequalities on the Incidence of Type 2 Diabetes Mellitus: A Systematic Review

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Abstract

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Background:

Type 2 diabetes mellitus (T2DM) is a rising global burden, and socioeconomic inequalities may shape risk through differential resources, environments, and access to prevention and care. We synthesised evidence on the association between socioeconomic position (SEP) and incident T2DM.

Methods:

We conducted a PRISMA 2020–guided systematic review of PubMed, EMBASE, and Scopus (inception to 18 January 2026). Observational studies of adults without diabetes at baseline that measured SEP (education, income, occupation and/or area deprivation) prior to diagnosis and reported incident T2DM were eligible. Random-effects meta-analyses pooled relative risks (RRs), treating hazard ratios as approximations. Risk of bias was assessed (NOS).

Result:

From 1,580 records, 25 studies met inclusion criteria and 23 contributed to quantitative synthesis. Studies were mainly prospective cohorts or nested case–control designs, largely from high-income countries, with follow-up from 3 to 34 years and participants aged 18–86 years. Lower education was associated with higher T2DM incidence (least vs most educated: RR 1.55, 95% CI 1.37–1.75). Lower occupational position was also associated with increased risk (lowest vs highest: RR 1.60, 95% CI 1.25–2.05). Income was not statistically conclusive (lowest vs highest: RR 1.37, 95% CI 0.94–2.01).

Conclusions:

Socioeconomic disadvantage, particularly lower education and occupational status, is consistently associated with higher risk of incident T2DM. Prevention and screening should incorporate SEP to better target upstream determinants.

Introduction

Type 2 diabetes mellitus (T2DM) is a rapidly growing global clinical and public health burden, driving cardiovascular

disease, kidney failure, vision loss, and lower-limb amputation.¹ The World Health Organization reports that the number of people living with diabetes increased to 830

million in 2022, with faster growth in low- and middle-income countries (LMICs) than in high-income settings.² Consistent with this trajectory, the International Diabetes Federation estimates 537 million adults (20–79 years) were living with diabetes in 2024, contributing to about USD 1 trillion in health expenditure and 3.4 million deaths in that year alone.³ In clinical practice, these numbers translate into rising demand for earlier risk identification, durable prevention strategies, and long-term management of complications.⁴

Socioeconomic inequalities are increasingly recognized as “upstream” drivers of T2DM incidence through multiple, clinically relevant pathways: differential access to healthy foods, opportunities for physical activity, exposure to chronic stress, occupational risks, and barriers to preventive care and timely diagnosis.⁵ Population-level evidence supports a consistent socioeconomic gradient in incident T2DM. Socioeconomic context may also modify risk from common exposures seen in routine histories (e.g., work patterns): long working hours (≥ 55 /week) were linked to incident T2DM only among low socioeconomic status groups (risk ratio 1.29), but not among high socioeconomic status groups.⁶

Despite substantial literature, findings vary by how socioeconomic status is measured (education, income, occupation, area deprivation), by geography and health-system context, and by the degree to which studies adjust for mediators such as obesity, diet, and access to care. A focused synthesis is therefore needed to clarify the strength and consistency of the association between socioeconomic inequalities and new-onset T2DM, and to inform clinically actionable approaches (risk stratification, targeted screening, and prevention strategies tailored to deprived populations). Accordingly, the objective of this study is to systematically review and synthesize the available evidence on the impact of socioeconomic inequalities on the incidence of type 2 diabetes mellitus.

Material And Methods

This systematic review was conducted and reported in accordance with the

PRISMA 2020 guideline, and the study selection process was documented using a PRISMA flow diagram.⁷ A protocol was developed a priori to specify the research question, eligibility criteria, outcomes, and synthesis approach.

A comprehensive literature search was performed in PubMed, EMBASE, and Scopus from inception until 18 January 2026. The search strategy combined controlled vocabulary (e.g., MeSH and Emtree terms) and free-text keywords related to type 2 diabetes incidence and socioeconomic inequalities, including terms such as “type 2 diabetes,” “incident diabetes,” “new-onset,” “socioeconomic status,” “education,” “income,” “occupation,” “deprivation,” and “inequality.” Reference lists of included studies and relevant reviews were also screened to identify additional eligible studies.

The study question was defined using the PICO framework. The population comprised adults (≥ 18 years) without diabetes at baseline. The exposure of interest was socioeconomic disadvantage measured at the individual level (income, education, occupation) and/or area level (deprivation indices). The comparator was the most advantaged socioeconomic group or the least deprived area, depending on how the original study categorized socioeconomic position. The outcome was incident type 2 diabetes mellitus, ascertained by physician diagnosis, medical records or registries, validated algorithms, laboratory criteria, or the initiation of glucose-lowering medication consistent with type 2 diabetes.

Eligible studies were observational analytic designs (prospective or retrospective cohort studies, nested case–control studies, or case–control studies) that evaluated socioeconomic status as an exposure prior to the onset of type 2 diabetes and reported incident outcomes. Studies were required to provide an effect estimate (preferably risk ratio, but hazard ratios and odds ratios were also accepted) with confidence intervals or sufficient data to calculate an effect estimate. We excluded cross-sectional studies, ecological analyses without individual-level

linkage, case reports or series, editorials, narrative reviews, and conference abstracts lacking full extractable data, as well as studies focusing only on type 1 or gestational diabetes, studies reporting prevalence rather than incidence, and studies without extractable socioeconomic comparisons.

Records were de-duplicated prior to screening. Title and abstract screening and full-text eligibility assessment were conducted independently by all authors, and all authors also independently performed data extraction and risk of bias assessment. Any discrepancies at any stage were resolved through discussion until consensus was reached. Extracted data included study characteristics (country, setting, design), participant characteristics, follow-up duration, definitions and ascertainment methods for socioeconomic exposures and incident type 2 diabetes, covariates included in adjusted models, and effect estimates for comparisons between disadvantaged and advantaged socioeconomic groups.

Risk of bias was assessed using the Newcastle–Ottawa Scale (NOS) for observational studies, covering selection, comparability, and outcome or exposure ascertainment domains.⁸ The NOS results were used to support interpretation of the evidence base and inform sensitivity analyses.

The primary effect measure for synthesis was the risk ratio (RR) for incident type 2 diabetes mellitus comparing lower versus higher socioeconomic position; hazard ratios were treated as approximations of RR when pooled. Meta-analyses were conducted in R within RStudio-Posit IDE (R version 4.5.2 and RStudio 2026.01). Pooled estimates were calculated using random-effects models for all analyses, applying the DerSimonian–Laird estimator for between-study variance and Wald-type confidence intervals. Primary analyses were implemented using the meta package (version 8.2-1) and supported using metafor (version 4.8-0). Statistical heterogeneity was assessed using Cochran’s Q and quantified using I^2 and τ^2 . Where sufficient studies were available, publication bias was explored

using funnel plot inspection and standard asymmetry tests. No external funding was received for this study.

Result

Following database searching, 1,580 records were identified (PubMed n=322, EMBASE n=291, Scopus n=967). After removing 47 duplicates, 933 records were screened and 887 were excluded. Forty-six full-text reports were sought and retrieved (none were missing), and all 46 were assessed for eligibility; 21 were excluded (type 1 diabetes or gestational diabetes or autoimmune outcomes n=7, pre-diabetes or metabolic syndrome n=6, reviews or meta-analyses, editorials, or conference abstracts n=3, and inappropriate socioeconomic position (SEP) exposure measurement n=5), leaving 25 studies included in the review (Figure 1).^{9–33}

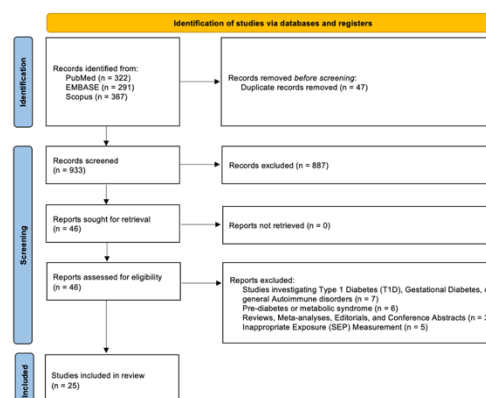


Figure 1. PRISMA flow diagram of the study selection process.

A total of 23 studies were included in the final quantitative synthesis, predominantly originating from high-income countries including the USA, Sweden, UK, and Finland, with fewer studies representing middle-income settings such as Brazil, Lithuania, and Mauritius (Table 1). The study designs were primarily prospective cohorts and nested case-control studies, with follow-up durations ranging from 3 to 34 years and participant

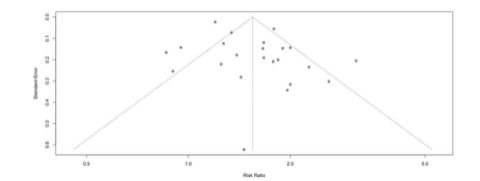
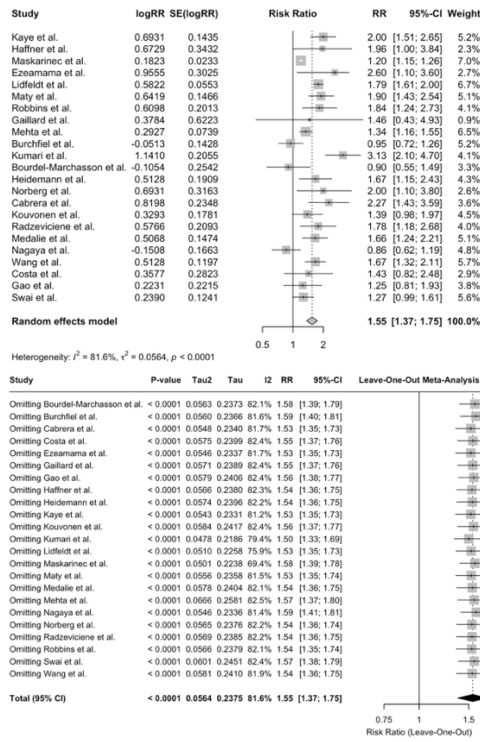


Figure 2. Meta-analysis of the association between educational attainment and type 2 diabetes risk: comparison of the least educated versus the most educated groups.

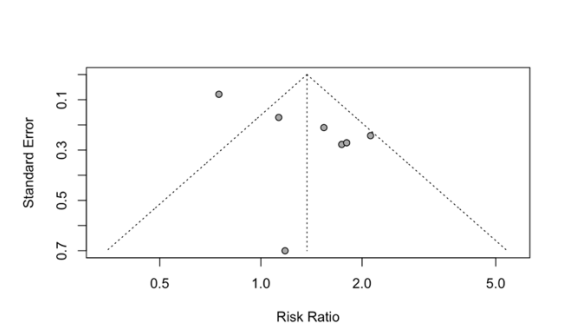
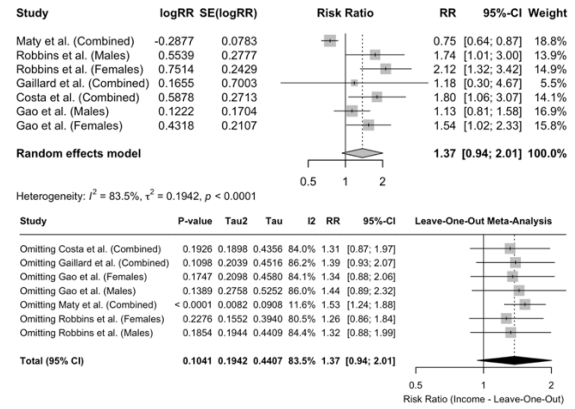


Figure 3. Impact of socioeconomic disadvantage on disease incidence: aggregated relative risks of type 2 diabetes comparing the lowest and highest income tiers.

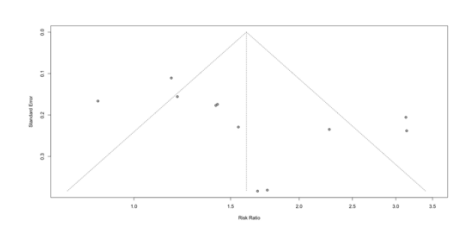
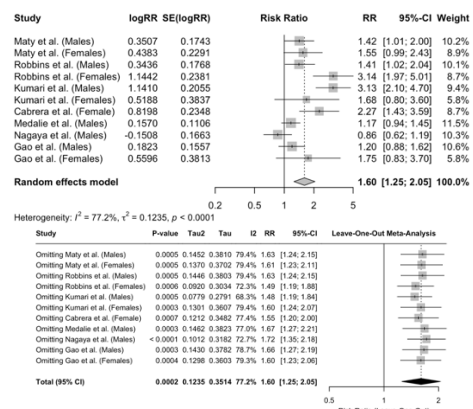


Figure 4. Pooled risk estimates for type 2 diabetes comparing individuals in the lowest occupational categories to those in the highest.

Discussion

This systematic review and meta-analysis synthesised evidence from 25 eligible studies (23 contributing to quantitative synthesis) to evaluate associations between SEP and incident type 2 diabetes. Across the three principal SEP indicators assessed we observed a consistent pattern whereby lower SEP was associated with higher risk of type 2 diabetes. The clearest and most robust associations were seen for educational attainment and occupational position. Compared with the highest categories, individuals in the least educated group had a 55% higher pooled risk of type 2 diabetes (RR 1.55, 95% CI 1.37–1.75), and those in the lowest occupational categories had a 60% higher pooled risk (RR 1.60, 95% CI 1.25–2.05). In contrast, the pooled association for income, while suggestive of increased risk in the lowest income groups, was not statistically conclusive (RR 1.37, 95% CI 0.94–2.01). Collectively, these findings support the presence of a social gradient in type 2 diabetes risk, with stronger evidence for education- and occupation-based inequalities than for income.

A key implication of the observed education and occupation gradients is that SEP likely influences diabetes risk through multiple, interrelated pathways operating across the life course. Educational attainment may shape health literacy, problem-solving skills, and the capacity to engage with preventive health information and services, while also influencing employment opportunities and working conditions.³⁴ Occupational position can reflect both material circumstances and psychosocial exposures, including job strain, shift work, lower decision latitude, and chronic stress, all of which have plausible links to cardiometabolic risk through behavioural and biological mechanisms.³⁵ Lower education and

occupational status are also commonly associated with less favourable neighbourhood environments and constrained access to healthy foods, safe spaces for physical activity, and preventive healthcare.³⁵

The inconclusive pooled estimate for income may reflect several factors rather than a true absence of association. First, income is often measured with greater error and temporal variability than education, which is usually fixed in adulthood) and occupation (which can capture long-term social standing.³⁶ Misclassification or use of broad categories can bias associations toward the null and increase uncertainty. Second, income may act differently across settings with varying welfare systems, healthcare access, and social safety nets, potentially increasing between-study heterogeneity and widening confidence intervals.³⁶ Third, income can be particularly sensitive to reverse causation when baseline health affects employment, productivity, or earning capacity; although included studies were largely prospective, varying degrees of baseline health adjustment and exclusion of early cases could influence income-related estimates more than education-related estimates.³⁷ Finally, income may exert effects primarily through mediators such as diet and adiposity; differences in analytic adjustment strategies across studies (e.g., controlling for BMI or behaviours) could attenuate pooled effects in some analyses.³⁸

The predominance of studies from high-income countries (e.g., USA, Sweden, UK, Finland) is important when interpreting generalisability. In such contexts, gradients by education and occupation may be particularly pronounced due to patterned exposure to obesogenic environments, occupational risks, and healthcare navigation barriers. However, the smaller number of studies from middle-income settings (e.g., Brazil, Lithuania, Mauritius) limits inference about how these

relationships manifest under different labour markets, nutrition transitions, healthcare systems, and social policies. It is plausible that SEP–diabetes gradients may differ in magnitude during periods of rapid economic and dietary change, underscoring the need for more evidence from diverse geographic and economic contexts.³⁹

Several methodological considerations may also influence the magnitude and comparability of pooled estimates. SEP measurement varied across studies in how categories were defined and when exposures were assessed, which can introduce heterogeneity and dilute pooled effects. Education, occupation, and income capture overlapping but distinct dimensions of SEP; therefore, differences in operationalisation can yield different effect sizes even when the underlying social gradient is similar. Outcome ascertainment likewise varied, including self-report (often verified), medical records, and glucose measurements, each with different sensitivity and specificity.⁴⁰ Non-differential misclassification of diabetes status could attenuate associations, whereas differential diagnosis rates by SEP (e.g., greater healthcare access among higher SEP leading to earlier diagnosis) could bias estimates in either direction depending on the setting and case-finding approach.⁴¹

Residual confounding and differences in adjustment strategies across studies are further considerations. Some studies may have adjusted for factors that are plausibly mediators (e.g., BMI, diet, physical activity), which would be expected to reduce SEP effect estimates and potentially underestimate the total impact of SEP on diabetes risk. Conversely, inadequate control for early-life factors, ethnicity, or neighbourhood deprivation could overstate associations in some contexts. The long follow-up periods (3–34 years) strengthen temporal inference but also introduce complexity due to SEP mobility, changes in exposures and behaviours over time, and

evolving diagnostic criteria and treatment patterns.⁴²

Despite these limitations, the consistency and magnitude of associations for education and occupation suggest that socioeconomic inequalities are a meaningful determinant of type 2 diabetes risk. From a public health perspective, these findings highlight the importance of addressing structural and upstream drivers, such as educational opportunities, working conditions, and equitable access to prevention and early detection, alongside individual-level behavioural interventions. Policies that reduce material hardship, improve food and built environments, and mitigate hazardous or stressful work exposures may contribute to reducing diabetes incidence and narrowing health inequalities.^{43,44} Preventive strategies may be most effective when proportionate to need, ensuring greater intensity and accessibility for populations in lower SEP strata who carry a disproportionate burden of risk.

Future research should prioritise (i) increased representation from low- and middle-income countries, (ii) standardised SEP measurement to improve comparability, (iii) repeated SEP assessments over time to capture mobility and cumulative disadvantage, and (iv) careful modelling of mediation to distinguish total effects from pathways through adiposity and health behaviours. Studies examining intersectionality (e.g., SEP combined with sex, ethnicity, and migration status) and contextual determinants (e.g., welfare regimes, labour protections, neighbourhood deprivation) would further clarify where and for whom the SEP gradient is steepest and which policy levers are most likely to be effective.

Conclusion

This review provides evidence that lower educational attainment and lower

occupational position are associated with substantially higher risk of incident type 2 diabetes, while evidence for income differences remains suggestive but imprecise. The findings reinforce the need to embed diabetes prevention within broader social and policy actions aimed at reducing socioeconomic disadvantage and improving the conditions that shape cardiometabolic health across the life course.

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