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Review

The association between adverse childhood experiences (ACEs) and gestational diabetes mellitus (GDM): A systematic review and *meta*-analysis

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ABSTRACT

Gestational diabetes mellitus (GDM) has consequences for maternal and offspring health. Adverse childhood experiences (ACEs) contribute to GDM risk, but findings have been inconsistent. We synthesised evidence using a systematic review and *meta*-analysis of observational studies on ACEs before age 18 and GDM, including dose–response effects. Nine databases were searched from inception to 30 May 2025 based on a PROSPERO-registered protocol (CRD420251035754). Studies without extractable estimates or assessing diabetes not restricted to gestational onset were excluded. Two reviewers independently screened studies, extracted data, and assessed risk of bias using ROBINS-E. Random-effects models pooled adjusted odds ratios (aORs), with dose–response analyses using the Greenland–Longnecker method. Thirteen studies met eligibility criteria, 11 contributed to *meta*-analysis (n = 326,797). ACE exposure was associated with higher odds of GDM (aOR 1.15, 95% CI 1.12–1.18; I^2 = 0%). A cumulative dose–response effect was observed, with each additional ACE increasing GDM odds (aOR 1.13, 95% CI 1.08–1.19). This study extends prior work by incorporating dose–response modelling, updated pooled estimates from recent cohorts, a pregnancy-focused risk window, and consideration of mediating and moderating pathways. Overall, ACEs are associated with increased risk of GDM, supporting a life-course approach to maternity care.

1. Introduction

Gestational diabetes mellitus (GDM), defined as glucose intolerance first identified during pregnancy [1], affects 14% of pregnancies worldwide [2,3]. Its prevalence has increased over the past two decades [4], driven by demographic shifts, widening inequities, and improved detection [5,6]. Previously thought to fully resolved after birth, it is now clearly recognised that GDM is an independent risk factor for type 2 diabetes mellitus (T2DM) and cardiometabolic disease in women [7,8]. Furthermore, it is also associated with adverse metabolic and neurobehavioural outcomes in the offspring [9–11]. Pregnancy has therefore been described as a physiological stress test that can reveal underlying metabolic susceptibility [9].

Psychosocial determinants are increasingly recognised as important contributors to metabolic health alongside traditional biomedical factors [12]. Adverse Childhood Experiences (ACEs), such as abuse, neglect, or household dysfunction before age 18 [13], are a common form of early-life stress. Globally, most adults report at least one ACE [14–16], with higher prevalence among socioeconomically disadvantaged groups and those facing structural adversity [17–19]. Increases in reported ACEs may reflect both greater awareness and widening social inequities [20,21].

ACEs are associated with long-term alterations in stress-responsive systems [22–24], including dysregulation of the hypothalamic–pituitary–adrenal axis (HPA) and low-grade inflammation [25–28], which overlap with biological pathways implicated in insulin resistance and GDM [4]. Evidence suggests that early-life adversity may shape cardiometabolic risk across the life course [29–31], while

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pregnancy represents a physiological context in which these vulnerabilities may be amplified. The extent to which these processes influence GDM remains incompletely understood.

Empirical findings on the association between ACEs and GDM have been inconsistent. Some studies report increased risk among women exposed to childhood adversity [32], while others show attenuated or null associations after adjustment for key confounders [33]. Existing reviews have examined ACEs in relation to broader perinatal or cardiometabolic outcomes, limiting detailed exploration of GDM-specific pathways [13,34]. In parallel, evidence from systematic reviews on ACEs and T2DM demonstrates a long-term metabolic dysregulation across the life-course [3], while GDM is a known risk factor for subsequent T2DM [7]. Taken together, these findings suggest a potential continuum linking ACEs, GDM, and later-life metabolic disease. However, a focused synthesis quantifying the ACEs-GDM association and examining dose–response relationship remains limited. This study addresses this gap by isolating GDM as a primary outcome and integrating a life-course perspective.

This study aimed to systematically review and *meta*-analyse evidence on the association between maternal ACEs exposure and GDM, quantify dose–response relationships, and summarise psychosocial and physiological factors reported across studies that may shape this association.

2. Methods

2.1. Data sources

This systematic review and *meta*-analysis followed PRISMA 2020 guidelines and was registered with PROSPERO (CRD420251035754). Methodological considerations were guided by the Meta-analysis of Observational Studies in Epidemiology (MOOSE) reporting guidelines. The protocol is available online (https://www.crd.york.ac.uk/PROSPERO/view/CRD420251035754).

We searched nine databases from inception to 30th May 2025 (MEDLINE, Embase, PsycINFO, CINAHL, Scopus, Web of Science, Psychiatry Online, Global Health, and MIDIRS) using terms related to ACEs,

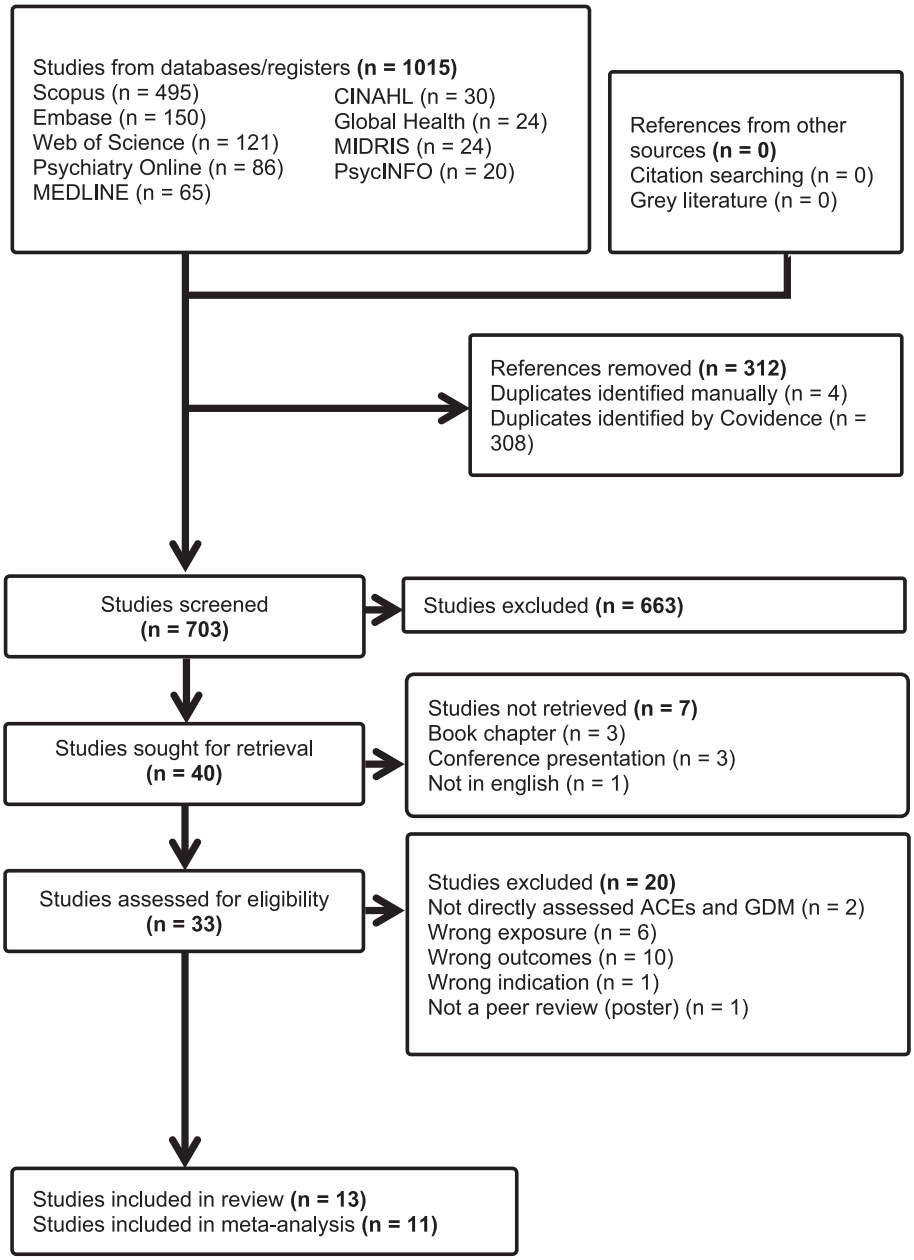

Fig. 1. PRISMA diagram illustrating the study selection process.

trauma, early-life stress, and GDM. Equivalent search strategies were adapted for each database (Table S1). Additional studies were identified through reference list screening. Grey literature was explored via WorldCat and the Conference Proceedings Citation Index.

Eligible studies were peer-reviewed quantitative observational designs (cohort or cross-sectional) that examined ACEs before age 18 and reported GDM diagnosis. Studies had to present an effect estimate (OR, relative risk [RR], or hazard ratio), or sufficient data to calculate one. Exclusion criteria were qualitative studies, conference abstracts without full data, non-English reports, and studies examining diabetes not restricted to gestational onset. Inclusion decisions and reasons for exclusion are detailed in the PRISMA flow diagram (Fig. 1).

Two reviewers (SNF and MB) independently screened titles, abstracts, and full texts in Covidence, resolving disagreements through discussion or consultation with a third reviewer (SB).

2.2. Data extraction and risk-of-bias assessment

Data extraction was conducted by one reviewer using a standardised form and independently verified by a second reviewer. Extracted variables included study design, sample characteristics, ACE assessment, GDM diagnostic criteria, covariate adjustment, and effect estimates. When multiple estimates were available, most adjusted estimates were used. Studies were included if sufficient information was provided in the main text or Supplementary materials to facilitate calculation, including raw counts to reconstruct 2 × 2 tables. Duplicate data from overlapping cohorts were resolved by retaining the study with the largest or most complete dataset. Where information was unclear or missing, corresponding authors were contacted; studies with unresolved gaps were retained qualitatively but excluded from quantitative pooling.

Risk of bias was assessed independently by two reviewers using the ROBINS-E tool [35], across domains of confounding, exposure classification, participant selection, missing data, outcome measurement, and selective reporting. Ratings reflected the highest-risk domain. Disagreements were resolved by consensus.

2.3. Data analysis

The primary outcome was the association between any ACE exposure (≥1 ACE vs none) and GDM, expressed as pooled aORs. Secondary outcomes included high vs low exposure and dose–response effects per additional ACE. Study-specific cut-offs were harmonised by classifying low ACE exposure as 0–1 ACEs and high exposure as ≥2 or ≥3 ACEs, depending on each study's categorisation. Because the included studies used different ACE measurement tools and reporting formats (e.g., dichotomous any vs none, categorical low vs high, or continuous score), studies were grouped into three comparable analytical subsets (Fig. 2).

Effect estimates from 11 studies [32,33,36–44], were eligible for inclusion in the *meta*-analysis, as shown in the appendix (Table S3 and S4). One study by Swedo et al. [42] reported findings for two distinct populations: North and South Dakota (NSD) and Kansas, Michigan, and Rhode Island (KMRI), which were treated as separate estimates.

Random-effects *meta*-analysis was conducted in R (version 4.0.2) with the metafor package [45]. Dose-response analysis applied the Greenland and Longnecker method via dosresmeta [46]. Heterogeneity was quantified with I2 and Cochran's Q [47].

Prespecified subgroup analyses examined study design (prospective vs retrospective), country income level (high-income (HIC) vs low- and middle-income country (LMIC)), and risk-of-bias category (low, medium, high). However, because all included studies were conducted in HIC settings, the prespecified subgroup analysis by country income level could not be performed. We instead examined population risk profile: general vs at-risk population (e.g., low-income clinics, mental health referrals, or adversity-exposed groups) as this better reflected variation across studies.

A narrative synthesis was conducted to summarise potential

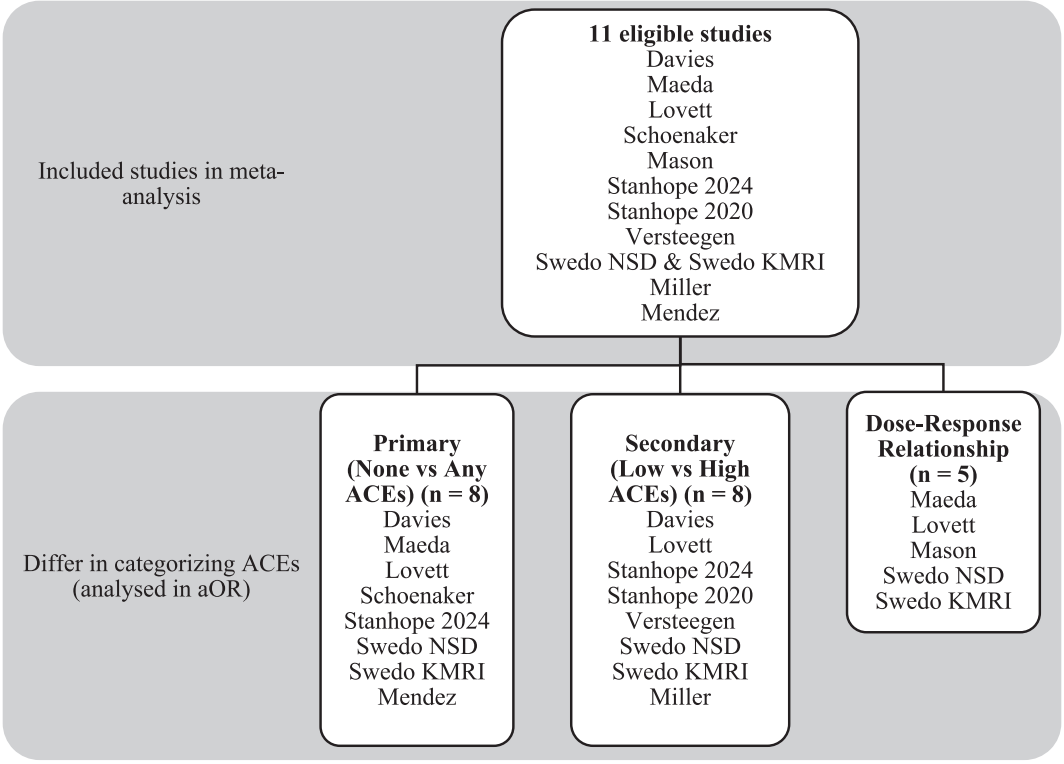

Fig. 2. Flow diagram of included studies for the primary, secondary, and dose–response *meta*-analyses. Effect estimates from 11 studies,[32,33,41-46,48,49,51] were eligible for inclusion in the *meta*-analysis, as shown in the appendix (Table S3 and S4). One study by Swedo et al.[48] reported findings for two distinct populations: North and South Dakota (NSD) and Kansas, Michigan, and Rhode Island (KMRI), which were treated as separate estimates.

psychosocial and biological moderators and mediators. Sensitivity analyses omitted one study at a time ("leave-one-out"). Publication bias was assessed using funnel plots and Egger's regression [48]. Evidence certainty was evaluated using GRADE [49].

3. Results

3.1. Study characteristics

The search identified 1015 records, of which 13 studies met inclusion criteria. Eleven studies were included in the *meta*-analysis, comprising 326,797 participants (Fig. 1). Studies were published between 2016 and 2025, and most were conducted in high-income countries (HICs), primarily the USA (n = 8). [33,38,40–44,50] Study characteristics are summarised Table 1.

ACEs were most commonly assessed using the CDC–Kaiser ACE-10 questionnaire (n = 5) [33,41,43,44,50], although several studies used the culturally-modified version [32,37,51], or different instruments. Risk of bias was generally moderate, mainly reflecting retrospective ACE reporting and sampling limitations (Supplementary Table S2).

3.2. Meta-analysis results

In the primary analysis (eight studies) [32,36–38,40,42,44], exposure to any ACE was associated with increased odds of GDM (pooled aOR 1.15, 95% CI 1.12–1.18; $I^2 = 0\%$; Fig. 3a). In the secondary analysis (eight studies) [33,36,38,40–43], high ACE exposure was associated with higher odds of GDM compared with low exposure (pooled aOR 1.29, 95% CI 1.08–1.53; $I^2 = 46.9\%$; Fig. 3b).

Dose–response modelling, based on five studies [37–39,42], demonstrated a cumulative association, with increasing odds of GDM per additional ACE (pooled aOR 1.13, 95% CI 1.08–1.19; $I^2 = 25.6\%$; Fig. 4). Effect estimates were consistent in direction across studies, although confidence intervals varied according to study size and exposure categorisation.

3.3. Heterogeneity, sensitivity, and certainty of evidence

Subgroup analyses for the primary outcome showed no statistically significant differences by study design (prospective vs retrospective), population risk profile (general vs at-risk populations), or risk-of-bias category (all $p > 0.05$; Supplementary Table S5). For the secondary analysis, moderate heterogeneity was observed, with studies rated as having some concerns contributing most to between-study variability.

Sensitivity analyses indicated that pooled estimates were robust to exclusion of individual studies (Supplementary Figs. S1 and S3). No small-study effects were detected (Egger $p > 0.10$; Supplementary Figs. S2 and S4). Overall certainty of evidence for the primary and secondary analyses was rated as moderate using GRADE criteria (Supplementary Table S7).

4. Discussion

4.1. Main findings

This systematic review of 13 studies (11 *meta*-analysed) provides evidence for a significant, dose-dependent association between maternal ACEs and GDM. Exposure to any ACE increased GDM odds by 15%, with risk rising cumulatively for each additional ACE. These findings support a cumulative-risk model consistent with allostatic load theory [52], in which chronic stress exerts physiological strain revealed during the metabolic challenge of pregnancy [53].

The associations were consistent across analytical approaches and remained robust in sensitivity analyses, despite heterogeneity in study design and exposure assessment. The absence of detectable small-study effects suggests that the pooled estimates are unlikely to be driven by selective publication. While effect sizes were modest, their consistency across large population-based cohorts underscores their public health relevance, particularly given the high global prevalence of both ACE exposure and GDM [2–4,14–16,54].

The pooled estimates are broadly consistent with previous reviews of ACEs and adverse pregnancy outcomes, which reported increased risk of metabolic-related complications with similar effect sizes [14,34]. However, these reviews treated GDM as part of composite outcomes, limiting interpretation of GDM-specific pathways. By isolating GDM as the primary outcome, this study provides more precise evidence of a dose–response relationship, supporting a cumulative biological embedding mechanism.

These findings also align with evidence linking ACEs to T2DM [7], where early-life adversity is associated with long-term metabolic dysregulation. Pregnancy may represent a clinical context in which this vulnerability becomes apparent, with GDM as an early expression of underlying risk.

GDM is a well-established risk factor for subsequent T2DM, and offspring of affected pregnancies are also at increased metabolic risk. Together, these findings suggest a potential intergenerational pathway linking ACE exposure, maternal metabolic dysregulation, and long-term risk in both mother and child, positioning ACEs as an upstream determinant of metabolic disease.

This study contributes by isolating GDM from broader pregnancy outcomes, demonstrating a dose–response relationship, and integrating a life-course perspective linking childhood adversity, pregnancy, and later metabolic disease.

4.2. Possible mechanisms

The association between ACEs and GDM likely reflects convergent neuroendocrine, inflammatory, metabolic, and placental pathways originating early in development. ACEs have been linked to long-term dysregulation of the HPA axis, including altered cortisol awakening responses and blunted reactivity that persist into adulthood and re-emerge during pregnancy [55]. These endocrine changes contribute to chronic inflammation, reduced glucocorticoid sensitivity, and impaired insulin signalling [56].

Psychosocial factors may also influence risk expression (Supplementary Table S6). Associations were stronger among women with preconception depression or high work stress [32,44], whereas race/ethnicity and childhood social support did not consistently modify estimates [40,41,44]. The non-linear associations observed in some groups may reflect protective cultural or social environments [6,37,51]. Adiposity appeared to have a modifying rather than mediating role; adversity-exposed women had elevated GDM risk even after adjustment for BMI, suggesting that physiological embedding of stress may contribute independently of fat mass while still influencing visceral adiposity and metabolic flexibility [32,39,40].

Placental pathways may also contribute to risk. Maternal adversity has been associated with reduced placental 11β-hydroxysteroid dehydrogenase type 2 activity and altered cortisol transport, which may affect maternal metabolic adaptation and fetal glucocorticoid exposure [57].

The cumulative dose–response pattern observed across studies is consistent with models of stress accumulation rather than single-exposure effects [52]. This pattern suggests that repeated or clustered adversities may progressively erode metabolic resilience, lowering the threshold at which pregnancy-related insulin resistance manifests as GDM [53]. Such cumulative processes may operate through both biological embedding and behavioural adaptation, whereby early adversity shapes long-term stress responsivity, health behaviours, and access to protective resources [12]. Pregnancy may therefore act as a critical inflection point at which these latent vulnerabilities become clinically apparent [9].

Table 1
Study characteristics.

Authors	Country	Participants N = 335,293	Study Design	Study Overviews	Adjustment	ACEs tools	Overall Outcomes	Meta-Analysis	Overall risk of bias
Davies et al. 2024.[41]	Denmark	208,207	Pro-spective cohort	**Ethnicity:** predominantly European origin, **SES:** high income and education level **Parity:** Nulliparous, **Mean maternal age:** 27.1 (low adversity) vs 24.1 (high adversity **ACEs prevalence:** 52% **GDM prevalence:** 2.6%	Maternal age, SGA, parental education, birth year	Administrative records, maternal questionnaires, and child reports from DANLIFE to construct domains of adversity.	Any adversity (vs. low) associated with higher GDM risk, but increasing adversity does not show a linear or graded increase in risk beyond a certain point.	Included	Low Risk
Maeda et al. 2024.[42]	Japan	5,444	Cross-sectional	**Ethnicity:** Japanese **SES:** high income and education level **Mean maternal age:** ~32 years old **ACEs prevalence:** 6.9% **GDM prevalence:** 5.8%	Parity, maternal age at delivery, maternal pre-pregnancy BMI, household income, maternal academic backgrounds, smoking status, gestational weight gain	ACE-J	The highest GDM risk was in the 1 ACE group, showing possibility of buffer effects (e.g. support from welfare system).	Included	Moderate Risk
Lovett et al. 2024.[43]	US	34,879	Pro-spective cohort	**Ethnicity:** predominantly NHW **SES:** College or associate degree (34%), middle income (41%), never smoke (58%) **Parity:** 2nd (46%) **Median age:** 40–50 s **ACEs prevalence:** 67% **GDM prevalence:** 4.1%	Age at baseline, race and ethnicity, childhood SES	Brief Betrayal Trauma Survey (BBTS)	22% increase in the risk for GDM was observed among women with a history of any trauma in early life. The high trauma class had nearly double the risk of GDM compared to the low trauma group; moderate was second.	Included	Moderate Risk
Schoenaker et al. 2019.[32]	Australia	6,317	Pro-spective cohort	**Ethnicity:** predominantly Australian born (White) **SES:** highly educated **Parity:** nulliparous 88.7% **Mean maternal age:** ~28 years **ACEs prevalence:** 45.3% **GDM prevalence:** 8%	Family history of diabetes, age at menarche, PCOS, preconception BMI, diet quality, parity, maternal age, and antenatal depression.	Modified CDC–Kaiser ACE-10 into Custom 9-item questionnaire (covering common ACE domains)	The association between ACEs and a greater GDM risk was confined to pregnancies where the mother had symptoms of depression before conceiving; a dose–response pattern was also observed in this group.	Included	High Risk
Mason et al. 2016.[44]	UK	45,500	Pro-spective cohort	**Ethnicity:** predominantly NHW **Mean maternal age:** 28 years **SES:** mean over 12 years of education **Parity:** mean 2.3 **Physical abuse:** 53% **Sexual abuse:** 32.7% **GDM prevalence:** 5%	Demographics (maternal age, birth year, race), SES (parental education, occupation, home ownership), and health history (parental diabetes, child's body size at age 5).	Revised Conflict Tactic Scales; sexual abuse screening	A statistically significant link emerged between a history of childhood physical and sexual abuse and an increased GDM risk. This risk was greatest for women exposed to both severe physical and forced sexual abuse, and a graded, dose–response relationship was also observed for physical abuse.	Included	Moderate Risk

(*continued on next page*)

Table 1 (*continued*)

Authors	Country	Participants N = 335,293	Study Design	Study Overviews	Adjustment	ACEs tools	Overall Outcomes	Meta-Analysis	Overall risk of bias
Stanhope et al. 2024.[45]	US	1,033	Pro-spective cohort	**Ethnicity:** white (53.8%) and black (46.2%) **SES:** predominantly 12–15 years of school, and unmarried (73.1%) **Parity:** predominantly nulliparous **ACEs prevalence:** 2.6% **GDM prevalence:** 13.1%	Parental education level, race, maternal age during pregnancy, and parity.	Childhood Family Environment Scale (CFE)	A statistically significant link emerged between higher childhood abuse scores and an increased likelihood of GDM, with the odds of GDM rising by 30% for every point increase on the abuse subscale.	Included	High Risk
Stanhope et al. 2020.[46]	US	2319	Cross-sectional	**Ethnicity:** Hispanic / Latina women **SES:** predominantly greater than high school graduates, and never smokes (72.6%) **Parity:** mean 2.9 **Mean maternal age:** 46.9 years **ACEs prevalence:** 77.4% **GDM prevalence:** 9.2%	Childhood SES, including education, race, age at US arrival, and migration status.	CDC–Kaiser ACE-10	No significant association between ACEs and GDM was identified	Included	High Risk
Versteegen et al. 2021.[33]	US	266	Pro-spective cohort	**Ethnicity:** NHW (52.4%) **SES:** predominantly low-income (16%), post-high school graduates (62%), and unmarried (62.4%) **ACEs prevalence:** 70.6% **GDM prevalence:** 8.3%	Age, BMI	CDC–Kaiser ACE-10	No significant relationship between ACEs and GDM both for continuous and categorical ACEs.	Included	Moderate Risk
Imanishi et al. 2025.[47]	Japan	5693	Retro-spective cohort	**Ethnicity:** Japanese **SES:** predominantly high income **Parity:** nulliparous **Mean maternal age:** 32 years **ACEs prevalence:** 37.8% **GDM prevalence:** 5.6%	–	ACE-J	No association between ACEs and GDM nor linear or graded increase of GDM risk with greater ACEs exposure.	Excluded from *meta*-analysis. Reason: double data possibility with Maeda et al.	Moderate Risk
Swedo et al. 2023.[48]	US	14,510	Cross-sectional	**Ethnicity:** predominantly NHW **SES:** high school or higher **Maternal age:** predominantly 25–34 years **Parity:** predominantly multiparous **ACEs prevalence:** 51.3% **GDM prevalence NSD:** 10.3% **GDM prevalence KMRI:** 9%	Age, race / ethnicity, marital status, parity, education, and insurance status.	8 items Behavioral Risk Factor Surveillance System (BRFSS)	There is a significant association between ACEs and GDM especially at higher exposure.	Included	Moderate Risk

(*continued on next page*)

Table 1 (*continued*)

Authors	Country	Participants N = 335,293	Study Design	Study Overviews	Adjustment	ACEs tools	Overall Outcomes	Meta-Analysis	Overall risk of bias
Miller et al. 2021.[49]	US	1274	Cross-sectional	**Ethnicity:** White (58.8%) **SES:** no data **Mean maternal age:** 32–33 years **Parity:** predominantly nulliparous **ACEs prevalence:** 93% **GDM prevalence:** 6.8%	Maternal age, insurance, marital status, tobacco use, chronic medical conditions, and obesity.	CDC–Kaiser ACE-10	No statistically significant association between ACEs and GDM, while GDM prevalence appeared higher in group with higher ACEs exposure (7.4% vs 6.7%), the trend was weak and insignificant.	Included	High Risk
Jasthi et al. 2023.[50]	US	192	Retro-spective cohort	**Ethnicity:** black (91.7%) **SES:** high school or higher graduate (75.6%), low-income women referred to a specialist in mental health case management to elevated psychosocial risk **Mean maternal age:** 25.5 years **Parity:** mean 3.2 **ACEs prevalence:** 47.4% **GDM prevalence:** 7.3%	–	CDC–Kaiser ACE-10	No significant association was found between ACEs and GDM. While there was a slight, non-significant increase in GDM cases at higher levels of ACE exposure, the magnitude of this difference was minor	Excluded from *meta*-analysis. Reason: not enough data, the number of GDM cases is too low.	High Risk
Mendez et al. 2024.[51]	US	1163	Pro-spective cohort	**Ethnicity:** 45% African American, 35% White, 16% Hispanic **ACEs prevalence:** 48% **GDM prevalence:** 5.9%	Age, education, Medicaid status at delivery, marital status, race, employment status, tobacco use during pregnancy, BMI, parity, and care group.	CDC–Kaiser ACE-10	No significant association for the general exposre to ACEs, however those with both childhood abuse and high work stress had nearly 4 times the odds of developing GDM.	Included	Moderate Risk

SES: Socioeconomic status, SGA: Small for gestational age, BMI: body mass index, DANLIFE: the DANish Life-course Cohort, ACE-J: Culturally adapted version of the CDC-Kaiser ACE-10 questionnaire; NHW: Non-hispanic white, PCOS: Poly-cystic ovarian syndrome.

4.3. Strength and limitations

Several limitations should be considered when interpreting these findings. Most included studies relied on cross-sectional or retrospective ACE reporting, limiting causal inference and introducing potential recall bias [58]. ACE definitions, severity, and timing were inconsistently measured across studies, and adjustment for key confounders, such as pre-pregnancy BMI, socioeconomic position, and maternal mental health, varied.[59] The predominance of evidence from HIC restricts generalisability to LMIC where adversity patterns and metabolic risk structures may differ [60,61].

Heterogeneity in exposure definition and outcome assessment represents a key challenge in synthesising observational evidence in this field. However, the directionally consistent associations observed across studies using different ACE instruments and GDM criteria suggest that the relationship is not artefactual. The use of adjusted estimates mitigates concerns regarding residual confounding, although unmeasured factors such as lifetime stress or intergenerational adversity may still influence results. These considerations support cautious interpretation while reinforcing the need for more harmonised measurement.

Despite these limitations, this review has notable strengths, including a comprehensive search across nine databases, dual-reviewer screening and extraction, structured risk-of-bias assessment using ROBINS-E, and dose–response *meta*-analytic modelling. Together, these methodological features strengthen confidence in the robustness of the findings.

4.4. Implications for research and practices

These findings support viewing GDM as a manifestation of cumulative life-course adversity. Trauma-informed approaches in maternity services may therefore be essential. Best-practice recommendations emphasise sensitive enquiry about past adversity, clear communication, continuity of care, psychological safety, and staff training to avoid re-traumatisation [62]. Such approaches are relevant given the heightened psychological and metabolic vulnerability among women with ACE histories [63].

Within diabetes research and clinical practice, these findings support broader recognition of psychosocial history as a relevant component of metabolic risk stratification in pregnancy. While ACE screening is not intended as a diagnostic tool, incorporating trauma-informed principles into routine antenatal care may enhance engagement, reduce stress-related barriers to care, and improve uptake of preventive interventions among women at elevated risk. Such approaches complement, rather than replace, established biomedical risk assessment and may be particularly relevant in settings serving socially disadvantaged populations where both ACE exposure and GDM prevalence are high.

The results also identify a population at increased risk for GDM for

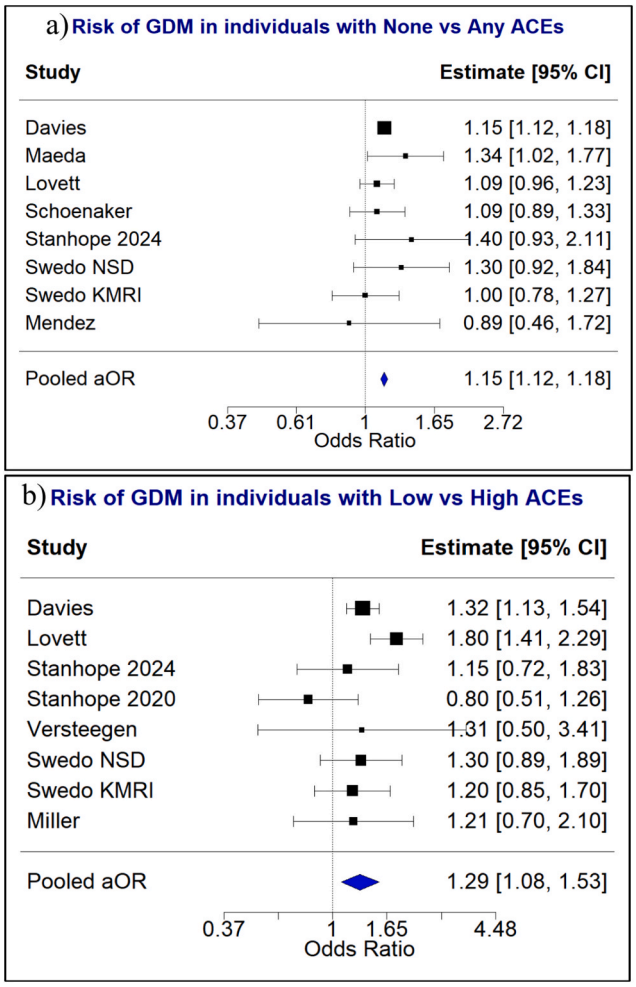

Fig. 3. Risk of GDM in individuals with No ACE vs Any ACEs and Low ACEs vs High ACEs.

whom targeted preventive and management strategies may be beneficial [5,12]. This includes tailored metabolic and psychological support during pregnancy and postpartum, when the risk of developing type 2 diabetes is substantially increased [3,64]. At a population level, the findings reinforce the importance of framing GDM prevention as an intergenerational challenge [65]. Preventing childhood adversity should therefore be considered a long-term public health strategy to reduce chronic disease burden across generations [66].

Intervention need not be limited to early childhood. Because ACEs may increase GDM risk through physiological and behavioural pathways, the preconception period represents a critical secondary window for intervention [67]. However, women face substantial barriers to engaging in preconception care, including limited information, time constraints, affordability, and inconsistent primary-care support, although peer support may help mitigate these challenges [68]. For women with known adversity histories, preconception care may need to extend beyond routine supplementation to include targeted metabolic screening, psychological support, and interventions aimed at normalising stress physiology and inflammation, consistent with upstream life-course strategies [5,55–57,69]. Later-life supports, including trauma-focused therapies, social support programmes, and workplace stress management, may also help buffer the long-term physiological imprint of adversity and reduce cumulative metabolic strain [63].

Together, these implications align with evidence linking ACEs to multiple pregnancy outcomes, suggesting shared biopsychosocial pathways. Unlike broader reviews [14,34], this synthesis clarifies the metabolic sequelae of ACE exposure and highlights opportunities to interrupt the trajectory from early adversity to GDM and later cardiometabolic disease [70].

4.5. *Future research*

Future research should prioritise large, prospective *peri*-conceptional cohorts to strengthen causal inference, reduce recall bias, and test biopsychosocial pathways linking ACEs to GDM. Incorporating placental epigenetic analyses may clarify biological transmission [63]. Standardised ACE and GDM measures and improved capture of adversity timing would enhance comparability [1,5,59,71]. Developmental neuroscience highlights adolescence as a sensitive window in which

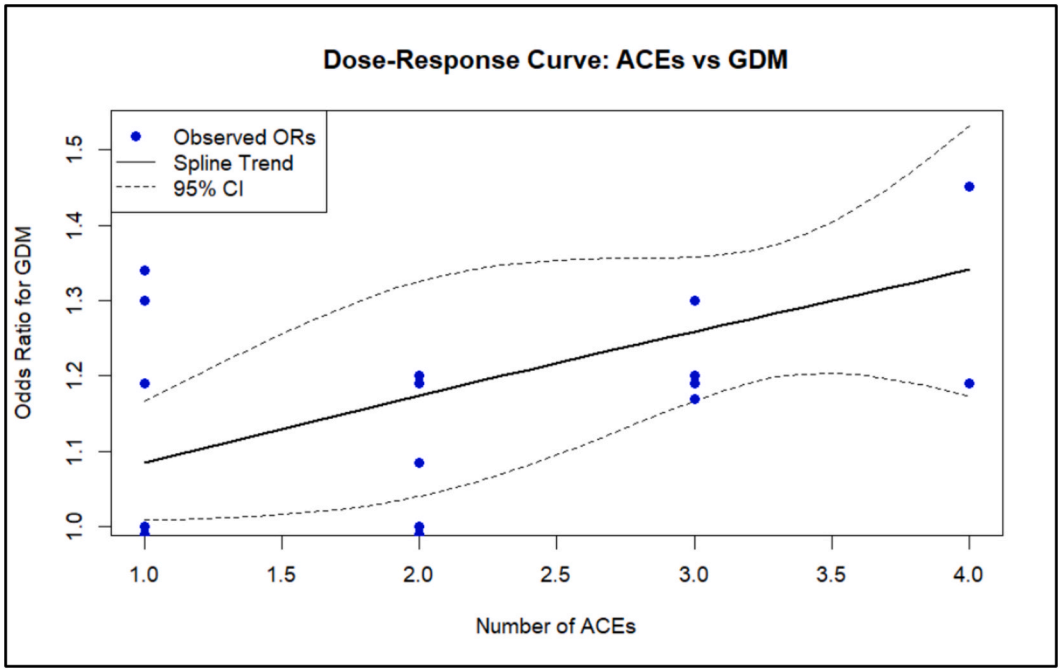

Fig. 4. Dose-response relationship of ACEs and GDM.

stress may shape later metabolic trajectories [72,73], underscoring the importance of longitudinal designs. Embedding HPA-axis and inflammatory biomarkers into future cohorts will further advance mechanistic understanding [55–57]. Research in diverse populations, particularly in LMIC, remains essential to understand how adversity interacts with structural inequities, nutritional transitions and sociocultural buffering [6,21,60].

5. Conclusion

Maternal exposure to ACEs is associated with increased risk of GDM in a dose-dependent manner. Although limitations in the available evidence preclude causal inference, the consistency of findings across studies supports the relevance of early-life adversity to metabolic vulnerability during pregnancy. These results reinforce a life-course perspective on GDM, in which psychosocial exposures contribute alongside established biomedical risk factors. Integrating trauma-informed principles into maternity care and strengthening prevention of childhood adversity may therefore represent important, complementary strategies for improving maternal and offspring metabolic health.

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Data sharing

The protocol for this review is publicly available on PROSPERO (CRD420251035754). Data extracted from included studies and the R scripts used for statistical analyses are available from the corresponding author upon reasonable request. Summary data used in the *meta*-analyses are provided in the Supplementary Materials.

CRediT authorship contribution statement

Salma Nur Fadhilah: Writing – review & editing, Writing – original draft, Validation, Resources, Project administration, Methodology, Formal analysis, Data curation, Conceptualization. **Sam Burton:** Writing – review & editing, Supervision, Methodology, Formal analysis, Data curation, Conceptualization. **Claire A. Wilson:** Writing – review & editing. **Madeleine Benton:** Writing – review & editing, Validation, Supervision, Methodology, Data curation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary material including detailed search strategies, risk of bias assessment (ROBINS-E), study-level effect estimates, dose–response data, subgroup analyses, GRADE assessment, and additional sensitivity and publication bias analyses can be found online at https://doi.org/10.1016/j.diabres.2026.113269.

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