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Prenatal alcohol exposure and associations with physical size, dysmorphology and neurodevelopment: a systematic review and meta-analysis

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Abstract

Background Fetal alcohol spectrum disorder (FASD) is a significant public health concern, yet there is no internationally agreed set of diagnostic criteria or summary of underlying evidence to inform diagnostic decision-making. This systematic review assesses associations of prenatal alcohol exposure (PAE) and outcomes of diagnostic assessments, providing an evidence base for the improvement of FASD diagnostic criteria.

Methods Six databases were searched (inception–February 2023). Case-controls or cohort studies examining associations between participants with/without PAE or a FASD diagnosis and the domains of physical size, dysmorphology, functional neurodevelopment and/or brain structure/neurology were included. Excluded studies were non-empirical, sample size < 10, PAE determined via biological markers only, or no suitable comparison group. Summary data were extracted and associations between outcomes and standardised levels of PAE or FASD diagnosis determined using random-effects meta-analyses. Certainty of the evidence was assessed using GRADE.

Results Of the 306 included studies, 106 reported physical size, 43 dysmorphology, 195 functional neurodevelopment and 110 structural/neurological outcomes, with 292 different outcomes examined. There was a dose–response relationship between PAE and head circumference, as well as measures of physical size, particularly at birth. There was also an association between higher PAE levels and characteristic sentinel facial dysmorphology, as well as many of the current functional neurodevelopmental outcomes considered during diagnosis. However, data were often lacking across the full range of exposures. There was a lack of evidence from studies examining PAE to support inclusion of non-sentinel dysmorphic features, social cognition, speech-sound impairments, neurological conditions, seizures, sensory processing or structural brain abnormalities (via clinical MRI) in diagnostic criteria. GRADE ratings ranged from very low to moderate certainty of evidence.

Conclusions This comprehensive review provides guidance on which components are most useful to consider in the diagnostic criteria for FASD. It also highlights numerous gaps in the available evidence. Future well-designed

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pregnancy cohort studies should specifically focus on dose–response relationships between PAE and dysmorphology, neurodevelopment and brain structure/neurological outcomes.

Systematic review registration PROSPERO: CRD42021230522.

Keywords Fetal alcohol spectrum disorder, FASD, Birth weight, Prenatal alcohol exposure, Head circumference, Functional neurodevelopment, Diagnostic criteria, Facial features, Dysmorphology

Background

Prenatal alcohol exposure (PAE) is common in over 76 countries [1] and can lead to various adverse outcomes in pregnancy and childhood. Fetal alcohol spectrum disorder (FASD) is the leading cause of non-genetic developmental disability in many countries, affecting an estimated 7.7 per 1000 individuals [1]. Consequently, FASD is a serious public health issue, associated with significant costs for the individual, family and society [2]. FASD is under-diagnosed globally, in part owing to the current lack of a unified diagnostic approach [3]. Due to the complex and heterogeneous nature of FASD, over ten different diagnostic criteria are currently employed internationally [4]. This lack of standardised diagnostic criteria contributes to variations in the identification of FASD cases, making it difficult to establish accurate prevalence and impact. Establishing uniform diagnostic criteria is crucial for improving FASD identification and facilitating appropriate services for those affected.

The key clinical components in diagnostic criteria are physical size (i.e. birth weight, birth length, postnatal weight and/or postnatal height); dysmorphology (i.e. characteristic facial features [small palpebral fissures, smooth philtrum and thin vermilion], other dysmorphic features and birth defects); functional neurodevelopmental outcomes (e.g. general intelligence, memory, attention, executive function); structural neurodevelopmental outcomes (e.g. head circumference, structural brain abnormalities); and neurological outcomes (e.g. seizures of unknown origin, cerebral palsy, hearing and vision impairment) [5–10]. Whilst there have been previous systematic reviews on isolated diagnostic features (e.g. executive function, motor skills, birth weight) [11–13], none have provided a comprehensive summary to inform evidence-based decisions regarding diagnostic criteria. We systematically reviewed and synthesised the existing evidence examining the association of PAE with diagnostic outcomes to provide an evidence base for the improvement of diagnostic criteria, in the context of revising the Australian Guide for the Diagnosis of FASD [8].

Methods

We used Cochrane Systematic Review methodology to conduct this systematic review and meta-analysis and followed the 2020 PRISMA guideline for reporting [14]. This review was pre-registered with PROSPERO (reference: CRD42021230522).

Search strategy and selection criteria

Criteria for study inclusion were case–control and cohort studies with summary estimates examining the association between PAE (exposure studies) or FASD (diagnosed studies) and one or more outcomes related to physical size, dysmorphology, functional neurodevelopment or structural/neurological (see full population, exposure, comparator and outcome (PECO) components and detailed outcomes list in Additional file 1: Tables S1 and S2) [6, 15–24]. Articles were excluded if they were preclinical studies, the wrong publication type (letters, editorials, conference abstracts, higher degree dissertations, reviews of commentaries), sample size < 10 participants, PAE measured only using biological markers and no appropriate comparison group: individuals with no/minimal exposure (for exposure studies) or typically developing controls (for diagnosed studies). Six electronic databases (CINAHL, the Cochrane Library, EMBASE, PsychINFO, PubMed and Web of Science Core Collection) were searched (LA) from inception until January 30, 2021, and updated February 28, 2023. The search strategies included alcohol-related terms (and specifically those focussed on alcohol exposure during pregnancy) combined with terms related to the diagnostic criteria for FASD. Details of search strategies applied to each database are provided in Additional file 1: Table S3. Manual screening of reference lists of retrieved full-text publications and previous relevant systematic reviews was performed to identify additional relevant publications. Retrieved references were imported into an EndNote library and duplicate records removed. Remaining references were uploaded to Covidence (www.covidence.org) for screening against the inclusion and exclusion criteria. Title and abstracts were independently screened for eligibility by two reviewers (NR, LA). Full-text publications

of the remaining references were then retrieved and independently assessed by two reviewers (NR, LA). Discrepancies were resolved via discussion and consensus with a third reviewer (NH). A list of studies excluded at the full text level is provided (Additional file 1: Table S4).

Standardisation of prenatal alcohol exposure (PAE) levels across studies

For exposure studies, PAE levels were standardised and classified into six categories: light, moderate, heavy, very heavy, any (dichotomised as yes/no), and confirmed/unquantifiable (level not reliably collected but generally reported to be heavy or very heavy). Light PAE was defined as 1–20 g alcohol/ week (equivalent to 2 standard drinks in Australia), as per O'Leary et al. [25]. This study described different patterns of alcohol use during pregnancy and defined low exposure in terms of both dose per week (never more than 2 drinks per occasion) and maximum weekly amount (up to 7 drinks in a week). Most papers did not provide both dose and weekly amount so we chose ≤ 20 g/week to ensure that exposure could never be more than 2 drinks per occasion (i.e. no possibility of a 'binge' exposure, defined as 4 drinks per occasion). The definition for heavy PAE (> 100 g/week) was based on the Australian National Health and Medical Research Council (NHMRC) Guidelines that recommend no more than 10 standard drinks per week (equivalent to 100 g alcohol), with > 10 standard drinks/week defined as 'risky drinking' [26]. Therefore, moderate PAE was between the light and heavy levels of exposure (21–100 g/week). Very heavy PAE was defined by doubling the minimum level for heavy exposure (i.e. > 200 g alcohol/week).

In instances where PAE group mean alcohol level was not reported in the study, the PAE category definitions reported in the study methods were used to quantify and classify PAE level using procedures described by Patra et al. [13]. When a range of alcohol intake level was given, the midpoint of the range was used (e.g. 10–20 g per week = 15 g per week). In cases where no upper boundary was provided for the highest category of PAE (e.g. 40 + g per week), three-quarters of the length of the immediate previous category range was added to the lower boundary to estimate the amount per week. Where consumption was reported in drinks and not in grams, the grams of pure alcohol per drink (if defined in each article) was used. If the amount of alcohol per standard drink was not defined, conversion was based on geographical location: for Canada 13.6 g, USA 14 g, UK 8 g and for both New Zealand and Australia 10 g pure alcohol per standard drink (see https://iard.org/science-resources/detail/drinking-guidelines-general-population/ for definitions of standard drinks). For all other countries without any clear specifications, 12 g pure alcohol was used per standard drink. Where consumption was reported over some other timeframe (e.g. per day or per month), this was converted to weeks. Where multiple study PAE categories were classified into the same exposure level defined in this review, the higher PAE category from the study was used in the analyses. Example calculations for standardising PAE levels are provided in Additional file 1: Table S5.

Standardisation of diagnostic categories

For diagnosed studies, four categories were used to group diagnoses: FASD, FAS (fetal alcohol syndrome), pFAS (partial fetal alcohol syndrome) and ARND/other (alcohol-related neurodevelopmental disorder). FASD was used when a study grouped all individuals with an FASD diagnostic outcome together. FAS included diagnoses of FAS, FASD with three sentinel facial features, syndromal, and where FAS and pFAS were grouped together but FAS participants were reported to be in the majority. When a study had a pFAS/FAS group and pFAS participants were the majority, or if participant numbers were not reported, the study was classified in the pFAS category. ARND/other included static encephalopathy/alcohol exposed (SE/AE), neurobehavioural disorder/alcohol exposed (ND/AE), and heavily exposed non-syndromal. If a study reported multiple ARND/other groups, SE/AE was used in favour of ND/AE and ARND was used in favour of heavily exposed.

Data extraction and analysis

Data extraction was performed by multiple reviewers (LA, NH, NR, CV) and checked by a second reviewer (LA, JL, NH, NR or CV). When multiple studies reported the same population/cohort and outcome, the study with the largest sample size was included. Exceptions were studies where participant groups were reported with greater specificity (e.g. multiple PAE levels or FASD diagnostic subgroups).

Meta-analyses were conducted using Review Manager 5·4 software (RevMan desktop, Cochrane, London, UK) to investigate effects of PAE or FASD diagnoses on outcomes. Effect estimates were pooled across studies (when ≥ 2 studies) using a random effects model with study weightings adjusted using the generic inverse-variance method. For binary outcomes, odds ratios (OR) or frequency data were used. For continuous data, means/standard deviations or mean differences were used. Where available, adjusted estimates were prioritised. Separate meta-analyses were conducted for each PAE level or diagnostic category where available. Associations between diagnostic outcomes and

FASD diagnoses are often interdependent due to inclusion of these features in the diagnostic criteria. Unlike exposure studies, which classify participants based on the level of PAE and then determine whether particular features are present, diagnostic studies base their inclusion of participants on a diagnosis based on the presence or absence of these features. We included these diagnostic studies for a complete analysis of all available evidence, particularly where there may be gaps in available exposure data. However, presentation of exposure study data were prioritised as these studies are the most informative in understanding associations between PAE and diagnostic features. Subgroup analyses were used where possible to examine the effect of risk of bias (low versus moderate–high risk of bias), or adjustment for confounders (adjusted versus unadjusted data), on pooled effect estimates. Subgroup analysis investigating timing of PAE during pregnancy was not possible due to lack of available data or inconsistent reporting of exposure timing. Most studies did not stratify their effect estimates by sex so we could not include a sex-based analysis. Separate meta-analyses were conducted based on data availability for different measures and instruments across age groups.

Risk of bias assessment of included studies

Risk of bias assessment was performed independently by three reviewers (LA, NH, CV) and checked by a third reviewer (NR) using a modified version of the RTI Item Bank for Assessing Risk of Bias and Confounding for Observational Studies of Interventions or Exposures [27]. Ten items were included assessing selection bias, detection bias, performance bias, attrition bias and confounding. Risk of bias was assessed at the outcome level. Therefore, where relevant, studies that reported multiple outcomes were assessed for risk of bias multiple times for the different outcomes and analyses (e.g. raw data and regression analyses). Overall risk of bias was rated as low, moderate, serious or critical:

- Studies were rated as *low risk of bias* if there were no concerns across all areas of the assessment.
- Studies were rated as *moderate risk of bias* if they had some minor methodological concerns, but no major methodological concerns.
- Studies were rated as *serious risk of bias* if they had one or more major methodological flaws or five or more areas where enough information was not provided.
- Studies were rated as *critical risk of bias* and excluded from analysis if they did not measure and even partially consider confounding variables.

GRADE assessment of the certainty of evidence

Certainty of evidence for each meta-analytic finding was made using the GRADE (Grading of Recommendations Assessment, Development, and Evaluation) approach [28, 29]. The GRADE approach is international best practice when considering evidence for clinical practice guideline development and provides an assessment of degree of certainty that the observed effects from each meta-analytic finding are true and reliable. The following domains were assessed, and a judgement made as to whether there were *serious* or *not serious* concerns:

- Risk of bias: A *serious* rating was provided when > 50% of the studies included in a meta-analysis had a moderate or high risk of bias.
- Inconsistency: A *serious* rating was provided when the overall heterogeneity chi-square statistic was significant ($P < 0.05$) and I^2 was > 50%. Where the outcome included only a single study, inconsistency was rated as *not serious*.
- Indirectness: A *serious* rating was provided when > 50% of studies included samples not likely to be comparative to an Australian population (e.g. studies were undertaken in South Africa, Ukraine or Chile).
- Imprecision: A *serious* rating was provided when the overall 95% confidence intervals (CI) for the meta-analysis crossed the line of no effect, were wide or when optimal sample size criteria were not met (i.e. for dichotomous data, ≥ 300 abnormal events or sample size ≥ 2000; for continuous data, required sample size of ≥ 400). A *very serious* rating was provided when all three *serious* criteria above were present. 95% CIs were considered 'wide' based on clinically meaningful differences between the lower and upper confidence intervals for each of the outcomes (following discussion with clinical members of the Guidelines Development Group).
- Other considerations: Publication bias was assessed with funnel plots generated for outcomes with 10 or more studies. Publication bias was rated as *strongly suspected* in the presence of an asymmetrical funnel plot.

GRADE Profiler (GRADEPro, McMaster University and Evidence Prime, 2022) was used to complete the assessments and generate the overall GRADE certainty rating for each meta-analytic outcome. The GRADE approach for prognostic factors was used whereby ratings started out as high certainty and were rated down due to the GRADE domains mentioned above. Overall GRADE ratings for each meta-analysis were reported in summary figures as: ⊕◯◯◯ very low certainty,

⊕⊕◯◯ low certainty, ⊕⊕⊕◯ moderate certainty, and ⊕⊕⊕⊕ high certainty. Note that GRADE domain and overall certainty of evidence ratings are made at the meta-analytic level and not at an individual study level. Ratings did not exclude individual studies or meta-analyses from inclusion in the review findings. Further details of the GRADE ratings for each meta-analysis can be found here: https://zenodo.org/records/10649619.

Data presentation

Information from meta-analyses (i.e. number of studies, number of participants, pooled effect estimates, 95% CIs and I^2%), and the GRADE ratings, were combined in composite figures created using GraphPad Prism 9 (GraphPad Software, Boston MA, USA). This approach facilitated visual comparison across PAE levels or FASD diagnoses. Due to the large amount of data available for physical size outcomes, we did not include outcomes reported in only single studies in the summary figures (reported instead here: https://doi.org/https://doi.org/10.5281/zenodo.10783892). For functional neurodevelopmental outcomes, due to the diversity of assessments and outcome types, the more clinically relevant outcomes were included in summary figures (including outcomes with single studies) and results for other outcomes are provided elsewhere (see https://doi.org/https://doi.org/10.5281/zenodo.10783892). For dysmorphology and structural/neurological outcomes, aside from head circumference, single studies were included in summary figures due to the limited data available.

Results

Study selection and characteristics

The initial search identified 18,422 records (Fig. 1). After removal of duplicates and screening at the title/abstract and full text level, 306 studies were included: 106 reporting physical size outcomes, 43 dysmorphology, 195 functional neurodevelopmental outcomes, and 110 structural/neurological measures (many studies reported more than one outcome).

Characteristics for all included studies are presented in Additional file 1: Table S6 [22, 25, 30–332]. Studies originated from 23 countries, including 136 (44%) from USA, 44 (14%) from Europe/UK (including Ukraine), 42 (14%) from South Africa, 41 (13%) from Canada, 20 (7%) from Australia/New Zealand, 5 (2%) from Japan and 7 (2%) from other countries (e.g. India, Ethiopia). Eleven studies (4%) were multinational. Of the 306 included studies, 216 (71%) were case-controls (99 nested case-controls).

Risk of bias assessments

Of the 505 risk of bias assessments completed at the outcome level, 360 (72%) studies with outcomes analysed were rated as having serious risk of bias, 107 (21%) as moderate, and 33 (7%) as low risk of bias. Two studies were rated as critical risk of bias and were excluded from further analysis (see Additional file 1: Table S4 for details). A summary of the risk of bias assessments at the level of each diagnostic component is presented in Table 1.

Meta-analyses and GRADE ratings summaries

Descriptive and summary statistics for the > 900 meta-analyses conducted are presented in Table 2 and Additional file 1: Table S7. Meta-analyses averaged ~ 2–4 studies per analysis but for many, there was only one study available at that specific exposure level/diagnosis for a specific diagnostic outcome. For most diagnostic components, heterogeneity across studies within each meta-analysis was generally high (where this could be assessed), excluding dysmorphology. There was no evidence of publication bias, assessed where possible using funnel plots. Further details of GRADE assessments, meta-analyses and funnel plots are provided here: https://doi.org/https://doi.org/10.5281/zenodo.10783892.

Studies reporting regression analyses were narratively synthesised and included for completeness (Additional file 1: Table S8) [34, 35, 45–47, 55, 61, 63, 67–69, 74, 75, 80, 90, 106, 107, 109–111, 123, 128, 129, 132, 133, 141, 146–150, 175, 176, 192, 217, 271, 275, 284, 292, 293, 302, 310, 312, 313, 321]. Whilst these analyses included adjustment for potential confounders, they showed a similar pattern of association between various diagnostic outcomes and PAE levels to the meta-analyses, with limited evidence for effects at light to moderate levels of PAE but sustained impacts at higher levels of exposure.

Physical size outcomes

There was an inverse dose–response relationship with the level of PAE (Fig. 2, PAE and birth measures; all other outcomes in Additional file 1: Fig. S2). Certainty of the evidence was higher for the odds of small for gestational age (SGA) and low birthweight (LBW), compared to raw measures of weight and length/height. More comprehensive and higher certainty evidence was available for birth outcomes (Fig. 2), compared to postnatal outcomes (Additional file 1: Fig. S2). FASD diagnoses were also associated with reduced physical size, particularly FAS and pFAS (Additional file 1: Fig. S1 and S3).

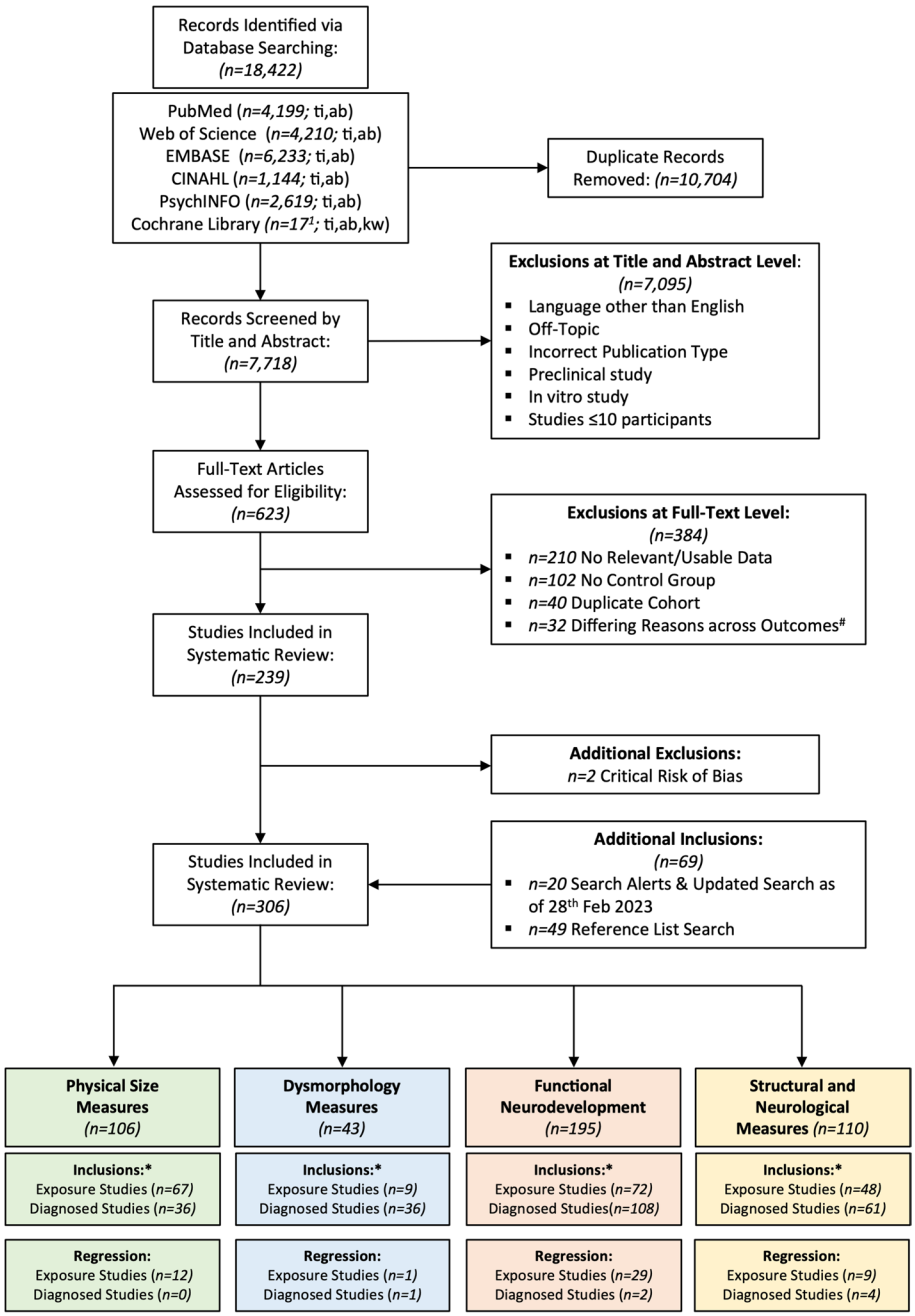

Fig. 1 PRISMA flow chart. # studies where exclusion reasons differed across outcomes. *Studies included in both exposure and diagnosed groups (*n* = 3). Note: Some studies reported on more than one diagnostic domain. ti = title, ab = abstract, and kw = keyword

Dysmorphology outcomes

There were increased odds of the characteristic facial features of smooth philtrum (OR 3·3–6·2) or short palpebral fissures (OR 8·3–9·2) with moderate, very heavy and confirmed/unquantifiable PAE levels and thin vermilion with confirmed/unquantifiable PAE (OR 5·3, 95% CI 3·6–7·7; Additional file 1: Fig. S4A). Few exposure studies examined other facial and non-facial dysmorphic features (Additional file 1: Fig. S5), with substantial variability across outcomes. A small number of exposure studies reported composite dysmorphology scores, with an association with moderate and very heavy PAE (Additional file 1: Fig. S7). As expected, diagnostic groups with dysmorphic features showed a positive association with dysmorphology outcomes (Additional file 1: Figs. S4, S6, S7). Certainty of evidence across dysmorphology outcomes was very low to low.

Table 1 Risk of bias assessment ratings summary

Diagnostic component	Low	Moderate	Serious	Critical[a]
Physical size	9 (7%)	32 (27%)	76 (64%)	2 (2%)
Dysmorphology	-	6 (12%)	41 (84%)	2 (4%)
Functional neurodevelopment	19 (9%)	42 (20%)	153 (70%)	1 (1%)
Structural/neurological	5 (4%)	27 (22%)	90 (73%)	2 (2%)

Data shown as the number of studies per outcome, *n* (%). Note that some studies reported on more than one diagnostic outcome

[a] The two studies with critical risk of bias were excluded from further analysis. See Additional file 1: Table S4 for more details

Functional neurodevelopment outcomes

For functional neurodevelopmental outcomes, few exposure studies examined light or moderate PAE levels. Caregiver-reported *attention problems* were associated with very heavy and confirmed/unquantifiable PAE when reported as a standardised mean difference, with very low to low certainty ratings (Fig. 3A). Results were more variable when reported as an OR (Additional file 1: Fig. S8). All diagnostic groups demonstrated increased attention problems on both caregiver and teacher reports, although there was wide variability for the pFAS group,

Table 2 Descriptive statistics of meta-analyses

Diagnostic component	Number of meta-analyses	Studies per meta-analysis			Meta-analyses with high heterogeneity[a]
		Mean	Mode	Range	
Physical size	104	3.9	1	1–14	48.4%
Dysmorphology	58	2.1	1	1–12	1.9%
Functional neuro-development	663	1.8	1	1–16	45.9%
Structural/ neurological	118	2.4	2	1–9	54.5%

[a] Defined as $I^2 > 75\%$, calculated using a random-effects model where the meta-analysis included ≥ 5 studies. Number of meta-analyses per diagnostic outcome where this could be calculated: physical size, $n = 31$; dysmorphology, $n = 16$; functional neurodevelopment, $n = 37$; structural/neurological, $n = 11$. Note: Publication bias was also assessed using funnel plots where the meta-analysis included ≥ 10 studies. Number of meta-analyses per diagnostic outcome where this could be calculated: physical size, $n = 10$; dysmorphology, $n = 3$; functional neurodevelopment, $n = 3$. There was no evidence of publication bias. All analyses conducted using Revman 5.4

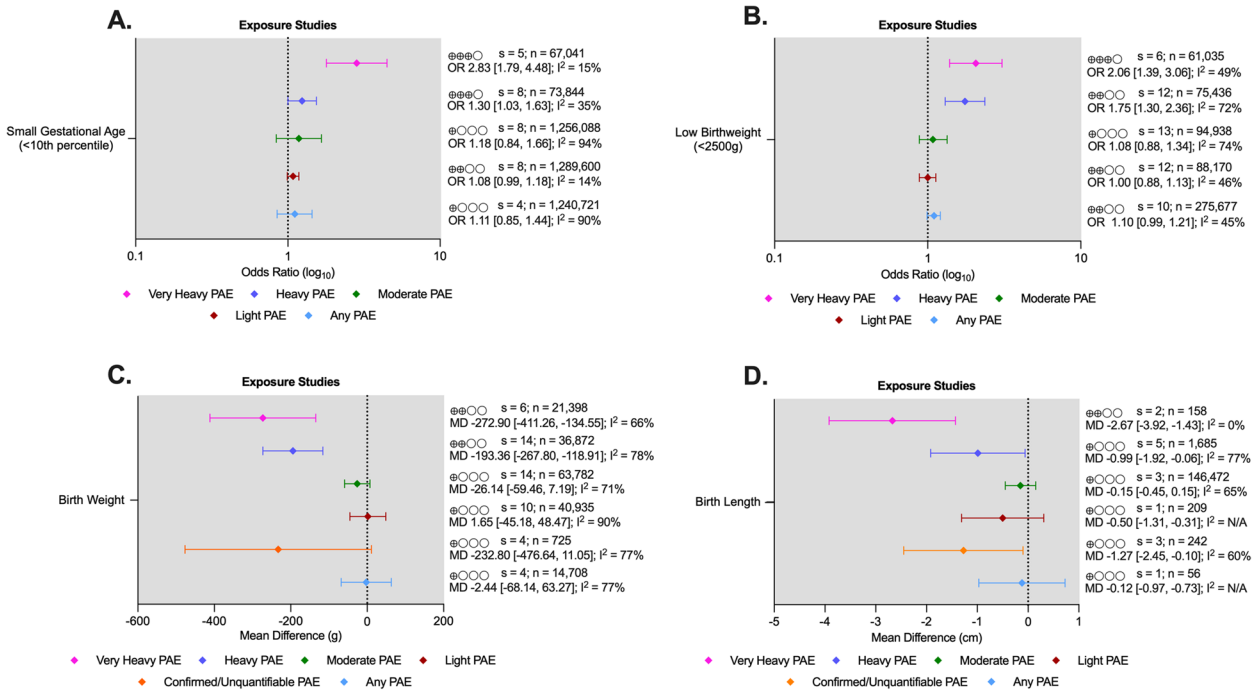

Fig. 2 Association between prenatal alcohol exposure (PAE) and size at birth. **A** Small for gestational age. **B** Low birth weight.**C** Birth weight. **D** Birth length. GRADE Ratings: ⊕◯◯◯=very low certainty; ⊕⊕◯◯=low certainty; ⊕⊕⊕◯=moderate certainty. s=number of studies included in each meta-analysis. *n*=overall number of participants included in each meta-analysis. OR=odds ratio [95% confidence interval (CI)]. MD=mean difference [95% CI]. I^2=indicator of heterogeneity

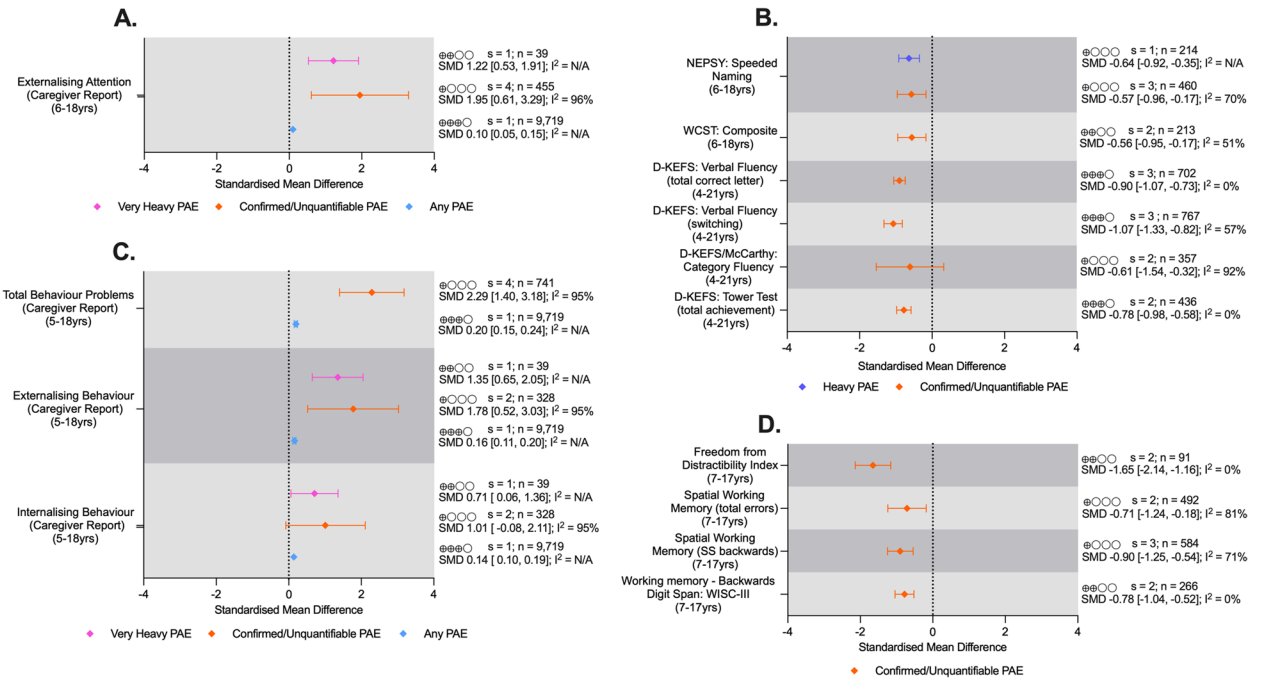

Fig. 3 Association between prenatal alcohol exposure (PAE) and neurodevelopmental measures related to behaviour and executive function. **A** Externalising attention. **B** Measures of executive function. **C** Caregiver-reported measures of behaviour. **D** Measures of working memory. Test details provided in Additional file 1: p 90. Lower scores indicate better performance for (**A**) and (**C**); higher scores indicate better performance for (**B**) and (**D**). GRADE Ratings: ⊕◯◯◯=very low certainty; ⊕⊕◯◯=low certainty; ⊕⊕⊕◯=moderate certainty; ⊕⊕⊕⊕=high certainty. s=number of studies included in each meta-analysis. *n*=overall number of participants included in each meta-analysis. OR=odds ratio [95% confidence interval (CI)]. MD=mean difference [95% CI]. I^2=indicator of heterogeneity. NEPSY=NEuroPSYchological. WCST=Wisconsin Card Sorting Test. D-KEFS=Delis-Kaplan Executive Function System. WISC-III=Wechsler Intelligence Scale for Children

and overall, very low to moderate certainty of the evidence (Additional file 1: Fig. S8). For *executive function* (EF), poorer performance on most direct measures was associated with heavy and confirmed/unquantifiable PAE, with very low to moderate evidence certainty (Fig. 3B; Additional file 1: Fig. S9). Caregiver and teacher reports of EF were only associated with poorer performance in the confirmed/unquantifiable PAE group, and no associations were found with other PAE levels (Additional file 1: Fig. S10). Most diagnosed groups demonstrated poorer performance on all measures of EF, with very low to low certainty evidence (Additional file 1: Figs. S9, S10). *Total and externalising behaviour problems,* as assessed using the Child Behaviour Checklist (CBCL), were increased in the very heavy and confirmed/unquantifiable exposure groups, whilst results were more variable for *internalising behaviour problems*, all with very low to low certainty ratings (Fig. 3C). Scores for CBCL sub-scales such as aggression, anxiety/depression and rule-breaking were consistently increased in very heavy and confirmed/unquantifiable PAE groups (Additional file 1: Fig. S11). Odds of scoring in the clinical range for behavioural problems using other measures were increased at moderate PAE levels, but not all heavier levels of exposure were examined (Additional file 1: Fig. S11). FASD diagnoses were consistently associated with increased behaviour problems across most measures (Additional file 1: Figs. S12, S13). The evidence for these associations was generally of moderate certainty. Similar poorer performance was found for measures of *working memory,* although only studies with confirmed/unquantifiable PAE were available (Fig. 3D). Most diagnosed groups demonstrated poorer performance compared to controls on measures of working memory, with very low to low evidence certainty (Additional file 1: Fig. S14).

For *language,* confirmed/unquantifiable PAE showed significant associations with poorer language abilities, with very low to low certainty of the evidence, but there were no studies with heavy or very heavy PAE (Fig. 4A, Additional file 1: S15). Diagnosed groups demonstrated poorer language performance compared to controls, except for studies using the NEPSY (a developmental neuropsychological test), where scores were variable (Additional file 1: Fig. S16). Certainty of the evidence for diagnosed studies was very low to low. For *academic achievement*, there was an inverse dose–response relationship with PAE levels, with significant associations found only with very heavy PAE (Fig. 4B), and very low

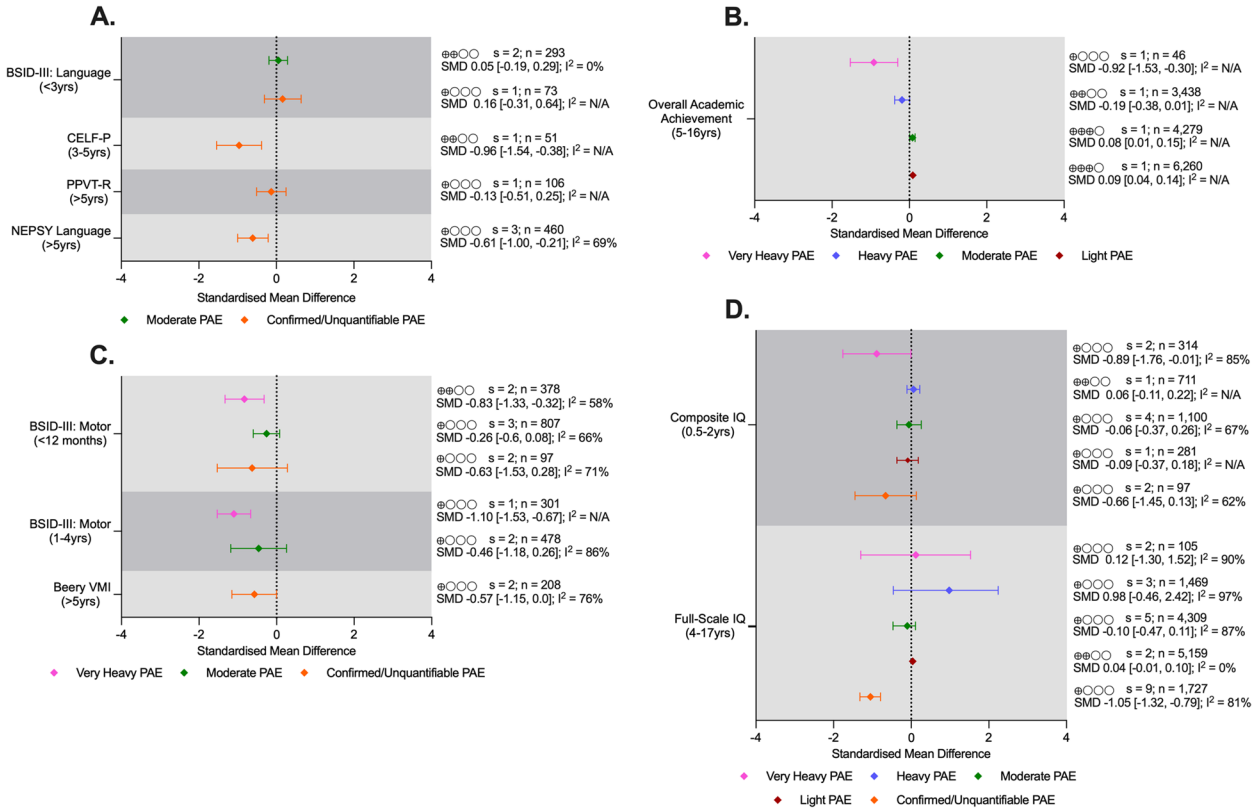

Fig. 4 Association between prenatal alcohol exposure (PAE) and neurodevelopmental measures of motor function and academic performance. **A** Language abilities. **B** Overall academic achievement. **C** Motor function. **D** General intellectual abilities. Test details are provided in Additional file 1: (p 90). Higher scores indicate better performance for all measures. GRADE Ratings: ⊕◯◯◯=very low certainty; ⊕⊕◯◯=low certainty; ⊕⊕⊕◯=moderate certainty; ⊕⊕⊕⊕=high certainty. s=number of studies included in each meta-analysis. *n*=overall number of participants included in each meta-analysis. OR=odds ratio [95% confidence interval (CI)]. MD=mean difference [95% CI]. I^2=indicator of heterogeneity. BSID-III=Bayley's Scales of Infant Development. CELF-P=Clinical Fundamentals of Language Preschool. PPVT-R=Peabody Picture Vocabulary Test-Revised. NEPSY=NEuroPSYchological. VMI=Visual Motor Integration

to moderate certainty of evidence. There were also associations between FASD diagnoses and academic ability, with those diagnosed with pFAS showing the greatest impairments (Additional file 1: Fig. S17). For *motor skills*, only very heavy PAE was associated with significant reductions in motor abilities, although this was only in infants and pre-school aged children (Fig. 4C). Available evidence at other levels showed no associations with moderate and confirmed/unquantifiable PAE levels. Diagnosed groups generally demonstrated poorer motor abilities compared to controls, although studies reporting data as ORs showed more variable results (Additional file 1: Fig. S16). For *intellectual abilities*, lower composite IQ (< 2 years) or full-scale IQ (4–17 years) scores were found at very heavy or confirmed/unquantifiable PAE levels, although there was often imprecision around the effect estimates (i.e. wide 95% CIs), and very low to low certainty evidence (Fig. 4D). Component IQ measures (e.g. verbal and performance) were similarly variable and imprecise (Additional file 1: Fig. S18). All FASD diagnoses were associated with lower intellectual ability scores (Additional file 1: Fig. S19). Few studies assessed *memory*, *adaptive behaviour*, *social functioning*, *sensory processing* or *soft neurological signs* (Additional file 1: Figs. S20-23), often examining only one exposure level or diagnostic group. Associations were generally seen for heavy and confirmed/unquantifiable PAE. Certainty of evidence ranged from very low to moderate.

Structural/neurological outcomes

Head circumference at birth showed a dose–response with PAE level (Fig. 5A), but only where data were expressed as the mean difference of absolute measurements, and not percentiles (Fig. 5B). There were fewer studies examining head circumference postnatally and results were variable. A dose–response was evident for odds of a small head circumference (< 10th percentile; Fig. 5C). Evidence certainty ranged from very low to moderate. Diagnoses of FAS and pFAS were generally associated with lower head circumference at birth and

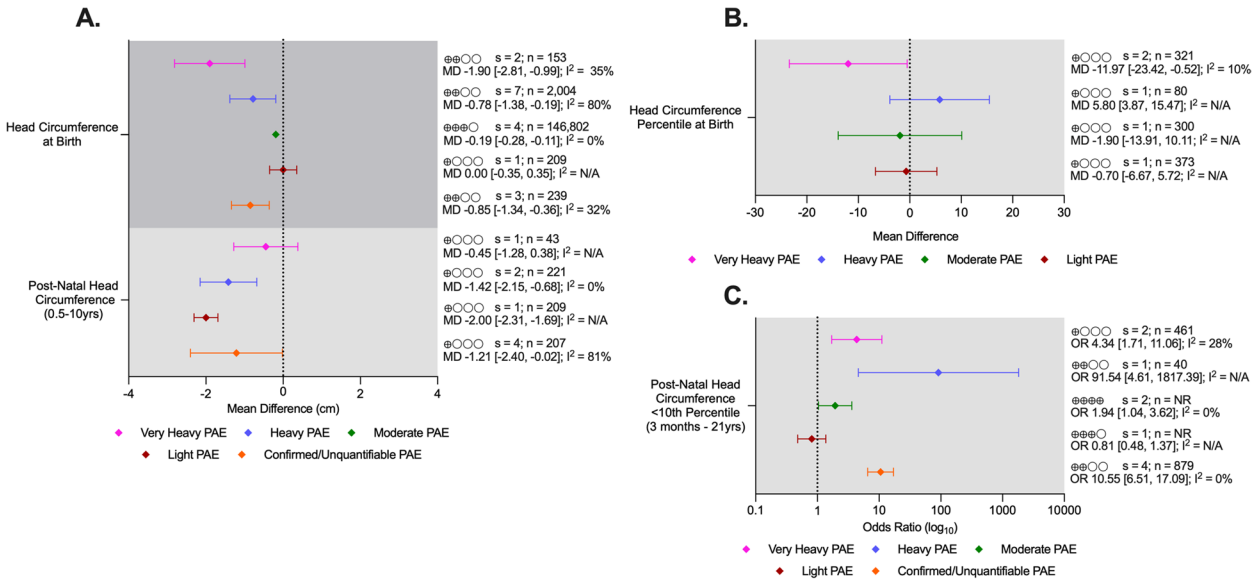

Fig. 5 Association between prenatal alcohol exposure (PAE) and head circumference. **A** Head circumference (cm) at birth and post-natally. **B** Head circumference at birth as a percentile. **C** Odds ratio of small post-natal head circumference (<10th percentile).GRADE Ratings: ⊕◯◯◯=very low certainty; ⊕⊕◯◯=low certainty; ⊕⊕⊕◯=moderate certainty; ⊕⊕⊕⊕=high certainty. s=number of studies included in each meta-analysis. *n*=overall number of participants included in each meta-analysis. OR=odds ratio [95% confidence interval (CI)]. MD=mean difference [95% CI]. I^2=indicator of heterogeneity

postnatally, with very low to low evidence certainty (Additional file 1: Fig. S24). There was only one *clinical MRI* exposure study, with no clearly increased odds of clinically relevant incidental MRI findings with confirmed/unquantifiable PAE (Additional file 1: Fig. S25). There were only two diagnosed studies, providing some evidence for FAS/pFAS diagnosis being associated with clinically relevant incidental MRI findings (Additional file 1: Fig. S25). Whilst not currently included as part of the clinical assessment process, outcomes for quantitative MRI findings have been summarised to provide information regarding potential changes in brain structure in relation to PAE or FASD diagnosis (Additional file 1: Figs. S26, S27). Several *other neurological outcomes* (e.g. hearing and vision impairments, seizures, cerebral palsy) often included in diagnostic criteria were examined. However, there were mostly single studies examining each exposure level or diagnostic group relative to controls (Additional file 1: Fig. S28). Evidence ranged from very low to low certainty, with moderate certainty for the one exposure study assessing seizures.

Discussion

Diagnostic criteria are a set of signs and symptoms used in clinical practice to support the accurate identification of a health condition. These criteria must capture the heterogeneity of a condition, including the key clinical features. However, developing diagnostic criteria for conditions without 'gold standard' diagnostic tests or biomarkers is a significant challenge that requires both rigor and flexibility [3]. This systematic review comprehensively summarises the available evidence on the association between PAE and key diagnostic components of FASD. Perhaps not surprisingly, higher PAE levels were associated with the largest and most consistent effects, including smaller physical size, increased rates of dysmorphic features, and poorer functional neurodevelopmental outcomes across a range of domains. There was also a dose–response relationship between PAE and head circumference, with higher PAE levels associated with smaller head circumference at birth. However, evidence was limited and inconsistent for clinically available structural MRI and neurological outcomes. Additionally, we examined the relationship of key diagnostic components across the spectrum of FASD diagnoses. A significant limitation of studies using diagnosed individuals is the interdependency between outcomes and diagnosis (i.e. individuals were identified based on diagnostic features). Consequently, exposure studies provide the highest quality evidence to inform decision making about associations between PAE and diagnostic features, and thus which features should be prioritised for diagnostic purposes. Diagnostic studies that prospectively assess and compare currently available tools and norms and

collect and report all diagnostic information, rather than only reporting to one set of diagnostic criteria, could still make important contributions to improved assessment and diagnostic practices for FASD.

A key difference among current diagnostic criteria internationally is whether a specific PAE threshold is required for diagnosis. The results of the current review provide no evidence to support the inclusion of light alcohol exposure (defined as up to 20 g per week) for diagnostic purposes, and mixed evidence for moderate alcohol exposure (21–100 g per week). There was however consistent evidence for higher levels of exposure (>100 g per week). These findings are generally consistent with previous research [333–336]. PAE is a risk factor for FASD, but not all exposures will result in a diagnosis of FASD. Minimum exposure thresholds increase certainty that the observed impairments can be attributable to PAE, aiding in more accurate diagnosis of FASD. The current review did not aim to determine a 'safe' PAE level, but the findings provide empirical evidence regarding the level of PAE at which clinically meaningful impairments are likely to be observed for individuals and thus support clinicians in diagnostic decision making [334]. Whilst an increasing number of FASD diagnostic criteria include a PAE minimum threshold [5, 337, 338], this is for diagnostic purposes. Public health recommendations in many countries advise that people should not consume alcohol when planning a pregnancy to prevent the wide range of possible adverse outcomes other than FASD that may result from PAE.

Included studies were inconsistent or lacked information on timing or consumption patterns (e.g. 'binge,' prior to pregnancy recognition), preventing sub-group analyses. This is a key limitation of the available evidence, as previous research suggests that the timing of exposure can be related to the type and extent of possible adverse outcomes [25, 339]. Recent findings also highlight the importance of considering alcohol use frequency and amount per occasion separately in characterising PAE levels and adverse outcomes [334]. We also acknowledge that there are limitations with self-reports of alcohol use during pregnancy; however, self-reports are currently the best available method to assess PAE [340].

Whilst adjusted effect estimates were included where possible, many studies lacked consideration of other pre- and post-natal risk factors, which could exacerbate or ameliorate the impacts of PAE. For example, there is evidence that improving nutrition during pregnancy [341, 342] and early childhood [343] can positively influence the severity of deficits following PAE. Thus, care is required when applying PAE thresholds in clinical practice at an individual level due to the wide range of determinants of PAE impacts. Diagnosis of FASD is a complex process that is best undertaken using a holistic interprofessional approach [344].

The current review revealed a lack of evidence for the utility of neurological or clinically available MRI assessments to determine effects of PAE. Whilst there is variability in how structural brain abnormalities, seizures of unknown origin, hearing and vision impairment, and other neurological conditions are considered, current FASD diagnostic criteria include many of these outcomes. Due to lack of data, there is insufficient evidence for inclusion of these currently. However, we suggest that brain abnormalities and neurological conditions be considered in the assessment process to inform individual support planning and future research to better understand potential associations with PAE.

This comprehensive systematic review examined the components included in international diagnostic criteria for FASD across a range of PAE levels and FASD diagnostic groups. A strength of this review is the standardisation of PAE categories, enabling synthesis and comparison of evidence across studies at equivalent PAE levels, rather than comparing studies based on their author-defined levels. Another strength was the application of GRADE, which provides an indication of the degree of certainty in the review findings, supporting evidence-based decision-making related to FASD diagnostic criteria. Overall, GRADE ratings ranged from very low to moderate certainty. Certainty in findings was impacted by the risk of bias in many included studies, which were often rated as serious. The most common reasons for risk ratings included inadequate control for confounding variables, lack of reliable PAE measurement across participant groups and/or insufficient details being reported across most risk of bias assessment areas.

Another key limitation of the available evidence is that few studies stratified results by sex, and thus potential sex differences could not be examined. Additionally, there was wide variability in the outcomes examined, whether due to the type of assessment used, or differences in the reporting of outcomes (i.e. ORs and mean differences), which limited the ability for larger meta-analyses. This was particularly evident in the functional neurodevelopmental area, where an extensive range of measures and differences in reporting were found. Use of a standardised approach to reporting research outcomes would strengthen future research efforts. Further, aside from the components of physical size and head circumference at birth, there were limited data available across PAE levels for the same outcome; often only confirmed/unquantifiable levels were available (i.e. studies reporting suspected heavy or very heavy exposure, but specific levels were not reliably collected). This prevents examination of potential dose–response relationships across all

diagnostic components. Future well-designed pregnancy cohort studies are required, focussed on dysmorphology, functional neurodevelopmental and structural/neurological outcomes with stratified exposure data collected across well-defined PAE levels.

Conclusions

Overall, evidence from this systematic review provides direction regarding which components should currently be considered for inclusion in diagnostic criteria for FASD. The results have also highlighted key research gaps that can be targeted to improve understanding of the potential associations between PAE and diagnostic outcomes. Importantly, a collaborative international approach, driven by a goal of continuous quality improvement is required to advance assessment and diagnostic practices for FASD, with the united goal of improving quality of life for individuals with FASD and their families.

Abbreviations

ARND	Alcohol-related neurodevelopmental disorder
BSID	Bayley Scales of Infant Development
CBCL	Child behaviour checklist
CELF-P	Clinical Fundamentals of Language—Preschool
CI	Confidence interval
D-KEFS	Delis-Kaplan Executive Function System
EF	Executive function
FAS	Fetal alcohol syndrome
FASD	Fetal alcohol spectrum disorder
GRADE	Grading of Recommendations Assessment, Development, and Evaluation
IQ	Intelligence quotient
LBW	Low birth weight
MD	Mean difference
MRI	Magnetic resonance imaging
ND/AE	Neurobehavioural disorder/alcohol exposed
NEPSY	NEuroPSYchological
NHMRC	National Health and Medical Research Council (Australia)
OR	Odds ratio
PAE	Prenatal alcohol exposure
PECO	Population, Exposure, Comparator and Outcome
pFAS	Partial fetal alcohol syndrome
PPVT-R	Peabody Picture Vocabulary Test-Revised
PRISMA	Preferred Reporting Guidelines for Systematic reviews and Meta-analyses
SE/AE	Static encephalopathy/alcohol exposed
SGA	Small for gestational age
VMI	Visual Motor Integration
WCST	Wisconsin Card Sorting Test
WISC	Wechsler Intelligence Scale for Children

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12916-024-03656-w.

Additional file 1: Table S1. Population, Exposure, Comparator and Outcome (PECO) Components. Table S2. Definitions of included outcomes. Table S3. Search strategies for each database. Table S4. List of studies excluded at the full text level. Table S5. Example calculations for standardising prenatal alcohol exposure levels across studies. Table S6. Study characteristics. Table S7. Summary of exposure study meta-analysis results. Table S8. Narrative summary of studies reporting regression data. Fig. S1. Association between fetal alcohol spectrum disorder (FASD) diagnoses and birth measures. Fig. S2. Association between prenatal alcohol exposure (PAE) and various measures of postnatal size. Fig. S3. Association between fetal alcohol spectrum disorder (FASD) diagnoses and various measures of postnatal size. Fig. S4. Association between prenatal alcohol exposure (PAE) or fetal alcohol spectrum disorder (FASD) diagnosis and sentinel facial features. Fig. S5. Association between prenatal alcohol exposure (PAE) and minor dysmorphology features. Fig. S6. Association between fetal alcohol spectrum disorder (FASD) diagnoses and minor dysmorphology features. Fig. S7. Association between prenatal alcohol exposure (PAE) or fetal alcohol spectrum disorder (FASD) diagnosis and dysmorphology scores. Fig. S8. Association between prenatal alcohol exposure (PAE) or fetal alcohol spectrum disorder (FASD) diagnosis and measures of attention. Fig. S9. Association between fetal alcohol spectrum disorder (FASD) diagnosis and direct measures of executive functioning. Fig. S10. Association between prenatal alcohol exposure (PAE), fetal alcohol spectrum disorder (FASD) diagnosis and parent and teacher measures of every day executive function abilities. Fig. S11. Association between prenatal alcohol exposure (PAE) and caregiver and teacher reported sub-scale measures of behaviour. Fig. S12. Association between fetal alcohol spectrum disorder (FASD) diagnosis and composite measures of behaviour. Fig. S13. Association between fetal alcohol spectrum disorder (FASD) diagnosis and sub-domain measures of behaviour. Fig. S14. Association between fetal alcohol spectrum disorder (FASD) diagnosis and measures of working memory. Fig. S15. Association between prenatal alcohol exposure (PAE) and language or motor measures. Fig. S16. Association between fetal alcohol spectrum disorder (FASD) diagnosis and language or motor measures. Fig. S17. Association between prenatal alcohol exposure (PAE) or fetal alcohol spectrum disorder (FASD) diagnosis and academic achievement. Fig. S18. Association between prenatal alcohol exposure (PAE) and general intellectual abilities (sub-scales). Fig. S19. Association between fetal alcohol spectrum disorder (FASD) diagnoses and general intellectual abilities. Fig. S20. Association between prenatal alcohol exposure (PAE) or fetal alcohol spectrum disorder (FASD) diagnosis and measures of memory. Fig. S21. Association between prenatal alcohol exposure (PAE) and adaptive and social behaviour. Fig. S22. Association between fetal alcohol spectrum disorder (FASD) diagnosis and adaptive and social behaviour. Fig. S23. Association between prenatal alcohol exposure (PAE) or fetal alcohol spectrum disorder (FASD) diagnosis and sensory processing/neurological signs. Fig. S24. Association between fetal alcohol spectrum disorder (FASD) diagnosis and head circumference. Fig. S25. Association between prenatal alcohol exposure (PAE) or fetal alcohol spectrum disorder (FASD) diagnosis and structural brain abnormalities from clinical magnetic resonance imaging (MRI). Fig. S26. Association between prenatal alcohol exposure (PAE) and quantitative magnetic resonance imaging (MRI). Fig. S27. Association between Fetal Alcohol Spectrum Disorder (FASD) and quantitative magnetic resonance imaging (MRI) measures. Fig. S28. Association between prenatal alcohol exposure (PAE) or fetal alcohol syndrome (FAS) diagnosis and other neurological outcomes.

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Authors' contributions
LA, NH, ZM, PM, KM, and NR contributed to study conceptualisation and the development of the review protocol. LA did the literature search. LA, NH, and NR performed the title/abstract and full-text screening. LA, NH, CV, and NR did risk of bias assessments and data extraction. LA, NH, CV, and JL conducted the data analysis and created the summary figures. LA and NR wrote the first draft of the manuscript. All authors edited or substantively reviewed the publication. All authors have had full access to all data in the study and had final responsibility for the decision to submit for publication. All authors read and approved the final manuscript.

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Availability of data and materials
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Declarations

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