Review title

The association between prenatal exposure to environmental tobacco smoke and childhood obesity: a systematic review protocol

Reviewers

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Review question/objective

To determine if women’s exposure to environmental tobacco smoke (ETS) during pregnancy increases the risk of obesity in their children.

Background

Childhood obesity has reached epidemic proportions across the world. This is alarming because obesity is a major health risk and is linked to an increased incidence of hypertension, metabolic disorders and other chronic cardiovascular conditions at very young ages.¹, ² One of the contributing factors is prenatal exposure to tobacco smoke.³, ⁴ Maternal smoking during pregnancy increases the risk of obesity in exposed children.³, ⁵⁻⁶ While there is a body of literature linking maternal smoking during pregnancy to the increased risk of childhood obesity, there are few studies that have explored the association of exposure to ETS or secondhand smoke during pregnancy to the body mass index (BMI) of children. The proposed systematic review will analyze data from identified quantitative studies on the subject and explore the strength of the relationship between prenatal exposure to ETS and childhood obesity in the offspring.

Childhood obesity

Prevalence of obesity among children has risen at an alarming rate over the last few decades.¹⁰⁻¹⁴ According to the World Health Organization (WHO) 24% of children in European countries are overweight.¹² The United Kingdom reported nearly 30% of children were overweight in 2012.¹³ Similar findings were reported by Australia and China.¹¹, ¹⁴ In the USA the prevalence has tripled since 1980 and at present, approximately 17% of children between the ages of two and 19 are obese.¹⁵ Obesity is measured by the BMI and classifications are based on the BMI for age and gender. BMI is expressed as weight in kilograms divided by height in meters squared (kg/m²).¹⁵ In children, BMI is plotted on growth charts and classifications are based on ranges. Children with BMI values at or above the 95th percentile of the United States Centers for Disease Control and Prevention (CDC) sex-specific BMI growth charts are categorized as obese, while those between the 85th and 95th percentiles are considered at risk for obesity.¹⁰, ¹⁵, ¹⁶ The National Health Service (NHS) in the UK uses similar measures.¹³ Although the CDC uses BMI as a measure of obesity, its use in children, especially those under two years, is arguable. Some researchers use Ponderal Index (kg/m³) as a predictor of childhood obesity; however literature suggests that BMI is a better predictor after age two.¹⁷

Exposure to environmental tobacco smoke

Even though the rates of smoking have decreased in developed countries and ETS levels have also decreased, 40% of children across the globe live with a smoker.¹⁸ In China nearly 50% of the population smokes, while the rates in the UK, Australia and Canada are around 25%.¹⁸ The scope and magnitude of exposure can be gauged by the USA as an example. In the USA 19.3%
of the population still smokes and the cotinine level (a biological marker of exposure to tobacco smoke) among non-smokers is still at 40%. Cotinine levels are higher in children overall, among black non-smokers and people living below the federal poverty level. The CDC estimates nearly 54% of children aged three to 11 are exposed to ETS and 19% of children (three to 11 years of age) live with a smoker. With one in seven low-income children obese, there are also significant racial and ethnic disparities in the rates of obesity among minority children in the USA. Furthermore, the rates of smoking are higher among minority and low-income populations, and exposure to ETS is higher among minority children.

**Association between maternal smoking during pregnancy and weight and growth in the offspring**

The etiology of childhood obesity is multifactorial and complex. Maternal smoking has been linked to an increased prevalence of childhood obesity. The risk is dose-dependent and increases as the number of cigarettes smoked increases. Birth weight was decreased in babies born to smokers and there was an increased prevalence in childhood obesity in these children but there were gender differences with boys at a greater risk of obesity in childhood. In a longitudinal cohort study Howe et al (2012) found that maternal smoking was associated with shorter height of babies, slightly more in girls. The association with dose dependency was stronger for girls than boys.

Babies born to smokers grew faster during their first year of life but thereafter growth slowed until age 10; girls born to smokers were 1.2 cm shorter when compared to those born to non-smokers. Moreover, the association between maternal smoking and weight gain in children is not limited to the prenatal period but remains significant during early childhood.

**Secondhand smoke or ETS?**

The WHO and the US Environmental Protection Agency use both terms to define involuntary smoking of a mixture of exhaled mainstream smoke and sidestream smoke emitted from a burning cigarette or pipe, cigar, etc., and diluted with ambient air. For the purpose of this review we will use ETS.

**Association between exposure to environmental tobacco smoke and childhood obesity**

Although there is a body of literature linking maternal smoking during pregnancy to an increased prevalence of childhood obesity, very little is known about the association of exposure to ETS and childhood obesity. The exposure to ETS begins in utero. Exposure can be the direct effect of mothers who smoke or indirect if they are exposed to ETS. The number of pregnant women exposed to ETS is not known but 40% of the population who did not smoke had cotinine levels suggesting exposure.

Braun et al (2010) found that 50% of their sample of 292 mother-child dyads (self-reported non-smoker mothers) had cotinine levels indicating exposure to ETS when only 15% reported exposure to ETS. However, those with self-reported exposure to ETS had higher cotinine levels. Women who had a history of prenatal exposure to ETS or cotinine levels indicative of exposure to ETS had children with high BMIs. Birth weight was lower, height was shorter but weight gain was higher during the first few years of childhood.

Clearly there is a need to further explore the role of exposure to ETS and the increasing rate of childhood obesity. To date there have been only a few studies that have explored this association. A systematic review of the available studies will add to the body of literature and provide an opportunity to calculate an effect size and measure the strength of this relationship.

**Inclusion criteria**

**Types of participants**

This review will consider studies that include non-smoking pregnant women of any age who reported exposure to ETS, and subsequently gave birth and their children, aged up to eighteen years. Comparator groups will comprised parturient women who were non-smokers and did not report exposure to ETS who subsequently gave birth, and their children.

**Types of intervention(s)/phenomena of interest**

Studies that explore exposure to ETS during pregnancy will be included. This can be measured by

1. Self report of exposure e.g. partner smoked
2. Cotinine levels (as measured by serum cotinine levels).
Types of outcomes
Weight, height and BMI and/or Ponderal Index of the children.

Types of studies
This review will consider epidemiological study designs including before and after studies, prospective and retrospective cohort studies, case control studies and analytical cross sectional studies.
This review will also consider descriptive epidemiological study designs including case series, individual case reports and descriptive cross sectional studies.

Search strategy
The search strategy incorporates both published and unpublished studies. A three-step search strategy will be utilised in this review. An initial limited search of MEDLINE and CINAHL will be undertaken followed by an analysis of the text words contained in the title and abstract, and of the index terms used to describe the article. A second search using all identified keywords and index terms will then be undertaken across all included databases. Thirdly, the reference list of all identified reports and articles will be searched for additional studies. Only studies published in the English language will be considered for inclusion in this review. No search range will be set to allow greater sensitivity.
The databases to be searched include:
MEDLINE, CINAHL, EMBASE, ScienceDirect, Proquest, NHS Research Register, Wiley InterScience, Controlled Clinical Trials, Clinical Trials.gov, Google Scholar, Centers for Disease Control website and WorldCat
The search for grey literature will include:
ProQuest Dissertations and Theses Database, the directory of grey literature via the New York Academy of Medicine website and Mednar
Initial keywords to be used will be:
Second hand smoke
Environmental tobacco smoke
Passive smoking
Paternal smoking
Childhood obesity
Childhood body mass index
Ponderal Index
Weight, height or length
Cotinine level

Assessment of methodological quality
Papers selected for retrieval will be assessed by two independent reviewers for methodological validity prior to inclusion in the review using standardized critical appraisal instruments from the Joanna Briggs Institute Meta Analysis of Statistics Assessment and Review Instrument (JBI-MAStARI) (Appendix I). Any disagreements that arise between the reviewers will be resolved through discussion, or with a third reviewer. Reviewers will try to contact authors of primary studies for missing information or to clarify unclear data.

Data collection
Data will be extracted from papers included in the review using the standardized data extraction tool from JBI-MAStARI (Appendix II). The data extracted will include specific details about the
interventions, populations, study methods and outcomes of significance to the review question and specific objectives.

**Data synthesis**

Quantitative data will, where possible, be pooled in statistical meta-analysis using JBI-MAStARI. All results will be subject to double data entry. Effect sizes expressed as odds ratio (for categorical data) and weighted mean differences (for continuous data) and their 95% confidence intervals will be calculated for analysis. Heterogeneity will be assessed statistically using the standard Chi-square and also be explored using subgroup analyses based on the different study designs included in this review. Where statistical pooling is not possible the findings will be presented in narrative form including tables and figures to aid in data presentation where appropriate.

**Conflicts of interest**

No conflict of interest anticipated.

**References**


Appendix I: Appraisal instruments

MAStARI appraisal instrument

JBI Critical Appraisal Checklist for Randomised Control / Pseudo-randomised Trial

<table>
<thead>
<tr>
<th>Question</th>
<th>Yes</th>
<th>No</th>
<th>Unclear</th>
<th>Not Applicable</th>
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<tbody>
<tr>
<td>1. Was the assignment to treatment groups truly random?</td>
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<td>2. Were participants blinded to treatment allocation?</td>
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<td>3. Was allocation to treatment groups concealed from the allocator?</td>
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<td>4. Were the outcomes of people who withdrew described and included in the analysis?</td>
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<td>5. Were those assessing outcomes blind to the treatment allocation?</td>
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<td>6. Were the control and treatment groups comparable at entry?</td>
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<td>7. Were groups treated identically other than for the named interventions</td>
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<td>8. Were outcomes measured in the same way for all groups?</td>
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<td>9. Were outcomes measured in a reliable way?</td>
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<td>10. Was appropriate statistical analysis used?</td>
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Overall appraisal: Include □ Exclude □ Seek further info. □

Comments (Including reason for exclusion)

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________________________________________________________________________
# JBI Critical Appraisal Checklist for Descriptive / Case Series

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<tr>
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<th>Yes</th>
<th>No</th>
<th>Unclear</th>
<th>Not Applicable</th>
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<tbody>
<tr>
<td>1.</td>
<td>Was study based on a random or pseudo-random sample?</td>
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<td>2.</td>
<td>Were the criteria for inclusion in the sample clearly defined?</td>
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<td>3.</td>
<td>Were confounding factors identified and strategies to deal with them stated?</td>
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<td>4.</td>
<td>Were outcomes assessed using objective criteria?</td>
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<td>5.</td>
<td>If comparisons are being made, was there sufficient descriptions of the groups?</td>
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<td>6.</td>
<td>Was follow up carried out over a sufficient time period?</td>
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<td>7.</td>
<td>Were the outcomes of people who withdrew described and included in the analysis?</td>
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<td>9.</td>
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**Overall appraisal:**  
- Include □  
- Exclude □  
- Seek further info □

**Comments (Including reason for exclusion):**

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### JBI Critical Appraisal Checklist for Comparable Cohort/Case Control

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<td>1. Is sample representative of patients in the population as a whole?</td>
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<td>2. Are the patients at a similar point in the course of their condition/illness?</td>
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<td>3. Has bias been minimised in relation to selection of cases and of controls?</td>
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<td>4. Are confounding factors identified and strategies to deal with them stated?</td>
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Overall appraisal: Include □ Exclude □ Seek further info. □

Comments (Including reason for exclusion)

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Appendix II: Data extraction instruments

MAStARI data extraction instrument

**JBI Data Extraction Form for Experimental / Observational Studies**

Reviewer: ___________________________ Date: ___________________________

Author: ___________________________ Year: ___________________________

Journal: ___________________________ Record Number: ___________________________

**Study Method**

- RCT: □
- Quasi-RCT: □
- Longitudinal: □
- Retrospective: □
- Observational: □
- Other: □

**Participants**

Setting: ___________________________________________

Population: _________________________________________

**Sample size**

Group A: ___________________________ Group B: ___________________________

**Interventions**

Intervention A: _______________________________________

Intervention B: _______________________________________

Authors Conclusions:

________________________________________________________________________

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________________________________________________________________________

Reviewers Conclusions:

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________________________________________________________________________
Study results

Dichotomous data

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<th>Outcome</th>
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<th>Intervention ( ) number / total number</th>
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Continuous data

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<th>Outcome</th>
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