Prenatal Tobacco and Cannabis Exposure on Child Obesity Outcomes: A Systematic Review

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Key Terms

PTE = prenatal tobacco exposure	PCE = prenatal cannabis exposure
PTCE = prenatal tobacco-cannabis co-exposure	OWO = overweight/obese

Current Literature

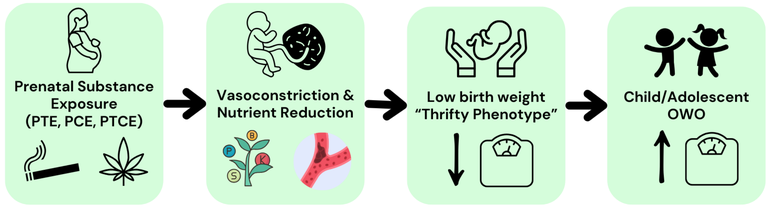
Tobacco & cannabis are the most widely used substances during pregnancy[1]
- ~12.6 to 14.9% of infants in the US born exposed to tobacco[2]
- ~5.5% of infants in the US born exposed to cannabis[1]

PTE and PTCE frequently co-occur[1, 5, 6]
- ***Up to 70% of adult cannabis users report past month tobacco use***[5]

PTE > ***LOW*** birth weight (LBW), OWO[3]
PCE > some evidence of LBW[4]
- Gap in the literature on effects of cannabis, and in combination with PTE

Both tobacco & cannabis metabolites freely cross the fetal-placental barrier[7, 8]

Figure 1. Proposed Mechanism



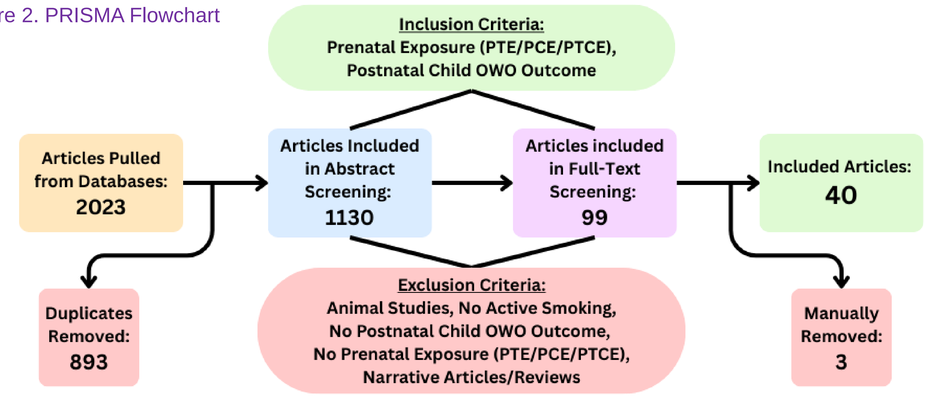
Developmental Origins of Health and Disease[9]
- Prenatal environment = high fetal stress, relative resource scarcity
- Predictive adaptive [fetal] response[10] – response to an environmental cue meant to improve fitness ***LATER*** in development
- "Thrifty phenotype" [11] = metabolic function altered based on early environmental cues

Methodological Flaws

- Reliance on single item self-report
- No biochemical verification
- Demographic differences in control versus exposure groups
- Effects of PTE/PTCE in the context of other substance exposures (e.g., alcohol, cocaine, etc.)
- Varying age and measure of OWO child outcomes
- Lack of control for postnatal exposure

Article Selection

Figure 2. PRISMA Flowchart



Preliminary Results

Figure 3. Found Significant Results

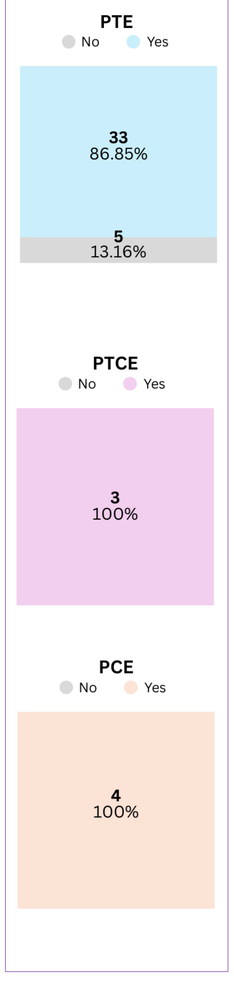


Figure 4. Dose-Response Effects

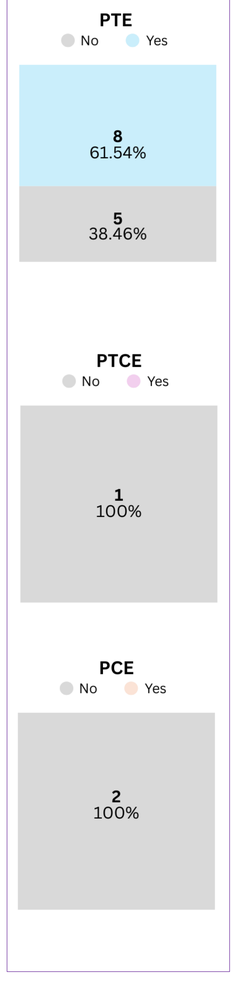


Figure 5. Timing-Specific Effects

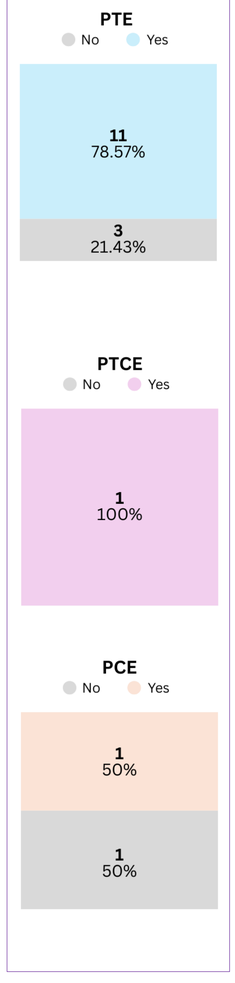


Figure 6. Examined Sex-Specific Effects

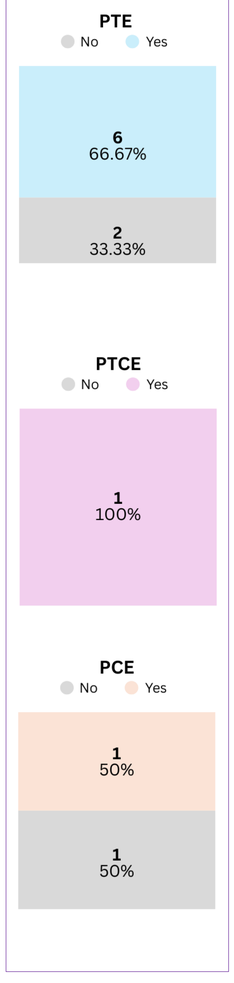
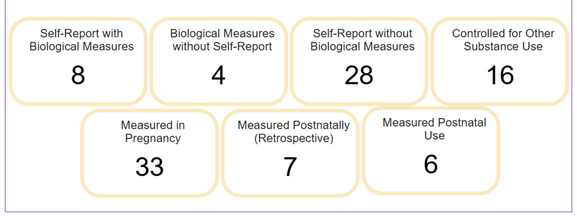


Figure 7. **Measurement of Prenatal Substance Use**



Preliminary Conclusions

Examples of Mixed Sex-Specific Effects:

- **Neonatal Period**: Among girls only, maternal continued smoking during pregnancy was associated with lower neonatal phospholipid metabolite concentrations, which may increase susceptibility to obesity[12]
- **Early Childhood (36 months)**: Males showed greater weight than females in heavy smoking group[13]
  - Effect disappeared when controlling for maternal prenatal marijuana use
- **Middle Childhood (9-12 years):** Both PTE and PCE groups showed steeper increases in BMI from birth to mid-childhood [14]
  - Among PTCE children, girls demonstrated steeper increases than boys
- **Adolescence (16/17 years)**: The association of smoking with thicker retinal walls was driven by the girls, whereas no difference was seen in the boys[15]
  - Overall effects included smaller stature and increased obesity-related indices

Examples of Mixed Dose-Response Effects:

- The odds ratio for higher child BMI was 2.42 times greater for mothers who smoked 11+ cigarettes/day in the third trimester compared to mothers who didn't smoke[16]
- Reducing the number of cigarettes during pregnancy did not lower the risks of childhood overweight[17]
- No dose-response relationship between PTE or PTCE and BMI trajectories from birth to middle childhood[14]
- PTE dose–response association displayed the highest effect estimates of obesity-related metabolites in children whose mothers continued smoking ≥5 cigarettes per day[12]
- PCE (nonsmokers, light, and heavy) dose-response analyses found were no differences for weight or height[18]
- PCE dose–response analyses found highest effect estimates for child BMI in children whose mothers used daily cannabis[12]

References & Lab