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Perspective

Caffeine consumption during pregnancy and birth malformations

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Abstract

Caffeine is a habit-forming substance consumed daily by the majority of pregnant women. Accordingly, it is important that women receive sound evidence-based advice about potential caffeine-related harm. This narrative review examines evidence of association between maternal caffeine consumption and negative pregnancy outcomes, and assesses whether current health advice concerning maternal caffeine consumption is soundly based. The articles reported results for one or more of six major categories of negative pregnancy outcomes: miscarriage, stillbirth, low birth weight and/or small for gestational age, preterm birth, childhood acute leukaemia, and childhood overweight and obesity. The three remaining meta-analyses were also unanimous in reporting absence of a reliable association between maternal caffeine consumption and preterm birth. No meta-analyses were identified for childhood overweight and obesity, although four of five original observational studies reported significant associations linking maternal caffeine consumption to that outcome category.

Keywords: Caffeine, Pregnancy, Infants, Anthropometry.

INTRODUCTION

Maternal exposures such as excessive caffeine consumption during pregnancy, are likely associated to low birth weight (LBW) and preterm birth. This issue has been the subject of several epidemiological studies. However, the results of these studies are contradictory, which is partly explained by the heterogeneity in study design by the measurement of caffeine intake, and by the identification of confounders. Knowledge of caffeine pharmacology suggests high biological plausibility for potential fetal harm from maternal consumption. When consumed during pregnancy, caffeine readily crosses the placenta, exposing the fetus to concentrations of the drug similar to systemic levels in the mother. The extent of diffusion of caffeine from maternal to fetal circulation is indicated by the presence of caffeine in fetal hair, with concentrations in the hair of newborns being found to correlate well with maternal caffeine consumption during the third trimester (Dumas et al., 1982).

The symptoms of nausea and vomiting that frequently accompany pregnancy-related changes in hormonal milieu and associated reduced rate of caffeine clearance help to explain the observation that women often spontaneously reduce their intake of caffeine when pregnant. Reduced intake means that plasma caffeine concentrations tend to be maintained at levels comparable to the pre-pregnant state instead of reaching the appreciably higher levels that would occur if intake remained unchanged (Fulgoni et al., 2015).

Furthermore, habitual caffeine consumption leads to physical dependence, indicated by behavioural, physiological and subjective withdrawal effects (caffeine withdrawal syndrome) in response to even brief abstinence. Sleepiness, lethargy and headache are common symptoms, which may occur following cessation of habitual intake of as little as per day and less. With reference to standard criteria, principally those of the Diagnostic and Statistical Manual of Mental Disorders (DSM-V), caffeine has been labelled a 'drug of abuse'. Indeed, newborn infants of caffeine-consuming mothers have been reported to experience caffeine withdrawal symptoms including disturbed sleep,

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vomiting, increased frequency of irregular heartbeat and respiration, and increased fine tremors similar to neonatal narcotic abstinence syndrome (Kline et al., 2016).

Rational health advice about caffeine exposure during pregnancy depends crucially on the extent to which the evidence of association is thought to be indicative of causation. Therefore, despite extensive consistency in the reporting of increased risk of harm associated with maternal caffeine consumption, it is reasonable to ask: Do methodological shortcomings limit inferences of causation within the large body of evidence of association? In reply, apart from specific potential limitations examined below, it may be said that the likelihood of causation (rather than mere association) is supported by the many reports of observed dose response relationships between the amount of caffeine consumed during pregnancy and the risk of negative pregnancy outcomes. Implication of causation is also rendered more salient by reports of no threshold of consumption below which associations are absent. Overall, then, likely causation is supported by a compelling body of evidence, both theoretical (ie, biological plausibility) and empirical, including a strong consensus among observational studies and particularly meta-analyses, dose–response relationships and reported absence of threshold effects (Pearlman et al., 1989).

The substantial majority finding from observational studies and meta-analyses is that maternal caffeine consumption is reliably associated with major negative pregnancy outcomes. Reported findings were robust to threats from potential confounding and misclassification. Among both observational studies and meta-analyses, there were frequent reports of significant dose response associations suggestive of causation, and frequent reports of no threshold of consumption below which associations were absent (Sasaki et al., 2017).

CONCLUSION

It is generally accepted that chronic exposure to chemicals during pregnancy is cause for concern. When the chemical of interest is caffeine, a near universally consumed habit-forming substance of no nutritional value, the need for caution is compelling. There is substantial cumulative evidence of an association between maternal caffeine consumption and diverse negative pregnancy outcomes. Indicative of causation, observational studies and meta-analyses alike have reported dose–response associations with some studies also finding no threshold of consumption below which associations with negative outcomes are absent. Crucially, the evidence has proved to be decidedly robust to threats from potential confounding and misclassification.

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