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BPA Study Report Card

The criteria identified in this Report Card were established in the *Environment International* article, "A proposal for assessing study quality: Biomonitoring, Environmental Epidemiology, and Short-lived Chemicals (BEES-C) instrument." The BEES-C instrument is designed to evaluate the quality of research studies that incorporate biomonitoring data on short-lived chemicals. More detailed explanation on the various criteria and the ranking system are included in the publication, which is available online.

Study Meets Criteria	Study Criteria Unknown or not applicable	Study fails criteria

Study: Bisphenol A and Adiposity in an Inner-City Birth Cohort
Authors: Lori A. Hoepner, Robin M. Whyatt, Elizabeth M. Widen, Abeer Hassoun, Sharon E. Oberfield, Noel T. Mueller, Diurka Diaz, Antonia M. Calafat, Frederica P. Perera, and Andrew G. Rundle
Journal: ENVIRONMENTAL HEALTH PERSPECTIVES

CRITERIA	SCORE	COMMENTS
Biological relevance: exposure biomarker (level of quantitative relationship between biomarker and external exposure, internal dose, or target dose)		Urinary BPA concentrations were measured by spot analysis at prenatal, age 3, and age 5. No information was collected on possible BPA exposures (diet, medical devices). Single spot analyses have low precision and accuracy.
Biological relevance: effect biomarker (level of specificity of biomarker to reported effect)		Biological relevance unknown. Authors acknowledge that body mass index (BMI) and fat mass index (FMI) may not be suitable measure of adiposity in prepubescent children. No biological mechanisms provided for a potential BPA-obesity association.
Specificity (one parent compound with one biomarker or multiple parent chemicals with varying effects)		Study measured urinary total BPA concentrations. Study also measured urinary concentrations of four DEHP metabolites.
Method sensitivity/detection limits (accuracy and precision of methods used to quantify the biomarker)		The urinary concentrations of the sum of free and conjugated BPA species (total BPA) were measured using online solid-phase extraction coupled to high-performance liquid chromatography–isotope dilution tandem mass spectrometry with peak focusing. The LOD and coefficients of variations were reported, but coefficients of variations were higher than expected for the low concentrations of BPA detected.
Known or documented stability of biomarker		The limit of detection was provided for BPA. In addition to study samples, low concentration and high concentration quality control materials prepared with spiked urine samples and reagent blanks were included.
Prevention of sample contamination		Study did not describe methods to prevent BPA contamination.
Method requirements (appropriateness and description of measurement method)		Methods described by citing a previous publication.
Matrix adjustments (appropriate reporting and weighting of differences in collection requirements and sample processes)		Study adjusted for urinary dilution using specific gravity. Only quantified total BPA, no measurement of free (biologically active) BPA or conjugated BPA.
Study design and execution: temporality (claim of causation supported by observation of the putative causal exposure preceding the outcome)		Study measured urine BPA at three time points in prenatal and early childhood. While the exposure did occur prior to outcome measurement, the study did not conduct a longitudinal analysis (*i.e.*, changes in exposure overtime and changes in outcome overtime).

Study design and execution: exposure variability and misclassification (sufficient number of samples)		Misclassification of urinary BPA concentrations likely substantial due to intrapersonal variation in dietary intake and spot urine samples.
Study rationale (specific design to evaluate hypothesis)		Previous evidence from cross-sectional studies on urine BPA and obesity was cited. Study employed a longitudinal cohort design.
Study participants (unbiased selection)		African American or Dominican women (N = 727) in third trimester recruited. Considerable loss-to-follow-up was observed (N = 498 at age 5; N = 511 at age 7). Study did not report whether there is significant differential loss to follow-up.
Data analysis (control of extraneous factors, distinction between causal and predictive)		Models with prenatal BPA adjusted for maternal pre-pregnancy BMI, race/ethnicity, prenatal ∑DEHP, and sex, birth weight, and gestational age of the child. Models with childhood BPA adjusted for maternal ln∑DEHP, race/ethnicity, dichotomous pre-pregnancy obesity, and child sex, birth weight, and gestational age. No total caloric intake (a critical confounder for BPA-obesity association) was adjusted for in the statistical analyses. Sensitivity analyses were conducted. Also, as mentioned previously, no longitudinal analysis was conducted.
Reporting (Study clearly states its aims and allows the reader to evaluate the number of tested hypotheses)		Study clearly stated aims and different hypotheses tested. However, study did not fully discuss the inconsistencies within results (only prenatal BPA associated with adiposity measures, not early childhood BPA), nor did study sufficiently discuss various limitations and their impact on the results.

Notes:

The study is a longitudinal cohort and obtained repeated measurements of exposure and outcome. However, the study did not use the more appropriate and informative statistical approach to evaluate a potential BPA-adiposity association.

Total caloric intake, a critical confounder associated with both BPA exposure and adiposity, was not adjusted for in the analyses.

Inconsistencies in results were not fully discussed by the authors. Prenatal BPA levels were much lower than early childhood BPA levels, yet only prenatal BPA was associated with adiposity measures.

Considerable misclassification in exposure is likely because of intrapersonal variation in diet and spot urine samples.