BPA linked to diminished childhood lung function in Bizzaro World

By ACSH Staff — October 8, 2014

A recent study examining the association between prenatal BPA (bisphenol-A) exposure and lung function has been making headlines, often with titles similar to BPA linked to asthma. However, the actual study is about as bad as they come. You can just as easily conclude anything about BPA exposure and lung function as you can who will win the All Star Game in 2019.

The study [1], published in JAMA Pediatrics, examined whether pre- and post-natal BPA exposure was associated with lung function in children at 4 and 5 years old. BPA exposure was measured through maternal urine samples collected at the 16th and 26th weeks of pregnancy, and child urine samples were collected annually. The lung function of the children was examined by two measures: parent-reported incidence of wheezing and forced expiratory volume (FEV). The study participants included almost 400 pregnant women and their babies.

The authors reported that child urinary BPA concentrations were not associated with FEV or wheeze at either age 4 or 5. The average maternal urinary BPA concentrations were statistically associated with a decrease in the percentage predicted FEV at four years old but not at five years old. These results may seem inconsistent, and the authors agree. We found that prenatal BPA exposure that occurred during early pregnancy was inconsistently associated with diminished lung function, the authors conclude. Because the study only examined statistical associations, the word inconsistently is very important however it was omitted from almost every headline covering this study which is akin to forgetting to include the word no before saying there was [no] significant linkage found.

As for the second measurement of lung function, parent-reported incidence of wheezing, the authors report that average maternal urinary BPA concentration was marginally associated with an increased odds of wheezing. However, the unacceptable p-value of 0.11 actually suggests that there was, in fact, no association. Also, wheezing incidence based on parental reporting can be a serious limitation, as this self-reported method of data collection can often be inaccurate or inconsistent.
Another limitation, which the authors also acknowledged, is that BPA concentration was weakly correlated at 16 and 26 weeks, and it is known to vary widely over time. Because urine BPA levels vary widely over short and long periods of time, spot samples may not adequately represent exposure even over short periods of time.

ACSH’s Dr. Gil Ross had this comment: This is truly one of the worst junk science studies ever published. And yet, it got published, in a JAMA journal, no less. This travesty just goes to show us all, yet again, the failure of peer review when faced up against a PC-faux-study, alleging to find some link between some targeted chemical and some adverse outcome, especially when the children are involved. To the objective observer, this bizarre conglomeration of datapoints does not even amount to a study; the suggested association was manufactured in the fevered brains of the co-authors, who should be embarrassed for submitting this as a scientific study. Even the low bar of statistical significance was bypassed on several occasions to suit the pre-ordained outcome. But none of this will deter the spin-meisters at JAMA and the green blogosphere, and the mainstream media, from reporting on a link between BPA and children’s respiratory dysfunction. Nothing such was shown in this article, and all associated with it should be ashamed.

ACSH’s Dr. Josh Bloom was less kind: I would be disappointed in a gerbil that published this. Yes it is that bad.

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