8.78; 95% CI, -16.78 to -0.79). In contrast, maternal TSH, FT4, and TT4 levels were not significantly associated with child FSIQ scores. Maternal primary hypothyroidism did not significantly mediate the relationship between maternal water fluoride concentration and child FSIQ (p natural indirect effect= .35).
Conclusions: Fluoride in drinking water may increase the risk of hypothyroidism in pregnancy. Thyroid dysfunction in pregnancy may be one mechanism underlying developmental neurotoxicity of fluoride.

Categories: Drug/Toxin-Related Disorders (including Alcohol)
Keyword 1: neurotoxicity
Keyword 2: endocrine disorders
Keyword 3: intelligence
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4 Urinary Fluoride Levels and Metal Co-Exposures Among Pregnant Women in Los Angeles, California

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Objective: Approximately 73% of the United States (US) population on public water systems receives fluoridated water for tooth decay prevention. In Los Angeles (LA) County, 89% of cities are at least partially fluoridated. Drinking water is the primary source of fluoride exposure in the US. Studies conducted in Mexico and Canada suggest that prenatal fluoride exposure, at levels relevant to the US, may contribute to poorer neurodevelopment in offspring. However, data on biomarkers and patterns of fluoride exposure among US pregnant women are scarce. This study examined urinary fluoride levels according to sociodemographic factors and metal co-exposures among pregnant women in the US.
Participants and Methods: Participants were from the Maternal and Developmental Risks from Environmental and Social Stressors (MADRES) cohort based in Los Angeles, California. There were 293 and 490 women with urine fluoride measured during the first and third trimesters of pregnancy, respectively. An intra-class correlation coefficient examined consistency of specific gravity-adjusted maternal urinary fluoride (MUFsg) between trimesters. Kruskal-Wallis and Mann-Whitney U tests examined associations of MUFsg with sociodemographic variables. Spearman correlations examined associations of MUFsg with blood and urine metals within and between trimesters. A False Discovery Rate (FDR) correction accounted for multiple comparisons. The criterion for statistical significance was an alpha of 0.05.
Results: Participants were approximately 29 years old on average, and 80% were Hispanic or Latina. Median (IQR) MUFsg during trimesters one and three was 0.65 (0.5) mg/L and 0.8 (0.59) mg/L, respectively. MUFsg levels were moderately consistent between trimesters (N=292, ICC = 0.46, 95%CI: 0.32,0.57). Maternal age was positively associated with MUFsg during first ($\rho = 0.16$, $p = 0.006$) and third ($\rho = 0.18$, $p < 0.001$) trimesters. MUFsg differed by race/ethnicity during first and third trimesters (N = 293, H (3) = 7.99, $p = 0.046$; N = 486, H (3) = 25.31, $p < 0.001$, respectively). Specifically, MUFsg was higher for White, Non-Hispanic participants (first trimester Median (IQR) =1.03 (1.31) mg/L; third trimester Median (IQR) = 1.32 (1.24) mg/L) than for Hispanic participants in both trimesters (first trimester Median (IQR) =0.64 (0.48) mg/L; third trimester Median (IQR) = 0.76 (0.55) mg/L). Additionally, during trimester three, MUFsg was higher for White, Non-Hispanic participants than for Black Non-Hispanic participants (Median (IQR) = 0.82 (0.49) mg/L). MUFsg also differed by education during trimester one (N = 293, H (4) = 10.61, p = 0.031), and was higher for participants with some graduate training than for those with high school or some college/technical school education (ps = 0.03 and 0.04, respectively). After FDR correction, MUFsg was associated with blood lead (N =91, $\rho = 0.29$, $p = 0.024$) and urinary cadmium (N =279, $\rho = 0.19$, $p = 0.042$), copper (N=279, $\rho = 0.16$, $p = 0.042$), and tungsten (N=279, $\rho = 0.16$, $p = 0.049$) during trimester three.
Conclusions: Consistent with studies conducted in Canada and Mexico, MUFsg

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increased across pregnancy. Lower MUFsg among Hispanic and Non-Hispanic Black participants may reflect lower tap water consumption. Metal co-exposures are important to consider when examining potential neurodevelopmental impacts of fluoride.

Categories: Drug/Toxin-Related Disorders (including Alcohol)
Keyword 1: environmental pollutants / exposures
Keyword 2: ethnicity
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5 Translating developmental neurotoxicity for the public: A large, international, randomized-control trial investigating children's environmental health literacy

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Objective: Exposure to toxic chemicals during early brain development increases the risk of neurodevelopmental problems in children. Parents' and prospective parents' understanding of the impact of toxic chemicals on brain development and the efficacy of translation tools for children's environmental health literacy are poorly understood. We developed and validated a questionnaire, PRevention of Toxic chemicals in the Environment for Children Tool (PRoTECT) to assess knowledge of toxic chemicals and neurodevelopment, intentions to reduce exposures to toxic chemicals, and preferences for actions by government and industry to prevent neurodevelopmental disorders. Using PRoTECT, we surveyed people of child-bearing age across five countries (Canada, United States (US), United Kingdom (UK), India, and Australia) to identify general patterns of responses on this questionnaire by demographic characteristics, including country, age, gender, parental status, pregnancy status, and education. We also employed a randomized control design to examine the efficacy of a knowledge translation video to instill knowledge and prompt behavioral changes to reduce exposures to toxic chemicals immediately following its presentation and after a six-week follow-up period.
Participants and Methods: We recruited 15,594 participants, ages 18 to 45, via CloudResearch's Prime Panels between October-December 2021. After completing the PRoTECT survey, participants were randomly assigned to watch the video Little Things Matter: Impact of Toxic Chemicals on Brain Development (i.e., the experimental group) or to serve as the control group. Next, both groups answered a series of questions to assess their knowledge of toxic chemicals, their intentions to reduce exposures to toxic chemicals, and barriers to changing their behaviours. After six-weeks, we recontacted a subset (N=4,842) of participants to repeat PRoTECT and answer the same series of behavioural questions assessing whether they modified any of their behaviours to reduce exposure and why or why not.
Results: Most participants (i.e., 75-85%) agreed that toxic chemicals can impact brain development and endorsed preferences (~85%) for allocating more resources to prevent neurodevelopmental disorders, especially people with higher education, parents and pregnant women, and people who lived in India. Despite this, a large proportion of participants (~50%) trusted industry and believed that government effectively regulated toxic chemicals. After the six-week follow-up, experimental participants showed greater changes in scores on PRoTECT (i.e., between 5-15% change), indicating greater knowledge about harms posed by toxic chemicals, more intentions to reduce exposure, and stronger preferences for prevention as compared to the control group. Differences were larger among people from the US, those who were more highly educated, and people in their thirties. However, the differences between groups in making behavioural changes to reduce exposures were attenuated at the six-week follow up as compared to baseline. Significant barriers to reduce exposure to toxic chemicals were reported by both groups and included cost, inconvenience, and not knowing how to determine whether a product is non-toxic or where to purchase non-toxic products.

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