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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Conclusions: We observed greater knowledge and concerns about toxic chemicals among more affluent respondents, pregnant women and parents, and people living in India across both groups. While the video enhanced participants' knowledge about toxic chemicals and intentions to reduce exposure, they indicated that barriers hindered them from making behavioral changes.

Categories: Drug/Toxin-Related Disorders (including Alcohol)
Keyword 1: environmental pollutants / exposures
Keyword 2: neurotoxicity
Keyword 3: brain development
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Paper Session 19: Aging topics: section 4

10:45am - 12:10pm
Saturday, 4th February, 2023
Town & Country Ballroom C

Moderated by: Kayci Vickers

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1 Quantifying the Role of Social Determinants of Health in Racial Disparities

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Objective: In the United States, Black individuals have suffered from 300 years of racism, bias, segregation and have been systematically and intentionally denied opportunities to accrue wealth. These disadvantages have resulted in disparities in health outcomes. Over the last decade there has been a growing interest in examining social determinants of health as upstream factors that lead to downstream health disparities. It is of vital importance to quantify the contribution of SDH factors to racial disparities in order to inform policy and social justice initiatives. This demonstration project uses years of education and white matter hyperintensities (WMH) to illustrate two methods of quantifying the role of a SDH in producing health disparities.
Participants and Methods: The current study is a secondary data analysis of baseline data from a subset of the National Alzheimer's Coordinating Center database with neuroimaging data collected from 2002-2019. Participants were 997 cognitively diverse, Black and White (10.4% Black) individuals, aged 60-94 (mean=73.86, 56.5% female), mean education of 15.18 years (range= 0-23, SD=3.55). First, mediation, was conducted in the SEM framework using the R package lavaan. Black/White race was the independent variable, education was the mediator, WMH volume was the dependent variable, and age/sex were the covariates. Bootstrapped standard errors were calculated using 1000 iterations. The indirect effect was then divided by the total effect to determine the proportion of the total effect attributable to education. Second, a population attributable fraction (PAF) or the expected reduction in WMH if we eliminated low education and structural racism for which Black serves as a proxy was calculated. Two logistic regressions with dichotomous (median split) WMH as the dependent variable, first with low (less than high school) versus high education, and second with Black/White race added as predictors. Age/sex were covariates. PAF of education, and then of Black/White race controlling for education were obtained. Subsequently, a combined PAF was calculated.
Results: In the lavaan model, the total effect of Black/White race on WMH was not significant (B=.040, se=.113, p=.246); however, Black/White race significantly predicted education (B= -.108, se=.390, p=.001) and education significantly predicted WMH burden (B=-.084, se=.008, p=.002). This resulted in a significant indirect effect (effect=.009, se=.014, p=.032). 22.6 % of the relationship between Black/White race and WMH was mediated by education. In the logistic models, the PAF of education was 5.3% and the additional PAF of Black/White race was 2.7%. The combined PAF of Black race and low education was 7.8%.
Conclusions: From our mediation we can conclude that 22.6% of the relationship between Black/White race and WMH volume is explained by education. Our PAF analysis suggests that we could reduce 7.8% of the cases with high WMH burden if we eliminated low education and the structural racism for which Black serves as a

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