SATURDAY

388 Climate Extremes Impact the Infant Microbiome to Increase Risks for Allergy and Asthma

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RATIONALE: Climate change is increasing the frequency and severity of weather extremes, such as hurricanes. It is urgent to evaluate the impacts of such climate extremes on health. The early microbiome has lasting implications for health. An allergy- and asthma- promoting microbiome has been characterized, especially in the infant airway and gut. Whether climate extremes shift the infant microbiome towards the allergy- and asthma-promoting patterns has not been well investigated.

METHODS: We utilized a natural experiment by recruiting a unique birth cohort after the devastating Hurricane Maria in San Juan Puerto Rico. In total 63 full-term infants aged two to six months were included in this study, including n=29 infants who were exposed *in utero* to the hurricane and n=34 infants who were conceived at least five months after the Hurricane as controls. We performed 16S ribosomal RNA gene sequencing and shotgun metagenomic sequencing on infant stool swab and nasal swab samples.

RESULTS: Infants who were exposed *in utero* to the hurricane harbored allergy- and asthma- promoting microbiomes, including increased pathogenic bacteria in the nasal microbiome and decreased diversity in the gut microbiome. The infant nasal microbiome influences the gut microbiome, with a stronger effect being detected among the hurricane-exposed infants.

CONCLUSIONS: Climate extremes, such as a devastating hurricane, may increase risks for allergy and asthma via shifting the early microbiome. Strategies targeting at modifying the microbiome may mitigate health impacts caused by climate change.

389 Degrees of Climate Change (CC): The Significance of 2°C and 3°C Thresholds in Pollen Season Dynamics

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RATIONALE: CC pushes temperature, resulting in increased Growing Degree Days (GDD), extending pollen season lengths (PSL), and exacerbating allergic airway diseases (AAD). Calculating GDD for 2°C and 3°C warming scenarios, we aim to estimate the increase in PSL due to CC.

METHODS: Employing linear regression models to project pollen season lengths for tree, grass, and weed pollens (TP/WP/GP) under 2°C and 3°C pre-industrial warming scenarios by calculating growing degree days (GDD) from PSL defined by EAACI criteria across Zone 6 (2023 USDA Map). GDD were calculated using NOAA temperature records and tbase=50°F. We identified PSL using initial data from AAAAI - National Allergy Bureau pollen counting stations reporting from 2003-2009 (PCS n=37) including 21 pollen species. Regression modeling for projected PSL under 2°C and 3°C conditions utilized initial slope and y-intercept and projected GDDs.

RESULTS: A 2°C increase generated an increase in PSL duration (days) for TP, WP, and GP ranging from 2-11, 0-7, and 0-14; 3°C increase generated 2-6, 1-9, and 1-23, respectively. Similarly, a 2°C generated an increase in the ΔPSL% for TP (+34%), WP (+25%), and GP (+22%); 3°C was +51%, +36%, and +22%, respectively. R^2 values for TP, WP, and GP were 0.644, 0.889, and 0.987, respectively.

CONCLUSIONS: An increase in 2°- 3°C led to an increased GDD, resulting in longer projected PSL. These extended pollen seasons with each degree of warming may intensify the clinical impact on patients suffering from AAD.

390 Mice Repeatedly Exposed to Vapor Containing Peracetic Acid Exhibit Airway Hyperresponsiveness

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RATIONALE: Acute or repeated employment-related exposure to vapor containing peracetic acid (PAA), an organic oxidant found in disinfectant cleaners, may lead to occupational asthma (OA), which is incidental asthma caused by exposure to workplace irritants or sensitizers. Nevertheless, no animal model currently exists to elucidate the molecular bases underlying OA incited by exposure to PAA. Therefore, our goal was to develop a mouse model of PAA-induced OA, which is characterized, in part, by airway hyperresponsiveness (AHR), a cardinal feature of asthma.

METHODS: Male C57BL/6J mice were exposed to either filtered room air (FRA) or vapor containing PAA (4 parts/million) for six hours *per* day, for four days per week, for nineteen days. Twenty-four hours following cessation of the final exposure, indices of airway responsiveness to aerosolized methacholine [airway resistance (R_{aw}), coefficients of lung tissue damping (G) and elastance (H), and lung tissue hysteresivity (η)] were measured using the forced oscillation technique.

RESULTS: Mice repeatedly exposed to PAA weighed significantly less than mice exposed to FRA twenty-four hours following cessation of the final exposure. As compared to mice exposed to FRA, exposure to PAA significantly enhanced methacholine-induced increases in R_{aw}, G, H, and η.

CONCLUSIONS: Repeated exposure of mice to vapor containing PAA causes AHR, which is consistent with diagnoses of OA in humans that are exposed to this irritant in the workplace. Therefore, this newly developed animal model may be useful to elucidate the mechanistic bases for the development of OA following employment-related exposure to disinfectant cleaners.

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