

Antimony Compounds Associate with Atopic Dermatitis and Influence Models of Itch and Dysbiosis

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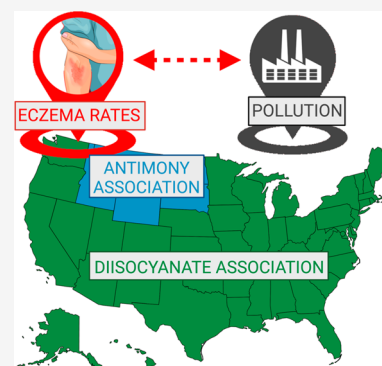
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ABSTRACT: Compared to the myriad of known triggers for rhinitis and asthma, environmental exposure research for atopic dermatitis (AD) is not well established. We recently reported that an untargeted search of U.S. Environmental Protection Agency (EPA) databases versus AD rates by United States (U.S.) postal codes revealed that isocyanates, such as toluene diisocyanate (TDI), are the pollutant class with the strongest spatiotemporal and epidemiologic association with AD. We further demonstrated that (di)isocyanates disrupt ceramide-family lipid production in commensal bacteria and activate the thermo-itch host receptor TRPA1. In this report, we reanalyzed regions of the U.S. with low levels of diisocyanate pollution to assess if a different chemical class may contribute. We identified antimony compounds as the top associated pollutant in such regions. Exposure to antimony compounds would be expected from brake dust in high-traffic areas, smelting plants, bottled water, and dust from aerosolized soil. Like TDI, antimony inhibited ceramide-family lipid production in *Roseomonas mucosa* and activated TRPA1 in human neurons. While further epidemiologic research will be needed to directly evaluate antimony exposure with surrounding AD prevalence and severity, these data suggest that compounds which are epidemiologically associated with AD, inhibit commensal lipid production, and activate TRPA1 may be causally related to AD pathogenesis.

KEYWORDS: Atopic dermatitis, TRPA1, antimony, diisocyanate, isocyanate, eczema, microbiome



INTRODUCTION

A connection between allergic diseases and environmental exposures has been demonstrated since early research revealed pollen as a trigger for rhinitis and pollution as a trigger for asthma.^{1–3} However, environmental exposure research for atopic dermatitis (AD) is not as well established. Particularly high rates of AD are associated with living in urbanized sectors of industrialized nations, especially after the year 1970.⁴ We recently reported that an untargeted search of U.S. Environmental Protection Agency (EPA) databases versus AD rates by United States (U.S.) postal codes revealed that isocyanates, such as toluene diisocyanate (TDI), are the pollutant class with the strongest spatiotemporal and epidemiologic association with AD.⁵ We further demonstrated exposure of health-associated commensal bacteria to TDI shifts the microbial metabolism toward an AD-like phenotype by disrupting ceramide-family lipid production and negating the modeled benefit in commensal isolates of *Roseomonas mucosa* through disrupting nitrogen fixation.⁵ Beyond impacts on commensal bacteria, we have additionally shown that TRPA1 is directly activated by TDI^{6–11} in a targetable manner operating through the balance of catecholamine signaling.¹²

While it is possible that products containing TDI brought into the home, wildfires, and car exhaust could generate (di)isocyanate exposure in areas without diisocyanate-produc-

ing factories, not every area in the U.S. has high levels of (di)isocyanate pollution.⁵ In this report, we aimed to reanalyze regions of the U.S. with low levels of diisocyanate pollution to assess if a different chemical class may contribute. We identified antimony compounds as the top associated pollutant in regions with low diisocyanate exposure. Antimony is a natural element (elemental signal Sb), to which exposure would be expected from both brake dust in high-traffic areas and dust from aerosolized soil.¹³ To further assess for a shared mechanism, we demonstrate that antimony, like TDI,^{5,12} inhibited ceramide-family lipid production in *R. mucosa* and activated TRPA1. While further epidemiologic research will be needed to directly evaluate antimony exposure with surrounding AD prevalence and severity, these data suggest that compounds which are epidemiologically associated with AD, inhibit commensal lipid production, and activate TRPA1 may be causally related to AD pathogenesis.

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Figure 1. Antimony compounds associate with AD in low TDI areas of the U.S. (A) Map of the U.S. with per capita rate of atopic dermatitis (AD) clinic visits as blue circles (larger circles indicate high visit rates), diisocyanate exposure per EPA databases depicted as green overlays, and major highways drawn in orange as indicated. (B) Colinearity plot for top variables selected by random forest. (C, D) Random forest (C) and Lasso (D) for top variables associated with AD visits in Montana, Idaho, Wyoming, North Dakota, and South Dakota. (E) Map as in A with clinic visits as blue circles, diisocyanate and/or antimony exposure depicted as green overlays, and major highways drawn in orange. TDI = toluene diisocyanate, iso = isomer, DIC = diisocyanate.

METHODS

Spatial Mapping and Environmental Survey. As detailed previously,⁵ data on pollution derived from the EPA's Risk-Screening Environmental Indicators (RSEI) system from 2015 to 2019 were contrasted against the Definitive Health database, which contains 1.2 billion diagnostic billing codes per year from 2019 across 20,000 postal codes in the United States, including Hawaii, Alaska, and U.S. territories for ICD-10 code L40.9. Elemental compounds, including antimony, can be reported in TRI as pure elemental release or a metal-containing compounding (that may also be mixed with pure element). For our analyses, the metal and metal compounds were *a priori* combined into one variable. Additional data on air pollution in 2015 from the Center for Air, Climate, and Energy Solutions (CACES) were used to incorporate CO, NO₂, particulate matter of 2.5 μm or less (PM_{2.5}), PM₁₀, and SO₂.¹⁴ For each postal code tabulation area where ICD codes are aggregated, the concentration of pollution from the surrounding census tracts within a 50-mile radius was averaged in a weighted manner based on both population in each census tract and the proximity to the postal code centroid (based on a Gaussian constructed assuming the average distance to a primary care doctor is 8.6 miles¹⁵). Other covariates included the population density, bracketed age ranges (from American Community Survey), the Area Deprivation Index (from Neighborhood Atlas¹⁶), and the proportion of visits to specialists (i.e., rheumatologists, dermatologists). The spatially lagged y autocovariate was constructed with inverse distance weights. Poisson lasso regression with 10-fold cross-validation was implemented with the glmnet package. Random forest regression was implemented with the ranger package, with case weights equal to the total number of visits, mtry untuned to one-third the number of features, and 1000 trees.

Calcium Flux Assessment. Schwann cells were seeded in glass-bottomed dishes for the Ca²⁺ imaging experiment. The next day, cells were loaded with a calcium sensitive dye (Fluo-4 AM, 2 μM for 30 min; ThermoFisher). After cells were washed with warm PBS, fresh culture media were added. The cells were imaged with a Leica SP8 confocal microscope. Cells were stimulated with specific agonists or antagonists (TDI 100 μM and toluene used at 0.6 mM and HC030031 used at 10 mM concentration) or diluent control as described during the imaging. The live images were quantified using ImageJ software, and graphs were generated with GraphPad Prism software.

Bacteriology. Isolates of *R. mucosa* were selected as previously described.^{17–19} *Roseomonas* was grown in R2A broth (Teknova; Hollister, CA) or on R2A agar (Remel; San Diego, CA) at 32 °C. Bacterial plates were placed in anaerobic chambers, either Torbal AJ-2, BBL Gaspak Jar, or a Torbal AJ-3 equipped with a digital Dwyer gauge (Grainger; Rockville, MD). Custom gas mixes were purchased from Roberts Oxygen (Rockville, MD). 2,4-TDI and toluene were purchased from Sigma and applied to a glass lid of the agar plate (Fisher). Antimony is itself water insoluble and nonvolatile, but when diluted in the highly volatile methylene chloride (Sigma), it could be applied to the glass lid of the dish and compared with methylene chloride alone. Bacterial pellets from broth cultures or loops (Fisherbrand #22-363-606) were placed into a 20 mg/mL 2,5-dihydroxybenzoic acid (DHB) (#149357-10G Sigma-Aldrich) matrix solution in 70% methanol (Sigma)

and 0.1% trifluoroacetic acid (TFA; Sigma). Samples were then plated on spot plates (Bruker; Billerica, MA). Plates were cleaned between uses in 100% methanol in an E+ Easy Elmasonic instrument (Elma; Quebec, Canada). All MALDI data were collected using timsControl (Bruker). MS settings: scan range 20–2500 m/z on negative MS scan mode. TIMS settings of 1/K0 0.8–1.8 V s/cm². Ramp time of 73 ms, Accu time of 20 ms, duty cycle = –27.4%, and ramp rate of 12.64 Hz. Annotations were performed on Metaboscape 2021b as were *t* tests for comparison between indicated conditions. In select experiments, annotations were searched for “Cer”, and total intensity for ceramide containing annotations were exported and summed. Cytation 5 and Gen5 software were also used to take images at 1.25 $\times$ of agar plates (BioTek).

RESULTS

Antimony Associates with Atopic Dermatitis in Diisocyanate-Low U.S. Postal Codes. Our prior results evaluating TDI^{5,12} suggested that compounds which modulate commensal lipid production and influence TRPA1 may contribute to AD. However, the potential role of other air pollutants that may colinearize with TDI had not been evaluated. Therefore, we tested to see whether TDI may be correlated with other compounds which could activate TRPA1^{11,20,21} or if alternate chemicals may impact TDI-low areas of the U.S. First, we mapped our previous environmental assessments⁵ which used the EPA Toxin Release Inventory (TRI) and Risk-Screening Environmental Indicators (RSEI) versus rates of AD. A strong spatial relationship was evidence contrasting visit rates for AD with RSEI concentration of diisocyanate, TDI, and major highways (a major source of isocyanate)⁵ (Figure 1A). Assessment for collinearity between the diisocyanates and variables identified by random forest (Figure 1B) did not show collinearity between TDI and other variables, suggesting TDI was not being selected as a representative from a group of collinear features.

Notably, the region spanning Montana, Idaho, Wyoming, and the Dakotas appeared disparate in its association with AD and factory-produced diisocyanates (Figure 1A; Supplemental Table 1). The correlation coefficient between AD visits to pediatricians in 2019 versus the 2015–2019 exposure to diisocyanates in these five states was 0, compared to a coefficient of 0.102 in the remaining U.S. states (Supplemental Table 1). We thus ran an additional model limited to only this North Central region and identified another known influencer of bacterial nitrogen metabolism, antimony,²² as the most predictive variable (Figure 1C, D). A correlation between AD and antimony was seen for in Montana, Idaho, Wyoming, and the Dakotas (coefficient = 0.84) versus –0.014 in the remaining states. Combining the isocyanate exposures to antimony on the RSEI map enhanced the visual appearance of correlation with AD (Figure 1E).

Antimony Impacts Lipid Production from *R. mucosa*. Our prior work established the importance of bacterial-sourced, ceramide-family lipids in the modeled benefits of *R. mucosa* treatment.⁵ We determined that TDI inhibited ceramide production in healthy isolates of *R. mucosa* through disruption of nitrogen fixation.⁵ Thus, we hypothesized that *R. mucosa* ceramide production and nitrogen metabolism may be viable screens to assess other compounds associated with AD that may contribute to the pathogenesis through identical means. Similar to TDI,⁵ antimony was able to protect *R. mucosa* against the damages of nitrogen deprivation (Figure

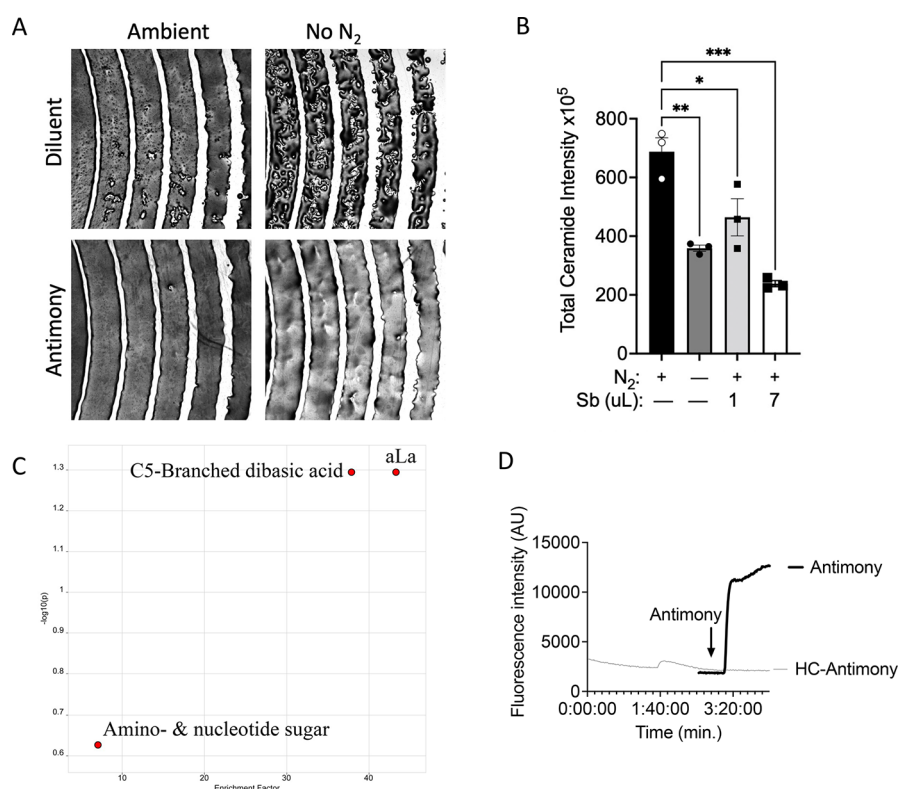


Figure 2. Antimony compounds modify nitrogen-containing lipid biology in *R. mucosa*. Representative images (A) and total annotated ceramide compounds by MALDI mass spec (B) of healthy control *R. mucosa* isolate incubated in ambient air or nitrogen deprivation (No N₂) with and without antimony (Sb). (C) Metaboanalyst selected pathways impacted by antimony exposure for *R. mucosa* in ambient air. (D) Average fluorescence intensity for calcium flux in Schwann cell neurons stimulated with antimony at time indicated by arrow; cells were pretreated with either diluent or TRPA1 inhibitor HC030031 (HC). Data represent four independent experiments and are shown as mean $\pm$ SEM * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$.

2A), inhibited total annotated ceramide levels (Figure 2B), and enriched metabolic impacts on the alpha-linoleic acid (aLa) pathway (Figure 2C).

Antimony Modulates TRPA1 in Human Neuron Cultures. Finally, to test if antimony compounds had similar impacts on TRPA1 as we saw with TDI,¹² we exposed human Schwann cell neurons to antimony or diluent and tested for calcium flux, a sign of activation for all TRP receptors.²³ Indeed, similar to TDI,¹² antimony induced Ca²⁺ flux in Schwann cells but could be blocked with a TRPA1 antagonist using HC030031 (Figure 2D; Supplemental Figure 1).

DISCUSSION

Atopic dermatitis has afflicted patients since well before the industrial age; the earliest potential description of eczema was sometime between 69 and 140 CE.²⁴ Therefore, despite the post-1970 rise in AD rates, identifying potential environmental triggers that may be more relevant in nonurbanized environments could further elucidate AD pathogenesis. Antimony is a natural element most often found in soil and would be an expected natural exposure during dust storms.²⁵ Additional routes of exposures may include metal work, mining, and smelting.^{25,26} Oral exposure through food and drinking water (especially near the runoff from mining facilities) would also be expected in nonindustrialized areas.²⁷ Urbanized exposures would be expected by proximity to automobiles, which have been associated with risk of AD⁵ and begets antimony exposure through aerosolization of particles during brake pad wear.^{13,25} In-home exposures to antimony might include

bottles made of polyethylene terephthalate plastic, flame retardant coating on fabric and plastics, or microwaved ready-to-eat foods,¹³ each of which would be expected to be more common in the modern era. Our analysis suggests antimony exposure may be a separate path to AD pathogenesis but does not exclude isocyanates as contributing to AD in the areas mapped to have low factory production of diisocyanates. For example, automobile exhaust, isocyanate-containing in home products, and wildfires would all remain risk factors⁵ for the North Central U.S. but would not be captured by the EPA databases for factory output.

Our proposed mechanism for antimony's impact on AD is similar to that of our previous work on toluene diisocyanate (TDI).⁵ TDI alters commensal lipid production through disruption of nitrogen fixation and alters host physiology through activation of the thermo-itch receptor TRPA1.¹² Antimony is an established influencer of nitrogen fixation in plant biology,²⁸ and thus, our findings of nitrogen fixation disruption are consistent with the prior literature. Furthermore, nitrogenase-mediated nitrogen fixation and activation of TRPA1 demonstrate a shared biology centered around direct alteration of specific cysteine residues.^{11,20,21,29} Although limited to epithelia, in vitro studies evaluating how antimony compounds may impact AD pathways would predict a mixed effect: antimony's ability to inhibit beta-defensins and STAT3³⁰ would be predicted to worsen AD,³¹ while antimony's described inhibition of glucose transporter 1 and interleukin-23³⁰ would likely improve outcomes.^{32,33}

Our findings are limited by a lack of in vivo correlations such as mouse models or direct challenge studies in humans. Further work will be necessary to bridge this gap between our population-level associations and our proposed molecular mechanisms. Direct measurement of ambient antimony levels should be paired with longitudinal tracking of the incidence and severity of AD in the surrounding population. However, it may be noteworthy that antimony exposure from dust storms and isocyanate exposure from wildfires would be among the few, if not the only, exposures that could have contributed to AD during both antiquity and modern times. Therefore, overall, this work suggests antimony, as well as (di)isocyanates, should be prioritized for further assessments as potential contributors to AD through effects on commensal lipid biology and host TRPA1.

■ ASSOCIATED CONTENT

SI Supporting Information

The Supporting Information is available free of charge at <https://pubs.acs.org/doi/10.1021/acs.estlett.3c00142>.

Supplemental Table 1: Correlation coefficients for TDI, diisocyanate, and antimony for Montana, Wyoming, Idaho, and North and South Dakotas compared to the correlation coefficients for the remaining U.S. Supplemental Figure 1: Full activation set for antimony and Schwann neurons. (PDF)

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Author Contributions

J.Z. and T.T.T. performed the environmental surveying and mapping work. M.Y. conducted experiments and analyzed data

for calcium flux assessment. P.P.C. and B.N.D'S. assisted in conducting experiments and analyzed data for murine experiments. G.R. assisted in data analysis for MALDI-IMS. S.G. performed experiments on calcium flux. I.A.M. designed experiments, oversaw the project, and wrote the manuscript.

Notes

The authors declare no competing financial interest.

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