A COMPARATIVE ANALYSIS OF THE ROLE RACE AND SOCIOECONOMIC STATUS PLAY IN CHEMICAL EXPOSURE IN THE UNITED STATES

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Abstract

Environmental justice concerns arise when historically underrepresented groups are disproportionately exposed to toxins in the environment. Analysis of environmental biomonitoring data provides a method to analyze chemicals for race/ethnicity and income-related disparity. Using data from the US National Health and Nutrition Examination Survey 2013-2014, biomarker concentrations of 167 chemicals were analyzed. Ten subgroups were defined on the basis of race/ethnicity and income. To examine disparity, geometric mean (GM) concentrations of chemical biomarker for each subgroup were compared to a reference group (i.e., the non-Hispanic white individuals with poverty to income ratio ≥ 2). Of the 167 compounds considered, 95 were detected in >60% of samples and were evaluated for disparity. There was evidence of an environmental justice concern for 42 compounds (GM ratios significantly > 1) in at least one of the identified subgroups. For 21 of these compounds, disparity was present only in the low-income non-Hispanic Black subpopulation. Disparity was particularly pronounced for cotinine, propyl paraben, and dichlorophenol. GM ratios were significantly <1 for 16 chemicals, indicating higher exposure among high-income non-Hispanic whites. Cumulatively, this project demonstrates disproportionate exposure to environmental contaminants by income and race/ethnicity. Results suggest that the low-income non-Hispanic Black subpopulation experiences much higher instances of disparity. Comparing with prior research, results also suggest that disparity in environmental exposure may be increasing.

Introduction

Chemical exposures are ubiquitous throughout populations in the United States. Daily, people are exposed to chemicals from a variety of sources, including cosmetics, personal care products, off-gassing from materials in the home, and plant protection materials. Exposures also originate from discharge at industrial facilities, mining sites, roadway runoff, and chemical combustion [2]. Chemical exposure is a serious public health concern, as many contaminants found in our everyday environment have been linked to a number of diseases in the human population, including IQ loss [3-5], anxiety and depression [6-8], obesity [9-11], ADHD [6, 12], and cancer [5, 13, 14]. A 2016 report from the world health organization estimates that in 2012 1.3 million lives and 43 million disability-adjusted life-years were lost due to exposure to selected environmental contaminants [15].

Environmental justice [1] concerns arise when historically underrepresented groups – people of color (POC) or people of low socioeconomic status [16] – are disproportionately exposed to toxins in the environment, either intentionally or otherwise [1]. The EJ movement began as a response to the proposed construction of a polychlorinated biphenyl (PCB) landfill in Warren Co., NC in 1982 [17]. Since the protests in response to the landfill siting, instances of environmental injustice have been studied and cited widely in the literature [18]. To name a few -- Adamkiewicz et al. [19] found socioeconomic disparities in exposure to indoor air pollutants, Kraft & Scheberle [20] cite consistent disparity in lead exposure on the basis of both race and SES, and Bell & Ebisu [21] found that exposure to particulate matter exposure varied based on race and socioeconomic status. To date, many detailed studies of instances of environmental injustice rely on proximity analysis – i.e. what is the population distribution of the communities surrounding facilities that produce toxic waste [22-25]. This type of analysis, however, may

overlook instances of environmental injustice that arise outside of the disproportionate siting of hazardous facilities.

Measures of chemical biomarkers – parent compounds or their metabolites in blood, urine, or other specimens – provide an approach to examine potential disparities and understand EJ concerns beyond disproportionate citing. Biomarker concentrations are an integrated measure of exposure which represents the total amount of chemical exposure from all potential sources (air, water, food, soil, dust, and the use of consumer products) and all potential routes of exposure (ingestion, inhalation, dermal absorption) [26]. Thus, analyzing the diversity of biomarker concentrations across race and SES may shed light on environmental justice concerns that arise from differences in everyday activities, such as food preference or personal care product use.

The U.S. Centers for Disease Control and Prevention [26] collects environmental biomonitoring data through the National Health and Nutrition Examination Survey (NHANES). Although the NHANES biomarker concentrations are freely available, the CDC does not statistically compare biomarkers across the various racial and socioeconomic subgroups. In 2013, Belova et al. [27] published a method to screen NHANES biomonitoring data for instances of environmental injustice. They cited environmental justice concerns with exposure to lead, dichlorodiphenyldichloroethylene (DDE), parabens, phthalates, dichlorophenols, antimony and tin. To understand how trends in EJ in the United States are changing over time, we analyzed all biomarkers of environmental exposure in the NHANES 2013-2014 datasets for differences in U.S. population subgroups defined by race/ethnicity and income.

Building on the work of Belova et al. [27], we (1) model the joint impacts of race/ethnicity and income with the most recently published data; (2) test for statistically

significant evidence of disparity with adjustments for multiple comparisons; and (3) analyze how trends in disparity are evolving.

Methods

Study Population

NHANES, a cyclical study meant to analyze the health of children and adults in the United States, utilizes a complex survey design method to collect nationally representative biomonitoring data from around 5,000 people every two years. Every year 15 different counties nationwide are selected for examination [15]. Participants from within the selected counties complete a physical examination in a mobile exam unit and provide biospecimens for laboratory analysis. Questionnaires used to obtain demographic, dietary, and health information are administered to participants (or their legal guardian for children) by trained study personnel. Because data are published and freely available online, and anonymous, institutional review board (IRB) approval is not necessary.

The demographic information collected includes, among other things, race/ethnicity/ancestry and income. Race/ethnicity/ancestry categories include Mexican American (MA), non-Hispanic Black (NHB), non-Hispanic White (NHW), non-Hispanic Asian (NHA). Surveyed participants self-report their race, ethnicity, and ancestry. If a participant indicated 'Mexican American' they were coded as such, regardless of any other race/ethnicity/ancestries reported, otherwise those that self-identified as 'Hispanic' were coded as 'Other Hispanic'. Only those who did not indicate Hispanic origins were separated into the remaining categories. NHANES also specifies a 'mixed-race' variable, but this category was not included in the present analyses due to the small number of individuals reporting mixed race. Since 2011, NHANES has intentionally oversampled in the NHA, OH, NHB, and NHW-L subgroups. Because the total yearly sample size is fixed by operational constraints, the increased

sampling of NHA persons has led to decreased sample size in the MA, OH persons and non-low-income (at or below 185% of the federal poverty level) NHW subgroups [28]. Despite the sampling scheme, the sample sizes of all subgroups were adequate to include in the analysis (excepting for mixed-race individuals as described above).

Individuals participating in NHANES were also asked to report their income which was used to calculate the poverty to income ratio (PIR), a ratio of a family's total income to the family size-specific federal poverty level. This poverty level is determined yearly by the Department of Health and Human Services [29] and is issued in the Federal Register. The poverty level varies both by family size and by geographic location [20]. For the purposes of this analysis, the PIR was dichotomized to be consistent with Belova et al. [27]. "Low" income (L) was defined as PIR < 2, and "High" income (H) was defined as PIR >= 2. The unweighted median PIR in the data set is near 2, and a PIR threshold of 2 has been utilized in income-based analyses in the past.

These race/ethnicity/ancestry and income data were used to classify individuals into ten subgroups. Because limited biospecimen volumes are available in NHANES, participants were separated into subsamples for biomarker analysis. The participants in each subsample were tested for a portion of the biomarkers analyzed that cycle. Table 1 details the sample sizes for the 10 subgroups in each of the subsamples utilized.

Biomarkers of Exposure

Urine and blood samples are collected from NHANES participants in the mobile exam unit. As indicators of exposure, biomarkers (i.e., the chemicals or their metabolites) are measured in these samples. Data from 17 of the most recently available (as of January of 2019) NHANES laboratory and demographic files, corresponding to 167 different chemicals were

included in these analyses. In instances where one chemical was measured in multiple media, each measurement was treated as a separate biomarker. Per CDC convention, compounds with a detection rate <60% were excluded from the analysis [26]. For compounds detected in >60% of samples but not detected in all samples, concentrations below the limit of detection were replaced by the limit of detection divided by √2 for statistical analyses.

Statistical Analysis

Biomarker concentrations were generally lognormally distributed; accordingly, descriptive statistics included the geometric mean (GM) concentration for all biomarker [26, 27]. Geometric means were calculated using the 'RNHANES' package [30] with R Version 3.6.0 [31] which accounts for the complex sampling design.

Race/ethnicity and income-related disparity were assessed using a geometric mean ratio (GMR):

$$GMR_{bs} = \frac{GM_{bs}}{GM_{br}},$$

where GMR_{bs} is the ratio of the GM of biomarker *b* in subgroup *s* (GM_{bs}) with respect to the GM of biomarker b in the reference subgroup r (GM_{br}) Belova et al. [27]. NHW-H was used as the reference subgroup for all analyses. Nine GMR ratios (one for each subgroup compared to NHW-H) were calculated in Stata/SE 11.2 with a linear regression utilizing Stata's survey analysis package.

A subgroup-specific GMR was considered to be equivalent to the reference subgroup when $GMR_{bs} = 1$. Thus, for each biomarker *b* and subgroup *s* we tested the null hypothesis that GMRbs = 1 using two-sided tests.

The analyses involved multiple testing of the hypothesis $GMR_{bs} = 1$ for each and a large number of biomarkers. Thus, a high number of false positives was expected. The Holm-

Bonferroni procedure was utilized to account for this occurrence of false positives as recommended by the U.S. Food and Drug Administration [27].

Results

In the 2013- 2014 NHANES cycle a totally of 9,813 people were examined, ranging in age from <1 year to 80+ years. This includes 4,831 males, and 4,982 females. Laboratory tests (i.e. biomarker analysis) was not conducted on children younger than 6 years of age (1,430 participants), and blood samples were not taken for children under 12 years of age. The 8,401 participants older than 6 years of age were grouped into subsamples for laboratory analysis. A detailed breakdown of the subsample populations are detailed in Table 1.

Table 1. Demographic composition of the subsamples utilized in the NHANES survey

	NHW		MA		OH		NHB		NHA	
	H	L	H	L	H	L	H	L	H	L
Subsample A	513	458	135	273	87	144	219	352	205	72
Subsample B	466	383	95	215	84	107	173	299	170	67
Subsample C	553	444	118	273	90	130	243	332	190	91
Blood Metal	990	944	279	675	187	328	429	822	414	152
Environmental B 2-year	522	442	123	276	90	140	196	378	194	74

The abbreviations in this table are as follows: High-income (H), Low-income (L), non-Hispanic White (NHW), non-Hispanic Black (NHB), Other Hispanic (OH), Mexican American (MA), and non-Hispanic Asian (NHA).

Of the 167 chemicals analyzed, 95 had detection frequencies >60% and were included in GMR analyses (Table 2). There were 55 chemicals with significant evidence of disparity (after correcting for multiple comparisons): 42 chemicals with at least one GMR significantly >1, indicating possible incidence of environmental injustice (Table 3), and 13 chemicals with at least one GMR significantly <1 (Table 4), indicating a higher exposure in the NHW-H reference subgroup. Of the 42 biomarkers flagged for potential EJ concern, 21 (50%) were unique to the NHB-L subpopulation. The GMR screening results are provided in Tables 3 and 4.

Figure 1 provides a visual overview of the results and illustrates some important trends. First, there were a large number significantly elevated GMRs for the NHB-L subpopulation which indicates an environmental justice concern concentrated within one subgroup. Second, there were instances where certain subgroups, namely NHA and MA, had lower exposures than that of the reference subgroup. Additionally, aside from a select few compounds, biomarker concentrations in NHW do not differ widely based on income.

Considering specific biomarkers, cotinine, phthalates, arsenic and arsenic metabolites, metals, personal care products, polycyclic aromatic hydrocarbons (PAHs), and volatile organic compounds (VOCs) all show evidence of environmental injustice (i.e. GMR >1, significant at $p < 0.05$). On the other hand, flame retardant metabolites and PFAS chemicals exhibited GMR <1, indicating a higher exposure in the NHW-H (reference) subgroup. Certain personal care product metabolites, such as benzophenone – a biomarker for sunscreen, and triclosan – an antibiotic used in soaps, were found to be significantly higher in the reference subgroup.

Table 3 presents information of the 42 chemicals corresponding to 74 GMRs significantly >1, indicating potential environmental justice concern for cotinine, phthalates, arsenic, metals, parabens, phenols, PAHs, and VOCs. Of the 74 GMRs, there were 40 for the NHB-L, 8 for the

NHB-H, 3 for the NHW-L, 3 for the MA-H, 7 for the MA-L, 2 for the OH-L, 6 for the NHA-H, and 5 for the NHA-L subgroups.

Table 4 presents information on the 13 chemicals corresponding to 36 GMRs significantly <1, indicating higher biomarker concentration in the reference subgroup. These chemicals were distributed throughout the chemical classes. Of the 36 GMRs, there were 4 for the NHW-L subgroup, 1 for the MA-H Subgroup, 8 for the MA-L Subgroup, 2 for the OH-H subgroup, 3 for the OH-L subgroup, 3 for the NHB-H subgroup, 3 for the NHB-L subgroup, 6 for the NHA-H subgroup, and 9 for the NHA-L subgroup.

Because NHANES analyzes different biomarkers from cycle to cycle, comparisons to disparity analyzed in 2008 are limited. However, we were able to make comparisons for 11 biomarkers spanning 5 chemical classes (cotinine, metals, pesticides, phenols, and phthalates). These comparisons, provided in Table 5, show that although overall chemical concentrations seem to be decreasing, the GMRs are increasing, suggesting growing disparity. This is especially pertinent for the lead in urine biomarker, which fell below reportable detection limits in the 2013 – 2014 NHANES cycle for many participants.

Table 2. This table shows the detection frequency for each compound in the sample examined.

Biomarker	% Detect	Biomarker	% Detect	Biomarker	% Detect	Biomarker	% Detect	Biomarker	% Detect
Mono(carboxyisononyl) phthalate	0.99	Diphenyl phosphate	0.92	Urinary Uranium	0.84	1-Hydroxypyrene	0.76	Blood Octane	0.01
Mono(carboxyisoctyl) phthalate	1.00	Bis(1,3-dichloro-2-propyl) phosphate	0.93	Nitrate, urine	1.00	2-Hydroxyphenanthrene & 3-Hydroxyphenanthrene	1.00	Blood 1,2-Dichlorobenzene	0.00
MECP phthalate	1.00	Bis(1-chloro-2-propyl) phosphate	0.61	Perchlorate, urine	1.00	Urinary N-Acetyl-S-(2-carbamoylethyl)-L-cysteine	0.99	Blood 1,2-Dichloroethane	0.02
Mono-n-butyl phthalate	0.98	Bis(2-chloroethyl) phosphate	0.90	Thiocyanate, urine	1.00	Urinary N-Acetyl-S-(N-methylcarbamoyl)-L-cysteine	1.00	Blood 1,3-Dichlorobenzene	0.00
Mono-(3-carboxypropyl) phthalate	0.90	Di-p-cresyl phosphate	0.14	Perfluorohexane sulfonic acid	0.79	Urinary 2-Aminothiazoline-4-carboxylic acid	0.95	Blood Tetrachloroethene	0.07
Mono-ethyl phthalate	1.00	Di-o-cresyl phosphate	0.00	2-(N-Methyl-PFOSA) acetic acid	0.99	Urinary N-Acetyl-S-(benzyl)-L-cysteine	0.99	Blood Bromoform	0.05
MEHP phthalate	0.99	Dibutyl phosphate	0.81	Pefluorodecanoic acid	0.44	Urinary N-Acetyl-S-(n-propyl)-L-cysteine	0.69	Blood Bromodichloromethane	0.12
MHNCH	0.30	Dibenzyl phosphate	0.00	Perfluorobutane sulfonic acid	0.01	Urinary N-Acetyl-S-(2-carboxyethyl)-L-cysteine	0.99	Blood Benzene	0.25
Mono-(2-ethyl)-hexyl phthalate	0.62	2,3,4,5-tetrabromobenzoic acid	0.05	Perfluoroheptanoic acid	0.13	Urinary N-Acetyl-S-(2-cyanoethyl)-L-cysteine	0.90	Blood Cyclohexane	0.02
Mono-isobutyl phthalate	0.97	Fluoride, Plasma	1.00	Perfluorononanoic acid	0.99	Urinary N-Acetyl-S-(1,2-dichlorovinyl)-L-cysteine	0.00	Blood Chlorobenzene	0.00
Mono-isononyl phthalate	0.40	2-Amino-9H-pyrido[2,3-b]indole (A-a-C)	0.50	Perfluoroundecanoic acid	0.44	Urinary N-Acetyl-S-(2,2-dichlorovinyl)-L-cysteine	0.00	Blood Chloroform	0.49
MEOH phthalate	1.00	2-Amino-6-methyldipyrido[1,2-a:3',2'-d]imidazole (Glu-P-1)	0.02	Perflurododecanoic acid	0.17	Urinary N-Acetyl-S-(3,4-dihydroxybutyl)-L-cysteine	1.00	Blood Dibromochloromethane	0.12
Mono-benzyl phthalate	0.98	2-Aminodipyrido[1,2-a:3',2'-d] imidazole (GLU-P-2)	0.00	Linear perfluorooctanoate	0.99	Urinary N-Acetyl-S-(dimethylphenyl)-L-cysteine	0.00	Blood Carbon Tetrachloride	0.00

Metabolite	% Detect	Metabolite	% Detect	Metabolite	% Detect	Metabolite	% Detect	Metabolite	% Detect
Arsenic (total)	1.00	Harman	1.00	Branched isomers of perfluorooctanoate	0.19	Urinary N-Acetyl-S-(2-carbamoyl-2-hydroxyethyl)-L-cysteine	0.41	Blood 1,4-Dichlorobenzene	0.78
Urinary arsenous acid	0.72	2-Amino-3-methyl-3H-imidazo[4,5-f]quinoline (IQ)	0.02	Linear perfluorooctane sulfonate (PFOS)	0.99	Urinary N-Acetyl-S-(2-hydroxyethyl)-L-cysteine	0.46	Blood 1,2-Dibromoethane	0.00
Urinary arsenic acid	0.02	2-Amino-3-methyl-9H-pyriodo[2,3-b]indole (MeA-a-C)	0.15	Monomethyl branched isomers of PFOS	0.98	Urinary N-Acetyl-S-(3-hydroxypropyl)-L-cysteine	1.00	Blood Diethyl Ether	0.00
Urinary arsenobetaine	0.44	*Norharman*	1.00	Urinary Benzophenone-3	0.95	Urinary N-Acetyl-S-(2-hydroxypropyl)-L-cysteine	0.94	Blood 1,4-Dioxane	0.00
Urinary arsenocholine	0.16	2-Amino-1-methyl-6-phenylimidazo[4,5-b]pyridine (PhIP)	0.57	Urinary Bisphenol A	0.96	Urinary N-Acetyl-S-(3-hydroxypropyl-1-methyl)-L-cysteine	1.00	Blood Ethyl Acetate	0.02
Urinary Dimethylarsinic Acid	0.76	3-Amino-1,4-dimethyl-5H-pyrido[4,3-b]indole (Trp-P-1)	0.02	Urinary Bisphenol F	0.65	Urinary Mandelic acid	0.98	Blood Ethylbenzene	0.27
Urinary Monomethylarsonic Acid	0.70	1-Methyl-3-amino-5H-pyrido[4,3-b]indole (Trp-P-2)	0.02	Urinary Bisphenol S	0.90	Urinary 2-Methylhippuric acid	0.90	Blood Chloroethane	0.00
Blood Lead	1.00	Iodine	0.99	Urinary Triclocarban	0.39	Urinary 3- and 4-Methylhippuric acid	0.99	Blood Furan	0.15
Blood Cadmium	0.71	Urinary mercury	0.66	Urinary Triclosan	0.76	Urinary N-Acetyl-S-(1-hydroxymethyl-2-propenyl)-L-cysteine	0.01	Blood Isopropylbenzene	0.00
Blood Mercury	0.74	Urinary Barium	1.00	Butyl paraben	0.31	Urinary N-Acetyl-S-(2-hydroxy-3-butenyl)-L-cysteine	0.10	Blood Methylene Chloride	0.00
Blood Selenium	1.00	Urinary Cadmium	0.82	Ethyl paraben	0.47	Urinary N-Acetyl-S-(4-hydroxy-2-butenyl)-L-cysteine	0.98	Blood Methylcyclopentane	0.02
Blood Manganese	1.00	Urinary Cobalt	1.00	Methyl paraben	0.99	Urinary N-Acetyl-S-(phenyl-2-hydroxyethyl)-L-cysteine	0.36	Blood MTBE	0.02

Metabolite	% Detect	Metabolite	% Detect	Metabolite	% Detect	Metabolite	% Detect	Metabolite	% Detect
Serum copper	1.00	Urinary Manganese	1.00	Propyl paraben	0.99	Urinary Phenylglyoxylic acid	0.99	Blood Nitrobenzene	0.00
Serum selenium	1.00	Urinary Molybdenum	1.00	Urinary 2,4-dichlorophenol	0.98	Urinary N-Acetyl-S-(phenyl)-L-cysteine	0.38	Blood o-Xylene	0.27
Serum zinc	1.00	Urinary Lead	0.30	Urinary 2,5-dichlorophenol	0.95	Urinary N-Acetyl-S-(trichlorovinyl)-L-cysteine	0.00	Blood Trichloroethene	0.01
Serum cotinine	0.68	Urinary Antimony	0.99	1-Hydroxynapthalene (1-Naphthol)	1.00	Urinary 2-Thioxothiazolidine-4-carboxylic acid	0.35	Blood 1,1,1-trichloroethane	0.01
Serum hydroxycotinine	0.48	Urinary Strontium	0.78	2-Hydroxynapthalene (2-Naphthol)	1.00	Blood 2,5-Dimethylfuran	0.18	Blood ααα-Trifluorotoluene	0.00
DEET	0.05	Urinary Thallium	0.91	3-Hydroxyfluorene	0.98	Blood 1,1,1,2-Tetrachloroethane	0.00	Blood Tetrahydrofuran	0.01
DEET acid	0.82	Urinary Tin	1.00	2-Hydroxyfluorene	1.00	Blood Hexane	0.02	Blood Toluene	0.96
Desethyl hydroxy DEET	0.08	Urinary Tungsten	0.99	1-Hydroxyphenanthrene	0.99	Blood Heptane	0.02	Blood 1,2,3-Trichloropropane	0.00
						Blood m-/p-Xylene	0.64	Blood Vinyl Bromide	0.00

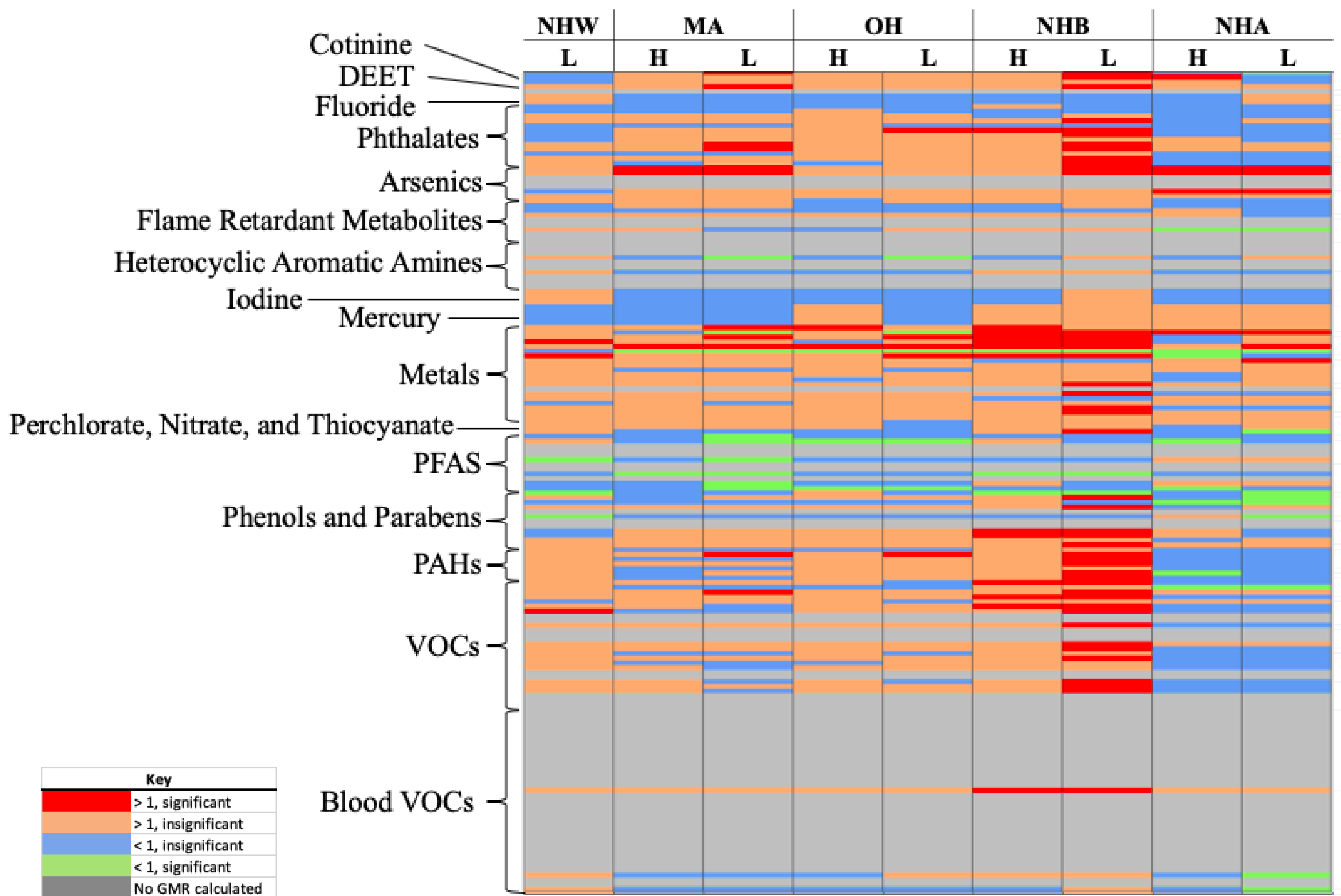

Figure 1. A visual representation of GMR for NHANES 2013 - 2014 for all subgroups. Each cell summarizes the output of the GMR and significance. The cells are color-coded to represent the direction of the relationship as well as significance determined by $p < 0.05$, corrected for multiple comparisons. The columns correspond to the 9 race/ethnicity and income-based subgroups being compared to the reference subgroup (NHW-H). Each row in the matrix corresponds to one of the 167 biomarkers analyzed, grouped into similar compounds (i.e. phthalates, personal care product metabolites, VOCs, PAHs, etc.).

Table 3. Chemicals demonstrating environmental justice concern for at least one subgroup (GMR > 1, significant at a level of $p < 0.05$).

Chemical Class	Biomarker (units, media)	Subgroup		Subgroup geometric mean	Reference subgroup geometric mean
		Race/Ethnicity	Income		
Cotinine	Serum cotinine (ng/mL)	NHW	L	0.99	0.11
	Serum cotinine (ng/mL)	NHB	H	0.20	0.11
	Serum cotinine (ng/mL)	NHB	L	1.58	0.99
Pthalates	Mono-n-butyl phthalate (ng/mL)	NHB	L	12.99	8.38
	Mono-ethyl phthalate (ng/mL)	OH	L	64.22	29.53
	Mono-ethyl phthalate (ng/mL)	NHB	H	72.68	29.53
	Mono-ethyl phthalate (ng/mL)	NHB	L	71.39	29.53
	Mono-(2-ethyl)-hexyl phthalate (ng/mL)	MA	L	1.64	1.14
	Mono-(2-ethyl)-hexyl phthalate (ng/mL)	NHB	L	1.70	1.14
	Mono-isobutyl phthalate (ng/mL)	MA	L	9.34	6.15
	Mono-isobutyl phthalate (ng/mL)	NHB	L	11.80	6.15
	MEOH phthalate (ng/mL)	NHB	L	5.93	3.93
	Mono-benzyl phthalate (ng/mL)	NHB	L	7.74	3.81
Arsenics	Arsenic (total) (ng/mL)	MA	H	6.84	5.87
	Arsenic (total) (ng/mL)	MA	L	5.23	5.87
	Arsenic (total) (ng/mL)	NHB	L	7.89	5.87
	Arsenic (total) (ng/mL)	NHA	H	10.80	5.87
	Arsenic (total) (ng/mL)	NHA	L	14.06	5.87
	Urinary arsenous acid (ug/mL)	MA	H	0.45	0.26
	Urinary arsenous acid (ug/mL)	MA	L	0.39	0.26
	Urinary arsenous acid (ug/mL)	NHB	L	0.42	0.26
	Urinary arsenous acid (ug/mL)	NHA	H	0.45	0.26
	Urinary arsenous acid (ug/mL)	NHA	L	0.46	0.26
	Urinary Dimethylarsinic Acid (ug/mL)	NHA	H	5.21	2.88
	Urinary Dimethylarsinic Acid (ug/mL)	NHA	L	6.19	2.88
Metals	Blood Cadmium (ug/L)	NHA	H	0.33	0.22
	Blood Cadmium (ug/L)	NHA	L	0.37	0.22
	Blood Mercury (ug/L)	NHA	H	1.71	0.77
	Blood Manganese (ug/L)	MA	H	10.52	9.30
	Blood Manganese (ug/L)	MA	L	11.33	9.30
	Blood Manganese (ug/L)	NHA	H	12.29	9.30
	Blood Manganese (ug/L)	NHA	L	12.85	9.30
	Serum copper (ug/dL)	NHB	L	125.57	111.84
	Urinary Molybdenum (ug/L)	NHB	L	45.16	29.08
Metals	Urinary Antimony (ug/L)	NHB	L	0.07	0.04
	Urinary Tin (ug/L)	NHB	L	0.91	0.36
	Urinary Tungsten (ug/L)	NHB	L	0.09	0.05
	Thiocyanate, urine (ng/mL)	NHB	L	1823.71	968.27
PCPs	Urinary Bisphenol A (µg/L)	NHB	L	1.90	1.17
	Urinary Bisphenol S (ug/L)	NHB	L	0.64	0.38
	Methyl paraben (µg/L)	NHB	H	106.58	40.62
	Methyl paraben (µg/L)	NHB	L	113.03	40.62
	Propyl paraben (µg/L)	NHB	H	14.94	4.90
	Propyl paraben (µg/L)	NHB	L	15.02	4.90
	Urinary 2,5-dichlorophenol (µg/L)	NHB	L	12.35	1.80
PAHs	2-Hydroxynapthalene (2-Naphthol)	MA	L	5574.62	3355.59
	2-Hydroxynapthalene (2-Naphthol)	OH	L	5196.33	3355.59
	2-Hydroxynapthalene (2-Naphthol)	NHB	L	7773.14	3355.59
	3-Hydroxyfluorene	NHB	L	177.61	66.19
	2-Hydroxyfluorene	NHB	L	342.71	153.70
	1-Hydroxypyrene	NHB	L	222.86	117.98
	2-Hydroxyphenanthrene & 3-Hydroxyphenan	NHB	L	197.96	103.07

Chemical Class	Biomarker (units, media)	Subgroup		Subgroup geometric mean	Reference subgroup geometric mean
		Race/Ethnicity	Income		
VOCs	Urinary N-Acetyl-S-(2-carbamoylethyl)-L-cy	NHB	H	69.12	40.66
	Urinary N-Acetyl-S-(2-carbamoylethyl)-L-cy	NHB	L	70.82	40.66
	Urinary 2-Aminothiazoline-4-carboxylic acid	MA	L	148.58	88.12
	Urinary 2-Aminothiazoline-4-carboxylic acid	NHB	L	155.72	88.12
	Urinary N-Acetyl-S-(benzyl)-L-cysteine	NHB	H	9.96	5.46
	Urinary N-Acetyl-S-(benzyl)-L-cysteine	NHB	L	11.26	5.46
	Urinary N-Acetyl-S-(2-carboxyethyl)-L-cyste	NHB	H	136.35	71.14
	Urinary N-Acetyl-S-(2-carboxyethyl)-L-cyste	NHB	L	135.71	71.14
	Urinary N-Acetyl-S-(2-cyanoethyl)-L-cystein	NHW	L	90.96	71.14
	Urinary N-Acetyl-S-(2-cyanoethyl)-L-cystein	NHB	L	9.15	2.53
	Urinary N-Acetyl-S-(3,4-dihydroxybutyl)-L-c	NHB	L	332.01	227.52
	Urinary N-Acetyl-S-(3-hydroxypropyl)-L-cys	NHB	L	305.67	186.85
	Urinary N-Acetyl-S-(2-hydroxypropyl)-L-cys	NHB	L	36.53	24.51
	Urinary Mandelic acid	NHB	L	160.00	102.69
	Urinary N-Acetyl-S-(4-hydroxy-2-butenyl)-L	NHB	L	9.23	5.23
	Urinary N-Acetyl-S-(phenyl-2-hydroxyethyl)-	NHB	L	1.02	0.69
	Urinary Phenylglyoxylic acid	NHB	L	227.43	157.96
	Blood 1,4-Dichlorobenzene (ng/mL)	NHB	H	0.11	0.04
	Blood 1,4-Dichlorobenzene (ng/mL)	NHB	L	0.14	0.04

Table 4. Chemicals demonstrating environmental justice concern (GMR < 1, significant at a level of $p < 0.05$)

Chemical Class	Biomarker (units, media)	Subgroup		Subgroup geometric mean	Reference subgroup geometric mean
		Race/Ethnicity	Income		
Flame Retardent	Dibutyl phosphate (μg/L)	NHA	H	0.12	0.19
Metabolites	Dibutyl phosphate (μg/L)	NHA	L	0.08	0.19
HCAA	Harman	MA	L	84.23	147.52
	Harman	OH	L	98.79	147.52
Metals	Blood Mercury (ug/L)	NHW	L	0.45	0.77
	Blood Mercury (ug/L)	MA	L	0.49	0.77
	Blood Manganese (ug/L)	NHB	H	8.09	9.30
	Blood Manganese (ug/L)	NHB	L	8.36	9.30
	Thiocyanate, urine (ng/mL)	NHA	L	476.09	968.27
PFAS	Perfluorohexane sulfonic acid (ng/mL)	MA	L	0.87	1.64
	2-(N-Methyl-PFOSA) acetic acid (ng/mL)	MA	L	0.10	0.13
	2-(N-Methyl-PFOSA) acetic acid (ng/mL)	OH	H	0.08	0.13
	2-(N-Methyl-PFOSA) acetic acid (ng/mL)	OH	L	0.09	0.13
	2-(N-Methyl-PFOSA) acetic acid (ng/mL)	NHA	H	0.09	0.13
	Perfluorononanoic acid (ng/mL)	NHW	L	0.54	0.77
	Perfluorononanoic acid (ng/mL)	MA	L	0.49	0.77
	Linear perfluorooctanoate (ng/mL)	MA	H	1.58	2.27
	Linear perfluorooctanoate (ng/mL)	MA	L	1.15	2.27
	Linear perfluorooctanoate (ng/mL)	NHB	H	1.51	2.27
	Linear perfluorooctanoate (ng/mL)	NHB	L	1.35	2.27
	Linear perfluorooctane sulfonate (PFOS) (ng/mL)	MA	L	2.22	3.87
	Monomethyl branched isomers of PFOS (ng/mL)	MA	L	0.88	1.74
	Monomethyl branched isomers of PFOS (ng/mL)	OH	H	1.05	1.74
	Monomethyl branched isomers of PFOS (ng/mL)	OH	L	0.90	1.74
	Monomethyl branched isomers of PFOS (ng/mL)	NHA	H	1.06	1.74
PCPs	Urinary Benzophenone-3 (μg/L)	NHW	L	13.43	45.48
	Urinary Benzophenone-3 (μg/L)	NHB	H	13.62	45.48
	Urinary Benzophenone-3 (μg/L)	NHB	L	9.90	45.48
	Urinary Benzophenone-3 (μg/L)	NHA	L	7.97	45.48
	Urinary Bisphenol A (μg/L)	NHA	L	0.60	1.17
	Urinary Bisphenol F (ug/L)	NHA	H	0.28	0.60
	Urinary Bisphenol F (ug/L)	NHA	L	0.27	0.60
	Urinary Triclosan (μg/L)	NHW	L	7.13	12.04
	Urinary Triclosan (μg/L)	NHA	L	4.78	12.04
PAHs	1-Hydroxypyrene	NHA	H	86.49	117.98
VOCs	Urinary N-Acetyl-S-(N-methylcarbamoyl)-L-cysteine	NHA	H	61.44	124.00
	Urinary N-Acetyl-S-(N-methylcarbamoyl)-L-cysteine	NHA	L	52.00	124.00
	Blood Toluene (ng/mL)	NHA	L	0.05	0.08
	Blood m-/p-Xylene (ng/mL)	NHA	L	0.04	0.06

Table 5. Comparison of geometric means from 2008 to 2013/2014 NHANES cycle

Chemical Class	Biomarker (concentration, matrix)	Race/Ethnicity	Income	GMR	
				2008	2014
Cotinine	Cotnine (ng/mL, serum)	NHB	Low	1.7	1.58
	Cotnine (ng/mL, serum)	NHW	Low	1.3	0.99
Metals	Antimony (ug/L, urine)	NHB	High	0.082	0.06
	Antimony (ug/L, urine)	NHB	Low	0.088	0.07
	Lead (ug/dL, blood)	NHB	Low	1.5	0.93
	Lead (ug/dL, blood)	NHB	Low	0.67	No detect
	Thallium (ug/L, urine)	NHB	High	0.18	0.18
Phenols	2,4-Dichlorophenol (ug/L, urine)	NHB	High	1.8	0.90
	2,4-Dichlorophenol (ug/L, urine)	NHB	Low	1.7	1.21
	2,5-Dichlorophenol (ug/L, urine)	NHB	High	28	5.98
	2,5-Dichlorophenol (ug/L, urine)	NHB	Low	28	12.35
Parabens	Methyl Paraben (ng/mL, urine)	NHB	High	170	106.58
	Methyl Paraben (ng/mL, urine)	NHB	Low	140	113.03
	Propyl Paraben (ng/mL, urine)	NHB	High	24	14.94
Phthalates	Mono-ethyl Phthalate (ng/mL, urine)	MA	Low	180	45.92
	Mono-ethyl Phthalate (ng/mL, urine)	NHB	High	280	71.39
	Mono-ethyl Phthalate (ng/mL, urine)	NHB	Low	240	72.68
	Mono-isobutyl Phthalate (ng/mL, urine)	MA	Low	10	9.34
	Mono-isobutyl Phthalate (ng/mL, urine)	NHB	Low	11	11.80
	Mono-n-butyl Phthalate (ng/mL, urine)	NHB	Low	27	12.99

Discussion

Analyzing biomarker concentrations in subgroups by race, ethnicity, and ancestry and income can shed light on complicated dynamics that and use-patterns that may result in disproportionate exposures. Understanding this disparity is paramount to understanding, targeting, and mitigating risk in specific subgroups. If researchers understand which exposures are likely to be present in certain groups, they may be able to better protect the health and well-being of vulnerable individuals. Many are more familiar with disproportionate exposure on the basis of income. The results of this analysis, however, indicate that race is a more important determinant in biomarker concentration levels than income. When we controlled for income (i.e. look across each income category), differences in exposure by race remained. For example, bisphenol A (BPA) and bisphenol S (BPS) are elevated in the NHB-L subgroup, but not the NHW-L subgroup.

The high percentage of disparate exposures unique to the NHB subpopulation suggests that larger dynamics at play most seriously affect this unique population. An analysis of potential sources for these exposures may shed light onto such dynamics. Four probable exposure sources are discussed below: smoking exposure, housing-based exposure, dietary exposure, and personal care product use exposure.

Smoking

Elevated concentrations of several biomarkers indicate exposure to cigarette smoke, either from personal smoking, or second hand some inhalation in the home. The most commonly cited smoking biomarker is cotinine, though thiocyanate [32], metabolites of fluorene (3-

hydroxfluroene and 2-hydroxylfluorene) [33], 2-naphthol [34], and several VOC metabolites can also be used as smoking biomarkers.

In our work, cotinine and 2-naphthol levels were elevated in the NHB-L, NHW-L, and MA-L subgroups. Urinary 2-Aminothiazoline-4-carboxylic acid (ATC), a biomarker for cyanide, is elevated in both the Mexican American and NHB-L subpopulations. Additionally, thiocyanate and fluorene were elevated solely in the NHB-L subpopulation.

These data are consistent with prior research indicating that low-income people, particularly those living in public housing, smoke at higher rates than high-income non-Hispanic whites [35] For example, Galvez et al. [36] found an association between residing in low-income housing and cotinine levels. Other studies have found that tobacco advertising campaigns target low-income areas [37] and that there are higher densities of tobacco retailers in low-income neighborhoods [38]. Additionally, studies have shown that individuals living in low-income neighborhoods may not have informal social control to decrease “bad” behaviors such as smoking [39]. This phenomenon, as well as the increased marketing and prevalence of tobacco stores in low-income areas, may explain these observed disparities.

Housing

Housing based exposures include sources such as polyvinyl chloride flooring (phthalates), indoor insecticides (2,5-dichlorophenol), in-home insulation (VOCs) and vehicular traffic in urban areas (Metals, PAHs, and VOCs). All of the biomarkers associated with housing-based exposures were elevated only in the NHB-L subpopulation.

Three of the detected phthalate metabolites are associated with in-home sources (DBP, DEHP, BBP). Indoor air and dust are thought to be major sources of exposure to DBP (monobutyl phthalate -- dibutyl phthalate).The highest concentrations of DBP are measured in

city air, or in homes with new carpets or heavy nail polish use [40]. A separate analysis found high levels in rooms recently covered with polyvinyl chloride tiles [40], and a New York study found that DBP likely has building-related sources [41]. BBP (Mono-benzyl phthalate -- Benzylbutyl phthalate) and DEHP (MEOH -- Di(2-ethylhexyl) phthalate)) also have known in-home sources such as wall coverings, floor tiles, furniture upholstery, shower curtains, some toys, automobile upholstery and packaging film and sheets [29]. This suggests that the disparity in phthalate exposure may arise due to differences in living situations amongst subgroups.

Urban areas have more vehicular traffic and high density of industrial activity. Thus, individuals who live in urban areas may have higher biomarker concentrations for chemicals commonly associated with vehicle operation or industrial activity. Antimony, copper, and phenanthrene associated with the combustion of fossil fuels (i.e. vehicular operation) [42] [43] [44] and the volatile organic chemical (VOC) biomarkers for acrylamide, toluene, acrolein, 1,3-butadiene, and ethyl benzene which are associated with vehicular traffic were all flagged for environmental justice concern. Tungsten, molybdenum and the VOC biomarkers for acrylonitrile, associated with industrial activity, were also flagged for environmental justice concern. Disparities in exposure to acrylamide, toluene and acrolein were also detected in NHB-H populations. The VOC biomarker for acrylonitrile was also elevated for the low-income Non-Hispanic White subpopulation.

Individuals in the minority (especially Black) populations are more likely to live in high-density poor urban neighborhoods than whites, and the difference is not explained by differences in income alone [39]. Instead, this disparity can be better explained by policies like red lining and restrictive covenants that were put in place decades ago to control where people of color could and could not live [45]. Red lining was a policy that prevented people of color from

acquiring mortgages and thus prevented them from building intergenerational wealth [46]. Restrictive covenants were contracts drafted by homeowners associations that prohibited people of color from occupying specific buildings [47]. Although those policies are no longer actively enforced, their influence on housing segregation seems to significantly influence exposures, particularly for the NHB-L people.

Dietary

Biomarkers associated with decreased access to fresh food (tin, phenols, phthalates) were unique to the NHB-L subpopulation, while compounds commonly associated with seafood exposures (mercury) were unique to the non-Hispanic Asian subpopulations.

Measures of total mercury include methylmercury, that is often from the diet, inorganic mercury, and elemental mercury. According to the CDC, measures of blood mercury are most important to evaluate exposures from interior latex paints [48]. However, mercury exposure can come from a wide range of sources including light bulbs, old medical products, and dental amalgam [49]. The most important source, however, is likely a diet high in seafood (apex fish predators, shellfish, etc.), are at a higher risk of methylmercury exposure because methylmercury accumulates up the food chain. The CDC found that non-Hispanic Asian adults consume significantly higher amounts of seafood per week than other race/ethnicity/ancestry groups [50]. This consumption pattern may explain the high levels of detected mercury.

Phenols flagged for environmental justice concern include bisphenol A (BPA), bisphenol S (BPS). BPA and BPS are found in similar products, as BPA has been slowly phased out and replaced with BPA [51]. Increases in BPA and tin exposure have been associated with reduced access to fresh food and low food security. A large portion of emergency food assistance is canned goods [52]. Consistent with our findings, other studies have cited racial disparities in

biomarker concentrations of BPA [53, 54]. Although environmental exposures form tin can be the result of a number of sources, including industrial processing, the most significant source of tin is the consumption of canned foods. One study found a significant relationship between consumption of canned goods, especially canned fruits, and urinary tin concentration [55].

Phthalate parent compounds DEHP (Mono(2-ethyl)-hexyl – (Di(2-ethylhexyl) phthalate) and DiP (Mono-Isobutyl -- Di-isobutyl phthalate) are also thought to be sourced mainly through food [56, 57]. Adamkiewicz et al. [19] point to fast food as a possible source of DEHP exposure, though the results of the study need to be confirmed.

Interestingly, disparity in compounds associated with dietary exposures (phenols, phthalates, tin), may also be explained by housing segregation. The phenols, phthalates, and tin associated with dietary exposure are correlated with decreased access to fresh food. In a comprehensive review of food desert literature, Walker et al. [58] noted that several studies show that black neighborhoods have fewer grocery stores than their white counterparts, even when controlling for income. Thus, the NHB-L subpopulation must rely more heavily on food assistance, pre-packaged food, and fast food – all of which have been associated with increased chemical exposures.

Personal Care Products

Paraben exposure disparity was unique to the non-Hispanic Black subpopulation. Parabens are a class of compounds commonly associated with personal care product use, though metals and phthalates may also play a role in these exposures. Methyl paraben and propyl paraben, the biomarkers elevated in the non-Hispanic Black subpopulations, are two of the most common parabens used [59] in personal care products, and researchers have posited that disparity in exposure is attributable to differences in personal care product use. This disparity

was observed regardless of income classification. Colorism, the perception that whiteness/lightness is associated with power, wealth, and beauty [60], may subconsciously influence the personal care products that people of color elect to utilize. For example, women of color may use hair relaxing creams to achieve straight hair, a look that can be attributed to the white standard of beauty. The same trend was observed for mono-ethyl phthalate (MEP), a metabolite of a phthalate utilized to manufacture of personal care products. Di-ethyl phthalate (MEP parent compound) has been reported in the formulas of over 60 different cosmetic products [61]. Zota & Shamasunder [62] found similar trends for multiple beauty products related chemical exposures

Unfortunately, we do not have detailed information on personal care product use and were unable to evaluate the contribution of these products to exposure; however, previous research has linked personal care product use with higher body burdens of parabens, suggesting a possible explanation for observed patterns.

Compounds with unclear trends

Several of the detected biomarkers – cadmium, manganese and arsenic – have a number of potential sources that are not easily attributed to one of the four hypothesized exposure scenarios. Cadmium, Manganese (blood), and arsenic metabolites were elevated for both Mexican American and non-Hispanic Asian subpopulations. Cadmium exposure can occur through a number of sources, including fertilizers, plastics, pigments, and sewage sludge [63]. Manganese is most commonly used for the production of iron and steel [48]. Manganese exposure can also be the result of certain pesticides, or as a fuel additive in gasoline [64]. Arsenic is a naturally occurring metal ubiquitous in the environment. Exposures can occur through crops grown in arsenic soil, drinking water (particularly wells drilled in arsenic-

containing rocks), personal care products where arsenic is used as a preservative, and cigarette smoke [65]. It should be noted that not all compounds will follow the hypothesized exposure pathways. However, the observed disparity for these compounds may have important exposure pathways that should be analyzed in future research.

Developing Trends

As evidenced in Table 5, the observed geometric means for all compounds have decreased from 2008 to 2014. However, for several compounds, particularly those unique to the NHB-L subgroup, the geometric mean ratios have increased (e.g. The two most significant examples of this change are observed for 2,5-dichlorophenol and cotinine. Deeper analysis into these trends reveals that although exposures are decreasing for all groups, there is much more significant reduction in the NHW-H subgroup. This results in an increased GMR in 2014 compared to previous NHANES cycles. Interestingly, the same trend does not hold for the NHB-H subgroup; both the geometric mean and GMR for 2,5-dichlorophenol (as well as other compounds) have decreased from 2008 – 2014. This suggests that there are dynamics at play, significant only for the NHB-L subgroup that prevent exposures from decreasing to the same extent as other populations.

Conclusions & Limitations

There are several limitations to this study design. First, although this study included large sample size, further stratifying the analysis by age and by sex was not possible without sacrificing statistical power. A finer analysis may shed light on which people are truly at risk. However, because many disparities seem to be related to housing, sex and age may not play a

significant role. Second, biomarker concentrations only provide a snapshot of time. Variability of biomarker concentrations over time has been well documented in the literature. That being said, the large sample size likely captures and corrects for any normal variability in each population. Finally, this analysis may not detect class instances of environmental injustice (i.e. disproportionate siting), or, if the sampling counties are home to a poorly sited industrial facility, may overestimate exposures for particular groups.

It is important to note that exposure does not always necessarily equate to harm. Most chemical biomarkers provide a measure of internal chemical concentration for a snapshot in time, as many chemicals metabolize and are eliminated rapidly from the human body. Although biomarkers reflect the toxicokinetics of a given chemical (i.e. how an individual absorbs, distributes, metabolizes, and excretes the chemical) [[66] [67]], the detected presence of the chemical does not indicate a link to disease [26]. This does not negate the importance of the disparity highlighted in this study. Many of the compounds that have been flagged for environmental justice concern and are also known to be toxic to humans and expert panels have been organized to determine the percentage of disease incidence that can be attributed to exposures to various endocrine disrupting chemicals [68]. It is, however, beyond the scope of this analysis to consider the health implications of the observed disparities.

In summary, although there is disparity observed amongst several of the observed subgroups, the disparity is most pronounced for the NHB-L subpopulation. This may be due to housing patterns, and the availability of resources in neighborhoods where POC typically reside. The results suggest that more research should be done to understand the dynamics of health, place, and race.

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