Neighborhood Factors and Urinary Metabolites of Nicotine, Phthalates, and Dichlorobenzene

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abstract

BACKGROUND AND OBJECTIVES: Exposures to environmental chemicals are ubiquitous in the US. Little is known about how neighborhood factors contribute to exposures.

METHODS: Growing Up Healthy is a prospective cohort study of environmental exposures and growth and development among Hispanic and African American children (n = 506) in New York City. We sought to determine associations between neighborhood-level factors (eg, housing type, school, time spent indoors versus outdoors) and urinary biomarkers of chemical exposures suspected to be associated with these characteristics (cotinine, 2, 5-dichlorophenol, and phthalate metabolites) adjusted by age, sex, race, and caregiver education and language.

RESULTS: Urinary cotinine concentrations revealed a prevalent exposure to secondhand smoke; children living in public housing had higher concentrations than those in private housing. In homes with 1 smoker versus none, we found significant differences in urinary cotinine concentrations by housing, although not in homes with 2 or more smokers. Children in charter or public schools had higher urinary cotinine concentrations than those in private schools. School type was associated with exposures to both low- and high-molecular-weight phthalates, and concentrations of both exposure biomarkers were higher for children attending public versus private school. 2,5-Dichlorophenol concentrations declined from 2004 to 2007 (P = .038) and were higher among charter school children.

CONCLUSIONS: Housing and school type are associated with chemical exposures in this minority, inner city population. Understanding the role of neighborhood on environmental exposures can lead to targeted community-level interventions, with the goal of reducing environmental chemical exposures disproportionately seen in urban minority communities.

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Through biomonitoring conducted by the Centers for Disease Control and Prevention (CDC) for the National Report on Human Exposure to Environmental Chemicals, it is demonstrated that environmental chemical exposures are widespread in the US population.[1] Many of these exposures may be higher in children than adults, with children ages 6 to 11 years often having higher biomarker concentrations than their adult counterparts, whereas adolescents have intermediate concentrations.[1] Certain biomarker concentrations are higher in racial and ethnic minority groups compared with non-Hispanic white individuals.[1] In addition to sociodemographic factors, the contribution of individual behaviors, such as smoking frequency or personal care product use, and their respective contributions to exposure have also been examined.[2–5] Thus, what is known about exposure sources has focused largely on individual factors. For these reasons, much of the clinical counseling on exposure reduction has targeted behavioral changes at the individual level.

In emerging research, authors have identified how exposures might be magnified by neighborhood-level factors, including housing or school type.[6–8] This is rooted in the concept of environmental justice, which arose from the knowledge that persons living in high poverty communities can have higher exposures.[9–11] This is true whether we are considering established chemicals of concern, such as nicotine, or emerging chemicals of concern, including those classified as potential endocrine disruptors (EDs). EDs are chemicals that may influence hormone action and thus have the potential to disrupt the endocrine system.[12] Researchers have demonstrated a potential role of EDs such as phthalates in a wide variety of health conditions from reproductive effects to alteration of growth and neurodevelopment.[12]

We sought to determine associations between neighborhood-level factors and chemicals that are both potential respiratory irritants and suspected to be associated with neighborhood characteristics in inner city, low income, and minority girls and boys using urinary concentrations of cotinine, phthalate metabolites, and 2,5-dichlorophenol. Cotinine is the primary metabolite of nicotine.[1] Phthalates are a class of chemicals used to make plastics more flexible and durable. Phthalates can be found in products such as vinyl flooring, adhesives, detergents, lubricating oils, automotive plastics, toys, plastic clothes (eg, raincoats), medical tubing, and personal care products (eg, soaps, shampoos, hair sprays, and nail polishes; see Supporting Information, Supplemental Table 6).[1] 2,5-Dichlorophenol is a metabolite of 1,4-dichlorobenzene (paradichlorobenzene), which can be found in mothballs and room and toilet deodorizers.[1] Environmental exposures to these chemicals or their precursors by neighborhood factors in our sample compared with the US general population in NHANES may in part contribute to disparities in adverse childhood health conditions.[13]

METHODS

Growing Up Healthy in East Harlem is a community-based, prospective cohort study in which researchers examine environmental factors and their influence on the growth and development of boys and girls ages 6 to 8 years. Children (n = 506) were recruited from 2005 to 2007 from East Harlem and the greater New York City area, and recruited subjects were predominantly girls because of a parallel study of puberty in girls. Informed consent was obtained from a parent or guardian along with child assent, and the study was approved by the institutional review boards of Mount Sinai and the CDC. Eligibility included age (6–8 years), having no underlying endocrine medical conditions, and African American or Hispanic race and/or ethnicity.

Urinary concentrations of cotinine, phthalate metabolites, and 2, 5-dichlorhophenol were measured at the CDC and analyzed, correcting for creatinine.[14,15] Cotinine levels, however, are only available for girls because of the parallel study of puberty in girls, which allowed for these additional urinary analyses. We examined molar sums of the phthalate metabolites as low- and high-molecular-weight compounds on the basis of their usual sources of exposure[16] to reduce multiple comparisons and stabilize the variance. We fit multivariable models predicting geometric means (GMs) of biomarker concentrations according to neighborhood variables. We considered additional covariates as potential confounders that should be controlled for in the analysis. We included child age, sex, and race in the models. Additional covariates were retained by using backward elimination if they altered estimates for the neighborhood variables by >10%. We also verified that exclusion of a covariate did not degrade the precision of neighborhood estimates. Final models that included >1 neighborhood variable were further examined with each variable singly. In addition, because charter schools may have unique population characteristics that distinguish them from either private or public school populations, final models with school type as a predictor were further tested for the influence of charter schools (N = 23 children). We tested interactions by including in the models the cross products for biologically plausible effect modification; for example, we included housing by smoking and school type by year of urine collection.

RESULTS

Our cohort of inner city minority children ages 6 to 8 years were mainly from East Harlem in New York

City (70%). They were predominantly Hispanic (61%), English-speaking (59%), from nonsmoking households (65%), with caregivers who had less than a high school education (61%), living in privately owned housing (61%), and attending public school (89%) (Table 1). Urine specimens and interview data were collected mostly during the school year (Table 1). Boys and girls had similar characteristics, except boys were recruited mainly between 2005 and 2006, whereas some girls were recruited between 2004 and 2007.

We observed significant differences between the biomarker GMs ($P \leq .1$) and school type, public versus private housing ownership, and more outside activity time. Neighborhood variables that showed no associations with biomarker levels were not further examined (owning versus renting a home, number in household, number of rooms in home, number of plastic household items, use of an afterschool program, urine collection site, neighborhood safety perception).

The urinary cotinine concentrations in girls revealed prevalent exposures to secondhand smoke (median 1.3 μg/g creatinine compared with <0.5 μg/g creatinine in the NHANES (Tables 2 and 3).[1] Children who lived in public housing had higher urinary cotinine concentrations than those whose homes were in privately owned buildings. When reported household smoking was taken into account, this difference was significant among homes with 1 smoker (GM = 4.2 vs 1.9 μg/g respectively); there were no cotinine differences in children who lived in public or private homes with 2 or more smokers. Children in charter and public schools had higher cotinine concentrations than those in private schools, even after adjustment for demographics.

Urinary phthalate metabolites (Supplemental Table 6), both high-molecular-weight phthalates (HMWPs) and low-molecular-weight phthalates (LMWPs), in this cohort were higher than among 2005 to 2006 NHANES children (medians shown in Table 2), which is consistent with the disproportionate burden of environmental exposures often seen in high poverty, predominantly minority populations.[1] Boys and girls also had similar concentrations of urinary biomarkers. Phthalate metabolites differ by demographic factors including across race-specific subgroups. For example, LMWP concentrations in children in NHANES from 2005 to 2006 were lower than in adults, but monoethyl phthalate, the major LMWP, was higher among African American and Hispanic children.[1]

HMWPs and LMWPs were both higher in children who attended public school compared with private school or charter school; however, the difference was only significant for HMWPs in public school children when compared with private school children (Table 4). Increasing time spent in outdoor activity was associated with decreasing concentrations of HMWPs and increasing concentrations of LMWPs, but neither association remained significant in adjusted models ($P > .1$). In addition, LMWP concentrations were ~30% higher among samples collected in summer. Models in which researchers used only 1 of these factors to predict LMWPs or HMWPs were similar to those presented in Table 3. Removing charter schools from these models did not change the findings.

Urinary 2,5-dichlorophenol concentrations were elevated in the children in our study compared with the US sample. Median concentrations (overall median 68 μg/g creatinine) were twice as high as NHANES 75th percentile (28 μg/g creatinine in 2005–2006) (Table 2). 2,5-Dichlorophenol concentrations declined over the years the samples were collected ($P = .038$ for adjusted model; see Table 5). 2,5-Dichlorophenol was higher among children in charter schools and in homes with no smokers. An apparent relationship of 2,5-dichlorophenol with seasonality was no longer significant after adjustment for covariates.

Interactions between housing and smoking as well as school type and year of urine collection for all urinary biomarkers were not significant (data not shown).

DISCUSSION

We report associations between neighborhood characteristics, specifically, type of housing where a child lives, type of school a child attends, and amount of time spent outdoors, with exposures to chemicals or their precursors known to be high in our study cohort, including cotinine, LMWPs and HMWPs, and 2,5-dichlorophenol. A strength of this analysis is the availability of both descriptive neighborhood-level data along with individual level biomarker data, which allows for a detailed assessment of neighborhood factors unique to the urban built environment and its potential role in every day chemical exposures.

Urinary cotinine concentrations revealed a prevalent exposure to secondhand smoke; girls living in public housing had higher concentrations than those in privately owned housing. Secondhand smoke exposures in multiunit housing have been documented despite implementation of smoke-free housing policies.[17,18] In homes with 1 smoker versus none, we found significant differences in urinary cotinine concentrations by housing type, although these differences by housing type were not seen in homes with 2 or more smokers. Girls in charter or public

TABLE 1 Participant Characteristics ($N = 506$)

Characteristics	Variable	Category	All	%	Girls	%	Boys	%	χ^2 *P*
			n		*n*		*n*		
Individual characteristics	Language of interview	English	300	0.59	248	0.61	52	0.51	.07
		Spanish or Spanish and English	206	0.41	157	0.39	49	0.49	
	Child's race and/or ethnicity	African American	135	0.27	111	0.27	24	0.24	.69
		Hispanic	307	0.61	242	0.6	65	0.64	
		Hispanic African American	64	0.13	52	0.13	12	0.12	
	Age at baseline questionnaire (in y)	6–6.99	189	0.37	151	0.37	38	0.38	.92
		7–7.99	159	0.31	126	0.31	33	0.33	
		8–8.99	158	0.31	128	0.32	30	0.3	
	Caregiver's education	≤ HS diploma	303	0.61	235	0.59	68	0.69	.06
		≥ Some college	192	0.39	162	0.41	30	0.31	
Neighborhood characteristics: housing	Public or private housing	Privately owned housing	294	0.61	238	0.62	56	0.58	.53
		Public housing	187	0.39	147	0.38	40	0.42	
	No. HH smokers	0 HH smokers	328	0.65	258	0.64	70	0.69	.11
		1 HH smokers	124	0.25	98	0.24	26	0.26	
		2+ HH smokers	54	0.11	49	0.12	5	0.05	
Neighborhood characteristics: school	Type of school child attends	Charter school	23	0.05	22	0.05	1	0.01	.07
		Private school	33	0.07	29	0.07	4	0.04	
		Public school	450	0.89	354	0.87	96	0.95	
	Y of urine sample collection	2004	20	0.04	20	0.05	0	0	<.001
		2005	153	0.3	117	0.29	36	0.36	
		2006	230	0.45	169	0.42	61	0.6	
		2007	103	0.2	99	0.24	4	0.04	
	In school yesterday	In school yesterday	277	0.55	222	0.55	55	0.54	.89
		Not in school yesterday	226	0.45	180	0.45	46	0.46	
Indoor time	≥ 3 h/wk in outside play	< 3 h outside in play per wk	258	0.51	213	0.53	45	0.45	.14
		≥ 3 h outside in play per wk	248	0.49	192	0.47	56	0.55	
	≥8.5 (median) of sedentary activity per d	< 8.5 h of sedentary activity per wk	242	0.48	190	0.47	52	0.51	.41
		≥ 8.5 h of sedentary activity per wk	264	0.52	215	0.53	49	0.49	
	D of week yesterday	Weekday yesterday	423	0.84	341	0.84	82	0.81	.47
		Weekend yesterday	83	0.16	64	0.16	19	0.19	
	Season of urine collection	Fall and winter: September to February	250	0.49	203	0.5	47	0.47	.96
		Spring: March to June	185	0.37	145	0.36	40	0.4	
		Summer: July to August	71	0.14	57	0.14	14	0.14	
Total			—	—	405	—	101	—	—

HH, household; HS, high school; —, not applicable.

TABLE 2 Median Concentrations of Urinary Metabolites in Growing Up Healthy Children, 2004 to 2007 (μg/g Creatinine)

	NHANES	All			Girls			Boys		
	Child Median (2005–2006)	*n*	Median	IQR	*n*	Median	IQR	*n*	Median	IQR
Cotinine (girls)	<0.5[a]	386	1.3	0.6–4.7	386	1.3	0.6–4.7	—	—	—
HMWPs[b]	~150	506	281	158–500	405	270	149–479	101	316	185–583
Mono-benzyl phthalate[b]	35.6	506	26	13–54	405	24	13–48	101	35	21–71
Mono-(3-carboxypropyl) phthalate	5.06	506	5.1	3.4–8.9	405	5.1	3.3–9.4	101	5.2	3.6–8.1
Sum di-(2-ethylhexyl) phthalate	~120	506	212	113–420	405	206	110–410	101	242	131–471
Mono-(2-ethyl-5-carboxypentyl) phthalate	54.2	506	110	62–215	405	110	61–208	101	117	69–240
Mono-2-ethylhexyl phthalate	3.26	506	6.2	3.2–12.9	405	6	3.1–12.6	101	6.8	3.7–13.9
Mono-(2-ethyl-5-hydroxyhexyl) phthalate	37	506	68	35–133	405	66	33–128	101	74	40–156
Mono-(2-ethyl-5-oxohexyl) phthalate	24.4	506	43	22–84	405	41	21–82	101	51	26–98
LMWPs	~70	506	212	136–405	405	216	135–409	101	198	141–368
Mono-ethyl phthalate	75.9	506	116	67–261	405	117	67–287	101	98	67–184
Mono-isobutyl phthalate	9.46	506	22	14–36	405	22	13–36	101	23	15–36
Mono-n-butyl phthalate	33.9	506	63	39–97	405	61	38–95	101	73	46–113
2,5-Dichlorophenol[b]	8[c]	505	68	31–189	405	64	30–160	100	79	33–259

See Supplemental Table 6 phthalates. IQR, interquartile range; —, not applicable.

[a] Urine test as 5*serum cot: 0.050; 75th percentile, 0.22; 90th percentile, 1.22.

[b] Wilcoxon rank test for boys and girls differ, $P < .05$.

[c] 75th percentile: 24.7 μg/g creatinine.

schools had higher urinary cotinine concentrations than those in private schools. Both housing and school type are associated with chemical exposures in this minority, inner city population. These findings are of particular concern given the higher prevalence of asthma in children living in public housing, providing further support for the need for environmental interventions partnered with individualized medical treatment to reduce asthma exacerbations.[19]

Although in an emerging body of literature researchers have demonstrated an association between housing factors and phthalate exposures, such as indoor dust concentrations of phthalates, and presence of polyvinylchloride products, or vinyl flooring,[20,21] we did not see differences between broader neighborhood characteristics, including public or private housing type and urinary concentrations of phthalate biomarkers. With these findings, we suggest that for this particular population, housing type is not a good surrogate for indoor characteristics that have been associated with phthalate exposures in previous studies. An important limitation, however, is that the population characteristics of our study may not capture the full variability in public versus private housing that one typically sees across the economic spectrum given that our population is almost entirely low income. A second limitation is the lack of environmental exposure data for both homes and schools to further assess their contribution to children's exposure levels.

We originally hypothesized that time outdoors would reduce environmental chemical exposures, reflecting exposure sources in the indoor setting to a variety of

TABLE 3 Relationships Between Urinary Cotinine Concentrations (μg/g Creatinine) and Neighborhood Characteristics

Cotinine (*n* = 386 Girls)	*n*	Unadjusted GMs (μg/g Creatinine)	*P*	*n*	Adjusted GMs (μg/g Creatinine)	*P*
Private housing						
0 smoker	213	1.0 (0.8, 1.2)	.006*	208	0.9 (0.7, 1.2)	<.01*
1 smoker	59	2.5 (1.8, 3.4)	—	59	1.9 (1.3, 2.9)	—
2+ smokers	22	11.7 (6.7, 20.2)	—	22	7.0 (3.9, 12.4)	—
Public housing						
0 smoker	100	0.9 (0.7, 1.2)	—	97	0.7 (0.5, 0.9)	—
1 smoker	56	6.9 (4.8, 9.9)	—	56	4.2 (2.6, 6.6)	—
2+ smokers	31	10.4 (6.8, 16.0)	—	29	6.1 (3.7, 10.1)	—
School						
Charter	23	3.21 (1.70, 6.06)	.01*	21	2.9(1.7, 5.1)	.06
Private	33	0.93 (0.54, 1.61)	—	29	1.7(1.1, 2.7)	—
Public	450	1.86 (1.59, 2.18)	—	421	3.0(2.5, 3.5)	—

Adjusted for child age, race, sex, caregiver education, and Spanish language of caregiver. —, not applicable.
* $P < .05$.

TABLE 4 Relationships Between Urinary Phthalate Metabolite Concentrations (μg/g Creatinine) and Neighborhood Characteristics (*N* = 506)

	Neighborhood Characteristics	*n*	Unadjusted GMs	*P*	*n*	Adjusted GMs	*P*
HMWPs	School		μg/g creatinine			μg/g creatinine	
	Charter	23	269 (186 387)	.009*	23	301 (208 437)	.01*
	Private	33	191 (141 259)		33	201 (148 273)	—
	Public	450	311 (286 338)		447	321 (291 355)	—
	Hours outside in play per week						
	<3 h	258	324 (290 362)	.04*	255	287 (236 349)	.11
	≥3 h	248	275 (246 308)		248	252 (210 303)	
LMWPs	School						
	Charter	23	192 (136 272)	.06	22	184 (126 268)	.10
	Private	33	190 (142 254)		33	190 (139 258)	—
	Public	450	254 (235 275)		437	245 (215 279)	—
	Hours outside in play per week						
	<3 h	258	231 (208 256)	.09	251	194 (157 241)	.22
	≥3 h	248	263 (236 292)		241	215 (178 260)	—
	Season sample collected						
	Fall and winter: September to February	250	236 (212 262)	.008*	245	189 (156 229)	.04*
	Spring: March to June	185	233 (206 263)		179	182 (148 223)	—
	Summer: July to August	71	329 (271 401)		68	249 (190 325)	—

Adjusted for child age, race, and sex. HMWPs were further adjusted for having been a weekday. LMWPs were adjusted for caregiver education and Spanish language. —, not applicable.
* $P < .05$.

chemicals. However, in adjusted models, no significant relationships were seen with time outdoors.

School factors were associated with a number of exposures that we assessed, including attendance at public schools versus charter or private schools. Schools differ on a number of factors, from age of building to renovation status to years in operation to class composition, all of which may potentially play a role in why school type influenced exposure levels.[22] It is further possible that school type (public versus private versus charter) is a surrogate marker for socioeconomic status and hence may in part explain socioeconomic status differences on a population level rather than neighborhood level, although the possibility of an association because of chance cannot be excluded.[22]

Notably, urinary cotinine[1] and 2, 5-dichlorophenol[23] concentrations declined in general over the course of the study years, and these trends were also noted in NHANES from 2003 to 2010. A 2004 ban on use of paradichlorobenzene, the precursor of 2,5-dichlorophenol, in school buildings[24] and the ban on smoking in indoor public spaces and certain outdoor areas in New York City[25] may in part account for these trends, demonstrating the importance of public policy in reducing exposures.

CONCLUSIONS

Understanding existing health disparities commonly seen in low income, predominantly minority communities requires an enhanced understanding of neighborhood-level contributors to health.[7–9] This is especially true for environmental exposures,

TABLE 5 Relationships Between Urinary 2,5-Dichlorophenol Concentrations (µg/g Creatinine) and Neighborhood Characteristics *N* = 506

Neighborhood Characteristics	*n*	Unadjusted GMs (µg/g Creatinine)	*P*	*n*	Adjusted GMs (µg/g Creatinine)	*P*
School						
Charter	23	211 (115–390)	.02*	22	197 (99–394)	.07
Private	33	200 (148–270)	—	33	76 (43–134)	—
Public	450	87 (76–100)	—	440	99 (74–131)	—
Household smokers						
0 smoker	328	100 (85–118)	.10	320	146 (101–210)	.03*
1 smoker	124	72 (55–94)	—	124	99 (65–153)	—
2+ smokers	54	81 (54–121)	—	51	102 (61–170)	—
Year sample collected			—			
2004	20	177 (92–342)	.06	20	185 (89–387)	.19
2005	153	95 (75–121)	—	149	108 (74–156)	—
2006	230	93 (77–113)		226	100 (69–144)	—
2007	103	69 (52–92)	—	100	84 (53–134)	—
Season sample collected						
Fall and winter: September to February	250	106 (88–127)	.04*	246	129 (89–186)	.25
Spring: March to June	185	73 (59–91)	—	181	100 (67–149)	—
Summer: July to August	71	90 (64–128)	—	68	114 (69–189)	—

Adjusted for child age, race, sex, caregiver education, year of urine collection, and whether yesterday was a weekday. —, not applicable.
* $P < .05$.

which are disproportionately higher in these same communities and are increasingly linked with a wide range of health outcomes, the most common of which includes respiratory conditions, such as asthma.[26,27] Enhanced understanding of neighborhood-level factors and their association with concentrations of exposure biomarkers can lead to targeted community-level interventions, with the goal of reducing the cumulative burden of environmental chemicals exposure disproportionately seen in urban minority communities.

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ABBREVIATIONS

CDC: Centers for Disease Control and Prevention
ED: endocrine disruptor
GM: geometric mean
HMWP: high-molecular-weight phthalate
LMWP: low-molecular-weight phthalate

The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the California Department of Public Health.

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