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RESEARCH ARTICLE

Are urinary polyaromatic hydrocarbons associated with adult hypertension, heart attack, and cancer? USA NHANES, 2011–2012

Ivy Shiue [1,2]

Abstract Links between environmental chemicals and human health have emerged over the last few decades, but the effects from polyaromatic hydrocarbons were less studied, compared to other commonly known environmental chemicals such as heavy metals, phthalates, arsenic, phenols and pesticides. Therefore, it was aimed to study the relationships of urinary polyaromatic hydrocarbons and adult cardiovascular disease and cancer using human sample in a national and population-based study in recent years. Data was retrieved from US National Health and Nutrition Examination Surveys, 2011–2012, including demographics, self-reported health conditions and urinary polyaromatic hydrocarbons. Statistical analyses included chi-square test, t test, survey-weighted logistic regression modeling and population attributable risk (PAR) estimation. Of 5560 American adults aged 20–80 and included in the statistical analysis, urinary polyaromatic hydrocarbons (representatively in one-third sample) were observed to be higher in people with cardiovascular disease and total cancer. In particular, urinary 4-hydroxyphenanthrene was associated with hypertension (odds ratio (OR) 1.33, 95 % confidence interval (CI) 1.00–1.76, $P=0.048$, PAR 5.1 %), urinary 1-hydroxypyrene was significantly associated with heart attack (OR 1.47, 95 %CI 1.05–2.06, $P=0.027$, PAR

1.7 %), and urinary 2-hydroxynapthalene (2-naphthol) was associated with cancer (OR 1.46, 95 %CI 1.12–1.90, $P=0.008$, PAR 3.9 %). Urinary polyaromatic hydrocarbons were associated with adult hypertension, heart attack and cancer, although the causality cannot be established. From the research perspective, future studies with a longitudinal or experimental approach would be suggested. From the law and public health perspectives, regulation on minimizing exposure to polyaromatic hydrocarbons might need to be considered in future health and environmental policies and intervention programs.

Keywords Polyaromatic hydrocarbons · Risk factor · Hypertension · Heart attack · Cancer · Angina

Introduction

Links between environmental chemicals and human health in American adults including hypertension, cardiovascular disease, food allergy, oral health, emotional support and cognitive function have emerged (Shiue 2013a, b, c, 2014a, b, 2015a, b), but the effects from polyaromatic hydrocarbons (PAHs) were less studied, compared to other commonly known environmental chemicals such as heavy metals, arsenic, phenols and phthalates. PAHs constitute a group of chemicals that people could be exposed via vehicle exhausts, asphalt, coal tar, wild fires, agricultural burning, soil, charbroiled foods and tobacco smoke (Centers for Disease Control and Prevention 2012). PAH pollution may have significant health implications, and the extent of damage to organisms from PAH exposure could be dependent on several factors including degree and type of PAH exposure (Ball and Truskewycz 2013). The potential for PAHs to adversely affect

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human immunologic health could have traditionally been evaluated in rodents, under laboratory conditions (Luebke, et al 1997). Providing evidence from human sample might help public health promotion in the next few years. Following this context, therefore, it was aimed to examine the relationships of urinary PAHs and adult cardiovascular disease by subtypes and cancer using a large human sample in a national and population-based setting in recent years.

Methods

Study sample

As described elsewhere (Centers for Disease Control and Prevention 2012), US National Health and Nutrition Examination Surveys (NHANES) has been a national, population-based, multiyear, cross-sectional study since the 1980s. Study sample is representative of the civilian, noninstitutionalized US population. Information on demographics (more details via http://wwwn.cdc.gov/nchs/nhanes/2011-2012/DEMO_G.htm), serum cotinine (more details via http://wwwn.cdc.gov/nchs/nhanes/2011-2012/COTNAL_G.htm), and self-reported health conditions (more details via http://wwwn.cdc.gov/nchs/nhanes/2011-2012/MCQ_G.htm) was obtained by household interview using questionnaires. In the current analysis, the 2011–2012 study cohort as the most recent wave was selected. Informed consents were obtained from the participating subjects by the NHANES researchers.

Biomonitoring

Urines were only collected in a subsample within 10 days of the household interview, being one third of the whole study cohort with representation (more details via http://www.cdc.gov/nchs/data/nhanes/nhanes_09_10/homeurine.pdf), to measure environmental chemical concentrations in urines among people aged 6 and above (more details via http://www.cdc.gov/nchs/nhanes/nhanes2011-2012/labdoc_g.htm). Urine specimens from urinary polyaromatic hydrocarbon were processed, stored under appropriate frozen (−20 °C) conditions, and shipped to the Division of Environmental Health Laboratory Sciences, National Center for Environmental Health, Centers for Disease Control and Prevention, for analysis. According to the NHANES website (more details via http://www.cdc.gov/nchs/data/nhanes/nhanes_11_12/PAH_G_met.pdf), the procedure involved enzymatic hydrolysis of glucuronidated/sulfated OH-polyaromatic hydrocarbon metabolites in urine, extraction, derivatization, and analysis using isotope dilution capillary gas chromatography tandem mass spectrometry (GC-MS/MS). Ion transitions specific to each analyte and carbon-13-labeled internal standards are monitored, and the abundances

Table 1 Characteristics of the included participants aged 20–80 ($n=5560$)

	Number (%)
Age	48.9±17.9
20–39	1957 (35.2 %)
40–59	1812 (32.6 %)
60–80	1791 (32.2 %)
Sex	
Male	2740 (49.3 %)
Female	2820 (50.7 %)
Body mass index	28.8±6.9
<18.5	1103 (1.9 %)
18.5–24.9	1577 (28.4 %)
25–29.9	1684 (30.3 %)
30+	2196 (39.5 %)
Ratio of family income to poverty	
0–4.9	4199 (75.5 %)
5+	1361 (24.5 %)
Education level	
Less than 9th grade	550 (9.9 %)
9–11th grade	782 (14.1 %)
High school graduate or equivalent	1169 (21.0 %)
Some college or AA degree	1657 (29.8 %)
College graduate or above	1397 (25.2 %)
Serum cotinine (ng/mL)	52.1±120.2
Alcohol status	
>12 drinks	3413 (72.8 %)
Less than 12 drinks	1275 (27.2 %)
Physical activity level	
Engaging moderately	2297 (41.3 %)
None	3262 (58.7 %)

of each ion are measured. Since urinary PAH concentrations were highly skewed, they were all log transformed when performing the statistical analyses.

Table 2 Prevalence of cardiovascular disease by subtypes in American adults ($n=5560$)

	Number (%)
Hypertension	770 (16.2 %)
Diabetes (borderline included)	821 (14.8 %)
Heart failure	187 (3.4 %)
Coronary heart disease	196 (3.5 %)
Angina	129 (2.3 %)
Heart attack	203 (3.7 %)
Stroke	229 (4.1 %)
Cancer	488 (8.8 %)
Blood transfusion	673 (12.2 %)

Table 3 Associations between 2-hydroxyfluorene (ng/L) and adult health (*n*=1670)

	Present	Absent	*P* value	OR (95 %CI)[a]	*P* value	Population attributable risk
Hypertension	647.2 (1081.8)	619.7 (1116.5)	0.772	1.18 (0.88–1.58)	0.252	–
Diabetes	557.0 (1022.6)	609.5 (1069.1)	0.466	0.98 (0.76–1.25)	0.845	–
Heart failure	486.3 (623.2)	605.4 (1073.1)	0.439	1.13 (0.68–1.88)	0.621	–
Coronary heart disease	793.0 (2183.8)	595.4 (1001.4)	0.164	0.83 (0.56–1.23)	0.333	–
Angina	491.1 (791.4)	604.1 (1066.7)	0.584	0.85 (0.61–1.18)	0.313	–
Heart attack	1028.6 (2266.0)	585.4 (987.4)	0.001	1.27 (0.73–2.22)	0.373	–
Stroke	580.0 (850.1)	602.2 (1068.5)	0.880	0.96 (0.63–1.47)	0.851	–
Cancer	551.6 (733.9)	605.6 (1086.0)	0.567	1.33 (0.89–1.97)	0.150	–
Blood transfusion	641.2 (1317.9)	593.9 (1016.1)	0.552	0.85 (0.69–1.06)	0.138	–

[a] Adjusted for urine creatinine, age, sex, body mass index, ratio of family income to poverty, education level, serum cotinine, alcohol habit, physical activity level, and subsampling weighting

Statistical analysis

Adults aged 20 and above were included in the current statistical analysis since chronic diseases were commonly reported in adults. Associations of urinary PAHs and adult health problems were examined by performing *t* test and survey-weighted logistic regression model, presenting with mean values, odds ratios (OR), and 95 % confidence intervals (CI). Covariates including urinary creatinine, age, sex, ratio of family income to poverty (proxy of socioeconomic status), body mass index, education level, serum cotinine (biomarker of smoking status), alcohol status and physical activity level were adjusted. In addition, population attributable risks from urinary PAHs, which significant associations were found, were calculated based on the formula introduced by Fleiss (1979). Statistical software Stata version 13.0 (Stata, College Station, Texas, USA) was used to perform all the analyses. Since the present study was only carrying out secondary data analysis by extracting data from the US NHANES website (more details via http://wwwn.cdc.gov/nchs/nhanes/search/nhanes11_12.aspx), no further ethics approval was required.

Results

Of 5560 American adults aged 20–80 and included in the statistical analysis, their characteristics are shown in Table 1. The presence of cardiovascular disease and cancer in the American adult population varied. They are shown in Table 2. In Tables 3, 4, 5, 6, 7, 8, 9, 10, 11, and 12, associations of 10 urinary PAHs and adult cardiovascular disease and cancer are presented separately. It was observed that urinary PAHs were higher in people with cardiovascular disease and total cancer. In particular, urinary 4-hydroxyphenanthrene was associated with hypertension (OR 1.33, 95 %CI 1.00–1.76, *P*=0.048, population attributable risk (PAR) 5.1 %), urinary 1-hydroxypyrene was significantly associated with heart attack (OR 1.47, 95 %CI 1.05–2.06, *P*=0.027, PAR 1.7 %), and urinary 2-hydroxynapthalene (2-naphthol) was associated with cancer (OR 1.46, 95 %CI 1.12–1.90, *P*=0.008, PAR 3.9 %). In addition, urinary 2-hydroxynapthalene (2-naphthol) was inversely associated with angina (OR 0.56, 95 %CI 0.33–0.95, *P*=0.034).

Table 4 Associations between 3-hydroxyfluorene (ng/L) and adult health (*n*=1670)

	Present	Absent	*P* value	OR (95 %CI)[a]	*P* value	Population attributable risk
Hypertension	322.5 (648.6)	307.1 (645.3)	0.779	1.18 (0.90–1.55)	0.207	–
Diabetes	242.1 (572.7)	305.2 (620.8)	0.130	0.96 (0.78–1.17)	0.657	–
Heart failure	209.5 (345.6)	298.6 (620.6)	0.318	1.07 (0.62–1.87)	0.796	–
Coronary heart disease	341.7 (842.9)	294.5 (605.2)	0.565	0.82 (0.55–1.22)	0.309	–
Angina	193.3 (319.7)	297.8 (618.0)	0.381	0.92 (0.63–1.34)	0.647	–
Heart attack	458.0 (914.3)	289.5 (599.4)	0.035	1.18 (0.69–2.04)	0.525	–
Stroke	301.3 (529.3)	295.5 (616.8)	0.946	1.00 (0.71–1.41)	0.986	–
Cancer	273.9 (458.6)	297.5 (625.9)	0.666	1.34 (0.92–1.94)	0.115	–
Blood transfusion	324.6 (766.1)	289.6 (581.1)	0.444	0.87 (0.66–1.15)	0.319	–

[a] Adjusted for urine creatinine, age, sex, body mass index, ratio of family income to poverty, education level, serum cotinine, alcohol habit, physical activity level, and subsampling weighting

Table 5 Associations between 9-hydroxyfluorene (ng/L) and adult health (*n*=1670)

	Present	Absent	*P* value	OR (95 %CI)[a]	*P* value	Population attributable risk
Hypertension	624.7 (1437.9)	518.6 (821.4)	0.169	1.27 (0.98–1.65)	0.073	–
Diabetes	483.4 (660.0)	525.4 (900.3)	0.475	0.93 (0.77–1.12)	0.419	–
Heart failure	448.3 (418.4)	518.5 (870.0)	0.574	1.04 (0.62–1.77)	0.863	–
Coronary heart disease	536.1 (868.6)	519.0 (869.2)	0.883	0.87 (0.65–1.17)	0.346	–
Angina	371.8 (295.7)	521.9 (874.9)	0.373	0.97 (0.71–1.34)	0.857	–
Heart attack	525.2 (682.7)	518.7 (874.4)	0.954	1.05 (0.68–1.62)	0.812	–
Stroke	556.1 (853.3)	517.9 (869.0)	0.751	1.03 (0.63–1.68)	0.900	–
Cancer	495.2 (686.8)	521.0 (882.4)	0.738	1.20 (0.91–1.57)	0.181	–
Blood transfusion	512.6 (736.5)	522.1 (889.7)	0.886	0.95 (0.72–1.26)	0.717	–

[a] Adjusted for urine creatinine, age, sex, body mass index, ratio of family income to poverty, education level, serum cotinine, alcohol habit, physical activity level, and subsampling weighting

Discussion

Previous research

Exposure to PAHs has been reported to elevate cardiovascular risk by affecting atherogenesis, thrombosis, or blood pressure regulation (O'Toole, et al 2008; Gentner and Weber 2011). Recently, it was also found that urinary 1-hydroxypyrene is associated with oxidative stress and inflammatory biomarkers in acute myocardial infarction (Freitas, et al 2014; Kraus, et al 2011; Trochimowicz, et al 1976) while 2-naphthol could be linked with lung cancer and breast cancer (Eom, et al 2013; Lee, et al 2010). Consistent with these studies, in the current analysis, adults with hypertension, heart attack and coronary heart disease had higher levels of urinary PAHs. The pathway might go from exposure to PAHs to human biomarkers leading to health conditions presenting. Also consistent with other literature, people with angina and heart failure had lower levels of PAHs (Mendis, et al 1995). However, the mechanism is unknown. As mentioned earlier, the literature on the effects of PAHs on human health is still less than that on other environmental chemicals, such as heavy metals, pesticides, arsenic, phthalates and phenols. Therefore, there is a need to further study with a longitudinal or experimental approach in future research.

Strengths and limitations

The present study has a few strengths. First, this study was conducted in a large and nationally representative human sample with mixed ethnicities and socioeconomic status. Second, this is the first time examining the risk effects of urinary PAHs on adult cardiovascular disease and cancer. Third, population attributable risks from urinary PAHs for cardiovascular disease and cancer were calculated. However, there are also a few limitations that cannot be ignored. First, there could be still other emerging chemicals from the living environments through different channels/vehicles that we might not yet know and would need future research to further identify and examine. Second, it was not possible to examine cancer by

Table 6 Associations between 1-hydroxyphenanthrene (ng/L) and adult health (*n*=1670)

	Present	Absent	*P* value	OR (95 %CI)[a]	*P* value	Population attributable risk
Hypertension	217.2 (261.5)	206.0 (302.6)	0.661	1.20 (0.92–1.56)	0.172	–
Diabetes	183.8 (233.9)	205.1 (293.8)	0.272	0.93 (0.75–1.14)	0.461	–
Heart failure	139.9 (118.4)	203.1 (287.7)	0.125	0.73 (0.39–1.34)	0.284	–
Coronary heart disease	337.9 (991.8)	197.2 (223.1)	0.0002	1.08 (0.75–1.57)	0.657	–
Angina	150.0 (106.4)	202.8 (287.7)	0.341	1.02 (0.57–1.83)	0.938	–
Heart attack	318.2 (958.9)	197.5 (224.7)	0.001	1.14 (0.65–2.00)	0.630	–
Stroke	168.8 (211.5)	202.9 (287.7)	0.389	0.93 (0.54–1.60)	0.786	–
Cancer	203.0 (266.0)	201.7 (287.3)	0.958	1.36 (0.84–2.20)	0.193	–
Blood transfusion	198.6 (236.7)	202.7 (292.5)	0.848	0.84 (0.61–1.15)	0.253	–

[a] Adjusted for urine creatinine, age, sex, body mass index, ratio of family income to poverty, education level, serum cotinine, alcohol habit, physical activity level, and subsampling weighting

Table 7 Associations between 2-hydroxyphenanthrene (ng/L) and adult health (*n*=1670)

	Present	Absent	*P* value	OR (95 %CI)[a]	*P* value	Population attributable risk
Hypertension	119.6 (189.2)	110.0 (154.6)	0.479	1.10 (0.78–1.55)	0.572	–
Diabetes	106.6 (137.2)	109.1 (154.7)	0.807	1.07 (0.86–1.35)	0.517	–
Heart failure	85.6 (74.5)	108.8 (151.6)	0.284	1.06 (0.56–2.01)	0.852	–
Coronary heart disease	170.2 (447.6)	106.6 (130.1)	0.002	1.11 (0.73–1.69)	0.606	–
Angina	84.3 (65.8)	109.0 (153.0)	0.401	1.10 (0.55–2.20)	0.774	–
Heart attack	153.8 (420.0)	107.0 (132.2)	0.018	1.32 (0.80–2.17)	0.265	–
Stroke	100.0 (171.6)	109.0 (151.6)	0.670	0.81 (0.45–1.47)	0.473	–
Cancer	105.5 (141.4)	109.0 (153.1)	0.797	1.35 (0.84–2.19)	0.203	–
Blood transfusion	101.0 (122.4)	109.9 (155.9)	0.435	0.86 (0.63–1.16)	0.293	–

[a] Adjusted for urine creatinine, age, sex, body mass index, ratio of family income to poverty, education level, serum cotinine, alcohol habit, physical activity level, and subsampling weighting

Table 8 Associations between 3-hydroxyphenanthrene (ng/L) and adult health (*n*=1670)

	Present	Absent	*P* value	OR (95 %CI)[a]	*P* value	Population attributable risk
Hypertension	138.8 (202.7)	125.1 (245.2)	0.505	1.24 (0.87–1.77)	0.211	–
Diabetes	103.5 (140.3)	126.0 (240.7)	0.145	1.02 (0.82–1.29)	0.829	–
Heart failure	78.4 (90.2)	123.3 (229.6)	0.172	0.76 (0.42–1.40)	0.360	–
Coronary heart disease	228.9 (883.7)	119.0 (163.1)	0.0003	0.83 (0.60–1.14)	0.235	–
Angina	73.1 (62.2)	123.4 (230.2)	0.257	0.95 (0.57–1.58)	0.833	–
Heart attack	225.2 (858.9)	118.8 (163.6)	0.0003	1.10 (0.67–1.81)	0.676	–
Stroke	117.5 (213.1)	122.8 (229.0)	0.866	0.91 (0.57–1.45)	0.676	–
Cancer	111.7 (136.5)	123.5 (234.8)	0.558	1.31 (0.81–2.13)	0.250	–
Blood transfusion	118.5 (186.6)	123.2 (233.9)	0.784	0.84 (0.63–1.12)	0.220	–

[a] Adjusted for urine creatinine, age, sex, body mass index, ratio of family income to poverty, education level, serum cotinine, alcohol habit, physical activity level, and subsampling weighting

Table 9 Associations between 1-hydroxypyrene (ng/L) and adult health (*n*=1670)

	Present	Absent	*P* value	OR (95 %CI)[a]	*P* value	Population attributable risk
Hypertension	198.9 (240.1)	212.3 (345.9)	0.640	1.12 (0.86–1.48)	0.375	–
Diabetes	149.9 (237.7)	212.4 (342.3)	0.005	0.91 (0.68–1.21)	0.485	–
Heart failure	143.1 (233.4)	204.5 (331.4)	0.198	0.99 (0.59–1.66)	0.976	–
Coronary heart disease	294.4 (747.0)	199.9 (304.6)	0.032	1.10 (0.83–1.47)	0.488	–
Angina	148.9 (145.4)	204.1 (331.7)	0.388	1.21 (0.71–2.05)	0.461	–
Heart attack	282.7 (739.0)	200.0 (303.7)	0.054	*1.47 (1.05–2.06)*	*0.027*	1.7 % (0.2–3.8 %)
Stroke	145.9 (187.4)	204.6 (332.8)	0.198	1.05 (0.68–1.63)	0.814	–
Cancer	186.5 (335.5)	204.4 (328.8)	0.541	1.24 (0.79–1.94)	0.325	–
Blood transfusion	193.5 (280.8)	204.0 (332.5)	0.668	1.00 (0.72–1.38)	0.994	–

Numbers in italics denote significant associations

[a] Adjusted for urine creatinine, age, sex, body mass index, ratio of family income to poverty, education level, serum cotinine, alcohol habit, physical activity level, and subsampling weighting

Table 10 Associations between 1-hydroxynapthalene (1-naphthol) (ng/L) and adult health (*n*=1670)

	Present	Absent	*P* value	OR (95 %CI)[a]	*P* value	Population attributable risk
Hypertension	87,436.3 (965,110.4)	34,738.2 (597,282.7)	0.338	1.05 (0.81–1.36)	0.701	–
Diabetes	68,535.9 (760,485.6)	31,736.3 (568,097.6)	0.366	0.88 (0.75–1.02)	0.089	–
Heart failure	587,977.6 (2,900,984.0)	21,023.9 (344,529.1)	<0.001	1.00 (0.64–1.57)	0.984	–
Coronary heart disease	7881.3 (25,540.2)	38,386.9 (611,939.5)	0.704	0.71 (0.47–1.07)	0.096	–
Angina	7121.7 (16,061.2)	37,828.6 (606,275.6)	0.793	0.92 (0.70–1.20)	0.504	–
Heart attack	477,352.6 (2,604,519.0)	20,942.2 (345,359.4)	<0.001	1.01 (0.67–1.51)	0.967	–
Stroke	321,046.1 (2,265,530.0)	28,000.9 (453,156.1)	0.0004	1.21 (0.95–1.56)	0.118	–
Cancer	9489.3 (28,027.2)	39,705.9 (626,537.9)	0.571	1.11 (0.90–1.36)	0.312	–
Blood transfusion	213,762.8 (1,693,955.0)	12,625.5 (145,458.6)	<0.001	1.00 (0.80–1.24)	0.981	–

[a] Adjusted for urine creatinine, age, sex, body mass index, ratio of family income to poverty, education level, serum cotinine, alcohol habit, physical activity level, and subsampling weighting

Table 11 Associations between 2-hydroxynapthalene (2-naphthol) (ng/L) and adult health (*n*=1670)

	Present	Absent	*P* value	OR (95 %CI)[a]	*P* value	Population attributable risk
Hypertension	10,098.6 (14,429.9)	8957.0 (11,404.4)	0.254	1.04 (0.76–1.42)	0.783	–
Diabetes	8986.9 (16,128.3)	8897.2 (11,237.6)	0.913	0.89 (0.67–1.18)	0.384	–
Heart failure	7179.9 (6432.3)	8968.8 (12,230.7)	0.308	1.01 (0.68–1.51)	0.959	–
Coronary heart disease	8601.9 (15,545.2)	8929.2 (11,972.0)	0.840	0.58 (0.34–1.00)	0.050	–
Angina	6739.0 (7180.1)	8950.7 (12,167.6)	0.347	*0.56 (0.33–0.95)*	*0.034*	–
Heart attack	10,042.4 (12,784.6)	8864.7 (12,069.4)	0.455	0.95 (0.50–1.79)	0.864	–
Stroke	8866.9 (8735.3)	8906.7 (12,193.4)	0.981	1.28 (0.85–1.92)	0.225	–
Cancer	8478.2 (12,029.0)	8944.6 (12,102.5)	0.664	*1.46 (1.12–1.90)*	*0.008*	3.9 % (1.0–7.3 %)
Blood transfusion	8902.0 (12,158.0)	8894.5 (12,112.4)	0.993	0.93 (0.69–1.26)	0.627	–

Numbers in italics denote significant associations

[a] Adjusted for urine creatinine, age, sex, body mass index, ratio of family income to poverty, education level, serum cotinine, alcohol habit, physical activity level, and subsampling weighting

Table 12 Associations between 4-hydroxyphenanthrene (ng/L) and adult health (*n*=1670)

	Present	Absent	*P* value	OR (95 %CI)[a]	*P* value	Population attributable risk
Hypertension	47.5 (108.8)	34.6 (42.6)	0.005	*1.33 (1.00–1.76)*	*0.048*	5.1 % (0–11.0 %)
Diabetes	38.3 (81.2)	34.5 (44.1)	0.281	1.07 (0.86–1.32)	0.534	–
Heart failure	21.8 (18.4)	35.3 (51.3)	0.067	0.70 (0.39–1.29)	0.237	–
Coronary heart disease	44.8 (101.5)	34.8 (48.8)	0.146	0.76 (0.48–1.18)	0.204	–
Angina	24.5 (18.9)	35.2 (51.7)	0.280	1.08 (0.61–1.91)	0.791	–
Heart attack	40.5 (89.9)	34.9 (49.4)	0.400	1.02 (0.56–1.85)	0.941	–
Stroke	30.9 (54.4)	35.2 (51.3)	0.542	0.73 (0.42–1.28)	0.258	–
Cancer	31.5 (42.6)	35.4 (52.1)	0.404	1.36 (0.72–2.57)	0.319	–
Blood transfusion	37.0 (77.9)	34.8 (46.9)	0.573	0.96 (0.72–1.29)	0.781	–

Numbers in italics denote significant associations

[a] Adjusted for urine creatinine, age, sex, body mass index, ratio of family income to poverty, education level, serum cotinine, alcohol habit, physical activity level, and subsampling weighting

subtypes due to small number of disease events. Therefore, it is not possible to compare with the literature directly. Third, causality cannot be established in the present study due to the cross-sectional study design in nature. The significance observed in the present study could be just by chance, but the values in urine were indeed higher in people with these health conditions. Future studies with a longitudinal or experimental approach to confirm or refute the current findings and, if at all, to understand the persisting risk effects along the life course from those environmental chemicals mentioned above would be suggested.

Conclusion

In sum, urinary PAHs were positively associated with adult hypertension, heart attack and cancer while inversely associated with angina. From the research perspective, future studies with a longitudinal or experimental approach would be suggested. From the law and human health perspectives, regulation on minimizing exposure to PAHs for humans might need to be considered in future health and environmental policies and intervention programs.

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