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Association of Urinary Phenols with Increased Body Weight Measures and Obesity in Children and Adolescents

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Abstract

Objective—To examine the association of urinary levels of the environmental phenol pesticides 2,5-dichlorophenol, 2,4-dichlorophenol, and triclosan with body weight outcomes in children and adolescent participants in the National Health and Nutrition Examination Survey 2007-2010.

Study design—We performed multivariate linear and multinomial logistic regressions to analyze the association of body mass index (BMI) z-score, waist circumference (WC), and obesity with urinary pesticide concentration in children and adolescents.

Results—After adjustment for covariates, we found a statistically significant positive association ($P < .05$) between both 2,5-dichlorophenol and 2,4-dichlorophenol with BMI z-score, WC, and obesity in children and adolescents. After stratification by age, the significant associations remained only in adolescents (ages 12-19). No associations were found between triclosan and any of the body weight outcomes.

Conclusions—We found an association between dichlorophenols and increased body weight measures (BMI z-score, WC, and obesity) in adolescents. However, further studies, such as a longitudinal study, are needed to confirm and elucidate on our findings.

Obesity has become an increasingly important public health concern, as the prevalence in children, adolescents, and adults has increased over the past few decades both within the US and worldwide. The most accepted explanation of this rise in obesity is an adoption of an unhealthy diet and lifestyle. However, an increasing body of evidence suggests that exposure to environmental chemicals, known as “obesogens,” may increase the risk of obesity both in children and adults.[1–10]

Because of their vast usage throughout the US as well as the rest of the world, pesticides have become ubiquitous in our environment, creating the potential for human exposure through contaminated food, soil, water, air, and other sources. Pesticides have been shown to have many adverse effects in child development including neurologic and metabolic outcomes, such as diabetes and obesity.[7,8] Dichlorophenols are one such category of pesticides; the National Health and Nutrition Examination Survey (NHANES 2007-2010) has detected them in over 90% of the urine samples of participants. 2,5-Dichlorophenol (2,5-

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DCP) is a metabolite of 1,4-dichlorobenzene (1,4-DCB), which is commonly used in moth balls, room and toilet deodorizers, and as a fumigant insecticide.[9,10] The main source of exposure to 1,4-DCB is through inhalation.[11]

Studies have reported an association between 2,5-DCP and obesity and body mass index (BMI) in children and adolescents[12] and adults.[13] These researchers found a statistically significant increase in the rate of obesity among these populations. The objective of this study was to build on these studies and investigate the correlation between 2,4-dichlorophenol (2,4-DCP), 2,5-DCP, and triclosan (TCS) with not only obesity but with other weight outcomes—BMI z-score and waist circumference (WC), an indication of abdominal fat—using data from NHANES 2007-2010 in children and adolescents (6-19 years old).

Methods

NHANES is a program of cross-sectional, nationally representative surveys of the noninstitutionalized civilian population of the US conducted by the National Center for Health Statistics (NCHS), Centers for Disease Control and Prevention (CDC).[14] Beginning in 1999, the survey was conducted continuously and released in 2-year cycles. For our study, we merged the publicly available files for NHANES cycles 2007-2008 and 2009-2010 using the recommendations of NCHS.[15] The survey employs a multistage stratified probability sample based on selected counties, blocks, households, and persons within households.

NCHS-trained professionals conducted interviews in participants' homes and extensive physical examinations, which included blood and urine collection, conducted at mobile examination centers. CDC's National Center for Environmental Health, Division of Laboratory Sciences coordinates the National Biomonitoring Program, which offers an assessment of nutritional status and the exposure of the US population to environmental chemicals and toxic substances. All procedures were approved by the NCHS Research Ethics Review Board (Continuation of Protocol #2005-2006 http://www.cdc.gov/nchs/nhanes/irba98.htm), and all participants provided written informed consent.

In the 2007-2008 and 2009-2010 dataset, urinary metabolites of chlorinated phenols and TCS were measured in a randomly selected one-third subsample of persons 6 years and older. For our analysis, we included all participants (age 6-19 years) who had measurements for dichlorophenols and TCS (n = 1603). In addition, participants with missing covariables included in the multivariable-adjusted models were excluded (n = 305). Therefore, the total number of participants in our analyses was 1298.

Spot urine samples were collected from study participants and stored at −20°C until analysis. The urine samples were analyzed by the National Center for Environmental Health, Division of Laboratory Sciences for TCS and 2 chlorophenols: 2,4-DCP and 2,5-DCP using solid phase extraction coupled on-line to high performance liquid chromatography and tandem mass spectrometry.

Urinary 2,4-DCP, 2,5-DCP, and TCS were detected in 90%, 98.5%, and 79% of the samples, respectively. Therefore, as reported in the NHANES dataset, urinary concentrations of 2,4-

DCP, 2,5-DCP, and TCS below the level of detection were assigned the limit of detection divided by the square root of 2. To account for variation in dilution in spot urinary samples, urinary creatinine was entered in the analyses as an independent variable as suggested by previous studies.[16,17] Urinary creatinine was determined using a Jaffé rate reaction measured with a CX3 analyzer (Beckman Instruments Inc, Brea, California).[18]

BMI is calculated by the weight divided by height squared (kg/m^2). However, the relation between BMI in children depends on age and sex; therefore, it is more appropriate to calculate the BMI z-score. The BMI z-score is the number of SDs by which a child differs from the mean BMI of children of the same age and sex. Thus, the BMI z-score allows comparison of children of different ages and sexes. Age- and sex-specific BMI z-scores were calculated using the methodology provided by the CDC (http://www.cdc.gov/nccdphp/dnpao/growthcharts/resources/sas.htm).

BMI z-score was then used to classify individuals as overweight and obese defined as BMI greater than or equal to the 95th percentile (obese) or between the 85th and less than the 95th percentile (overweight).

Data on WC (cm) was obtained during the examination. WC was measured at the high point of the right iliac crest at minimal respiration to the nearest 0.1 cm (http://www.cdc.gov/nchs/data/nhanes/nhanes_07_08/manual_an.pdf; http://www.cdc.gov/nchs/data/nhanes/nhanes_09_10/BodyMeasures_09.pdf).

In the regression models, we adjusted for a priori covariates[12,19]: age, race/ethnicity, sex, urinary creatinine, poverty income ratio, calorie intake, and serum cotinine as a biomarker of exposure to environmental tobacco smoke. Race/ethnicity was categorized as non-Hispanic white, non-Hispanic black, Mexican American, other Hispanic, and other. Poverty income ratio is a measure of socioeconomic status and represents the calculated ratio of household income to the poverty threshold after accounting for inflation and family size. Caloric intake was categorized as “normal” and “excessive” based on the US Department of Agriculture calorie intake guidelines by age and sex (http://health.gov/dietaryguidelines/dga2010/DietaryGuidelines2010.pdf). The individual cut-off caloric need was the highest value for the range by age and sex assuming a moderate physical activity level. After stratification by age, we included television, video game, and computer usage for children (ages 6-11 years) and physical activity for adolescents (ages 12-19 years). Information on daily hours of television, video, or computer use was obtained by questionnaire, and the covariate was categorized with a cut point of ≥2 hours/day. Information on physical activity came from the NHANES questionnaire; participants were asked whether they engaged in regular moderate and/or vigorous recreational activities (categorized as yes or no).

Statistical Analyses

Specific sample weights for this subsample were used for analyses to account for the complex sampling design and nonresponse of NHANES. Weights for combined NHANES survey cycles were calculated according to NHANES guidelines.[20] We estimated sampling errors using the Taylor series linearized method. Linear regression analyses were used to investigate the correlation between BMI z-scores and WC and urinary 2,4-DCP, 2,5-DCP, and TCS categorized by weighted quartile distribution. We used multinomial logistic regression models to simultaneously estimate aORs for obesity and overweight status as distinct outcomes (compared with normal/underweight) in association with categorical urinary compounds. In addition to estimating associations for all observations combined, we performed separate analyses stratified by age (6-11 years and 12-19 years). We also assessed possible interactions between pesticides and sex, but because the interaction was not statistically significant, it was not included in the models. SAS 9.3 (SAS Institute, Cary, North Carolina) was used for all statistical analyses and SAS-Callable SUDAAN 10 (Research Triangle Institute, Research Triangle Park, North Carolina) was used to account for the NHANES complex sample design. *P* values from Satterthwaite statistics were presented at the significance level of <.05.

Results

Table 1 illustrates the weighted characteristics of participants aged 6-19 years from NHANES 2007-2010 included in this study. The mean age of the participants was slightly higher than 12 years, and 51% were male. Non-Hispanic whites accounted for 58.3% of the total study group; 14.2% were non-Hispanic blacks, and 13.5 % were Mexican-American. Twenty-four percent of the participants were from families with income at or below the poverty level. The mean and SE of the mean for BMI z-score was 0.51 (0.03) and for WC was 74.27 (0.38) (Table I). Eighteen percent of all participants were obese, and 15% were overweight (data not shown). The geometric mean (SE) of 2,4-DCP, 2,5-DCP, and TCS were 8.81 (0.98) μg/L, 1.04 (0.06) μ/L, and 13.19 (0.99) ng/mL, respectively.

In addition, Table I shows the characteristics of the obese, overweight, and normal/underweight participants. Participants categorized as obese had higher urinary levels of 2,4-DCP and 2,5-DCP compared with normal and overweight participants but had lower levels of urinary TCS.

A statistically significant association was found between BMI z-score and WC with 2,4-DCP and 2,5-DCP. The second, third, and fourth 2,4-DCP quartiles had a higher BMI z-score (β [95% CI]: 0.33 [0.09, 0.57]; 0.40 [0.15, 0.66] and 0.30 [0.02, 0.58], respectively) and higher WC (β [95% CI]: 3.54 [0.98, 6.10], 4.59 [1.82, 7.35], and 3.80 [0.69, 6.92], respectively) compared with the lowest referent quartile (Table II). Therefore, participants in the fourth quartile compared with the first have an increase of 0.30 SDs in BMI z-score and an increase of 3.80 cm in WC. The second, third, and fourth 2,5-DCP quartile had a higher BMI z-score (β [95% CI]: 0.38 [0.15, 0.62]; 0.43 [0.14, 0.72], and 0.35 [0.01, 0.69], respectively) and WC (β [95% CI]: 3.80 [1.62, 5.97], 5.17 [2.11, 8.23], and 4.75 [0.82, 8.67], respectively) compared with the lowest referent quartile (Table II). Therefore, participants in the fourth quartile of 2,5-DCP exposure have an increase of 0.35 SDs in BMI z-score and an increase of 4.75 cm in WC compared with participants in the first quartile. No association was found between BMI z-score and TCS. However, the highest quartile of TCS was associated with a lower WC compared with the lowest quartile (β [95% CI]: −2.44 [−4.53, −0.34]) (Table II).

After stratification by age, the statistically significant associations between 2,4-DCP and 2,5-DCP with BMI z-score and WC remained in adolescents in the second, third, and fourth quartiles (Table II). The association between TCS and WC dissipated after stratification by age group.

Analyses run using chemicals as log-natural transformed variables did not find any statistically significant associations, other than for TCS and WC for all participants (data not shown). The difference in the results suggests that the relationship between these chemicals and the outcomes of interest are not linear; this suggests that the use of quartiles is more appropriate for these analyses.

In multinomial logistic regression, we found a statistically significant positive association between 2,4-DCP and 2,5-DCP with obesity in all participants after controlling for a priori covariates; these statistically significant associations were found only in adolescents after stratification by age (Table III). Compared with persons in the lowest 2,4-DCP quartile, adolescents in the highest 2,4-DCP quartiles had a statistically significant higher aORs for obesity (aOR = 2.39, 95% CI, 1.17, 4.90). This relationship was only seen for those participants in the highest quartile of 2,4-DCP exposure, but not for those participants in the second or third quartiles (Table II). Compared with persons in the lowest 2,5-DCP quartile, adolescents in the second, third, and highest 2,5-DCP quartiles had a statistically significant higher aORs for obesity (aOR = 4.03, 95% CI, 1.81, 8.99; aOR = 5.32, 95% CI:2.21, 12.80, and aOR = 4.86, 95% CI, 1.83, 12.90, respectively) (Table III). No association was found between TCS and obesity (Table III). Furthermore, no associations were found between any of the chemicals with overweight outcomes.

Discussion

Previous studies investigated the association between dichlorophenols and obesity outcomes using the NHANES databases. Twum and Wei[12] reported a statistically significant association between 2,5-DCP and childhood (ages 6-19 years) obesity using NHANES 2003-2006. Compared with the lowest quartile, individuals in third and fourth 2,5-DCP quartiles had an increased prevalence of obesity (OR: 1.47; 95% CI, 1.09, 1.97 and OR: 1.44; 95% CI, 1.06, 1.95, respectively). This is in agreement with our findings. However, they did not find any association with 2,4-DCP, whereas we found a statistically significant association of 2,4-DCP with obesity.

Despite some animal studies finding decreased body weight after exposure to 2,4-DCP or 1,4-DCB, these depressed body weights were only observed after treatment with high doses of the chemicals. At the lower-level exposures, no alterations in body weight were observed. However, levels of human exposure in the general population are much lower than the doses used in animal studies; therefore, comparisons may not be appropriate.

There are several plausible biological mechanisms through which dichlorophenols may act to affect body weight. Recent studies have suggested that dichlorophenols may have endocrine disrupting potential, in part because of the structure of the chemicals, which is similar to that of estradiol. However, results from in vitro studies have been inconsistent.

Another potential mechanism by which 2,4-DCP may act as an endocrine disruptor is through the thyroid hormone system, which regulates the body's metabolic processes. Although there are no data on a direct relationship between 2,4-DCP and the thyroid, an effect on the thyroid gland was reported for the parent compound of 2,4-DCP, dichlorophenoxyacetic acid. Rats exposed to dichlorophenoxyacetic acid showed a decrease in thyroid weight and a decrease in circulating thyroid hormone levels.[21]

Recently, there have been studies undertaken to investigate the effects of dichlorophenols on birth weight. Given the possible association between lower birth weight and the risk of obesity during childhood and adolescence, the results from these studies could provide insight into a potential mechanism for our findings. Philippat et al[22] found that maternal urinary concentrations of 2,4-DCP and 2,5-DCP are associated with lower birth weight, whereas no association was found with urinary TCS. In addition, an earlier study by Wolff et al[23] found a similar association with 2,5-DCP. The authors found higher prenatal exposure was statistically significantly associated with lower birth weight in male offspring. Specifically, there was a 210 g (95% CI, 71-348 g) average birth weight difference between third and first tertiles. Furthermore, a study on 2,5-DCP's parent compound, 1,4-DCB, also found an association with lower birth weight.[24]

There are few studies on the association between TCS and body weight, and the results are conflicting. Data suggest that TCS may alter thyroid function. A study in rats found that prenatal exposure to TCS impaired thyroid homeostasis and resulted in lower body weights in the pups at postnatal day 20.[25]

There are several limitations to our study. The cross-sectional study design of NHANES limits the inferences that can be made because all data are collected at a single time-point; therefore, causation cannot be explored. A major limitation is the use of single-spot urine measures as an estimate of exposure because these compounds are rapidly metabolized and excreted, so a single exposure measurement may not reflect long-term exposure. However, the high detection rate of these compounds in NHANES suggests that exposure to these compounds is likely constant over time. In addition, reverse causation cannot be excluded because overweight and obese people will have more fat mass, they may store more pesticides, thus, leading to higher excretion. Furthermore, because NHANES uses questionnaires, there is the possibility of information bias because of a reliance on participants providing accurate information, particularly pertaining to the dietary information. Recently, the accuracy of the self-reported data on energy intake for obesity research has been questioned because participants may be misreporting their caloric intake.[26] In our analyses, removal of the dietary information as a covariate did not change the significance of our results (data not shown). Although we controlled for several factors associated with body weight and obesity, other risk factors that we have not accounted for might have influenced the findings.

Dichlorophenols are found in over 90% of the US population (NHANES 2007-2010). Because of their prevalence within the environment, it is important to understand their health impact in humans; furthermore, it is important to elucidate the potential mechanisms through which these pesticides act so preventive measures can be taken to curb their effects. These results, along with previous studies, suggest that this association warrants further investigation, such as a longitudinal study that would allow for long-term exposure to be evaluated and causality to be potentially elucidated.

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Glossary

1,4-DCB	1,4-Dichlorobenzene
2,4-DCP	2,4-Dichlorophenol
2,5-DCP	2,5-Dichlorophenol
BMI	Body mass index
CDC	Centers for Disease Control and Prevention
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
TCS	Triclosan
WC	Waist circumference

References

1. Grun F, Blumberg B. Environmental obesogens: organotins and endocrine disruption via nuclear receptor signaling. Endocrinology 2006;147:S50–5. [PubMed: 16690801]
2. Janesick A, Blumberg B. Endocrine disrupting chemicals and the developmental programming of adipogenesis and obesity. Birth Defects Res C Embryo Today 2011;93:34–50. [PubMed: 21425440]
3. Grüm F Obesogens. Curr Opin Endocrinol Diabetes Obes 2010;17:453–9. [PubMed: 20689419]
4. Karoutsou E, Polymeris A. Environmental endocrine disruptors and obesity. Endocr Regul 2012;46:37–46. [PubMed: 22329821]
5. Newbold RR, Padilla-Banks E, Jefferson WN. Environmental estrogens and obesity. Mol Cell Endocrinol 2009;304:84–9. [PubMed: 19433252]
6. Casals-Casas C, Desvergne B. Endocrine disruptors: from endocrine to metabolic disruption. Annu Rev Physiol 2011;73:135–62. [PubMed: 21054169]
7. Meeker JD. Exposure to environmental endocrine disruptors and child development. Arch Pediatr Adolesc Med 2012;166:952–8. [PubMed: 23367522]
8. Thayer KA, Heindel JJ, Bucher JR, Gallo MA. Role of environmental chemicals in diabetes and obesity: a National Toxicology Program workshop review. Environ Health Perspect 2012;120:779–89. [PubMed: 22296744]
9. ATSDR: Agency for Toxic Substances and Disease Registry. Toxicological Profile for Dichlorobenzenes. Atlanta, GA: US Department of Health and Human Services, Public Health Service; 2006.

10. Yoshida T, Andoh K, Fukuhara M. Urinary 2,5-dichlorophenol as biological index for p-dichlorobenzene exposure in the general population. Arch Environ Contam Toxicol 2002;43:481–5. [PubMed: 12399920]
11. Hill RH Jr, Ashley DL, Head SL, Needham LL, Pirkle JL. P-dichlorobenzene exposure among 1000 adults in the United Sates. Arch Environ Health 1995;50:227–80.
12. Twum C, Wei Y. The association between urinary concentrations of dichlorophenol pesticides and obesity in children. Rev Environ Health 2011;26:215–9. [PubMed: 22206198]
13. Wei Y, Zhu J, Nguyen A. Urinary concentrations of dichlorophenol pesticides and obesity among adult participants in the U.S. National Health and Nutrition Examination Survey (NHANES) 2005-2008. Int J Hyg Environ Health 2013;217:294–9. [PubMed: 23899931]
14. NCHS: National Center for Health Statistics. National Health and Nutrition Examination Survey Home Page. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Health Statistics. 2008a. Available at: http://www.cdc.gov/nchs/nhanes.htm. Accessed March 2012.
15. NCHS: National Center for Health Statistics. National Health and Nutrition Examination. Survey analytic guidelines. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Health Statistics. 2013. Available at: http://www.cdc.gov/nchs/data/series/sr_02/sr02_161.pdf. Accessed October 31, 2013.
16. Ikeda M, Ezaki T, Tsukahara T, Moriguchi J, Furuki K, Fukui Y, et al. Bias induced by the use of creatinine-corrected values in evaluation of beta2-microgloblin levels. Toxicol Lett 2003;145:197–207. [PubMed: 14581173]
17. Barr DB, Wilder LC, Caudill SP, Gonzalez AJ, Needham LL, Pirkle JL. Urinary creatinine concentrations in the U.S. population: implications for urinary biologic monitoring measurements. Environ Health Perspect 2005;113:192–200. [PubMed: 15687057]
18. NCHS: National Center for Health Statistics. National Health and Nutrition Examination Survey 2005–2006. Laboratory Procedure Manuals. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Health Statistics. 2007. Available at: http://www.cdc.gov/nchs/nhanes/nhanes2005-2006/manuals05_06.htm. Accessed October 31, 2013.
19. Trasande L, Attina TM, Blustein J. Association between urinary bisphenol A concentration and obesity prevalence in children and adolescents. JAMA 2012;308:1113–21. [PubMed: 22990270]
20. NCHS: National Center for Health Statistics. Continuous National Health and Nutrition Examination Survey (NHANES) Web Tutorial. Constructing Weights for Combined NHANES Survey Cycles. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Health Statistics. 2008c. Available at: http://www.cdc.gov/nchs/tutorials/nhanes/SurveyDesign/Weighting/intro.htm. Accessed October 31, 2013.
21. Charles JM, Bond DM, Jeffries TK, Yano BL, Stott WT, Johnson KA, et al. Chronic dietary toxicity/oncogenicity studies on 2,4-dichlorophenoxyacetic acid in rodents. Fundam Appl Toxicol 1996;33:166–72. [PubMed: 8921335]
22. Philippat C, Mortamais M, Chevrier C, Petit C, Calafat AM, Ye X, et al. Exposure to phthalates and phenols during pregnancy and offspring size at birth. Environ Health Perspect 2012;120:464–70. [PubMed: 21900077]
23. Wolff MS, Engel SM, Berkowitz GS, Ye X, Silva MJ, Zhu C, et al. Prenatal phenol and phthalate exposures and birth outcomes. Environ Health Perspect 2008;116:1092–7. [PubMed: 18709157]
24. Marsman D NTP technical report on the toxicity studies of dibutyl phthalate (CAS No. 84-74-2) administered in feed to F344/N rats and B6C3F1 mice. Toxic Rep Ser 1995;30:1–G5. [PubMed: 12209194]
25. Rodríguez PE, Sanchez MS. Maternal exposure to triclosan impairs thyroid homeostasis and female pubertal development in Wistar rat offspring. J Toxicol Environ Health A 2010;73:1678–88. [PubMed: 21058171]
26. Archer E, Hand GA, Blair SN. Validity of U.S. nutritional surveillance: National Health and Nutrition Examination Survey caloric energy intake data, 1971-2010. PLoS One 2013;8:e76632. [PubMed: 24130784]

Table I.

Weighted characteristics of the NHANES 2007-2010 participants, aged 6-19 years

	All	Normal/underweight	Overweight	Obese
2,5-DCP (μg/L), GM (SE)	8.81 (0.98)	7.50 (0.91)	9.20 (1.59)	14.71 (2.36)
2,4-DCP (μg/L), GM (SE)	1.04 (0.06)	0.97 (0.06)	1.07 (0.14)	1.31 (0.14)
Urinary TCS (ng/L), GM (SE)	13.19 (0.99)	13.99 (1.36)	12.92 (1.39)	11.00 (0.77)
Age (y), mean (SE)	12.56 (0.10)	12.59 (0.12)	12.57 (0.23)	12.43 (0.19)
BMI z-score, mean (SE)	0.51 (0.03)	−0.11 (0.02)	1.31 (0.01)	2.08 (0.02)
BMI, mean (SE)	21.41 (0.15)	18.77 (0.08)	23.40 (0.19)	29.30 (0.30)
WC (cm), mean (SE)	74.27 (0.38)	67.70 (0.22)	79.26 (0.58)	93.83 (0.64)
Serum cotinine (ng/mL), GM (SE)	0.11 (0.01)	0.10 (0.01)	0.11 (0.02)	0.19 (0.04)
Urinary creatinine (mg/dL), GM (SE)	99.27 (2.49)	96.88 (2.83)	95.19 (5.38)	112.48 (3.72)
Age				
Children (ages 6-11 years), % (SE)	42.22 (1.13)	41.65 (1.33)	42.11 (3.17)	44.42 (2.40)
Adolescents (ages 12-19 years), % (SE)	57.78 (1.13)	58.35 (1.33)	57.89 (3.17)	55.58 (2.40)
Sex				
Male % (SE)	51.03 (1.22)	50.71 (1.55)	47.31 (2.58)	55.37 (2.51)
Female % (SE)	48.97 (1.22)	49.29 (1.55)	52.69 (2.58)	44.63 (2.51)
Race				
White (Non-Hispanic) % (SE)	58.33 (2.76)	62.18 (2.79)	52.82 (3.88)	49.02 (4.42)
Non-Hispanic Black % (SE)	14.23 (1.20)	12.31 (1.19)	15.82 (1.88)	19.88 (2.28)
Mexican-American % (SE)	13.54 (1.99)	11.57 (1.82)	16.81 (2.33)	17.93 (3.21)
Other Hispanic % (SE)	6.47 (1.34)	5.89 (1.24)	6.17 (1.70)	8.84 (2.01)
Other % (SE)	7.42 (0.93)	8.05 (1.10)	8.37 (1.99)	4.32 (1.11)
Income				
Poverty income level (PIR ≤ 1) % (SE)	24.09 (1.57)	21.63 (1.69)	23.18 (1.75)	33.70 (2.90)
Above poverty income level (PIR > 1) % (SE)	75.91 (1.57)	78.38 (1.69)	76.82 (1.75)	66.30 (2.90)
Caloric intake				
Normal intake % (SE)	68.31 (1.68)	66.48 (1.80)	73.86 (2.88)	70.24 (2.57)
Excessive intake % (SE)	31.69 (1.68)	33.52 (1.80)	26.16 (2.88)	29.76 (2.57)
Television and video games use				

	All	Normal/underweight	Overweight	Obese
≤2 hours, % (SE)	54.67 (2.38)	56.37 (3.45)	55.36 (3.74)	48.18 (3.39)
>2, % (SE)	45.33 (2.38)	43.63 (3.45)	44.64 (3.74)	51.82 (3.39)
Moderate recreational activities				
Yes % (SE)	52.50 (2.01)	53.69 (2.61)	51.23 (4.25)	49.01 (4.19)
No % (SE)	47.50 (2.01)	46.31 (2.61)	48.77 (4.25)	50.99 (4.19)
Vigorous recreational activities				
Yes % (SE)	62.28 (1.59)	62.39 (2.08)	71.30 (4.10)	53.50 (4.49)
No % (SE)	37.72 (1.59)	37.61 (2.08)	28.70 (4.10)	46.50 (4.49)

GM, geometric mean; *PIR*, poverty income ratio.

Table II.

Adjusted* multivariate linear regression (b coefficient and 95% CI) for the association between BMI z-score and WC with quartiles of 2,5-DCP, 2,4-DCP, and TCS in NHANES 2007-2010 participants

	Children/adolescents (6-19 y)		Children (6-11 y)		Adolescents (12-19 y)	
	BMI-z score	**WC**	**BMI-z score**	**WC**	**BMI-z score**	**WC**
n	1298	1284	572	565	671	665
2,5-DCP Q1	Referent	Referent	Referent	Referent	Referent	Referent
2,5-DCP Q2	0.38 (0.15, 0.62)	3.80 (1.62, 5.97)	0.25 (−0.12, 0.62)	2.93 (−0.71, 6.56)	0.48 (0.22, 0.73)	5.01 (2.15, 7.88)
2,5-DCP Q3	0.43 (0.14, 0.72)	5.17 (2.11, 8.23)	0.46 (0.04, 0.89)	4.41 (0.71, 8.12)	0.46 (0.12, 0.80)	6.40 (2.39, 10.42)
2,5-DCP Q4	0.35 (0.01, 0.69)	4.75 (0.82, 8.67)	0.07 (−0.30, 0.45)	1.37 (−2.01, 4.74)	0.46 (0.08, 0.85)	6.83 (1.86, 11.80)
P-trend	.01	<.01	.08	.07	<.01	<.01
2,4-DCP Q1	Referent	Referent	Referent	Referent	Referent	Referent
2,4-DCP Q2	0.33 (0.09, 0.57)	3.54 (0.98, 6.10)	0.29 (−0.00, 0.58)	3.58 (1.32, 5.83)	0.39 (0.13, 0.66)	4.11 (1.08, 7.14)
2,4-DCP Q3	0.40 (0.15, 0.66)	4.59 (1.82, 7.35)	0.47 (0.14, 0.80)	5.30 (2.08, 8.53)	0.35 (0.02, 0.67)	4.24 (0.47, 8.01)
2,4-DCP Q4	0.30 (0.02, 0.58)	3.80 (0.69, 6.92)	0.09 (−0.25, 0.44)	1.42 (−1.37, 4.22)	0.36 (0.01, 0.72)	5.14 (1.14, 9.14)
P-trend	.02	.01	<.01	<.01	.04	.03
TCS Q1	Referent	Referent	Referent	Referent	Referent	Referent
TCS Q2	−0.02 (−0.20, 0.16)	−0.13 (−2.21, 1.95)	−0.15 (−0.50, 0.20)	−1.05 (−3.85 1.75)	0.08 (−0.16, 0.32)	1.49 (−1.94, 4.91)
TCS Q3	0.12 (−0.12, 0.36)	1.24 (−1.60, 4.08)	0.05 (−0.30, 0.40)	0.07 (−3.32, 3.47)	0.21 (−0.07, 0.49)	2.85 (−0.77, 5.46)
TCS Q4	−0.11 (−0.32, 0.09)	−2.44 (−4.53, −0.34)	0.02 (−0.26, 0.31)	−0.58 (−3.07, 1.90)	−0.14 (−0.40, 0.13)	−2.11 (−4.81, 0.59)
P-trend	.21	.01	.47	.76	.11	.03

*Adjusted for age, sex, race/ethnicity, calorie intake, television and video game use (6-11 y), recreational activity (12-19 y), serum cotinine, income level, and urinary creatinine. Quartiles 2,5-DCP (mg/L): Q1: ≤2.03; Q2:2.04-6.14; Q3:6.15-27.91; Q4: >27.91. Quartiles 2,4-DCP (μg/L): Q1: ≤0.38; Q2: 0.39-0.80; Q3: 0.81-1.97; Q4: >1.97. Quartiles TCS (ng/mL): Q1: ≤3.53; Q2:3.54-10.13; Q3: 10.14-36.53; Q4: >36.53.

Table III.

Adjusted* multinomial logistic regression (OR and 95% CI) of obese and overweight with quartiles of 2,5-DCP, 2,4-DCP, and TCS in NHANES 2007-2010 participants

	Children/adolescents (6-19 y)		Children (6-11y)		Adolescents (12-19 y)	
	Obese	Overweight	Obese	Overweight	Obese	Overweight
N						
2,5-DCP Q1	Referent	Referent	Referent	Referent	Referent	Referent
2,5-DCP Q2	2.27 (1.35, 3.80)	1.41 (0.71, 2.78)	1.55 (0.69, 3.49)	1.39 (0.42, 4.56)	4.03 (1.81, 8.99)	1.59 (0.81, 3.13)
2,5-DCP Q3	3.31 (1.77, 6.18)	1.12 (0.68, 1.87)	2.62 (1.09, 6.29)	1.50 (0.70, 3.19)	5.32 (2.21, 12.80)	0.97 (0.37, 2.55)
2,5-DCP Q4	2.63 (1.34, 5.15)	1.11 (0.54, 2.28)	1.23 (0.62, 2.44)	0.95 (0.36, 2.49)	4.86 (1.83, 12.90)	1.27 (0.50, 3.18)
2,4-DCP Q1	Referent	Referent	Referent	Referent	Referent	Referent
2,4-DCP Q2	2.35 (1.26, 4.37)	1.21 (0.69, 2.11)	2.91 (1.43, 5.91)	1.18 (0.43, 3.24)	2.08 (0.92, 4.74)	1.21 (0.49, 2.99)
2,4-DCP Q3	2.32 (1.29, 4.15)	1.50 (0.77, 2.91)	2.70 (1.27, 5.74)	1.60 (0.61, 4.22)	1.75 (0.80, 3.83)	1.47 (0.55, 3.93)
2,4-DCP Q4	2.42 (1.41, 4.17)	1.00 (0.46, 2.17)	1.91 (0.94, 3.90)	1.18 (0.41, 3.42)	2.39 (1.17, 4.90)	0.83 (0.31, 2.22)
TCS Q1	Referent	Referent	Referent	Referent	Referent	Referent
TCS Q2	0.91 (0.59, 1.41)	1.25 (0.68, 2.31)	0.57 (0.30, 1.09)	1.34 (0.49, 3.70)	1.36 (0.69, 2.67)	1.18 (0.44, 3.18)
TCS Q3	1.34 (0.77, 2.35)	1.28 (0.73, 2.24)	1.21 (0.56, 2.62)	1.17 (0.60, 2.29)	1.62 (0.78, 3.37)	1.34 (0.56, 3.21)
TCS Q4	0.74 (0.51, 1.09)	1.16 (0.63, 2.16)	0.84 (0.41, 1.73)	1.24 (0.52, 2.92)	0.81 (0.44, 1.50)	0.99 (0.47, 2.10)

*Adjusted for age, sex,race/ethnicity,calorie intake, television and video game use (6-11 y),recreational activity (12-19 y), serum cotinine, income level, and urinary creatinine. Quartiles 2,5-DCP (μg/L): Q1: ≤2.03; Q2:2.04-6.14; Q3:6.15-27.91; Q4: >27.91. Quartiles 2,4-DCP (μg/L): Q1: ≤0.38; Q2:0.39-0.80; Q3:0.81-1.97; Q4: >1.97. Quartiles TCS (ng/mL): Q1: ≤3.53; Q2:3.54-10.13; Q3: 10.14-36.53; Q4: >36.53.