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Fruit and vegetable intake, as reflected by serum carotenoid concentrations, predicts reduced probability of PCB-associated risk for type 2 diabetes: NHANES 2003–2004

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Abstract

Type 2 diabetes has been shown to occur in response to environmental and genetic influences, among them nutrition, food intake patterns, sedentary lifestyle, body mass index (BMI), and exposure to persistent organic pollutants (POPs), such as polychlorinated biphenyls (PCBs). Nutrition is essential in the prevention and management of type 2 diabetes and has been shown to modulate the toxicity of PCBs. Serum carotenoid concentrations, considered a reliable biomarker of fruit and vegetable intake, are associated with the reduced probability of chronic diseases, such as type 2 diabetes and cardiovascular disease. Our hypothesis is that fruit and vegetable intake, reflected by serum carotenoid concentrations, is associated with the reduced probability of developing type 2 diabetes in US adults with elevated serum concentrations of PCBs 118, 126, and 153. This cross-sectional study utilized the CDC database, National Health and Nutrition Examination Survey (NHANES) 2003–2004 in logistic regression analyses. Overall prevalence of type 2 diabetes was approximately 11.6% depending on the specific PCB. All three PCBs were positively associated with the probability of type 2 diabetes. For participants at higher PCB percentiles (e.g., 75th and 90th) for PCB 118 and 126, increasing serum carotenoid concentrations were associated with a smaller probability of type 2 diabetes. Fruit and vegetable intake, as

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Online Supplementary Materials

See Tables S1, S2, S3, and S4.

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reflected by serum carotenoid concentrations, predicted notably reduced probability of dioxin-like PCB-associated risk for type 2 diabetes.

Keywords

nutrition; polychlorinated biphenyls; PCBs; serum carotenoids; NHANES; type 2 diabetes; environmental health

1. Introduction

Recent decades have seen increased rates of type 2 diabetes, and it is now estimated to affect 25.8 million Americans and 346 million people worldwide [1,2]. Type 2 diabetes has been shown to occur in response to environmental and genetic influences [3–9], among them food intake patterns, sedentary lifestyle, body mass index (BMI) and exposure to persistent organic pollutants (POPs), such as polychlorinated biphenyls (PCBs) [10–15]. Though not produced in the U.S. since 1977, PCBs persist in the environment and concentrate in adipose tissue of organisms. They remain detectable in soil, air, water, and sediment, where they enter the food chain [16]. The primary route of PCB exposure today is through dietary intake of contaminated foods and through inhalation of airborne pollutants [17]. Increasing evidence from animal [10,11,12,18,19] and epidemiological research suggests that background exposure to PCBs is associated with type 2 diabetes, including studies examining National Health and Nutrition Examination Survey (NHANES) data [13], low dose PCB exposure [14,15], five-year prospective data from an elderly population in Sweden [20], the PCB-exposed population of Anniston, AL [21], a review of epidemiological studies from a National Toxicology Program Workshop [22], and prospective data from the Nurses' Health Study as part of a meta-analysis [23].

Nutrition is essential in the prevention and management of type 2 diabetes [6–9] and has been shown to modulate the toxicity of PCBs [19,24–29]. Consistent intake of fruits and vegetables has been associated with a healthy weight, positive antioxidant status, and a reduced risk of chronic diseases. Dietary antioxidants, such as vitamin C and the carotenoids, when consumed at adequate levels, can provide a balanced defense against the harmful effects of reactive oxygen species (ROS) [30]. Serum carotenoids, a family of lipophilic plant pigments with potent antioxidant activity, are considered a reliable biomarker of fruit and vegetable intake [31]. Serum responses to carotenoids have been reported to depend on a variety of factors, including the amount consumed, food matrix, and half-life variability of individual carotenoids [32]. The predominant carotenoids in human sera are α -carotene, β -carotene, β -cryptoxanthin, lycopene, lutein, and zeaxanthin [33]. Observational studies of carotenoids reveal inverse associations with type 2 diabetes [34], CVD [35,36], and all-cause mortality [37,38], although individual carotenoid effects have been inconsistent.

In the present study, we tested the hypothesis that fruit and vegetable intake, reflected by serum carotenoid concentrations, is associated with the reduced probability of developing type 2 diabetes in U.S. adults with elevated serum concentrations of PCBs 118, 126, and 153. The objective was to use the CDC database, National Health and Nutrition Examination

Survey (NHANES) 2003–2004 to establish whether serum carotenoid concentrations are associated with a reduced risk of developing type 2 diabetes in adult participants with elevated serum concentrations of PCB118, PCB126, and PCB 153.

2. Methods and materials

2.1. Procedure and study population

NHANES is a series of nationally representative, cross-sectional surveys of the civilian, non-institutionalized U.S. population. NHANES 2003–2004 survey procedures, laboratory assays, and ethics review board approval are published in detail [39]. Approval for our analysis of the NHANES data was granted by the University of Kentucky Institutional Review Board.

In NHANES 2003–2004, a random, 1/2 subsample was selected for fasting plasma glucose measurements, and a separate random, but overlapping, 1/3 subsample was selected for serum PCB measurements. Measurement of serum carotenoids was a fixed component of the mobile examination center (MEC) examination without dedicated subsample status.

For our study, we used the following inclusion criteria for a larger subpopulation ($n = 5,041$, Table 1): adults: 20 years of age examined at the MEC, having measurements for serum carotenoid concentrations, and either a fasting glycosylated hemoglobin value or history of type 2 diabetes diagnosis. A prototype individual represents this group. Mean values were drawn from this larger subpopulation. The prototype was a fifty year old non-Hispanic, white male, at the 50th percentile of PIR (2.23), the 50th percentile of body mass index (BMI) (27), and the 50th percentile serum carotenoid concentrations (1.7 $\mu\text{mol/L}$). Approximately 1200 of these subjects had at least one PCB concentrations measured.

The BMI of the prototype individual was varied to help understand the changing relationship between the variables in our models. The National Institutes of Health (NIH), National Heart, Lung, and Blood Institute (NHLBI) defines BMI, as weight (kg) divided by height squared (m^2) and its use, as a measure of body fat based on height and weight in adults [40]. The Center for Disease Control and Prevention (CDC) interprets BMI using standard weight status categories: <18.5 = underweight; 18.5 – 24.9 = normal weight; 25.0 – 29.9 = overweight; 30.0 – 39.9 = obese; 40.0 = extremely obese [43].

Socio-demographic covariates included gender, age, race/ethnicity (non-Hispanic white, non-Hispanic black, Mexican American, and other ethnicities), PIR, and BMI. PIR was determined by the ratio of total family income to poverty, as determined by the U.S. Department of Health and Human Services annual poverty guidelines. Women and non-Hispanic whites comprised over 50% of the sample (Table 1). Approximately 22% of participants reported being current cigarette smokers although 79–81% had serum cotinine levels >0.015 ng/mL, considered a positive indicator of passive or active smoking, thus we did not include smoking in our models.

2.2. Exposure variables

2.2.1 Serum carotenoids—Individual carotenoids were pooled and assessed as total carotenoids. The carotenoids of interest in this study were α -carotene, β -carotene, α -cryptoxanthin, β -cryptoxanthin, lycopene, lutein, and zeaxanthin. Lutein and zeaxanthin measures were combined within the same peak and consequently analyzed together in the NHANES analysis. Carotenoids were measured by absorbance at 450 nm using high performance liquid chromatography (HPLC) with multi-wavelength photodiode-array absorbance detection.

2.2.2. PCBs—PCBs were measured in serum samples and were lipid-standardized, representing the quotient of PCB concentration and total serum lipid content (mg/dL). This has been considered a better reflection of body burden in epidemiological studies. Three PCBs were selected for analysis representing one from each subclass: PCB118 mono-ortho-substituted, PCB126 non-ortho-substituted (coplanar or dioxin-like), and PCB153 di-ortho-substituted (non-coplanar or non-dioxin-like). To avoid bias at lower concentrations, PCBs were selected with $\geq 60\%$ of participants over the limit of detection (LOD) (see Online Supplementary Materials, Table S1). PCB118 and PCB153 had zero observations below the LOD. PCBs were measured in 5–10 ml serum by high-resolution gas chromatography/isotope-dilution high-resolution mass spectrometry.

2.3 Response variable

2.3.1 Type 2 Diabetes—Participants were evaluated for type 2 diabetes from the examination and questionnaire data files of NHANES. A positive finding of type 2 diabetes was found if one of two criteria was met: (1) a participant's glycosylated hemoglobin result was $\geq 6.5\%$; or (2) they answered “yes” to the question, “Have you ever been told by a doctor or health professional that you have diabetes or sugar diabetes”. Annually, the American Diabetes Association publishes the most current clinical guidelines for the diagnosis of diabetes, which include a glycosylated hemoglobin (A1C) $\geq 6.5\%$ [42].

2.4. Statistical analyses

All analyses were performed using SAS 9.2 (SAS Institute, Inc., Cary, NC). In the individual analysis, participants were categorized linearly by quantile for each PCB congener. In this manner, the slope of each PCB quantile represented the probability of chronic disease per total serum carotenoid concentration. Quantiles were categorized at 25th, 50th, 75th, and 90th percentiles; however, all analyses were set at the lower LOD of 60%, and were variable by compound and extractable volume. Variables in Table 1 were considered for inclusion in our models. If a variable was significant in one PCB model, it was included in all three models. Interactions as well as quadratic terms found to be significant ($p < 0.05$) were added to the models.

Logistic regression models included covariates for age, race/ethnicity, PIR, BMI, and gender, rather than NHANES subsample weights. This method of covariate modeling has been regarded as a good alternative in subpopulation methodology and a suitable compromise between efficiency and bias [43,44]. A p-value of < 0.05 was used to indicate statistical significance.

Survey logistic regression models were also run using the NHANES subsample weights (Online Supplementary Materials, Tables S2,S3,S4). The standard errors were calculated using Taylor series approximations, which rely upon all of the cases in the data file to arrive at appropriate estimates. Therefore, in order to use the full sample, the subpopulation options were activated with the survey logistic commands. The variable that identifies the subpopulation is coded 0 for cases that are excluded and 1 for cases that are included.

3. Results

Age was directly associated with the probability of type 2 diabetes in all analyses ($p < 0.01$). BMI was associated with an increased probability of disease with congener-specific effects observed for the obese in diabetes modeling.

Prevalence of type 2 diabetes was specific for each PCB: PCB 118 was 11.6%, PCB 126 was 11.5% and PCB 153 was 11.6%. All three PCBs were significantly associated with an increased probability of type 2 diabetes ($p < 0.01$) (Tables 2,3,4). The mean ($\pm$ SD) serum carotenoid concentration was 1.83 ± 0.87 $\mu\text{mol/L}$. Statistically significant interactions were identified between PCB 118 and serum carotenoids and PCB 126 and serum carotenoids ($p < 0.05$).

For models including PCB118, there was a lack of observable effect at any serum carotenoid concentration at the 25th PCB percentile (Figure 1) for the prototype individual at the 50th percentile BMI (27). A similar lack of effect was noted in the 25th first percentile of PCB126. Conversely, at higher concentrations of these two PCBs, the probability of type 2 diabetes declined with increasing serum carotenoid concentrations to levels similar to that observed at the 25th and 50th PCB percentiles.

Similar trends were noted for our prototype individual at the 90th percentile of BMI (Figure 2). For PCBs 118 and 126, diabetes probability was higher at the 75th and 90th percentiles. As serum carotenoid concentrations increased, diabetes risk diminished to levels similar to participants in the 25th and 50th PCB percentiles.

As an alternative analysis, subsample weights for subjects with PCB measurements and then NHANES subsample weights were used in survey logistic regression models (Online Supplementary Materials, Tables S2,S3,S4). These models found similar results.

While PCB153 was associated with an increased probability of T2DM, serum carotenoids did not correlate with any variable in this model nor were statistical interaction effects observed.

4. Discussion

These data support our hypothesis that increased serum carotenoids exhibited a protective effect in the probability of developing type 2 diabetes for this adult population with elevated serum concentrations of PCBs 118 and 126. PCB153, also considered relatively resistant to metabolism [45], however, showed no significant benefit of serum carotenoids at any concentration. To our knowledge, this is the first study to investigate associations among

serum carotenoids, a nutrition biomarker indicative of fruit and vegetable intake; serum concentrations of PCBs; and the probability of developing type 2 diabetes in a representative sample of U.S. adults.

In our analysis, increased serum carotenoids provided benefits in reducing the risk of type 2 diabetes for models at higher serum concentrations of PCBs 118 and 126 at both BMI percentiles. For those at the 90th percentile BMI (BMI 37) for both PCB 118 and 126, our models showed a greater decrease in probability of type 2 diabetes than the decrease observed at the 50th percentile (BMI 27). It is notable that the purpose of BMI is to assign morbidity and mortality risk status to individuals based on their weight (kg) to height (m²) ratio [46].

Carotenoid absorption and utilization is complex and known to be influenced by many factors, one of which is cytochrome P450 (CYP) induction. Carotenoids and their derivatives influence the activity of several nuclear families [47,48], and through gene regulation [49] exhibit modulating effects in diabetes [50]. Cross-sectional studies have reported an inverse association between serum carotenoids and the mean blood glucose status of participants with normal glucose tolerance and those with type 2 diabetes [36] and between total serum carotenoids and T2DM in nonsmokers [51]. Studies have also found a fairly consistent inverse correlation between BMI and carotenoid status [52,53]. In general, effects of individual carotenoids have been inconsistent, although nutrients seldom appear in the diet in isolation.

In supplement form, a pro-oxidant effect has been found and is not recommended [54,55]. The two studies cited most often for these effects, randomized controlled trials which supplemented the diet of smokers with β -carotene and either retinol (Beta-Carotene and Retinol Efficacy Trial (CARET)) [54] or α -tocopherol (Alpha-Tocopherol, Beta-Carotene Cohort (ATBC) Study) [55], were terminated early due to increased incidence of disease and deaths. Follow-up dietary analysis, however, revealed significantly lower disease risk in both studies, with reported high weekly fruit and vegetable intake and/or elevated dietary lycopene, lutein/zeaxanthin, beta-cryptoxanthin, and total carotenoids [56,57].

Of all the carotenoids, lycopene is the most effective antioxidant (58). Studies have shown protection of lycopene against PCB-induced reproductive dysfunction [59] and in glucose homeostasis. Lycopene supplementation when administered to rats concurrent with Aroclor 1254, a highly toxic mixture of 54% PCBs, prevented decreased expression of the glucose transporter protein, GLUT4, which normalized glucose uptake in skeletal muscle [60].

No one etiologic mechanism or pattern has been able to explain the association of PCBs with diabetes. Various studies have examined their capacity to disrupt normal hormone regulation with the pathogenesis of obesity [10–12,18, 61], insulin resistance [12,13,14], and nonalcoholic fatty liver disease (NAFLD) [62]. A synergistic interaction was observed between PCB153 and high-fat diet, increasing obesity and NAFLD, beyond any effect seen with high-fat diet alone. PCB153 with control diet had no effect on these parameters, suggesting the influence of PCBs on obesity and NAFLD may depend more on nutritional interactions with PCB congener than on either factor alone [62]. Other factors have been

shown to complicate studies; obesity alone is a risk factor for type 2 diabetes and increased body fat may suggest increased storage of and, therefore, exposure to PCBs. While cross-sectional surveys have established associations between POPs and diabetes [14], insulin resistance [15], and NAFLD [63], the influence of diet was not addressed in these studies.

While increasing evidence suggests that environmental pollutants increase susceptibility to type 2 diabetes, little research overall has been conducted on the effects of nutrition in mitigating this risk. PCBs and isoforms of β -carotene derived from foods and supplements were significant for type 2 diabetes among 543 environmental factor and genetic markers [3]. Endothelial cells treated with quercetin [64] or green tea catechins [65] were protected against coplanar PCB-induced inflammation. In vitro and in vivo supplementation of resveratrol diminished PCB77-induced oxidative stress in cells and adipose tissue by enhancing antioxidant signaling pathways associated with insulin and glucose tolerance [19]. Modifying the composition of dietary fatty acids from primarily omega-6 (linoleic acid) to omega-3 (alpha-linolenic acid) inhibited pro-inflammatory signaling pathways and completely blocked PCB-induced effects when pretreated with alpha-linolenic acid [24]. These and related findings may be best confirmed in longitudinal studies, which could prove critical in determining the magnitude of nutrition in preventing disease in pollutant-exposed populations.

Environmental PCB levels have been declining in the decades since PCB production was banned in the U.S., yet rates of obesity and type 2 diabetes have increased. It has been shown that these effects may still occur at low-dose, persistent exposures [14,15]. Dietary intake is a major route for PCB exposure [17], with one market study estimating daily PCB intake from typical American foods at 33 ng per day [66]. The foods highest in PCBs tend to be those with a lipid compartment; marine, mammal, and dairy foods [17]. Regular consumption of fruits and vegetables, which are naturally low in lipid content and high in antioxidants, would reduce exposure and protect against toxic insults. Reporter gene assays have identified fruits, vegetables, and herbs that may modulate PCB transformation and toxicity through CYP enzyme induction [67,68], or as aryl hydrocarbon receptor (AhR) agonists and antagonists [69]. More research is needed to understand how various ligands activate the AhR, and how this can result in clinically relevant outcomes [29].

While nutritional supplementation of carotenoids is not advised, the benefits of whole food nutrition at normal physiological levels should be promoted. Diet has been repeatedly shown to mitigate the effects of progressive chronic diseases, especially type 2 diabetes [70, 71], on persons at elevated risk. Studies with participants consuming diets that are high in vegetables and comprised of a variety of fruits and vegetables [72], increased servings of fruits, particularly blueberries, grapes, and apples [73], and Mediterranean diets, also known to be high in fruits, vegetables, olive oil, nuts, and grains [74,75], are associated with a reduced risk of type 2 diabetes. Two studies of the Mediterranean diet supplemented with extra-virgin olive oil or nuts in a Spanish cohort showed reduced CVD risk factors in persons with type 2 diabetes [74] and reduced risk of diabetes in older individuals (55 to 80 yrs) at high CVD risk [75]. Women with history of gestational diabetes, a condition placing them at greatly increased diabetes risk subsequent to pregnancy [76], were placed on one of three healthy meal plans [77], and experienced a 40% (alternate Mediterranean), 46%

(Dietary Approaches to Stop Hypertension), or 57% (alternate Healthy Eating Index) lower risk of type 2 diabetes. Current recommendations for an 1800–2000 calorie meal plan are to consume eight to nine servings of fruits and vegetables per day for optimal health [78].

There are limitations with the current study, and results should be interpreted with caution. Due to the cross-sectional nature of the study, it was not possible to determine time or extent of the original chemical exposures. Direction of causation cannot be determined, allowing for the possibility that perturbations of metabolic disease could affect serum concentrations. Neither was it possible at this time to measure all potential confounders that could influence results. Last, other POPs, including PCBs not investigated in this study, may contribute to the compromised health of these participants.

This study has several strengths as well. NHANES 2003–2004 is representative of the U.S. population for those two years. Results are based on actual clinical parameters and nutritional examination to assess health status, whereas most research studies have examined one pollutant and one nutrient at concentrations that may not reflect true environmental risk ratios.

Nutrition is an ideal modulator of PCB toxicity. PCBs are widely known to cause oxidative stress [10–12,18,19] and exposure to them continues, primarily through dietary intake of contaminated foods and through inhalation of airborne pollutants. Chronic PCB exposure is associated with a persistent inflammatory state associated with diseases, such as type 2 diabetes [13–15,20–23]. Daily exposure to foods rich in beneficial nutrients, such as carotenoid-containing fruits and vegetables, may provide a dynamic barrier against the chemical, physical, and biological stressors that are a part of today's life. Further studies are needed to advance these findings.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

A1C	glycosylated hemoglobin
AhR	Aryl hydrocarbon receptor
ATBC	Alpha-Tocopherol, Beta-Carotene Cancer Prevention Trial
BMI	Body mass index
CARET	Beta-Carotene and Retinol Efficacy Trial
CDC	Center for Disease Control and Prevention

CVD	Cardiovascular disease
CYP	Cytochrome P450
HDL	High density lipoprotein
LOD	Limit of detection
MEC	Mobile examination center
NAFLD	nonalcoholic fatty liver disease
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
PCBs	Polychlorinated biphenyls
PIR	Poverty income ratio
POPs	Persistent organic pollutants
T2DM	Type 2 diabetes mellitus

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Figure 1.

Logistic model for adult NHANES 2003–2004 participants for the prototype individual: 50 year old white male at the 50th percentile poverty income ratio (2.23), 50th percentile body mass index (BMI 27), 50th percentile serum carotenoid concentrations (1.7 $\mu\text{mol/L}$). All three PCBs were positively associated with an increased probability of type 2 diabetes ($p < 0.01$). (A) PCB118. (B) PCB126. (C) PCB 153. At higher PCB percentiles for PCB 118 and 126, increasing serum carotenoid concentrations were associated with a smaller probability of type 2 diabetes ($p < 0.05$). No serum carotenoid benefit and no statistical interaction effects were observed for PCB 153.

Figure 2.

Logistic model for adult NHANES 2003–2004 participants for the prototype individual: 50 year old white male at the 50th percentile poverty income ratio (2.23), 90th percentile body mass index (BMI 37), 50th percentile serum carotenoid concentrations (1.7 $\mu\text{mol/L}$). All three PCBs were associated with an increased probability of type 2 diabetes ($p < 0.01$). (A) PCB118. (B) PCB126. (C) PCB 153. Similar results at higher PCB percentiles for PCB 118 and 126, increasing serum carotenoid concentrations were associated with a smaller probability of type 2 diabetes ($p < 0.05$). No serum carotenoid effects were observed for PCB 153.

Table 1

Characteristics of study participants from National Health and Nutrition Examination Survey (NHANES), 2003–2004, Age 20 years of age ($n = 5041$).

Characteristic	Value ^a
Male (%)	2418 (47.97)
Age (y), mean	50.85 ($\pm$ 9.70)
Race/ethnicity (%)	
Non-Hispanic white	2689 (53.34)
Non-Hispanic black	994 (19.72)
Mexican American	985 (19.54)
Other race/ethnicity	373 (7.40)
PIR ^b , mean	2.57 ($\pm$ 1.59)
BMI (kg/m ²) (%)	
<18.5	170 (1.39)
18.5–24.9	1408 (27.93)
25.0–29.9	1631 (32.35)
30.0–39.9	1299 (25.77)
40.0	239 (4.74)
Total carotenoids (umol/L), mean	1.83 ($\pm$ 0.87)
Use dietary supplements (%)	2658 (52.80)
Current cigarette smoker (%)	1131 (22.48)
Serum cotinine (%)	
0.015 ng/mL	3632 (81.14)
<0.015 ng/mL	844 (18.86)
Physical activity (%)	
Sedentary	1379 (47.97)
Low activity	498 (17.32)
Moderate to vigorously active	995 (34.61)
Alcohol consumption (%)	
Non-drinker	980 (19.44)
Non-excessive drinker	1664 (33.01)
Excessive drinker	1032 (20.47)

^aValues shown are mean $\pm$ SD or n (%).

^bAbbreviations: PIR: poverty income ratio, BMI: body mass index

Table 2

Logistic model for adult NHANES 2003–2004 participants with serum PCB118 concentrations.

Type 2 Diabetes Mellitus (<i>n</i> = 1195)		
Parameter	Estimate^b	P-value
Male	0.2573	0.0127
Age	0.0360	<0.0001
Race/Ethnicity		
Mexican American	1.1257	0.0829
Other Hispanic	0.7815	0.3398
Non-Hispanic White	0.3419	0.5862
Non-Hispanic Black	0.5503	0.4049
PIR ^a	−0.0375	0.5780
PCB118	0.1222	0.0008
PCB118*118	−0.00257	0.0012
BMI	0.0421	0.0127
Serum Carotenoids	0.0677	0.7338
PCB118*Sera Carotenoids	−0.0306	0.0161
PCB118*PCB118*Sera Carotenoids	0.000538	0.0023
PCB118*PCB118*BMI	0.000024	0.0118

^a Abbreviations: PIR: poverty income ratio, BMI: body mass index

^b Logistic regression coefficient estimates including covariates for age, race/ethnicity, PIR, BMI, and gender

Table 3

Logistic model for adult NHANES 2003–2004 participants with serum PCB126 concentrations.

Type 2 Diabetes Mellitus (<i>n</i> = 1188)		
Parameter	Estimate	P-value
Male	0.5922	0.0041
Age	0.0322	<0.0001
Race/Ethnicity		
Mexican American	1.2236	0.0617
Other Hispanic	1.0374	0.2087
Non-Hispanic White	0.6019	0.3465
Non-Hispanic Black	0.7885	0.2391
PIR ^a	−0.0405	0.5486
PCB126	0.0664	0.0011
PCB126*126	−0.00045	0.0013
BMI	0.0496	0.0022
Serum Carotenoids	0.0695	0.7807
PCB126*Sera Carotenoids	−0.0155	0.0746
PCB126*PCB126*Sera Carotenoids	0.000139	0.0124

^a Abbreviations: PIR: poverty income ratio, BMI: body mass index

^b Logistic regression coefficient estimates including covariates for age, race/ethnicity, PIR, BMI, and gender

Table 4

Logistic model for adult NHANES 2003–2004 participants with serum PCB153 concentrations

Type 2 Diabetes Mellitus (<i>n</i> = 1204)		
Parameter	Estimate^b	P-value
Male	0.3542	0.0708
Age	0.0348	<0.0001
Race/Ethnicity		
Mexican American	1.1303	0.0809
Other Hispanic	0.6129	0.4542
Non-Hispanic White	0.2691	0.6673
Non-Hispanic Black	0.3593	0.5876
PIR ^a	−0.0301	0.6496
PCB153	0.0158	0.0051
PCB153*153	−0.00005	0.0266
BMI	0.0647	<0.0001
Serum Carotenoids	−0.1597	0.1815

^a Abbreviations: PIR: poverty income ratio, BMI: body mass index

^b Logistic regression coefficient estimates including covariates for age, race/ethnicity, PIR, BMI, and gender