

Sex-Specific Adverse Association of Endocrine Disrupting Chemicals on Peak Anaerobic Power and Skeletal Muscle Mass of Active Young Adults

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Abstract

dos Santos, JM, Kennedy, T, Zhu, J, McPike, A, Ray, M, Demirci, J, and Dechant, J. Sex-specific adverse association of endocrine disrupting chemicals on peak anaerobic power and skeletal muscle mass of active young adults. *J Strength Cond Res* XX(X): 000–000, 2025—Endocrine disrupting chemicals, such as bisphenol A (BPA) and bisphenol S (BPS), and polycyclic aromatic hydrocarbons (PAHs) are a broad class of industrial compounds that induce adverse physiologic outcomes dependent on biologic sex. These chemicals stimulate oxidative stress and impair pathways involved in anaerobic and aerobic energy generation, key drivers of athletic performance. This study aims to analyze the association of BPA, BPS, 2-naphthol (a marker of PAH), and oxidative stress with peak anaerobic power (PAP) and anthropometric characteristics. Peak anaerobic power was assessed in active men and women ($n = 54$, age 20 ± 1.4 , body mass index $25.4 \pm 3.8 \text{ kg}\cdot\text{m}^{-2}$) using a vertical jump test. Fasting levels of urinary BPA, BPS, 2-naphthol, oxidative stress, blood glucose, and muscle mass were assessed by ELISA, capillary sample analysis, and body composition analyzer, respectively. Peak anaerobic power was negatively associated with BPA and positively with percentage of skeletal muscle and oxidative stress ($p < 0.05$). Multivariate regression, moderated by nonmodifiable variables associated with PAP such as sex, height, and age, indicated that only BPA was a predictor of PAP ($\beta = -0.186$, $p = 0.021$). Relationships of variables were assessed in each sex, and PAP was negatively associated with levels of BPA and 2-naphthol in woman. Multivariate regression confirmed that those variables are predictors of PAP ($p < 0.01$). Results support the idea of a sex-specific physiologic impact of BPA and 2-naphthol on PAP, independently of levels of oxidative stress and percentage of muscle. Controlling BPA and 2-naphthol exposure could be a potential strategy to improve PAP in female athletes.

Key Words: bisphenol, 2-naphthol, percentage of muscle, athletic performance, oxidative stress

Introduction

Endocrine disrupting chemicals (EDCs) are natural or man-made broad class of compounds that can mimic, impair, or interfere with hormonal activity (13). They are found in many products used on a routine basis, such as food packaging, pesticides, pharmaceutical agents, and personal care products (5,36). Large-scale epidemiologic studies have associated levels of EDCs in human specimens with the risk of hormone-related health conditions (5,9). Polycyclic aromatic hydrocarbons (PAHs) and bisphenols are EDCs widely present in the environment and can either be inhaled through air particles and/or ingested through food or beverage (10,11,31). Polycyclic aromatic hydrocarbons, formed by incomplete combustion of oil, gas, garbage, and coal, are present in vehicle exhaust, tobacco, and charbroiled food (10,11,14,31). Bisphenols, such as bisphenol A (BPA) and bisphenol S (BPS), are components used in the manufacture of food packaging, and they can leach into the food/beverage

content and be ingested. Bisphenol A and BPS can also be released into the atmosphere through thermal destruction of plastic products and be inhaled (5,24,33).

On entering the human body, bisphenols and PAHs can be metabolized by the liver (15,32). However, in excess, because of their lipophilic property, EDCs can easily cross the cell membrane and affect various cellular pathways (15,31). In skeletal muscle cells, these chemicals induce oxidative stress and impair glucose metabolism and mitochondria homeostasis, which are key elements in anaerobic and aerobic energy generation (24,31,33,34). Moreover, BPA, BPS, and PAHs can affect the activity of hormones that regulate glucose metabolism and skeletal muscle hypertrophy such as testosterone and estrogen, with potential sex-specific differences in the extent of the disruption (3,12,20,31,34).

Skeletal muscle mass, its distribution, and the ability of skeletal muscle cells to generate energy are essential factors in athletic performance and key drivers of sex differences in strength, aerobic, and anaerobic capacity (19,27). Peak anaerobic power (PAP), defined as the highest power generated by skeletal muscle during any 3–5 seconds interval, is a critical element that dictates sex-specific differences in athletic performance (4,17,28,37). The endocrine system controls several pathways that underlie skeletal muscle homeostasis, growth, and differentiation; therefore, hormonally active xenobiotics such as EDCs could affect athletic

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performance. Although the effect of EDCs on aerobic capacity was previously studied by our group (24), their impact on PAP has yet to be characterized. Therefore, the aim of this study is to analyze the associations between BPA, BPS, 2-naphthol (a marker of PAH, 11), and oxidative stress with PAP in active female and male young adults.

Methods

Experimental Approach to the Problem

This study aimed to analyze the associations between levels of urinary EDCs and oxidative stress with PAP in physically active young adults. Because EDCs can affect the activity of hormones with sex-specific differences in the extent of the disruption, men and women were recruited to the study. A validated test to assess PAP was conducted in subjects (4,6,22,28). Urinary BPA, BPS, and 2-naphthol levels were assessed after overnight fasting to rule out the diet's acute effect on EDCs exposure. Peak anaerobic power results were associated with urinary EDCs, oxidative stress, body composition, and blood glucose.

Subjects

Healthy, physically active students, men ($n = 24$) and women ($n = 30$) between 18 and 23 years old, attending Fairmont State University in Fairmont (FSU), West Virginia, participated in the study. The university's campus is located 0.4 miles from Fairmont downtown in an area not classified as a food desert according to the U.S. Environmental Protection Agency environmental justice tool. The environmental burden indicators, such as particulate matter 2.5 micrometers in diameter (PM 2.5), ozone, diesel PM, and toxins released in the air in the area 5 miles surrounding the FSU campus, are 40–80% lower than the national average. The risk of wildfire at Fairmont is <1%. Subjects of this study were living on campus or within 5 miles of campus. The inclusion criteria required subjects to perform exercise at least 5 times a week for at least 2 years, be familiar with the vertical jump test, and be healthy according to the Physical Activity Readiness Questionnaire. Subjects were asked not to change their daily diet or routine except for fasting for at least 12 hours (water only) before the sample collection date, performed between 7 and 9 AM. The study was approved by the institutional review board of FSU and the University of Pittsburgh (protocol ID STUDY22060168). Written informed consent was obtained from each subject before project participation.

Procedures

Subject's urine samples were collected after overnight fasting, before the PAP test, and stored at -20°C . InBody 570 Body Composition Analyzer (Philadelphia, PA) was used to assess body composition (percentage of body fat and skeletal muscle) as previously described (20,23,30). Blood glucose levels were assessed through a True Track™ glucose meter (Starter Kit, True Metrix, Fort Lauderdale, FL) (24). Blood pressure was assessed before PAP assessment using standard guidelines, wherein the subject will remain in a sitting position after a 5-minute rest period before the assessment. Three blood pressures were recorded 1–2 minutes apart by a Oron 3 Series blood pressure monitor. The average of the last 2 measurements was recorded.

Peak Anaerobic Power Assessment. PAP was assessed using a vertical jump test on the Just Jump System mat (SimpliFaster, Alachua, FL) according to a validated protocol (6,22). All the subjects were familiar with the test. Subjects began in a normal anatomical start position. Three trials of jumps were completed. Subjects were instructed to perform a countermovement jump with free arm movement, jumping as high as possible and landing back on the Just Jump System mat. Between each trial, subjects took a 3- to 5-minute rest period to minimize fatigue. The highest jump height out of the 3 trials was recorded. No additional restrictions were placed on arm position, and subjects were allowed to use their arms to generate momentum during the jump. The Sayer equation (Peak Power (W) = $60.7 \times (\text{jump height cm}) + 45.3 \times (\text{body mass [kg]}) - 2055$) was used to calculate peak anaerobic muscular power (4,28).

Bisphenol A, Bisphenol S, 2-Naphthol and 8-Isoprostane Analyses. Urine samples were thawed, and BPA, BPS, 2-naphthol, and 8-isoprostane (a marker of oxidative stress) were assessed by ELISA kit (Detroit R&D, Inc, Detroit, MI), as previously described by our group (8,24). Endocrine disrupting chemical levels in urine were normalized by creatinine [(One-step Creatinine Urinary Assay; Detroit R&D, Inc, Detroit, MI)] to account for the impact of urine dilution on chemical levels as standardized by several investigators (9,10,18,24).

Statistical Analyses

Data were analyzed by SPSS software 29 (IBM, Chicago, IL). Outliers were identified using a boxplot graph, and values above or below 3 SDs were deleted. Levene's test for equality of variance was used to test variable distribution, and comparisons between men and women were tested by independent t test. The Shapiro-Wilk normality test was used to assess the distribution of the data. Associations between variables were tested by non-parametric correlations using Kendall's tau b test. Potential predictors of PAP were selected based on the significance obtained at correlation analyses ($p < 0.05$). Multivariate linear regression was performed with PAP as the dependent variable and the following modifiable PAP predictors and EDCs as independent variables: percentage of skeletal muscle, 8-isoprostane, BPA, BPS, and 2-naphthol. To assess whether these associations differed by the nonmodifiable variables sex, age, and height, we conducted moderation analyses. Also, relationships between variables separately by sex (i.e., male and female) were tested by applying the nonparametric correlation Kendall's tau b test. We completed multivariate linear regression with PAP as the dependent variable and BPA and 2-naphthol as independent variables, and height as a moderator, among women only. p -values < 0.05 was considered statistically significant.

Results

Results for non-normalized urinary EDCs are presented in Supplemental Digital Content (see Table 1, <http://links.lww.com/JSCR/A671>). Bisphenol A level normalized by creatinine was higher in women, while BPS and 8-isoprostane normalized by creatinine were higher in men. Body mass index, body mass height, systolic blood pressure, percentage of skeletal muscle mass, and PAP were higher in men, while percentage of fat was higher in women. No differences were found between sex on

Table 1**Levels of urinary endocrine disrupting chemicals and oxidative stress, peak anaerobic power, and subjects characteristics.*†**

Variables	Sex			95% conf. int. of the diff			
	Male	Female	<i>p</i>	Min	Max	Upper	Lower
BPA (ng·mg ⁻¹)	10.40 ± 9.21	19.10 ± 16.85	0.03*	0.71	61.64	-16.42	-0.97
BPS (ng·mg ⁻¹)	11.76 ± 9.60	4.13 ± 3.71	0.01**	0.27	30.87	2.04	12.94
2-NAP (ng·mg ⁻¹)	6.32 ± 8.31	9.10 ± 10.73	0.32	0.03	34.30	-8.13	2.57
8-Iso (ng·mg ⁻¹)	0.86 ± 0.87	0.18 ± 0.26	<0.01**	0.025	3.96	0.27	1.05
Crea (mg·ml ⁻¹)	1.50 ± 1.05	1.34 ± 0.70	0.26	0.03	3.81	-0.35	0.66
Age (years)	19.61 ± 1.41	20.19 ± 1.28	0.10	18.00	23.00	-1.32	0.15
Body Mass (kg)	91.02 ± 13.04	69.61 ± 12.30	<0.01**	54.27	115.50	14.43	28.37
Height (m)	1.84 ± 0.06	1.69 ± 0.07	<0.01**	1.55	1.96	0.11	0.19
BMI (kg·m ⁻²)	26.72 ± 2.80	24.48 ± 4.15	0.01*	17.90	36.10	0.22	4.24
Fat (%)	15.07 ± 5.60	25.37 ± 8.38	<0.01**	6.80	46.70	-14.34	-6.24
Musc. (%)	48.86 ± 3.40	41.64 ± 4.90	<0.01**	29.71	53.95	4.82	9.60
Syst. BP (mm Hg)	128.41 ± 12.53	118.61 ± 10.41	0.02*	100.00	156.00	3.24	16.34
Dia BP (mm Hg)	72.73 ± 9.68	75.16 ± 10.44	0.40	53.00	92.00	-8.10	3.23
PAP (watts)	5,755.88 ± 778.96	3,442.83 ± 491.14	<0.01**	2,286.04	7,236.89	1963.05	2,663.04
Blood glucose (mg·dl ⁻¹)	85.61 ± 10.91	89.42 ± 8.50	0.80	71.00	113.00	-9.10	1.48

*BPA = urinary bisphenol A; BPS = bisphenol S; 2-NAP = 2-naphthol; 8-Iso = 8-isoprostane normalized by creatinine, respectively. BMI = body mass index; Fat = percentage of body fat; Musc. = muscle; Syst BP = systolic blood pressure; Dia BP = and diastolic blood pressure; PAP = peak anaerobic power; blood gluc. = fasting blood glucose. Data are presented as mean ± SD.

†**p* < 0.05; ** <0.01 by independent *t* test.

urinary creatinine levels, age, diastolic blood pressure, and blood glucose (Table 1).

Correlations between all examined variables are presented in Table 2. The relationships between PAP and 2-naphthol, BPA, and muscle percentage overall (men and women) and stratified by sex are also presented in Figure 1. Peak anaerobic power was negatively associated with BPA (*p* < 0.05), age, height, and percentage of fat (*p* < 0.01), and positively associated with 8-isoprostane and percentage of muscle (*p* < 0.01). Levels of BPA were positively associated with blood glucose (*p* < 0.01) and age (*p* < 0.05). Bisphenol A was positively associated with 8-isoprostane levels. 2-Naphthol and 8-isoprostane were negatively associated with age (*p* < 0.05). 8-Isoprostane was also positively associated with height (*p* < 0.01). Blood glucose was positively associated with age and percentage of fat and negatively associated with the percentage of skeletal muscle (*p* < 0.01).

Multivariate regression of modifiable variables (BPA, BPS, 2-naphthol, percentage of muscle mass, and 8-isoprostane) with PAP was statistically significant ($R^2 = 0.614$, R^2 adjusted =

0.556, $F = 10.5$, $p < 0.001$) and moderated by sex ($R^2 = 0.814$, R^2 adjusted = 0.779, $F = 23.2$, $p < 0.001$) or by sex, age, and height ($R^2 = 0.868$, R^2 adjusted = 0.833, $F = 24.7$, $p < 0.001$). As given in Table 3, unadjusted multivariate regression of all the modifiable variables predicted PAP; however, when moderated by sex, BPA and 2-naphthol predicted PAP, and when further moderated by sex, age, and height, only BPA was a PAP predictor.

The associations between variables for men and women are presented in Table 4 and Figure 1D–I. In men, PAP was negatively associated with age (*p* < 0.01). In women, PAP was negatively associated with levels of BPA and 2-naphthol (*p* < 0.05) and positively with height (*p* < 0.05). In men, levels of BPA were negatively associated with BPS (*p* < 0.05) and positively associated with blood glucose (*p* < 0.05). In women, BPA was positively associated with oxidative stress (*p* < 0.01) and age (*p* < 0.05). Blood glucose was negatively associated with muscle mass percentage (*p* < 0.01) and positively with fat percentage (*p* < 0.01). Because only 1 variable was associated with PAP in men, multivariate regression was not conducted for this group. In women,

Table 2**Association between peak anaerobic power, urinary levels of endocrine disrupting chemicals, and oxidative stress, and all subjects characteristics.*†**

	PAP (Watts)	BPA (ng·mg ⁻¹)	BPS (ng·mg ⁻¹)	2-NAP (ng·mg ⁻¹)	8-Iso (ng·mg ⁻¹)	B. Gluc (mg·dl ⁻¹)	Age (yr old)	Height (m)	Fat (%)	Musc. (%)
PAP (watts)	1.00									
BPA (ng·mg ⁻¹)	-0.23*,‡	1.00								
BPS (ng·mg ⁻¹)	0.19	-0.21	1.00							
2-NAP (ng·mg ⁻¹)	-0.19‡	-0.02	-0.16	1.00						
8-Iso (ng·mg ⁻¹)	0.33**	0.01	0.32**	-0.05	1.00					
B. Gluc (mg·dl ⁻¹)	-0.16	0.30**	-0.11	0.09	-0.11	1.00				
Age (years)	-0.34**	0.23*	0.07	0.08	-0.14	0.22*	1.00			
Height (m)	0.56**,‡	-0.11	0.19	-0.22*	0.31**	-0.16	-0.29**	1.00		
Fat (%)	-0.37**	0.19	-0.08	0.05	-0.20	0.27**	0.18	-0.41**	1.00	
Musc. (%)	0.42**,‡	-0.18	0.01	-0.06	0.21	-0.29**	-0.18	0.42**	-0.93**	1.00

*Urinary bisphenol A, bisphenol S, 2-naphthol and 8-isoprostane normalized by creatinine (BPA, BPS, 2-NAP, and 8-Iso, respectively), age, height, percentage of body fat (Fat) and muscle (Musc.) and fasting blood glucose (blood gluc). Correlation coefficient *p* is presented on the table.

†**p* < 0.05; ** <0.01 by Kendall's tau *b* correlation test.

‡Also presented in Figure 1.

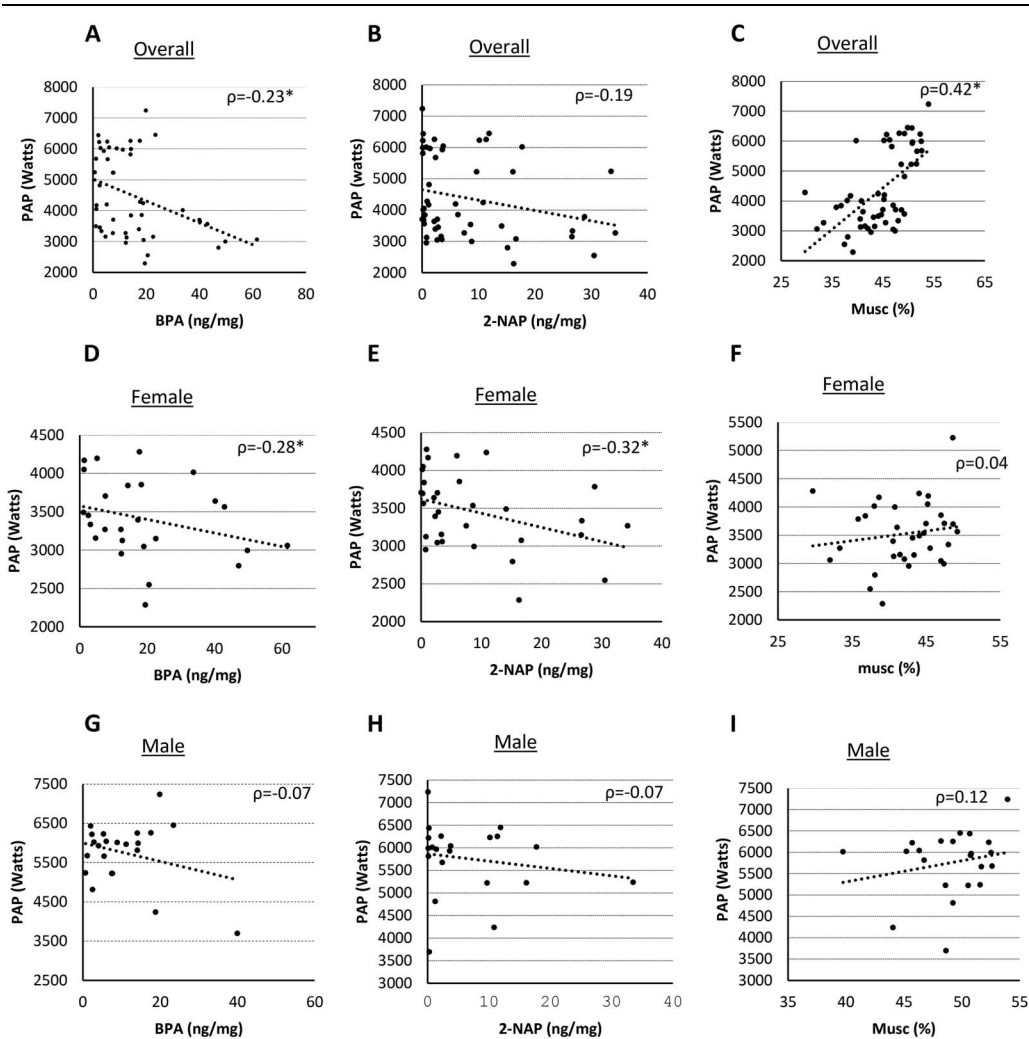


Figure 1. Peak anaerobic power (PAP) with bisphenol-A, 2-naphthol (2-NAP), and percentage of skeletal muscle (Musc %): Overall (women and men together), women and men association of PAP to BPA (A, D, G), 2-NAP (B, E, H), and Musc (%) (C, F, I) by Kendall's tau *b* correlation test. Correlation coefficient ρ is presented. * $p < 0.05$.

unadjusted multivariate regression with PAP as the dependent variable and BPA and 2-naphthol as predictors were statistically significant ($R^2 = 0.339$, R^2 adjusted = 0.279, $F = 5.6$, $p < 0.05$). When height was included as a moderator, the model improved ($R^2 = 0.514$, R^2 adjusted = 0.445, $F = 7.413$, $p < 0.001$), confirming that BPA and 2-naphthol ($\beta = -0.429$, $p = 0.010$) were negative predictors of PAP (Table 5).

Discussion

The adverse impacts of EDCs on the human body depend on chemical dose, frequency of exposure, age, and sex (3,9,13,14). Findings from this study support a sex-specific impact of BPA and 2-naphthol on PAP, which opens the window for further inquiry into EDCs as one of the multiple modifiable mediators of sex differences in athletic performance. To the best of our knowledge,

Table 3

Multivariate regression: peak anaerobic power as dependent variable and endocrine disrupting chemicals and oxidative stress as independent variables of all subjects.*

	Unadjusted			Adjusted by sex			Adjusted by sex, age height		
	Stand. coef. β	T	Sig	Stand. coef. β	T	Sig	Stand. coef. β	t	Sig
Constant		1.10	0.28		5.40	<0.001		2.15	0.04
BPA/crea (ng·mg ⁻¹)	-0.26	-2.21	0.03*	-0.22	-2.63	0.01**	-0.19	-2.44	0.02*
2-NAP/crea (ng·mg ⁻¹)	-0.23	-2.04	0.05*	-0.19	-2.40	0.02*	-0.14	-1.95	0.06
Musc. (%)	0.28	2.23	0.03*	0	-0.00	0.99	-0.10	-1.02	0.31
BPS/crea (ng·mg ⁻¹)	0.32	2.81	0.01*	0.10	1.17	0.25	0.08	1.04	0.31
8-iso/crea (ng·mg ⁻¹)	0.28	2.38	0.02*	0.09	1.02	0.31	0.09	1.12	0.27

*Bisphenol A, bisphenol S, 2-naphthol and 8-isoprostane (BPA, BPS, 2-NAP, and 8-iso, respectively) and percentage of skeletal muscle (Musc.) with PAP as dependent variable unadjusted and moderated by sex (male = 1 and female = 2), or the combination of sex, age, and height. Standard coefficient beta (Stand. coef. β). * $p < 0.05$, ** < 0.01 . Dependent variable: peak anaerobic power (watts).

Table 4**Associations between peak anaerobic power, urinary endocrine disrupting chemicals and oxidative stress levels, and sample characteristics in men and women.***

Variables	PAP (watts)	BPA (ng·mg ⁻¹)	BPS (ng·mg ⁻¹)	2-Nap (ng·mg ⁻¹)	8-iso (ng·mg ⁻¹)	B gluc (mg·dl ⁻¹)	Age (years old)	Height (m)	Fat (%)	Musc. (%)
Male										
PAP (watts)	1.00	-0.07	0.28	-0.07	0.07	-0.04	-0.47**	0.20	0.07	0.12
BPA (ng·mg ⁻¹)	-0.28*	1.00	-0.43*	-0.15	-0.11	0.33*	0.04	0.01	-0.12	0.16
BPS (ng·mg ⁻¹)	-0.15	0.11	1.00	-0.38*	0.07	-0.28	-0.38*	0.04	0.12	-0.08
2-NAP (ng·mg ⁻¹)	-0.32*	0.04	-0.06	1.00	0.07	0.15	0.17	-0.16	-0.06	0.02
8-iso (ng·mg ⁻¹)	0.01	0.41**	0.23	-0.03	1.00	0.05	-0.07	-0.09	0.04	-0.09
B. Gluc (mg·dl ⁻¹)	0.02	0.22	0.24	0.02	0.08	1.00	0.21	-0.08	-0.09	0.05
Age (years)	-0.26	0.27*	0.45**	-0.06	0.12	0.09	1.00	-0.3	-0.01	-0.01
Height (m)	0.27*	0.05	-0.07	-0.28*	0.08	-0.05	-0.15	1.00	-0.07	0.04
Fat (%)	-0.10	0.14	0.21	-0.02	0.16	0.42**	-0.18	-0.20	1.00	-0.93**
Musc. (%)	0.04	-0.12	-0.23	0.02	-0.15	-0.41**	0.17	-0.18	-0.95**	1.00
Female										

*Peak anaerobic power (PAP), urinary bisphenol A, bisphenol S, 2-naphthol and 8-isoprostane (BPA, BPS, 2-NAP, and 8-iso, respectively) age, body mass height, body mass index (BMI), percentage of body fat (Fat), percentage of body muscle (Musc), and fasting blood glucose (blood gluc). Correlation coefficient ρ is presented on the table. * $p < 0.05$; ** < 0.01 by Kendall's tau b correlation test.

this is the first study providing evidence that indicates that PAP could exhibit susceptibility to BPA and PAHs toxicity.

Apart from the technique, tactics, and skill, athletic performance is dependent on the integrated status of physiologic mechanisms of body systems, namely skeletal muscle (4,19,27). Studies have shown that muscle fiber type, high percentage of muscle mass, and skeletal muscle substrate availability (such as muscle glycogen) affect PAP (19,27). Men tend to have higher PAP than women because of several factors, including increased skeletal muscle mass and decreased overall fat accumulation (19,21,27), which is also found in the sample population of this study.

Extrinsic factors, including nutrition, climate elements, and exposure to toxicants, are known modifiable factors that affect physiologic outcomes (16,17,23,24,29,37). Although the effect of the first 2 aforementioned factors on athletic performance has received substantial attention from the scientific community (16,17,23,29,37), scant research has been performed on environmental toxicants. Based on our previous findings showing that urinary level of BPA was highly associated with a decreased ability of active subjects to run for 30 minutes, our group concluded that BPA could affect aerobic capacity and, therefore, athletic performance (24). The results presented here strengthen the hypothesis that BPA plays a major role in performance by

being inversely related to PAP in men and women and acts as a predictor of PAP even when key nonmodifiable variables such as age, sex, and height were used as a mediator.

For decades, awareness of the adverse health effects of BPA and BPS has been evident, and even with this knowledge, these chemicals are abundantly used by the food packaging industry (3,5,12,34,36). Despite the adverse effect of BPA and BPS on testosterone secretion (34), these chemicals are known as “environmental estrogen” because of their heavy impact on estrogen activity, which negatively affects the female reproductive system (3). Bisphenol A and BPS effects on estrogen hinder skeletal muscle cell differentiation, impairing skeletal muscle growth in women to a higher magnitude than men (3,38). Sex-dependent differences in insulin action are also manifested, because women generally exhibit greater insulin sensitivity, mainly due to the influence of estrogen (3). The impact of BPA on estrogen might be the basis for justifying our current findings regarding the potential role of BPA as a negative predictor for PAP in women, but not in men. The result of this study strengthens the assumption of the interaction between BPA and physical performance in female athletes and opens the window for further inquiries on potential mediators of BPA-adverse action, such as estrogen and insulin.

Polycyclic aromatic hydrocarbons are a group of chemicals that can be found in plastics and rubbers, and their exposure to humans is mainly through inhalation produced during the incomplete combustion of organic materials (10,11). Naphthalene is the main PAH component of PM 2.5. Once in the human body, naphthalene is metabolized to 2-naphthol (10). Sun et al. (31) tested the association of 8 types of PAHs, including 2-naphthol, on female and male skeletal muscle mass and anaerobic power. The authors used grip strength to assess anaerobic power. They found that urinary level of 2-naphthol was negatively associated with skeletal muscle mass in men, not women. No association was found between 2-naphthol and anaerobic power (31). The authors further strengthen the assumption that the male-specific effect of PAHs involves testosterone levels by showing that PAHs impair testosterone secretion in an animal model (31). In contrast, 2-naphthol was not related to the percentage of muscle mass in both sexes in the present study, but it was negatively associated with PAP in women. Sample characteristics might explain this divergence, because Sun et al. tested the hypothesis in subjects

Table 5**Multivariate regression: Peak anaerobic power as dependent variable and endocrine disrupting chemicals as independent variables of female subjects.***

Female	Unadjusted			Adjusted by height		
	Stand. coef. β	t	Sig	Stand. coef. β	T	Sig
Constant		24.30	<0.01		5.40	<0.01
BPA/crea (ng·mg ⁻¹)	-0.33	-1.90	0.07	-0.22	-2.63	0.01*
2-NAP/crea (ng·mg ⁻¹)	-0.50	-2.87	0.01*	-0.20	-2.40	0.02*

*Peak anaerobic power (PAP) predictor in female subjects: Multiple regression with PAP as dependent variable with urinary bisphenol A and 2-naphthol (BPA and 2-NAP, respectively) unadjusted and moderated by height. Standard coefficient beta (Stand. coef. β). * $p < 0.05$. Dependent variable: peak anaerobic power (watts).

with a vast age range, including women in postmenopause (lower levels of sex hormone estrogen), and the subjects in this study were between 18 and 24 years old and active. The impact of 2-naphthol on estrogen levels and skeletal muscle content in women awaits further scientific inquiry.

Sex differences in the ability to detoxify EDCs could also justify differences in EDCs associations between men and women found in this study (1,14). Once in the human body, BPA, BPS, and 2-naphthol can be conjugated with glucuronic acid by the enzyme uridine 5'-diphosphate-glucuronosyltransferase (15,32). The conjugated form of these chemicals is less likely to cross the cell membrane and is quickly excreted through the urine (15,32). In excess, unconjugated BPA, BPS, and 2-naphthol become biologically active, impairing several intracellular pathways (31,33). Owing to their lipophilic properties, these chemicals accumulate in adipocytes and lipid-rich fluids such as breast milk (21). Several factors indicate that women are at more risk for relevant adverse EDCs consequences. On average, women have a higher ratio of adipocytes per body mass than men, which results in increased accumulation sites for EDCs (21,27) as shown in this study. In addition, women have lower expression of uridine 5'-diphosphate-glucuronosyltransferase and decreased glomerular filtration rate, and, therefore, they have less capacity to detoxify and eliminate these chemicals (1,15). These factors could strengthen the assumption that BPA and 2-naphthol have a higher impact on PAP in women.

Urine samples normalized by creatinine are widely used to estimate EDCs exposure (9,10,18,24,25). However, a few studies have suggested the potential of within-person EDCs variability (21,35). Leading world organizations have appointed diet as the primary driver of EDCs daily variation (26,36). Thus, to control this covariant, all samples were collected in the morning after overnight fasting. We acknowledge that even with this methodological approach, levels of EDCs have varied >100-fold between subjects. Still, EDCs values detected in this study have varied within the range reported by other authors in a population with a similar age group (18,24,25). Regular behaviors not measured in this study, such as high intake of processed or canned food containing high levels of EDCs, might be driving this variance.

Reactive oxidative species are generated during acute exercise; however, regular exercise increases antioxidant capability and decreases systemic levels of oxidative stress (2,7,8). Studies have suggested that oxidative stress mediates the detrimental effect of PAHs, BPA, and BPS on the human body (10,24). Moreover, our previous report demonstrates that levels of both BPA and BPS were associated with levels of 8-isoprostane (oxidative stress metabolite) in urine, which was, in part, consistent with this study (24). Bisphenol A was positively associated with 8-isoprostane when data were analyzed together (men and women), and BPA was positively associated in women. Nevertheless, to our surprise, the level of 8-isoprostane was positively associated with PAP when both groups (men and women) were analyzed together, which partially refutes our initial hypothesis. The reason for this divergence could be a matter for further studies. Therefore, our findings indicate that oxidative stress might not mediate EDCs effects on PAP.

This study is the first to suggest that BPA and 2-naphthol, EDCs widely present in the environment, could affect PAP and, therefore, athletic performance. However, the authors acknowledge that this study has a few limitations, including the sample size and a distinct number of subjects in each sex. In addition, the conclusions are based on association rather than cause and effect, like most of the studies were performed with the purpose of

investigating the impact of EDCs on human subjects. Nevertheless, the results of this study highlight the need for further inquiry into the impact of EDCs in the human performance field.

The results of this study provide evidence suggesting that 2 EDCs, BPA and 2-naphthol, could influence athletic performance by affecting PAP. These findings affect the athletic field, because interventional studies have shown the efficacy of strategies to minimize BPA and 2-naphthol levels in the human body, and such nonexercise interventions could provide synergistic effects to athletic performance when combined with athletic training (26,36). Moreover, the idea that women are more at risk for adverse effects of BPA and 2-naphthol opens the window for further investigations on EDCs being one of the several determinants of sex differences on athletic performance.

Practical Applications

This study serves as one of the first comprehensive analyses of the impact of EDCs exposure on anaerobic performance. Our results inform athletes and coaches that exposure to specific toxicants, such as BPA and 2-naphthol, could negatively affect PAP and further athletic performance, particularly in female athletes. Simple daily behavior changes, including prioritizing fresh over canned and packaged food, avoiding heating food in plastic containers, and minimizing airborne inhalation, can drastically decrease BPA and 2-naphthol levels in the human body. Therefore, athletes can follow the recommendations above to reduce their exposure to those harmful EDCs, particularly in training seasons that aim to achieve maximal athletic performance.

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References

1. Anderson GD. Gender differences in pharmacological response. *Int Rev Neurobiol* 83: 1–10, 2008.
2. Benite-Ribeiro SA, Lucas-Lima KL, Jones JN, Dos Santos JM. Transcription of mtDNA and dyslipidemia are ameliorated by aerobic exercise in type 2 diabetes. *Mol Biol Rep* 47: 7297–7303, 2020.
3. Caporossi L, Papaleo B. Exposure to bisphenol A and gender differences: From rodents to humans evidences and hypothesis about the health effects. *J Xenobiot* 5: 5264, 2015.
4. Coratella G, Longo S, Rampichini S, et al. Quadriceps and gastrocnemii anatomical cross-sectional area and vastus lateralis fascicle length predict peak-power and time-to-peak-power. *Res Q Exerc Sport* 91: 158–165, 2020.
5. Darbre PD. Overview of air pollution and endocrine disorders. *Int J Gen Med* 11: 191–207, 2018.
6. Dobbin N, Hunwicks R, Highton J, Twist C. Validity of a jump mat for assessing countermovement jump performance in elite rugby players. *Int J Sports Med* 38: 99–104, 2017.
7. Dos Santos JM, Tewari S, Mendes RH. The role of oxidative stress in the development of diabetes mellitus and its complications. *J Diabetes Res* 2019: 4189813, 2019.
8. Dos Santos JM, De Oliveira DS, Moreli ML, Benite-Ribeiro SA. The role of mitochondrial DNA damage at skeletal muscle oxidative stress on the development of type 2 diabetes. *Mol Cell Biochem* 449: 251–255, 2018.
9. Fan Y, Tao C, Li Z, et al. Association of endocrine-disrupting chemicals with all-cause and cause-specific mortality in the U.S.: A prospective cohort study. *Environ Sci Technol* 57: 2877–2886, 2023.

10. Gong J, Zhu T, Kipen H, et al. Urinary polycyclic aromatic hydrocarbon metabolites as biomarkers of exposure to traffic-emitted pollutants. *Environ Int* 85: 104–110, 2015.
11. Jeng HA, Pan CH, Chang-Chien GP, Diawara N, Peng CY, Wu MT. Repeated measurements for assessment of urinary 2-naphthol levels in individuals exposed to polycyclic aromatic hydrocarbons. *J Environ Sci Health A Tox Hazard Subst Environ Eng* 46: 865–873, 2011.
12. Jones J, Reneau P, Dos Santos JM. Metabolically healthy obese vs. Metabolic syndrome—The crosslink between nutritional exposure to bisphenols and physical exercise. *Med Hypotheses* 149: 110542, 2021.
13. La Merrill MA, Vandenberg LN, Smith MT, et al. Consensus on the key characteristics of endocrine-disrupting chemicals as a basis for hazard identification. *Nat Rev Endocrinol* 16: 45–57, 2020.
14. Lin TJ, Guo YL, Hsu JC, Wang IJ. 2-Naphthol levels and allergic disorders in children. *Int J Environ Res Public Health* 15: 1449, 2018.
15. Liu W, Kulkarni K, Hu M. Gender-dependent differences in uridine 5'-diphospho-glucuronosyltransferase have implications in metabolism and clearance of xenobiotics. *Expert Opin Drug Metab Toxicol* 9: 1555–1569, 2013.
16. Margolis LM, Pasiakos SM. Performance nutrition for cold-weather military operations. *Int J Circumpolar Health* 82: 2192392, 2023.
17. Martinez N, Campbell B, Franek M, Buchanan L, Colquhoun R. The effect of acute pre-workout supplementation on power and strength performance. *J Int Soc Sports Nutr* 16: 13–29, 2016.
18. Moura HSRP, Rocha PRS, Amato AA, Sodré FF. Quantification of bisphenol A in urine samples from children studying in public schools from the Brazilian capital. *Microchem J* 152: 104347, 2020.
19. Nuzzo JL. Narrative Review of sex differences in muscle strength, endurance, activation, size, fiber type, and strength training participation rates, preferences, motivations, injuries, and neuromuscular adaptations. *J Strength Cond Res* 37: 494–536, 2023.
20. Park WC, Kim M, Kim S, et al. Introduction of fall risk assessment (FRA) system and cross-sectional validation among community-dwelling older adults. *Ann Rehabil Med* 43: 87–95, 2019.
21. Porucznik CA, Cox KJ, Wilkins DG, et al. A preliminary study of bio-monitoring for bisphenol-A in human sweat. *J Anal Toxicol* 39: 562–566, 2015.
22. Pueo B, Lipinska P, Jiménez-Olmedo JM, Zmijewski P, Hopkins WG. Accuracy of jump-mat systems for measuring jump height. *Int J Sports Physiol Perform* 12: 959–963, 2017.
23. Richmond P, Zhu J, Kennedy T, et al. The effect of fat distribution and nutritional behavior on balance and equilibrium of acrobatics and tumbling athletes: Body composition and Performance. *Proc W Va Acad Sci* 95: 29–35, 2023.
24. Riffe JNJ, Wade M, Sine T, et al. The link of environmental estrogens exposure to oxidative stress and their association with insulin- and exercise-induced glucose uptake. *Obes Med* 42: 100503, 2023.
25. Rocha PRS, Moura HSRP, Silva NG, Neves FAR, Sodré FF, Amato AA. Exposure of elementary school-aged Brazilian children to bisphenol A: Association with demographic, social, and behavioral factors, and a worldwide comparison. *Sci Rep* 14: 24355, 2024.
26. Rudel RA, Gray JM, Engel CL, et al. Food packaging and bisphenol A and bis(2-ethylhexyl) phthalate exposure: Findings from a dietary intervention. *Environ Health Perspect* 119: 914–920, 2011.
27. Sandbakk Ø, Solli GS, Holmberg HC. Sex differences in world-record performance: The influence of sport discipline and competition duration. *Int J Sports Physiol Perform* 13: 2–8, 2018.
28. Sayers SP, Harackiewicz DV, Harman EA, Frykman PN, Rosenstein MT. Cross-validation of three jump power equations. *Med Sci Sports Exerc* 31: 572–577, 1999.
29. Shi Y, Lian Z, Hu S, Hu Y. Effects of multiple indoor environmental factors on anaerobic exercise performance. *J Therm Biol* 108: 103280–2022, 2022.
30. Sirirat R, Heskey C, Wilson C, et al. A comparison of body composition measurements between bioelectrical impedance analysis (InBody 570) and air displacement plethysmography (BOD POD). *Curr Dev Nutr* 4: nzaa063_087, 2020.
31. Sun L, Chen T, Wang Z, et al. The gender-specific adverse association of polycyclic aromatic hydrocarbons on skeletal muscle mass and strength in the general adults and the possible mechanisms in experimental rats. *Chemosphere* 287: 132066, 2022.
32. Vergara AG, Watson CJW, Chen G, Lazarus P. UDP-glycosyltransferase 3A metabolism of polycyclic aromatic hydrocarbons: Potential importance in aerodigestive tract tissues. *Drug Metab Dispos* 48: 160–168, 2020.
33. Wade M, Delawder V, Reneau P, Dos Santos JM. The effect of BPA exposure on insulin resistance and type 2 diabetes—the impact of muscle contraction. *Med Hypotheses* 140: 109675, 2020.
34. Wisniewski P, Romano RM, Kizys MM, et al. Adult exposure to bisphenol A (BPA) in Wistar rats reduces sperm quality with disruption of the hypothalamic-pituitary-testicular axis. *Toxicology* 329: 1–9, 2015.
35. Ye X, Wong LY, Bishop AM, Calafat AM. Variability of urinary concentrations of bisphenol A in spot samples, first morning voids, and 24-hour collections. *Environ Health Perspect* 119: 983–988, 2011.
36. Yilmaz B, Terekci H, Sandal S, Kelestimur F. Endocrine disrupting chemicals: Exposure, effects on human health, mechanism of action, models for testing and strategies for prevention. *Rev Endocr Metab Disord* 21: 127–147, 2020.
37. Ziegenfuss TN, Rogers M, Lowery L, et al. Effect of creatine loading on anaerobic performance and skeletal muscle volume in NCAA Division I athletes. *Nutrition* 18: 397–402, 2002.
38. Zou Z, Harris LK, Forbes K, Heazell AEP. Sex-specific effects of bisphenol A on the signaling pathway of ESRG in the human placenta. *Biol Reprod* 106: 1278–1291, 2022.