Urinary bisphenol-A output in plastic industry workers: A possible indicator of occupational exposure

Article *in* Tropical Journal of Medical Research · July 2014

DOI: 10.4103/1119-0388.140435

CITATIONS
6

READS
345

5 authors, including:

Maduka Ignatius Chukwudi
University of Nigeria-Teaching Hospital
58 PUBLICATIONS **700** CITATIONS
SEE PROFILE

Francis Ezeonu
Nnamdi Azikiwe University
46 PUBLICATIONS **459** CITATIONS
SEE PROFILE

Emeka Neboh
Enugu State University of Science and Technology (ESUT), Enugu State.
61 PUBLICATIONS **771** CITATIONS
SEE PROFILE

Elvis Shu
University of Nigeria
114 PUBLICATIONS **1,776** CITATIONS
SEE PROFILE

ORIGINAL ARTICLE

Urinary bisphenol-A output in plastic industry workers: A possible indicator of occupational exposure

Ignatius C. Maduka, Francis C. Ezeonu[1], Emeka E. Neboh[2], Elvis N. Shu[3], Ebele J. Ikekpeazu[4]

Department of Human Biochemistry, College of Medicine, Nnamdi Azikiwe University (NAU), Nnewi, [1]Department of Applied Biochemistry, Nnamdi Azikiwe University (NAU), Awka, Anambra State, [2]Department of Medical Biochemistry, College of Medicine, Enugu State, University of Science and Technology (ESUT), Enugu State, [3]Departments of Pharmacology and Therapeutics and [4]Medical Biochemistry, College of Medicine, University of Nigeria Enugu Campus, Enugu State, Nigeria

Abstract

Background: Bisphenol-A (BPA) is a known endocrine disruptor and a plasticizer, and occupational exposure has been said to be a potential source of endogenous BPA in humans. **Objective:** The study is aimed at evaluating residual urinary BPA of plastic industry workers in comparison with the controls, as an indicator of possible predisposition of these workers to xenoestrogens. **Materials and Methods:** BPA was evaluated in the urine of 108 (80 male and 28 female) workers of a plastic industry at Enugu, Nigeria, who were aged 21-40 years and who had not spent more than 10 years in the industry. The control group consisted of 88 (48 male and 40 female) age-matched and apparently healthy non-plastic industry workers. BPA was analyzed by enzyme-linked immunosorbent assay (ELISA) using Ecologenia® Supersensitive BPA ELISA kit. Data analysis was done by using GraphPad Prism computer software. **Results:** There was significant increase (P = 0.0002) in the mean urinary BPA output by the industry workers compared with the controls. Those who had spent ≥6 years in the industry showed a significant increase (P = 0.0001) in the BPA output compared to those who had spent <6 years. When the subjects were grouped according to gender, there were significant increases in urinary BPA in male subjects compared to the females (P = 0.0006 and 0.0487) both in control subjects and plastic industry workers, respectively. There was, however, no significant difference (P = 0.0825) when the subjects were grouped according to age. **Conclusions:** The study showed that urinary output is not dependent on age, but on sex and duration of exposure to the raw materials. Subjects working in plastic industries, especially males, were found to be more exposed to the endocrine disruptor and should be checked for bioaccumulation.

Keywords: Bisphenol-A, exposure, plastic industry workers, urine

Introduction

The possibility that some chemicals may disrupt the endocrine systems in humans and animals has received considerable attention in the scientific and public community.[1] Human and other animal bodies depend upon a complexly integrated and timed series of events, of which the delivery of hormones to various organs is vital.[2] When the delivery time and/or amount of a hormone is upset, the result can be devastating and permanent.[2] Because the disturbance of the endocrine system is a very sensitive topic, scientific findings and/or observations are often controversially discussed among scientists, environmentalist, and authorities.[1] In recent years, a growing body of scientific research indicates that substances in the environment may interfere with the normal function of the endocrine system of humans and wildlife.[3] These compounds may be man-made (known as xenoestrogens) and include industrial chemicals, crop protection chemicals, or they may be natural such as the phytoestrogens.[1]

Access this article online	
Quick Response Code:	Website: www.tjmrjournal.org
	DOI: 10.4103/1119-0388.140435

Address for correspondence: Mr. Emeka E. Neboh, Department of Medical Biochemistry, College of Medicine, Enugu State University of Science and Technology (ESUT), Enugu State, Nigeria.
E-mail: emmyneboh@yahoo.com

Bisphenol-A (BPA) belongs to the category of bis (hydroxyphenyl) methane, for which several endocrine effects have been reported.[1] BPA is one of the most important chemicals worldwide. In 2000, the United States Environmental Protection Agency (USEPA) reported that over 1 million pounds of BPA is released into the environment annually.[4] BPA can enter the environment either directly or through degradation of products such as ocean-borne plastic trash. Leaching of BPA from plastic and metal waste in landfills is one potential source of environmental contamination.[5] BPA serves as a stabilizer for plasticizers in polyvinyl chloride (PVC), a thermal stabilizer for PVC resins, and an antioxidant in rubber and plastics.[6] It is also used in the manufacture of hard, clear plastic known as polycarbonate, used in packaging many consumer products.[6] BPA is a well-known endocrine disruptor which can also lead to various health hazards.[7-10] It can easily be absorbed into the body by inhalation of its aerosols and by ingestion. Occupational exposure appears to be a potential source of endogenous BPA in the human system.[11] BPA is largely excreted unaltered in humans and in the form of glucuronides in the urine and feces.[12] Urinary BPA level, therefore, can be a good tool for assessment of BPA exposure,[13] hence the need for this study. The aim of the study is to estimate the urinary BPA output of plastic industry workers in comparison with the duration of exposure to this xenobiotic, as a possible indicator of occupational exposure.

Materials and Methods

Subjects

The subjects consisted of 108 volunteer workers (80 male and 28 female workers) at a plastic and chemicals industry at Emene, Enugu. They had spent 10 years or less in the industry, and were aged between 21 and 40 years. Also, 88 age-matched apparently healthy non-plastic and paint industry workers were used as control subjects.

The sampling technique used is the stratified random sampling technique such that the different strata (control and plastic industry workers, male and female subjects, etc.) are all represented.

Informed consent was obtained from each of the subjects and a well-explained questionnaire was distributed to each of them. Only those adults who returned their questionnaire were enlisted for the study. The questionnaire used was a structured type of questionnaire (fixed response format) containing information that actually helped in the stratification of the sample population. The questionnaire was also validated (standardized) through pilot testing using face validation by lecturers in the educational statistics department in the Enugu State University of Science and Technology.

The contents of the questionnaire included name, age, sex, period of time spent in the industry, department in the industry, marital status, duration of marriage, whether or not there was difficulty in conception, etc.

The study was performed in the period between March and June 2009 in Enugu Metropolis, south-east Nigeria. Approval was also given by the ethics committee of University of Nigeria Teaching Hospital (UNTH) Enugu.

Sample collection

The subjects were asked to collect about 10 ml spot urine samples directly into clean glass tubes. The urine samples were stored in a refrigerator and analyzed within 48 h.

Analytical techniques

Sample pre-treatment

The refrigerated samples were allowed to come to room temperature. All the urine samples were first diluted 1 in 20 with distilled water prior to analysis. Methanol was then added to the well-mixed and diluted urine samples to form a methanol concentration of 10% (v/v).

Sample analysis

BPA was analyzed by enzyme-linked immunosorbent assay (ELISA) method using Ecologenia® Supersensitive BPA ELISA kit Batch No. T2KSI of 2007 from Envirochemicals Ltd, Osaka, Japan. Samples were analyzed in duplicate and the average was used for the calculation.

Analysis of results

Results were expressed as mean ± standard deviation (SD). Significant differences between means were determined using Student's *t*-test for equality of means. Statistical analysis was done by the use of GraphPad Prism computer software program.

Results

Table 1 shows the mean ± SD of urinary BPA output (g/l) in plastic industry workers and their age-matched control subjects. The table shows a highly significant statistical increase ($P = 0.0002$) in the urine BPA level of the test subjects compared to the control subjects.

A comparison of duration of occupational exposure at the plastic industry shows that those who had worked for greater than 5 years showed a highly significant increase ($P < 0.0001$) in the mean urinary BPA output

when compared with those who had worked for only 0-5 years [Table 2].

Table 3 shows the effect of sex on urinary BPA level in both the control and plastic industry subjects. The table shows an increase in the urinary output of BPA by male subjects compared to their female counterparts in both the control and test subjects ($P = 0.0006$ and 0.0487, respectively).

However, a comparison of two different age groups of 21-30 and 31-40 years shows no statistical difference ($P = 0.0825$) in their mean urinary BPA level [Table 4].

Discussion

The result of this study clearly shows that there is an increase of BPA in the urine of occupationally exposed plastic industry workers. This suggests that plastic industry workers are at greater risk of the deleterious effect of exposure to BPA ($P = 0.0002$) compared to a non-exposed work population. This could be attributed to the high exposure to the compound which is highly used in the industry. The values recorded in our study for the plastic industry workers (43.88 ± 4.76 µg/l) and controls (25.10 ± 2.72 µg/l) appear to be high enough to cause endocrine-related effects, with higher values in the test subjects. There is a widespread variation in the reported values for endocrine-related effects, but many fall in the range of 1.9-4.2 ng/ml,[14] and the mean was reported to be 1.20 µg/l in a reference population.[11,15] The high mean BPA value obtained in the present study for both exposed and control subjects suggests great exposure of the subjects in this environment to BPA-containing substances (such as drinking water packaged in light polythene bags and stored under high tropical heat for long periods of time) which are freely consumed without any control measure.

Table 1: Urinary BPA output (g/l) in plastic industry workers

Variables	Control (*n*=88)	Plastic industry subjects (*n*=108)
Mean	25.10	43.88
Standard deviation	2.72	4.76
P value	*P*=0.0002	

BPA=Bisphenol-A

Table 2: Duration of occupational exposure and urine BPA level (g/l)

Variables	Duration of occupational exposure (years)	
	0-5 (*n*=60)	>5-10 (*n*=48)
Mean	32.91	61.29
Standard deviation	2.50	4.02
P value	*P*<0.0001	

BPA=Bisphenol-A

Table 3: The effect of gender on urinary BPA level (g/l)

Variables	Control subjects		Plastic industry subjects	
	Male (*n*=48)	Female (*n*=40)	Male (*n*=80)	Female (*n*=28)
Mean	32.25	17.53	46.18	31.14
Standard deviation	2.32	1.83	4.84	1.65
P value	*P*=0.0006		*P*=0.0487	

BPA=Bisphenol-A

Table 4: The effect of age on urinary BPA level (g/l)

Variables	Age group (years)	
	21-30 (*n*=64)	31-40 (*n*=44)
Mean	29.86	46.00
Standard deviation	4.54	
P value	*P*=0.0825	2.74

BPA=Bisphenol-A

This shows that although the controls were also exposed to BPA, the plastic industry workers were exposed to greater risk of the effects, having almost twice the concentration of the controls. Also, there was a significant increase ($P = 0.0001$) in the urinary output of BPA by the workers who had worked in the industry for more than 5 years, compared with those who had worked for 5 years or less. This indicates that length of continuous exposure to the chemical increases the risk to the subject. This reveals that occupational exposure among the plastic industry workers must have been responsible for this accumulation with increasing years of service in the industry. Although a report[12] indicates that the compound can be excreted unaltered in urine and feces, exposure-dependent bioaccumulation is still possible despite the increased excretion. This is dangerous as BPA is known to exhibit toxic effects at very high exposures.

Male subjects were found to excrete more BPA than their female counterparts in both the controls and plastic industry workers. This suggests that BPA might preferentially accumulate in males, where they affect their endocrine functions (anti-androgenic activity).[11] The reason for this sex-dependent bioaccumulation is not clear. However, the WHO International Program on Chemical Safety (IPCS) reported in 1986 that a higher metabolic rate and oxygen consumption leads to a greater intake of air per unit body weight and subsequent greater exposures to these chemicals.[16] The increased excretion of BPA by the males relative to the females in this study might be related to their higher metabolic rate. There was, however, no statistical variation in the level of urinary BPA

output as a result of the subjects' ages, when the different age ranges were compared.

Conclusion

The study showed that the endocrine disruptor (BPA) was significantly increased in the urine of plastic industry workers, mostly in males. This suggests that these individuals are predisposed to the compound via occupational exposure, since most workers have protective covering over their nostrils only. Subjects working in plastic and other related industries should be evaluated for BPA levels in both blood and urine regularly to check bioaccumulation and its various biochemical effects like hormone disruption, especially in males.

Also, more precautionary measures should be employed by such industries to reduce exposure of the workers through ingestion, inhalation, and absorption through the skin, in order to minimize the effect of this chemical on both the workers and the general population.

References

1. Beronius A, Rudén C, Håkansson H, Hanberg A. Risk to all or none? A comparative analysis of controversies in the health risk assessment of Bisphenol A. Reprod Toxicol 2010;29:132-46.
2. Erler C, Novak J. Bisphenol-A exposure: Human risk and health policy. J Pediatr Nurs 2010;25:400-7.
3. Newbold RR, Padilla-Banks E, Jefferson WN. Environmental estrogens and obesity. Mol Cell Endocrinol 2009;304:84-9.
4. United States Environmental Protection Agency (US. EPA). Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-P-Dioxin (TCDD) and related Compounds. National Academy Sciences (External Review Draft) US. Washington, DC: Environmental Protection Agency; 2004. p. 16-30. EPA /600 /P-00 /001 Cb.
5. Bromer JG, Zhou Y, Taylor MB, Doherty L, Taylor HS. Bisphenol-A exposure in utero leads to epigenetic alterations in the developmental programming of uterine estrogen response. FASEB J 2010;24:2273-80.
6. Vogel SA. The politics of plastics: The making and unmaking of bisphenol a 'safety'. Am J Public Health 2009;99 Suppl 3:S559-66.
7. Ryan KK, Haller AM, Sorrell JE, Woods SC, Jandacek RJ, Seeley RJ. Perinatal exposure to bisphenol-A and the development of metabolic syndrome in CD-1 mice. Endocrinology 2010;151:2603-12.
8. Benachour N, Aris A. Toxic effects of low doses of bisphenol-A on human placental cells. Toxicol Appl Pharmacol 2009;241:322-8.
9. vom Saal FS, Myers JP. Bisphenol A and risk of metabolic disorders. JAMA 2008;300:1353-5.
10. Chou K, Wright RO. Phthalates in food and medical devices. J Med Toxicol 2006;2:126-35.
11. Li D, Zhou Z, Qing D, He Y, Wu T, Miao M, *et al*. Occupational exposure to bisphenol-A (BPA) and the risk of self-reported male sexual dysfunction. Hum Reprod 2010;25:519-27.
12. Lakind JS, Naiman DQ. Daily intake of bisphenol-A and potential sources of exposure: 2005-2006 National Health and Nutrition Examination Survey. J Expo Sci Environ Epidemiol 2011;21:272-9.
13. Teeguarden JG, Calafat AM, Ye X, Doerge DR, Churchwell MI, Gunawan R, *et al*. Twenty-four hour human urine and serum profiles of bisphenol A during high-dietary exposure. Toxicol Sci 2011;123:48-57.
14. Center for Disease Control (CDC). New Data from CDC Confirms Human Exposure to Bisphenol A in the United States is Far Below Safe Limits. November 1, 2007.
15. Calafat AM, Kuklenyik Z, Reidy JA, Caudill SP, Ekong J, Needham LL. Urinary concentrations of Bisphenol-A and 4-nonylphenol in a human reference population. Environ Health Perspect 2005;113:391-5.
16. WHO International Programme on Chemical Safety (IPCS). Principles for evaluating health risks from chemicals during infancy and early childhood: The need for a special approach. Environ Health Criteria 1986;59:24-38.

How to cite this article: Maduka IC, Ezeonu FC, Neboh EE, Shu EN, Ikekpeazu EJ. Urinary bisphenol-A output in plastic industry workers: A possible indicator of occupational exposure. Trop J Med Res 2014;17:117-20.

Source of Support: Nil, **Conflict of Interest:** None declared.