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ARTICLE



Human health risk assessment of bisphenol A released from polycarbonate drinking water bottles and carbonated drinks exposed to sunlight in Nigeria

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ABSTRACT

This study investigated the release of a known endocrine disruptor—bisphenol A (BPA), in drinking water and carbonated drinks stored in polycarbonate bottles and exposed to sunlight with a view of assessing the health risk involved in their consumption. BPA in the samples was extracted using solid-phase extraction method and determined with high performance liquid chromatography equipped with ultra-violet detector. Results showed that there were significant increase ($p < 0.05$) in BPA concentration of bottled water and carbonated drink exposed at 1, 3, 7 and 21 days. Also, there were enrichment of BPA levels ranging 31–868% in the bottled water and 17–920% carbonated drinks from 1 to 60 days when compared with the control. However, BPA migrating rate decreases with increase in exposure duration and later remains fairly constant as exposure duration progresses. The chronic daily intake of BPA in carbonated drinks and bottled water were low. The health risk index of BPA in polycarbonate bottled carbonated drinks and water was less than one in both adult and child and thus, there may be no probable adverse health effect on consumers of such products. However, caution should be taken as BPA is also released from other food packaging materials.

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Bisphenol A; polycarbonate; carbonated drinks; drinking water; sunlight; health risk

1. Introduction

Bisphenol A (BPA) is known to be one of the highest man-made chemicals produced. It is a monomer and plasticiser used in the manufacture of certain plastics especially food contact materials, such as polycarbonate (PC) plastic and epoxy resins. This is because it is cheap and effective and could produce strong plastics that wouldn't break or shatter when heated or cooled [1]. BPA has been discovered to be an endocrine disruptor that mimics the body's hormone and disrupts normal hormone levels, thus causing a significant impact on human health [2]. Research has shown that BPA has been detected in human serum, urine, breast milk, amniotic fluid, follicular fluid, placental tissue and umbilical cord blood [3]. At low doses, BPA affects development and reproduction in animals [4]. It does not occur naturally in the environment, it is released during the

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synthesis and manufacturing, processing and transportation [3]. Humans are exposed to BPA through different routes, such as: direct contact from food packaged with plastics containing BPA [5], and through aquatic environment contaminated with wastewater containing BPA [6].

PC bottled water and carbonated drinks in Nigeria have become an important commodity believed by consumers to be safe, pure, healthy and portable. There is great concern about the possible migration of chemicals from PC bottles into water or carbonated drink. These migrations can be as a result of exposure to higher temperature [7]. Mercea et al. [8] and Cao et al. [9] have reported that PC in contact with hot water, higher temperatures and alkaline conditions increases the hydrolytic depolymerisation resulting in formation of monomeric BPA with increased BPA migration.

In Nigeria, there are no regulations guiding exposure of plastics bottled water or carbonated drinks to high temperature. Most retail shops and supermarkets display their goods outside in order to advertise them thereby exposing these products to high temperature especially in dry season. This season (November–March) is usually characterised with harmattan, dust and temperatures as high as 40°C. Continuous exposure of these products to sunlight could lead to potential health effects to consumers over time due to bio-accumulation [10]. This necessitates the design of this study which aimed at determining the release of BPA in drinking water and carbonated drinks stored in PC bottles after exposure to sunlight and its associated health risks to consumers. This study is the first of its kind in Nigeria since no available literature from Nigeria has reported it. Information from this study can serve as a baseline for Government and policymakers in providing BPA regulation in Nigeria.

2. Experimental

2.1. Reagents and chemicals

BPA was purchased from Sigma-Aldrich (St. Louis, USA). High performance liquid chromatography (HPLC) grade water and methanol were purchased from J. T. Baker (Phillipsburg, NJ). Multi-walled carbon nanotubes (MWCNTs) with average external diameter of 30–60 nm was purchased from Chengdu Organic Chemicals Co. Ltd. (Chengdu, China). Sodium hydroxide and hydrochloric acid were purchased from Beijing Chemicals Corporation (Beijing, China).

The BPA standard stock solution of 1.0 µg/L was prepared by dissolving an appropriate amount of BPA in methanol. The standard stock solution was appropriately diluted in water to get the working standard solutions 0.1, 1, 2, 4 and 5 ng/L. Calibration curve was prepared by injecting standard solutions into HPLC and then plotting the resulting peak height values against the corresponding concentration for BPA (Supplementary 1) while Supplementary 2 presents the BPA standard chromatogram.

2.2. Sample collection and preparation

Eighteen samples each of plastic carbonated drink and water were randomly purchased from retail shops and supermarkets in Nsukka, Enugu and Lagos. In purchasing these samples, care was taken to obtain the bottled drinks and water that has not been exposed

to sunlight. A total of 36 samples were collected within December 2017–February 2018. Meanwhile, six samples were obtained at the point of production for both PC bottled carbonated drink and water to serve as control. The pH of the control samples was taken and the values ranged 2.82–3.24 for PC carbonated drinks and 6.8–7.2 for bottled water. The sampled bottled carbonated drinks and water were left to stand outside under the sun for 1, 3, 7, 21, 30 and 60 days, respectively at an average of 8 h/day at a temperature of $35 \pm 1^\circ\text{C}$.

2.3. Extraction and analysis

The novel carbonaceous material MWCNTs packed column, was washed with 5 mL of methanol and activated with 5 mL of water. About 25 mL of the sample was passed through the column at a flow rate of 2 mL/min. After the sample solution had passed through the column, it was washed with 5 mL of 10% methanol to remove the matrix materials from the column. The BPA retained on the column was eluted with 2.5 mL of methanol and 20 μL of methanol elute was injected into the HPLC system for determination [11].

2.4. HPLC working conditions

HPLC, Agilent 1200 series was attached to Ultraviolet Detector Agilent 1260 series. The wavelength selected for the analysis was 230 nm and Perkin Elmer C8 column was used with a column temperature of 40°C . Dimension of 5 μm , 4.6×150 mm was used with Hamilton Microlitre Syringe. Injection volume of 20 μL was used at a flow rate of 1.00 mL/min. Acetonitrile/de-ionised water was the mobile phase used at a ratio of (60/40).

2.5. Quality control

A clean laboratory environment was ensured during the preparation of solutions and analysis. The glassware was washed thoroughly with distilled water and detergent and dried in an oven. The reagents and solvents used for the analysis were of analytical grade and of high purity. Blank and recovery tests were done to check the performance of the procedures and instruments. The recovery was conducted by standard addition method. About 100 mL of the sample was spiked with 1.0 $\mu\text{g/L}$ of the BPA stock solution. After the spiking, the extraction procedure was followed as described in Section 2.3. The percent recovery was calculated with the equation below

$$\% \text{Recovery} = \frac{F - I}{A} \times 100 \quad (1)$$

Where F = conc. of spiked

I = conc. of unspiked

A = spiking conc. (i.e. BPA conc. added). The limit of detection (LOD) and limit of quantification (LOQ) were determined by the signal-to-noise ratio method. The peak-to-peak noise for the analyte retention time was measured, and subsequently, the concentration of the analyte that yielded a signal equal to the noise-to-signal ratio was estimated.

Table 1. Recovery analysis of BPA concentration in polycarbonate bottled water and carbonated drink.

	Samples	Carbonated drink ($n = 3$)	Bottled water ($n = 3$)
Spiked samples ($\mu\text{g/L}$)	60 days	9.27390	9.51021
	60 days	9.82183	8.94037
	60 days	10.24727	9.47525
Un-spiked samples ($\mu\text{g/L}$)	60 days	8.33149	8.47401
	60 days	8.87948	8.00425
	60 days	9.31035	8.54862
Recovery ($\mu\text{g/L}$)	60 days	0.94241	0.93620
	60 days	0.94235	0.93612
	60 days	0.93422	0.92663
Mean % recovery		93.96 ± 0.0047	93.29 ± 0.0055
LOD ($\mu\text{g/L}$)		0.0042	0.0042
LOQ ($\mu\text{g/L}$)		0.0068	0.0068

A signal-to-noise ratio of three was used to estimate LOD and signal-to-noise ratio of 10 was used to estimate LOQ (Table 1).

2.6. Data analysis

Descriptive statistics was used to present the data obtained as mean and standard deviation. One way analysis of variance (ANOVA) was used to compare the BPA concentration in carbonated drinks and bottled water in plastic containers. Tukey's test was used to determine significance for mean duration effect on BPA concentration. All statistical analysis was performed with SPSS version 20 for Windows.

2.7. Percent enrichment and migration rate

Percent enrichment of BPA in the drinking water and carbonated drinks compared with the control samples were determined with Equation 2 while migration rate of BPA ($\mu\text{g/L/d}$) from the PC bottles into the drinking water and carbonated drinks was determined with the Equation 3

$$\% \text{Enrichment} = \frac{C_t - C_o}{C_o} \times 100 \quad (2)$$

$$\text{Migration rate} = \frac{C_t - C_o}{D} \quad (3)$$

Where C_t is BPA concentrations at various exposure durations, C_o is BPA concentration in control sample and D is exposure duration.

2.8. Health risk assessment

Health risk index (HRI) was calculated to determine possible chronic non-carcinogenic health effects among consumers due to consumption of water and carbonated drinks from PC bottles. HRI is defined as ratio of chronic chemical daily intake to the acceptable daily intake (ADI). HRI greater than one is regarded as a probable potential risk.

$$\text{HRI} = \frac{\text{CDI}}{\text{ADI}} \quad (4)$$

$$\text{CDI} = \frac{C \times \text{IR}}{\text{BW}} \quad (5)$$

Where HRI is health risk index, CDI = chronic daily intake ($\mu\text{g}/\text{kg}$ body weight (BW)/day), ADI = acceptable daily intake ($4 \mu\text{g}/\text{kg}$ BW/day), C = BPA concentrations in samples ($\mu\text{g}/\text{L}$), IR = ingestion rate of water/carbonated drink (L/day). The IR for water used was 2.4 L/day for adult [12], 1 L/day for children [13] while IR for carbonated drinks was 1.479 L/day for adults [14] while for children 0.426 L/day was used as IR [15]. BW = body weight (60 kg for adult, 16.7 kg for children) [16].

3. Results and discussions

3.1. BPA levels in PC bottled water and carbonated drinks

Table 1 presents the results of the recovery analysis. The percentage recovery of BPA in carbonated drink and bottle water was $93.83\% \pm 0.41\%$ and $93.14\% \pm 0.48\%$, respectively. LOD and LOQ are 0.0042 and 0.00068 $\mu\text{g}/\text{L}$, respectively. The mean concentrations of BPA in drinking water and carbonated drinks stored in PC bottles are presented in Table 2, while Supplementary 3 presents the chromatogram of the samples. The BPA concentrations ($\mu\text{g}/\text{L}$) in PC bottled water ranges from 0.308 for 1-day exposure to 8.343 for 60-day exposure while that in carbonated drinks ranges from 0.176 for 1-day exposure to 8.841 for 60-day exposure. The study showed that BPA was present in all of the carbonated drinks and bottled water exposed to sunlight and the BPA concentrations increased with increase in exposure time compared to the control. This result is in tandem with the reports of Tokuanaga et al. [17] who also identified BPA in drinking water bottles. Amiridou and Voutsas [18] also detected plastic residues in water stored in plastic bottles. Cao et al. [9] detected BPA in the soft drinks analysed with about 75% and 85% of the products having BPA concentrations of 50 and 1000 ng/L, respectively with mean BPA concentration of 570 ng/L. All the BPA concentrations measured in PC bottled water and carbonated drinks in this study were below the maximum concentration level (MCL) of BPA (10 $\mu\text{g}/\text{L}$) as stipulated by China drinking water regulation [19]. The BPA levels in PC bottled water obtained in this study were higher than values ranging from 0.0136 $\mu\text{g}/\text{L}$ in 1 week to 0.168 $\mu\text{g}/\text{L}$ in 8 weeks reported on PC bottled water exposed to 50°C by Wen et al. [20]. Also, Fan et al. [21] reported lower BPA concentrations ($\mu\text{g}/\text{L}$) in ranges of

Table 2. Mean concentration of BPA in carbonated drink and bottled water ($\mu\text{g}/\text{L}$).

Days	Carbonated drink	Bottle water
Control (at production)	0.0096 ± 0.00004	0.0096 ± 0.0003
1	0.1757 ± 0.0100	0.3082 ± 0.0100
3	0.5683 ± 0.0700	0.8174 ± 0.0200
7	1.1784 ± 0.0700	1.5161 ± 0.0500
21	1.8891 ± 0.0500	2.0991 ± 0.0500
30	3.5841 ± 0.2300	3.5071 ± 0.0500
60	8.8413 ± 0.2800	8.3425 ± 0.1700

0.00031–0.0389 (1 week), 0.00723–0.078 (2 weeks) and 0.0144–0.119 (4 weeks) in bottled water at 70°C compared to those reported in this study.

The lowest BPA concentration in this study was found in the control sample which were samples collected at the point of production. The low values show that leaching of BPA from the bottle into the samples is very slow and insignificant under room temperature. On the other hand, the samples exposed to direct sunlight increased with increase to exposure time. As suggested by Omer et al. [22], this might be as an outcome of the fast migration of BPA from the bottle into water during the exposure to higher temperature. Amiridou and Voutsas [18] has attributed this leaching to photolytic formation/degradation of organic compounds in the PC bottles when exposed to high temperature. Also, Vom-Saal and Myers [23] have reported that high temperature, heat and repeated washing of PC products accelerate the migration of BPA. The differences in BPA content across the different brand can also be ascribed to the quality of the bottle used in packaging [18].

BPA is listed as a possible emerging priority substance subject to review for identification in the field of water policy. The ester bonds in BPA-based polymers are subject to hydrolysis and, therefore, BPA leaches into food and drinks from their storage containers.

3.2. Effect of exposure duration on BPA release from polycarbonate bottles

To evaluate the effect of exposure duration on BPA released, the concentration of BPA released from PC bottles after 1, 3, 7, 21, 30 and 60 days of exposure to sunlight at temperature of $35 \pm 1^\circ\text{C}$ for water and carbonated drinks were used to calculate percent enrichment and migration rate. Enrichment of BPA in the PC bottles for water and carbonated drinks exposed to sunlight was calculated with Equation 2 and the percent enrichment was presented in Figure 1. It was observed that BPA increased significantly ($p < 0.05$) with storage duration in both drinking water and carbonated drink PC bottles. It was also observed that exposure at 60 days has the highest percent enrichment (920%) in carbonated drink and 868% in bottled water with respect to the control sample. Exposures at 1 and 3 days gave percent enrichment below 100% in both water and

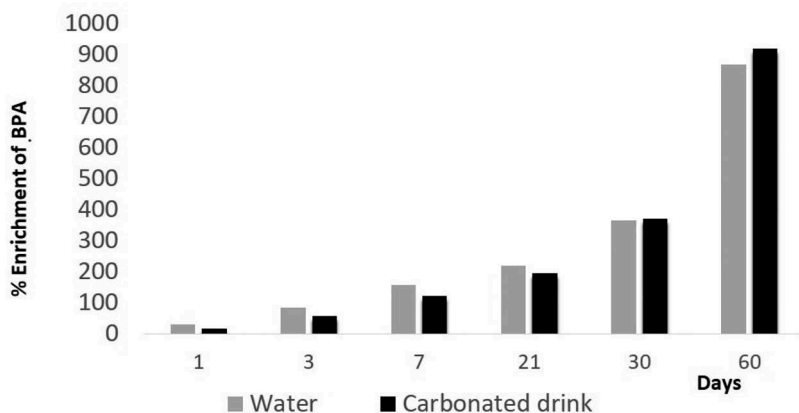


Figure 1. Percent enrichment of BPA in drinking water and carbonated drinks stored in polycarbonate bottles.

carbonated drinks. There was significant increase in BPA concentration of bottled water than carbonated drink at 1, 3, 7 and 21 days of exposure but at 30- and 60-day exposure, the BPA concentration in carbonated drink was higher than that of bottled water. BPA migration rates into the water and carbonated drinks exposed to sun were calculated (Equation 3). The migration rates decreased with exposure duration up to 21 days and remain fairly constant (Figure 2). This suggests that BPA levels may become stable over long exposure duration. Munguia-López et al. [24] have reported that BPA levels in food increases in the first 40 days. Similar decrease in migration rate as storage and exposure duration increases has been reported by some authors. Fan et al. [21] reported BPA migrating rate ($\mu\text{g}/\text{week}$) ranging 0.00378–0.0334 in 1 week, 0.00368–0.0294 in 2 weeks and 0.00174–0.0234 in 4 weeks in polyethylene terephthalate (PET) bottled water exposed at 70°C. Also, Wen et al. [20] reported BPA migrating rate ($\mu\text{g}/\text{week}$) of 0.02554 in 1 week, 0.02398 in 2 weeks, 0.02252 in 4 weeks and 0.02115 in 8 weeks in PC bottled water exposed to 50°C.

BPA can migrate from PC to liquids either by diffusion of the residual BPA present in PC after the manufacture or hydrolysis of polymers [25]. Le [26] has reported PC exposed to boiling water (100°C), to have BPA migration rate up to 55-fold higher than those exposed at 20°C. The greater migration has been reported to be as a result of increase in the permeability of the wall of containers that leads water to adhere to these walls [7].

3.3. Health risk assessment

Non-carcinogenic health risks due to BPA exposure from PC bottled water and carbonated drinks exposed to sunlight at different duration were calculated using HRI. The CDI was compared to the ADI of BPA at 4 $\mu\text{g}/\text{kg}$ BW/day. The CDIs and corresponding HRI associated with BPA in carbonated drink and bottled water for both adults and children across the different exposure duration are presented in Table 3. The CDI of BPA in carbonated drinks range from 0.0002 to 0.2179 $\mu\text{g}/\text{kg}$ BW/day in adults and from 0.0002

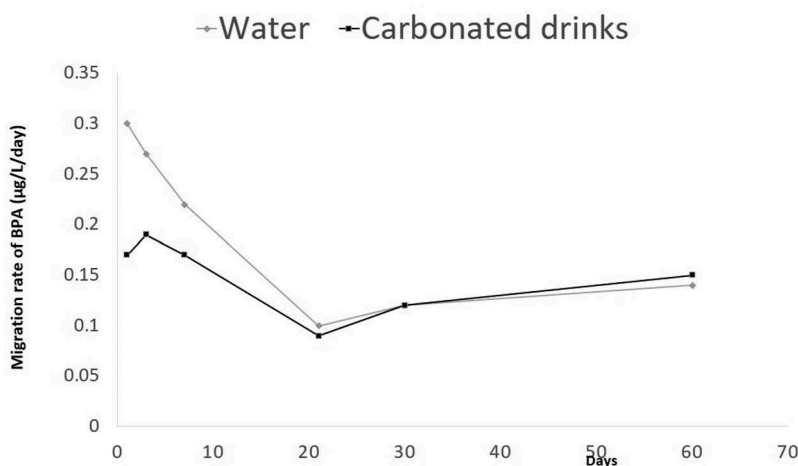


Figure 2. Migration rate of BPA into drinking water and carbonated drinks stored in polycarbonate bottles.



Table 3. Health risk assessment of BPA concentration in carbonated drink and bottled water.

Duration (days)	ADI ($\mu\text{g}/\text{kg BW}/\text{day}$)	CDI ($\mu\text{g}/\text{kg BW}/\text{day}$)						HRI						
		Carbonated drink			Bottle water			Carbonated drink			Bottle water			
		Adult	Children	Adult	Children	Adult	Children	HR	Children	HR	Adult	Children	HR	Children
At production (control)	4	0.0002	0.0002	0.0004	0.0006	0.0000	0.0000	N	0.0000	N	0.0000	N	0.0001	N
1	4	0.0043	0.0045	0.0123	0.0185	0.0011	0.0011	N	0.0011	N	0.0031	0.0011	0.0046	N
3	4	0.0140	0.0145	0.0327	0.0490	0.0035	0.0036	N	0.0035	N	0.0082	0.0036	0.0122	N
7	4	0.0291	0.0301	0.0606	0.0908	0.0073	0.0075	N	0.0073	N	0.0152	0.0075	0.0227	N
21	4	0.0466	0.0482	0.0840	0.1257	0.0116	0.0120	N	0.0116	N	0.0210	0.0120	0.0314	N
30	4	0.0883	0.0914	0.1403	0.2100	0.0221	0.0229	N	0.0221	N	0.0351	0.0229	0.0525	N
60	4	0.2179	0.2255	0.3337	0.4996	0.0545	0.0564	N	0.0545	N	0.0834	0.0564	0.1249	N

to 0.2255 $\mu\text{g}/\text{kg BW}/\text{day}$ in children. The CDI of BPA in bottled water range from 0.0004 to 0.3337 $\mu\text{g}/\text{kg BW}/\text{day}$ in adults and from 0.0006 to 0.4996 $\mu\text{g}/\text{kg BW}/\text{day}$ in children. The HRI of BPA in PC bottled water and carbonated drink exposed to sunlight in all the exposure durations was less than one. This implies that even though the PC bottled water and carbonated drinks assessed in this study contained BPA, the levels were still low to pose any risk to the consumers of such as at the time of the study. This, however, does not exclude the possible bio-accumulation and possible health risk of the consumption of water from the same source as the storage/exposure period increase. Also, BPA has been reported to be released from other material used for food or drinks storage like PET [20] and epoxy resins [27]. More so, Cao et al. [28] have reported high BPA levels in canned food. Humans are exposed to BPA through different routes, mostly by consuming food and drink that are stored in materials produced with BPA. Many water-packaging companies in Nigeria use PC plastic bottles for packaging of water [22]. The CDI and HRI obtained in this study were compared with those reported by other authors. The values in our study were higher than CDI (1.08–6.80 $\text{ng}/\text{kg}/\text{d}$ in adult and 3.79–31.3 $\text{ng}/\text{kg}/\text{d}$ in child) reported by Fan et al. [21] (2014) on PET bottled water exposed at 70°C. Also, lower CDI ranging 5×10^{-7} – 6.2×10^{-6} $\text{mg}/\text{kg}/\text{d}$ and HRI ranging 9.99×10^{-6} – 1.24×10^{-4} has been reported on PC bottled water exposed at 50°C by Wen et al. [20].

BPA is an endocrine disruptor, which can mimic the body's hormones. After entering the human body, BPA can disrupt normal cell function by acting as an oestrogen agonist [29], as well as an androgen antagonist [30], which may affect health. It has been suspected that BPA may affect human development throughout the foetal period [31], and may be carcinogenic, potentially leading to the precursors of breast cancer [32]. Due to its oestrogenic activity, it has been shown to reduce sperm count and sperm activity, be toxic to liver and may be even linked to obesity by affecting fat-cell activity [32]. In addition, exposure to BPA has been associated with chronic disease conditions in humans such as cardiovascular disease, diabetes, and is a serum marker of liver disease [33]. Bottled carbonated drinks and water are found to be sources for human exposure to BPA. However, the levels of BPA detected in PC bottled carbonated drinks and water were within low health risk range but prolonged storage and exposure to sunlight may cause increase level.

4. Conclusion

This study determined the effect of exposure duration on the concentrations of BPA in PC bottled water and carbonated drinks exposed to sunlight and also assessed its associated health risk. BPA concentration of carbonated drinks and bottled water in PC containers was found to increase with exposure time. As exposure time increases, the water and carbonated drinks were highly enriched with BPA which were leached from the PC bottles. However, it was observed that BPA migrating rate decreases with increase in exposure duration and later remains fairly constant at higher exposure time. The HRI showed that consumption of BPA from PC bottled water and carbonated drinks at the rate of ingestion reported in this study will probably have no adverse health effect on the consumers. However, as far as humans consume carbonated drinks and bottled water stored in plastic containers, and food packaged with cans, BPA exposure as a human health treat will continue to grow.

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Disclosure statement

The authors declare no conflict of interest.

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References

- [1] *Chemical Products Synopsis: Bisphenol A* (Mannsville Chemical Products Corp, Mannsville, 2008).
- [2] D. Berryman, F. Houde, C. DeBlois and M. O'Shea, *Chemosphere* **56**, 247 (2004). doi:10.1016/j.chemosphere.2004.02.030.
- [3] L. Vandenberg, R. Hauser, M. Marcus, N. Olea and W. Welshons, *Reprod. Toxicol.* **24**, 139 (2007). doi:10.1016/j.reprotox.2007.07.010.
- [4] W. Dekant and W. Völkel, *Toxicol. Appl. Pharmacol.* **228**, 114 (2008). doi:10.1016/j.taap.2007.12.008.
- [5] G. Lyons, WWF European Toxics Programme Report (WWF-UK, Godalming, Surrey) <http://www.wwfuk.org/filelibrary/pdf/bpa.pdf> (2000).
- [6] J. Kang, F. Kondo and Y. Katayama, *Toxicol* **226**, 79 (2006). doi:10.1016/j.tox.2006.06.009.
- [7] C. Brede, P. Fjeldal, I. Skjevraak and H. Herikstad, *Food Addit. Contam.* **20**, 684 (2003). doi:10.1080/0265203031000119061.
- [8] P. Mercea, *J. Appl. Polym. Sci.* **112**, 579 (2009). doi:10.1002/app.29421.
- [9] X.L. Cao, G. Dufresne, S. Belisle, G. Clement, M. Falicki, F. Beraldin and A. Rulibikiye, *J. Agric. Food Chem.* **56**, 7919 (2008). doi:10.1021/jf8008712.
- [10] C. La-or, S. Kriangsukand and O. Boonsong, *Expo. Health* **9**, 243 (2017). doi:10.1007/s12403-016-0235-5.
- [11] L. Pei, Z. Ehong, D. Qiongand and D.J. Dan, *Anal. Atomic Spectrochim. Acta B* **63**, 714 (2008).
- [12] United State Environmental Protection Agency, Human Health Ambient Water Quality Criteria, (2015). (Accessed Mar. 15, 2018) <https://www.epa.gov/sites/production/files/2015-10/documents/human-health-2015-update-factsheet.pdf>
- [13] United State Environmental Protection Agency, Drinking Water Standards and Health Advisories. EPA 822-S-12-001. (2012).
- [14] S.O. Awoyemi, J.A. Adekunle and C.I. Jolayemi, *J. Econ. Sust. Dev.* **5**, 16 (2014).
- [15] K. Hafekost, F. Mitrou, D. Lawrence and S.R. Zubrick, *BMC Pub. Health* **11**, 950 (2011). doi:10.1186/1471-2458-11-950.
- [16] A.O. Oyeyiola, O.T. Fatunsin, L.M. Akanbi, D.E. Fadahunsi and M.O. Moshood, *J. Health Pollut.* **15**, 63 (2017). doi:10.5696/2156-9614-7.15.63.
- [17] O.K. Tokuanaga, X. Liu, A. TakayMastushima and Y. Shimohigashi, *Environ. Health Perspect.* **116**, 32 (2008). doi:10.1289/ehp.10587.
- [18] D. Amiridou and D. Voutsas, *J. Haz. Mat.* **185**, 281 (2011). doi:10.1016/j.jhazmat.2010.09.031.
- [19] Ministry of health, PRC. Standard for Drinking Water Quality in China. GB 5749-2006. (2006).
- [20] M.Y. Wen, B.H. Yu, S.S.T. Eugenie and V. How, *Malaysian J. Med. Health Sci.* **14**, 18 (2018).
- [21] -Y.-Y. Fan, J.-L. Zheng, J.-H. Ren, J. Luo, X.Y. Cui and L.Q. Ma, *Environ. Pollut.* **192**, 113 (2014). doi:10.1016/j.envpol.2014.05.012.
- [22] L. Omer, H. Ahmed and A. Elbashir, *J. Environ. Chem. Ecotoxicol.* **8**, 51 (2016). doi:10.5897/JECE2015.0363.

- [23] F.S. Vom-Saal and J.P. Myers, *JAMA* **300**, 1353 (2008). doi:[10.1001/jama.300.11.1353](https://doi.org/10.1001/jama.300.11.1353).
- [24] E.M. Munguia-Lopez, E. Peralta, A. Gonzalez-Leon, C. Vargas-Requena and H. Soto-Valdez, *J. Agric. Food Chem.* **50**, 7299 (2002). doi:[10.1021/jf0257502](https://doi.org/10.1021/jf0257502).
- [25] K. Aschberger, P. Castello, E. Hoekstra, S. Karakitsios, S. Munn, S. Pakalin and D. Sarigiannis, *Bisphenol A and Baby Bottles: Challenges and Perspectives* (Publications Office of the European Union, Luxembourg, 2010), p. 5.
- [26] L. Le Corre, P. Besnard and M.C. Chagnon, *Crit. Rev. Food Sci. Nutr.* **55**, 769 (2015). doi:[10.1080/10408398.2012.678421](https://doi.org/10.1080/10408398.2012.678421).
- [27] A. Goodson, H. Robin, W. Summerfield and I. Cooper, *Food Addit. Contam.* **21**, 1015 (2004). doi:[10.1080/02652030400011387](https://doi.org/10.1080/02652030400011387).
- [28] X.L. Cao, C. Perez-Locas, G. Dufresne, G. Clement, S. Popovic, F. Beraldin and M. Feeley, *Food Addit. Contam.* **28**, 791 (2011). doi:[10.1080/19440049.2010.513015](https://doi.org/10.1080/19440049.2010.513015).
- [29] A.L. Wozniak, N.N. Bulayeva and C.S. Watson, *Environ. Health Perspect.* **113**, 431 (2005). doi:[10.1289/ehp.7505](https://doi.org/10.1289/ehp.7505).
- [30] H.J. Lee, S. Chattopadhyay, E.Y. Gong, R.S. Ahn and K. Lee, *Toxicol. Sci.* **75**, 40 (2003). doi:[10.1093/toxsci/kfg150](https://doi.org/10.1093/toxsci/kfg150).
- [31] B.S. Rubin and A.M. Soto, *Mol. Cell Endocrinol.* **304**, 55 (2009). doi:[10.1016/j.mce.2009.02.023](https://doi.org/10.1016/j.mce.2009.02.023).
- [32] European Food Safety Authority (EFSA), *Efsa J.* **428**, 1 (2006).
- [33] I.A. Lang, T.S. Galloway, A. Scarlett, W.E. Henley, M. Depledge and R.B. Wallace, *JAMA* **300**, 1303 (2008). doi:[10.1001/jama.300.11.1303](https://doi.org/10.1001/jama.300.11.1303).