REVIEW ARTICLE

J Korean Dent Sci. 2017;10(1):1-9
https://doi.org/10.5856/JKDS.2017.10.1.1
ISSN 2005-4742

Importance of Oral Environment for Environmental Hormones Interaction with Human Body for Future Research

Seong-Won Lee[1], Jae-Yoon Jeon[1,2], Byung-Cheol Oh[2], Jong-Won Choi[1], Seong-Su Ro[1], Chang-Joo Park[1,3], Kyung-Gyun Hwang[1,2,3]

[1]Department of Oral and Maxillofacial Surgery, Hanyang University Hospital,
[2]Department of Translational Medicine, Graduate School of Biomedical Science and Engineering, Hanyang University,
[3]Department of Dentistry/Oral and Maxillofacial Surgery, College of Medicine, Hanyang University, Seoul, Korea

There is increasing evidence that the environmental hormones may adversely affect the human body. The human reproductive system misrecognizes some of these endocrine disruptors with consequences to reproductive cell differentiation. Therefore, studies on the safety of these substances have been widely carried out to develop the science to create effective legislation to limit or prevent their use or require the development of inert, alternative substances. A few studies have reported that the oral cavity is the pathway for absorption of these substances released from plastic products or environmental hormone substances. This review suggests that the oral environment is vulnerable to exposure to environmental hormones and introduces supporting literature.

Key Words: Endocrine disruptors; Environmental hormones; Oral cavity

What Are Environmental Hormones?

Many plastic products made from natural or synthetic macromolecular substances are used in everyday life. Some of the synthetic macromolecular substances have structural characteristics similar to human endocrine hormones, and as such, manifest abnormalities to the body's systems. These substances are not easily metabolized and often accumulate in the body unlike human endocrine hormones, hence their name "endocrine disruptors." The 2002 International Programme on Chemical Safety (IPCS) of World Health Organization (WHO) categorized these substances as 'endocrine disruptors' and 'potential endocrine disruptors' and defined them as follows. A foreign

Corresponding Author: **Kyung-Gyun Hwang**
Department of Dentistry/Oral and Maxillofacial Surgery, College of Medicine, Hanyang University, 222 Wangsimni-ro, Seongdong-gu, Seoul 04763, Korea
TEL : +82-2-2290-8646, FAX : +82-2-2291-8673, E-mail : hkg@hanyang.ac.kr

Received for publication May 23, 2017; Returned after revision June 26, 2017; Accepted for publication June 29, 2017

substance or mixture that alters the functions of the endocrine system and as a result causes negative health effects in an organism, its progeny, or its biomass is called an 'endocrine disruptor', while a substance that is expected to disrupt the endocrine system is called a 'potential endocrine disruptor'[1]. These substances, often referred to as 'environmental hormones,' became a hot issue in the mid-1990s, and related research was carried out in various fields with great interest for about 20 years, leading to the accumulation of ample information. These substances are under strict regulation in many developed countries including South Korea. Research is associated with the quantification assessment of harmfulness and the development of substitutes[2].

Hormones become effective through several activation steps and regulate the physiology and behavior of an organism. From hormone synthesis, hormone release, hormone transport to target tissue, hormone-receptor recognition, binding and receptor activation to even modulation of gene expression and cell proliferation at the DNA level, the environmental hormones can have a wide range of impacts[3]. The most interesting effect of these materials is estrogenicity, a term that refers to having similar characteristics with estrogen (estradiol), the primary female sex hormone of the body. The molecular structure of these substances is a three-dimensional chain similar to that of estrogen (estradiol), which leads them to being misrecognized by the reproductive system and they affect reproductive cell differentiation (Fig. 1). A study on animal models and humans showed that these substances could cause autoimmune diseases, cardiovascular diseases, neurological diseases and reproductive/endocrine diseases such as breast cancer/prostate cancer, infertility, and endometriosis to the mother and offspring[1].

Types of Environmental Hormones

The research on environmental hormones that can cause endocrine disruption is being actively carried out, the types of related substances are diverse[2]. The WWF (World Wildlife Fund) has classified 67 types of harmful chemical substances as priority research subjects while in Japan, the Ministry of Health, Labor and Welfare has classified around 140 types of substances, such as industrial chemicals, medicines and food additives, as being the cause of endocrine disruption[3]. The most representative examples among them are industrial chemicals and related substances, pesticides and herbicides, organochlorinated chemicals, synthetic estrogens, etc.[2].

1. Dichlorodiphenyl-trichloroethane (DDT)

Dichlorodiphenyl-trichloroethane (Chemical Abstracts Service [CAS] No. 50-229-3, 1,1'-[2,2,2-trichloroethane-1,1-diyl] bis [4-chlorobenzene], DDT) was previously used as a raw material of pesticides and herbicides and a representative substance known as an environmental hormone (Fig. 2). DDT, which is not present in nature, is a colorless hydrophobic substance produced by reacting chloral (CCl_3CHO) and chlorobenzene (C_6H_5Cl) in the presence of a sulfuric acid catalyst. Müller discovered the characteristic of acting as an insecticide and received the Nobel Prize in Physiology or Medicine in 1948. After that, the consumption increased sharply, and in 1963, 83,000

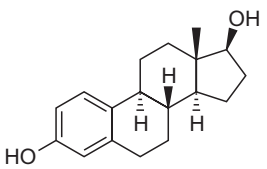

Fig. 1. Estrogen (estradiol).

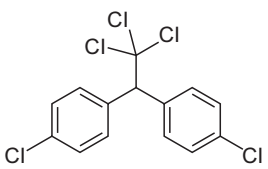

Fig. 2. Dichlorodiphenyl-trichloroethane (DDT).

tons, the maximum annual amount, was produced. There is now a worldwide ban on agricultural usage following the Stockholm Convention on Persistent Organic Pollutants, an international environmental treaty to eliminate or restrict the production of persistent organic pollutants in 2001[4]. However, the chemical is still used in areas where malaria is a primary health concern for disease vector control[5].

DDT persists in soil and sediment from 22 days to 30 years according to the given conditions. Because of its lipophilic properties, DDT also accumulates in the fat of all organisms in the food chain[6]. WHO classifies acute toxicity in humans as "moderate toxicity" based on an LD50 of 113 mg/kg in rats[6]. According to a review article in The Lancet, DDT shows the properties of so-called endocrine glands, which disturb semen quality, menstruation, gestational length and duration of lactation[7]. Several reports have shown that DDT and dichlorodiphenyl-dichloroethylene (DDE) also interfere with proper thyroid function in pregnant women and children[8,9].

2. Dioxin

Dioxin and benzofuran are the polyhalogenated organic compounds, representative environmental hormones and classified as anti-estrogenic material. Dioxin is colorless, odorless, and has been shown to accumulate *in vivo* due to its lipophilic properties and to cause developmental disturbances and cancer in organisms. The name dioxin comes from a dioxygenated ring in the molecular structure and connects the benzene rings to both sides, which is a very stable structure[10]. The most commonly used dioxins are polychlorinated dibenzodioxins (PCDD), 1,2,3,7,8-pentachlorodibenzofuran (PeCDF) derivatives, and 2,3,7,8-tetrachlorodibenzo-dioxin (TCDD), the most toxic component of the defoliant (Fig. 3)[11].

Dioxin was present in a low concentration in the natural state, but it was detected in all humans as a circulation process of rain, soil, and organisms through atmospheric layers as it was discharged as flue gas at chemical plants, fossil fuel engines, waste incinerators and some organic chlorine manufacturing processes[12]. In particular, dioxin present in the mother is transmitted to the newborn through breastfeeding, which increases the burden on the body compared to the child who does not breastfeed. For this reason, the WHO consultation group is evaluating pre-pregnancy dioxin exposure[13]. The accumulation of dioxin in adipose tissue, exposure to trace amounts can eventually reach dangerous levels. In 1994, the United States Environmental Protection Agency (US EPA) reported dioxin as a possible carcinogen and classified the most toxic TCDD in the International Agency for Research on Cancer (IARC) as a Group 1 carcinogen. However, the US EPA says that the non-cancer effects (reproductive and sexual development, immune system) could pose a greater threat to human health[14].

3. Synthetic Estrogen

Synthetic estrogens have a molecular structure similar to that of estrogen. Diethylstilbestrol (DES) and ethinylestradiol are typical examples of synthetic estrogens.

DES has been used as a medication for sexual hormone related symptoms such as remedies for patients suffering from recurrent miscarriage, hormone replacement therapy in hypoestrogenism patients, and emergency contraceptives (Fig.

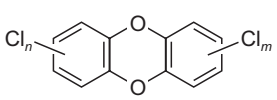

Fig. 3. Polychlorinated dibenzodioxins (PCDD).

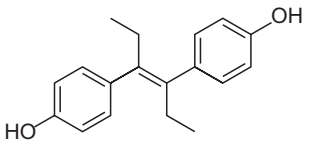

Fig. 4. Diethylstilbestrol (DES).

4). From 1941 to 1971, about 3 million pregnant women in the United States were prescribed DES and the synthetic estrogen was also widely used in Canada, the United Kingdom, Europe, Australia and New Zealand during the same period. Women who were prescribed DES during pregnancy had a slightly increased risk of breast cancer and breast cancer mortality[15]. Another problem is the impact on the next generation, which leads to the term DES daughters, DES sons. DES daughters have been reported to have an increased risk of infertility, spontaneous abortion, ectopic pregnancy, stillbirth, premature menopause, cervical epithelial cell cancer, and breast cancer aged 40 years and older[16]. In addition, cohort studies confirm that the risk of testicular cancer, premature ejaculation, infertility, and atypical genitourinary organs is increased in DES sons[17].

4. Industrial Chemicals and Related Substances

Phthalates (bis[2-ethylhexyl] phthalate [DEHP], benzyl butyl phthalate [BBP], dibutyl phthalate [DBP], dipentyl phthalate [DPP], dipropyl phthalate [DPrP], etc.), alkylphenols, bi-phenolic compounds (bisphenol A [BPA], bisphenol F [BPF]), and polychlorinated biphenyls (PCB), which are classified as "Industrial Chemicals and Related substances," are widely used in everyday life and can be easily contacted[18]. Among them, BPA and phthalate, which are representative of environmental hormone substances, are as follows.

1) Bisphenol A (BPA)

BPA (CAS No. 80-05-7, 4,4'-isopropylidene-diphenol, BPA) is an organic compound produced by the condensation of two molecules of phenol and one molecule of acetone (Fig. 5). BPA has been used as a basic raw material for the synthesis of polycarbonate plastic (used for materials such as compact discs, automobile parts, infant bottles, plastic bowls, spectacle lenses, and anti-shock agents) and epoxy resin (used for materials such as food cans, bottle caps, food packaging materials, and dental resins) for more than 50 years. It is also used as an antioxidant and a vinyl chloride stabilizer in the production of synthetic resins.

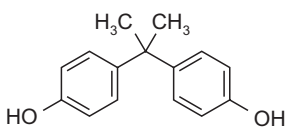

Fig. 5. Bisphenol A.

Most of the work on BPA efflux has been made in epoxy resins and sealants used in dental materials. The amount of BPA detected in saliva after restorative treatment with resin was reported to be 30 μg/ml for 1 hour post-treatment by Olea et al.[19] and 2.8 μg/ml by Arenholt-Bindslev et al.[20]. Fung et al.[21] reported that BPA concentrations in saliva were in the range of 5.8 to 105.6 ppb at 1 and 3 hours after sealant treatment and were not detected in serum. Results from Joskow et al.'s study[22] showed that BPA concentration in saliva was up to 96.2 ng/ml in subjects treated with two sealants and BPA in urine was also increased in one group.

BPA in the body is also found in umbilical cord blood and amniotic fluid, indicating that it is transferred from the embryonic development stage to the fetus[23]. In studies by Völkel et al.[24], the concentrations of BPA and metabolites in human urine exposed to D16-BPA at low concentrations (0.00028~0.00063 mg/kg bw) reached peak levels between 1 and 3 hours. Most of the absorbed BPA (85%~100%) was excreted through the urine and had a half-life of 4~5.4 hours[24].

As worldwide interest in BPA exposure has increased, countries have been predicting BPA exposure levels and providing safety standards. The European Union (EU) calculated the maximum amount of BPA that could be exposed to pathways such as food intake, based on the BPA transport from polycarbonate packaging and the BPA concentration in the epoxy lined metal can. In this report, it was suggested that the use of baby bottles may contain 50 μg/L of BPA, and adults could detect up to 650 μg/L when consuming

wine[25]. According to the Korea Food and Drug Administration, a large amount of BPA was detected in canned products, and up to 13.66 ppb could be detected in canned fish with long shelf life, and 4.2~29.4 ppb (μg/kg) was detected in an infant bottle containing water at 60°C. In the same report, the annual exposure to BPA shows a trend of decreasing the estimated exposure amount due to the increased interest in BPA[26].

2) Phthalate

Diethylhexyl phthalate (CAS No. 84-66-2, DEHP) is a plasticizer component used to increase plastic flexibility (Fig. 6). It is the most widely used material to make cellulose ester plastic such as photographic film and photo paper, blister packaging, tape application, insecticide spray, mosquito repellent, cosmetic and plastic more flexible. It may also be used as a diluent in polysulfides of fragrance diluents, alcohol modifiers, nitrocellulose and cellulose acetate solvents, plasticizers in solid rocket propellants, dye carriers, wetting agents, perfume fixatives, and dental impression materials.

Hauser et al.[27] reported that the phthalate metabolites detected in 379 men who were infertile were associated with increased sperm DNA damage. Acute toxicity has been reported in the nervous system, kidney, digestive system, and skin when exposed to high doses of a few grams or more in human or experimental animals. However, chronic toxicity, mutagenicity and carcinogenicity are unknown[28,29].

DEHP is known to be rapidly absorbed into the skin, intestinal tract, peritoneal membrane and lungs. In the experiment using human epithelial cells, the lag time was 6 hours and the normal absorption rate was 12.8 μg/cm^3/hr. Absorbed DEHP was excreted in monoester derivatives (67%~70%), phthalic acid (8%~9%), and parent compounds (0.1%~0.4%) within 24 hours in animal experiments, and about 85% to 93% of them were excreted within one week of administration, and the *in vivo* half-life was reported to be 2.22 days[30].

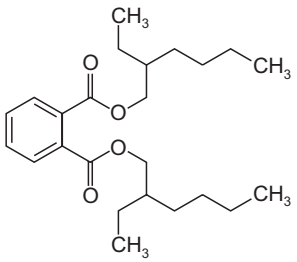

Fig. 6. Bis-phthalate.

The Specificity of Oral Environment, the Pathway of Environmental Hormones into the Body

Environmental hormones enter the body and function as body hormones. At this time, the beginning of the main absorption pathway is absolutely the mouth (oral cavity), and can be absorbed into the body through skin contact, sap, dialysis, and the like. Environmental hormone components are widely used in food additives that come into direct contact with the mouth, in medicine used for cultivation, and in dishes made of synthetic polymers. Therefore, the oral cavity is directly exposed to the absorption of the body. In fact, in early 1998, BPA was detected in food containers and baby bottles, and in 1999, dioxins were detected in livestock products in Belgium. Society has given the issue significant attention[3].

The oral environment is exposed to a much more dynamic situation than other body parts (Fig. 7). First, the oral cavity is known to be able to withstand temperatures higher than other body parts. Studies have shown that people drink black coffee at temperatures ranging from 60°C~88°C[31]. This study evaluates soft tissue mucosa and hard tissue teeth exposed to more extreme environments. In the oral cavity, saliva is always present and exposed to organic acids, forming a unique space. Most materials are characterized by their ability to degrade more easily than liquids in the air,

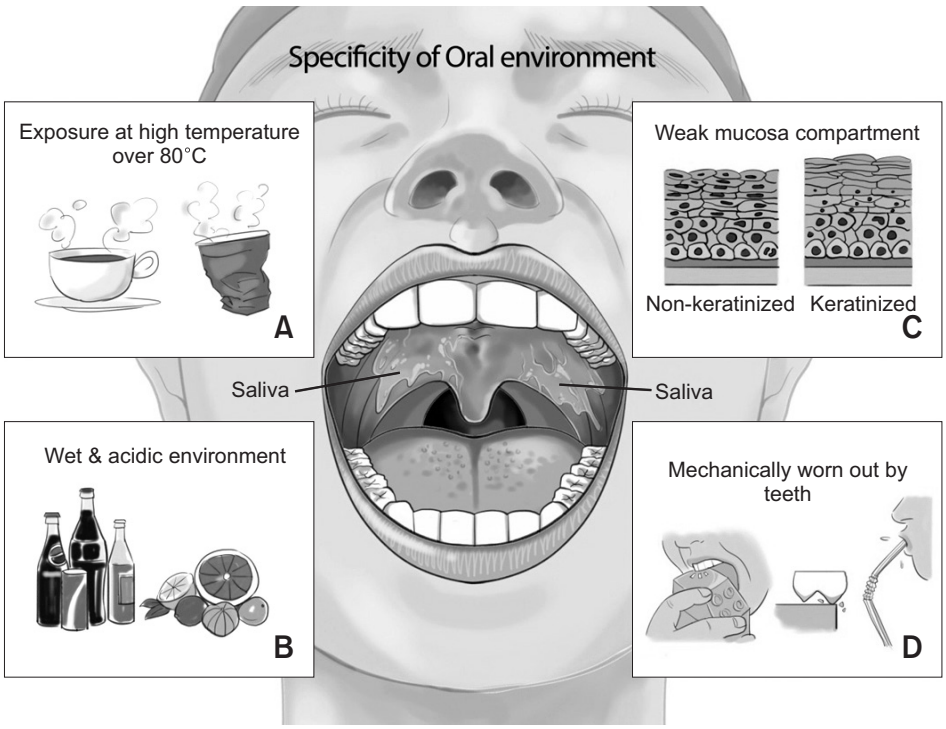

Fig. 7. (A~D) Specificity of oral environment.

especially when exposed to acids. In the oral cavity, the hard tissue, called the tooth, is exposed uniquely among the body parts, so that it can be mechanically chewed and subsequently experience wear of the components. And the oral cavity is composed of mucous membranes, and unlike skin, it is vulnerable to defend mechanical stimuli due to non-keratinized compartment.

Environmental hormones are used daily, so situations in which they come into contact with the mouth occur spontaneously. Among them, environmental hormones contained in tableware such as bottles, food containers, straws and inner coatings of beverage cans are most frequently exposed to the oral cavity and are responsible for a large part of the body's absorption. It has recently been noted that infant toys may inadvertently come in contact with the oral cavity during normal usage and attention has also been paid to the ingredients contained therein. In the field of medicine, extensive research on the safety of synthetic resin products used for liquid containers and dental material has been carried out. In summary, it is noted that among the synthetic resin products, there is a possibility of contact with the oral cavity. Therefore, the possibility of introducing toxic chemical components into the body through the oral cavity should be treated as an important health issue.

Needs for Studies about Environmental Hormone Substances in Oral Environments

The process of decomposition of an object in the natural world consists of two processes: physical decomposition and chemical decomposition. Similarly, the process by which constituent molecules can be liberated in already manufactured synthetic resin products can be largely accomplished by mechanical and chemical decomposition[32]. The oral environment is composed of a special anatomical structure called teeth, which causes mechanical abrasion to occur continuously, and constituents may leak out due to softening, abrasion, and mechanical grinding of synthetic resin products

that come into contact with the oral cavity[33].

In addition, the ester bond, which is usually present in polymeric materials, can be degraded by the hydrolysis process and can be promoted by water, pH, and the temperature of the reactants[33]. The oral cavity is a vulnerable environment for decomposition because it is easy to form low pH conditions due to the nature of organic acids and foods. In fact, when a solution of pH 2~12 (pH 2, 4, 5.6, 6, 8, 10, and 12) is applied to a polycarbonate glass containing BPA, BPF or their diglycidyl ethers (BFDGE, BADGE), the results were the worst at pH 2~4 and the optimum at pH 9[34]. Experiments comparing the release of phthalate at various temperatures that can occur in the oral environment have reported an increase in constituent molecules released at high temperatures. Fujii et al.[35] reported that a surface of plastic materials containing diethylhexyl phthalate (DEHP) at a temperature of 80°C released 100 times more DEHP than at 20°C. In a study of the amount of BPA incorporated in polycarbonate glass at 50°C and 70°C, a higher proportion of BPA was eluted with increasing temperature[34].

Environmental Hormone Research Direction Considering Specificity of Oral Environment

Over the last two decades, research on environmental hormones has been cumulative, but there are still many environmental hormones in everyday life. It is absolutely necessary that the oral cavity occupy the pathway of these substances into the human body. The problem of using materials as environmental hormones in the production of plastic tableware is already an issue and the industry is developing products that do not have environmental hormones. A lot of resin products are used extensively in the field of dental materials. There has been a rapid increase in the number of studies about the safety of environmental hormones when they are exposed to the oral environment. Because of these efforts, environmental hormones currently in dental materials are considered safe and guidelines for their safe use are presented. Recent studies have reported that BPA was not leaked when testing saliva of patients 5 minutes and 7 days after being treated with resin[36]. Despite these efforts, however, it is difficult to predict the elution of environmental hormones from dental resin material in the oral cavity because of its unique environment and extended exposure. Therefore, it is necessary to develop safe ingredients that can replace environmental hormone related substances contained in dental materials.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Acknowledgement

The authors wish to thank the financial support from Ministry of Science, ICT and Future Planning of Korea (Grant Number: NRF-2015M3C8A6A06014792).

References

1. Bergman Å, Heindel JJ, Jobling S, Kidd KA, Zoeller RT. Endocrine disrupting chemicals 2012. Geneva: United Nations Environment Programme and the World Health Organization; 2013.
2. Lee CW, Choi KH, Jeong SW, Kim HL, Seo YR. An overview and future perspective on endocrine disruptors. J Korean Endocr Soc. 2009; 24: 7-14.
3. Kang CG, Lee SH, Kim EK. Endocrine disruptors. J Korean Med Assoc. 2007; 50: 359-68.
4. Stockholm Convention on Persistent Organic Pollutants [Internet]. New York (NY): United Nations Environment Programme, 2001 [cited 2007 Jul 20]. Available from: http://www.pops.int/documents/convtext/convtext_en.pdf.
5. World Health Organization. The use of DDT in

malaria vector control: WHO position statement. Geneva: World Health Organization; 2011.
6. World Health Organization International Programme on Chemical Safety. The WHO recommended classification of pesticides by hazard and guidelines to classification 2009. Geneva: World Health Organization; 2010.
7. Rogan WJ, Chen A. Health risks and benefits of bis(4-chlorophenyl)-1,1,1-trichloroethane (DDT). Lancet. 2005; 366: 763-73.
8. Eskenazi B, Chevrier J, Rosas LG, Anderson HA, Bornman MS, Bouwman H, Chen A, Cohn BA, de Jager C, Henshel DS, Leipzig F, Leipzig JS, Lorenz EC, Snedeker SM, Stapleton D. The Pine River statement: human health consequences of DDT use. Environ Health Perspect. 2009; 117: 1359-67.
9. Chevrier J, Eskenazi B, Holland N, Bradman A, Barr DB. Effects of exposure to polychlorinated biphenyls and organochlorine pesticides on thyroid function during pregnancy. Am J Epidemiol. 2008; 168: 298-310.
10. Beychok MR. A data base of dioxin and furan emissions from municipal refuse incinerators. Atmos Environ. 1987; 21: 29-36.
11. Schecter A, Birnbaum L, Ryan JJ, Constable JD. Dioxins: an overview. Environ Res. 2006; 101: 419-28.
12. Buckley-Golder D, Coleman P, Davies M, King K, Petersen A, Watterson J, Woodfield M. Compilation of EU dioxin exposure and health data. Organohalogen Compd. 1999; 44: 79-82.
13. van Leeuwen FX, Feeley M, Schrenk D, Larsen JC, Farland W, Younes M. Dioxins: WHO's tolerable daily intake (TDI) revisited. Chemosphere. 2000; 40: 1095-101.
14. Geusau A, Schmaldienst S, Derfler K, Päpke O, Abraham K. Severe 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) intoxication: kinetics and trials to enhance elimination in two patients. Arch Toxicol. 2002; 76: 316-25.
15. Palmer JR, Wise LA, Hatch EE, Troisi R, Titus-Ernstoff L, Strohsnitter W, Kaufman R, Herbst AL, Noller KL, Hyer M, Hoover RN. Prenatal diethylstilbestrol exposure and risk of breast cancer. Cancer Epidemiol Biomarkers Prev. 2006; 15: 1509-14.
16. Hoover RN, Hyer M, Pfeiffer RM, Adam E, Bond B, Cheville AL, Colton T, Hartge P, Hatch EE, Herbst AL, Karlan BY, Kaufman R, Noller KL, Palmer JR, Robboy SJ, Saal RC, Strohsnitter W, Titus-Ernstoff L, Troisi R. Adverse health outcomes in women exposed in utero to diethylstilbestrol. N Engl J Med. 2011; 365: 1304-14.
17. Palmer JR, Herbst AL, Noller KL, Boggs DA, Troisi R, Titus-Ernstoff L, Hatch EE, Wise LA, Strohsnitter WC, Hoover RN. Urogenital abnormalities in men exposed to diethylstilbestrol in utero: a cohort study. Environ Health. 2009; 8: 37.
18. Oehlmann J, Oetken M, Schulte-Oehlmann U. A critical evaluation of the environmental risk assessment for plasticizers in the freshwater environment in Europe, with special emphasis on bisphenol A and endocrine disruption. Environ Res. 2008; 108: 140-9.
19. Olea N, Pulgar R, Pérez P, Olea-Serrano F, Rivas A, Novillo-Fertrell A, Pedraza V, Soto AM, Sonnenschein C. Estrogenicity of resin-based composites and sealants used in dentistry. Environ Health Perspect. 1996; 104: 298-305.
20. Arenholt-Bindslev D, Breinholt V, Preiss A, Schmalz G. Time-related bisphenol-A content and estrogenic activity in saliva samples collected in relation to placement of fissure sealants. Clin Oral Investig. 1999; 3: 120-5.
21. Fung EY, Ewoldsen NO, St Germain HA Jr, Marx DB, Miaw CL, Siew C, Chou HN, Gruninger SE, Meyer DM. Pharmacokinetics of bisphenol A released from a dental sealant. J Am Dent Assoc. 2000; 131: 51-8.
22. Joskow R, Barr DB, Barr JR, Calafat AM, Needham LL, Rubin C. Exposure to bisphenol A from bis-glycidyl dimethacrylate-based dental sealants. J Am Dent Assoc. 2006; 137: 353-62.
23. Engel SM, Levy B, Liu Z, Kaplan D, Wolff MS.

Xenobiotic phenols in early pregnancy amniotic fluid. Reprod Toxicol. 2006; 21: 110-2.
24. Völkel W, Bittner N, Dekant W. Quantitation of bisphenol A and bisphenol A glucuronide in biological samples by high performance liquid chromatography-tandem mass spectrometry. Drug Metab Dispos. 2005; 33: 1748-57.
25. Shelby MD. NTP-CERHR monograph on the potential human reproductive and developmental effects of bisphenol A. Ntp Cerhr Mon. 2008; (22): v, vii-ix, 1-64 passim.
26. Korea Food and Drug Administration (KFDA). Monitoring studies on bisphenol A and phthalates in food and container packaging. Cheongju: KFDA; 1999.
27. Hauser R, Meeker JD, Singh NP, Silva MJ, Ryan L, Duty S, Calafat AM. DNA damage in human sperm is related to urinary levels of phthalate monoester and oxidative metabolites. Hum Reprod. 2007; 22: 688-95.
28. Pohanish RP. Sittig's handbook of toxic and hazardous chemicals and carcinogens. Norwich: William Andrew; 2008.
29. Bingham E, Cohrssen B, Powell CH. Patty's toxicology. Volume 1-9. New York: Wiley; 2001.
30. World Health Organization. International Programme on Chemical Safety. Geneva: World Health Organization; 2003.
31. Lee HS, O'Mahony M. At what temperatures do consumers like to drink coffee?: mixing methods. J Food Sci. 2002; 67: 2774-7.
32. van Groeningen G, Jongebloed W, Arends J. Composite degradation in vivo. Dent Mater. 1986; 2: 225-7.
33. Göpferich A. Mechanisms of polymer degradation and erosion. Biomaterials. 1996; 17: 103-14.
34. Oca ML, Sarabia LA, Herrero A, Ortiz MC. Optimum pH for the determination of bisphenols and their corresponding diglycidyl ethers by gas chromatography-mass spectrometry. Migration kinetics of bisphenol A from polycarbonate glasses. J Chromatogr A. 2014; 1360: 23-38.
35. Fujii M, Shinohara N, Lim A, Otake T, Kumagai K, Yanagisawa Y. A study on emission of phthalate esters from plastic materials using a passive flux sampler. Atmos Environ. 2003; 37: 5495-504.
36. Lee JH, Yi SK, Kim SY, Kim JS, Son SA, Jeong SH, Kim JB. Salivary bisphenol A levels and their association with composite resin restoration. Chemosphere. 2017; 172: 46-51.