



In utero exposure to bisphenol-A and its effect on birth weight of offspring[☆]

Maohua Miao^{a,b}, Wei Yuan^{b,*}, Guoping Zhu^c, Xuefei He^c, De-Kun Li^{a,**}

^a Division of Research, Kaiser Foundation Research Institute, Kaiser Permanente, Oakland, CA, United States

^b Department of Epidemiology and Social Science on Reproductive Health, Shanghai Institute of Planned Parenthood Research, WHO Collaborating Center for Research in Human Reproduction, National Population & Family Planning Key Laboratory of Contraceptive Drugs and Devices, Shanghai, China

^c Yixing Center for Health Surveillance, Yixing, China

ARTICLE INFO

Article history:

Received 17 January 2011

Received in revised form 18 February 2011

Accepted 11 March 2011

Available online 31 March 2011

Keywords:

Bisphenol-A

Birth weight

In utero exposure

Occupational exposure

Endocrine disruptor

ABSTRACT

To examine the effect of *in utero* BPA exposure on the birth weight of offspring, a total of 587 children from families in which parent(s) did or did not have occupational exposure to BPA were examined. Their birth weights were obtained by an in-person interview of the mother. Parental BPA exposure level during the index pregnancy was determined through personal air sampling measurements and exposure history. After controlling for potential confounders, parental exposure to BPA in the workplace during pregnancy was associated with decreased birth weight. The association was stronger for maternal exposure which is statistically significant ($P=0.02$). A dose–response relationship was observed with increased BPA exposure levels in pregnancy associated with greater magnitude of decrease of birth weight in offspring ($P=0.003$). Our findings provide the new epidemiologic evidence suggesting that *in utero* exposure to BPA during pregnancy may be associated with decreased birth weight in offspring.

© 2011 Elsevier Inc. All rights reserved.

1. Introduction

Bisphenol-A (BPA) is a monomer used in the manufacture of polycarbonate plastics and resins. It is one of the more potent endocrine disrupting chemicals (EDCs) that have raised significant public health concerns because of widespread human exposure. BPA has been shown to leach from a variety of resin-based and plastic products, including baby bottles, dental sealants, food and water containers, as well as the lining of beverage and food cans [1–3]. Animal studies showed that BPA has both estrogenic and antiandrogenic properties. It can reduce the sperm quality of exposed animals, disturb hormonal balance, and cause reproductive organ damage and malformations in offspring [4–7]. The adverse effect of BPA is particularly potent when the exposure occurs during the prenatal period, because the developing fetus is especially susceptible to chemical effects, and the fetus' liver has limited capacity to metabolize BPA [6,8,9].

Abbreviations: BPA, bisphenol A; TWA₈, 8-h time-weighted average.

[☆] **Funding/support:** This study was supported by a grant from the U.S. National Institute of Occupational Safety and Health (NIOSH) (R01 OH007580) and National basic research program of China (973 program) 2010CB529505.

* Corresponding author.

** Corresponding author at: Division of Research, Kaiser Permanente Northern California, 2000 Broadway, Oakland, CA 94612, United States. Tel.: +1 510 891 3755.

E-mail addresses: yuanwei11@yahoo.com (W. Yuan), dkl@dor.kaiser.org (D.-K. Li).

Reproductive systems have been the major endpoint for assessing BPA toxicity. Some evidence suggests that prenatal exposure to BPA, like diethylstilbestrol (DES), can adversely affect fetal growth [10]. DES and BPA are similar in chemical structure and prenatal DES exposure has been known to lead to lower birth weight in offspring of mice [11]. In recent animal studies, prenatal exposure to BPA in varying doses has also been reported to be associated with a significant reduction in the body weight of mice or rats [12–14]. Decreased birth weight has also been found in sheep following BPA exposure from days 30 to 90 of gestation [15]. However, inconsistent reports also exist [16,17]. The discrepancy in animal species, administration route and exposure dosage make the comparison of existing studies difficult.

Epidemiological studies of the prenatal effect of BPA exposure remain very limited. In a study of 125 pregnant women, fetal head circumference and abdominal circumference in the third trimester determined through ultrasound examination were found to be negatively correlated with maternal BPA level measured during the first trimester [18]. Birth weight was found to be slightly lower in offspring whose mothers' blood BPA levels were higher (>5 ng/ml), while the difference was not statistically significant [19]. In another study, no association was found between birth length and weight and maternal BPA level examined in third trimester [20].

In the present study, we evaluated the effect of parental BPA exposure in the workplace during pregnancy on the birth weight of offspring in a retrospective cohort study.

2. Materials and methods

2.1. Study population

The study population was identified from the participants of a study evaluating the reproductive effects of BPA. BPA-exposed (from manufacturers of BPA and epoxy resin) and unexposed workers (from industries without BPA exposure) and their spouses and offspring were recruited from 2003 to 2008. A detailed description of the original study can be found elsewhere [21]. The study was approved by the committees for the protection of human subjects from all participating institutes. All participant families provided written informed consent.

The participant rate of BPA exposed and unexposed workers were 67.32% and 60.69%, respectively. Children of the BPA-exposed parents were eligible for the current study if: (1) they were a live-born singleton and (2) one of their parents was involved in BPA-exposed occupations during the index pregnancy. Children of BPA-unexposed workers were all eligible if they were live-born singletons. A total of 587 eligible children were identified, among whom 444 children were born to unexposed parents, 93 had a father who was exposed to BPA in the workplace while his wife was pregnant with the index child, and 50 had a mother who was exposed to BPA in the workplace during pregnancy.

2.2. Measurement of exposure

Parents were first classified into “exposed” or “unexposed” categories based on whether they were employed in the BPA-exposed factories during the index pregnancy. To further measure BPA exposure level, for exposed workers, personal air sample monitoring was carried out to obtain their current exposure level. TWA_8 (8-h time-weighted average) was then calculated to measure the BPA exposure level of a given job title based on the personal air sample monitoring results from this job title. Past exposure level was estimated based on the combination of the current exposure level, employment history, change of manufacturing process and work environment, and use of protective measures. A job/exposure matrix was then constructed to provide BPA exposure level (TWA_8) during the index pregnancy by individuals who were blinded to the workers’ current urinary BPA concentrations as well as the outcome data (birth weight).

We also assayed urine BPA level among those participants who provided their urine samples. The results have been previously reported [22,23]. Although current urine BPA may not reflect the actual exposure level during the index pregnancy, it nevertheless provided an additional measure for personal exposure level of both BPA-exposed and BPA-unexposed groups.

2.3. In-person interview

An in-person interview was performed by trained interviewers with all of the participating workers and their spouses to collect the following information: socio-demographic characteristics, reproductive and medical history, health related behaviors, exposure to environmental hazards, and employment history. In addition, for the mothers, information about the index pregnancy was collected on birth outcomes including infant sex, birth weight, gestational age, gravidity and parity and any gross abnormalities of newborns, as well as the mother’s height, weight, illnesses, medications, smoking and drinking before and during the index pregnancy.

2.4. Data analysis

Birth weight of offspring was compared between the exposed (mother or father who was exposed to BPA in their workplaces during the index pregnancy) and unexposed groups. Using birth weight as a continuous variable, we examined the relationship between parental BPA exposure and offspring’s birth weight using multiple linear regression models after controlling for maternal age at birth, maternal body weight before pregnancy, calendar year of birth, maternal education, family income and gravidity.

BPA exposure during the index pregnancy was further divided into several dose categories. Because paternal exposure is considered an indirect maternal exposure during pregnancy (e.g., through contamination or maternal visits to factories) which is more relevant for *in utero* exposure level, paternal exposure was classified in lower exposure categories than maternal exposure. We tested this assumption among those women who provided urine samples for BPA assay. The results confirmed that urine BPA levels showed a gradient reduction from exposed women (direct fetal exposure) to spouses of exposed male workers (indirect fetal exposure through paternal exposure) to unexposed women. The geometric mean (95% CI) of maternal current urine BPA was 15.98 (9.11–28.02), 2.22 (1.49–3.31) and 0.56 (0.70–0.88) $\mu\text{g/g Cr}$ in currently exposed mothers, spouses of exposed fathers and unexposed mothers (including unexposed mothers and spouses of unexposed fathers), respectively. To evaluate the dose–response relationship, among each of the paternal and maternal exposed categories, we further divided the exposure into high and low categories based on their TWA_8 during the index pregnancy. TWA_8 measurement appeared to be a reasonably good measure of actual personal BPA exposure. Using current TWA_8 against current urine BPA level, we found that among the exposed women, the geometric means (95% CI) of urine BPA level were 33.01 (15.06–72.33) and 10.79 (3.69–31.50) $\mu\text{g/g Cr}$ for high and low TWA_8 category (with

the median TWA_8 as cutoff), respectively, supporting the rationale for using TWA_8 as a measure for BPA exposure level.

Categorical variables were used to examine the effects of different exposure levels using the classification of parental unexposed, paternal low TWA_8 , paternal high TWA_8 , maternal low TWA_8 , and maternal high TWA_8 . We also used continuous variables (with the value of 1, 2, 3, 4, 5 corresponding to the above categories) to examine the trend of a dose–response relationship. *P*-values of <0.05 were considered statistically significant.

3. Results

As shown in Table 1, maternal age at birth, maternal weight before the index pregnancy, and the number of weeks worked during pregnancy were similar among the mother-exposed group, father-exposed (mother-indirectly-exposed) group, and parents-unexposed group, as was the rate of paternal smoking during pregnancy. Higher maternal education level and higher family income were found in the mother-exposed group. No mothers smoked during the index pregnancy. Distribution of gender of offspring was similar among the three groups. Offspring in the mother-exposed group were less likely to be the result of the first pregnancy. In addition, they were slightly younger at the time of interview. There was a slightly higher rate of preterm birth in the mother-exposed group.

After controlling for maternal age at birth, maternal weight before pregnancy, calendar year of birth, maternal education, family income and gravidity, parental exposure to BPA in the workplace during pregnancy was correlated with decreased birth weight in offspring: compared with offspring from the families without parental BPA exposure in the workplace. Birth weight of offspring with paternal BPA exposure was 90.75 g less on average ($P=0.10$), and 168.40 g less for those with maternal BPA exposure ($P=0.02$). To reduce the influence of preterm delivery, we restricted the analyses among term births. The association remained largely the same. The number of preterm births was too small to produce any reliable estimates (Table 2).

To further reduce the recall error for birth weight, we repeated analyses among those whose offspring were less than 15 years of age, which represented children born after the early 1990s when vital statistical records were better maintained. The same association was observed, though it was slightly strengthened, likely due to reduced recall error (Table 2).

Based on TWA_8 level, we also observed a dose–response relationship between increased BPA TWA_8 level and decreased birth weight. Compared to the unexposed group, the average birth weight was lowered by 57.48 g, 153.00 g, 196.10 g, and 234.75 g for paternal low TWA_8 , paternal high TWA_8 , maternal low TWA_8 , and maternal high TWA_8 , respectively. The *P*-values of the trend tests were 0.003 (Table 3).

4. Discussion

Although it needs to be confirmed by other studies, this study provides the first epidemiological evidence that parental exposure to BPA in the workplace during pregnancy was associated with decreased birth weight of offspring. The association was strengthened after restricting to term birth and offspring whose current age was less than 15 to reduce recall errors. We also observed a dose–response relationship, with a greater magnitude of decrease in birth weight in children whose mothers were directly exposed to high BPA levels during pregnancy, followed by those whose mothers were exposed to low BPA levels, then by those whose mothers had BPA exposure through paternal high BPA exposure, and finally, the least decrease of birth weight in the offspring whose mothers had BPA exposure through low paternal BPA exposure.

Although adverse effect of prenatal BPA exposure on birth weight has not been reported in human studies, some studies

Table 1
Characteristics of BPA-exposed and unexposed parents.

	BPA-exposed		BPA-unexposed (n = 444)
	Mother exposed (n = 93)	Father exposed ^a (n = 50)	
Average maternal age at birth (year) ^b	26.20 (2.45)	25.76 (2.80)	25.4 (2.64)
Average maternal weight before index pregnancy (kg) ^b	51.56 (9.43)	51.29 (8.89)	50.45 (5.99)
Average working weeks of mother during the index pregnancy ^b	29.31 (12.88)	34.17 (11.54)	31.42 (12.04)
Maternal education			
Middle school or less	6 (12.24%)	39 (44.83%)	200 (45.87%)
High school	32 (65.31%)	40 (45.98%)	187 (42.89%)
College or more	11 (22.45%)	8 (9.20%)	49 (11.24%)
Family income (RMB ^c /month/person)			
≤500	10 (20.41%)	41 (47.13%)	119 (27.23%)
500–1000	34 (69.39%)	43 (49.43%)	287 (65.68%)
>1000	5 (10.20%)	3 (3.45%)	31 (7.09%)
Paternal smoking during pregnancy	27 (54.00%)	48 (51.61%)	234 (52.94%)
Gravidity			
1	25 (50.00%)	52 (56.52%)	252 (60.14%)
2	21 (42.00%)	27 (29.35%)	125 (29.83%)
>2	4 (8.00%)	13 (14.13%)	42 (10.02%)
Gestational age (weeks)			
<37	3 (6.00%)	2 (2.15%)	21 (4.73%)
37–42	46 (92.00%)	91 (97.85%)	418 (94.14%)
>42	1 (2.00%)	0 (0.00%)	5 (1.13%)
Gender of offspring			
Male	26 (52.00%)	50 (53.76%)	242 (54.50%)
Female	24 (48.00%)	43 (46.24%)	202 (45.50%)
Current age of offspring (year)			
<5	20 (40.00%)	35 (37.63%)	107 (24.10%)
5–10	15 (30.00%)	17 (18.28%)	115 (25.90%)
10–15	5 (10.00%)	20 (21.51%)	85 (19.14%)
15	10 (20.00%)	21 (22.58%)	137 (30.86%)

^a Father exposed considered as mother indirectly exposed.^b Mean (Std).^c RMB: Chinese currency.**Table 2**
Parental BPA exposure during pregnancy and birth weight in offspring.

BPA exposure	n	Birth weight (g) Mean (std)	Regression coefficient ^a	P
Unexposed	444	3398.74 (523.61)	Reference	
Father Exposed	93	3308.60 (539.91)	−90.75	0.10
Mother Exposed	50	3299.40 (428.00)	−168.40	0.02
Among termed birth				
Unexposed	418	3416.36 (512.75)	Reference	
Father exposed	91	3330.22 (515.64)	−90.75	0.10
Mother exposed	46	3309.13 (441.45)	−176.04	0.02
Among termed birth and current age less than 15				
Unexposed	288	3464.15 (492.89)	Reference	
Father exposed	70	3378.57 (514.68)	−104.92	0.09
Mother exposed	38	3337.37 (477.05)	−217.98	0.007

^a With categorical variables in linear regression model, adjusted for maternal age at birth, maternal weight before pregnancy, calendar year of birth, maternal education, family income, gravidity.**Table 3**
Dose–response relationship between parental BPA exposure during pregnancy and birth weight in offspring.

	n	Birth weight (g) Mean (std)	Regression coefficient ^a	Trend test ^b
Both parents unexposed	288	3464.15 (492.89)	Reference	0.003
Paternal low TWA8	40	3417.50 (588.84)	−57.48	
Paternal high TWA8	30	3326.67 (398.86)	−153.00	
Maternal low TWA8	22	3343.18 (392.28)	−196.10	
Maternal high TWA8	16	3329.38 (588.05)	−234.75	

^a With categorical variables in linear regression model, adjusted for maternal age at birth, maternal weight before pregnancy, calendar year of birth, maternal education, family income, gravidity.^b Trend test with BPA exposure from low to high as a continuous variable in the model, adjusted for maternal age at birth, maternal weight before pregnancy, calendar year of birth, maternal education, family income, gravidity.

reported growth restriction after maternal BPA exposure on other growth parameters such as head circumference and fetal growth in the third trimester [18,19]. Therefore, our finding is consistent with the limited reports of human studies although inconsistent finding also exists [20].

Exposure to BPA has been reported to reduce birth weight in animal studies at relatively high levels of exposure (>5 mg/kg/day) [12–14,24]. This finding provides support for our results observed in human population. However, BPA exposure at lower doses has been reported to increase birth weight in some animal studies [16,25,26], suggesting that the effect of BPA on birth weight may be dose-dependent [1]. We were not able to evaluate this aspect of the BPA effect (decreasing birth weight at high levels and increasing birth weight at low levels) in our study population.

Both animal and human studies have shown that BPA can pass through the placental barrier and that fetuses are likely to be exposed to similar (if not higher) levels of BPA as those of mothers [19,27,9]. In the present study, the current urine BPA level among exposed mothers (direct maternal exposure) was much higher than among un-exposed mothers. Combined with the fact that all mothers in the mother-exposed group worked for at least three months during pregnancy, it is conceivable that all offspring in this group had a relatively high level of *in utero* BPA exposure than those in other groups. For the spouses of exposed fathers, although not directly exposed to BPA in the workplace, they were likely to have a higher BPA exposure level than women in the unexposed group (whose spouse and they themselves were not exposed BPA in the workplace), through indirect exposure to contaminated clothing, their own visits to the spouses' (male workers) factories (BPA exposure in the workplace), and residence in the vicinity of the factories. Indeed, through the in-person interviews, we found that 45% of exposed fathers did not routinely change their uniforms before discharge. Most of them (64%) did not routinely take a shower before going home. In addition, 19% of spouses of exposed male workers regularly visited their husbands' factories. Finally, many workers' residences were close to the factories. All of these factors were likely to have led to a secondary exposure to BPA for those female spouses of exposed male workers. Consequently, the offspring of these women would have a relatively higher *in utero* BPA exposure than those whose parents were neither exposed to BPA in the workplace. Our urine assays of BPA levels confirmed that urine BPA level was highest among women with direct BPA exposure in the workplace, followed by spouses of male exposed workers. The unexposed mothers have lowest urine BPA levels. Therefore, our classification of *in utero* BPA exposure levels likely reflected the underlying BPA exposure gradient.

The biologic mechanism for the effect of BPA on birth weight has not been well understood. It was reported that inappropriate exposure to sex hormones or hormone-like chemicals (e.g., EDCs) has an effect on fetal growth. For instance, *in utero* exposure to diethylstilbestrol (DES), an agent with estrogenic and anti-androgen effect and a similar chemical structure as BPA, is associated with intrauterine growth restriction (IUGR) [10,11]. A similar association was found in some other endocrine disruptors [28,29]. Thus, it is possible that increased estrogen or anti-androgen signaling during critical times of fetal development due to exposure to BPA may lead to decreased birth weight.

The present study was limited by the small sample size in the exposed group, which limited our ability to conduct more detailed analyses. Due to the retrospective nature of the study, TWA₈, rather than maternal urine BPA level, was used to classify the exposure dosage during the index pregnancy. Although we have demonstrated that current TWA₈ was highly correlated with current urine BPA, it is still possible that the association between BPA exposure and birth weight was impacted due to inaccurate classification of BPA exposure categories. The misclassification may also come

from the unexposed group: since exposure to BPA can be achieved through consumer product use, some of the workers in the unexposed group may have high BPA exposure level due to exposure to other BPA sources. Given that any misclassification of BPA exposure level is likely to be non-differential (not related to birth weight), the effect of such misclassification, if existed, would have attenuated the association toward null.

The validity of parental recall of birth weight has been reported to be good for up to 16 years after delivery [30]. The accuracy of recall in our case may be better because most of the families in our study had just one child. Nonetheless, the misclassification derived from recall error could not be ruled out. The strengthened association after restricting the analysis to less than 15 years supports the argument that such misclassification, if it does exist, might have diluted the observed association.

While the results need to be replicated in other studies, our findings provide preliminary evidence suggesting that exposure to BPA during pregnancy may have an adverse effect on fetal growth. It should be noted that the observed association may be dose-dependent, and only apply to high level BPA exposure. A similar effect at lower environmentally exposed levels remains unclear.

Conflict of interest statement

No conflicts of interest are to be declared.

Acknowledgement

We would like to thank Roxana Odouli for her help in developing data collection instruments and preparing the manuscript and the participating factories for their cooperation.

References

- [1] vom Saal FS, Hughes C. An extensive new literature concerning low-dose effects of bisphenol A shows the need for a new risk assessment. *Environ Health Perspect* 2005;113(8):926–33.
- [2] Le HH, Carlson EM, Chua JP, Belcher SM. Bisphenol A is released from polycarbonate drinking bottles and mimics the neurotoxic actions of estrogen in developing cerebellar neurons. *Toxicol Lett* 2008;176(2):149–56.
- [3] Calafat AM, Ye X, Wong LY, Reidy JA, Needham LL. Exposure of the U.S. population to bisphenol A and 4-tertiary-octylphenol: 2003–2004. *Environ Health Perspect* 2008;116(1):39–44.
- [4] Ranjit N, Siefert K, Padmanabhan V. Bisphenol-A and disparities in birth outcomes: a review and directions for future research. *J Perinatol* 2010;30(1):2–9.
- [5] Diamanti-Kandarakis E, Bourguignon JP, Giudice LC, Hauser R, Prins GS, Soto AM, et al. Endocrine-disrupting chemicals: an endocrine society scientific statement. *Endocr Rev* 2009;30(4):293–342.
- [6] Richter CA, Birnbaum LS, Farabolini F, Newbold RR, Rubin BS, Talsness CE, et al. In vivo effects of bisphenol A in laboratory rodent studies. *Reprod Toxicol* 2007;24(2):199–224.
- [7] Salian S, Doshi T, Vanage G. Perinatal exposure of rats to Bisphenol A affects the fertility of male offspring. *Life Sci* 2009.
- [8] Elsby R, Maggs JL, Ashby J, Park BK. Comparison of the modulatory effects of human and rat liver microsomal metabolism on the estrogenicity of bisphenol A: implications for extrapolation to humans. *J Pharmacol Exp Ther* 2001;297(1):103–13.
- [9] Takahashi O, Oishi S. Disposition of orally administered 2,2-bis(4-hydroxyphenyl)propane (bisphenol A) in pregnant rats and the placental transfer to fetuses. *Environ Health Perspect* 2000;108(10):931–5.
- [10] Giusti RM, Iwamoto K, Hatch EE. Diethylstilbestrol revisited: a review of the long-term health effects. *Ann Intern Med* 1995;122(10):778–88.
- [11] Newbold RR, Padilla-Banks E, Snyder RJ, Phillips TM, Jefferson WN. Developmental exposure to endocrine disruptors and the obesity epidemic. *Reprod Toxicol* 2007;23(3):290–6.
- [12] Kim JC, Shin HC, Cha SW, Koh WS, Chung MK, Han SS. Evaluation of developmental toxicity in rats exposed to the environmental estrogen bisphenol A during pregnancy. *Life Sci* 2001;69(22):2611–25.
- [13] Nunez AA, Kannan K, Giesy JP, Fang J, Clemens LG. Effects of bisphenol A on energy balance and accumulation in brown adipose tissue in rats. *Chemosphere* 2001;42(8):917–22.
- [14] Takagi H, Shibutani M, Masutomi N, Uneyama C, Takahashi N, Mitsumori K, et al. Lack of maternal dietary exposure effects of bisphenol A and nonylphenol during the critical period for brain sexual differentiation on the reproductive/endocrine systems in later life. *Arch Toxicol* 2004;78(2):97–105.

- [15] Savabieasfahani M, Kannan K, Astapova O, Evans NP, Padmanabhan V. Developmental programming: differential effects of prenatal exposure to bisphenol-A or methoxychlor on reproductive function. *Endocrinology* 2006;147(12):5956–66.
- [16] Rubin BS, Murray MK, Damassa DA, King JC, Soto AM. Perinatal exposure to low doses of bisphenol A affects body weight, patterns of estrous cyclicity, and plasma LH levels. *Environ Health Perspect* 2001;109(7):675–80.
- [17] Seidlova-Wuttke D, Jarry H, Christoffel J, Rimoldi G, Wuttke W. Effects of bisphenol-A (BPA), dibutylphthalate (DBP), benzophenone-2 (BP2), procymidone (Proc), and linurone (Lin) on fat tissue, a variety of hormones and metabolic parameters: a 3 months comparison with effects of estradiol (E2) in ovariectomized (ovx) rats. *Toxicology* 2005;213(1–2):13–24.
- [18] Lee B, Ha E, Park H, Kim B, Seo J, Chang M, et al. Exposure to bisphenol A in pregnant women and early fetal growth. *Epidemiology* 2010;19(6):S365.
- [19] Padmanabhan V, Siefert K, Ransom S, Johnson T, Pinkerton J, Anderson L, et al. Maternal bisphenol-A levels at delivery: a looming problem? *J Perinatol* 2008;28(4):258–63.
- [20] Wolff MS, Wolff Mary S, Engel Stephanie M, Berkowitz Gertrud S, Ye Xiaoyun, Silva Manori J, et al. Prenatal phenol and phthalate exposures and birth outcomes. *Environ Health Perspect* 2008;116(8):1092–7.
- [21] 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(2):519–27.
- [22] He Y, Miao M, Herrinton LJ, Wu C, Yuan W, Zhou Z, et al. Levels in blood and urine in a Chinese population and the personal factors affecting the levels. *Environ Res* 2009;109(5):629–33.
- [23] He Y, Miao M, Wu C, Yuan W, Gao E, Zhou Z, et al. Occupational exposure levels of bisphenol A among Chinese workers. *J Occup Health* 2009;51(5):432–6.
- [24] NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Bisphenol A. NTP CERHR MON 2008;(22):i–III1.
- [25] Elobeid MA, Allison DB. Putative environmental–endocrine disruptors and obesity: a review. *Curr Opin Endocrinol Diabetes Obes* 2008;15(5):403–8.
- [26] Rubin BS, Soto AM, Bisphenol A. Perinatal exposure and body weight. *Mol Cell Endocrinol* 2009;304(1–2):55–62.
- [27] Nishikawa M, Iwano H, Yanagisawa R, Koike N, Inoue H, Yokota H. Placental transfer of conjugated bisphenol a and subsequent reactivation in the rat fetus. *Environ Health Perspect* 2010.
- [28] Konishi K, Sasaki S, Kato S, Ban S, Washino N, Kajiwara J, et al. Prenatal exposure to PCDDs/PCDFs and dioxin-like PCBs in relation to birth weight. *Environ Res* 2009;109(7):906–13.
- [29] de Siqueira MT, Braga C, Cabral-Filho JE, Augusto LG, Figueiroa JN, Souza AI. Correlation between pesticide use in agriculture and adverse birth outcomes in Brazil: an ecological study. *Bull Environ Contam Toxicol* 2010;84(6):647–51.
- [30] O'Sullivan JJ, Pearce MS, Parker L. Parental recall of birth weight: how accurate is it? *Arch Dis Child* 2000;82(3):202–3.