Systematic Review and Meta-Analysis

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Association between prenatal exposure to bisphenol a and birth outcomes

A systematic review with meta-analysis

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Abstract

Background: Previous studies investigated the relation of prenatal exposure to bisphenol A (BPA) and birth outcomes, but these results were inconsistent. The aim of this study was to investigate the relation of prenatal exposure to BPA and birth outcomes, provide comprehensive results based on current studies.

Methods: The PubMed, Cochrane databases, and Web of Science databases were searched systematically by two researchers respectively from their inceptions to Oct. 2018, using the following keywords "bisphenol A, birth weight, birth length, head circumference, gestational age, birth outcomes". We extracted β coefficient and 95% confidence interval (CI) or β coefficient and standard deviation (SD) from included study. The subgroup analysis was performed to evaluate the potential heterogeneity between studies. We conducted sensitivity analysis by excluding the each individual study to assess the results whether were stable. Finally, the publication bias was performed by accumulative forest plot.

Results: Seven studies with 3004 participants met the inclusion criteria. BPA had significant positively association with birth weight (β = 21.92, 95%CI: 1.50–42.35, $P = .04$). No significant associations were found between BPA and birth length, head circumference and gestational age (All of $P > .05$).

Conclusion: This meta-analysis demonstrated that the BPA was positively associated with birth weight. Therefore, further studies are needed to investigate the critical sensitive period of influencing fetal development and to investigate the difference on gender.

Abbreviations: BPA = bisphenol A, CI = confidence interval, EDCs = endocrine disrupting chemicals, GM = geometric mean, LOD = limits of detection, SD = standard deviation.

Keywords: birth outcomes, birth weight, bisphenol A

1. Introduction

Bisphenol A (BPA) is used widely in the manufacture of polycarbonate plastics, epoxy resins which are used to line food cans, food and beverage containers, dental sealants, medical tubing, and thermal receipt papers.[1,2] BPA is ubiquitous in our daily life, people may get exposed to it through many ways. The studies indicate that BPA can release from the polycarbonate drinking bottles, food and beverage containers, dental sealants,[1,3,4] but ingesting food and water in daily life can be a main exposure approach.[1,5] Some studies have demonstrated that BPA can be detected from human plasma, urine, amniotic fluid, follicular fluid, placental tissue, breast milk and umbilical cord blood, adipose tissue.[6–9]

BPA is an endocrine disrupting chemicals (EDCs) that can exert estrogenic and anti-androgenic activities, disturb immune system, influence thyroid and neural function.[5,10] The studies confirm that BPA can pass through the placenta,[11–13] influence fetal growth in the uterus, result in adverse birth outcomes finally.[14] Pregnant People are susceptible to EDCs in gestational period and fetus is sensitive to environmental toxicants.[15] Thus, there are increasing concerns about the influence of BPA on birth outcomes. Many cohort studies have been done to investigate the association between BPA and birth outcomes, but these consequences are inconsistent.[16–22] The latest a published meta-analysis only provides evidence of the association between prenatal exposure to BPA and birth weight, and the results are not widely representative.[23] Hence, the aim of this meta-analysis is to provide summarized evidence on the association between prenatal exposure to BPA and birth outcomes based on current published cohort studies.

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2. Materials and methods

2.1. Search strategy

The PRISMA (preferred reporting items for systematic review and meta-analyses) protocol was prospectively conducted.[24] The PubMed, Cochrane databases, and Web of Science databases were searched systematically by 2 researchers respectively from their inceptions to Oct. 2018, using the keywords "bisphenol A", "birth weight", "birth length", "head circumference", "gestational age", "birth outcomes" without language restrictions. We also searched the reference lists of all acquired studies to avoid missing. The titles and abstracts were screened firstly. Then the remaining studies were reviewed by full text and identified based on the inclusion criteria. The disagreement between two researchers was solved by discussion. The study began in Oct. 2018. Ethical approval was not necessary, as this study was a meta-analysis based on published studies and did not need handle individual patient data.

2.2. Inclusion criteria

(1) A cohort study.
(2) The time of exposure to BPA for pregnant women was prenatal period.
(3) The exposure way of BPA for pregnant women was in daily life.
(4) The birth outcomes included birth weight, birth length, head circumference, or gestational age.

2.3. Data extraction

The following information was extracted through predesigned data extraction content by 2 researchers respectively from each included study: publication year, country, sample size, sample, time of sample collection, limits of detection (LOD), time period, eligible criteria of pregnant women, urinary BPA categorization, adjustment in the model, birth outcomes, results expressed as β coefficient (95%CI)or β coefficient (SD). The discrepancy was solved by discussion.

2.4. Assessment of quality

We used the Newcastle-Ottawa Scale (NOS) to assess the methodological quality of included studies.[25] The NOS included 3 categories (Selection, Comparability and Outcome) and 8 items. The NOS ranged from 0 to 9 stars: 4 stars for Selection, 2 stars for Comparability, 3 stars for Outcome. If the total stars was ≥ 6, we regarded the study as high quality, if the total stars was from 3 to 5, we regarded the study as middle quality; if the total stars was <3, we regarded the study as low quality,[26] and we excluded low quality study. The assessment was conducted by 2 researchers respectively, the disagreement was solved by discussion.

2.5. Statistical analysis

The association between prenatal exposure to BPA and birth outcomes was assessed by calculating pooled β coefficient and 95% confidence interval (CI). The heterogeneity of studies was assessed using Chi-squared test and quantified by calculating the I^2 statistic. When $I^2 > 50\%$ or P value $<.05$ was identified for heterogeneity among studies, we used the random effect model; Otherwise, a fixed effect model was adopted. We conducted subgroup analyses to evaluate the heterogeneity between studies based on country, sample size, LOD, BPA concentration. The sensitivity analysis was performed to assess whether the consequences were influenced by the single study. Finally, we evaluated the publication bias by cumulative forest plot. Meta-analysis was performed using Stata 12.0 version (Stata Corp., College Station, TX). $P<.05$ was considered statistically significant.

3. Results

3.1. Studies selection and characteristics

The detailed study selection progress was shown in Figure 1. Firstly, 209 studies were identified from PubMed, Web of Science, and Cochrane databases. An additional article was included by scanning the reference lists. Finally, seven studies with 3004 participants were selected into the meta-analysis.[17–19,21,27–29] The data of 2 studies [β (SD)] was acquired by formula transformation.[17,28]

Table 1 showed the main characteristics of seven studies. Three studies were from USA and Europe,[19,28,29] the remaining studies were from Asia[17,18,21,27]; 6 studies were urine sample,[17–19,21,27,29] 1 study was amniotic fluid sample[28]; 7 studies included birth weight,[17–19,21,27–29] 6 studies included birth length,[17–19,21,27,29] 4 studies included head circumference and gestational age.[18,19,27,29] Table 3 showed the result of quality assessment of included studies. Five studies were high quality,[17–19,27,29] 2 studies were middle quality,[21,28]

3.2. Main outcomes

3.2.1. Birth weight. The pooled results of 7 studies showed in Figure 2. Heterogeneity was not observed across studies (I^2 = 31.8%, P = .137), so fixed effect model was used. There was positively significant association of BPA with birth weight (β = 21.92, 95%CI: 1.50–42.35, P = .04).

3.2.2. Birth length. The pooled results of 6 studies showed in Figure 3. Heterogeneity was not observed across studies (I^2 = 0.0%, P = .996), so fixed effect model was used. There was no significant association of BPA with birth length (β = 0.12, 95% CI: −0.01–0.25, P = .07).

3.2.3. Head circumference. Heterogeneity was not observed across studies (I^2 = 33.0%, P = .188), so fixed effect model was used. There was no significant association of BPA with head circumference (β = –0.03, 95%CI: –0.14–0.08, P = .60).

3.2.4. Gestational age. Heterogeneity was observed across studies (I^2 = 55.4%, P = .062), so random effect model was used. There was no significant association of prenatal exposure to BPA with gestational age (β = –0.07, 95%CI: –0.19–0.06, P = .31).

3.3. Subgroup analysis and sensitivity analysis

The subgroup analysis was conducted based on country, sample size, LOD (Table 2), there was no significant association was found ($P>.05$). When BPA concentration was ≤ 0.76 μg/L and 0.76–1.3 μg/L, there were positive correlation between BPA and birth weight (β = 70.72, 95%CI: 16.42–125.02; β = 39.63, 95% CI: 7.36–71.91, respectively) (Fig. 4).

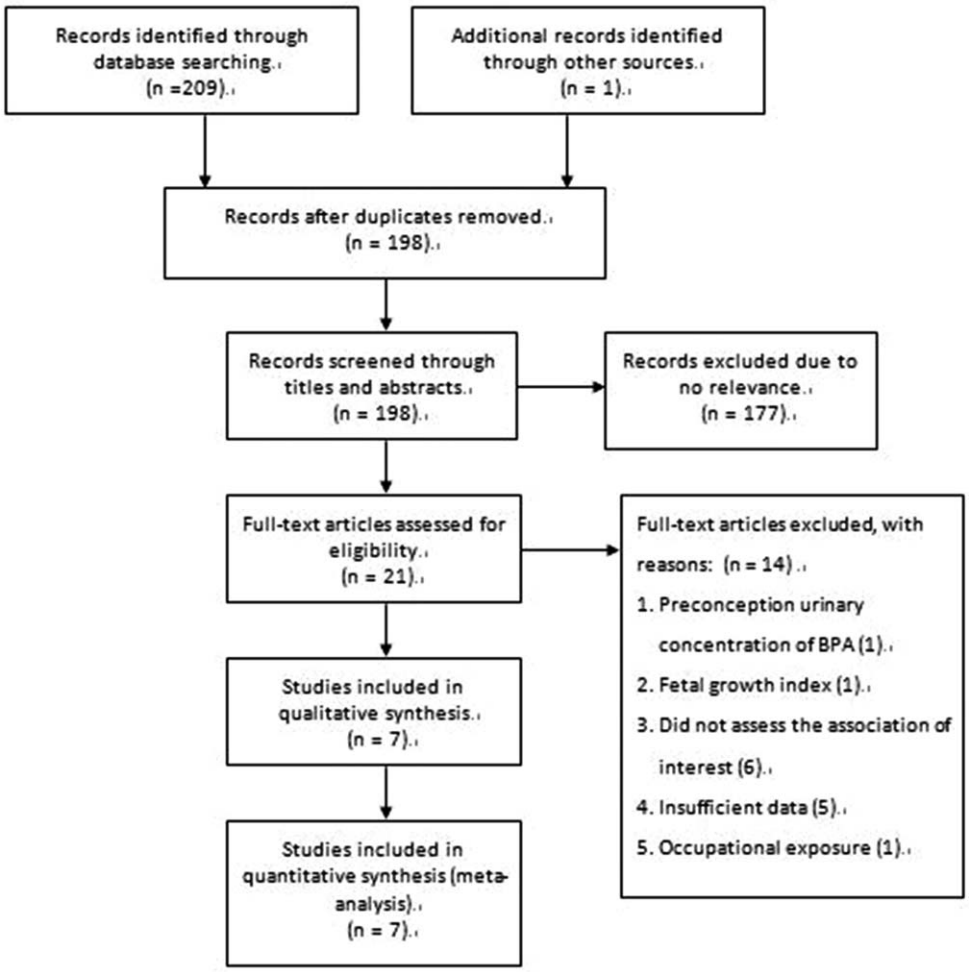

Figure 1. Flow diagram of studies selection. CI = confidence interval, SD = standard interval.

We performed sensitivity analysis by excluding the each individual study, these research results did not change evidently.

3.4. Publication bias

The publication bias was evaluated by accumulative forest plot, we did not observe publication bias.

4. Discussion

This meta-analysis indicated that BPA was positively associated with birth weight, however, not associated with birth length, head circumference and gestational age. The sensitivity analysis showed that the results were consistent after excluding small sample study. The publication bias was not found in the study. The results were almost accordant in the subgroup of country, sample size, publication year and LOD.

This result was not consistent with the latest published meta-analysis,[23] which indicated that prenatal exposure to BPA was not associated with birth weight. That may be because the inclusion criteria of studies and analysis methods were different between the 2 studies. The published meta-analysis included preconception exposure and prenatal exposure, and included case-control studies. Our study only included prenatal exposure, and all of the included studies were cohort studies. In addition, our study included every concentration group of BPA, but the published meta-analysis only included the third trimester or the high concentration group, which can make the result present bias. A European meta-analysis also demonstrated that occupational exposure to BPA was not associated with birth weight.[30] But in this European meta-analysis, the BPA exposure way and countries from which the participants come were different from our study, which can make inconsistent results.

The result suggested that there was positive correlation between prenatal exposure to BPA and birth weight. The animal study also indicated that BPA exposure group had higher birth weight compared to the unexposed group.[31] In the current mechanism researches, BPA may cause adverse health effects by acting on nuclear receptors (NRs). The study showed that BPA can promote Adipogenesis by stimulating the activity of glucocorticoid receptor (GR) in 3T3-L1 preadipocytes.[32] Also, BPA can increase adipocyte number by blinding to estrogen receptor (ER).[33] The subgroup analysis showed that this correlation was more pronounced at relative low concentration of exposure. The animal experiments also showed that BPA can affect birth weight at low concentration,[34,35] but the relevant mechanisms can still need to be further explored. Currently, there were less epidemiological studies to explore the association

Table 1

Characteristics of included studies.

Studies	Country	publication year	Total participants	Sample	Time of sample collection	Limits of detection (μg/L)	Time period	Eligible criteria of pregnant women	Urinary BPA categorization (μg/L)	Adjustment in the model	Birth outcomes
Wolff et al[29]	USA	2008	404	Urine	Third trimester of gestation	0.36	1998–2002	Primiparas, singleton pregnancy, no medical complications, no change of hospital or residence outside New York City, can collect biological specimens	Creatinine-corrected BPA as continuous variable, GM: 1.3	Creatinine, race, infant sex, gestational age at delivery (except in models predicting gestational age), maternal pre-pregnancy BMI, education, marital status, smoking during pregnancy	Birth weight, birth length, head circumference, gestational age
Tang et al[21]	China	2013	567	Urine	Delivery	0.36	2010-2012	Age>18 years, singleton pregnancy, no assisted reproduction and medical complications or preexisting diabetes, hypertension, HIV infection	Creatinine-corrected BPA: low, middle, high, GM: 0.91	Urinary creatinine (CR), parihty, gestational age, maternal age, BMI in late pregnancy	Birth weight, body length, length of gestation
Casas et al[19]	Spain	2016	488	Urine	First and third trimester	0.1	2004-2006	Age >16 years, singleton pregnancy, intention to deliver in reference hospital unassisted conception, no communication problems	Creatinine-corrected BPA as continuous variable, GM: 2.3	Maternal education, parity, smoking during pregnancy, birth season, type of delivery, urinary cotinine	Birth weight, birth length, head circumference, gestational age, placental weight
Huang et al[18]	Taiwan	2017	162	Urine	11 and 26 weeks of gestation delivery	0.16	<13weeks pregnant until delivery	age of 18–45 years, <13weeks pregnant with detection of the fetal heart beat at the first prenatal visit, and planning to deliver at CGH	Creatinine-corrected BPA: GM: 1st:0.17, 2nd: 0.37, 3rd:0.34	Maternal age, pre-pregnancy BMI, gestational age, weight gain, infant sex, parity, adverse pregnancy outcomes	Birth weight, birth length, head circumference, chest circumference
Ding et al[27]	China	2017	496	Urine	Delivery	0.1	2010–2013	Age >18 years, singleton pregnancy, residence in the area for at least 3 years, spontaneous conception, no history of diabetes mellitus or gestational diabetes, chronic or pregnancy-associated hypertension, thyroid disorders, HIV infection or AIDs, illicit drug use	Creatinine-corrected BPA as continuous variable, GM: 1.07	Maternal age, pre-pregnancy BMI, gestational weight gain during pregnancy, passive smoking, gestational age, household monthly income, infant gender, parity	Birth weight, birth length, head circumference, gestational age, ponderal index
Lee et al[17]	Korea	2014	757	Urine	Third trimester	0.12–0.28	Period of pregnancy (less than 20 weeks of gestation) until delivery	Period of pregnancy <20 weeks, singleton pregnancy, no congenital anomalies and stillbirths	Creatinine-corrected BPA: 1st, 2nd, 3rd tertiles, GM: 1.87	Gestational age, education, pre-pregnant MBI, infant gender, parity	Birth weight, birth length
Pinney et al[28]	USA	2017	130	Amniotic fluid	Second trimester	0.25	2004-2006	Singleton pregnancy, without any reported maternal health conditions, pregnancy complications, fetal anomalies or exposure to maternal smoking or illicit drugs	Group1:≤0.25, group2: 0.25–0.40, group3: 0.40–2.0, group4: >2.0	Sex of offspring, gestational age, at amniocentesis, parity, gravidity	Birth weight

BPA = bisphenol A, GM = geometric mean.

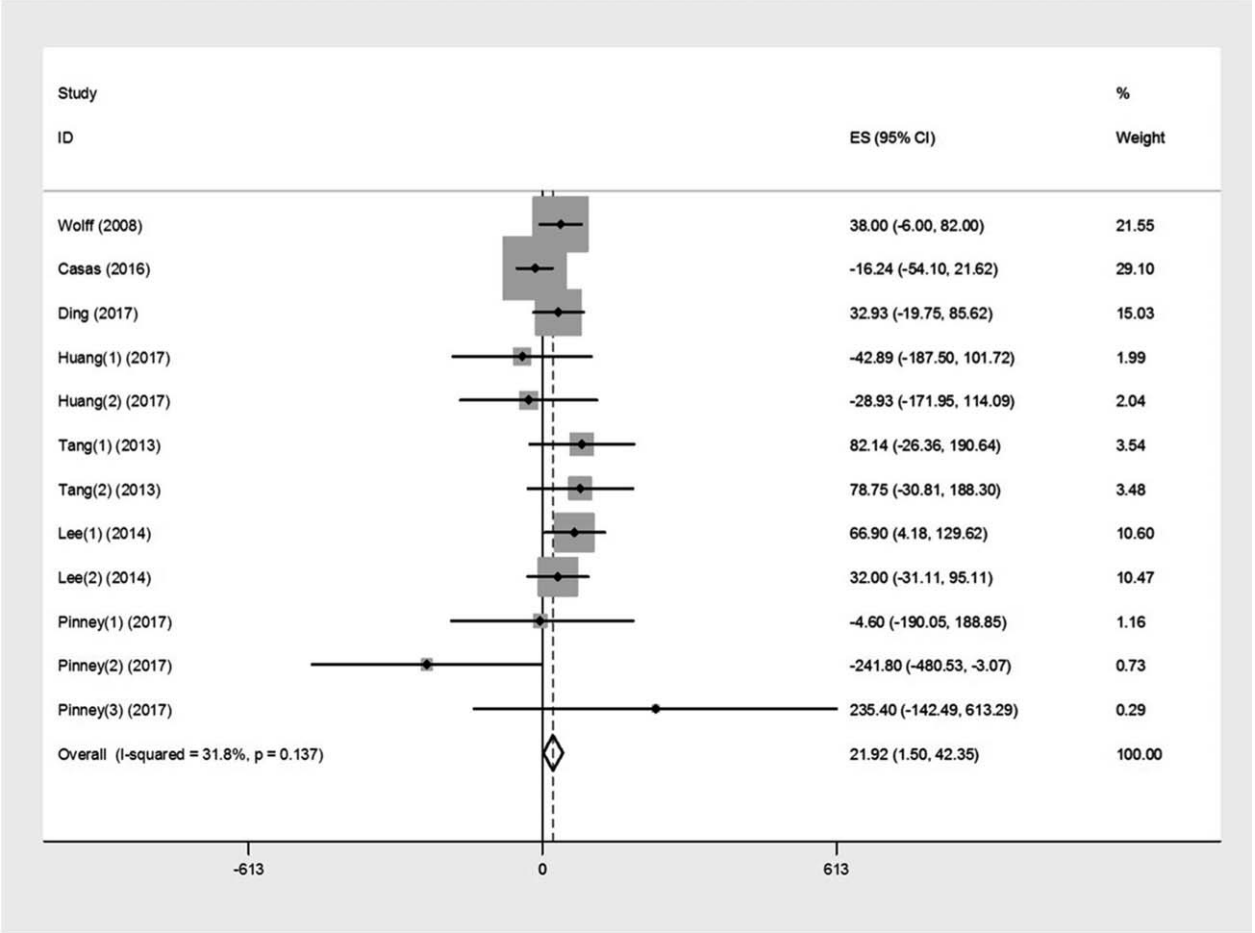

Figure 2. Forest plot of the association between prenatal exposure to BPA and birth weight.

Table 2

Subgroup analysis of the relation of prenatal BPA exposure with birth weight, birth length, head circumference, and gestational age.

Variable	Categories	Study size	β (95%CI)	*P* value
Birth weight				
Country	Europe and America	3	4.40 (−23.70–32.50)	.08
	Asia	4	41.55 (11.81–71.30)	
Sample size	>=450	4	22.59 (−1.44–46.62)	.92
	<450	3	20.19 (−18.57–58.95)	
LOD (μg/L)	>=0.36	2	48.43 (10.22–86.65)	.11
	<0.36	5	11.32 (−12.84–35.49)	
Birth length				
Country	Europe and America	2	0.10 (−0.15–0.35)	.83
	Asia	4	0.13 (−0.02–0.29)	
Sample size	>=450	4	0.15 (−0.02–0.32)	.61
	<450	2	0.08 (−0.13–0.29)	
LOD (μg/L)	>=0.36	2	0.10 (−0.11–0.31)	.78
	<0.36	4	0.14 (−0.03–0.31)	
Head circumference				
Country	Europe and America	2	0.09 (−0.09–0.27)	.09
	Asia	2	−0.12 (−0.26–0.03)	
Sample size	>=450	2	−0.07 (−0.27–0.12)	.56
	<450	2	0.00 (−0.16–0.16)	
LOD (μg/L)	>=0.36	1	0.08 (−0.11–0.27)	.13
	<0.36	3	−0.11 (−0.27–0.05)	
gestational age				
Country	Europe and America	2	0.05 (−0.13–0.23)	.09
	Asia	2	−0.17 (−0.34–0.01)	
Sample size	>=450	3	−0.15 (−0.32–0.03)	.17
	<450	1	0.03 (−0.16–0.22)	
LOD (μg/L)	>=0.36	2	−0.08 (−0.24–0.08)	.70
	<0.36	2	−0.03 (−0.24–0.18)	

CI = confidence interval, LOD = limits of detection.

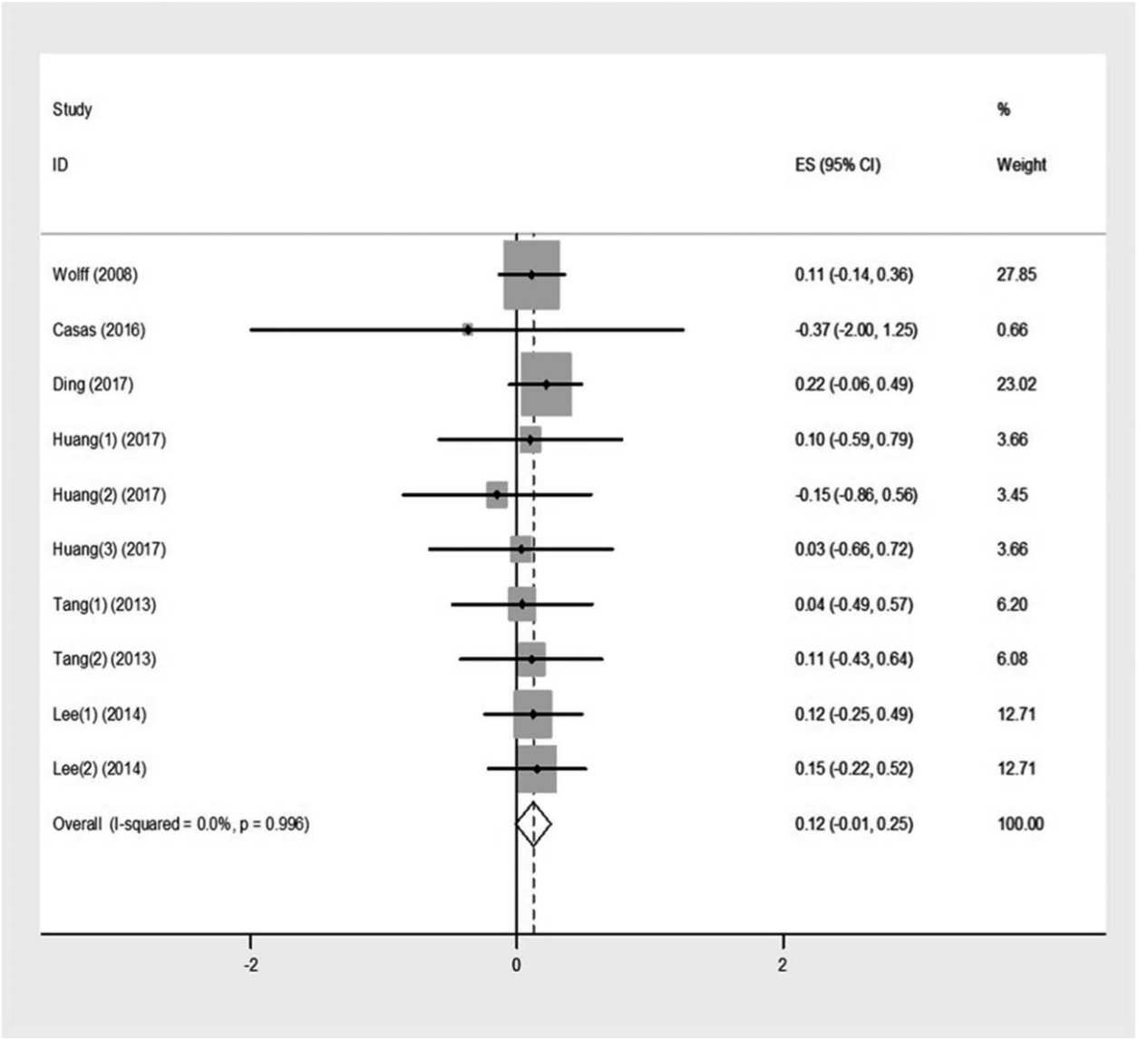

Figure 3. Forest plot of the association between prenatal exposure to BPA and birth length.

Table 3

Assessment of methodological quality of included individual studies.

	Selection				Comparability	Outcome				
Study	Representativeness of the exposed cohort	Selection of the non-exposed cohort	Ascertainment of exposure	Demonstration that outcome of interest Was not present at start of study	Comparability of cohorts on the basis of the design or analysis	Assessment of outcome	Was follow-up long enough for outcomes to occur	Adequacy of follow up of cohorts	Total smile face	Quality level
Wolff et al[29]	☺	☹	☺	☺	☹	☺	☺	☺	6	High
Tang et al[21]	☺	☹	☺	☺	☹	☺	☺	☹	5	Middle
Casas et al[19]	☺	☹	☺	☺	☹	☺	☺	☺	6	High
Huang et al[18]	☺	☹	☺	☺	☹	☺	☺	☺	6	High
Ding et al[27]	☺	☹	☺	☺	☹	☺	☺	☺	6	High
Lee et al[17]	☺	☹	☺	☺	☹	☺	☺	☺	6	High
Pinney et al[28]	☺	☹	☺	☺	☹	☺	☺	☹	5	Middle

☺: Yes ☹: No.

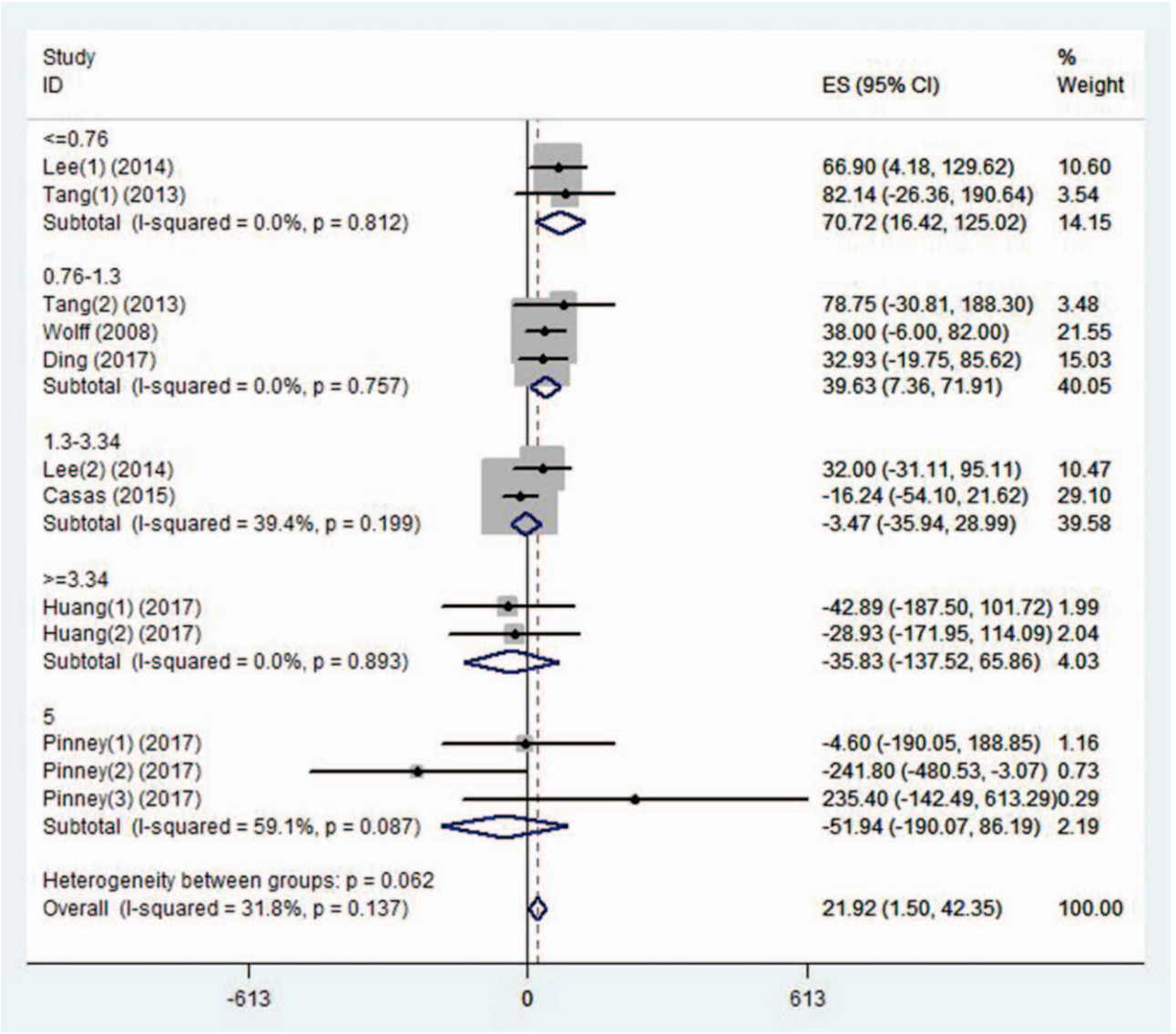

Figure 4. Forest plot of the association between prenatal BPA exposure levels and birth weight.

between the correlation and BPA concentration. Therefore, more prospective studies should be done to investigate the impact of BPA concentration on birth outcomes.

Gender may be a source of heterogeneity, but subgroup analysis was not performed due to data limitation. Relevant studies revealed that there were gender differences on the association between prenatal exposure to BPA and birth outcomes.[17,20,21,27,36,37] Animal experiments also observed gender-specific association.[38,39] Thus, further studies are needed to investigate the association in gender. Gestational period can cause heterogeneity; the subgroup analysis was also not performed due to limited data. The study suggested that late pregnancy can be a sensitive period for exposing to BPA.[40] More researches were needed to explore a sensitive period of BPA exposure in pregnant women.

This study had strict inclusion criteria and exclusion criteria, so the results were reliable. And this study provided summarized evidence about the association between prenatal exposure to BPA and more birth outcomes. But it still had some limitations. First, the sample was not uniform and sample could not represent the authentic exposure level of pregnant women. Second, the definition for study quality cannot be relatively strict. Third, we were unable to analyze the dose-response relationship due to differences in the data description of included studies.

In summary, this meta-analysis reveals that BPA is positively associated with birth weight, but not associated with birth length, head circumference and gestational age. Therefore, further studies are needed to investigate the critical sensitive period of influencing fetal development and to investigate the difference on gender.

Author contributions

Conceptualization: Zhitong Zhou, Xiaofeng Li, Xin Chen.
Data curation: Zhitong Zhou, Yuyang Lei, Wei Wei, Yuxin Zhao, Yizhou Jiang, Ningning Wang.
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Funding acquisition: Xin Chen.
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Software: Zhitong Zhou, Yuyang Lei, Wei Wei, Yuxin Zhao, Yizhou Jiang, Ningning Wang.
Supervision: Xiaofeng Li, Xin Chen.
Writing – original draft: Zhitong Zhou, Yuyang Lei, Xin Chen.

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