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REVIEW ARTICLE

Maternal prepregnancy BMI or weight and offspring's blood pressure: Systematic review

Helena Ludwig-Walz [ID] | Milan Schmidt | Anke L. B. Günther | Anja Kroke

Department of Nutritional, Food and Consumer Sciences, Fulda University of Applied Sciences, Fulda, Germany

Correspondence
Anja Kroke, Department of Nutritional, Food and Consumer Sciences, Fulda University of Applied Sciences, eipziger Str. 12336037 Fulda, Germany.
Email: anja.kroke@oe.hs-fulda.de

Abstract

Emerging evidence suggests that maternal prepregnancy body mass index or weight (MPBW) may be associated with offspring's blood pressure (BP). Therefore, we conducted a systematic review—following the Preferred Reporting Items for Systematic Reviews and Meta-analyses statement—to assess and judge the evidence for an association between MPBW with offspring's later BP. Five data bases were searched without limits. Risk of bias was assessed using the "Tool to Assess Risk of Bias in Cohort Studies," and an evidence grade was allocated following the World Cancer Research Fund criteria. Of 2,011 publications retrieved, 16 studies (all cohort studies) were included in the systematic review; thereof, 5 studies (31%) were rated as good-quality studies. Overall, data from 63,959 participants were enclosed. Systolic BP was analysed in 15 (5 good quality), diastolic BP in 12 (3 good quality), and mean arterial pressure in 3 (no good quality) studies. Five good-quality studies of MPBW with offspring's systolic BP as the outcome and 1 good-quality study with offspring's diastolic BP as the outcome observed a significant association. However, after adding offspring's anthropometry variables to the statistical model, the effect attenuated in 4 studies with systolic BP to nonsignificance, the study with diastolic BP remained significant. No good-quality studies were found with respect to offspring's later mean arterial pressure. In conclusion, this systematic review found suggestive, but still limited, evidence for an association between MPBW with offspring's later BP. The available data suggest that the effect might be mainly mediated via offspring's anthropometry.

KEYWORDS

blood pressure, Developmental Origins of Health and Disease, offspring, perinatal programming, prepregnancy BMI, prepregnancy weight

1 | INTRODUCTION

Accumulating evidence suggests that long-term health is influenced by determinants acting in the time window between conception and approximately the end of the second year of life (Hanson & Gluckman, 2015; Koletzko, 2015; Plagemann, 2011). These first "1,000 days" are characterized by developmental plasticity of the young organism, allowing perinatal programming effects exerted by nutritional, metabolic, and/or hormonal environmental influences that determine metabolic and functional processes (Hanson & Gluckman, 2015; Plagemann, 2011). The development of obesity (Oddy et al., 2014; Yan, Liu, Zhu, Huang, & Wang, 2014), type 2 diabetes (Mitanchez et al., 2015; Pereira, Alfenas, & Araújo, 2014), high blood pressure (BP; Mu et al., 2012; Pacce et al., 2016; Taal et al., 2013; Zhang et al., 2013), adverse

lipid profile (Wijnands, Obermann-Borst, & Steegers-Theunissen, 2015), and cardiovascular disease (CVD; Barker et al., 1993; Drake & Reynolds, 2010; Mitanchez et al., 2015) have already been described to be influenced by these early life factors.

Accordingly, increasing evidence suggests maternal peri-pregnancy body mass index (BMI) or weight—meaning the maternal BMI or weight just before or in early pregnancy—as an important determinant in the programming of offspring's metabolic profile (Drake & Reynolds, 2010; O'Reilly & Reynolds, 2013). Especially maternal (prepregnancy) obesity is discussed as having an important influence on the fetus, eventually leading to programming of an adverse metabolic profile, and, consequently, the predisposition to metabolic disorders and CVD as well as other diseases in offspring's later life (Mesman et al., 2009; O'Reilly & Reynolds, 2013; Pacce et al., 2016; Yu et al.,

2013). Although the possible underlying mechanisms are not completely understood, there is evidence from animal and human studies that maternal peri-pregnancy BMI or weight may independently influence the offspring's BP later in life (Nuyt, 2008; Symonds, Sebert, Hyatt, & Budge, 2009; Thornburg, 2015).

Due to their major public health importance, CVD and their putative developmental origins are of particular interest (World Health Organization [WHO], 2014). Elevated BP—as a leading as well as modifiable risk factor for cardiometabolic impairments (Lim et al., 2012; Rapsomaniki et al., 2014), which tracks from early life periods into adulthood (Regnault et al., 2014)—is responsible for a considerable global disease burden (WHO, 2014). Thus, the evaluation of early life factors contributing to elevated BP is considered as highly relevant.

However, for evidence-based health promotion and preventive activities, the summary and evaluation of available evidence is a fundamental prerequisite (Knorpp & Kroke, 2012). Therefore, we conducted a systematic review to assess and judge the evidence for an association between maternal peri-pregnancy BMI or weight with offspring's BP later in life. Because both direct effects of maternal prepregnancy BMI or weight (MPBW) on offspring's BP (Ojala et al., 2009; Samuelsson, 2014; Thornburg, 2015) and indirect effects have been hypothesized, for example, via offspring's anthropometric characteristics mediated effects (Gademan et al., 2013; Gaillard et al., 2014; Gaillard et al., 2016), both underlying mechanisms were considered.

2 | MATERIALS AND METHODS

The systematic review was conducted and presented according to the Preferred Reporting Items for Systematic Reviews and Meta-analyses statement (Moher, Liberati, Tetzlaff, & Altman, 2009) and was registered in the international prospective register of systematic reviews PROSPERO (CRD42015026639; Booth et al., 2012). The Preferred Reporting Items for Systematic Reviews and Meta-analyses checklist with the reported page numbers is provided in **Appendix S1**.

2.1 | Search strategy and information sources

A two-step systematic literature search was performed in the databases MEDLINE, EMBASE (PubMed/EMBASE), Cochrane Library, CINAHL, and Web of Science. The first systematic literature search included search terms regarding the association of MPBW and offspring's BP, and the second included search terms regarding the association of maternal BMI or weight in the first trimester and offspring's BP. The five databases were searched until December 4, 2016. A summarized overview of the search terms used is presented in Table 1. The full search strategies for all five databases are provided in **Appendix S2**.

All search terms were searched both as controlled vocabulary terms (Medical Subject Headings or Emtree) and as free words in title and abstract. No limits were set regarding language, age, year of publication, or study type. If several publications from the same population or cohort and approximately the same offspring's age were found, only data from the most relevant report were included (e.g., exclusion of congress papers or descriptions of ongoing studies, if the full study paper is also available). In addition, reference lists of the articles

Key messages

- Evidence suggests that long-term health is influenced by determinants acting in the first "1,000 days" (e.g., maternal prepregnancy body mass index or weight [MPBW]).

- Blood pressure is a leading risk factor for cardiometabolic impairments.

- This systematic review shows suggestive, but still limited, evidence for an association of MPBW with offspring's later blood pressure. However, offspring's anthropometric characteristics entirely explained the observed associations.

- MPBW could be an important, albeit indirect, determinant with respect to offspring's metabolic pathology.

- Regarding the still rising rates of obesity, further efforts should be made to clarify the role of MPBW on offspring's later health.

included were checked for further relevant studies. Research progress was monitored in the PROSPERO database as well as current conferences relating to the search question.

2.2 | Study eligibility criteria

The eligibility criteria were defined following the population–intervention–comparison–outcome scheme (Higgins & Green, 2011). To accommodate the fact that we did not expect to retrieve intervention studies but only observational studies, we replaced the category "intervention" (I) in this scheme with "exposure" (E):

1. Population (P): Studies that included the general population; exclusion of studies of ill or institutionalized participants, participants on antihypertensive medication, participants from low- and middle-income countries (classification according to the World Bank Group, 2016) and pregnancy impairments (e.g., low birthweight, intrauterine growth restriction, and maternal prenatal hypertensive disorders).

2. Exposure (E): Studies with measured or self-reported maternal peri-pregnancy BMI or weight.

3. Comparison (C): Not applicable.

4. Outcome (O): Studies with measurement of offspring's systolic BP (SBP), diastolic BP (DBP) or mean arterial pressure (MAP).

2.3 | Study selection

As suggested by the *Cochrane Handbook for Systematic Reviews* (Higgins & Green, 2011) two reviewers (H. L.-W. and M. S.) independently searched the databases to identify potentially relevant studies. In a first step, titles and abstracts were screened and irrelevant studies

TABLE 1 Summarized overview of the search terms used regarding both systematic searches

Population/exposure regarding the association between <u>maternal prepregnancy BMI or weight</u> and blood pressure in offspring's later life
(prepregnan* OR pre-pregnan* OR preconception* OR pre-conception* OR periconception* OR peri-conception* OR pregestation* OR pre-gestation* OR pregravid* OR pre-gravid* OR "before pregnancy") **AND** ("body mass index" OR "body weight" OR weight OR overweight OR obesity OR adiposity)
Population/exposure regarding the association between <u>maternal BMI or weight in the first trimester</u> and blood pressure in offspring's later life
(conception* OR antenatal* OR "early pregnancy" OR post-conception* OR postconception* OR "first trimester") **AND** ("body mass index" OR "body weight" OR weight OR overweight OR obesity OR adiposity)
Outcome (used for both searches)
(hypertension OR "cardiovascular risk" OR "cardiovascular profile" OR "cardiometabolic profile" OR "blood pressure" OR bloodpressure OR "blood tension" OR normotension OR "normo tension" OR "vascular pressure" OR "intravascular pressure")
Limits (used for both searches)
Humans

Note. BMI = body mass index;

= truncation.

were excluded. In a second step, the full text of the remaining articles was obtained and assessed for eligibility according to the study's inclusion criteria. Any discrepancies between the two reviewers were discussed extensively and, if necessary, resolved by a third author (A. L. B. G. or A. K.). The reasons for exclusion in the second step are reported in **Appendix S3**.

2.4 | Data extraction

Data extraction was conducted independently by two authors (H. L.-W. and M. S.) using specially developed data collection forms (Higgins & Green, 2011). These forms were pilot tested with a broad sample of the studies to be enclosed. Information was collected on study framework, characteristics of the study population, details on exposure and outcome assessment, and statistical analysis. Any discrepancies between the two reviewers were discussed extensively and, if necessary, resolved by a third author (A. L. B. G. or A. K.).

2.5 | Risk of bias assessment

Risk of bias within the selected studies was assessed using the "Tool to Assess Risk of Bias in Cohort Studies" (Busse & Guyatt, 2008). This tool is framed as questions and comprises eight categories with a four-category scale from low to high risk of bias. The selected publications were separately assessed by two independent reviewers (H. L.-W. and A. K.), and disagreement was resolved by discussion with involvement of a third author (A. L. B. G.) where necessary.

For the evaluation of an adequate adjustment and the assessment of an independent or mediated association, two sets of potential confounders, covariates, and mediators were defined. An intensive literature search was conducted to designate the most relevant variables to be included into the statistical models. A Variable Set 1 was defined, including three maternal variables (=confounders: maternal age at enrolment in pregnancy or at delivery, smoking during pregnancy, and maternal socio-economic status) and two offspring variables (=covariates: offspring's sex and age). Offspring's and mother's age were included because of the age dependency of BP (Wills et al., 2011; Wojciechowska et al., 2012). In addition, several studies pointed out that smoking during pregnancy affects offspring's later BP (Taal et al., 2013; Yang, Decker, & Kramer, 2013) as well as socio-economic status

(e.g., income, education, and occupation) as an important predictor for high BP (WHO, 2013). Thus, a low social status may influence a child's BP and accompanying later cardiovascular impairments (Brummett et al., 2011; Kivimäki et al., 2006). The full adjustment for Variable Set 1 defined a good-quality study in this systematic review. Furthermore, a Variable Set 2 was defined because some but not all studies further included characteristics of offspring's anthropometry at outcome assessment in their regression models. This Variable Set 2 thus included the potential mediators' birthweight and offspring's anthropometric characteristics (e.g., mostly BMI) at outcome assessment. Various studies described a strong relation between birthweight and offspring's later BP (Mu et al., 2012; Zhang et al., 2013) as well as offspring's weight status and later BP (American Heart Association, 2014; Chen & Wang, 2008). The included studies were checked for adjustment for Variable Set 1 and, if adjustment was complete, additionally checked for Variable Set 2. The rating criteria and the risk of bias assessment are reported in **Appendix S4** and in **Appendix S5**, respectively.

2.6 | Assessment of blood pressure

To estimate the quality of outcome measurement, the criteria of the fourth report on the diagnosis, evaluation, and treatment of high BP in children and adolescents (Falkner, 2005); the European Society of Hypertension; and the European Society of Cardiology (Mancia et al., 2013) as well as the recommendations of the European Society of Hypertension for BP measurement in children and adolescent (Lurbe et al., 2009) were considered. A high rating was selected, when BP measurement was carried out according to the recommended auscultatory method or a validated oscillometric method and when repeated measures of BP were taken in a rested position. A detailed description of the criteria is given in **Appendix S6**.

2.7 | Evidence assessment

The final evidence assessment was conducted in accordance with the criteria for grading evidence by two reviewers (H. L.-W. and A. K.), as described in the second report of the World Cancer Research Fund (WCRF, 2007). The classification tool is provided in **Appendix S7**.

3 | RESULTS

3.1 | Study selection

In the first systematic literature search regarding the association of MPBW with offspring's later BP, 2,011 publications were identified. After screening and exclusions, 16 publications met the inclusion criteria. Figure 1 illustrates the selection process. Reasons for exclusion of the full-text screened studies are described in **Appendix S3**.

In the second systematic literature search that focused on maternal BMI or weight in the first trimester of pregnancy as the exposure variable, none of the 2,196 studies retrieved could be included. Therefore, all the following results exclusively refer to the first search.
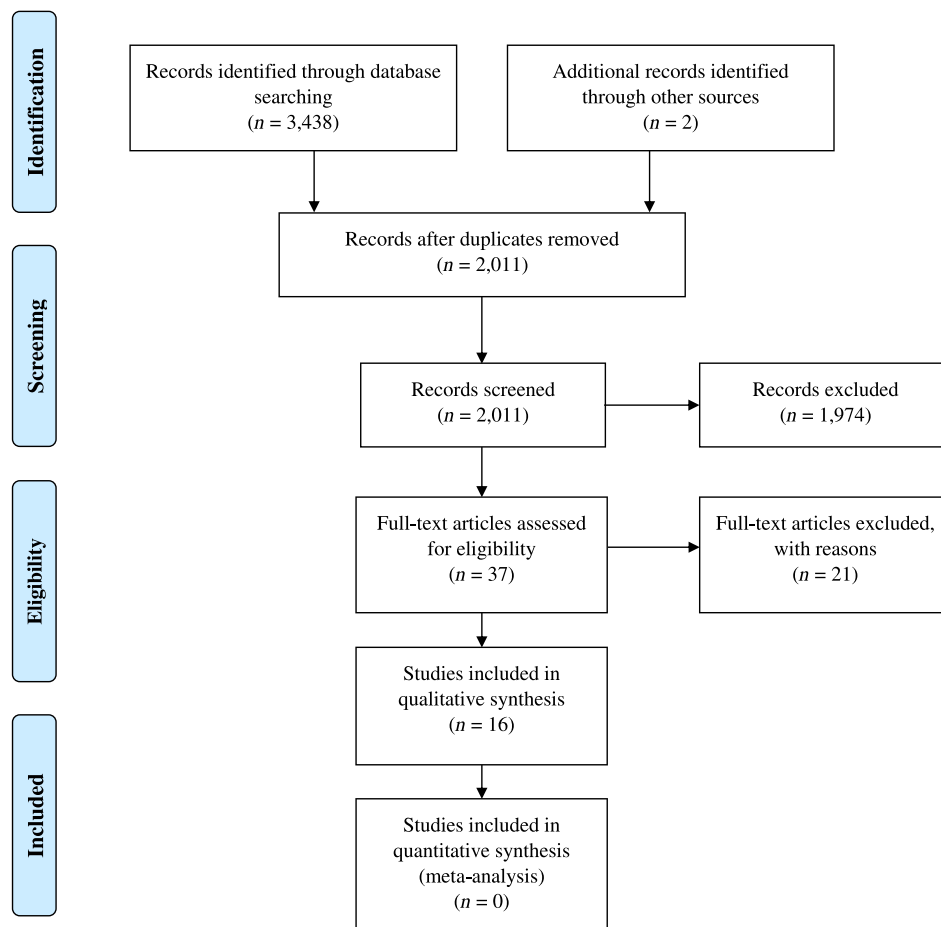
3.2 | Study characteristics

Table 2 presents the summarized study characteristics of the 16 studies included. In total, data from 63,959 participants were included. Of the total population, 43% was contributed by one study (Wen, Triche, Hogan, Shenassa, & Buka, 2011). The offspring's age at outcome assessment ranged from 0 years (newborns) to 32 years. From the "Jerusalem Perinatal Study," two publications were included, as offspring's age was 17 years (Laor, Stevenson, Shemer, Gale, & Seidman, 1997) in the first and 32 years in the second publication (Hochner et al., 2012). Most studies were conducted in Europe and the United States. Maternal BMI was used either as continuous or as class variable with varying classifications; the BMI classification was defined in five studies (Daraki et al., 2015; Eisenman, Sarzynski, Tucker, & Heelan, 2010; Gademan et al., 2013; Gaillard et al., 2014; Perng, Gillman, Mantzoros, & Oken, 2014) according to the WHO (2016) and in one study (Fraser et al., 2010) according to the Institute of Medicine (1990) guidelines.

3.3 | Risk of bias

Risk of bias of the 16 included studies was assessed with the "Tool to Assess Risk of Bias in Cohort Studies" (**Appendix S5**). The third ("Can we be confident that the outcome of interest was not present at start of study?") and eighth ("Were co-interventions similar between groups?") categories were not applicable to the included studies. In summary, in four of six categories, the risk of bias was low (1. participant selection; 2. exposure assessment; 5. covariate assessment; and 6. outcome assessment). However, the fifth category was chosen as the leading one for the classification as a good-quality study:

BMI, body mass index; BP, blood pressure;

ª according to Moher et al., 2009

FIGURE 1 Flow diagram for the study selection regarding the association of maternal prepregnancy BMI or weight with the later offspring's BP

TABLE 2 Study characteristics of the 16 studies included

First author; Year; Country	Study setting; population	Sample size (% female offspring)	Maternal characteristics: Age at enrolment in pregnancy or at delivery (years)	Measurement prepregnancy BMI or weight	Offspring's characteristics: Age at outcome measurement (years)	Outcome measurement's characteristics: Method (firm name), validation for children or adolescents	Technique of outcome measurement
Daraki; 2015; Greece	Population-based cohort study; recruitment of pregnant women (Greek and immigrants) at the time of the first comprehensive ultrasound examination	618 mother–child pairs (48), sample size for BP analyses: 488	AD: mean ± SD; no excess weight: 29.87 ± 0.2; overweight or obese: 29.98 ± 0.3	Self-reported prepregnancy BMI	Mean ± SD: 4.2 ± 0.2	Oscillometric method (Dinamap ProCare 400), NR	Seated position, 5 min rest, 1-min intervals, average of 5 consecutive measurements, child's right arm, and cuff of appropriate size for arm circumference.
Derraki; 2015; New Zealand	Retrospective analyses of a cohort; participants were identified from the obstetrics database at the National Women's Health, Auckland City Hospital (Auckland, New Zealand)	54 mothers, 70 offspring (39)	AD: mean ± SD: 33.0 ± 4.9 AD: range: 17.9–42.0	Self-reported prepregnancy BMI	Mean ± SD: 8.9 ± 1.9 Range: 4–11	Oscillometric method (Spacelab 90217), validated (Redwine, James, O'Riordan, Sullivan, & Blumer, 2015)	Measurements were performed every 20 min from 7:00 to 22:00 and every 30 min from 22:00 to 07:00 (24-hr ambulatory BP) on the nondominant arm, Spacelabs monitor fitted on the nondominant arm.
Eisenman; 2010; USA	Secondary data analysis of a longitudinal project; population was recruited in a rural U.S. Midwestern community through written and/or verbal advertisements	144 mother–child pairs (48.6)	NR: mean ± SD: 30 ± 4	Self-reported prepregnancy BMI	Mean ± SD: 7.3 ± 2.0 Range: 2.9–11.9	Auscultation method (NR), recommended method (Lurbe et al., 2009)	3 measurements, seated for 10 min, 1-min intervals, mean of 3 values; MAP was calculated as (SBP − DBP/3) + DBP.
Filler; 2011; UK	Prospective cohort study, sample of patients attending Children's Hospital, London Health Science Centre	3,024 offspring (45.4)	NR	Self-reported prepregnancy BMI	Range: 2.05–18.58	Oscillometric method (Welch Allyn Spot Vital Signs LXi or Dinamap Pro 100, Pro 300, and Dinamap XL Vital Signs Monitor), NR	Seated, calm, second of two measurements performed 5 min apart.
Fraser; 2010; UK	Prospective population-based cohort study; Avon Longitudinal Study of Parents and Children	5,154 mother–child pairs (50.5)	AD: mean ± SD: 29.2 ± 4.5	Self-reported prepregnancy weight	~9	Oscillometric method (Dinamap 9301 Vital Signs Monitor), NR	2 readings of SBP and DBP, child rested and seated, mean value was used, and arm supported at chest level on a table.
Gademan; 2013; Netherlands	Community-based birth cohort; Amsterdam Born Children and their Development study	3,074 mother–child pairs (49.9)	NR: mean ± SD: 31.8 ± 4.6	Self-reported prepregnancy BMI	~5–6	Oscillometric method (Omrom 705 IT), validated (Stergiou, Yiannes, & Rarra, 2006)	2 measurements, 1 min of rest, SBP and DBP were calculated by taking the mean values of the repeated measurements, validated cuff size for children.

(Continues)

TABLE 2 (Continued)

First author; Year; Country	Maternal characteristics: Study setting; population	Sample size (% female offspring)	Age at enrolment in pregnancy or at delivery (years)	Measurement prepregnancy BMI or weight	Offspring's characteristics: Age at outcome measurement (years)	Outcome measurement's characteristics: Method (firm name), validation for children or adolescents	Technique of outcome measurement
Gaillard; 2014; Netherlands	Population-based prospective cohort study; Generation R Study	4,871 children and their parents (49.8)	AP: median (95% range); 30.9 (19.9–39.4)	Self-reported prepregnancy BMI	Median (95% range); 6.0 (5.6–8.0)	Oscillometric method (Datascope Accutor Plus), validated (Wong, Sung, Yn, & Leung, 2006)	4 measurements, 1-min intervals, mean measure of the last 3 BP measurements, measured at the right brachial artery.
Gaillard; 2016; Australia	Population-based prospective cohort study; Western Australian Pregnancy (Raine) Cohort	1,392 mother–children pairs (49.3)	AD: mean ± SD: 29.0 ± 5.8	Self-reported prepregnancy BMI	Median (95% range); 17.0 (16.7; 17.7)	Oscillometric method (Dinamap 8100), NR	5 measurements (every 2 min for 10 min), subjects rested supine for 5 min, average values (exclusion of the first measurement).
Hochner; 2012; Israel	Population-based cohort; the Jerusalem Perinatal Family Follow-up Study	1,256 offspring (50.5)	AD: mean ± SD: 28 ± 5.47	Self-reported prepregnancy BMI	~32	Oscillometric method (Omron M7), NR	3 consecutive measurements, sitting position, 5-min rest, right arm.
Laor; 1997; Israel	Population-based cohort; the Jerusalem Perinatal Study	10,833 offspring (38.6)	NR	Self-reported prepregnancy BMI	~17	Auscultation method (Baumann), recommended method (Lurbe et al., 2009)	Sitting position, right arm, appropriate cuff size, end point for DBP was the disappearance of the Korotkoff sounds (Phase V).
Lawlor; 2004; Australia	Prospective study; Mater-University Study of Pregnancy and its outcomes	3,864 (NR)	AD: mean ± SD: 25.0 ± 5.1	Self-reported prepregnancy BMI	~5	Oscillometric method (NR), NR	2 measurements, child seated and at rest, 5 min apart, mean of 2 readings.
Marshall; 2011; USA	Mixed-longitudinal study; NR	71 children (49.3)	NR	NR	Range: 3.4–8.8	NR	NR
Morrison; 2013; Canada	Longitudinal cohort study; Family Atherosclerosis Monitoring in Early Life	901 mother–newborn pairs (49.6), sample size for BP analyses: 488	AD: mean ± SD: 32.0 ± 5.4	Self-reported prepregnancy weight	~2.6 days after birth	Oscillometric method (Dinamap Pro100 V2), NR	3 measurements, baby was sleeping or lying quietly, 2 min intervals.
Perng; 2014; USA	Prospective cohort study; recruitment of pregnant women in Massachusetts (USA) to examine prenatal diet and other factors in relation to maternal and child health (Project Viva)	1,090 mother–child pairs (50.3), sample size for BP analyses: 1,084	AP: n, age category; 106,15–24; 647, 25–34; 337, 35–44	Self-reported prepregnancy BMI	Median: 7.7 Range: 6.6–10.9	Oscillometric method (NR), NR	5 measurements, 1 min apart, mean value.
Wen; 2011; USA	Cohort study; Collaborative Perinatal Project	30,461 mother–child pairs (50.7), sample size for BP analyses: 27,625	AP: mean ± SD: 24.1 ± 6.1	Self-reported prepregnancy BMI	~7	Auscultation method (NR), recommended method (Lurbe et al., 2009)	Rest in a recumbent position, right arm.

(Continues)

Adjustment for Variable Set 1 was considered as a necessary prerequisite. Only five studies (31%) adjusted for Variable Set 1; thus, only these five studies (Gademan et al., 2013; Gaillard et al., 2014; Gaillard et al., 2016; Lawlor et al., 2004; Perng et al., 2014) were rated as good-quality studies. Due to the low methodological quality of the majority of studies, a meta-analysis was not possible.

3.4 | Association of MPBW and offspring's later SBP

Of the 15 studies available, five studies (33%) were rated as good-quality studies, which described a significant association after adjustment for Variable Set 1 (Gademan et al., 2013; Gaillard et al., 2014; Gaillard et al., 2016; Lawlor et al., 2004; Perng et al., 2014; Table 3 and Figure 2). After further inclusion of the (potential) effect mediating variables of the offspring's anthropometric characteristic (Variable Set 2), only in one study the association remained significant (Lawlor et al., 2004). Hence, evidence for an independent or direct association had to be rated as "limited—no conclusion." According to the WCRF criteria, this grading means that the present evidence is so limited that no final conclusion can be made. However, it does not necessarily indicate that there is evidence of no relationship. With further good quality research, the classification could be upgraded (in the direction of a probable or convincing effect as well as no substantial effect); see also **Appendix S7**.

The five studies that reported a significant association between MPBW and offspring's SBP were therefore interpreted as indicating an indirect effect, mediated via offspring's anthropometric characteristics. The evidence for this association was assessed as "limited—suggestive." This grade includes—following the WCRF criteria—that there is suggestive evidence of a generally consistent direction of the effect but is too limited for a probable or convincing causal judgement (**Appendix S7**).

3.5 | Association of MPBW and offspring's later DBP

Of the 12 studies included, three papers adjusted for Variable Set 1 (25%; Gademan et al., 2013; Gaillard et al., 2014; Gaillard et al., 2016) and one study (Gademan et al., 2013) described a significant association (Table 3 and Figure 2). Further inclusion of Variable Set 2 did not change the significant association in this study (Gademan et al., 2013). Due to the availability of three studies, of which only one study reported a statistically significant result, the evidence grade "limited—no conclusion" had to be chosen.

3.6 | Association of MPBW and offspring's later MAP

MAP was investigated in three studies (Derraik, Ayyavoo, Hofman, Biggs, & Cutfield, 2015; Eisenman et al., 2010; Marshall, Laurson, Heelan, & Eisenmann, 2011), all describing a positive significant association (Table 3 and Figure 2). Because covariate adjustment was insufficient (no complete adjustment for Variable Set 1), the evidence grade "limited—no conclusion" had to be chosen.

TABLE 2 (Continued)

First author; Year; Country	Study setting; population	Maternal characteristics		Offspring's characteristics		Outcome measurement's characteristics	
		Sample size (% female offspring)	Age at enrolment in pregnancy or at delivery (years)	Measurement prepregnancy BMI or weight	Age at outcome measurement (years)	Method (firm name), validation for children or adolescents	Technique of outcome measurement
West; 2011; USA	Retrospective cohort; mothers were members of the Kaiser Permanente of Colorado health plan	521 mother–child pairs (50.1)	NR	Prepregnancy BMI obtained from medical records or self-reported	Children exposed to diabetes in utero: mean ± SD: 9.5 ± 1.7. Children not exposed to diabetes in utero: mean ± SD: 10.6 ± 1.4	NR	2 measurements, sitting position, mean value.

Note. AD = age at delivery; AP = age at enrolment in pregnancy; BMI = body mass index; BP = blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; NR = not reported; SBP = systolic blood pressure; SD = standard deviation.

TABLE 3 Associations reported between maternal prepregnancy BMI or weight and offspring's later BP of the 16 studies included

First author; Year; Country	Statistical analysis	Adjustment	Outcome	Adjusted effect estimate
Daraki; 2015; Greece	Multivariable regression analyses	Maternal variables: Age (delivery), smoking during pregnancy, parity, education level (stratified low, medium, and high), and GWG. Offspring variables: Birthweight, breastfeeding duration, and TV watching at 4 years of age.	SBP DBP	ß [95% CI], 0.21 [−0.24, 0.67]; $p \geq .05$ ß [95% CI], −0.10 [−0.54, 0.33]; $p \geq .05$
Derraik; 2015; New Zealand	Multivariable regression analyses	Maternal variable: Height. Offspring variables: Sex, age, birthweight standard deviation scores, and birth order.	SBP (daytime) SBP (night-time) DBP (daytime) DBP (night-time) MAP	ß [95%], 0.79 [0.20, 1.39]; $p = .01$ ß [95%], 0.80 [0.15, 1.45]; $p = .017$ ß [95%], 0.44 [−0.01, 0.89]; $p \geq .05$ ß [95%], 0.20 [−0.27, 0.66]; $p \geq .05$ ß [95%], 0.51 [0.07, 0.95]; $p = .025$
Eisenman; 2010; USA	Comparison of offspring of prepregnancy normal-weight mothers (BMI < 25 kg/m^2) versus offspring of prepregnancy overweight mothers (BMI > 25 kg/m^2)		SBP DBP MAP	Normal-weight mmHg ± SD, 104.2 ± 8.8 Overweight mmHg ± SD, 106.6 ± 9.1; $p \geq .05$ Normal-weight mmHg ± SD, 69.3 ± 7.0 Overweight mmHg ± SD, 72.2 ± 7.2; $p \geq .05$ Normal-weight mmHg ± SD, 80.9 ± 6.7 Overweight mmHg ± SD, 83.7 ± 7.0; $p \geq .05$
		Additional adjustment for offspring variables: Sex, age, height, and %BF	MAP	Normal-weight mmHg ± SD, 81.2 ± 0.5 Overweight mmHg ± SD, 83.2 ± 0.9; $p < .05$
Filler; 2011; UK	Correlation	NR	SBP DBP	Spearman rank correlation, $r = .09$; $p < .0001$ Spearman rank correlation, $r = .06$; $p = .0007$
Fraser; 2010; UK	Multivariable regression analyses	Maternal variables: Age at birth, smoking in pregnancy, GWG in previous period, parity, head of household social class, mode of delivery, and prepregnancy weight. Offspring variables: Sex, age, height squared, and fat mass for height.	SBP DBP	ß [95%], 0.108 [0.087, 0.130]; $p < .05$ ß [95%], 0.028 [0.013, 0.043]; $p < .05$
Gademan; 2013; Netherlands	Multivariable regression analyses	Maternal variables: Ethnicity, age, smoking, parity, education, height, and hypertension during pregnancy. Offspring variables: Age at time of outcome measurement, sex, gestational age, and height. Additional adjustment for offspring's variable: Birthweight Additional adjustment for offspring's variable: Current BMI	SBP DBP SBP DBP SBP DBP	ß [95%], 0.14 [0.07, 0.21]; $p < .05$ ß [95%], 0.11 [0.05, 0.17]; $p < .05$ ß [95%], 0.16 [0.09, 0.23]; $p < .05$ ß [95%], 0.13 [0.07, 0.19]; $p < .05$ ß [95%], 0.07 [0.00, 0.14]; $p \geq .05$ ß [95%], 0.07 [0.01, 0.13]; $p < .05$
Gaillard; 2016; Netherlands	Multivariable regression analyses	Maternal variables: Age, ethnicity, smoking during pregnancy, parity, education level, caesarean delivery, alcohol consumption during pregnancy, folic acid supplement use, and total calorie intake during pregnancy. Paternal variables: Age, education level, and ethnicity.	SBP DBP	ß [95%], 0.08 [0.05, 0.11]; $p < .05$ ß [95%], 0.02 [−0.01, 0.05]; $p \geq .05$

(Continues)

TABLE 3 (Continued)

First author; Year; Country	Statistical analysis	Adjustment	Outcome	Adjusted effect estimate
		Offspring variables: Sex, age, breastfeeding duration, average duration of TV watching, and timing of introduction of solid food.		
		Additional adjustment for offspring's variable: Birth characteristics	SBP	ß [95%], 0.08 [0.05, 0.11]; $p < .05$
			DBP	ß [95%], 0.03 [0.0, 0.06]; $p \geq .05$
		Additional adjustment for offspring's variable: Current BMI	SBP	ß [95%], 0.02 [−0.01, 0.05]; $p \geq .05$
			DBP	ß [95%], 0.0 [−0.03, 0.03]; $p \geq .05$
		Fully adjusted model: Including all potential variables (see above) and intermediates (pregnancy complications, GWG, birth characteristics, infant growth, and current BMI)	SBP	ß [95%], 0.04 [0.01, 0.07]; $p < .05$
			DBP	ß [95%], 0.01 [−0.03, 0.04]; $p \geq .05$
Gaillard; 2014; Australia	Multivariable regression analyses	Maternal variables: Age, ethnicity, smoking during pregnancy, parity, education level, GWG rate, household income, gestational hypertension disorders, caesarean delivery, and gestational diabetes. Paternal variable: BMI. Offspring variables: Sex, age, gestational age at birth, weight and length at birth, breastfeeding duration, infant length and weight growth, adolescent Tanner stage, alcohol consumption, dietary intake, physical activity, and sedentary behaviour.	SBP	ß [95%], 0.08 [0.03, 0.14]; $p < .01$
			DBP	ß [95%], 0.0 [−0.06, 0.07]; $p \geq .05$
		Additional adjustment for offspring's variable: Current BMI	SBP	ß [95%], 0.01 [−0.05, 0.07]; $p \geq .05$
			DBP	ß [95%], 0.01 [−0.06, 0.08]; $p \geq .05$
Hochner; 2012; Israel	Multivariable regression analyses	Maternal variables: Age, ethnicity, smoking, parity, years of education, SES, and medical condition. Offspring variables: Sex, birthweight, gestational week, physical activity, smoking status, and years of education.	SBP	ß [95%], 0.44 [0.15, 0.73]; $p = .003$
			DBP	ß [95%], 0.29 [0.05, 0.05]; $p = .017$
		Additional adjustment for offspring's variable: Current BMI	SBP	ß [95%], 0.08 [−0.21, 0.36]; $p = .59$
			DBP	ß [95%], −0.003 [−0.23, 0.23]; $p = .983$
Laor; 1997; Israel	Multivariable regression analyses	Maternal variables: Ethnic origin and BMI. Offspring variables: Birthweight and weight at age 17.	SBP	Women: ß [95%], −0.12 [−0.37, 0.14]; $p \geq .05$ Men: ß [95%], −0.03 [−0.23, 0.18]; $p \geq .05$
			DBP	Women: ß [95%], 0.001 [−0.18, 0.18]; $p \geq .05$ Men: ß [95%], 0.11 [−0.03, 0.25]; $p \geq .05$
Lawlor; 2004; Australia	Multivariable regression analyses	Maternal variables: Age, smoking, maternal education, family income during year of pregnancy, and prepregnancy BMI (continuous). Offspring variables: Sex, age, and birth order.	SBP	ß [95%], 0.70 [0.39, 1.04]; $p < .05$
		Additional adjustment for offspring's variable: Birthweight	SBP	ß [95%], 0.65 [0.20, 0.91]; $p < .05$
		Additional adjustment for offspring's variables: Weight and height at age 5 years	SBP	ß [95%], 0.38 [0.04, 0.72]; $p < .05$

(Continues)

TABLE 3 (Continued)

First author; Year; Country	Statistical analysis	Adjustment	Outcome	Adjusted effect estimate
Marshall; 2011; USA	Correlation	NR	MAP	Correlation coefficient, $r = .35–.53$; $p < .05$
Morrison; 2013; Canada	Multivariable regression analyses	Offspring variables: Sex, age, and newborn's age at birth visit.	SBP DBP	Data not reported; $p \geq .05$ Data not reported; $p \geq .05$
Perng; 2014 USA	Multivariable regression analyses	Maternal variables: Age, race or ethnicity, smoking habits during pregnancy, parity, and annual household income. Paternal variable: BMI. Offspring variables: Sex, age at midchildhood examination, and height z-score.	SBP	ß [95%], 0.77 [0.27, 1.27]; $p < .05$
		Additional adjustment for maternal variable: GWG	SBP	ß [95%], 0.74 [0.22, 1.25]; $p < .05$
		Additional adjustment for offspring's variable: DXA total fat mass index.	SBP	ß [95%], 0.29 [−0.28, 0.86]; $p \geq .05$
Wen; 2011; USA	Multivariable regression analyses	Maternal variables: Age at pregnancy, race, parity, family socio-economic status percentile, and marital status. Offspring variables: Sex, gestational age, and small for gestational age.	SBP	Underweight: ß [95%], −0.85 [−1.14, −0.56]; $p < .05$ Overweight: ß [95%], 0.89 [0.52, 1.26]; $p < .05$
		Additional adjustment for offspring's variables: Childhood BMI at birth, change from birth to 1 year, and change from 1 to 7 years.	SBP	Underweight: ß [95%], 0.02 [−0.27, 0.30]; $p \geq .05$ Overweight: ß [95%], −0.04 [−0.40, 0.31]; $p \geq .05$
West; 2011; USA	Multivariable regression analyses	Offspring variable: Current BMI.	SBP DBP	Data not reported; $p > .10$ Data not reported; $p > .10$

Note. BMI = body mass index; BF = body fat; BP = blood pressure; DBP = diastolic blood pressure; DXA = dual energy X-ray absorptiometry; GWG = gestational weight gain; MAP = mean arterial pressure; NR = not reported; SBP = systolic blood pressure; SD = standard deviation; SES = socio-economic status.

4 | DISCUSSION

4.1 | Summary of evidence

To our knowledge, this is the first review that systematically searched and summarized the evidence regarding an association of MPBW, or with early pregnancy BMI or weight, respectively, with offspring's BP in later life. For the latter exposure, no studies could be identified. For the exposure MPBW, 16 studies were finally included. Only five (31%) of them were rated as good-quality studies. These studies described a significant association of MPBW with offspring's SBP (Gademan et al., 2013; Gaillard et al., 2014; Gaillard et al., 2016; Lawlor et al., 2004; Perng et al., 2014). However, when these studies were further stratified by presenting results from regression models that included offspring's anthropometry (Variable Set 2), only one study (Lawlor et al., 2004) reported a remaining significant association of maternal prepregnancy BMI with offspring's SBP at 5 years of age. Because the inclusion of offspring's anthropometry into the analytic models in almost all but two studies removed the association described before, an indirect or mediated effect was assumed. With respect to DBP, one study (Gademan et al., 2013) described a significant association both with and without inclusion of offspring's anthropometry characteristics at 5 or 6 years of age into the regression model. No good-quality studies were found with respect to offspring's later MAP. Applying the WCRF (2007) evidence classifications, the evidence

for an independent or direct association of MPBW with offspring's later SBP, DBP, and MAP was graded as "limited—no conclusion." Upon interpreting the reported findings after inclusion of potentially mediating variables into the regression models as hints for an indirect association between MPBW with offspring's later SBP, the respective evidence was rated as "limited—suggestive."

The hypothesis that there might be a direct association between MPBW and offspring's BP was based on insights regarding the sympathetic nervous system as a key regulation system (Schlaich et al., 2004; Zhou, Xie, Wang, & Yang, 2012) and possible environmental influences during fetal life that may lead to a lifetime programming of the autonomic nervous system and related metabolic pathways (Samuelsson, 2014; Thornburg, 2015). Both animal (Samuelsson, 2014) and human studies (Ojala et al., 2009) point to such a link. The discussed explanatory approaches include a dysregulation of the offspring's sympathetic activity through a surplus of leptin ("sympathoexcitatory hyperresponsiveness"), while the metabolic effects of leptin are suppressed ("selective leptin resistance"; Samuelsson et al., 2010; Taylor, Samuelsson, & Poston, 2014). Another possible explanation, based on animal studies, is related to an overactivity of the renal sympathetic system due to excessive leptin levels that altered central hypothalamic sensitivity to leptin and in turn increased BP (Prior et al., 2014; Samuelsson, 2014; Samuelsson et al., 2010).

Although the available evidence did not support a direct link between MPBW and offspring's BP, this review identified indications

	All Studies	Inadequate adjustment	Adjustment for variable-set 1[a]	Adjustment for variable-set 1[a] and 2[b]
SBP	**15 studies** Daraki et al., 2015; Derraik et al., 2015; Eisenman et al., 2010; Filler et al., 2011; Fraser et al., 2010; Gademan et al., 2013; Gaillard et al., 2014; Gaillard et al., 2015; Hochner et al., 2012; Laor et al., 1997; Lawlor et al., 2004; Morrison et al., 2013; Perng et al., 2014; Wen et al., 2011; West et al., 2011	**10 studies** Daraki et al., 2015; Derraik et al., 2015[c]; Eisenman et al., 2010; Filler et al., 2011; Fraser et al., 2010; Hochner et al., 2012; Laor et al., 1997; Morrison et al., 2013; Wen et al., 2011; West et al., 2011	**5 studies** Gademan et al., 2013; Gaillard et al., 2014; Gaillard et al., 2015; Lawlor et al., 2004; Perng et al., 2014	**5 studies** Gademan et al., 2013; Gaillard et al., 2014; Gaillard et al., 2015; Lawlor et al., 2004; Perng et al., 2014
DBP	**12 studies** Daraki et al., 2015; Derraik et al., 2015; Eisenman et al., 2010; Filler et al., 2011; Fraser et al., 2010; Gademan et al., 2013; Gaillard et al., 2014; Gaillard et al., 2015; Hochner et al., 2012; Laor et al., 1997; Morrison et al., 2013; West et al., 2011	**9 studies** Daraki et al., 2015; Derraik et al., 2015; Eisenman et al., 2010; Filler et al., 2011; Fraser et al., 2010; Hochner et al., 2012; Laor et al., 1997; Morrison et al., 2013; West et al., 2011	**3 studies** Gademan et al., 2013; Gaillard et al., 2014; Gaillard et al., 2015	**3 studies** Gademan et al., 2013; Gaillard et al., 2014; Gaillard et al., 2015
MAP	**3 studies** Derraik et al., 2015; Eisenman et al., 2010; Marshall et al., 2011	**3 studies** Derraik et al , 2015; Eisenman et al., 2010; Marshall et al., 2011	**No studies**	**No studies**

BMI, body mass index; DBP, diastolic blood pressure; MAP, mean arterial pressure; SBP, systolic blood pressure;

[a] variable-set 1 contains maternal age at enrollment in pregnancy or at delivery, smoking during pregnancy, maternal socio-economic status, offspring's sex and offspring's age; [b] variable-set 2 contains birth weight and offspring's anthropometric characteristics at outcome assessment; [c] underlined studies, significant association

FIGURE 2 Overview of the associations of maternal prepregnancy BMI or weight with offspring's later SBP, DBP, and MAP

for an indirect or mediated association: As shown by our adjustment handling, offspring's anthropometry may almost entirely explain the relation between MPBW and offspring's BP. Similarly, other thematically close, recently published studies point to such an indirect relation of MPBW with cardiometabolic risk factors such as metabolic syndrome (including BP, high-density lipoprotein, triglycerides, waist circumference, and HbA1c; Delpierre et al., 2016) and cardiac structure (including left ventricular mass, left ventricular mass index, relative wall thickness, fractional shorting, and eccentric left ventricular hypertrophy; Toemen et al., 2016); the association attenuated into nonsignificance after adding participants' BMI to the multivariate model. Previous studies already described a strong relation of MPBW with higher rates of offspring's overweight or obesity (Pacce et al., 2015; Yu et al., 2013), whereas childhood BMI is in turn related to cardiometabolic outcomes (Black et al., 2014; Di Bonito et al., 2014), including BP (Friedemann et al., 2012; Onis et al., 2013). Hence, it could be concluded that MPBW is associated with offspring's BMI, which in turn affects cardiometabolic outcomes and the risk of CVD. Thus, MPBW could be an important determinant in offspring's metabolic pathology.

Regarding the influence of the maternal weight status also gestational weight gain (GWG) is an often discussed exposure. Previous studies outlined that an independent association of GWG with offspring's BP appears not to be clear (Fraser et al., 2010; Gaillard et al., 2016; Hochner et al., 2012; Mamun et al., 2009; Perng et al., 2014). However, in some studies, particularly, GWG in early pregnancy seemed to play a critical role in influencing offspring's BP (Hochner

et al., 2012; Mamun et al., 2009). Similar to our study, these results also point to an explanation via offspring's anthropometry (Hochner et al., 2012; Mamun et al., 2009).

Upon explaining these associations on the biological level, insulin has been suggested as a central determinant (Plagemann, 2011). Fetal insulin production was shown to be stimulated by the availability of glucose and amino acids (offered by the mother), thereby programming the insulin set point. A high glucose and amino acid load due to maternal overnutrition might therefore contribute to a permanent hyperinsulinaemia in the offspring, which in turn increases the risk for obesity and other metabolic disorders (Plagemann, 2011), which have been consistently described to be closely linked to BP levels and the risk of hypertension (Aneja, El-Atat, McFarlane, & Sowers, 2004; Chandra et al., 2014; Lim & Meigs, 2014; Park et al., 2013; Wang et al., 2015; WHO, 2013). Alternatively, the epigenetic modification of the neonatal epigenome via intrauterine mechanisms (e.g., increased DNA methylation) during the developmental period was suggested to contribute to alterations in key regulatory pathways (Bruce & Hanson, 2010; Desai, Jellyman, & Ross, 2015; Sharp et al., 2015; Symonds et al., 2009).

For a correct placement of the described associations, it is important to note that adjustment for mediator variables (e.g., birthweight and/or offspring's anthropometric characteristics) could induce a noncausal association (collider stratification bias [CSB]) between MBPW and possible unmeasured confounding variables, block the effect of these variables as well as open a backdoor path and cause confounding within the association of MPBW and offspring's BP via unmeasured

confounding variables (Porta, Vineis, & Bolúmar, 2015; Whitcomb, Schisterman, Perkins, & Platt, 2009). Hence, the assessment of the direct effect of MPBW on BP could be biased (Porta et al., 2015). Such pathways are usually illustrated through directed acyclic graphs (DAGs; Porta et al., 2015; Whitcomb et al., 2009). So far, evidence regarding the impact of CSB in applied research is limited, especially in the field of perinatal epidemiology (Whitcomb et al., 2009). With our adjustment handling, we tried to separate the possible associations in direct and indirect ones. However, in the studies enclosed, none considered possible CSB or applied a DAG. Therefore, it cannot be excluded that unmeasured variables biased the analysed association of MPBW and offspring's BP.

4.2 | Limitations within the included studies

First, the lack of an adequate adjustment in most of the included studies has to be mentioned. Second, no causal path analyses were presented and neither mediating nor colliding effects of variables were considered. Third, the included studies displayed a broad heterogeneity in terms of exposure and outcome assessments and had several methodological limitations. The exposure (MPBW) was mainly self-reported, which implies a certain risk of underreporting (Gorber, Tremblay, Moher, & Gorber, 2007; Han, Abrams, Sridhar, Xu, & Hedderson, 2016); however, self-reported and measured weight showed a high correlation (r = .96 [Iii, Paulet, & Rajpura, 2016], r = .95 [Mamun et al., 2011]). In addition, no study defined the time frame for the ascertainment of "prepregnancy." Measurement of BP varied and was not consistently performed according to recommended guidelines (Lurbe et al., 2009; Makatsariya, Akinshina, Bitsadze, & Khizroeva, 2013). In addition, for the few studies with complete adjustment, residual confounding cannot be excluded.

Therefore, more good-quality studies are required for a concluding judgement. First, these studies should follow standardized reporting guidelines (e.g., CONSORT and STROBE). Second, more attention should be put on the determination of causal pathways (e.g., through DAGs) as well as the identification of all variables that may potentially confound or mediate the association between exposure and outcome. Finally, the use of validated assessment instruments and procedures as well as clearly defined exposure and outcome variables is desirable.

4.3 | Limitations of the systematic review

This systematic review has several limitations. First, no standardized definition exists for essential variables for which should be included in the analytic model. The definitions for Variable Sets 1 and 2 were based on a broad literature search, with the attempt to identify the most relevant variables that should be adjusted for. Therefore, the evaluation of the studies might be different if other requirements for variable control would be applied. Second, so far, no validated and recommended instrument or scale for quality assessment of cohort studies exists (Sanderson, Tatt, & Higgins, 2007). Third, BMI as a proxy for body fat mass is an imperfect marker with a high interindividual variance (Tomiyama, Hunger, Nguyen-Cuu, & Wells, 2016). We used MPBW as an indicator for the peri-pregnancy metabolic environment.

This could be misleading because also fat mass, fat distribution, waist circumference, or waist-to-hip ratio might also be relevant indicators (Czernichow, Kengne, Stamatakis, Hamer, & Batty, 2011; Dhana et al., 2016; Staiano et al., 2012). The association of offspring's BP with other prepregnancy anthropometric indicators should be analysed in further studies.

In conclusion, this systematic review found suggestive, but still limited, evidence for an association of MPBW with offspring's later BP. The interpretation of the available data suggests that the effect may be mainly mediated via offspring's anthropometry.

Given the high and still rising rates of overweight and obesity (NCD Risk Factor Collaboration, 2016), and consequently, rising numbers of pregnancies with a suboptimal weight (Hildingsson, Cederlöf, & Widén, 2011) on the one hand, and high prevalence of both suboptimal BP and hypertension on the other hand (WHO, 2014), this topic remains to be of highest public health relevance. Therefore, further efforts should be made to elucidate the role of maternal weight and anthropometric characteristics before and during early pregnancy on offspring's later health, including BP and hypertension.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

CONTRIBUTIONS

AK is the project leader. HL-W and AK formulated the research question. Study selection and data extraction were conducted by two reviewers (HL-W and MS). HL-W and AK performed the risk of bias assessment and evidence assessment. HL-W, MS, ALBG, and AK analysed and interpreted the data. HL-W and AK wrote the article, and ALBG revised the article.

ORCID

Helena Ludwig-Walz http://orcid.org/0000-0003-1558-8241

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

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