ORIGINAL ARTICLE

# Waist Circumference Measurement for Prediction of Preeclampsia: A Population-Based Cohort Study

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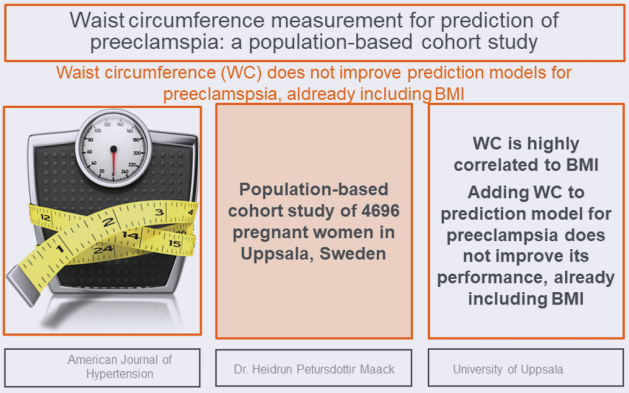
**BACKGROUND**
Identifying women at high risk for preeclampsia is essential for the decision to start treatment with prophylactic aspirin. Prediction models have been developed for this purpose, and these typically incorporate body mass index (BMI). As waist circumference (WC) is a better predictor for metabolic and cardiovascular outcomes than BMI in nonpregnant populations, we aimed to investigate if WC is a BMI-independent predictor for preeclampsia and if the addition of WC to a prediction model for preeclampsia improves its performance.

**METHODS**
We used a population-based cohort of 4,696 women with WC measurements taken in the first trimester. The influence of WC on the risk of developing preeclampsia was evaluated by multivariable logistic regression. We generated receiver operating characteristic curves and calculated the area under the curve (AUC) to evaluate the usefulness of WC measurements for prediction of preeclampsia.

**RESULTS**
Women who developed preeclampsia had greater early pregnancy WC than women who did not (85.8 ± 12.6 vs. 82.3 ± 11.3 cm, $P < 0.001$). The risk of preeclampsia increased with larger WC in a multivariate model, adjusted odds ratio 1.02 (95% confidence interval 1.01–1.03). However, when adding BMI into the model, WC was not independently associated with preeclampsia. The AUC value for preeclampsia prediction with BMI and the above variables was 0.738 and remained unchanged with the addition of WC to the model.

**CONCLUSIONS**
Large WC is associated with a higher risk of preeclampsia, but adding WC to a prediction model for preeclampsia that already includes BMI does not improve the model's performance.

**GRAPHICAL ABSTRACT**

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The incidence of obesity is increasing worldwide at an alarming rate. In 2016, 39% of adults were overweight and 13% were obese according to the World Health Organization (WHO), and the obesity epidemic also affects women of childbearing age.[1] Obesity is strongly associated with several adverse gestational and perinatal outcomes, including preeclampsia.[2] Preeclampsia complicates about 3%–8% of pregnancies and may lead to devastating outcomes for both mother and fetus.[3] Treatment of women with a high risk of preeclampsia with low-dose aspirin has been shown to lower the incidence of preterm preeclampsia.[4] Thus, identification of high-risk individuals is becoming increasingly important in clinical practice as it allows for both treatment and intensified surveillance during pregnancy. In order to

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identify women at risk, a number of prediction models have been developed to estimate the risk for preeclampsia. These models are based on maternal characteristics, including body mass index (BMI), and medical history.[5] Although obesity, defined as increased BMI, is a risk factor for preeclampsia,[2,6] more than half of all obese women will have event-free pregnancies.[7] BMI does not account for the amount of muscle mass or fat distribution, and it has been proposed that not all individuals with obesity are metabolically unhealthy.[8] Waist circumference (WC) is a simple method for measuring fat distribution and obesity. Since WC is superior to BMI in reflecting central abdominal fat deposits, it is used in the metabolic syndrome criteria.[9] Large WC has been associated with increased cardiovascular risk, elevated blood pressure, hyperlipidemia, elevated insulin concentration, and type 2 diabetes.[10,11] Obesity and preeclampsia have many common pathophysiological mechanisms. Abdominal obesity leads to an imbalanced production of fat-derived metabolic markers such as adiponectin, leptin, proinflammatory cytokines, and disturbances in the nitric oxide pathway. These markers are known to be part of the pathophysiological events leading to preeclampsia.[12]

Surprisingly, little effort has been made to establish if WC is a valid measure to distinguish between healthy and unhealthy obesity in pregnancy and if it can be used as a predictive marker of adverse obstetric and neonatal outcomes. As a marker of central obesity, WC is a BMI-independent risk factor for gestational diabetes, birth of infants with macrosomia,[13–15] and preterm birth.[16] Only a few studies on the association between WC and pregnancy hypertensive disorders have been published,[14,17–20] of which 4 have studied preeclampsia specifically. None of these studies, however, addressed if WC is a BMI-independent predictor or if WC can improve prediction in models already including BMI. Further, most of these studies were relatively modest in size, and only 2 measured WC before gestational week 16,[14,17] the latter specifically important if prophylactic aspirin treatment is considered.[4,21]

We hypothesized that greater WC in early pregnancy is associated with preeclampsia later in pregnancy, independent of BMI. Further, we hypothesized that incorporation of WC into a preeclampsia prediction model would improve its' performance in detecting women at high risk for developing preeclampsia.

Our aim in this population-based study, encompassing around 5,000 women with early pregnancy WC measurements, was to explore the association between early pregnancy WC measurements and preeclampsia, taking early pregnancy BMI into account.

## METHODS

### Study population

Between 5 January 2015 and 29 December 2017, we implemented WC measurement at the first antenatal booking in the County of Uppsala, Sweden. During this period, first-trimester WC was measured in 5,827 pregnancies. We reviewed the individual electronic medical records of these pregnancies. We identified and excluded 590 pregnancies that ended in a miscarriage or termination of pregnancy or pregnancies where women gave birth outside of Sweden. For the remaining pregnancies ($n$ = 5,237), we extracted first-trimester systolic and diastolic blood pressures measurements from the electronic medical records. Thereafter, we linked data to the Medical Birth Register held by the Swedish National Board of Health and Welfare. Linkage was enabled through the unique personal registration numbers assigned to all Swedish residents at birth or immigration. The Medical Birth Register includes prospectively collected information for all births at 22 gestational weeks or later in Sweden. Information includes demographic data as well as data on pregnancy, delivery, and neonatal outcomes. The Swedish National Board of Health and Welfare provided data from both the index pregnancy, during which WC was measured and previous pregnancies, enabling us to identify women with any past pregnancy complicated by preeclampsia. Linkage between WC data and the birth register failed in 60 pregnancies, which were excluded. After linkage, the study population was pseudo-anonymized. We further excluded multiple pregnancies ($n$ = 155), pregnancies without available data on BMI in early pregnancy ($n$ = 49) and additional pregnancies in women who had already been included in the cohort ($n$ = 277). The final population included 4,696 singleton pregnancies with available information on early pregnancy BMI and WC that resulted in birth at 22 gestational weeks or later (Figure 1).

### Exposure

The exposure was WC, measured at the first antenatal visit. The mean time point for measurement was 9.1 gestational weeks (SD 2.2 weeks). The measurement was done midway between the lower rib margin and the iliac crest with the woman in a standing position.[22] All measurements were made with standardized measurement tapes. All midwives working at the healthcare centers were given verbal and written information on how to perform the measurements, and this information was repeated several times during the study period.

We used WC as a continuous variable or dichotomized with cutoffs >80 and >88 cm. The cutoffs were chosen based

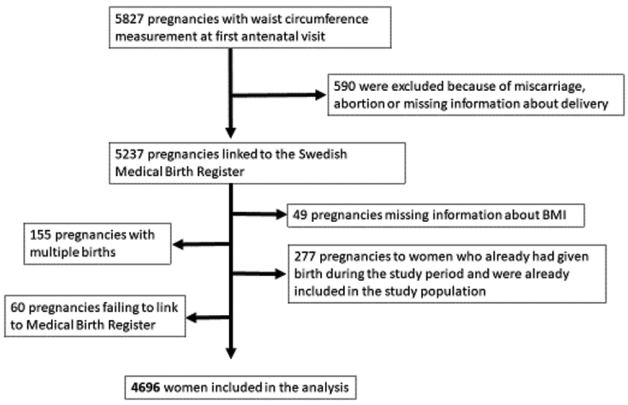


**Figure 1.** Flowchart of the study population. Abbreviation: BMI, body mass index.

on WHO sex-specific cutoff points for increased (>80 cm) and substantially increased (>88 cm) risk of metabolic complications in the general population.[22]

### Main outcome

The main outcome was preeclampsia, defined as *de novo* hypertension after 20 weeks of gestation combined with proteinuria, the clinical criteria for preeclampsia diagnosis in Sweden during the study period. Hypertension was defined as systolic blood pressure ≥140 mm Hg and/or diastolic blood pressure ≥90 mm Hg measured at 2 subsequent occasions, at least 6 hours apart. Proteinuria was defined as ≥2+ on dipstick or ≥300 mg/24 h in a urine collection.

### Maternal demographic and clinical outcomes

Mean arterial pressure was calculated from the systolic and diastolic blood pressures registered at the first antenatal visit. We collected all other data on maternal demographics and clinical outcomes from the Medical Birth Register. Demographic data included maternal height, weight, age, and parity. The women's height was self-reported or measured, while weight was measured at the first antenatal visit. BMI was calculated and classified as underweight (BMI <18.5 kg/m$^2$), normal weight (BMI 18.5–24.9 kg/m$^2$), overweight (BMI 25.0–29.9 kg/m$^2$), or obesity (BMI ≥30.0 kg/m$^2$). Self-reported smoking status (yes/no), if the pregnancy was conceived after ovarian stimulation or *in vitro* fertilization, and chronic diseases were also recorded at the first antenatal visit. Chronic diseases included chronic hypertension, type 1 or type 2 diabetes, systemic lupus erythematosus, and chronic kidney disease. These were identified based on the corresponding International Classification of Diseases (ICD)-10 codes (O10; O11; I10–I15; O240–243; E10–14; M32; N18; N19), entered by the discharging physician after delivery. Each woman's country of birth was categorized as within or outside the European Union.

Clinical variables included gestational length at birth, presented both as a continuous variable and as preterm birth, defined as birth <37 gestational weeks. Further, infant birthweight was included, both measured in grams and small for gestational age, defined as birthweight ≥2 SDs below the Swedish sex-specific reference curve of expected birthweight by gestational age.[23]

### Statistics

We compared demographic and clinical variables in women with preeclampsia and women without preeclampsia using independent *t*-tests or chi-squared tests. Correlation between WC measurement and BMI was drawn as a scatter plot and estimated with Pearson's *t*-tailed correlation tests.

We estimated the association between WC in early pregnancy and the risk of developing preeclampsia with logistic regression analyses. We used WC as a continuous variable (cm) or WC cutoffs of >80 or >88 cm as predictors. Risk was expressed as odds ratios (ORs) or adjusted OR (AOR) with corresponding 95% confidence intervals (CIs). We used established predictors from the National Institute for Health and Care Excellence (NICE) guidelines[24] and a prediction model proposed by Wright *et al.*[5] in our model. The variables in the prediction model included maternal BMI, age, parity, smoking, country of birth, chronic hypertension, type 1 or type 2 diabetes, systemic lupus erythematosus, chronic kidney disease, history of preeclampsia in a past pregnancy, conception with ovarian stimulation or *in vitro* fertilization, and mean arterial pressure at the first antenatal visit. To illustrate the influence of BMI in the prediction model, we performed the modeling in 2 steps. In the first step, we included WC and all the established predictors except BMI (model 1). Then, we added BMI to the model (model 2).

To determine the added value of WC to the prediction model we performed *c*-statistical analyses by generating receiver operating characteristic curves. The area under the curve (AUC), obtained following the logistic regression, was used to compare the models with and without WC. The likelihood ratio test was used to estimate if WC significantly changed the model.

For the statistical analyses, we used IBM SPSS Statistics, version 27 and R 4.0.5.

### Ethical approval

We obtained ethical approval from the Regional Ethical Review Board in Uppsala to implement WC measurements and evaluate their usefulness: 2014/363 and 2015/366.

## RESULTS

### Description of the study population

Among women in our study population, 209 (4.5%) developed preeclampsia.

Table 1 presents demographic and clinical variables in women with and without preeclampsia. The women who developed preeclampsia were younger (29.4 ± 5.2 vs. 30.3 ± 5.0 years), more often nulliparous (66% vs. 45%) and had higher BMI in early pregnancy (26.8 ± 5.5 vs. 25.1 ± 4.8 kg/m$^2$). They also had higher mean arterial pressure at the first antenatal visit than women who did not develop preeclampsia (88 ± 9 vs. 83 ± 8 mm Hg). There was no difference in smoking habits, country of birth, or if the pregnancy was conceived after ovarian stimulation or *in vitro* fertilization. Women who developed preeclampsia more often had prepregnancy type 1 or type 2 diabetes (3.3% vs. 0.9%) and a history of preeclampsia in past pregnancies (10.5% vs. 2.2%). However, they did not differ in rates of chronic hypertension, systemic lupus erythematosus, or chronic kidney disease. Pregnancies complicated by preeclampsia had shorter mean gestational length at birth (272 ± 16 vs. 278 ± 12 days) and resulted in smaller infants than pregnancies without preeclampsia (3,299 ± 712 vs. 3,549 ± 539 g).

### Correlation between WC and BMI

WC measured at the first antenatal visit was strong and positively correlated with early pregnancy BMI; $r = 0.82$, $P \leq 0.001$, see Figure 2.

**Table 1.** Demographic and clinical variables of the study population

| | Preeclampsia (*n* = 209) | Nonpreeclampsia (*n* = 4,487) | P |
|---|---|---|---|
| Waist circumference, cm | 85.8 ± 12.6 | 82.3 ± 11.3 | **<0.001** |
| Waist circumference >80 cm | 128 (61.1) | 2,167 (48.3) | **<0.001** |
| Waist circumference >88 cm | 73 (34.9) | 1,086 (24.2) | **<0.001** |
| First-trimester BMI (kg/m$^2$) | 26.8 ± 5.5 | 25.1 ± 4.8 | **<0.001** |
| <18.5 (underweight) | 3 (1.4) | 107 (2.4) | |
| 18.5–24.9 (normal weight) | 90 (43.1) | 2,526 (56.3) | |
| 25.0–29.9 (overweight) | 68 (32.5) | 1,187 (26.5) | |
| ≥30.0 (obese) | 48 (23.0) | 667 (14.9) | |
| Maternal age | 29.4 ± 5.2 | 30.3 ± 5.0 | **0.011** |
| Nulliparous | 138 (66.0) | 2,019 (45.0) | **<0.001** |
| Smoking | 10 (5.0) | 199 (4.7) | 0.831 |
| Mean arterial pressure (mm Hg) | 88 ± 9 | 83 ± 8 | **<0.001** |
| Born outside EU | 28 (13.4) | 824 (18.4) | 0.068 |
| Chronic hypertension | 3 (1.4) | 25 (0.6) | 0.107 |
| Prepregnancy diabetes[a] | 7 (3.3) | 39 (0.9) | **<0.001** |
| SLE | 0 (0.0) | 10 (0.2) | 0.494 |
| Chronic kidney disease | 0 (0.0) | 8 (0.2) | 0.541 |
| History of preeclampsia | 22 (10.5) | 100 (2.2) | **<0.001** |
| Ovarian stimulation | 0 (0.0) | 32 (0.7) | 0.221 |
| Conception with IVF | 6 (2.9) | 183 (4.1) | 0.385 |
| Gestational diabetes | 7 (3.3) | 109 (2.4) | 0.402 |
| Gestational length at birth, days | 272 ± 16 | 278 ± 12 | **<0.001** |
| Preterm birth (<37 weeks) | 28 (13.4) | 196 (4.4) | **<0.001** |
| Birthweight, g | 3,299 ± 712 | 3,549 ± 539 | **<0.001** |
| SGA | 15 (7.2) | 84 (1.9) | **<0.001** |

Data presented as mean ± SD or *n* (%). *P*-value in bold are significant values. Abbreviations: BMI, body mass index; EU, European Union; IVF, *in vitro* fertilization; SGA, small for gestational age, defined as birthweight under 2 SD of Swedish sex-specific reference curves for gestational age[21]; SLE, systemic lupus erythematosus. Missing data: smoking = 261, birthweight = 4, born outside EU = 2, mean arterial pressure = 84.

[a]Type 1 or type 2 diabetes mellitus.

### WC and risk for preeclampsia

Women who developed preeclampsia had greater early pregnancy WC (85.8 ± 12.6 cm) than women who did not (82.3 ± 11.3 cm), $P < 0.001$ (Table 1). The risk of preeclampsia increased by 2% for every cm increase in WC, i.e., women with greater WC more often developed preeclampsia, crude OR 1.02 (95% CI 1.01–1.04). Compared with women with a WC ≤80 cm, those with a WC >80 cm had 69% increased risk of developing preeclampsia; crude OR 1.69 (95% CI 1.27–2.25). A similar association was noted with the higher WC cutoff (>88 cm), crude OR 1.68 (95% CI 1.26–2.25).

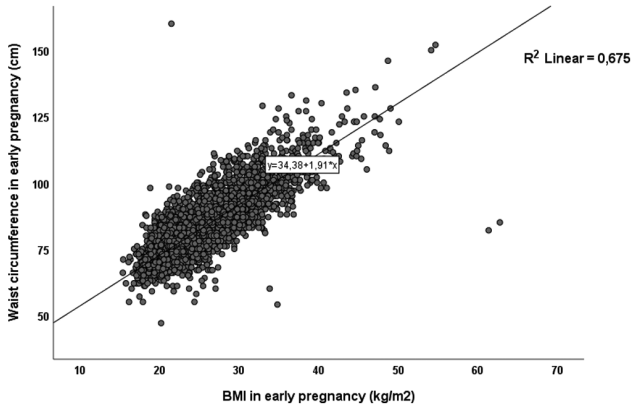


**Figure 2.** Correlation between body mass index (BMI) and waist circumference. Pearson's correlation shows that waist circumference is positively correlated to BMI in early pregnancy, $r = 0.822$, $P \leq 0.001$.

After including age, parity, smoking, country of birth, chronic hypertension, prepregnancy type 1 or type 2 diabetes, systemic lupus erythematosus, chronic kidney disease, history of preeclampsia, conception with ovarian stimulation or *in vitro* fertilization and mean arterial pressure in the model, the association between WC and preeclampsia remained unchanged; AOR 1.02 (1.01–1.03) for WC in cm, AOR 1.59 (1.16–2.17) for WC >80 cm, and AOR 1.50 (1.08–2.98) for WC >88 cm, model 1 (Table 2). When we added early pregnancy BMI into the model, the significant association between WC and preeclampsia disappeared, model 2 (Table 2).

### WC for improvement of a prediction model

Table 3 shows comparisons of receiver operating characteristic curves for the risk of developing preeclampsia using the established predictors, including BMI, without and with the addition of WC to the model. The AUC values for the model without and with WC were 0.738 (95% CI 0.704–0.771) and 0.739 (95% CI 0.705–0.773), respectively, $P = 0.318$. When we excluded BMI from the model, to test the model with WC as the only obesity marker, the AUC value did not change: 0.739 (95% CI 0.705–0.773), Table 3.

## DISCUSSION

Large WC, measured in early pregnancy, was associated with preeclampsia. However, first-trimester WC was closely correlated with first-trimester BMI, and our results indicate that the addition of WC to a prediction model that already incorporates BMI does not improve its performance.

Our findings are in agreement with earlier studies reporting an association between WC and development of pregnancy

**Table 2.** Association between waist circumference in early pregnancy and risk of developing preeclampsia

| | OR (95% CI) | P | Model 1 AOR (95% CI)[a] | P | Model 2 AOR (95% CI)[b] | P |
|---|---|---|---|---|---|---|
| Waist circumference (cm) | 1.02 (1.01–1.04) | **<0.001** | 1.02 (1.01–1.03) | **0.001** | 1.01 (0.99–1.04) | 0.318 |
| Waist circumference >80 cm | 1.69 (1.27–2.25) | **<0.001** | 1.59 (1.16–2.17) | **0.005** | 1.32 (0.90–1.93) | 0.155 |
| Waist circumference >88 cm | 1.68 (1.26–2.25) | **0.001** | 1.50 (1.08–2.08) | **0.015** | 1.12 (0.72–1.73) | 0.629 |

Risk is presented as odds ratio (OR) and illustrates increased risk in preeclampsia for each centimeter increase in waist circumference and stratified by waist circumference cutoffs >80 and >88 cm. *P*-value in bold are significant values. Abbreviations: AOR, adjusted odds ratio; CI, confidence interval.

[a]Model 1: adjusted for age (continuous), parity (nulliparous/multipara), smoking (yes/no), county of origin (born inside/outside European Union), chronic hypertension (yes/no), prepregnancy diabetes (yes/no), systemic lupus erythematosus (yes/no), chronic kidney disease (yes/no), history of preeclampsia (yes/no), conception with ovarian stimulation (yes/no), conception with *in vitro* fertilization (yes/no), and mean arterial pressure (continuous).

[b]Model 2: adjusted for the same variables as model 1 and additional for body mass index (continuous).

**Table 3.** Area under the curve (AUC) from receiver operating characteristic curves for possible prediction of preeclampsia

| | AUC | 95% CI |
|---|---|---|
| Prediction model[a] with BMI | 0.738 | 0.704–0.771 |
| Prediction model with WC | 0.739 | 0.705–0.773 |
| Prediction model with BMI and WC | 0.739 | 0.705–0.773 |

Abbreviations: BMI, body mass index; CI, confidence interval; WC, waist circumference.

[a]Prediction model includes maternal age (continuous), parity (nulliparous/multipara), smoking (yes/no), county of origin (born inside/outside European Union), chronic hypertension (yes/no), prepregnancy diabetes (yes/no), systemic lupus erythematosus (yes/no), chronic kidney disease (yes/no), history of preeclampsia (yes/no), conception with ovarian stimulation (yes/no), conception with *in vitro* fertilization (yes/no), and mean arterial pressure (continuous).

hypertensive disorders.[14,17–20] However, most earlier studies have not measured WC in early pregnancy,[18–20] making the results less useful in preeclampsia prediction.[21] Further, 2 of the studies included ethnic populations[18,20] with a fat distribution that has been shown to differ from WHO standards for nonpregnant populations.[22,25] Therefore, those results might not be generalizable to a European population.[11] We have only found 1 earlier study comparing the performance of BMI and WC in a prediction model for preeclampsia development.[19] The study included measurements in gestational weeks 20–28, and the prediction model included only maternal age, parity, and education. Nevertheless, their results are in agreement with our findings, i.e., that BMI and WC have similar predictive capacity. Our findings expand this knowledge since our measurements were performed in the first trimester, and were included in a prediction model including the most established maternal characteristic predictors. Further, to our knowledge, our study is the first to evaluate if the addition of early pregnancy WC to a prediction model that already includes BMI improves its performance. However, no such improvement could be noted.

In the general nonpregnant population, WC is a risk factor for developing cardiovascular disease,[11,26] and it is at least as good as BMI at predicting metabolic abnormalities, such as hypercholesterolemia, low high-density lipoprotein cholesterol and hypertension.[27] It reflects the volume of abdominal adipose tissue and is associated with an increased risk for cardiovascular disease and mortality, independent of BMI.[26] As the literature suggests that metabolic abnormalities, such as increased leptin, glucose, insulin, and lipids, may be a substantial part of the causal pathway between obesity and preeclampsia,[28] we hypothesized that adding WC to established predictors in a prediction model for preeclampsia would improve its performance. A possible reason why our hypothesis could not be proven is that pregnant women are young, in contrast with the general population at risk for cardiovascular disease. Among younger individuals, WC and BMI seem to be equally important predictors of blood pressure, whereas different effects are seen in older individuals.[29] WC increases with age, and this increase is larger than what can be expected from the increase in BMI.[30] Further, with aging substantial redistribution of fat tissue occurs, leading to decreased subcutaneous fat mass and increased intra-abdominal fat. This suggests that depot-specific changes in fat tissue function with aging may contribute to metabolic dysfunction and related health complications.[31]

A strength of our study was the population-based design, being one of the largest studies on the association between WC and preeclampsia, with almost 5,000 women included. Further, WC is a simple, inexpensive, and reliable tool to estimate central obesity. It can be easily implemented at the first antenatal visit, allowing for risk prediction in the first trimester. As WC measurements were performed in early pregnancy (mean 9 gestational weeks, all measurements before week 15), the size of the uterus should not significantly influence the WC of the women. Another strength is the specific outcome of preeclampsia instead of the more heterogeneous group of pregnancy hypertensive disorders. Further, we had information on other established predictors for preeclampsia, and no other studies have used models including those predictors together with WC and BMI.

There are also limitations to our study worth mentioning. Although 4.5% ($n$ = 209) of our study population developed preeclampsia, only 0.6% ($n$ = 28) developed preterm preeclampsia. Hence, we were not able to explore preterm and term preeclampsia separately. The prevalence of preeclampsia in our population is consistent with other registry studies on the Swedish population of pregnant women.[32,33] Our prevalence was somewhat higher than earlier reports on the association of WC and preeclampsia,[17–19] but at the same level as the study of Ebrahimi-Mameghani *et al.*[14] Another

limitation was that the WC measurement was performed by different midwives at different healthcare centers. However, all midwives were educated on how to measure WC, written information about the procedure was available, and only standardized measurement tapes were used. We did not have information about aspirin treatment during the pregnancies in our database, but national guidelines during the study period comprised a very selective recommendation concerning prophylactic use of aspirin for preeclampsia, with only 1.1% of nulliparous women being treated with aspirin during 2008–2013.[34] In addition, we did not have information about uterine artery pulsatility index or biomarkers commonly used in prediction models for preeclampsia.[4]

Obesity among pregnant women is a growing problem. Identifying those at the highest risk for severe complications, such as preeclampsia, is of great importance in order to offer prevention and individualized pregnancy surveillance. Our study showed that larger WC is associated with an increased risk of developing preeclampsia. However, our results do not support that WC in early pregnancy is associated with preeclampsia development independently of BMI or that adding WC to a prediction model including BMI would improve its performance. This is probably explained by the close correlation between WC and BMI in this relatively young cohort of pregnant women. As WC measures both visceral and subcutaneous adipose tissue, it remains unclear if different adipose tissue distribution influences the risk of preeclampsia in pregnant women. In the future, it would be of interest to evaluate if other anthropometric predictors, such as waist-to-hip ratio or subcutaneous/visceral adipose tissue thickness estimated by ultrasound, can improve risk prediction.

## DISCLOSURE

The authors declared no conflict of interest.

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