Preeclampsia and Prevalence of Microalbuminuria 10 Years Later

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Summary
Background and objectives A recent meta-analysis found that about 30% of women with a previous preeclamptic pregnancy had persistent microalbuminuria at follow-up. The analysis was, however, based on small studies, and more data are needed.

Design, setting, participants, & measurements Using data from the Medical Birth Registry in Norway, this study identified women with or without preeclampsia in their first pregnancy 9–11 years previously (1998–2000). Women with diabetes, rheumatic disease, essential hypertension, or renal disease before pregnancy and/or preeclampsia in later pregnancies were excluded. Eighty-nine women with and 69 women without preeclampsia participated in the study. Urinary albumin-to-creatinine ratio (ACR) was measured in three morning urine samples. Estimated GFR (eGFR) was calculated using the CKD-Epidemiology Collaboration formula.

Results Median urinary ACR in follow-up urine samples was 0.53 mg/mmol for women with and 0.50 mg/mmol for women without preeclampsia (P=0.54). Only one woman (1%) with previous preeclampsia had urinary ACR >2.5 mg/mmol in two of three urine samples. Preeclampsia was not associated with urinary ACR above the 75th percentile. Women with preeclampsia did not have significantly higher eGFR than women without preeclampsia (107.9 versus 104.9 ml/min per 1.73 m²; P=0.12), but preterm preeclampsia was significantly associated with eGFR above the 75th percentile (P=0.03).

Conclusions In this population-based study of otherwise healthy women, preeclampsia 10 years earlier was not associated with increased risk of persisting microalbuminuria. Estimated GFR was not significantly different between women with and those without preeclampsia, but preterm preeclampsia was associated with high normal eGFR.


Introduction
In recent years, several studies have shown an association between preeclampsia and future kidney disease (1,2) and an unfavorable cardiovascular risk profile (3–5). Earlier studies have also reported a substantially higher risk of microalbuminuria after a preeclamptic pregnancy (6,7), and a recent meta-analysis of seven available studies showed that microalbuminuria was present in 31% of women 7 years after a preeclamptic pregnancy (2). We have previously shown that women with preeclampsia had a 4–15 times increased risk of ESRD compared with women without preeclampsia (1). A follow-up study showed that preeclampsia was not associated with faster progression of kidney disease after the time of kidney biopsy (8), suggesting that preeclampsia in itself was associated with development of kidney disease.

Only a limited number of studies have investigated the prevalence of microalbuminuria after preeclampsia. These studies were small and had highly variable patient groups (2). Few studies have investigated early kidney dysfunction in otherwise healthy women with previous preeclampsia.

To investigate the occurrence of early stages of CKD after a single preeclamptic pregnancy, we performed a larger study that included women with or without preeclampsia during their first pregnancy 10 years earlier. We obtained three morning urinary samples for all women for analysis of urinary albumin-to-creatinine ratio (ACR), and estimated GFR (eGFR) was calculated. Our main hypothesis was that women with previous preeclampsia would have increased prevalence of microalbuminuria compared with women without preeclampsia.

Materials and Methods
Registries Involved
Medical data on all births in Norway with a gestational age of at least 16 weeks are forwarded to the Medical Birth Registry of Norway by compulsory notification (9). The notification form includes extensive data on the mother and the newborn and is completed by the attending midwife and doctor. We used data from the Norwegian Population Registry to obtain current addresses. The study was
approved by the Regional Ethics Committee and the participants provided informed consent.

**Study Design**

We identified women living in the Bergen area (population count about 325,000) with preeclampsia in their first pregnancy from 1998 to 2000. Those diagnosed with diabetes, rheumatic disease, essential hypertension, or renal disease before first pregnancy were excluded. Because recurrent preeclampsia is rare (1), the clinical diagnosis of preeclampsia is more difficult in later pregnancies (10), and to avoid unnecessary subgroups, we also excluded women who had later preeclamptic pregnancies. One hundred ninety-one women fulfilled these criteria. Among all the women without preeclampsia in their first pregnancy and with the same exclusion criteria, 191 women matched on age, year of first birth, and municipality were randomly selected as a control group. Twenty women were excluded after invitation because they had moved away from the area or were currently pregnant or breastfeeding. Thus, the number of eligible women for inclusion was 182 in the preeclampsia group and 180 in the control group, and proportions have been calculated on the basis of these numbers.

The women who agreed to participate were examined between December 2009 and October 2010. Before the examination, the participants received a questionnaire on medical history, education, and household income. They also received three containers for urine samples and were instructed to take morning urine samples on three consecutive mornings and store them in the refrigerator until the examination day. At the examination visit, the questionnaire was completed, and body height, weight, waist circumference, and hip circumference were measured. Resting BP was measured manually according to European Society of Hypertension-European Society of Cardiology guidelines (11). All biochemical analyses were done at the Laboratory of Clinical Biochemistry, Haukeland University Hospital, Bergen, Norway. Urinary albumin was measured using nephelometry (Behring Nephelometer Analyzer II; Siemens Healthcare Diagnostics, Tarrytown, NY). Creatinine levels in urine and serum were analyzed using an isotope dilution mass spectrometry traceable enzymatic method (Roche Diagnostics, Basel, Switzerland). The calibrator was traceable to isotope dilution mass spectrometry. We calculated eGFR using the CKD-Epidemiology Collaboration equation (12).

**Exposure Variables**

Criteria for preeclampsia used by the reporting midwives and obstetricians have been in accordance with the recommendations by the American Congress of Obstetricians and Gynecologists (13): increased BP after 20 weeks’ gestation ($\geq 140/90$ mmHg) and proteinuria ($\geq 0.3$ g in a 24-hour urine specimen or $+1$ or greater on urinary dipstick). Other variables registered in the Medical Birth Registry of Norway were total number of pregnancies until follow-up, age, offspring birthweight, gestational age, and delivery mode of the first birth. Offspring birth weight is measured shortly after birth; a weight $< 2.5$ kg was categorized as low birthweight. Estimation of gestational age was based on routine ultrasonographic examination between gestational weeks 17 and 20. Birth at a gestational age $< 37$ weeks was defined as preterm.

**Statistical Analyses**

Mean values in women with and without preeclampsia were compared using unpaired t tests; values are given as mean $\pm$ SD. Urinary albumin excretion values were skewed, and we therefore analyzed these data using the Mann-Whitney test; median values are given. Chi-squared tests were used to compare frequencies between the groups. Because urinary albumin excretion is associated with important endpoints also within normal ranges (14), we investigated risk factors for having urinary albumin excretion above the 75th percentile (corresponding to a urinary ACR of 0.70 mg/mmol). Because women with previous preeclampsia tended to have higher levels of eGFR, we also analyzed risk factors associated with eGFR above the 75th percentile; adjusted models included adjustment for age, body mass index, marital status, annual household income, and highest education level. The analyses were performed using the statistical package SPSS 20 (SPSS Inc., Chicago, IL).

**Results**

Among the invited women, 89 with previous preeclampsia and 69 without preeclampsia agreed to participate in the study. The participation rates were 49% for the preeclampsia group, 38% for the control group, and 44% overall. The remaining women declined the invitation or did not respond. Mean duration from first birth to follow-up was $10.9 \pm 1.0$ years.

Clinical and socioeconomic characteristics, as well as pregnancy outcomes of the first pregnancy, were compared between women with preeclampsia and women without preeclampsia (Table 1). Age, weight, body mass index, waist-to-hip ratio, total number of pregnancies, weekly physical activity, smoking habits, and educational level were not significantly different between the groups. Significantly more women in the preeclampsia group had a high annual household income ($> 600,000$ NOK) than women who had not had preeclampsia ($P = 0.04$). As expected, women with preeclampsia in their first pregnancy had more adverse pregnancy outcomes in their first pregnancy than women without preeclampsia.

**BP**

There was a nonsignificant trend toward higher BP in women with previous preeclampsia than in women without preeclampsia: 118 versus 114 mmHg for systolic BP ($P = 0.10$) and 73 versus 71 mmHg for diastolic BP ($P = 0.18$) (Table 1). Eleven women had hypertension, defined as a BP $\geq 140/90$ mmHg (11) and/or use of antihypertensive medications and/or self-report of diagnosed hypertension: eight women with previous preeclampsia (9%) and three women without preeclampsia (4%) ($P = 0.26$).

**Microalbuminuria**

Only one woman with previous preeclampsia had microalbuminuria in at least two of three samples according to the recommended cutoff value (2.5 mg/mmol) (14), and no women without preeclampsia had microalbuminuria. Median urinary ACR was 0.53 mg/mmol for women
with preeclampsia and 0.50 mg/mmol for women without preeclampsia \((P=0.54)\) (Figure 1). Preeclampsia, preeclampsia with preterm birth, low-birthweight offspring, and birthweight for gestational age less than the 10th percentile were not associated with urinary ACR above the 75th percentile (Table 2). These results did not change after adjustments for age, body mass index, marital status, annual household income, and highest education level.

**eGFR**

Mean eGFR was not significantly different for women with preeclampsia compared with women without preeclampsia \((108 \text{ versus } 105 \text{ ml/min per } 1.73 \text{ m}^2; P=0.12)\) (Table 1). Thirty-nine women had eGFR above the 75th percentile \((114.3 \text{ ml/min per } 1.73 \text{ m}^2)\), and we defined this as high-normal GFR. In logistic regression analysis, preeclampsia was almost significantly associated with high-normal GFR (odds ratio, 2.1; \(P=0.06\)). In further analyses, preterm preeclampsia was significantly associated with high-normal eGFR (Table 3 and Figure 2). The strength of the association was preserved after adjustments, but the \(P\) value increased slightly. Similar but not significant trends were seen for preeclampsia with low-birthweight offspring or offspring small for gestational age. Similar results were found when we used the 90th percentile \((118.5 \text{ ml/min per } 1.73 \text{ m}^2)\) as the cutoff level, but the 75th percentile was chosen to obtain sufficient group sizes for adjusted analyses. Of the adjustment factors, only young age was a significant risk factor for high-normal eGFR; body mass index was not a significant risk factor.

**Comparison of Participants with Population Average**

We compared characteristics of first pregnancy for our participants with those of the total population of women with their first pregnancy registered in the Medical Birth Registry of Norway and the same inclusion and exclusion criteria as the participants (Supplemental Table 1). Compared with all women without preeclampsia, participants without preeclampsia were older \((28.0 \text{ versus } 26.6 \text{ years}; P<0.001)\), had low-birthweight offspring slightly more often \((7.2\% \text{ versus } 3.1\%; P=0.05)\), and more frequently had preterm birth \((11\% \text{ versus } 5.2\%; P=0.07)\); however, frequency of being single or having a caesarean delivery was similar compared with the population average. There were no differences between participants with preeclampsia and all women with preeclampsia except that the participants were somewhat older \((27.1 \text{ versus } 26.3 \text{ years}; P<0.001)\).

Using data from Statistics Norway, we examined self-reported education level in the participating women aged 30–49 years and compared these with the expected number from official statistics for all women aged 30–49 years in the relevant municipalities, weighted for the municipality composition of our participants. These analyses showed that our participants had completed higher education above high school level more often than did the general population \((68\% \text{ versus } 53\%; P<0.001)\).

**Discussion**

This study is one of the largest population-based, long-term follow-up studies of otherwise healthy women with preeclampsia in their first pregnancy and one of few studies that have investigated urinary albumin excretion obtained in three standardized sets of morning urinary samples. The study, surprisingly, showed no increase in median urinary ACR or in risk of having a urinary ACR above the 75th percentile in these women. In fact, only 1 of 89 women...
with previous preeclampsia had microalbuminuria in this study. In addition, although there were no significant differences in mean eGFR values between women with and without previous preeclampsia, preterm preeclampsia was statistically significantly associated with eGFR above the 75th percentile.

In 2010, McDonald et al. published a meta-analysis of seven studies that had reported data on risk of microalbuminuria after preeclampsia, with a total of 237 women with previous preeclampsia and 333 women with uncomplicated pregnancies. Thirty-one percent of women with previous preeclampsia had microalbuminuria after a mean follow-up of 7 years, much higher than the 7% observed in women without previous preeclampsia (2). Several factors might explain why our results differ from earlier findings. The meta-analysis included women from many different clinical settings (e.g., two of the studies were of women with the HELLP [hemolysis, elevated liver enzymes, low platelet count] syndrome [15] or type 1 diabetes mellitus [16]). Furthermore, studies based on otherwise healthy women most often included women with more severe preeclampsia (6,17). This is different from our cohort, in which the majority probably were less severely affected; we invited all previously healthy women with preeclampsia in their first birth in the given timeframe and area, and only 16% of the women with preeclampsia had preterm birth. An important factor may also be the fact that most previous studies combined women with preeclampsia in their first pregnancy with women with presumed preeclampsia in their second or later pregnancies (2). In a thoroughly performed kidney biopsy study, preeclampsia was a difficult diagnosis in multiparous women; it could be morphologically verified in only about 40% of the patients with a previous pregnancy, whereas 50% had various underlying kidney diseases (10). Such underlying kidney disease is more likely to persist than preeclampsia-associated changes and might complicate the interpretation of these studies. Finally, there may be an important publication bias because the number of previous studies is small and the results have been more or less the same. In line with our findings, two recent smaller studies found no microalbuminuria in previously preeclamptic patients (18,19).

Another possibly important factor may be the timing of follow-up. Previous studies have shown that glomerular damage and proteinuria often are extensive during a preeclamptic pregnancy (20,21) but normalize soon after birth in the majority (22). In a small subset of patients, the resolution might, however, take years (23), and as described earlier, follow-up studies have suggested that it persists in 31% of women. From these studies, it is unclear whether microalbuminuria often normalizes before it later

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**Figure 1.** Histograms of distributions of urinary albumin-to-creatinine ratios in women with and without preeclampsia. The one woman with previous preeclampsia and urinary albumin-to-creatinine ratio of 12 mg/mmol is not included in the histogram.
Table 2. Odds ratios for urinary albumin-to-creatinine ratio above the 75th percentile, according to adverse outcomes in first pregnancy

<table>
<thead>
<tr>
<th>Variable</th>
<th>Participants (n)</th>
<th>Participants with ACR &gt;75th percentile, n (%)&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Unadjusted OR (95% CI)</th>
<th>P Value for Trend</th>
<th>Adjusted for OR (95% CI)&lt;sup&gt;b&lt;/sup&gt;</th>
<th>P Value for Trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preeclampsia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>69</td>
<td>17 (25)</td>
<td>1.0 (reference)</td>
<td>0.87</td>
<td>1.0 (reference)</td>
<td>0.85</td>
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<tr>
<td>Yes</td>
<td>89</td>
<td>23 (26)</td>
<td>1.06 (0.51 to 2.19)</td>
<td>1.08</td>
<td>1.08 (0.50 to 2.33)</td>
<td>0.99</td>
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<tr>
<td>No preeclampsia&lt;sup&gt;c&lt;/sup&gt;</td>
<td>74</td>
<td>20 (27)</td>
<td>1.13 (0.53 to 2.40)</td>
<td>0.99</td>
<td>1.16 (0.52 to 2.59)</td>
<td>0.99</td>
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<tr>
<td>Preterm preeclampsia</td>
<td>14</td>
<td>3 (21)</td>
<td>0.82 (0.20 to 3.28)</td>
<td>0.78</td>
<td>0.78 (0.18 to 3.28)</td>
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<tr>
<td>No preeclampsia</td>
<td>69</td>
<td>17 (25)</td>
<td>1.0 (reference)</td>
<td>1.0</td>
<td>1.0 (reference)</td>
<td></td>
</tr>
<tr>
<td>Preeclampsia without LBW offspring</td>
<td>71</td>
<td>20 (29)</td>
<td>1.20 (0.56 to 2.55)</td>
<td>0.75</td>
<td>1.21 (0.54 to 2.69)</td>
<td>0.78</td>
</tr>
<tr>
<td>Preeclampsia with LBW offspring</td>
<td>18</td>
<td>3 (17)</td>
<td>0.60 (0.16 to 2.33)</td>
<td>0.64</td>
<td>0.64 (0.16 to 2.54)</td>
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</tr>
<tr>
<td>No preeclampsia</td>
<td>69</td>
<td>17 (25)</td>
<td>1.0 (reference)</td>
<td>1.0</td>
<td>1.0 (reference)</td>
<td></td>
</tr>
<tr>
<td>Preeclampsia without SGA offspring&lt;sup&gt;c&lt;/sup&gt;</td>
<td>61</td>
<td>18 (30)</td>
<td>1.29 (0.59 to 2.80)</td>
<td>0.72</td>
<td>1.27 (0.56 to 2.90)</td>
<td>0.78</td>
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<tr>
<td>Preeclampsia with SGA offspring</td>
<td>27</td>
<td>5 (19)</td>
<td>0.68 (0.22 to 2.08)</td>
<td>0.73</td>
<td>0.73 (0.23 to 2.31)</td>
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</tr>
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</table>

ACR, albumin-to-creatinine ratio; OR, odds ratio; CI, confidence interval; LBW, low birthweight (defined as ≤2.5 kg); SGA, small for gestational age (defined as birthweight <10th percentile for gestational age).

<sup>a</sup>Women with urinary albumin-to-creatinine ratio above the 75th percentile (0.70 mg/mmol).

<sup>b</sup>Adjusted for age, body mass index, marital status, annual household income, and highest education level.

<sup>c</sup>Missing information about gestational age for one woman.
Table 3. Odds ratios for estimated GFR above the 75th percentile, according to adverse outcomes in first pregnancy

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total Participants (n)</th>
<th>Participants with eGFR &gt;75th percentile, n (%)&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Unadjusted OR (95% CI)</th>
<th>( P ) Value for Trend</th>
<th>Adjusted OR (95% CI)&lt;sup&gt;b&lt;/sup&gt;</th>
<th>( P ) Value for Trend</th>
</tr>
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<tr>
<td>Preeclampsia</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>69</td>
<td>12 (17)</td>
<td>1.0 (reference)</td>
<td>0.06</td>
<td>1.0 (reference)</td>
<td>0.15</td>
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<tr>
<td>Yes</td>
<td>89</td>
<td>27 (30)</td>
<td>2.07 (0.96 to 4.46)</td>
<td>1.98 (0.79 to 4.95)</td>
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<tr>
<td>No preeclampsia</td>
<td></td>
<td>12 (17)</td>
<td>1.0 (reference)</td>
<td>1.0 (reference)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Term preeclampsia&lt;sup&gt;c&lt;/sup&gt;</td>
<td>74</td>
<td>21 (28)</td>
<td>1.89 (0.84 to 4.20)</td>
<td>0.03</td>
<td>1.79 (0.68 to 4.71)</td>
<td>0.10</td>
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<tr>
<td>Preterm preeclampsia</td>
<td>14</td>
<td>6 (43)</td>
<td>3.56 (1.04 to 12.16)</td>
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<td>2.90 (0.72 to 11.67)</td>
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<tr>
<td>No preeclampsia</td>
<td>69</td>
<td>12 (17)</td>
<td>1.0 (reference)</td>
<td></td>
<td>1.0 (reference)</td>
<td></td>
</tr>
<tr>
<td>Preeclampsia without LBW offspring</td>
<td>71</td>
<td>22 (31)</td>
<td>2.13 (0.96 to 4.75)</td>
<td>0.13</td>
<td>1.94 (0.75 to 5.05)</td>
<td>0.18</td>
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<tr>
<td>Preeclampsia with LBW offspring</td>
<td>18</td>
<td>5 (28)</td>
<td>1.83 (0.55 to 6.09)</td>
<td></td>
<td>2.12 (0.55 to 8.17)</td>
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<tr>
<td>No preeclampsia</td>
<td>69</td>
<td>12 (17)</td>
<td>1.0 (reference)</td>
<td></td>
<td>1.0 (reference)</td>
<td></td>
</tr>
<tr>
<td>Preeclampsia without SGA offspring</td>
<td>61</td>
<td>18 (30)</td>
<td>1.99 (0.87 to 4.56)</td>
<td>0.06</td>
<td>1.62 (0.60 to 4.37)</td>
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<td>Preeclampsia with SGA offspring&lt;sup&gt;c&lt;/sup&gt;</td>
<td>27</td>
<td>9 (33)</td>
<td>2.38 (0.86 to 6.55)</td>
<td></td>
<td>3.16 (0.97 to 10.34)</td>
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</tr>
</tbody>
</table>

eGFR, estimated GFR; OR, odds ratio; CI, confidence interval; LBW, low birthweight (defined as <2.5 kg); SGA, small for gestational age (defined as birth weight <10th percentile for gestational age).

<sup>a</sup>Women with estimated GFR above the 75th percentile (114.3 ml/min per 1.73 m<sup>2</sup>).

<sup>b</sup>Adjusted for age, body mass index, marital status, annual household income, and highest education level.

<sup>c</sup>Missing information about gestational age for one woman.
relapses or whether it is persistently present in a larger subset of preeclamptic women. The mechanisms are unclear; persistent renal damage could explain persistent microalbuminuria, but women with previous preeclampsia have also been shown to have endothelial dysfunction (24,25), another possible mechanism for microalbuminuria (26,27). Endothelial dysfunction is likely to progress with age and total cardiovascular risk factor load; thus, it could be fairly mild during the first decade after preeclampsia but become more important later on in life. It is conceivable that our findings of a low prevalence of microalbuminuria could be explained by our investigating the women at a time in life with only minor endothelial dysfunction and that increasing dysfunction could appear during the next decades.

More women with previous preeclampsia had a high-normal GFR (defined as eGFR greater than the 75th percentile); this was significant for preeclampsia with preterm birth, with a more than three-fold increased risk. This increase in GFR could represent an early stage of hyperfiltration, which has been found to predict the development of microalbuminuria in hypertensive patients (28), is likely to precede renal dysfunction in diabetic nephropathy (29,30), and could be an important marker of early renal disease in obese patients (31). Our findings might indicate an association between preeclampsia and later hyperfiltration, a possible nonalbuminuric sign of early renal dysfunction. However, although the CKD-Epidemiology Collaboration equation has been shown to be the best available estimate of GFR, the formulas are imprecise for high values of eGFR and should be used with caution in healthy individuals (32). In general, however, the imprecision in a formula does tend to reduce the effect in the analyses, and our estimate of the effect on preeclampsia on later GFR may thus be conservative. Our results suggest a need for future studies on the association between measured GFR and preeclampsia.

We have previously found that women with preeclampsia had a four to five times increased risk of ESRD compared with women without preeclampsia (1). The overall rate of ESRD after the first birth was 3.7 per 100,000 women per year (1). On the basis of the increased relative risks of ESRD in women with a history of preeclampsia, we expected to find an association between previous preeclampsia and microalbuminuria. The absence of such an association weakens the hypothesis that preeclampsia itself induces renal damage in a larger proportion of women with preeclampsia. Possible explanations might be that preeclampsia could uncover underlying renal disease in some women progressing to ESRD later in life. Alternatively, renal damage after preeclampsia...
could be rarer than previously expected according to microalbuminuria data, especially in women healthy before pregnancy.

The relatively low participation rate of 44% represents a weakness in this study. However, participation was 49% in women with previous preeclampsia, which is similar to that reported in previous studies (16). Further analyses showed that our participants were more educated than the general population, and, compared with other European studies on preeclampsia and risk of kidney outcomes, our participants had somewhat lower BP but similar body mass index (15–17,33). Some other studies of women with a history of preeclampsia, especially studies from the United States, have, however, shown higher rates of obesity (7,34,35). Furthermore, the participants with previous preeclampsia had a similar prevalence of preterm birth, low-birthweight offspring, and other pregnancy complications compared with the population average, indicating that our finding of a very low prevalence of microalbuminuria could not be explained by our participants having less severe preeclampsia than average. Among the participants, there were also only small differences between the women with and without preeclampsia, with a slightly higher income level among women with previous preeclampsia. A potential selection bias cannot be ruled out, but this seems to be fairly mild, and we do not believe that selection bias can explain the large difference between our study and previous studies.

Strengths of this study include the large number of participants, considerably larger than in previous singular studies, and a thorough and established method for evaluating the main outcome, microalbuminuria. Our main exposure variable, preeclampsia, was well documented; only women with preeclampsia in first pregnancy were included; and the severity of preeclampsia in our women was similar to the population average. Only Norwegian women without diabetes, hypertension, renal disease, or rheumatic disease before first pregnancy were included, and the results should not be applied to other populations without caution.

In conclusion, in our population-based study of otherwise healthy women, preeclampsia was not associated with increased prevalence of microalbuminuria or higher rates of urinary albumin excretion. An additional finding of uncertain importance was a statistically significant association between prerenal preeclampsia and having a high-normal eGFR. It is still uncertain whether preeclampsia itself causes renal damage, but it does not seem to be as common as might have been expected, and may be more pronounced in women with severe preeclampsia. Future studies should address this topic further.

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References


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See related editorial, “Preeclampsia and Subsequent Cardiovascular Disease: Villain or Innocent Bystander?,” on pages 1061–1063.