Cumulative Systolic BP and Changes in Urine Albumin-to-Creatinine Ratios in Nondiabetic Participants of the Multi-Ethnic Study of Atherosclerosis

Paul Zemaitis,* Kiang Liu,† David R. Jacobs, Jr.,‡ Mary Cushman,§ Ramon Durazo-Arvizu,* David Shoam,* Walter Palmas,¶ Richard Cooper,* and Holly Kramer***

Abstract
Background and objectives Cumulative exposure to elevated systolic BP (cumSBP) may affect progression of urine albumin excretion in the absence of diabetes. The objective of this study was to examine the association between cumSBP exposure and progression of spot urine albumin-to-creatinine ratio (UACR) in a multi-ethnic cohort of adults without diabetes.

Design, setting, participants, & measurements The analysis included 3789 participants without severely increased urine albumin excretion or diabetes in the Multi-Ethnic Study of Atherosclerosis, a cohort of 6814 adults aged 45–84 years. UACR was measured at baseline and approximately 1.6, 3.1, and 9.4 years after the baseline examination. cumSBP was calculated as the summed average systolic BP (SBP; mmHg) between two consecutive examinations multiplied by the time between the two examinations (mmHg × year) and categorized as ≤1128 (SBP < 120 mmHg), 1129–1222 (SBP ≥ 120–129 mmHg), 1223–1316 (SBP ≥ 130–139 mmHg), and > 1316 (SBP ≥ 140 mmHg). Baseline UACR was categorized as normal, mildly increased, or moderately increased, and definite progression of UACR was defined as a persistently higher UACR category at subsequent examinations. No UACR progression was defined as remaining in the same UACR category across all examinations or regressing.

Results In fully adjusted models, compared with cumSBP ≤ 1128 mmHg, cumSBP 1223–1316 mmHg was associated with a 85% and 130% significantly higher odds of definite UACR progression (95% confidence interval, 24% to 178% and 56% to 243%, respectively) versus no UACR progression. Every 100-mmHg higher level of cumSBP was associated with a 1.23-fold higher odds of definite UACR progression (95% confidence interval, 1.13 to 1.34) versus no UACR progression.

Conclusion Exposure to higher cumSBP was associated with increased UACR progression among adults without diabetes.

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Introduction
Increased urine albumin excretion may reflect increased glomerular pressure (1,2), which depends on systemic pressures and the kidney’s ability to autoregulate intraglomerular pressure. Cross-sectional studies demonstrate a strong association between elevated systolic BP (SBP) measured at a single time point and levels of urine albumin excretion in adults without diabetes (3–5). Although a few studies have examined the association between baseline SBP and incidence of increased urine albumin excretion in adults without diabetes (6,7), information on how cumulative exposure to higher SBP affects progression of urine albumin excretion remains limited. BP tends to increase with age (8), and future BP depends on multiple factors, including baseline levels, diet, use of antihypertensive therapy, and development of comorbid conditions that may influence BP. Risk for end-organ damage associated with diabetes is clinically assessed via a measure of cumulative glucose exposure, such as hemoglobin A1c or time-averaged fasting glucose levels (9,10). In kidney disease progression, as reflected by progressive increases in urine albumin excretion, cumulative SBP (cumSBP) exposure may represent a mediator of risk and a factor that may be targeted for prevention.

In the Multi-Ethnic Study of Atherosclerosis (MESA) cohort, we examined the association between cumSBP and progression of urinary albumin-to-creatinine ratio (UACR) measured at four separate examinations among adults without clinical cardiovascular disease, diabetes, or severely increased urine albumin excretion at baseline. We hypothesized that higher levels of cumSBP are associated with greater concurrent progression of UACR.

Materials and Methods
Population
MESA is a prospective cohort study of 6814 men and women aged 45–84 years without clinical cardiovascular
disease (CVD) at baseline, recruited from six United States communities (Baltimore, MD; Chicago, IL; Forsyth County, NC; Los Angeles County, CA; northern Manhattan, NY; and St. Paul, MN). MESA was designed to determine the characteristics of subclinical CVD and its progression. Adults with symptoms or history of medical or surgical treatment for CVD were excluded. Information on the sampling frame and study design was previously reported (11).

Participants who self-reported their race/ethnicity group as Caucasian or white, African-American or black, Chinese, or Spanish/Hispanic/Latino were asked to participate and were enrolled and examined between July 2000 and August 2002. Participants then completed four additional examinations after a median follow-up time of 1.6, 3.1, 5.1, and 9.4 years. Institutional review board approval was obtained at all MESA sites, and informed consent was obtained from all participants.

Spot urine samples were collected at baseline and again after a median (interquartile range) follow-up time of 1.6 (1.4–1.8), 3.1 (2.0–4.9), and 9.4 (8.0–11.4) years. Participants with missing urine samples at any of these time points were excluded (n=2086). We also excluded 859 participants with baseline diabetes, 31 participants with severely increased urine albumin excretion at baseline (see definition below), and 49 participants with missing data on SBP for at least one time point. This left a total of 3789 MESA participants without severely increased urine albumin excretion or diabetes at baseline with complete data on UACR and cumSBP for the analysis (Figure 1).

To account for the missing data on UACR, we performed sensitivity analyses. The first sensitivity analysis focused on the association between cumSBP and UACR progression in the 4995 MESA participants with complete information on cumSBP and UACR for the first three examinations. In the second sensitivity analysis, we examined UACR as a continuous variable using mixed-effects models; this analysis included data from 5865 participants without baseline diabetes or severely increased urine albumin excretion and with complete information on baseline UACR and at least one follow-up UACR value.

**UACR**

Spot urine specimens were collected in the morning immediately after the participant arrived for each examination. Urine albumin and creatinine were measured at the Clinical Chemistry Laboratory at Fletcher Allen Health Care (Burlington, VT). Urinary creatinine was measured using the Vitros 950IRC instrument (Johnson & Johnson Clinical Diagnostics, Inc., Rochester, NY) using the Jaffe method. Thin film technology was used to quantitatively measure creatinine via a colorimetric reaction. The assay range was 0.05–16.5 mg/dl with an analytical coefficient of variation range of 2.5%–2.9%. Urinary albumin was determined using the Array 360 CE Protein Analyzer (Beckman Instruments, Inc., Drea, CA). This system uses a nephelometer to measure the rate of light scatter formation resulting from an immunoprecipitation reaction. The minimum detectable level of albumin was 0.2 mg/dl.

To account for differences in creatinine excretion between men and women, sex-specific UACR cut-points were used to define levels of moderately increased urine albumin excretion ($\geq 17–249$ mg/g in men and $\geq 25–354$ mg/g in women) and severely increased urine albumin excretion ($\geq 250$ in men and $\geq 355$ mg/g in women) (12,13). Normal UACR was defined as $<9$ mg/g in men and $<13$ mg/g in women, and mildly increased UACR was considered $9–16.9$ mg/g and $13–24.9$ mg/g in men and women, respectively. These UACR cut-points represent the midpoint of distribution of UACR below the threshold for defining moderately increased urine albumin excretion (14,15).

To examine progression of sex-adjusted UACR, three categories of UACR progression were created: no UACR progression, intermediate UACR progression, and definite UACR progression. Participants with no UACR progression remained in the same UACR category (normal or high
normal) across the four examinations (examination 1, 2, 3, and 5) or improved to a lower UACR category. Definite UACR progression was defined as going to a higher UACR category at examination 2 or 3 (e.g., from normal UACR at baseline to mildly, moderately, or severely increased UACR at examination 2 or 3 or from mildly increased UACR at baseline to moderately or severely increased UACR at examination 2 or 3) and then either remaining in this higher UACR category or progressing to an even higher category at examination 5. Participants who did not fit either of these two categories were categorized as having intermediate UACR progression (16). The median (interquartile range) difference in UACR between the first and last measurement (examination 5 UACR—examination 1 UACR) was 0.12 (−1.59 to 1.91) mg/g in the no-UACR-progression category, 6.6 (0.5–14.8) mg/g in the intermediate-UACR-progression category, and 16.7 (8.93–16.7) mg/g in the definite-UACR-progression category.

\[ \text{cumSBP} = \frac{(SBP_1 + SBP_2)/2 \times \text{time1} - 2 + (SBP_2 + SBP_3)/2 \times \text{time2} - 3 + (SBP_3 + SBP_4)/2 \times \text{time3} - 4 + (SBP_4 + SBP_5)/2 \times \text{time4} - 5}{n} \]

where SBP1, SBP2, SBP3, SBP4, and SBP5 indicate SBP at examinations 1 (baseline), 2, 3, 4, and 5 and time1–2, time2–3, time3–4, time4–5, indicate the participant-specific time interval between consecutive examinations 1–5 in years. cumSBP was categorized in mmHg as <1128 (<120 mmHg×9.4 years), 1128–1222 (≥120–130 mmHg×9.4 years), 1223–1316 (≥130–139 mmHg×9.4 years), and ≥1317 (≥140 mmHg×9.4 years). cumSBP was also examined as a continuous variable centered at a cumSBP of 1128 mmHg.

Covariates

All MESA participants completed self-administered questionnaires, provided fasting blood samples, and were interviewed and examined by trained research staff. Self-administered questionnaires were available in English, Spanish, and Chinese and were completed at each examination. Presence of diabetes was defined as self-reported physician diagnosis, use of insulin or oral hypoglycemic agents, or fasting glucose ≥126 mg/dl. Waist circumference was measured midway between the lowest rib and the iliac crest using a tape measure. Information on socioeconomic factors, including highest degree or level of school completed and health insurance (private health insurance, health maintenance organization, Medicaid, Medicare, veterans health care, or none), was collected from the questionnaires. Use of antihypertensive medication was defined as self-reported treatment for hypertension with one of six common classes of antihypertensive medications at each of the five examinations.

Statistical Analyses

Characteristics across the cumSBP categories are shown as means±SD for continuous variables, frequency (percentage) for categorical variables, and median (interquartile range [IQR]) for variables with non-normal distributions (UACR). Multinomial logistic regression was used to calculate the adjusted odds of definite and intermediate UACR progression compared with no UACR progression according to cumSBP while simultaneously adjusting for covariates (Stata software, version 11.0; Stata Corp., College Station, TX). Several models were created to examine the effects of covariates, especially new-onset diabetes. Model 1 adjusted for age, sex, race/ethnicity, site, and baseline UACR to establish the minimally adjusted relationship between cumSBP and UACR progression. Model 2 added use of BP-lowering medications at baseline or at any follow-up examination. Model 3 then further adjusted for baseline waist circumference, smoking status, education, and health insurance. Model 4 added incident diabetes at any of the follow-up examinations to Model 3. In all models, age, baseline UACR, and waist circumference were fitted as continuous variables, whereas race, site, sex, baseline current smoking, education, health insurance, hypertensive medication use, and incident diabetes at any of the follow-up examinations were fitted as categorical variables. Interaction terms between incident diabetes at examinations 2, 3, or 5 and cumSBP on UACR progression were fitted in the full model to explore effect modification by incident diabetes.

In the first sensitivity analysis, we limited the analysis to participants with complete data on BP and UACR over the first three examinations (n=4995). UACR progression was then based on UACR values measured over the first three examinations. The same multinomial logistic regression models (Models 1, 2, 3, and 4) were fitted as the main analysis. The second sensitivity analysis analyzed sex-adjusted UACR as a continuous variable using mixed-effects models fitted with random intercepts (baseline UACR for each individual). In this analysis, we included MESA participants without severely increased urine albumin excretion or diabetes at baseline but participants had at least one follow-up UACR measurement (n=5865). UACR for each time point was first log-transformed because of the skewed distribution. The same models (Models 1, 2, 3, and 4) were then fitted using mixed-effects models. Differences in slope of log UACR was determined by cumSBP categories with the lowest category as the referent group with simultaneous adjustment for covariates and random intercepts.

Results

Baseline Characteristics

Overall, cumSBP in mmHg was <1128 in 47%, 1129–1222 in 23%, 1223–1316 in 15%, and ≥1317 in 15% (Table 1). Participants in the lowest cumSBP category were on average younger (57.8 years) than participants in the higher cumSBP categories (64.4 years in highest cumSBP category). The rate of new-onset diabetes ranged from 10.5% in the lowest cumSBP category to 16.9% in the highest cumSBP category. Baseline UACR values increased gradually across cumSBP categories from 3.9 (IQR, 2.8–6.4) mg/g in lowest to 6.3 (IQR, 3.9–13.6) mg/g in the
highest category. Figure 2 shows the distribution of SBP for each of the five MESA examinations by cumSBP categories. Across all five examinations, SBP in most individuals in the two lowest cumSBP categories (<122 mmHg) was <130 mmHg. In the highest cumSBP category (≥1317 mmHg), SBP was <130 mmHg in <20% for each examination. Figure 3 shows the frequency of definite and intermediate UACR progression by cumSBP categories. As cumSBP increased, the frequency of both definite and intermediate UACR progression increased.

Table 2 shows the adjusted odds of definite or intermediate UACR progression by cumSBP categories. Compared with cumSBP<1128 mmHg, cumSBP 1128–1222 mmHg was not significantly associated with definite UACR progression in any model but was significantly associated with a 1.47-fold (95% confidence interval [95% CI], 1.19 to 1.83) higher odds of intermediate UACR progression after adjustment for all covariates. Compared with the lowest cumSBP category, after adjustment for all covariates, a cumSBP of 1223–1316 mmHg was associated with a 1.85-fold higher odds of definite UACR progression (95% CI, 1.24 to 2.78) while cumSBP≥1317 mmHg was associated with a 2.3-fold higher odds of definite UACR progression (95% CI, 1.56 to 3.43). The associations were not substantially attenuated with adjustment for incident diabetes during the follow-up period. When cumSBP was examined as a continuous variable, every 100 mmHg higher level of cumSBP was associated with a 1.23-fold increase in the frequency of both definite and intermediate UACR progression by cumSBP categories compared with the lowest category as noted in the cumSBP category ≥1317 mmHg (slope, 0.034; P<0.001).

Discussion

In MESA, higher cumulative SBP exposure was associated with significantly higher odds of UACR progression and higher change in log UACR per year. Adjusting for development of type 2 diabetes or use of antihypertensive medications at each follow-up examination did not substantially attenuate these associations. These findings provide additional evidence that urine albumin excretion progresses during long-term exposure to higher SBP. Previous studies have demonstrated a direct correlation between UACR and left ventricular hypertrophy (14,17). Given that increased SBP remains an important risk factor for left ventricular hypertrophy (18), cumulative SBP exposure may link increased urine albumin excretion with elevated left ventricular mass and also with heightened cardiovascular risk and kidney disease progression (19,20).

Use of longitudinal changes in UACR remains poorly explored in clinical studies. Schmieder et al. used data from 23,480 adults with vascular disease or diabetes to examine

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>&lt;1128 mmHg (SBP &lt; 120 × 9.4 yr) (n=1782)</th>
<th>1128–1222 mmHg (SBP ≥120–129 × 9.4 yr) (n=881)</th>
<th>1223–1316 mmHg (SBP ≥130–139 × 9.4 yr) (n=553)</th>
<th>≥1317 mmHg (SBP ≥140 × 9.4 yr) (n=573)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>58±9</td>
<td>60±9</td>
<td>62±9</td>
<td>64±9</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>112±14</td>
<td>126±15</td>
<td>132.5±16.0</td>
<td>148±19</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>68±9</td>
<td>74±9</td>
<td>75±10</td>
<td>78±10</td>
</tr>
<tr>
<td>Women (%)</td>
<td>51.2</td>
<td>50.7</td>
<td>52.5</td>
<td>64.4</td>
</tr>
<tr>
<td>White (%)</td>
<td>49.8</td>
<td>39.7</td>
<td>40.5</td>
<td>34.2</td>
</tr>
<tr>
<td>African-American (%)</td>
<td>19.6</td>
<td>28.1</td>
<td>27.7</td>
<td>30.5</td>
</tr>
<tr>
<td>Chinese (%)</td>
<td>11.6</td>
<td>11.2</td>
<td>10.8</td>
<td>13.6</td>
</tr>
<tr>
<td>Hispanic (%)</td>
<td>18.9</td>
<td>20.9</td>
<td>21.0</td>
<td>21.6</td>
</tr>
<tr>
<td>ACR (mg/g)</td>
<td>3.9 (2.8–6.4)</td>
<td>4.8 (3.3–8.3)</td>
<td>5.0 (3.3–9.6)</td>
<td>6.3 (3.9–13.6)</td>
</tr>
<tr>
<td>eGFR (ml/min per 1.73 m²)</td>
<td>85.8±14.2</td>
<td>84.7±15.8</td>
<td>81.4±15.4</td>
<td>80.7±15.5</td>
</tr>
<tr>
<td>Waist in men (cm)</td>
<td>97±11</td>
<td>100±12</td>
<td>101±12</td>
<td>100±13</td>
</tr>
<tr>
<td>Waist in women (cm)</td>
<td>92±16</td>
<td>97±15</td>
<td>98±15</td>
<td>98±14</td>
</tr>
<tr>
<td>Current smoker (%)</td>
<td>11.7</td>
<td>11.4</td>
<td>12.3</td>
<td>11.3</td>
</tr>
<tr>
<td>Incident DM (%)</td>
<td>10.5</td>
<td>16.2</td>
<td>16.6</td>
<td>16.9</td>
</tr>
<tr>
<td>Less than high school education (%)</td>
<td>8.9</td>
<td>12.6</td>
<td>16.1</td>
<td>18.2</td>
</tr>
<tr>
<td>Health insurance (%)</td>
<td>93.1</td>
<td>91.9</td>
<td>94.2</td>
<td>91.1</td>
</tr>
</tbody>
</table>

Data shown as mean±SD or frequency (percentage). ACR data are median (interquartile range). eGFR was calculated with the CKD-Epidemiology Collaboration equation. Incident DM defined as fasting glucose ≥126 mg/dl and/or use of glucose-lowering medications at the second or third follow-up exam among participants without baseline diabetes. SBP, systolic BP; DBP, diastolic BP; ACR, albumin-to-creatinine ratio; DM, diabetes mellitus.
whether changes in UACR over 2 years predict mortality and morbidity (21). They found that patients with a 2-fold increase in UACR from baseline, which was observed in almost one third of the study participants, had a 48% higher risk of death compared with those who did not have this level of change in UACR. It is possible that the association between change in UACR and mortality in this study may be a function of the association between UACR
progression and cumSBP exposure. The Action to Control Cardiovascular Risk in Diabetes trial, which focused on adults with type 2 diabetes, showed that lowering SBP to a goal of ≤120 mmHg versus <140 mmHg was associated with a reduced risk of moderately increased urine albumin excretion and stroke (22). However, cardiovascular events were not reduced with BP lowering to SBP <120 mmHg. This may be due to the presence of BP autoregulation in the brain and kidney but not in the heart (23). The ongoing Systolic Blood Pressure Intervention Trial is examining whether an SBP goal of <120 mmHg versus <140 mm Hg is associated with reduced risk of cardiovascular events, memory loss, and progression of CKD in adults without diabetes (24). This trial will also provide information on whether intensive BP-lowering reduces urine albumin excretion in hypertensive adults without diabetes at baseline and whether this reduction in urine albumin excretion is associated with reduced CVD risk.

Urine albumin excretion varies widely among patients (25,26). To address the intraindividual variability in UACR, this study used UACR progression categories. These progression categories account for the fact that variability in UACR across time depends on baseline values (individuals who start with high values will show large changes and individuals with low values show low changes) and our analysis adjusted for baseline UACR. Redon et al. followed 187 adults with newly diagnosed hypertension in the absence of diabetes or moderately increased urine albumin excretion for an average of 2.7 years and examined factors associated with development of moderately increased urine albumin excretion (6). Overall, 11.7% developed moderately increased urine albumin excretion. While baseline SBP was not associated with development of moderately increased urine albumin excretion, the slope of SBP over time was associated with the slope of urine albumin excretion over time. Focusing solely on development of moderately increased urine albumin excretion ignores small changes in UACR among individuals who have normal or mildly increased UACR. Small changes in UACR may be clinically relevant given

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**Table 2. Odds of urine albumin/creatinine ratio progression by cumulative systolic blood pressure categories**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definite UACR progression</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1128 mmHg (SBP&lt;120×9.4 yr)</td>
<td>1.00 (Referent)</td>
<td>1.00 (Referent)</td>
<td>1.00 (Referent)</td>
<td>1.00 (Referent)</td>
</tr>
<tr>
<td>1128–1222 mmHg (SBP≥120–129×9.4 yr)</td>
<td>1.64 (1.30 to 2.04)</td>
<td>1.70 (1.31 to 2.19)</td>
<td>1.70 (1.32 to 2.20)</td>
<td>1.71 (1.32 to 2.21)</td>
</tr>
<tr>
<td>1223–1316 mmHg (SBP≥120–129×9.4 yr)</td>
<td>1.67 (1.32 to 2.11)</td>
<td>1.64 (1.28 to 2.11)</td>
<td>1.63 (1.28 to 2.10)</td>
<td>1.65 (1.29 to 2.13)</td>
</tr>
<tr>
<td>≥1317 mmHg (SBP≥140×9.4 yr)</td>
<td>1.83 (1.45 to 2.31)</td>
<td>1.70 (1.32 to 2.19)</td>
<td>1.70 (1.32 to 2.20)</td>
<td>1.71 (1.32 to 2.21)</td>
</tr>
</tbody>
</table>

**Table 3. Adjusted difference in log urinary albumin-to-creatinine ratio slopes by cumulative systolic BP categories**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1128 mmHg (SBP&lt;120×9.4 yr)</td>
<td>Referent</td>
<td>Referent</td>
<td>Referent</td>
<td>Referent</td>
</tr>
<tr>
<td>1128–1222 mmHg (SBP≥120–129×9.4 yr)</td>
<td>0.006 (0.004)</td>
<td>0.012 (0.004)a</td>
<td>0.012 (0.004)b</td>
<td>0.012 (0.004)b</td>
</tr>
<tr>
<td>1223–1316 mmHg (SBP≥120–129×9.4 yr)</td>
<td>0.018 (0.005)b</td>
<td>0.024 (0.005)a</td>
<td>0.024 (0.005)a</td>
<td>0.024 (0.005)a</td>
</tr>
<tr>
<td>≥1317 mmHg (SBP≥140×9.4 yr)</td>
<td>0.031 (0.005)b</td>
<td>0.034 (0.005)a</td>
<td>0.034 (0.005)a</td>
<td>0.034 (0.005)a</td>
</tr>
</tbody>
</table>

Values are expressed as mean (SEM). Model 1 is adjusted for age, race/ethnicity, sex, site and baseline urinary albumin-to-creatinine ratio. Model 2 consists of Model 1 with BP treatment at baseline and for each of the follow-up examinations. Model 3 adds waist circumference, cigarette smoking status, education, and health insurance to Model 2. Model 4 consists of Model 3 with incident diabetes during the follow-up period.

p<0.001.

p<0.01.
that numerous studies have shown a fairly linear association between UACR > 10 mg/g and CVD outcomes and mortality (19, 20, 27).

One of the limitations of this study is differences in follow-up time between visits. Due to the wider time interval between examinations 3 and 5 versus examinations 1, 2 and 3, SBP measured at examinations 3 and 5 gave more weight to the cumSBP exposure than SBP at earlier examinations. By necessity we excluded participants with missing UACR at any time point, but sensitivity analyses were completed using mixed effect models that included participants with missing UACR data at examinations after the baseline exam. Most studies examining the progression of urine albumin excretion do not have more than two measurements (baseline and follow-up), which may give misleading findings given the large intrindividual variability of urine albumin excretion (25). Regardless of these limitations, the findings of this study demonstrate the importance of encouraging antihypertensive medication compliance to decrease the long-term exposure to higher SBP for prevention of kidney disease progression. These findings also have implications for BP goals for young adults with hypertension (28).

In conclusion, higher cumSBP exposure increases the risk of UACR progression, and this association is independent of incident diabetes. Ongoing clinical trials, such as the Systolic Blood Pressure Lowering Trial, will provide more definitive data on whether aggressive BP lowering in adults without diabetes reduces UACR progression and whether this reduction in UACR is associated with a reduction in CVD events.

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Disclosures

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