Effects of Intensive Blood Pressure Control in Patients with and without Albuminuria

Post Hoc Analyses from SPRINT

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Abstract

Background and objectives It is unclear whether the presence of albuminuria modifies the effects of intensive systolic BP control on risk of eGFR decline, cardiovascular events, or mortality.

Design, setting, participants, & measurements The Systolic Blood Pressure Intervention Trial randomized nondiabetic adults ≥50 years of age at high cardiovascular risk to a systolic BP target of <120 or <140 mm Hg, measured by automated office BP. We compared the absolute risk differences and hazard ratios of ≥40% eGFR decline, the Systolic Blood Pressure Intervention Trial primary cardiovascular composite outcome, and all-cause death in those with or without baseline albuminuria (urine albumin-creatinine ratio ≥30 mg/g).

Results Over a median follow-up of 3.1 years, 69 of 1723 (4%) participants with baseline albuminuria developed ≥40% eGFR decline compared with 61 of 7162 (1%) participants without albuminuria. Incidence rates of ≥40% eGFR decline were higher in participants with albuminuria (intensive, 1.74 per 100 person-years; standard, 1.17 per 100 person-years) than in participants without albuminuria (intensive, 0.48 per 100 person-years; standard, 0.11 per 100 person-years). Although effects of intensive BP lowering on ≥40% eGFR decline varied by albuminuria on the relative scale (hazard ratio, 1.48; 95% confidence interval, 0.91 to 2.39 for albumin-creatinine ratio ≥30 mg/g; hazard ratio, 4.55; 95% confidence interval, 2.37 to 8.75 for albumin-creatinine ratio <30 mg/g; *P* value for interaction <0.001), the absolute increase in ≥40% eGFR decline did not differ by baseline albuminuria (incidence difference, 0.38 events per 100 person-years for albumin-creatinine ratio ≥30 mg/g; incidence difference, 0.58 events per 100 person-years for albumin-creatinine ratio <30 mg/g; *P* value for interaction =0.60). Albuminuria did not significantly modify the beneficial effects of intensive systolic BP lowering on cardiovascular events or mortality evaluated on relative or absolute scales.

Conclusions Albuminuria did not modify the absolute benefits and risks of intensive systolic BP lowering.

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Introduction

The Systolic Blood Pressure Intervention Trial (SPRINT) was terminated early due to clear evidence of benefit of intensive systolic BP lowering (1). Intensive systolic BP lowering to a target systolic BP <120 mm Hg (compared with <140 mm Hg) reduced the risk of the composite cardiovascular disease outcome by 25% and reduced the risk of death by 27%. SPRINT participants in the intensive systolic BP lowering group also experienced higher risks of eGFR decline and incident CKD, although this effect was more likely hemodynamic with unclear long-term consequences (1–3). Albuminuria defined as ≥30 mg/g is associated with heightened risk of cardiovascular disease, kidney failure, and death (4). It is currently uncertain whether the effects of intensive systolic BP lowering on cardiovascular risk and eGFR decline vary by albuminuria status.

In prespecified analysis of the African American Study of Kidney Disease and Hypertension (AASK) and secondary analysis of the Modification of Diet in Renal Disease (MDRD) trial, proteinuria modified the effect of BP lowering to a target mean arterial pressure <92 mm Hg (compared with 102–107 mm Hg) on the risk of progression of kidney disease (5,6). In these trials, intensive BP lowering reduced the risk of CKD progression in those with proteinuria (defined as >1 g/d in the MDRD trial and ≥0.22 g/g protein-creatinine ratio in the AASK), with no effect on those without baseline proteinuria. However, meta-analyses examining the effects of intensive BP lowering on CKD progression have come to differing conclusions, depending on whether long-term follow-up data are used (7,8).

Because individuals with albuminuria are at elevated risk of eGFR decline, it is important to understand

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whether the potential benefits and risks of intensive systolic BP lowering differ by baseline albuminuria to help clinicians weigh potential risks and benefits and promote shared decision making. Our objectives were to examine whether the effects of intensive systolic BP lowering on risk of cardiovascular disease or eGFR decline varied by baseline albuminuria level and to examine absolute risk differences between the intensive and standard systolic BP arms among those with and without elevated baseline albuminuria.

Materials and Methods

Study Design and Setting

The SPRINT was a randomized, controlled, open-label trial with blinded end point determination conducted at 102 United States clinical sites, with the primary objective of determining whether a lower systolic BP goal <120 mm Hg would reduce clinical events more than a standard goal; full details of trial methods, including the study protocol, can be found in the main publication (1). In brief, participants were ≥50 years of age, had systolic BP of 130–180 mm Hg, and had an increased risk of cardiovascular disease (one or more of the following: clinical or subclinical cardiovascular disease other than stroke, eGFR=20–60 ml/min per 1.73 m^2, 10-year risk of cardiovascular disease ≥15% using the Framingham Risk Score, and age ≥75 years). Main exclusion criteria included history of diabetes, stroke, proteinuria >1 g/d, polycystic kidney disease, symptomatic heart failure, or left ventricular ejection fraction <35%. The study was approved by institutional review boards at all participating institutions, and written informed consent was obtained from all participants. Eligible participants were randomized 1:1 to a systolic BP target of <120 mm Hg (intensive) or <140 mm Hg (standard). BP was defined as the mean of three BP readings measured at study clinic visits (1-minute intervals after a 5-minute rest period) using an automated device (Omron HEM-907XL; Omron Healthcare, Lake Forest, IL) and has been previously described (9). BP medications were adjusted by site investigators according to study protocol algorithms to achieve and maintain systolic BP in the assigned target range (1). Albuminuria was estimated by a random urine albumin-creatinine ratio (UACR). Serum creatinine and urine creatinine were measured using an enzymatic method (Roche, Indianapolis, IN) at the SPRINT central laboratory at the University of Minnesota. Serum creatinine specimens were obtained at study visits for the first 3 months and then quarterly; eGFR was calculated using the four-variable MDRD equation (10). Urine albumin was measured using a nephelometric method (Siemens, Tarrytown, NY).

Definition of Kidney Outcomes

A simulation study published after the SPRINT protocol was finalized supports ≥40% decline in eGFR as a surrogate marker for risk of progression to kidney failure (11). Hence, we used this as the primary kidney outcome in a *post hoc* analysis. For this outcome, we defined eGFR decline events as requiring at least two consecutive qualifying values or a single qualifying value at the last trial study visit. In addition, we conducted sensitivity analyses with eGFR decline ≥30% as well as SPRINT protocol–defined kidney end points (1). The main secondary kidney outcome, applicable for the CKD subgroup, was a composite of 50% decrease in eGFR or development of kidney failure requiring maintenance dialysis or kidney transplantation. An additional secondary kidney outcome was incident CKD, applicable to the non-CKD subgroup, and defined as >30% decline in eGFR with a confirmed value <60 ml/min per 1.73 m^2. However, there were only 31 kidney composite events in the CKD subgroup. Therefore, effect modification by baseline albuminuria could not be tested for this particular kidney outcome.

Cardiovascular and All-Cause Mortality Outcomes

Medical records were obtained to document events, and a committee, unaware of treatment assignment, adjudicated protocol-specified clinical outcomes. The primary composite outcome included nonfatal myocardial infarction, acute coronary syndrome, nonfatal acute decompensated heart failure, nonfatal stroke, and death from cardiovascular causes. We also examined all-cause death and the primary cardiovascular disease composite outcome or all-cause death.

For all analyses, we used the main trial data with follow-up until August 20, 2015, when the intervention was terminated due to a significantly lower rate of the primary composite outcome in the intensive group than in the standard group (1).

Statistical Analyses

Baseline characteristics were summarized for the full cohort and in subgroups with or without albuminuria by mean (SD) for numerical variables, median (25%, 75% percentile) for skewed variables, and *N* (percentage) for categorical variables. We compared numeric baseline characteristics using *t* test or Wilcoxon rank-sum test and categorical variables using chi-squared tests between groups with or without albuminuria. As recommended by the Consolidated Standards of Reporting Trials 2010 guideline, we examined both the absolute and relative effects of intensive systolic BP lowering (12).

Comparison of Absolute Risk Differences of the Effects of Intensive Systolic Blood Pressure Lowering in Groups with or without Albuminuria. We calculated incidence rates per 100 person-years for study outcomes in the intensive and standard interventions by groups with or without albuminuria; then, we estimated absolute incidence rate differences between the intensive and standard interventions and computed the difference in incidence rate difference and 95% confidence intervals (95% CIs) between groups with and without albuminuria. Analyses of kidney and nonfatal cardiovascular disease outcomes were performed using competing-risks models, in which non-cardiovascular disease death was treated as a competing risk for the cardiovascular disease outcomes and all-cause death was treated as a competing risk for kidney outcomes (13). Then, we tested the interactions of absolute incidence rate differences between groups with and without baseline albuminuria by comparing differences in the estimated incidence rate differences with the SEM of the differences (14). The study outcomes included ≥40% eGFR

decline, primary cardiovascular disease outcome, all-cause death, and primary cardiovascular disease outcome or death.

Comparison of Relative Risk of the Effects of Systolic Blood Pressure Lowering in Groups with or without Albuminuria. We used Cox proportional hazards models to estimate the effects of intensive systolic BP lowering. We tested the interactions of effects of intensive systolic BP lowering with presence of baseline albuminuria using likelihood ratio tests.

In sensitivity analyses, we repeated the same analyses for the outcomes by baseline CKD subgroups. We also conducted similar analyses for SPRINT protocol–defined kidney outcomes (incident CKD in the baseline non-CKD subgroup and the CKD composite outcome in the baseline CKD subgroup), with all-cause death treated as a competing risk.

We performed all analyses using STATA version MP 15.1 or SAS version 9.4, and we used two-sided $\alpha=0.05$ for hypothesis testing without adjustment for multiple comparisons.

Results

Study Participants

A total of 8885 (95%) of 9361 participants had baseline UACR and eGFR data and were included in this analysis. Baseline prevalence of albuminuria was 19%, whereas prevalence of eGFR<60 ml/min per 1.73 m^2 was 29%. Presence of albuminuria was associated with older age; lower eGFR; higher systolic BP; higher prevalence of cardiovascular disease and eGFR<60 ml/min per 1.73 m^2; and higher use of aspirin, statin, and antihypertensive medications (Table 1).

Effects of Intensive Systolic Blood Pressure Lowering on ≥40% Estimated Glomerular Filtration Rate Decline in Participants with and without Albuminuria

Over a median follow-up time of 3.1 (25th to 75th percentile =2.7–3.7) years, incidence rates of ≥40% eGFR decline in the intensive versus standard systolic BP arms in those without albuminuria were 0.48 versus 0.11 events per 100 person-years, with an absolute risk difference of 0.38 events per 100 person-years (95% CI, 0.23 to 0.52) (Figure 1A, Table 2). The corresponding incidence rates were much higher in those with albuminuria (1.74 versus 1.17), with an absolute risk difference of 0.58 events per 100 person-years (95% CI, −0.11 to 1.26). However, absolute risk differences for intensive versus standard BP arms in those with albuminuria compared with those without albuminuria were not different ($P=0.60$).

In contrast, in the time to event analysis, the interaction P value comparing the corresponding regression coefficients of intensive versus standard systolic BP arms in those without versus with albuminuria was significantly different ($P<0.001$) (Table 2). Intensive systolic BP lowering resulted

Table 1. Characteristics of participants in the Systolic Blood Pressure Intervention Trial according to baseline urine albumin-creatinine ratio

Characteristics	Urine Albumin-Creatinine Ratio <30 mg/g	Urine Albumin-Creatinine Ratio ≥30 mg/g
n	7162	1723
UACR, mg/g	8 (5–13)	70 (42–162)
Criterion for increased cardiovascular risk, *n* (%)		
Age≥75 yr	1852 (26)	667 (39)
GFR<60 ml/min per 1.73 m^2	1735 (24)	818 (47)
Cardiovascular disease	1378 (19)	421 (24)
Clinical	1064 (15)	335 (19)
Subclinical	314 (4)	86 (5)
Framingham 10-yr cardiovascular risk score	24 (12)	29 (14)
Age, yr	67 (9)	70 (10)
Women, *n* (%)	2559 (36)	576 (33)
Race, *n* (%)		
Hispanic	785 (11)	153 (9)
Non-Hispanic Black	2130 (30)	552 (32)
Non-Hispanic White	4112 (57)	989 (57)
Other	135 (2)	29 (2)
Smoking, *n* (%)		
Current	955 (13)	232 (13)
Former	3004 (42)	781 (45)
Never	3196 (45)	707 (41)
Systolic BP, mm Hg	139 (15)	144 (17)
Diastolic BP, mm Hg	78 (12)	78 (13)
BMI, kg/m^2	29.9 (5.7)	29.7 (6.1)
eGFR, ml/min per 1.73 m^2	74 (19)	63 (24)
Statin use, *n* (%)	3052 (43)	833 (48)
Aspirin use, *n* (%)	3751 (52)	923 (54)

Values reported as mean (SD) or *N* (%) except for UACR, which is reported as median (25th percentile to 75th percentile). There are 11 missing values for Framingham 10-year cardiovascular risk score, 53 missing values for BMI, and 10 missing values for smoking status. UACR, albumin-creatinine ratio; BMI, body mass index.

in increased risk of ≥40% eGFR decline among those without albuminuria (hazard ratio, 4.55; 95% CI, 2.37 to 8.75). Because the baseline hazard of ≥40% eGFR decline in the standard arm in those with albuminuria was already high, the hazard ratio comparing ≥40% eGFR decline in the intensive versus standard arm was not significant (hazard ratio, 1.48; 95% CI, 0.91 to 2.39) (Table 2).

Sensitivity Analyses of Kidney Outcomes

Patterns of absolute risk differences and hazard ratios for ≥30% eGFR decline for intensive versus standard systolic BP arms in those without and with albuminuria mirrored the above results for ≥40% eGFR decline (Supplemental Table 1). For SPRINT protocol prespecified kidney outcomes of incident CKD in the non-CKD subgroup and kidney composite outcome in the CKD subgroup, there was no evidence that the presence or absence of albuminuria modified the effects of the intensive systolic BP lowering (Supplemental Table 1).

Effects of Intensive Systolic Blood Pressure Lowering on Primary Cardiovascular Disease Outcome, All-Cause Death, and Primary Cardiovascular Disease Outcome/All-Cause Death in Participants with and without Albuminuria

The incidence rates, corresponding absolute risk differences, and hazard ratios for the primary cardiovascular disease outcome, all-cause death, and primary cardiovascular disease outcome or all-cause death in those without and with albuminuria are summarized in Figure 1, B–D and Table 2. In general, intensive systolic BP intervention appeared to lower the risk of these events irrespective of the presence of albuminuria, with all interaction *P* values nonsignificant.

Non-CKD and CKD Subgroups Analyses

When the above analyses of ≥40% eGFR decline, cardiovascular disease outcome, and all-cause deaths were repeated in the non-CKD and CKD subgroups, the results were similar to the main analyses (Supplemental Tables 2 and 3).

Discussion

In this *post hoc* SPRINT analysis, we found that intensive systolic BP lowering resulted in similar absolute effects on ≥40% eGFR decline in those with and without albuminuria. This observation was in contrast to our expectation that the effects of intensive systolic BP lowering on ≥40% eGFR decline may be augmented in those with albuminuria, given higher baseline risk among those with albuminuria. In time to event analyses, intensive systolic BP lowering resulted in a stronger relative risk of ≥40% eGFR decline in participants without albuminuria compared with those with albuminuria. In terms of cardiovascular events and all-cause mortality, intensive systolic BP lowering reduced risk similarly in patients with and without albuminuria on both the absolute and relative risk scales.

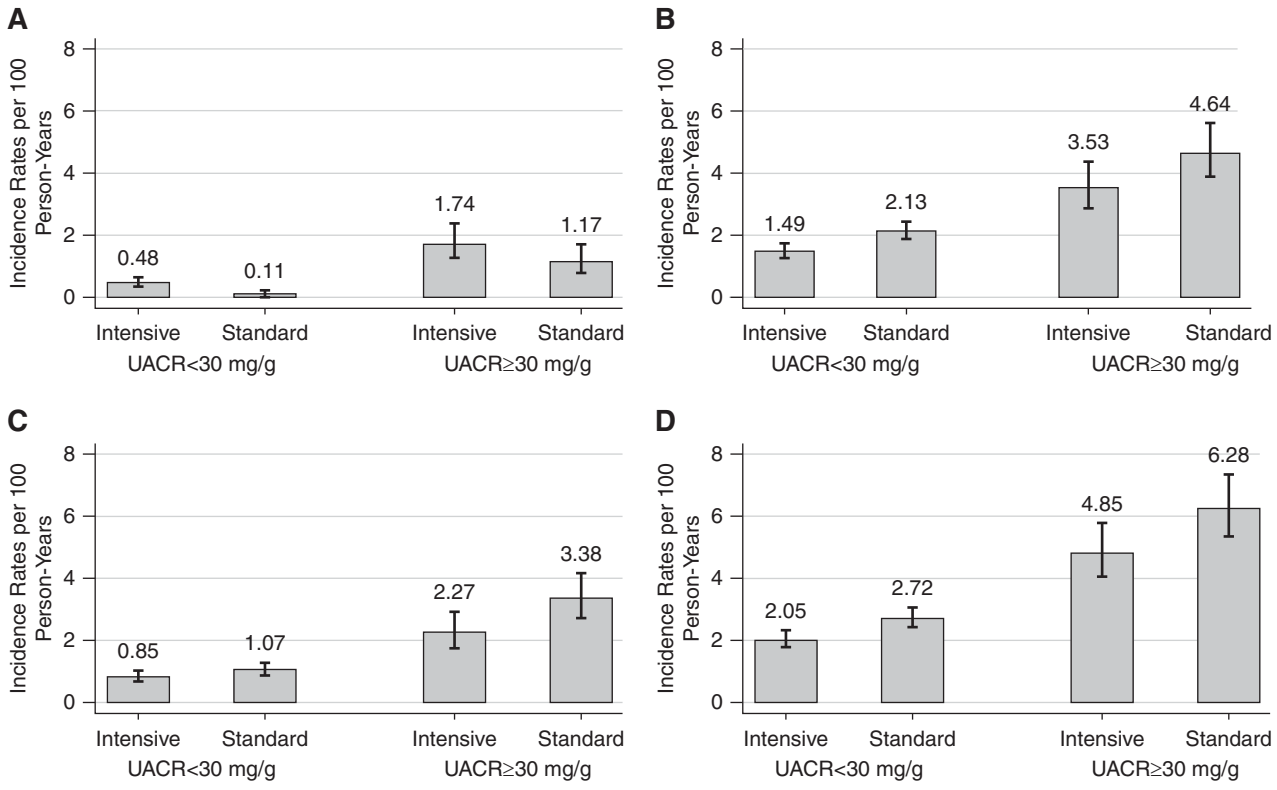

Figure 1. | **Albuminuria did not modify the absolute benefits and risks of intensive systolic BP lowering.** Incidence rates per 100 person-years of 40% eGFR decline (A), primary cardiovascular composite outcome (B), death (C), and primary cardiovascular composite outcome or death (D). Bars represent means, and error bars represent 95% confidence intervals. UACR, urine albumin-creatinine ratio.

Table 2. Effects of intensive systolic blood pressure lowering according to baseline urine albumin-creatinine ratio in the Systolic Blood Pressure Intervention Trial

Outcomes	*N* Events (Incidence Rate, Events per 100 Person-yr)		Absolute Differences (Events per 100 Person-yr)			Relative Differences		
	Intensive Arm	Standard Arm	Incidence Difference (Intensive versus Standard)[a]	Difference by Urine Albumin-Creatinine Ratio in Incidence Difference[b]	Interaction *P* Value[c]	Hazard Ratio (Intensive versus Standard)[d]	Difference by Urine Albumin-Creatinine Ratio in Hazard Ratio[e]	Interaction *P* Value[f]
40% reduction in eGFR (UACR<30 mg/g)	50 (0.48)	11 (0.11)	0.38 (0.23 to 0.52)	0.20 (−0.50 to 0.90)	0.57	4.55 (2.37 to 8.75)	0.22 (0.10 to 0.50)	<0.001
40% reduction in eGFR (UACR≥30 mg/g)	42 (1.74)	27 (1.17)	0.58 (−0.11 to 1.26)			1.48 (0.91 to 2.39)		
Primary cardiovascular composite (UACR<30 mg/g)	166 (1.49)	234 (2.13)	−0.64 (−0.99 to −0.28)	−0.47 (−1.65 to 0.72)	0.44	0.70 (0.57 to 0.85)	1.09 (0.77 to 1.53)	0.62
Primary cardiovascular composite (UACR≥30 mg/g)	89 (3.53)	113 (4.64)	−1.10 (−2.23 to 0.02)			0.76 (0.58 to 1.00)		
All-cause death (UACR<30 mg/g)	97 (0.85)	121 (1.07)	−0.22 (−0.47 to 0.04)	−0.89 (−1.84 to 0.06)	0.07	0.79 (0.61 to 1.04)	0.85 (0.56 to 1.30)	0.45
All-cause death (UACR≥30 mg/g)	60 (2.27)	87 (3.38)	−1.11 (−2.02 to −0.20)			0.67 (0.48 to 0.93)		
Primary cardiovascular composite or all-cause death (UACR<30 mg/g)	228 (2.05)	299 (2.72)	−0.67 (−1.08 to −0.27)	−0.76 (−2.14 to 0.61)	0.28	0.75 (0.63 to 0.89)	1.03 (0.76 to 1.38)	0.87
Primary cardiovascular composite or all-cause death (UACR≥30 mg/g)	122 (4.85)	153 (6.28)	−1.44 (−2.75 to −0.12)			0.77 (0.61 to 0.98)		

UACR, urine albumin-creatinine ratio.

[a]The estimated absolute incidence differences of study outcomes for intensive versus standard (reference) were calculated along with 95% confidence intervals. A positive value denotes higher incidence of the outcome in the intensive arm versus standard arm.

[b]The difference in incidence difference of study outcomes between those with albuminuria versus those without albuminuria (reference) was calculated. A positive value denotes the intensive arm causing a higher incidence of the outcome in those with albuminuria versus those without albuminuria.

[c]The interactions of absolute incidence differences of outcomes between groups with and without baseline albuminuria were tested by comparing differences in the estimated incidence differences with the SEM of the differences.

[d]Hazard ratios of intensive BP lowering versus standard (reference) were calculated for study outcomes in the albuminuria subgroups. A value of more than one denotes higher hazard of the outcome in the intensive arm versus standard arm.

[e]The ratio of hazard ratios comparing those with albuminuria versus those without albuminuria (reference) was calculated. A value of more than one denotes the intensive arm causing a higher hazard of the outcome in those with albuminuria versus those without albuminuria.

[f]The interactions of intensive systolic BP lowering on outcomes with presence of baseline albuminuria were tested using likelihood ratio tests.

On the basis of the results of ≥40% eGFR decline on the hazard ratio scale, one should not conclude that the absence of albuminuria somehow augments the risk for eGFR decline with intensive systolic BP lowering. Because relative risks are much more pronounced in populations at lower risk of that event than in populations that are at higher risk (15), the differential baseline hazard of ≥40% eGFR decline in the standard systolic BP arm in those with and without albuminuria affects the corresponding hazard ratios in those with and without albuminuria. As is evident from Figure 1, when comparing the hazard ratios of an intervention in different populations, baseline hazards and the absolute risk differences between the study arms in the two groups need to be considered for drawing conclusions. Indeed, a similar observation was noted when comparing the risk of incident CKD with intensive systolic BP lowering in the nondiabetic SPRINT population and the diabetic Action to Control Cardiovascular Risk in Diabetes Blood Pressure (ACCORD BP) study (2).

The SPRINT, the ACCORD BP study, and the Secondary Prevention of Small Subcortical Strokes trial were trials that predominantly included persons with normal kidney function or mild to moderate CKD; all found that intensive systolic BP lowering results in decline in eGFR (1,16,17). However, long-term follow-up of the AASK and the MDRD trial, which included individuals with more advanced CKD and higher magnitude of proteinuria than the SPRINT, suggested that BP lowering to a target mean arterial pressure <92 mm Hg might decrease the progression to kidney failure for some individuals (18). In particular, BP lowering to this target tended to be beneficial in those with higher levels of baseline proteinuria (AASK baseline protein-creatinine ratio, ≥0.22 g/g; MDRD baseline proteinuria, >1 g/d) (5,19). Level of proteinuria, number of kidney failure events, and follow-up time were much higher in the MDRD trial and the AASK than in the SPRINT, where median baseline UACR was 70 mg/g among the UACR≥30 mg/g subgroup. Although there is biologic plausibility that BP lowering reduced the risk of progression to kidney failure in the higher-risk subgroup of those with proteinuria in the MDRD trial and the AASK (20), less exists to explain why the risk for ≥40% eGFR decline was augmented only on the hazard ratio scale in those without albuminuria compared with those with albuminuria in this study.

Although ≥40% eGFR decline is considered a surrogate marker for risk of progression to kidney failure (11), it should be noted that ≥40% eGFR decline is presumably on the basis of progression of underlying intrinsic kidney disease. In contrast, ≥40% eGFR decline due to intensive systolic BP lowering might not have the same clinical connotations as ≥40% eGFR decline due to progression of intrinsic kidney disease. Additional analyses of the SPRINT and the ACCORD BP study have shown that intensive systolic BP lowering does not increase biomarkers of tubular cell damage despite lowering eGFR (3,21,22). Furthermore, a causal mediation analysis found no evidence that reduction of eGFR induced by intensive systolic BP lowering attenuated the cardiovascular or mortality benefits (23). In this study, we did not find evidence that baseline albuminuria modified the effects of intensive systolic BP lowering on cardiovascular disease outcome or all-cause death. A systematic review and meta-analysis of BP randomized controlled trials in participants with stages 3–5 CKD found that participants randomized to more intensive BP control had 14% lower risk of death than participants randomized to less intensive BP control (24). Regardless, longer-term data are needed to draw firm conclusions on the long-term effect of intensive systolic BP lowering on risk of kidney failure.

There were several strengths and some limitations of this study. First, the SPRINT is a large, randomized trial that was highly effective in achieving a sustained separation in BP between the intensive and standard arms. In order to provide clinicians and patients with useful information to promote shared decision making, we estimated absolute incidence rate differences, enabling comparison of the benefits of reduction of cardiovascular disease and death, alongside the risk of ≥40% eGFR decline. A limitation of our study was that we are unable to draw any conclusions on risk of kidney failure because the SPRINT was terminated early and there were few kidney failure events. The SPRINT findings may not be applicable to other study populations, such as individuals with low cardiovascular disease risk, history of diabetes, stroke, or proteinuria >1 g/d or those residing in nursing homes. There is the possibility of type 1 error given the multiple comparisons made in this *post hoc* analysis of the SPRINT.

In conclusion, the absolute effects of intensive systolic BP lowering on increased risk of ≥40% eGFR decline were not modified by baseline albuminuria. Furthermore, intensive systolic BP lowering reduced the risk of cardiovascular disease and mortality, regardless of baseline albuminuria status. These results support the adoption of the SPRINT findings in clinical practice in persons with or without albuminuria.

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All components of the SPRINT study protocol were designed and implemented by the investigators. The investigative team collected, analyzed, and interpreted the data. All aspects of manuscript writing and revision were carried out by the coauthors. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health, the US Department of Veterans Affairs, or the US Government.

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Data Sharing Statement

The data used in this paper are publicly available from the National Heart, Lung, and Blood Institute Biologic Specimen and Data Repository Information Coordinating Center.

Supplemental Material

This article contains the following supplemental material online at http://cjasn.asnjournals.org/lookup/suppl/doi:10.2215/CJN.12371019/-/DCSupplemental.

Supplemental Table 1. Absolute risk differences and hazard ratios for intensive systolic BP lowering on 30% eGFR decline and prespecified SPRINT kidney outcomes.

Supplemental Table 2. Incidence rates and hazard ratios for intensive systolic BP lowering on primary cardiovascular composite outcome, all-cause death, and 40% eGFR decline outcomes in participants with baseline eGFR ≥60 ml/min per 1.73 m^2.

Supplemental Table 3. Incidence rates and hazard ratios for intensive systolic BP lowering on primary cardiovascular composite, all-cause death, and 40% eGFR decline outcomes in participants with baseline eGFR <60 ml/min per 1.73 m^2.

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