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Home and office blood pressure measurements as determinants of kidney disease in the general population: The Finn-Home Study

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European and American guidelines for hypertension now advocate out-of-office blood pressure (BP) measurements for diagnosing patients, including those with chronic kidney disease (CKD). Self-monitoring of BP with self-titration of antihypertensive medication has been also shown to improve BP control and to be cost effective in high-risk patients.[1] However, prior inconclusive studies on the association between home BP measurements and CKD have mainly been performed in selected patient populations using a cross-sectional approach.[2–10] It is therefore still unclear whether home BP is more strongly associated with CKD than office BP in the general population. We therefore compared the associations of home and office BP with prevalent albuminuria, incident decreased glomerular filtration rate (GFR), and GFR decline in a nationwide population sample.

A total of 2120 participants drawn from the Finnish population register underwent a health examination and home BP monitoring in 2000–2001 as a part of the Health 2000 survey. We excluded 111 participants with missing data from the cross-sectional analyses. The Finn-Home study participants were invited to be re-examined in 2011–2012. A total of 1805 participants who were included in the cross-sectional analyses were still alive and 1350 (74.8%) agreed to participate. We excluded 82 participants with missing data from the longitudinal analyses.

All participants underwent a clinical examination with similar methods at baseline and follow-up. Fasting blood samples for serum creatinine were taken and estimated GFRs were calculated.[11] A spot urine sample for albumin and creatinine was only collected at baseline. Office BP was measured twice by a nurse with a mercury sphygmomanometer. Home BP was self-measured twice every morning and evening for seven days using a validated, automatic, oscillometric device. Office and home BP were defined as the means of all available measurements. We examined the association of baseline office and home BP with albuminuria and incident decreased GFR using logistic regression. The association of baseline office and home BP with a change in GFR between baseline and follow-up was assessed using linear regression. Home and office BP were included in all models simultaneously. Multivariable models were adjusted for baseline age, sex, diabetes and smoking as covariates. The longitudinal models also included baseline GFR as a covariate.

A total of 2009 participants (mean ± SD age 56.4 ± 8.5 years; 53.8% women) were included in the analyses for prevalent albuminuria. In the unadjusted models, only systolic and diastolic home BP, but not systolic or diastolic office BP, were associated with prevalent albuminuria (Table 1). In the adjusted model, both systolic home and systolic office BP were associated with prevalent albuminuria. Only diastolic home BP, but not diastolic office BP, was associated with prevalent albuminuria in the adjusted model.

A total of 1268 participants (mean ± SD age 55.2 ± 7.7 years; 55.3% women) with normal GFR at baseline were included in the analyses for incident decreased GFR and GFR change from baseline to follow-up. Baseline systolic home BP, but not systolic office BP, was related to incident decreased GFR in the unadjusted models. (Table 1). No significant association was observed in the unadjusted models between baseline diastolic home BP or diastolic office BP and

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Table 1. Associations of a one standard deviation increase in baseline home and office blood pressure with (a) prevalent albuminuria at baseline ($n = 2009$) and (b) incident decreased glomerular filtration rate and incident glomerular filtration rate change over an 11-year follow-up ($n = 1268$).

		Systolic BP				Diastolic BP			
		Unadjusted model		Adjusted model		Unadjusted model		Adjusted model	
Kidney injury	BP	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*
Albuminuria	Home	1.74 (1.40–2.17)	<0.001	1.41 (1.11–1.80)	0.006	1.59 (1.28–1.97)	<0.001	1.40 (1.11–1.76)	0.004
	Office	1.23 (0.99–1.53)	0.06	1.40 (1.11–1.76)	0.004	1.01 (0.81–1.27)	0.91	1.21 (0.96–1.52)	0.10
Incident dGFR	Home	1.62 (1.21–2.16)	0.001	1.31 (0.96–1.78)	0.09	1.21 (0.90–1.64)	0.21	1.11 (0.80–1.54)	0.54
	Office	1.00 (0.75–1.33)	0.99	0.93 (0.69–1.27)	0.91	0.91 (0.68–1.20)	0.50	0.94 (0.68–1.28)	0.67
		$\beta \pm$ SE	*P*	$\beta \pm$ SE	*P*	$\beta \pm$ SE	*P*	$\beta \pm$ SE	*P*
Change in GFR	Home	-1.23 ± 0.39	0.002	-0.73 ± 0.40	0.06	-0.13 ± 0.39	0.73	0.18 ± 0.37	0.63
	Office	0.03 ± 0.39	0.93	0.48 ± 0.39	0.22	0.07 ± 0.39	0.86	-0.20 ± 0.37	0.59

BP: blood pressure; CI: confidence interval; CKD: chronic kidney disease; dGFR: decreased glomerular filtration rate; GFR: glomerular filtration rate; SE, standard error; β: regression coefficient.

A total of 143 participants had baseline albuminuria (defined as albumin to creatinine ratio ≥2.5 mg/mmol for men and ≥3.5 mg/mmol for women) and 100 participants developed incident CKD (estimated GFR <60 ml/min/1.73 m^2) during follow-up. Systolic home and office BP or diastolic home and office BP were simultaneously included in the unadjusted models. Multivariable models also included baseline age, sex, diabetes, and smoking as covariates. All longitudinal models included baseline GFR as a covariate. The participants who were included in cross-sectional analyses had mean systolic/diastolic home BP $129.9 \pm 18.6/80.3 \pm 9.2$ mmHg, systolic/diastolic office BP $137.5 \pm 20.0/83.8 \pm 10.6$ mmHg, urine albumin to creatinine ratio 2.0 ± 13.1 mg/mmol and GFR 90.9 ± 13.5 ml/min/1.73 m^2. A total of 143 participants (7.1%) had albuminuria at baseline. Participants who were included in the longitudinal analyses had a mean systolic/diastolic baseline home BP of $127.2 \pm 17.7/79.5 \pm 9.1$ mmHg, a mean systolic/diastolic baseline office BP of $135.5 \pm 19.4/83.6 \pm 10.3$ mmHg and a mean change in GFR of -9.4 ± 10.0 ml/min/1.73 m^2 from baseline to follow-up. A total of 100 participants (7.9%) developed decreased GFR during follow-up.

incident decreased GFR. No significant association was observed between any baseline BP parameters and incident decreased GFR in the adjusted models. In the unadjusted model, only higher baseline systolic home BP, but not systolic office BP, was associated with GFR decline (Table 1). Baseline diastolic home BP and office BP did not predict a change in GFR. Baseline home and office systolic or diastolic BP did not predict a change in GFR in the multivariable-adjusted models.

To our knowledge, this is the first large-scale, longitudinal, population-based study that has examined the association of home and office BP with CKD. In the unadjusted models, home BP, but not office BP, was associated with prevalent albuminuria. In the models adjusted for other CKD risk factors, both systolic home and office BP, but only diastolic home BP, were associated with prevalent albuminuria. We also showed that systolic home BP was the only BP parameter associated with incident decreased GFR and GFR decline over an 11-year follow-up in the unadjusted models, whereas no significant association was observed in the multivariable-adjusted models.

Methodological limitations in many previous studies have made it difficult to establish whether home BP associates more strongly than office BP with CKD.[2–10] For example, many studies have been cross-sectional, which limits causal interference.[3,4,9,10] Furthermore, the samples of these studies have consisted of only 68–392 patients, leading to limited statistical power to test for significant differences between the two BP measurement methods. In addition, all prior studies have been performed in selected samples consisting of patients with diabetes,[7,10] CKD[2,6,8] or hypertension.[3–5,9]

We found four previous studies that examined the association between home versus office BP and albuminuria.[3,4,9,10] Three cross-sectional studies observed home BP to be an equal or stronger correlate of albuminuria than ambulatory or office BP.[3,4,9] The longitudinal study by Palmas et al.[10] observed that adding home BP to a statistical model that included office BP improved the prediction of albuminuria progression. Our study concurs with these previously published studies because systolic and diastolic home BP were strongly associated with prevalent albuminuria in all models, whereas systolic office BP was an inconsistent correlate of albuminuria.

Four previous studies have also assessed the association between home BP and incident decreased GFR or a change in GFR.[5–8] In addition, a single study has assessed the role of home BP as an independent predictor of progression from CKD to end-stage renal disease.[2] Although statistical comparisons of home versus office BP measurements was not

always performed, four of these studies suggested that home BP was a stronger predictor of a decline in GFR than office BP.[2,6–8] Our results are mostly in line with these previous findings from patient cohorts because we found that systolic home BP was the only BP parameter associated with incident decreased GFR and a decline in GFR in the unadjusted models.

In conclusion, our results suggest that home BP is more strongly associated with prevalent and incident CKD than office BP in the general population. Although the optimum home and office BP targets in patients with CKD and in the general population are still being debated[12], our findings are in line with previous work and provide additional evidence of the superiority of home BP over office BP.

Authorship

SS, IK, AJ and TN contributed to the conception and design of the work. JS contributed to the acquisition of data for the work. SS, VL and PP contributed to the analysis and interpretation of data for the work. SS and TN drafted the manuscript. VL, PP, JS, IK and AJ critically revised the manuscript. All gave final approval and agree to be accountable for all aspects of work ensuring integrity and accuracy.

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