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ORIGINAL RESEARCH

The Association Between Statin Use and Risk of Chronic Kidney Disease in Community-Dwelling Older People in Shanghai, China

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Purpose: The effects of statins on renal outcomes have already been studied in patients with chronic kidney disease (CKD); however, data on the general population are limited. We evaluated the association between statin use and risk of CKD in community-dwelling older people in Shanghai, China.

Patients and Methods: This registry-based cohort study was conducted in four communities in four districts in Shanghai. Participants with an estimated glomerular filtration rate (eGFR) $\geq$ 60 mL/min/1.73 m^2 in 2016 were eligible for the study, and new-onset CKD in 2017, 2018, and 2019 was recorded. Poisson generalized linear models were conducted to examine the relationships among statin therapy, dyslipidemia, and CKD; linear mixed-effects models were conducted to examine the relationships between statin therapy and changes in eGFR. All analyses were performed with both conventional adjustment and propensity score-matching methods.

Results: Of the study cohort of 2455 participants (41.1% men; average age, 68.06 years), 624 (25.4%) were treated with stains. Two propensity score-matched cohorts of 604 participants each were analyzed (statin users and nonusers). Statin use was significantly associated with a decreased risk of new-onset CKD with hazard ratios (HRs) and 95% confidence intervals (CIs) of 0.73 (0.59 to 0.91) ($p<0.01$) in the unmatched cohort and 0.75 (0.59 to 0.97) ($p=0.02$) in the matched cohort. There were significant differences in the eGFR decline between statin users and nonusers from baseline to 3 years in the unmatched and matched cohorts (both $p<0.05$). In addition, both statin users and nonusers with dyslipidemia experienced more new-onset CKD (both $p<0.05$).

Conclusion: Statin use was significantly associated with a decreased risk of new-onset CKD and a slower decline in eGFR in community-dwelling older people. Meanwhile, dyslipidemia was a risk factor for CKD progression among both statin users and nonusers.

Keywords: chronic kidney disease, statins, dyslipidemia, community-dwelling older people

Introduction

Chronic kidney disease (CKD) is a major health problem associated with increased risk of all-cause mortality, cardiovascular disease, and end-stage renal disease.[1–5] When CKD is defined as an estimated glomerular filtration rate (eGFR) less than 60 mL/min/1.73 m^2, its prevalence is 5.2% in England[6] and between 2.5% and 11.2% in Europe, North America, Asia, and Australia.[7] In 2012, the prevalence of CKD was 10.8% in China, suggesting that nearly 120 million adults had the disease.[8] The global incidence and mortality of CKD is continually rising, and its high medical costs impose a considerable burden on patients, their families, and the world.

Statins are the mainstay of the primary and secondary prevention of cardiovascular disease in the general population.[9,10] Statins also have beneficial effects in the treatment of diabetic nephropathy.[11–15] Growing numbers of studies have evaluated the renal protective effects of statins. The Assessment of Lescol in Renal Transplant (ALERT)

trial and the Scandinavian Simvastatin Survival Study (4S) demonstrated that statins slowed CKD progression.[16,17] However, the large Study of Heart and Renal Protection (SHARP), which included 6245 participants with advanced CKD, found that statin administration did not reduce the risk of kidney failure or rate of change in eGFR.[18] A previous systematic review and meta-analysis including 57 studies suggested that statin therapy does not reduce the risk of kidney failure events in adults not receiving dialysis but may modestly reduce proteinuria and the rate of eGFR decline.[19] Most of these studies were performed using patients with kidney disease. To our knowledge, no research has thus far been conducted into the associations between statins and renal outcomes in a community-dwelling population.

An abnormal lipid metabolism is common in patients with kidney disease.[20,21] Post hoc analyses of several large trials have demonstrated that dyslipidemia is significantly associated with increased risk of a reduced kidney function or faster eGFR decline in a general population without kidney disease.[22,23] Statins can significantly improve lipid profiles and the use of statins to lower lipid levels has renoprotective effects.[24,25] However, few studies have evaluated whether dyslipidemia has a differential impact on renal outcomes between statin users and nonusers.

In the present study, we investigated the association of statin use with the risk of CKD and changes in eGFR in community-dwelling older people in Shanghai, China. In addition, we evaluated the impact of dyslipidemia on CKD among statin users and nonusers.

Materials and Methods
Study Design and Participants
Data of this investigation were obtained from the "Research on Community Elderly Population of Tongji University" (RECEPT) study. The purpose of the RECEPT study was to describe the prevalence, incidence, and natural history of cardiovascular risk factors in community-dwelling older people in Shanghai, China. We adopted a multi-stage, stratified, random cluster sampling scheme. In the first stage, two districts were selected from each of the eight urban districts and eight suburbs of Shanghai; in the second stage, one community was randomly selected from each district. Free medical physical examination services are provided annually to community-dwelling people aged ≥ 60 years in Shanghai, China. We obtained the electronic medical records of health examinations from the Physical Examination System in these four districts in 2016, 2017, 2018, and 2019.

Given the focus of this investigation, we assembled a cohort, with participants with an eGFR≥60 mL/min/1.73 m^2 in 2016 eligible for the baseline study. Follow-up was conducted in 2017, 2018, and 2019 and new-onset CKD during follow-up was recorded. Therefore, using the physical examination data from 2016 to 2019, we analyzed the relationship of statin prescription with new-onset CKD and changes in eGFR and that of dyslipidemia with new-onset CKD in both statin users and nonusers. The study was performed in accordance with the Declaration of Helsinki and approved by the Ethics Committee of Tongji University. All procedures were performed in accordance with ethical standards. Written consent was obtained from each participant after they had been informed of the objectives, benefits, medical items, and confidentiality of personal information.

Participants were eligible for the study if they (1) were≥60 years old, (2) had a Shanghai household registration or were resident in this community for more than three years, (3) had an eGFR ≥ 60 mL/min/1.73 m^2 in 2016, and (4) had complete baseline data in 2016, and serum creatinine (Scr) measurements in 2017, 2018, and 2019. Exclusion criteria included (1) an eGFR < 60 mL/min/1.73 m^2 in 2016, (2) incomplete baseline data provided in 2016, (3) presence of severe mental illness, cognitive impairment or malignant tumors in 2016, and (4) incomplete Scr measurements provided in 2017, 2018, or 2019 (Figure 1).

Data Collection and Outcome Measures
For each patient, comprehensive data were extracted from electronic medical records. This included participant demographics (age and sex), body mass index (BMI), waist-to-hip ratio (WHR), systolic blood pressure (SBP) and diastolic blood pressure (DBP). We also collected information on participant clinical data (including the prescription of angiotensin-converting-enzyme inhibitors/angiotensin receptor blockers [ACEIs/ARBs], calcium channel blockers [CCBs], beta-blockers, diuretics, and antidiabetic drugs, and medical history of hypertension and diabetes mellitus) and laboratory
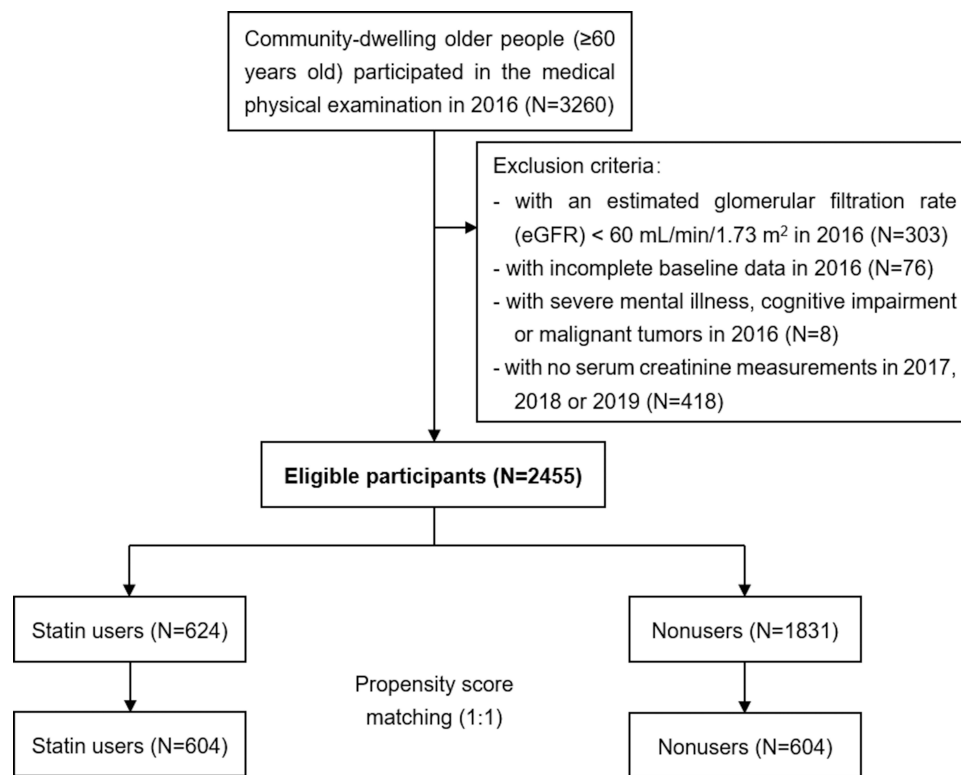
Figure 1 Flowchart of study inclusion.

data (including glycated hemoglobin [HbA1c], fasting plasma glucose [FPG], total cholesterol [TC], triglyceride [TG], high-density lipoprotein [HDL], low-density lipoprotein [LDL], aspartate transaminase [AST], alanine transaminase [ALT], blood urea nitrogen [BUN], total bilirubin [TBIL], uric acid [UA] and Scr).

The eGFR was calculated with the use of the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula: for males, $eGFR = 141 \times (Scr/0.9)^{-0.411} \times 0.993^{age}$ if $Scr \leq 80$ mmol/L and $eGFR = 141 \times (Scr/0.9)^{-1.209} \times 0.993^{age}$ if $Scr > 80$ mmol/L; for females, $eGFR = 144 \times (Scr/0.7)^{-0.329} \times 0.993^{age}$ if $Scr \leq 62$ mmol/L and $eGFR = 144 \times (Scr/0.7)^{-1.209} \times 0.993^{age}$ if $Scr > 62$ mmol/L.[26] New-onset CKD was defined as the occurrence of $eGFR < 60$ mL/min/1.73 m^2 during follow-up. Dyslipidemia was defined as $TC \geq 6.2$ mmol/L, $LDL \geq 4.1$ mmol/L, $HDL < 1.0$ mmol/L, or $TG \geq 2.3$ mmol/L.[27]

Statistical Analysis

To reduce potential confounding and selection bias, we used the propensity score-matching method. Propensity scores were calculated through a logistic regression model, with statin users versus nonusers as the dependent variable and all covariates measured at baseline as independent variables (Table 1). Matched pairs were obtained using a greedy nearest-neighbor matching algorithm (1:1 ratio, without replacement) and with a caliper width equal to 0.2 of the SD of the logit of the propensity score.[28] A standardized difference of less than 0.1 was used to indicate a negligible difference in covariates between the groups.

Poisson generalized linear models were conducted to examine the association between statin use and new-onset CKD rates. The covariates listed in Table 1 were adjusted in the unmatched cohort analyses. Linear mixed-effects models were conducted to examine the association between statin use and changes in eGFR. Participant ID was included as a random effect, with time (baseline, 1, 2, and 3 years), group (statin users versus nonusers), and time-group interactions included as fixed effects. The covariates listed in Table 1 were included as fixed effects in the unmatched cohort analyses.

Meanwhile, the impact of dyslipidemia on new-onset CKD was assessed among both statin users and nonusers. We repeated the propensity score matching (Supplementary Tables 1 and 2) and compared the event rates between

Table 1 Baseline Characteristics of Statin Users and Nonusers Before and After Propensity Score Matching

Characteristics	Statin Users (n=624)	Nonusers (n=1831)	Standard Difference	p	Statin Users (n=604)	Nonusers (n=604)	Standard Difference	p
Age, mean (SD), year	68.18 (2.58)	68.02 (2.68)	0.06	0.19	68.20 (2.60)	68.13 (2.61)	0.03	0.67
Male, N (%)	223 (35.74)	785 (42.87)	−0.15	<0.01	215 (35.60)	219 (36.26)	−0.01	0.81
Smoking, N (%)	69 (11.06)	310 (16.93)	−0.18	<0.01	66 (10.93)	64 (10.60)	0.01	0.85
ACEIs/ARBs, N (%)	345 (55.29)	691 (37.74)	0.36	<0.01	331 (54.80)	343 (56.79)	−0.04	0.49
CCBs, N (%)	237 (37.98)	459 (25.07)	0.28	<0.01	223 (36.92)	245 (40.56)	−0.08	0.19
Beta-blockers, N (%)	162 (25.96)	352 (19.22)	0.16	<0.01	158 (26.16)	156 (25.83)	0.01	0.90
Diuretics, N (%)	92 (14.74)	197 (10.76)	0.12	0.01	90 (14.90)	82 (13.58)	0.04	0.51
History of hypertension, N (%)	349 (55.93)	965 (52.70)	0.07	0.16	340 (56.29)	341 (56.46)	<−0.01	0.95
Antidiabetic drugs, N (%)	143 (22.92)	262 (14.31)	0.22	<0.01	136 (22.52)	139 (23.01)	−0.01	0.84
History of diabetes mellitus, N (%)	97 (15.54)	234 (12.78)	0.08	0.08	95 (15.73)	101 (16.72)	−0.03	0.64
SBP, mean (SD), mm Hg	136.19 (18.30)	135.40 (17.63)	0.04	0.34	136.06 (18.40)	135.99 (16.86)	<0.01	0.94
DBP, mean (SD), mm Hg	79.23 (8.98)	77.82 (9.53)	0.15	<0.01	79.16 (8.97)	79.62 (9.54)	−0.05	0.39
HbA1c, mean (SD), %	6.33 (0.88)	6.01 (0.84)	0.37	<0.01	6.30 (0.83)	6.31 (1.10)	−0.01	0.84
FPG, mean (SD), mmol/l	5.64 (1.11)	5.58 (1.24)	0.05	0.26	5.64 (1.11)	5.66 (1.28)	−0.02	0.75
TC, mean (SD), mmol/l	5.07 (1.05)	4.85 (0.90)	0.22	<0.01	5.05 (1.05)	5.03 (0.91)	0.02	0.79
LDL, mean (SD), mmol/l	2.93 (0.88)	2.87 (0.78)	0.08	0.09	2.93 (0.88)	2.95 (0.78)	−0.03	0.66
HDL, mean (SD), mmol/l	1.72 (0.50)	1.56 (0.45)	0.35	<0.01	1.71 (0.50)	1.68 (0.49)	0.06	0.32
TG, mean (SD), mmol/l	1.78 (1.02)	1.53 (0.83)	0.28	<0.01	1.73 (0.96)	1.73 (0.97)	0.01	0.92
WHR, mean (SD)	0.91 (0.06)	0.91 (0.06)	0.04	0.44	0.91 (0.06)	0.91 (0.06)	0.01	0.83
BMI, mean (SD), kg/m^2	24.90 (3.28)	24.77 (3.33)	0.04	0.41	24.81 (3.23)	24.71 (3.45)	0.03	0.61
AST, mean (SD)	23.99 (6.81)	22.97 (6.92)	0.15	<0.01	23.90 (6.57)	23.58 (7.49)	0.05	0.43
ALT, mean (SD)	20.93 (10.43)	18.98 (9.48)	0.20	<0.01	20.64 (10.11)	20.14 (10.87)	0.05	0.41
BUN, mean (SD), mmol/L	5.74 (0.47)	5.74 (0.47)	0.01	0.85	5.74 (0.46)	5.74 (0.48)	<0.01	0.98
TBIL, mean (SD), μmol/L	12.90 (4.98)	12.82 (5.02)	0.02	0.74	12.87 (4.94)	12.92 (4.94)	−0.01	0.85
UA, mean (SD), μmol/L	318.19 (78.32)	314.66 (79.50)	0.05	0.34	317.71 (77.82)	319.30 (82.98)	−0.02	0.73
eGFR, mean (SD), mL/min/1.73m^2	85.50 (12.29)	85.56 (11.81)	−0.01	0.90	85.26 (11.16)	84.99 (11.54)	0.02	0.68

Abbreviations: ACEIs/ARBs, angiotensin-converting-enzyme inhibitors/angiotensin receptor blockers; CCBs, calcium channel blockers; SBP, systolic blood pressure; DBP, diastolic blood pressure; HbA1c, glycated hemoglobin; FPG, fasting plasma glucose; TC, total cholesterol; LDL, low-density lipoprotein; HDL, high-density lipoprotein; TG, triglycerides; WHR, waist-to-hip ratio; BMI, body mass index; AST, aspartate transaminase; ALT, alanine transaminase; BUN, blood urea nitrogen; TBIL, total bilirubin; UA, uric acid; eGFR, estimated glomerular filtration rate.

participants with normal blood lipids and those with dyslipidemia. All analyses were conducted using SAS 9.4 (SAS Institute, Cary, NC, USA) and R 4.0.2. Two-sided p values less than 0.05 indicated statistical significance.

Results

The study cohort comprised 2455 older Chinese people. Their average age was (68.06 ± 2.65) years old, and 1008 (41.1%) were men. Statins were prescribed to 25.4% (624 of 2455) of the participants. Compared with the nonstatin group, the statin group had a higher prevalence of female participants, nonsmokers, antihypertensive drugs and antidiabetic drugs, and higher levels of DBP, HbA1c, TC, HDL, TG, AST, and ALT. Two propensity score-matched cohorts of 604 participants were defined according to statin use and analyzed. After propensity score matching, no significant differences in baseline characteristics were found between statin and nonstatin groups (Table 1).

Participants were followed up for 3 years. The incidences of new-onset CKD for one, two, and three times were 10.6% (66 of 624), 3.0% (19 of 624), and 0.5% (3 of 624) in the unmatched cohort for statin users, and 14.6% (267 of 1831), 3.6% (66 of 1831), and 1.0% (19 of 1831) for nonusers (p=0.03) (Figure 2A). After propensity score matching, the incidences were 10.4% (63 of 604), 3.1% (19 of 604), and 0.5% (3 of 604) for statin users and 13.9% (84 of 604), 3.6% (22 of 604), and 1.0% (6 of 604) for nonusers (p=0.18) (Figure 2B). Poisson generalized linear analyses showed that statin use was significantly associated with a decreased risk of new-onset CKD, with hazard ratios (HRs) and 95% confidence intervals (CIs) of 0.73 (0.59 to 0.91) (p<0.01) after adjustment for the covariates in the unmatched cohort and 0.75 (0.59 to 0.97) (p=0.02) in the matched cohort (Table 2).

The changes in eGFR from baseline to 1, 2, and 3 years in statin users and nonusers are demonstrated in Table 2. The declines in eGFR were −7.11 mL/min/1.73 m^2 (95% CI, −8.10 to −6.11) and −8.54 mL/min/1.73 m^2 (95% CI, −9.16 to −7.92) in the statin and nonstatin groups from baseline to 3 years in the unmatched cohort (p=0.02). These declines were −7.00 mL/min/1.73 m^2 (95% CI, −7.93 to −5.99) and −8.30 mL/min/1.73 m^2 (95% CI, −9.33 to −7.27) in the matched cohort (p=0.04).

We repeated the propensity score matching for statin users and nonusers with dyslipidemia and normal blood lipids (Supplementary Tables 1 and 2). Poisson generalized linear analyses showed that compared with individuals with normal blood lipids, those with dyslipidemia experienced more new-onset CKD, both statin users (HR: 1.61, 95% CI: 1.10 to 2.36, p=0.01) and nonusers (HR: 1.49, 95% CI: 1.22 to 1.81, p<0.01), after adjustment for the covariates in the unmatched cohort. In addition, the HRs (95% CIs) were 1.54 (1.01 to 2.36) (p=0.04) and 1.32 (1.05 to 1.68) (p=0.02) in the matched cohort, respectively (Table 3).
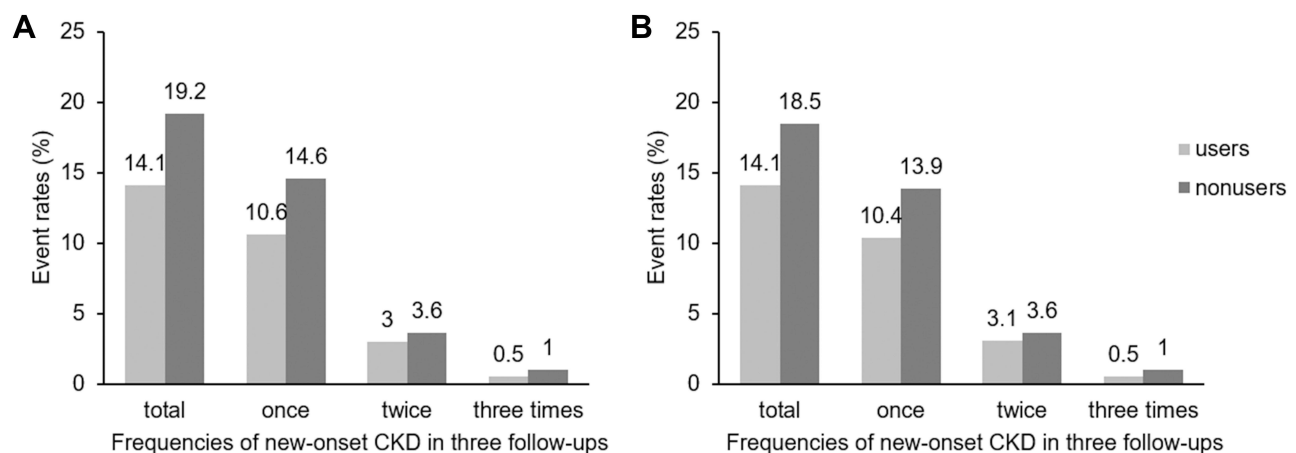

Figure 2 Frequencies of new-onset CKD in three follow-up visits among statin users and nonusers in the unmatched cohort (**A**) and matched cohort (**B**).

Table 2 Association of Statin Use with Risk of CKD Progression and Changes in eGFR

	Unmatched Cohort			Matched Cohort		
	Statin Users	Nonusers	*p*	Statin Users	Nonusers	*p*
New-onset CKD HR (95% CI)	0.73 (0.59 to 0.91)	1 (reference)	<0.01	0.75 (0.59 to 0.97)	1 (reference)	0.02
Changes in eGFR, mL/min/1.73 m^2						
1 year	−4.63 (−5.58 to −3.67)	−5.50 (−6.15 to −4.84)	0.18	−4.35 (−5.26 to −3.45)	−5.15 (−6.21 to −4.10)	0.22
2 years	−6.27 (−7.20 to −5.34)	−6.97 (−7.60 to −6.34)	0.27	−6.10 (−7.04 to −5.16)	−7.10 (−8.16 to −6.03)	0.11
3 years	−7.11 (−8.10 to −6.11)	−8.54 (−9.16 to −7.92)	0.02	−7.00 (−7.93 to −5.99)	−8.30 (−9.33 to −7.27)	0.04

Notes: HR (95% CI) was estimated by Poisson generalized linear models adjusted for age, sex, smoking, ACEIs/ARBs, CCBs, beta-blockers, diuretics, history of hypertension, antidiabetic drugs, history of diabetes mellitus, SBP, DBP, HbA1c, FPG, TC, LDL, HDL, TG, WHR, BMI, AST, ALT, BUN, TBIL, UA and baseline eGFR in the unmatched cohort analyses. Changes in eGFR were estimated by linear mixed-effects models.
Abbreviations: CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; HR, hazard ratio; CI, confidence interval.

Table 3 Association Between Dyslipidemia and Risk of CKD Progression in Statin Users and Nonusers

	Unmatched Cohort		Matched Cohort	
	HR (95% CI)	*p*	HR (95% CI)	*p*
Statin users				
Normal blood lipids	1 (reference)		1 (reference)	
Dyslipidemia	1.61 (1.10 to 2.36)	0.01	1.54 (1.01 to 2.36)	0.04
Nonusers				
Normal blood lipids	1 (reference)		1 (reference)	
Dyslipidemia	1.49 (1.22 to 1.81)	<0.01	1.32 (1.05 to 1.68)	0.02

Notes: HR (95% CI) was estimated by Poisson generalized linear models adjusted for age, sex smoking, ACEIs/ARBs, CCBs, beta-blockers, diuretics, history of hypertension, antidiabetic drugs, history of diabetes mellitus, SBP, DBP, HbA1c, FPG, WHR, BMI, AST, ALT, BUN, TBIL, UA and baseline eGFR in the unmatched cohort analyses.
Abbreviations: CKD, chronic kidney disease; HR, hazard ratio; CI, confidence interval.

Discussion

In the present study, we used both a conventional adjustment method and propensity score-matching method to evaluate the association between statin prescription and kidney function in community-dwelling older people. Statin use was significantly associated with a decreased risk of new-onset CKD with HRs (95% CIs) of 0.73 (0.59 to 0.91) (*p*<0.01) in the unmatched cohort and 0.75 (0.59 to 0.97) (*p*=0.02) in the matched cohort. There were significant differences in the eGFR decline between statin users and nonusers in the unmatched and matched cohorts from baseline to 3 years. Additionally, both statin users and nonusers with dyslipidemia experienced more new-onset CKD.

CKD is a global health concern.[29] Individuals with CKD have a higher risk of premature mortality related to cardiovascular disease, even during the early stages of the disease; this represents a significant challenge for physicians.[30] Epidemiological studies have determined an estimated 8% to 16% prevalence of CKD worldwide,[31] as well as 10.2% prevalence in adults in the United States.[32] With ever increasing global population aging, the disease burden of CKD is continually increasing. The effects of statins on kidney disease progression remain subject to debate. Although some trials evaluating the effects of statins on kidney disease outcomes have shown benefits of statins,[16,17,33] others have shown no effect.[18,34,35] However, most of these studies analyzed patients with kidney disease. In the present study, the cohort comprised individuals without kidney disease. To our knowledge, our study is the first to investigate the associations between statins and renal outcomes in community-dwelling older people. We found that statin use was associated with decreased new-onset CKD rates in the 3-year follow-up. Accumulating evidence shows the favorable efficacy and safety profiles of statin therapy in community-dwelling older populations.[36,37] Therefore, it can be expected

that the prevalence of statin use in the elderly population will continue growing. The additional benefits of statins in preventing incident CKD may be of great clinical importance.

Statins, which are inhibitors of the hydroxymethylglutaryl-CoA (HMG-CoA) reductase enzyme, are powerful cholesterol-lowering medications. Statins limit cholesterol biosynthesis by competitively inhibiting HMG-CoA reductase activity[38] and further upregulating the expression of LDL receptors in liver cell membranes, increasing the clearance of circulating LDL-cholesterol from the blood.[39] They then improve renal function by ameliorating the damage to glomerular and tubular caused by cell lipids.[40] Beyond lipid lowering, statins slow the decline of the GFR and may also reduce proteinuria.[41,42] The mechanisms involved may include effects on renal nitric oxide (NO), endothelin-1 bioactivity and reduction of prenylation.[41,43,44] Statins have other therapeutic benefits, such as anti-inflammatory and antioxidant properties, apoptosis induction, inhibition of vascular smooth muscle cell proliferation, platelet activation and aggregation reduction, and increased atherosclerotic plaque stability.[15,29,45] Many of these effects potentially arise from small G-protein disruption. Because of the resultant biological and genetic stress, activation of G-protein signaling is pivotal in renal pathologies.[46] Effects on endothelial function may also explain the apparent renal benefits of statin therapy, which may improve renal perfusion while reducing abnormal permeability to plasma proteins.[47,48] Accordingly, the mechanisms by which statin therapy reduces CKD risk remain controversial.

One of the strengths of our study is the application of robust statistical methods, including propensity score matching, to real-world patients. The propensity score matching was based on numerous covariates and allowed us to reduce the impact of selection biases and compare two matched groups. The participants in our study were enrolled from multiple medical centers in Shanghai, and a multi-stage, stratified, random cluster sampling scheme was adopted to verify data accuracy. In addition, we not only evaluated the relationship of statin therapy with new-onset CKD and changes in eGFR in community residents, but also analyzed the impact of dyslipidemia on kidney function in both statin users and nonusers. At the same time, we examined the frequency of new-onset CKD in three follow-up visits, not just whether it occurred, which is an additional strength of our study.

In light of these results, several limitations should also be considered. First, we could not conclude whether the association depends on statin type, dosage, or treatment duration, thus, further research is needed. Second, this was a retrospective observational study, which is not a perfect substitute for a randomized trial. Third, we did not collect data regarding the presence of albuminuria, which limited the interpretation of the results. Finally, our findings were obtained from random sampling in Shanghai and are therefore inadequate for generalization to China as a whole. Thus, further validation of studies incorporating other regions would better depict the results of a nationwide population.

Conclusions

The results of this study suggest that statin use was significantly associated with a decreased risk of new-onset CKD and a slower decline in eGFR in community-dwelling older people in Shanghai, China. Meanwhile, dyslipidemia was a risk factor for CKD progression among both statin users and nonusers. Further randomized controlled studies are needed to confirm the renoprotective efficacy of statins among community-dwelling older people.

Abbreviations

CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; Scr, serum creatinine; BMI, body mass index; WHR, waist-to-hip ratio; SBP, systolic blood pressure; DBP, diastolic blood pressure; ACEIs/ARBs, angiotensin-converting-enzyme inhibitors/angiotensin receptor blockers; CCBs, calcium channel blockers; HbA1c, glycated hemoglobin; FPG, fasting plasma glucose; TC, total cholesterol; TG, triglycerides; HDL, high-density lipoprotein; LDL, low-density lipoprotein; AST, aspartate transaminase; ALT, alanine transaminase; BUN, blood urea nitrogen; TBIL, total bilirubin; UA, uric acid; CKD-EPI, Chronic Kidney Disease Epidemiology Collaboration; HR, hazard ratio; CI, confidence interval; HMG-CoA, hydroxymethylglutaryl-CoA.

Data Sharing Statement

All the data supporting the study findings are within the manuscript. Additional detailed information and raw data are available from the corresponding author on reasonable request.

Ethics Approval and Informed Consent

The study was performed in accordance with the Declaration of Helsinki and approved by the Ethics Committee of Tongji University. Written consent was obtained from each participant after they had been informed of the objectives, benefits, medical items, and confidentiality of personal information.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors report no conflicts of interest in this work.

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