Prevalence and severity of chronic kidney disease in a population with type 1 diabetes from a United States health system: a real-world cohort study

Katherine R. Tuttle,[a,b,c,*] Christina L. Reynolds,[a] Lindsey M. Kornowske,[a] Cami R. Jones,[a] Radica Z. Alicic,[a,c] Kenn B. Daratha,[a] Joshua J. Neumiller,[a,d] Carla Greenbaum,[e] Meda E. Pavkov,[f] Fang Xu,[f] O. Kenrik Duru,[g] Susanne B. Nicholas,[h] and Keith C. Norris,[h] on behalf of the CURE-CKD Consortium

[a]Providence Medical Research Center, Providence Inland Northwest Health, Spokane, WA, USA
[b]Nephrology Division and Kidney Research Institute, University of Washington School of Medicine, Seattle, WA, USA
[c]Department of Medicine, University of Washington School of Medicine, Seattle, WA, USA
[d]College of Pharmacy and Pharmaceutical Sciences, Washington State University, Spokane, WA, USA
[e]Center for Intervention Immunology and Diabetes Program, Benaroya Research Institute, Seattle, WA, USA
[f]Division of Diabetes Translation, Centers for Disease Control and Prevention, Atlanta, GA, USA
[g]Division of General Internal Medicine and Health Services Research, David Geffen School of Medicine, University of California, Los Angeles, CA, USA
[h]Division of Nephrology, Department of Medicine, David Geffen School of Medicine, University of California, Los Angeles, CA, USA

The Lancet Regional Health - Americas 2025;47: 101130

Published Online 15 May 2025
https://doi.org/10.1016/j.lana.2025.101130

Summary

Background A contemporary description and estimates for rates of chronic kidney disease (CKD) in type 1 diabetes are needed to inform risk reduction strategies. The study aim was to assess prevalence and severity of CKD based on a population with type 1 diabetes receiving care at a large United States health system.

Methods Type 1 diabetes was identified through the Providence health system electronic health records during 2013–2022. Prevalent CKD was defined cross-sectionally by ≥ 90-day persistence of estimated glomerular filtration rate (eGFR) < 60 mL/min/1.73 m^2, urine albumin-to-creatinine ratio ≥30 mg/g, or urine protein-to-creatinine ratio ≥0.15 g/g. Multivariable logistic regression models analyzed variable associations with CKD and severe kidney disease (eGFR < 45 mL/min/1.73 m^2, dialysis, or transplant).

Findings The study population (N = 23,589) was 48.6% female with a mean ± SD age of 38 ± 17 years. CKD prevalence was 27.1%. Higher odds of CKD were found for females (odds ratio: 1.36 [95% confidence interval]: 1.26–1.47); age 60–79 years (reference 12–17 years; 2.22 [1.83–2.69]); Asian (reference White; 1.71 [1.20–2.44]), Black or African American (1.76 [1.45–2.14]), and Other race (1.33 [1.04–1.71]) populations. CKD odds were higher with hypertension, heart failure, and atherosclerotic cardiovascular disease. Severe kidney disease was present in 10.8% with higher odds among Black or African American (2.08 [1.23–3.54]) and Native Hawaiian or Pacific Islander (2.62 [1.28–5.38]) populations.

Interpretation CKD was present in nearly one of three persons with type 1 diabetes with higher risks for females, older adults, racial and ethnic minorities, and those with cardiovascular diseases. Severe kidney disease was found in over one-tenth and more likely in Black or African American and Native Hawaiian or Pacific Islander populations. Focus on disproportionately affected groups who may benefit from monitoring and interventions to improve clinical outcomes will be important for public health and health system strategies to reduce risks of CKD and severe kidney disease in type 1 diabetes.

Funding This work was supported in part by CDC project numbers 75D301-21-P-12254 and 75D301-23-C-18264, and in part by Brigham Research Institute.

Keywords: Diabetic kidney disease; Epidemiology; Kidney failure; Diversity; Cardiovascular disease

*Corresponding author. 105 W 8th Avenue, Suite 250E, Spokane, WA 99204, USA.
E-mail address: katherine.tuttle@providence.org (K.R. Tuttle).

Research in context

Evidence before this study

We conducted PubMed searches from June 15 to July 31, 2024, with no restrictions on time or language, using search terms including: "type 1 diabetes" AND "chronic kidney disease" or "CKD"; "type 1 diabetes" AND "diabetic kidney disease" or "DKD"; "type 1 diabetes" AND "kidney failure" or "dialysis" or "transplant"; type 1 diabetes AND "CKD epidemiology" or "CKD prevalence"; type 1 diabetes AND "DKD epidemiology" or "DKD prevalence". Chronic kidney disease (CKD) is a major diabetes complication. Racial and ethnic minorities have disproportionally high rates of CKD in type 2 diabetes, but data on prevalence and severity of CKD representing the diversity of the type 1 diabetes population were not found because prior studies were limited to mostly White or European populations.

Added value of this study

We estimated CKD prevalence as well as the likelihood of CKD or severe kidney disease in a large real-world population (N = 23,589) with type 1 diabetes from the United States during 2013–2022. CKD was present in 27.1% with no decrease over this decade despite many advances in diabetes care. Females, older adults, racial and ethnic minorities, and those with cardiovascular diseases were more likely to have CKD. In the overall type 1 diabetes population, more than one in ten had severe kidney disease with eGFR < 45 mL/min/1.73 m^2 in 5.3% and treatment for kidney failure by dialysis or transplant in 5.5%. Black or African American and Native Hawaiian or Pacific Islander populations had higher odds of severe kidney disease.

Implications of all the available evidence

CKD and severe kidney disease are common in type 1 diabetes. Females, older adults, racial and ethnic minorities, and those with cardiovascular diseases are disproportionately affected. Focus on these groups for awareness, detection, and intervention is an important strategy for reducing risks of CKD in type 1 diabetes.

Introduction

Type 1 diabetes is present in 8.75 million people worldwide.[1] Despite efforts to mitigate risks by management of hyperglycaemia and concurrent risk factors such as hypertension, chronic kidney disease (CKD) remains one of the most common diabetes complications. CKD has historically been expected in about one of three persons with type 1 diabetes and clusters with other microvascular complications.[2,3] Development of albuminuria heralds CKD onset, presaging glomerular filtration rate (GFR) decline, kidney failure, and death.[2] Racial and ethnic minorities have higher risk of developing CKD and kidney failure, especially from type 2 diabetes, but little is known about their CKD risks in type 1 diabetes because prior studies have been comprised of predominantly White or European populations.[4–8] Moreover, these studies have been limited by use of administrative codes to identify CKD, high rates of missingness for laboratory assessments, particularly albuminuria, and extrapolations from small sample sizes.[5–8]

Contemporary estimates and descriptions of CKD in type 1 diabetes are needed to inform risk reduction strategies now that highly efficacious therapies for CKD have become available. For type 2 diabetes, the four "pillars of therapy" for CKD include conventional renin-angiotensin system inhibitors as well as newer agents in the sodium glucose cotransporter 2 (SGLT2) inhibitor, glucagon-like peptide-1 (GLP-1) receptor agonist, and non-steroidal mineralocorticoid antagonist classes.[9–13] However, persons with type 1 diabetes were excluded from most clinical trials of newer agents for CKD, and thus have not similarly benefited from therapeutic advances. The last drug shown to improve CKD outcomes in type 1 diabetes was an angiotensin converting enzyme (ACE) inhibitor over three decades ago.[14] Therefore, the aim of this study was to assess CKD prevalence and severity in a large real-world population with type 1 diabetes to help identify individuals who may benefit from monitoring and interventions to improve clinical outcomes.

Methods

Data source

The Center for Kidney Disease Education and Hope (CURE-CKD) is a clinical registry derived from electronic health records (EHRs) of the Providence health system that contains demographics, physical and laboratory measurements, prescription records, and administrative codes.[15,16] The Providence health system includes a large clinical network spanning from tertiary care to community-based primary care centres across five western states (Washington, Montana, Oregon, Alaska, and California). Registry data are extracted and curated for persons with CKD or at risk of CKD due to diabetes or hypertension. Diabetes is identified by haemoglobin (Hb) A1c ≥ 6.5%; two fasting blood glucose measures ≥126 mg/dL, or two random blood glucose measures ≥200 mg/dL between 1 day and 2 years apart; a prescription for blood glucose-lowering agents; or diagnosis codes.[15] The Chronic Kidney Disease Epidemiologic 2021 equation is used to calculate estimated GFR (eGFR) from serum creatinine.[17] CKD is defined by the first of two laboratory measurements ≥90 days apart (eGFR < 60 mL/min/1.73 m^2, urine albumin-to-creatinine ratio (UACR) ≥30 mg/g, or urine protein-to-creatinine ratio (UPCR) ≥0.15 g/g). ICD-10

diagnosis and ICD-9 or ICD-10 procedure codes are also used to detect CKD, dialysis, or kidney transplant Supplementary Table S1).

Study population

Persons ≥ 12 years old were selected for a type 1 diabetes cohort from the overall diabetes population in the CURE-CKD registry by an EHR algorithm (Supplementary nathods) between January 1, 2013 and December 31, 2022. Those with type 1 diabetes and laboratory measures of serum creatinine, UACR, or UPCR were included. To assess for temporal trends in CKD prevalence, the 2013–2022 cohort was subdivided into two study periods (January 1, 2013 until December 31, 2017, and January 1, 2018 until December 31, 2022) resulting in three cross-sectional cohorts from years 2013–2022 (N = 23,589), 2013–2017 (N = 13,645), and 2018–2022 (N = 19,941; Supplementary Fig. S2).

At entry into the type 1 diabetes cohort, sex, race and ethnicity, and age were recorded. Systolic blood pressure, body mass index (BMI), and HbA1c measures were averaged over the study periods. Hypertension status,[15,16] primary health insurance type, International Classification of Diseases (ICD)-10 codes for atherosclerotic cardiovascular disease and heart failure (Supplementary Table S1), medication use by prescription records, and healthcare encounters by specialty type (Nephrology, Endocrinology, Internal Medicine, Family Medicine) were also recorded.

The study was conducted according to Reporting of Observational Studies in Epidemiology guidelines (Supplementary materials STROBE checklist), and was approved by the Providence Institutional Review Board, who determined a written informed consent was not required for analyses of a limited retrospective dataset.

Statistical analysis

The sample size was based upon the number of persons identified with type 1 diabetes in the Providence health system (Supplementary Figs. S1 and S2). Categorical variables were displayed as frequencies and percentages. Continuous normally distributed variables were reported as mean ± standard deviation (SD). Continuous skewed variables were reported as median and interquartile range (IQR). Persons with type 1 diabetes were described by demographic, clinical, and healthcare characteristics according to CKD status. The prevalence of CKD was estimated for each study period, and stratified by eGFR values of 45–59, 30–44, 15–29, and <15 mL/min/1.73 m^2 based on the most recent measurement.

Chi-squared tests assessed for differences in CKD prevalence between the study periods of 2013–2017 and 2018–2022. Multivariable logistic regression models examined primary and secondary outcomes for patients with available measures, respectively: 1. CKD (eGFR < 60 mL/min/1.73 m^2, UACR ≥ 30 mg/g, or UPCR ≥ 0.15 g/g, or CKD diagnosis code; n = 19,483) and 2. Severe kidney disease (eGFR < 45 mL/min/1.73 m^2, dialysis, or transplant; n = 12,021). Variable selection and cut points were based on clinically recognized CKD risk factors.[11] The exposure variables included: sex (reference: male), race and ethnicity (reference: White, non-Hispanic), age (reference: 12–17 years), BMI (≥30 kg/m^2 versus <30 kg/m^2), HbA1c (≥8% versus <8%),[18] commercial health insurance (reference: non-commercial), and dichotomous variables including medications, specialty type of healthcare encounters, hypertension, heart failure, and atherosclerotic cardiovascular disease. UACR ≥ 300 mg/g and UPCR ≥ 0.5 g/g were included as variables in the model for the secondary outcome. Both unadjusted and adjusted estimates for associations of exposure variables with the primary and secondary outcomes were calculated. In sensitivity analyses, models were also fit with baseline age (per decade), HbA1c (per 1%) and BMI (per 5 kg/m^2) as continuous variables. Missing values were not imputed. The significance level was set at 0.05. Analyses were performed in R version 4.2.2.

Role of the funding source

A sponsor of the study, the United States Centers for Disease Control and Prevention (CDC), reviewed and cleared the manuscript for publication, as required by CDC policy. Benaroya Research Institute provided funding to support the EHR algorithm to identify persons with type 1 diabetes. These institutions did not participate in the writing or decision to submit for publication. Two authors (MEP and FX) are employees of the CDC. CG is an employee of the Benaroya Research Institute.

Results

Characteristics of the type 1 diabetes population

During 2013–2022, the overall type 1 diabetes population (N = 23,589) included 48.6% females. Their racial and ethnic identities comprised 1.0% American Indian or Alaska Native, 2.5% Asian, 3.4% Black or African American, 0.4% Native Hawaiian or Pacific islander, 3.2% Hispanic, 6.6% Other race, 78.4% White, and 4.5% Unknown (Table 1). They were on average 38 ± 17 years of age at entry into the type 1 diabetes cohort. Their most recently recorded values were mean eGFR 91.1 ± 30.8 mL/min/1.73 m^2 and median UACR and UPCR 9.6 (5.0–31.0) mg/g and 0.3 (0.1–1.5) g/g, respectively. Measurement of eGFR was available for 99.0% with a median of 8 (3–19) measurements per patient. UACR and UPCR were measured in 54.4% and 3.8%, respectively with a median of 1 (0–2) measurement. Mean values for risk factors were: HbA1c 8.4 ± 1.8%, BMI 27 ± 6 kg/m^2, and systolic blood pressure 125 ± 14 mm Hg. The majority (>90%) of persons with type 1 diabetes were seen in primary care (Family Medicine or Internal Medicine) and half were seen

Persons, n (%) of total	Overall 23,589 (100.0)	With CKD 6396 (27.1)	Without CKD 17,193 (72.9)
Demographics and clinical characteristics			
Sex, n (%)			
Female	11,465 (48.6)	3274 (51.2)	8191 (47.6)
Male	12,124 (51.4)	3122 (48.8)	9002 (52.4)
Race and ethnicity, n (%)			
American Indian or Alaska Native	225 (1.0)	90 (1.4)	135 (0.8)
Asian	587 (2.5)	162 (2.5)	425 (2.5)
Black or African American	808 (3.4)	301 (4.7)	507 (2.9)
Hispanic or Latino(a)	752 (3.2)	171 (2.7)	581 (3.4)
Native Hawaiian or Pacific Islander	93 (0.4)	23 (0.4)	70 (0.4)
White	18,496 (78.4)	5106 (79.8)	13,390 (77.9)
Other[a]	1567 (6.6)	374 (5.8)	1193 (6.9)
Unknown	1061 (4.5)	169 (2.6)	892 (5.2)
Age, y, mean, SD	38, 17	48, 17	35, 16
Age category, y, n (%)			
12–17	3022 (12.8)	257 (4.0)	2765 (16.1)
18–39	10,319 (43.7)	1912 (29.9)	8407 (48.9)
40–59	7087 (30.0)	2448 (38.3)	4639 (27.0)
60–79	2999 (12.7)	1651 (25.8)	1348 (7.8)
≥80	162 (0.7)	128 (2.0)	34 (0.2)
eGFR, mL/min/1.73 m^2, n (%)	23,350 (99.0)	6385 (99.8)	16,965 (98.7)
Mean, SD	91.1, 30.8	61.8, 34.0	102.2, 20.7
UACR, mg/g, n (%)	12,844 (54.4)	3876 (60.6)	8968 (52.2)
Median (IQR)	9.6 (5.0–31.0)	49.0 (14.0–238.0)	7.0 (4.0–14.0)
UACR category, n (%)			
<30	9558 (40.5)	1524 (23.8)	8034 (46.7)
30–300	2287 (9.7)	1481 (23.2)	806 (4.7)
>300	999 (4.2)	871 (13.6)	128 (0.7)
UPCR, g/g, n (%)	900 (3.8)	607 (9.5)	293 (1.7)
Median (IQR)	0.3 (0.1–1.5)	0.6 (0.2–2.4)	0.1 (0.1–0.3)
UPCR category, n (%)			
<0.15	305 (1.3)	142 (2.2)	163 (0.9)
0.15–0.50	230 (1.0)	149 (2.3)	81 (0.5)
>0.50	365 (1.5)	316 (4.9)	49 (0.3)
HbA1c %, n (%)	20,048 (85.0)	5847 (91.4)	14,201 (82.6)
Mean, SD	8.4, 1.8	8.6, 1.8	8.4, 1.9
Systolic blood pressure, mm Hg, n (%)	22,648 (96.0)	6308 (98.6)	16,340 (95.0)
Mean, SD	125, 14	130, 15	123, 13
BMI, kg/m^2, n (%)	22,867 (96.9)	6319 (98.8)	16,548 (96.2)
Mean, SD	27, 6	28, 6	27, 6
Hypertension	14,932 (63.3)	5736 (89.7)	9196 (53.5)
Heart failure	1412 (6.0)	1158 (18.1)	254 (1.5)
Atherosclerotic cardiovascular diseases	3944 (16.7)	2460 (38.5)	1484 (8.6)
Medications, n (%)			
ACE inhibitor or ARB	11,281 (47.8)	5027 (78.6)	6254 (36.4)
GLP-1 receptor agonist	1146 (4.9)	375 (5.9)	771 (4.5)
SGLT2 inhibitor	694 (2.9)	259 (4.0)	435 (2.5)
Healthcare characteristics			
Clinical encounter types, n (%)			
Family medicine	14,033 (59.5)	4001 (62.6)	10,032 (58.3)
Internal medicine	7507 (31.8)	2655 (41.5)	4852 (28.2)
Pediatrics	741 (3.1)	175 (2.7)	566 (3.3)
Endocrinology	11,778 (49.9)	3091 (48.3)	8687 (50.5)
Nephrology	1260 (5.3)	1024 (16.0)	236 (1.4)
Primary health insurance, n (%)			
Commercial	14,675 (62.2)	2361 (36.9)	12,314 (71.6)

(Table 1 continues on next page)

by Endocrinology. Commercial health insurance was the primary payer for 62.2% while 25.2% had Medicare.

Similar patient characteristics were observed during the 2013–2017 (N = 13,645) and 2018–2022 (N = 19,941) study periods (Supplementary Table S2). The larger number of cases in the more recent timeframe is related to growth of the population accessing care in the Providence health system. The median observation window for 2013–2022 was 6.1 years (2.5–8.7). During 2013–2017 and 2018–2022 it was 3.0 years (1.1–4.4) and 3.5 years (1.4–4.7), respectively.

Frequency and severity of chronic kidney disease in type 1 diabetes

CKD prevalence in the overall type 1 diabetes population was 27.1% (6396/23,589) during 2013–2022 with prevalences of 27.0% in 2013–2017 and 27.3% in 2018–2022 (p = 0.53; Table 2). CKD prevalence by demography was also similar across study periods. CKD was more common in females (28.6%) than in males (25.8%) and highest among American Indian or Alaska Native (40.0%) and Black or African American (37.3%) populations (Table 2). The CKD group was older than those without CKD (48 ± 17 and 35 ± 16 years). While prevalence was higher with age such that 55.1% of those in the 60–79-year old group and 79.0% of those ≥80 years had CKD, 8.5% of the 12–17-year old group already had CKD. In the CKD group versus those without CKD, hypertension (89.7% and 53.5%), heart failure (18.1% and 1.5%), and atherosclerotic cardiovascular disease (38.5% and 8.6%) were more common. ACE inhibitors or ARBs were prescribed more often to those with versus without CKD (78.6% and 36.4%). GLP-1 receptor agonist and SGLT2 inhibitor use were low, but slightly higher in the CKD group (GLP-1 receptor agonists: 5.9% and 4.5%; SGLT2 inhibitors: 4.0% and 2.5%). A greater proportion with CKD than without CKD were seen by Nephrology (16.0% and 1.4%).

CKD was identified by eGFR < 60 mL/min/1.73 m^2 in 73.5% (n = 4701), while 19.0% (n = 1215) were identified by UACR ≥ 30 mg/g or UPCR ≥ 0.15 g/g in the overall type 1 diabetes population. Few persons were identified with CKD by only a diagnosis code (n = 428, 6.7%) or by codes for dialysis or kidney transplant (n = 52, 0.8%). With a more liberal definition not requiring more than one laboratory value ≥ 90 days apart for eGFR, UACR, or UPCR, CKD prevalence would be 33.3% (7844/23,589). An eGFR < 45 mL/min/1.73 m^2 occurred in 5.3%, and kidney replacement therapy by dialysis or transplant was present in 3.6% and 1.9%, respectively (Fig. 1A).

Among the type 1 diabetes population with CKD, the eGFR distribution was 15.6% for eGFR 45–59 mL/min/1.73 m^2, 9.8% for eGFR 30–44 mL/min/1.73 m^2, 5.7% for eGFR 15–29 mL/min/1.73 m^2, and 1.8% for eGFR < 15 mL/min/1.73 m^2 (Fig. 1B). Kidney replacement therapy by dialysis or transplant occurred in 13.3%, and

6.9% of the CKD population, respectively. Presence of elevated albuminuria defined as UACR 30–300 mg/g or UACR > 300 mg/g was present in 23.2% and 13.6% of those with CKD, respectively (Table 1). Elevated proteinuria defined as UPCR 0.15–0.50 g/g or UPCR > 0.50 g/g was present in 2.3% and 4.9%, respectively. Presence of both eGFR < 60 mL/min/1.73 m^2 and elevated UACR or UPCR occurred in 19.7% (n = 1259) of the type 1 diabetes population with CKD.

Associations of type 1 diabetes characteristics with chronic kidney disease or severe kidney disease

By multivariable adjusted logistic regression modelling, higher odds of CKD (Fig. 2 and Supplementary Table S3) were found for females (odds ratio 1.36 [95% confidence interval: 1.26–1.47]), and for Asian (1.71 [1.20–2.44]), Black or African American (1.76 [1.45–2.14]), and Other race (1.33 [1.04–1.71]) populations. Odds of CKD increased progressively with age particularly in those aged 60–79 years (2.22 [1.83–2.69]) and ≥80 years (10.43 [6.16–18.41]). Persons with a mean HbA1c ≥ 8.0% had higher odds of CKD (1.80 [1.66–1.95]). Higher odds of CKD were also observed in persons with hypertension (2.69 [2.41–3.02]), heart failure (3.69 [3.13–4.38]), atherosclerotic cardiovascular diseases (1.91 [1.73–2.11]), ACE inhibitor or ARB use (2.84 [2.59–3.10]), and Nephrology (7.70 [6.49–9.16]) or Internal Medicine (1.21 [1.12–1.31]) encounters. With covariate adjustment, lower odds of CKD were observed in the American Indian or Alaska Native population (0.79 [0.63–0.98]) and for persons with commercial insurance (0.46 [0.42–0.50]) or BMI ≥ 30 kg/m^2 (0.83 [0.76–0.90]). Inclusion of age, HbA1c, and BMI as continuous variables in the model, yielded similar results as the main analysis (Supplementary Fig. S3 and Table S5).

Similar results were found in the multivariable adjusted logistic regression model for severe kidney disease except that the Native Hawaiian or Pacific Islander population had higher odds (2.62 [1.28–5.38]) (Fig. 3 and Supplementary Table S4). Severe kidney disease was not significantly related to female sex, American Indian or Alaska Native, Asian and Other race populations. Lower odds were observed for persons with HbA1c ≥ 8.0% and Family Medicine encounters. Comparable results were observed with modelling age, HbA1c, and BMI as continuous variables (Supplementary Fig. S4 and Table S6) with significantly lower odds of severe kidney disease for higher degrees of HbA1c (per 1%, 0.92 [0.85–1.00]) and BMI (per 5 kg/m^2, 0.89 [0.80–0.98]).

Discussion

CKD was found in 27.1% of a racially and ethnically diverse population with type 1 diabetes from a large United States health system in 2013–2022. No decrease in CKD prevalence was observed during this decade despite many advances in diabetes care. In the overall type 1 diabetes population, 5.3% had an eGFR < 45 mL/min/1.73 m^2 and 5.5% received treatment for kidney failure by dialysis or transplant.

The representativeness of these estimates for prevalence of CKD and severe kidney disease in type 1 diabetes are reflected by similarities between the Providence health system in the United States and the national Danish Register.[3] Persons with type 1 diabetes in these systems may be receiving attention to kidney health assessments by laboratory testing. In contrast, lower rates were reported in national registries from Sweden and Norway where CKD was solely identified by diagnosis codes, which may underestimate prevalence due to under coding or less testing.[5,6] A single centre in Ireland reported a CKD prevalence of 18% in type 1

Persons, n (%) of total	Overall 23,589 (100.0)	With CKD 6396 (27.1)	Without CKD 17,193 (72.9)
(Continued from previous page)			
Medicare	5941 (25.2)	3402 (53.2)	2539 (14.8)
Medicaid	2659 (11.3)	603 (9.4)	2056 (12.0)
Unknown	313 (1.3)	30 (0.5)	283 (1.6)

CKD = chronic kidney disease; y = years; SD = standard deviation; eGFR = estimated glomerular filtration rate; UACR = urine albumin-to-creatinine ratio; IQR = interquartile range; UPCR-urine protein-to-creatinine ratio; Hb = haemoglobin; BMI = body mass index; ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; GLP-1 = glucagon-like peptide; SGLT2 = sodium-glucose co-transporter. [a]Includes persons who did not identify with main census categories.

Table 1: **Characteristics of the population with type 1 diabetes overall and by CKD status.**

CKD prevalence	2013–2022	2013–2017	2018–2022
Type 1 Diabetes, n with CKD/total N (%)	6396/23,589 (27.1)	3684/13,645 (27.0)	5447/19,941 (27.3)
n with CKD/total by sex N (%)			
Female	3274/11,465 (28.6)	1887/6706 (28.1)	2802/9694 (28.9)
Male	3122/12,124 (25.8)	1797/6939 (25.9)	2645/10,247 (25.8)
n with CKD/total race and ethnicity N (%)			
American Indian Alaska Native	90/225 (40.0)	51/146 (34.9)	79/195 (40.5)
Asian	162/587 (27.6)	86/292 (29.5)	134/503 (26.6)
Black or African American	301/808 (37.3)	166/431 (38.5)	248/675 (36.7)
Hispanic or Latino(a)	171/752 (22.7)	52/258 (20.2)	159/676 (23.5)
Native Hawaiian Pacific Islander	23/93 (24.7)	11/47 (23.4)	18/78 (23.1)
White	5106/18,496 (27.6)	3059/11,199 (27.3)	4347/15,662 (27.8)
Other[a]	374/1567 (23.9)	177/767 (23.1)	322/1297 (24.8)
Unknown	169/1061 (15.9)	82/505 (16.2)	140/855 (16.4)
n with CKD/total age category, y, N (%)			
12–17	257/3022 (8.5)	112/1577 (7.1)	248/2701 (9.2)
18–39	1912/10,319 (18.5)	1110/6000 (18.5)	1691/8648 (19.6)
40–59	2448/7087 (34.5)	1436/4146 (34.6)	2071/6046 (34.3)
60–79	1651/2999 (55.1)	929/1796 (51.7)	1363/2457 (55.5)
≥80	128/162 (79.0)	97/126 (77.0)	74/89 (83.1)

CKD = chronic kidney disease; y = years. [a]Includes persons who did not identify with main census categories.

Table 2: **CKD prevalence in type 1 diabetes population by demographics during study periods.**

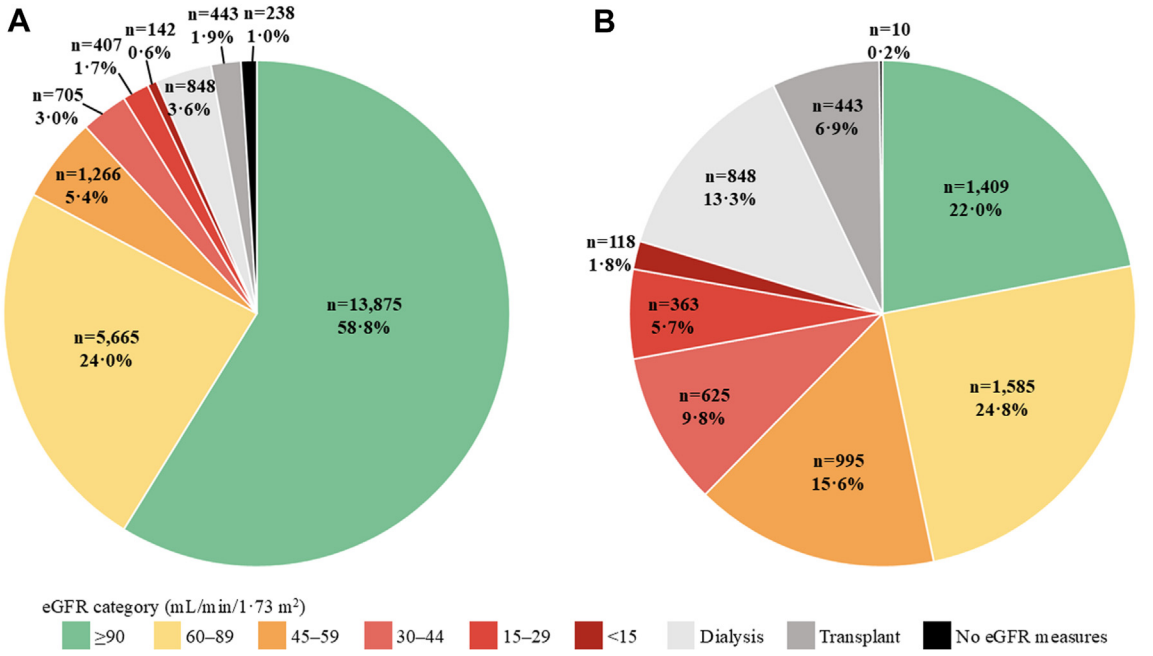

Fig. 1: Distribution of eGFR and kidney replacement therapy by dialysis or transplant in the type 1 diabetes population. A. Overall (N = 23,589) B. CKD (N = 6396). eGFR = estimated glomerular filtration rate; CKD = chronic kidney disease.

diabetes, but this is a low-range estimate due to high missingness for albuminuria.[7] An analysis of type 1 diabetes from the National Health and Nutrition Examination Survey in the United States estimated CKD prevalence at 21.5% based on a single laboratory assessment.[8] However, that analysis has considerable uncertainties because it was based on extrapolation with population weighting from just 40 individuals with type 1 diabetes who had both eGFR and UACR measurements. All these studies lacked the broad racial and ethnic diversity represented in the CURE-CKD data from the Providence health system.

Our findings underscore a persistently high CKD burden in a type 1 diabetes population with notable disparities that can help to guide strategies to improve awareness, detection, and intervention. Females were 36% more likely to have CKD, although they did not have higher odds than males of severe kidney disease, an observation aligned with the general CKD population and likely related to higher rates of disease progression and death in men.[4,6,16,19,20] Asian, Black or African American, and Other race populations were more likely to have CKD. However, only Black or African American and Native Hawaiian or Pacific Islander populations were more likely to have severe kidney disease by a magnitude of two-to-three-fold. Thus, among these populations with type 1 diabetes, higher odds of disease progression overlay their increased risks of incident CKD.[20] In the American Indian or Alaska Native population, the odds of CKD were lower after adjustment for covariates. Kidney failure incidence dropped by greater than half two decades after the Indian Health Service in the United States instituted a population health approach to optimize diabetes care and use of renin angiotensin system inhibitors.[21] As a result, persons with type 1 diabetes in the present American Indian or Alaska Native population also may have benefited by reduced risk of CKD. Although CKD independently associated with older age, it was common across all ages. Of note, the prevalence of CKD was 8.5% in adolescents aged 12–17 years, comparable to a prior study of type 1 diabetes in youth.[22] These observations highlight the importance of recognizing CKD early in type 1 diabetes and addressing lifelong risks.

Microvascular complications can be attenuated by intensive glycaemic control in type 1 diabetes.[23] Nonetheless, in our population, glycaemic control was suboptimal as reflected in an average HbA1c of approximately 8.5%. Higher HbA1c associated with CKD, although the reverse was the case for severe kidney disease. The latter observation may be explained by controlling for albuminuria or proteinuria, a strong risk factor for loss of kidney function that may confound an association with glycaemia. Higher BMI and CKD were also inversely associated, which may be due to an obesity paradox.[24] However, both observations could relate to reverse causality through effects of reduced kidney function to lower glycaemia and cause weight loss.[24,25] The association of CKD with renin-angiotensin system inhibitors probably signifies indication for use

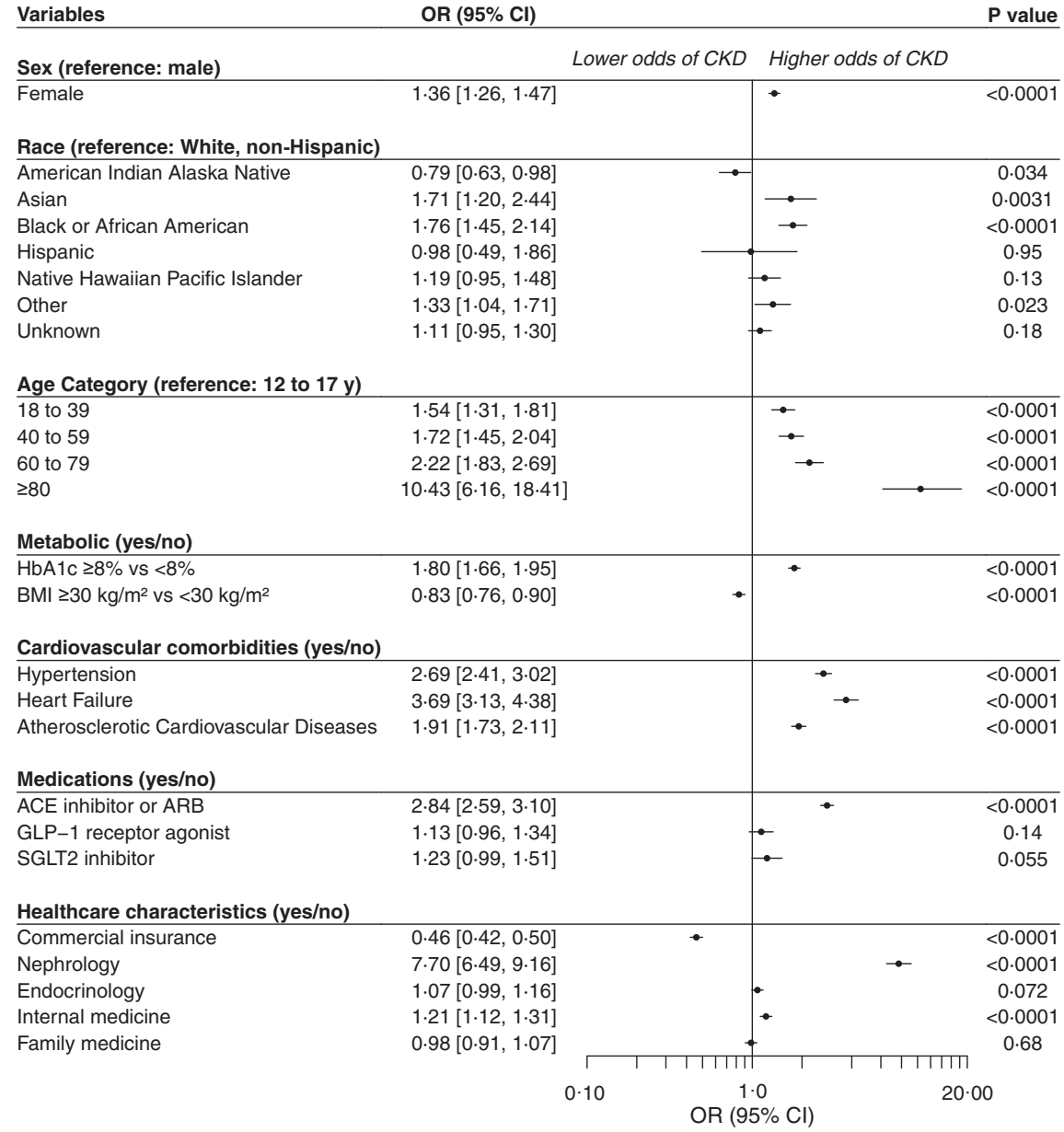

Fig. 2: Adjusted associations of demographic, clinical, and healthcare characteristics with CKD in type 1 diabetes and measures for BMI and HbA1c (N = 19,483). CKD: Administrative codes, eGFR < 60 mL/min/1.73 m^2, UACR ≥ 30 mg/g, or UPCR ≥0.15 g/g. CKD = chronic kidney disease; eGFR-estimated glomerular filtration rate; UACR-urine albumin-to-creatinine ratio; UPCR-urine protein-to-creatinine ratio; OR = odds ratio; CI = confidence interval; y = years; Hb = haemoglobin; BMI = body mass index; ACE = angiotensin converting enzyme; ARB = angiotensin receptor blocker; GLP-1 = glucagon-like peptide; SGLT2-sodium-glucose cotransporter.

by virtue of CKD or hypertension. Indeed, in the group with type 1 diabetes and CKD, blood pressure was well-controlled and ACE inhibitor or ARB use was 78.6%. However, these medicines are often stopped and not restarted, so their use may be overestimated.[26] The type 1 diabetes population with CKD is encumbered by multiple comorbidities. Hypertension was present in nearly 90%. Together, heart failure and atherosclerotic cardiovascular disease occurred in over half. An eGFR < 45 mL/min/1.73 m^2 or kidney replacement therapy by dialysis or transplant, were recorded for about one third of those with CKD. Once kidney function declined to this point, care for persons with type 1 diabetes was more likely from nephrologists than other specialties.

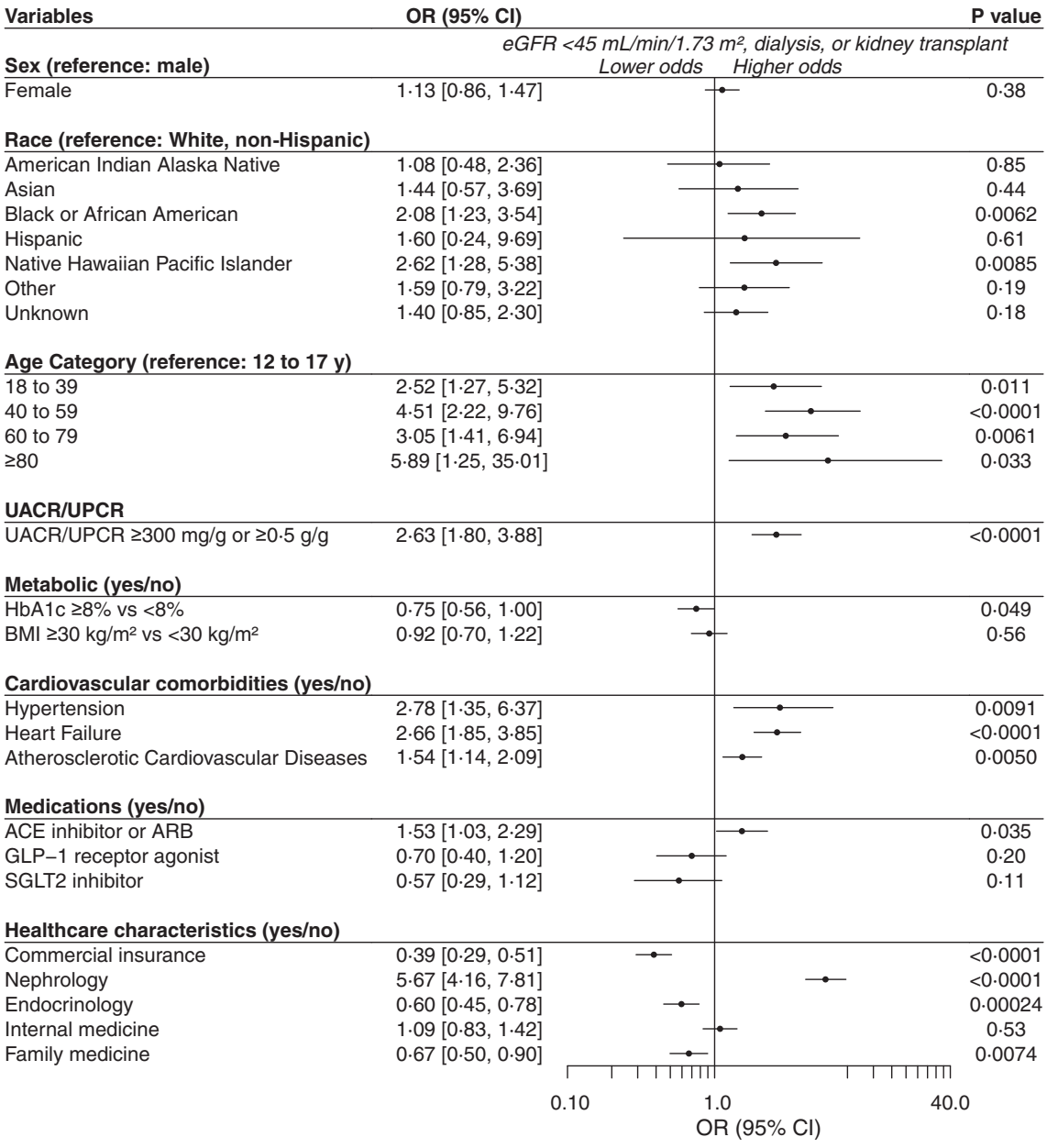

Fig. 3: Adjusted associations of demographic, clinical, and healthcare characteristics for severe kidney disease in type 1 diabetes and measures for UACR/UPCR (N = 12,021). Severe kidney disease: eGFR < 45 mL/min/1.73 m^2 or kidney replacement therapy by dialysis or transplant. eGFR = estimated glomerular filtration rate; OR = odds ratio; CI = confidence interval; y = years; UACR = urine albumin-to-creatinine ratio; UPCR = urine protein-to-creatinine ratio; Hb = haemoglobin; BMI = body mass index; ACE = angiotensin converting enzyme; ARB = angiotensin receptor blocker; GLP-1 = glucagon-like peptide; SGLT2 = sodium-glucose cotransporter.

People with advanced CKD, often develop disabilities accompanied by financial and employment hardships.[27] Correspondingly, we found commercial health insurance less frequently in the CKD group and inversely associated with severe kidney disease. The state-of-affairs in type 1 diabetes stands in stark contrast to the treatment advances that have significantly reduced kidney and cardiovascular risks in type 2 diabetes.[28–31]

The strengths of our study include representation of broad racial and ethnic diversity in a population of 23,589 persons with type 1 diabetes during a contemporary 10-year timeframe. We primarily relied on laboratory tests rather than on administrative codes to overcome problems of under coding for CKD in EHR.[15,16,32] We also had a rigorous requirement for persistence of laboratory abnormalities to identify CKD.

Therefore, our CKD prevalence estimate is conservative. For example, about 6% of persons without CKD identification had elevated levels of albuminuria or proteinuria because persistence was not confirmed. If based on only a single laboratory abnormality, CKD prevalence would have been 33.3% rather than 27.1%. UACR measurements were available in 54.4%, which may also have contributed to a lower range estimate for CKD in this population with type 1 diabetes. Furthermore, incidence-prevalence bias introduced by variable observation time between individuals and competing risk of death would also be likely to underestimate CKD. Our study has several other noteworthy limitations. The EHR algorithm might not have captured all persons with type 1 diabetes in the health system. However, we generated a sizeable sample to generally describe the prevalence and severity of CKD in a real-world population that can serve as a foundation for further study. Although we have reported temporal trends, longitudinal analyses are needed to more clearly delineate associations of individual characteristics, including use of guideline-directed medical therapy, such as ACE inhibitors and ARBs, with CKD and cardiovascular outcomes. We also did not have data on other microvascular complications, age at onset or duration of type 1 diabetes, although age is an indirect yet imperfect proxy for the latter. In some groups, particularly those ≥80 years old, sample sizes were relatively small such that odds ratios for CKD and severe kidney disease had wide confidence intervals reflecting more uncertainty in risk estimates than for other model variables. Finally, these observations from a single United States health system may not be generalizable to other systems or settings.

In conclusion, CKD was present in nearly one of three persons with type 1 diabetes who received care at the Providence health system. Females, older adults, racial and ethnic minority groups, and those with cardiovascular diseases were more likely to have CKD. Substantially reduced kidney function or kidney failure treated by dialysis or transplant was observed in over one tenth of the type 1 diabetes population with higher odds of severe kidney disease in Black or African American and Native Hawaiian or Pacific Islander populations. Focus on disproportionately affected groups who may benefit from monitoring and interventions to improve clinical outcomes will be important for public health and health system strategies to reduce risks of CKD and severe kidney disease in type 1 diabetes.

Contributors

All authors met the International Committee of Medical Journal Editors criteria for authorship for the article. KRT led project development from concept, study design, and funding acquisition through data acquisition, curation, analyses, interpretation, literature searches, and writing the first and subsequent drafts of the manuscript. KRT was responsible for the decision to submit the manuscript. CLR, LMK, CRJ, RZA, KBD, OKD, SBN, and KCN contributed to data acquisition, curation, analyses, and reviewing and editing the manuscript. CLR, LMK, and CRJ created the figures. JJN, CG, MEP, and FX reviewed and edited the manuscript. KRT, CLR, LMK, CRJ, and KBD accessed and verified the data.

Data sharing statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions and would require a data use agreement.

Declaration of interests

The findings and conclusions are those of the authors and do not necessarily represent the official position of the CDC.

KRT is supported by NIH research grants R01MD014712, U2CDK114886, UL1TR002319, U54DK083912, U01DK100846, OT2HL161847, UM1AI109568, OT2OD032581, and CDC project numbers 75D301-21-P-12254 and 75D301-23-C-18264. She has also received investigator-initiated grant support from Travere Therapeutics Inc., Bayer, Benaroya Research Institute, and the Doris Duke Charitable Foundation. She reports consultancy fees from AstraZeneca, Boehringer Ingelheim, Eli Lilly, Novo Nordisk, Travere Therapeutics Inc, and Pfizer; and speaker fees from Novo Nordisk. Additionally, she reports being chair of data safety monitoring boards for the National Institute of Diabetes and Digestive and Kidney Disease, and George Clinical Institute, and member of the data safety monitoring board for AstraZeneca. She reports leadership roles as chair for the Diabetic Kidney Disease Collaborative for the American Society of Nephrology, chair for Kidney Week 2025 Program Committee, and a member of the American Heart Association/American College of Cardiology Cardiovascular-Kidney-Metabolic Guideline Committee.

CLR is supported by NIH research grant OT2OD032581 and CDC project numbers 75D301-21-P-12254 and 75D301-23-C-18264, and reports other support from Travere Therapeutics Inc. and the Doris Duke Charitable Foundation.

LMK is supported by a NIH research grant OT2OD032581 and CDC project numbers 75D301-21-P-12254 and 75D301-23-C-18264; and reports other support from Benaroya Research Institute and Travere Therapeutics Inc.

CRJ is supported by NIH research grants R01MD014712, OT2OD032581 and CDC project numbers 75D301-21-P-12254 and 75D301-23-C-18264; and reports other support from Benaroya Research Institute, Travere Therapeutics Inc., and the Doris Duke Charitable Foundation.

RZA is supported by NIH research grants OT2HL161847, OT2OD032581, U24TR001608 and CDC project numbers 75D301-21-P-12254 and 75D301-23-C-18264; and reports other support from Benaroya Research Institute, Travere Therapeutics Inc., the Doris Duke Charitable Foundation, Bayer AG, AstraZeneca, Novo Nordisk, The George Institute for Global Health, CareDx, and KBP BioSciences, and personal fees from Boehringer Ingelheim, Bayer Pharmaceuticals, and Eli Lilly. She also reports being a member of the Executive Council for the American College of Physicians, Washington Chapter.

KBD is supported by NIH research grants R01MD014712, OT2OD032581 and CDC project numbers 75D301-21-P-12254 and 75D301-23-C-18264; and reports other support from Benaroya Research Institute, Travere Therapeutics Inc., and the Doris Duke Charitable Foundation.

JJN is supported by a NIH research grant OT2OD032581 and reports personal fees and other support from American College of Clinical Pharmacy, Bayer AG, Sanofi, Novo Nordisk, Boehringer Ingelheim, Eli Lilly, Proteomics International, and Dexcom. He also reports being on the Board of Directors for the American Diabetes Association.

CG has received research support from Sanofi, Bristol Myers Squibb, and Takeda.

MEP has no disclosures to report.

FX has no disclosures to report.

OKD is supported by NIH research grants R25HL161609, R18DK122372, R01DK124503, R01DK127733, R01AI135029, P30AG021684, the CDC cooperative agreement U18DP006535, and

receives research support from the Patient-Centered Outcomes Research Institute and the Terasaki Institute of Biomedical Innovation.

SBN is supported by NIH research grants R01MD014712, RF00250-2022-0038, U2CDK129496 and P50MD017366, and CDC project number 75D301-21-P-12254; received research support from Travere Therapeutics Inc, Terasaki Institute of Biomedical Innovation, and personal fees and other support from AstraZeneca, Bayer AG, Gilead, Novo Nordisk, Boehringer Ingelheim, Eli Lilly, and Vertex. She also reports having a pending US Patent Provisional Application for a Novel Small Molecule Drug to Treat Diabetic Kidney Disease. Additionally, she participates in an NIH/NIDDK and PCORI advisory board for Health System Outreach to Eliminate Disparities in Living Kidney Transplants (STEPS), is President of Women in Nephrology, and a board member of the National Kidney Foundation of Southern California.

KCN is supported in part by NIH research grants UL1TR001881, P30AG021684, U2CDK129496 and P50MD017366, and reports personal fees from Atlantis Dialysis Inc and AstraZeneca.

Acknowledgements

This work was supported in part by the CDC project numbers 75D301-21-P-12254 and 75D301-23-C-18264, and by Benaroya Research Institute.

Appendix A. Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.lana.2025.101130.

References

1 International Diabetes Federation. *Type 1 diabetes estimates in children and adults*; 2022. https://diabetesatlas.org/atlas/t1d-index-2022/. Accessed July 7, 2024.
2 Alicic RZ, Rooney MT, Tuttle KR. Diabetic kidney disease: challenges, progress, and possibilities. *Clin J Am Soc Nephrol*. 2017;12:2032–2045.
3 Bjerg L, Hulman A, Carstensen B, Charles M, Jørgensen ME, Witte DR. Development of microvascular complications and effect of concurrent risk factors in type 1 diabetes: a multistate model from an observational clinical cohort study. *Diabetes Care*. 2018;41:2297–2305.
4 Clark-Cutaia MN, Rivera E, Iroegbu C, Squires A. Disparities in chronic kidney disease-the state of the evidence. *Curr Opin Nephrol Hypertens*. 2021;30:208–214.
5 Kristófi R, Bodegard J, Norhammar A, et al. Cardiovascular and renal disease burden in type 1 compared with type 2 diabetes: a two-country nationwide observational study. *Diabetes Care*. 2021;44:1211–1218.
6 Swartling O, Rydell H, Stendahl M, Segelmark M, Trolle Lagerros Y, Evans M. CKD progression and mortality among men and women: a nationwide study in Sweden. *Am J Kidney Dis*. 2021;78:190–199.e1.
7 Majeed MS, Ahmed F, Teeling M. The prevalence of chronic kidney disease and albuminuria in patients with type 1 and type 2 diabetes attending a single Centre. *Cureus*. 2022;14:e32248.
8 Rossing P, Groop PH, Singh R, Lawatscheck R, Tuttle KR. Prevalence of chronic kidney disease in type 1 diabetes among adults in the U.S. *Diabetes Care*. 2024;47:1395–1399.
9 de Boer IH, Khunti K, Sadusky T, et al. Diabetes management in chronic kidney disease: a consensus report by the American diabetes association (ADA) and kidney disease: Improving global outcomes (KDIGO). *Kidney Int*. 2022;102:974–989.
10 Kidney Disease. Improving Global Outcomes (KDIGO) Diabetes Work Group. KDIGO 2022 clinical practice guideline for diabetes management in chronic kidney disease. *Kidney Int*. 2022;102:S1–S127.
11 Kidney Disease. Improving global outcomes (KDIGO) CKD work group. KDIGO 2024 clinical practice guideline for the evaluation and management of chronic kidney disease. *Kidney Int*. 2024;105:S117–S314.
12 American Diabetes Association Professional Practice Committee. 11. Chronic kidney disease and risk management: standards of care in diabetes-2024. *Diabetes Care*. 2024;47:S219–S230.
13 Naaman SC, Bakris GL. Diabetic nephropathy: update on pillars of therapy slowing progression. *Diabetes Care*. 2023;46:1574–1586.
14 Lewis EJ, Hunsicker LG, Bain RP, Rohde RD. The effect of angiotensin-converting-enzyme inhibition on diabetic nephropathy. The Collaborative Study Group. *N Engl J Med*. 1993;329:1456–1462.
15 Norris KC, Duru OK, Alicic RZ, et al. Rationale and design of a multicenter chronic kidney disease (CKD) and at-risk for CKD electronic health records-based registry: CURE-CKD. *BMC Nephrol*. 2019;20:416.
16 Tuttle KR, Alicic RZ, Duru OK, et al. Clinical characteristics of and risk factors for chronic kidney disease among adults and children: an analysis of the CURE-CKD registry. *JAMA Netw Open*. 2019;2:e1918169.
17 Inker LA, Eneanya ND, Coresh J, et al. New creatinine- and cystatin C-based equations to estimate GFR without race. *N Engl J Med*. 2020;383:2219–2229.
18 Shurraw S, Hemmelgarn B, Lin Met al, for the Alberta Kidney Disease Network. Association between glycemic control and adverse outcomes in people with diabetes mellitus and chronic kidney disease: a population-based cohort study. *Arch Intern Med*. 2011;171:1920–1927.
19 United States Renal Data System. *2024 USRDS annual data report: Epidemiology of kidney disease in the United States. National institutes of health, national Institute of diabetes and digestive and kidney diseases, Bethesda, MD*. 2024.
20 Tuttle KR, Jones CR, Daratha KB, et al. Incidence of chronic kidney disease among adults with diabetes, 2015–2020. *N Engl J Med*. 2022;387:1430–1431.
21 Narva A. Population health for CKD and diabetes: lessons from the Indian Health Service. *Am J Kidney Dis*. 2018;71:407–411.
22 Kahkoska AR, Nguyen CT, Adair LA, et al. Longitudinal phenotypes of type 1 diabetes in youth based on weight and glycemia and their association with complications. *J Clin Endocrinol Metab*. 2019;104:6003–6016.
23 de Boer IH, DCCT/EDIC Research Group. Kidney disease and related findings in the diabetes control and complications trial/epidemiology of diabetes interventions and complications study. *Diabetes Care*. 2014;37:24–30.
24 Rhee CM, Ahmadi SF, Kalantar-Zadeh K. The dual roles of obesity in chronic kidney disease: a review of the current literature. *Curr Opin Nephrol Hypertens*. 2016;25:208–216.
25 Rhee CM, Kalantar-Zadeh K, Tuttle KR. Novel approaches to hypoglycemia and burnt-out diabetes in chronic kidney disease. *Curr Opin Nephrol Hypertens*. 2022;31:72–81.
26 Nicholas SB, Daratha KB, Alicic RZ, et al. Prescription of guideline-directed medical therapies in patients with diabetes and chronic kidney disease from the CURE-CKD registry, 2019–2020. *Diabetes Obes Metab*. 2023;25:2970–2979.
27 Alma MA, van der Mei SF, Brouwer S, et al. Sustained employment, work disability and work functioning in CKD patients: a cross-sectional survey study. *J Nephrol*. 2023;36:731–743.
28 Nuffield Department of Population Health Renal Studies Group, SGLT2 inhibitor Meta-Analysis Cardio-Renal Trialists' Consortium. Impact of diabetes on the effects of sodium glucose co-transporter-2 inhibitors on kidney outcomes: collaborative meta-analysis of large placebo-controlled trials. *Lancet*. 2022;400:1788–1801.
29 Agarwal R, Filippatos G, Pitt B, et al. Cardiovascular and kidney outcomes with finerenone in patients with type 2 diabetes and chronic kidney disease: the FIDELITY pooled analysis. *Eur Heart J*. 2022;43:474–484. Erratum in: Eur Heart J 2022; 43: 1989.
30 Perkovic V, Tuttle KR, Rossing P, et al. Effects of semaglutide on chronic kidney disease in patients with type 2 diabetes. *N Engl J Med*. 2024;391:109–121.
31 Heerspink HJ, Cherney DZ, Groop PH, et al. People with type 1 diabetes and chronic kidney disease urgently need new therapies: a call for action. *Lancet Diabetes Endocrinol*. 2023;11:536–540.
32 Carrero JJ, Fu EL, Vestergaard SV, et al. Defining measures of kidney function in observational studies using routine health care data: methodological and reporting considerations. *Kidney Int*. 2023;103:53–69.