Kidney Medicine

RESEARCH LETTER

Prevalence of Obesity and CKD Among Adults in the United States, 2017-2020



To the Editor:

Obesity is associated with the development and progression of chronic kidney disease (CKD) through direct effects on the kidney as well as via intermediate diseases like type 2 diabetes and hypertension.¹ In light of obesity's public health importance, the epidemiological relationship between obesity and CKD warrants further elucidation.^{1,2} We therefore compared the prevalence of reduced estimated glomerular filtration rate (eGFR) in US adults with and without obesity, diabetes, and hypertension. We also present the latest available estimates of obesity, diabetes, and hypertension prevalence in US adults with and without reduced eGFR.

We used data from the 2017-2020 (prepandemic) National Health and Nutrition Examination Survey, a cross-sectional survey of the civilian noninstitutionalized US population that includes an in-person interview and physical examination.³ The National Health and Nutrition Examination Survey was approved by the National Center for Health Statistics Research Ethics Review Board and participants provided consent. The examination response rate among adults was 43.9%. Standardized measurements of weight, height, and blood presure were conducted and blood samples taken. Obesity was defined as body mass index \geq 30 kg/m². Hypertension was defined as systolic blood presure ≥130 mm Hg or diastolic blood presure $\geq 80 \text{ mm Hg}$ (average of up to 3 measurements) or current medication use. Diabetes was defined by a health care provider's diagnosis or hemoglobin A1c

 Table 1. Prevalence of Chronic Kidney Disease Among Adults by Demographic and Health Characteristics, United States, 2017-2020 (Prepandemic)

Characteristic	nª	Prevalence, % (95% Cl)	Unadjusted Prevalence Ratio, (95% CI)	Adjusted Prevalence Ratio, (95% Cl)
Overall	7,852	5.7 (4.8-6.7)	_	_
Age, y				
20-39	2,378	0.4 (0.1-1.2)	0.3 (0.1-0.7)	0.4 (0.1-1.4)
40-59	2,609	1.6 (1.0-2.4)	Reference	Reference
≥60+	2,865	16.7 (15.1-18.5)	10.4 (7.2-15.1)	8.7 (5.9-12.7)
Sex				
Men	3,789	4.8 (3.9-5.7)	Reference	Reference
Women	4,063	6.5 (5.5-7.8)	1.4 (1.2-1.6)	1.3 (1.1-1.5)
Race and Hispanic origin ^b				
Non-Hispanic White	2,774	6.4 (5.2-7.8)	Reference	Reference
Non-Hispanic Black	1,989	9.1 (7.6-10.8)	1.4 (1.1-1.8)	1.6 (1.3-2.0)
Non-Hispanic Asian	938	2.3 (1.3-3.6)	0.4 (0.2-0.6)	0.4 (0.2-0.8)
Hispanic	1,774	2.2 (1.6-3.0)	0.3 (0.2-0.5)	0.5 (0.3-0.8)
Men				
Non-Hispanic White	1,379	5.0 (3.7-6.5)	Reference	Reference
Non-Hispanic Black	934	9.4 (7.6-11.5)	1.9 (1.3-2.8)	2.2 (1.6-3.3)
Non-Hispanic Asian	426	3.0 (1.4-5.6)	0.6 (0.3-1.2)	0.8 (0.4-1.7)
Hispanic	845	1.8 (1.0-2.9)	0.4 (0.2-0.6)	0.6 (0.3-1.0)
Women				
Non-Hispanic White	1,395	7.7 (6.3-9.4)	Reference	Reference
Non-Hispanic Black	1,055	8.9 (6.8-11.4)	1.2 (0.9-1.5)	1.2 (1.0-1.5)
Non-Hispanic Asian	512	1.7 (0.8-3.2)	0.2 (0.1-0.4)	0.2 (0.1-0.5)
Hispanic	929	2.6 (1.7-3.8)	0.3 (0.2-0.5)	0.5 (0.3-0.7)
Diabetes				
Yes	1,529	13.8 (11.4-16.4)	3.2 (2.7-3.7)	1.5 (1.2-1.9)
No	6,323	4.3 (3.6-5.1)	Reference	Reference
Hypertension				
Yes	3,947	9.6 (8.2-11.2)	6.0 (4.0-9.2)	2.2 (1.5-3.2)
No	3,156	1.6 (1.0-2.4)	Reference	Reference
Obesity				
Yes	3,283	6.6 (5.4-7.9)	1.3 (1.1-1.6)	1.1 (0.9-1.3)
No	4,372	5.0 (4.1-6.0)	Reference	Reference

Notes: CKD defined as an eGFR of 15 to 59 mL/min/1.73 m². Source: National Health and Nutrition Examination Survey.

^aMay not add up to 7,852 for all characteristics because of missing data

^b"Other" not shown separately but included in totals.

Abbreviations: CI, confidence interval; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate.

Kidney Medicine



Figure 1. Unadjusted prevalence of obesity, diabetes, and hypertension among adults with and without CKD, United States, 2017-2020 (prepandemic). *Significantly different between adults with and without CKD (*P* < 0.05). CKD defined as an eGFR of 15 to 59 mL/min/1.73 m², Source: National Health and Nutrition Examination Survey, Abbreviations: CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate.

of \geq 6.5%. CKD was defined as a reduced eGFR of 15 to 59 mL/min/1.73 m² (stages 3 and 4) using a validated equation.⁴

Examination sample weights were used to adjust for oversampling, nonresponse, and noncoverage. Sex-specific prevalence of reduced eGFR estimates by race and Hispanic origin were presented because of significant interactions in a logistic regression model. Prevalence estimates were evaluated using National Center for Health Statistics presentation standards.⁵ Adjusted prevalence ratios were calculated from logistic regression models that included age category, sex, race and Hispanic origin, diabetes, hypertension, obesity, and interaction between sex and race and Hispanic origin. Differences in chronic disease prevalence between those with and without reduced eGFR were evaluated using t tests. A 2-sided P value of < 0.05determined statistical significance. SAS (version 9.4) and SUDAAN (version 11.0) were used for analyses. This analysis excluded 30 adults with stage 5 CKD and 662 adults with missing creatinine data, leaving 7,852 adults. Of these, 7,053 had complete data on health outcomes and were included in adjusted regression models.

In 2017-2020 (prepandemic), reduced eGFR prevalence among US adults was 5.7% (95% confidence interval [CI], 4.8%-6.7%) (Table 1). Prevalence increased with age and was higher among women compared with men. Reduced eGFR prevalence was also higher among non-Hispanic Black adults compared with non-Hispanic White adults but lower among non-Hispanic Asian and Hispanic adults. The unadjusted prevalence of reduced eGFR was higher among those with diabetes, hypertension, and obesity. However, after adjustment for demographic characteristics, diabetes, and hypertension, reduced eGFR prevalence was no longer significantly different between adults with and without obesity (adjusted prevalence ratio, 1.1; 95% CI, 0.9-1.3).

Figure 1 shows the prevalence of obesity, diabetes, and hypertension by reduced eGFR status. The prevalence of

obesity was 49.1% (95% CI, 43.5%-54.7%) in adults with reduced eGFR, higher than in adults without it (41.7%; 95% CI, 39.2%-44.2%). Compared to adults without reduced eGFR, adults with reduced eGFR also had a higher prevalence of diabetes (35.2%; 95% CI, 31.3%-39.3% vs 13.3%; 95% CI, 12.5%-14.1%) and hypertension (85.2%; 95% CI, 79.5%-89.9% vs 46.7%; 95% CI, 44.1%-49.3%).

In summary, US adults in 2017-2020 (prepandemic) with obesity, diabetes, or hypertension had a higher prevalence of reduced eGFR than adults without these conditions. However, after adjusting for demographic characteristics, diabetes, and hypertension, obesity was not independently associated with reduced eGFR. This suggests that at a population level, the association of obesity with reduced eGFR is primarily indirect through its association with diabetes and hypertension. This epidemiologic observation helps advance our understanding of the pathways through which reduced eGFR develops from obesity.

Nearly half of adults with reduced eGFR had obesity, making it a more common comorbid condition than diabetes but less common than hypertension. Among adults with reduced eGFR, obesity is a major risk factor for kidney failure, disability, death, and reduced access to kidney transplantation.^{1,2} The presence of reduced eGFR may also narrow treatment options for obesity.¹

This study has certain limitations, including those inherent to using body mass index as a maker for adiposity (no distinction between fluid retention and fat) and creatinine-based equations as a marker of eGFR in individuals with obesity.¹ Additionally, causality cannot be determined because of the cross-sectional design. Finally, if a small association between obesity and reduced eGFR exists independent of diabetes and hypertension, its detection may have been limited by the sample size.

Kidney Medicine

In conclusion, obesity is a highly prevalent condition in the US CKD population and one that is primarily associated with reduced eGFR via common intermediate disease states like diabetes and hypertension rather than solely through direct effects.

Allon N. Friedman, MD, Cynthia L. Ogden, PhD, and Craig M. Hales, MD

ARTICLE INFORMATION

Authors' Affiliations: Department of Medicine, Indiana University School of Medicine, Indianapolis, Indiana (ANF); and National Center for Health Statistics, Centers for Disease Control and Prevention, Hyattsville, Maryland (CLO, CMH).

Address for Correspondence: Allon Friedman, MD, 550 University Blvd, Suite 6100, Indianapolis, IN 46202. Email: Allfried@iu.edu

Authors' Contributions: Research idea: ANF; Study design: ANF, CLO, CMH; Data acquisition: CLO, CMH; Data interpretation: ANF, CLO, CMH; Statistical analysis: CMH. Each author contributed important intellectual content during manuscript drafting or revision and accepts accountability for the overall work by ensuring that questions pertaining to the accuracy or integrity of any portion of the work are appropriately investigated and resolved.

Support: None.

Financial Disclosure: Dr Friedman is on the scientific advisory board for GI Dynamics and Gila Therapeutics, is a consultant for Goldfinch Bio, and is on a Steering Committee for Astra Zeneca. He also owns Eli Lilly stock. The remaining authors declare that they have no relevant financial interests. Acknowledgements: The authors thank Mr Joseph Afful (Peraton) for his invaluable contributions to the analysis for this report.

Disclaimer: The findings and conclusions in this report are those of the authors and not necessarily the official position of the Centers for Disease Control and Prevention.

Peer Review: Received May 20, 2022. Evaluated by 1 external peer reviewer, with direct editorial input from the Statistical Editor and the Editor-in-Chief. Accepted in revised form October 9, 2022.

Publication Information: © 2022 The Authors. Published by Elsevier Inc. on behalf of the National Kidney Foundation, Inc. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/). Published online November 5, 2022 with doi 10.1016/j.xkme.2022.100568

REFERENCES

- Friedman AN, Kaplan LM, le Roux CW, Schauer PR. Management of obesity in adults with CKD. J Am Soc Nephrol. 2021;32(4):777-790. doi:10.1681/ASN.2020101472
- GBD 2015 Obesity Collaborators, Afshin A, Forouzanfar MH, et al. Health effects of overweight and obesity in 195 countries over 25 years. N Engl J Med. 2017;377(1):13-27.
- Akinbami LJ TC, Chen TC J, Davy O MK, et al. National Health and Nutrition Examination Survey, 2017-March 2020 prepandemic file: sample design, Estimation, and analytic guidelines. *Vital Health Stat 1*. 2022;(190):1-36.
- Inker LA, Eneanya ND, Coresh J, et al. New creatinine- and cystatin C-based equations to estimate GFR without race. *N Engl J Med.* 2021;385:1737-1749.
- Parker JD, Talih M, Malec DJ, et al. National Center for Health Statistics data presentation standards for proportions. *Vital Health Stat 2*. 2017;(175):1-22.