

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 pressure were conducted and blood samples taken. Obesity was defined as body mass index ≥ 30 kg/m². Hypertension was defined as systolic blood pressure ≥ 130 mm Hg or diastolic blood pressure ≥ 80 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 and Adjusted Prevalence of Chronic Kidney Disease Among Adults by Demographic and Health Characteristics, United States, 2017-2020 (Prepandemic)

| Characteristic | n ^a | Prevalence, % (95% CI) | Unadjusted Prevalence Ratio, (95% CI) | Adjusted Prevalence Ratio, (95% CI) |
|---|----------------|------------------------|---------------------------------------|-------------------------------------|
| 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 |
| $\geq 60+$ | 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 < 60 mL/min/1.73 m². Source: National Health and Nutrition Examination Survey.

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

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

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

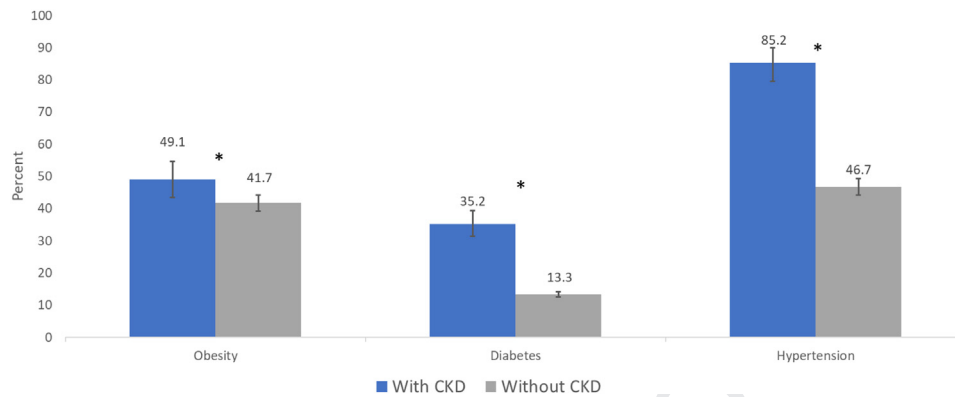


Figure 1. Unadjusted prevalence of obesity, diabetes, and hypertension among adults with and without CKD, United States, 2017-2020 (prepandemic). *Significantly different between adults with and without CKD ($P < 0.05$). CKD defined as an eGFR < 60 mL/min/1.73 m², Source: National Health and Nutrition 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. Reduced eGFR prevalence estimates were adjusted for sex, age, race and Hispanic origin, and having diabetes, hypertension, and obesity using predicted marginals from logistic regression models to evaluate independent effects. 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 < 0.05 determined 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, include 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.

225 Finally, if a small association between obesity and
 226 reduced eGFR exists independent of diabetes and hyper-
 227 tension, its detection may have been limited by the
 228 sample size.

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

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