HHS Public Access

Author manuscript

Ann Intern Med. Author manuscript; available in PMC 2017 February 14.

Published in final edited form as:

Ann Intern Med. 2016 November 15; 165(10): 743. doi:10.7326/L16-0407.

Metabolically Healthy Obesity and Development of Chronic Kidney Disease

Henry S. Kahn, MD and **Meda E. Pavkov, MD, PhD**

Centers for Disease Control and Prevention, Atlanta, Georgia

TO THE EDITOR

In their longitudinal study of metabolically healthy workers, Chang and colleagues (1) showed that higher BMI categories were associated with an increased incidence of CKD. They interpreted this finding as an adverse consequence of adiposity. However, their description of participant subgroups suggests that the association reported between elevated BMI and incident CKD was driven by relationships found primarily among older adults, men, and persons who exercise frequently. These are subgroups in which BMI status might reflect variations in the preservation or formation of muscle mass rather than the accumulation of adipose tissue. Greater muscle mass leads to increased production of creatinine, which in turn is associated with a reduced estimated glomerular filtration rate (GFR) (2) consistent with the final row of **Table 1** of the article. Reduction of the estimated GFR to less than 60 mL/min/1.73 m^2 was the quantitative threshold for identifying incident events of CKD.

Our curiosity leads to testable hypotheses. We suggest that Chang and colleagues use their Kangbuk Samsung Health Study to assess whether the decline in the estimated GFR might be associated more strongly with baseline lean mass rather than adipose tissue. As the authors recently reported (3), their healthy-worker cohort also provided measurements of bioelectric impedance and waist circumference. Thus, baseline values can be calculated for the percentage of fat and fat-free mass and the waist–height ratio.

Controversies exist about how to interpret BMI (4, 5). To clarify how it contributes to the decline in the estimated GFR, the authors could consider which tissue components or anatomical distributions of body mass best predict the described outcome.

References

1. Chang Y, Ryu S, Choi Y, Zhang Y, Cho J, Kwon MJ, et al. Metabolically healthy obesity and development of chronic kidney disease: a cohort study. Ann Intern Med. 2016; 164:305–12. [PMID: 26857595]. DOI: 10.7326/M15-1323 [PubMed: 26857595]
2. Levey AS, Becker C, Inker LA. Glomerular filtration rate and albuminuria for detection and staging of acute and chronic kidney disease in adults: a system-aticreview. JAMA. 2015; 313:837–46. [PMID:25710660]. DOI: 10.1001/jama.2015.0602 [PubMed: 25710660]

Disclosures: Authors have disclosed no conflicts of interest. Forms can be viewed at www.acponline.org/authors/icmje/ConflictOfInterestForms.do?msNum=L16-0407.

3. Zhao D, Kim MH, Pastor-Barriuso R, Chang Y, Ryu S, Zhang Y, et al. A longitudinal study of association between adiposity markers and intraocular pressure: the Kangbuk Samsung Health Study. PLoS One. 2016; 11:e0146057. [PMID: 26731527]. doi: 10.1371/journal.pone.0146057 [PubMed: 26731527]

4. Bastien M, Poirier P, Lemieux I, Després JP. Overview of epidemiology and contribution of obesity to cardiovascular disease. Prog Cardiovasc Dis. 2014; 56:369–81. [PMID: 24438728]. DOI: 10.1016/j.pcad.2013.10.016 [PubMed: 24438728]

5. Tomiyama AJ, Hunger JM, Nguyen-Cuu J, Wells C. Misclassification of cardiometabolic health when using body mass index categories in NHANES 2005-2012. Int J Obes (Lond). 2016; 40:883–6. [PMID: 26841729]. DOI: 10.1038/ijo.2016.17 [PubMed: 26841729]