Obesity Weight Loss Phenotypes in CKD: Measured GFR and Albumin-to-Creatinine Excretion Ratio Place for Stratification

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To the Editor: We have read with interest the recent published paper in *Kidney International Reports* entitled "Obesity Weight Loss Phenotypes in CKD: Findings From the Chronic Renal Insufficiency Cohort Study."[1] The authors of this interesting manuscript revealed that the pattern of weight loss (rapid vs. slow) and concurrent trends of nutritional, hemodynamic, and body composition indicators are important for understanding long-term mortality risk in persons with obesity and chronic kidney disease (CKD). Currently, obesity has been clearly identified as a cause of CKD with different phenotypes.[2] The manuscript by Harhay *et al.*[1] lacks in information regarding the urine albumin-to-creatinine excretion ratio, crucial for the diagnosis and prognosis of persons with CKD in terms of renal progression, independent marker of cardiovascular disease and mortality.[3] In addition, it has been previously found that in patients with obesity and CKD, estimated glomerular filtration rate is not a good method with a high variability that may reach to 30%, suggesting that in persons with obesity, measured glomerular filtration rate may avoid the estimation errors.[4] For all these mentioned reasons, we surmise that the results of the present study in *Kidney International Reports* should be at least adjusted by the baseline albumin-to-creatinine excretion ratio.

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Obesity Weight Loss Phenotypes in CKD: Findings from the Chronic Renal Insufficiency Cohort Study

The Author Replies: We thank Dr. Rico-Fontalvo and colleagues for their interest in our study. They suggest that we further adjust our statistical model for baseline albuminuria, which is an important predictor of kidney disease progression and cardiovascular outcomes. Due to missingness of baseline albuminuria data in our study population (33%), we present results after adjustment for baseline urine protein-to-creatinine ratio (UPCR), given that urinary protein excretion is an independent predictor of kidney function trajectory[1] and cardiovascular risk.[2] To examine the association between the estimated latent classes from the 6-class model in the primary analysis and the risk of death after further adjustment for baseline UPCR (mg/g), we excluded 150 Chronic Renal Insufficiency Cohort participants (5.3%) with missing baseline UPCR information, leaving 2681 participants that were eligible to be included in the analysis. We fit a Cox model with the 6 estimated latent classes from our primary analysis as predictors, adjusting for baseline age, sex, race/ethnicity, baseline diabetes status, baseline estimated glomerular filtration rate, baseline body mass index, baseline systolic blood pressure, baseline serum albumin level, initiation of