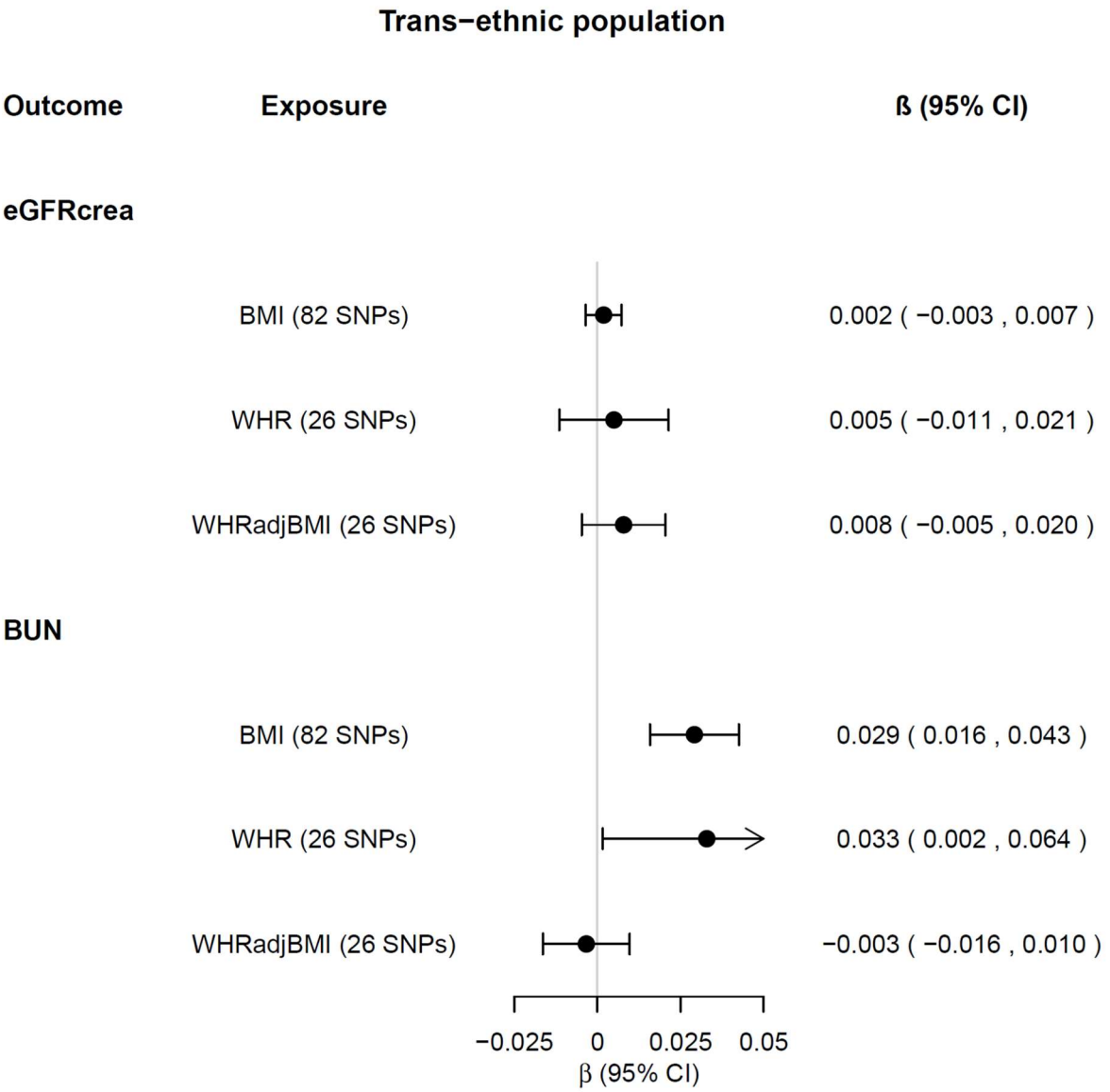


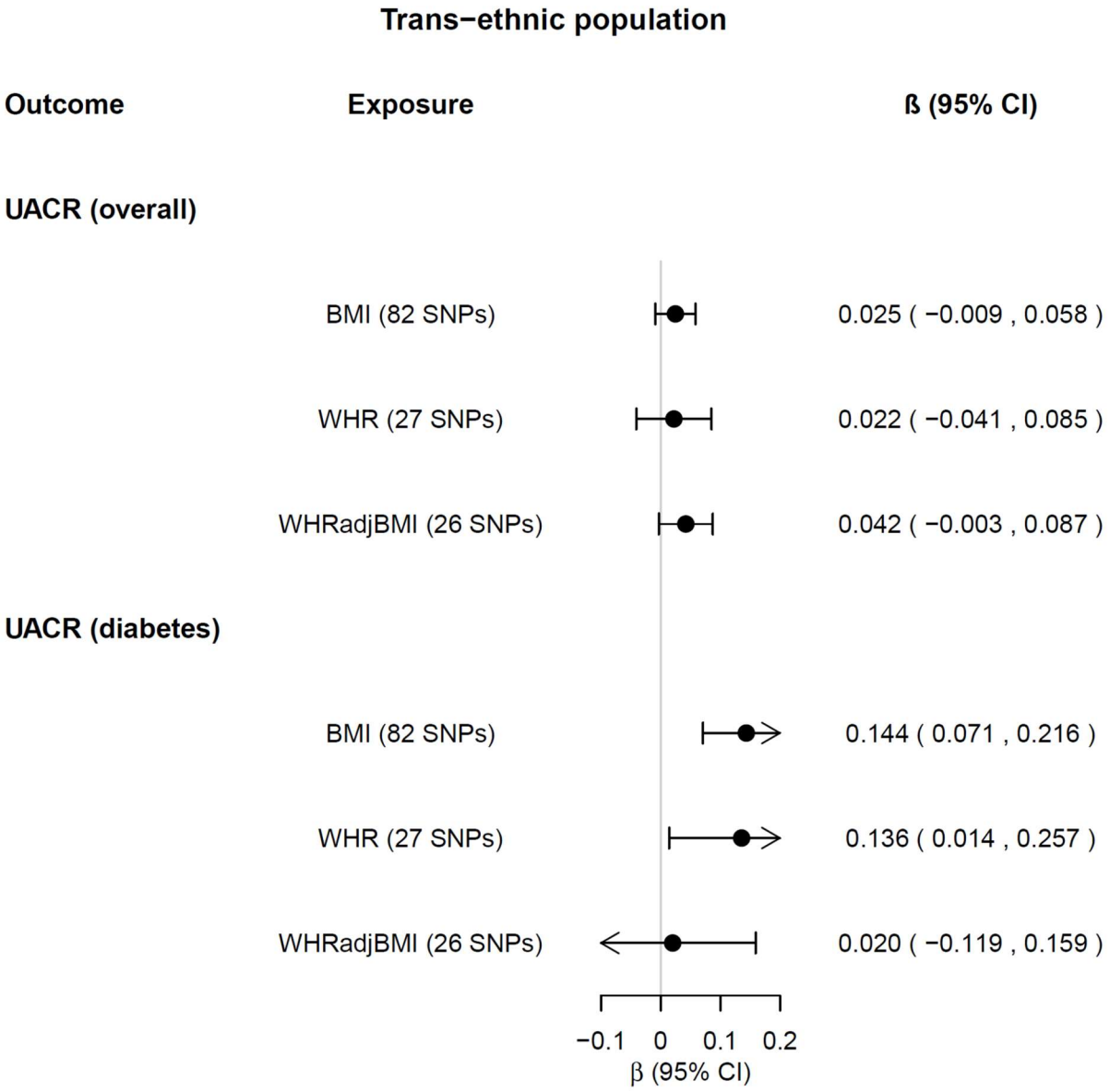
Supplemental Figure 1. Total causal effects of obesity on eGFR and BUN in the trans-ethnic population.

Estimates (β coefficients and 95% confidence intervals [CIs]) are from the inverse variance weighted random-effects Mendelian randomization analysis, and expressed in log units per standard deviation increase in the relevant exposure.

Obesity exposures from the GIANT Consortium were BMI (body mass index, N=339,224), WHR (waist-to-hip ratio, N=212,191), and WHRadjBMI (WHR adjusted for BMI, N=224,459). For each obesity exposure, the number of single nucleotide polymorphisms (SNPs) included in the analysis is shown in parenthesis.

Kidney function outcomes form the CKDGen Consortium were eGFRcrea (estimated glomerular filtration rate based on creatinine, calculated by the CKD-EPI equation, N=1,130,040) and BUN (blood urea nitrogen, N=852,680).

Sensitivity MR analyses are shown in Supplemental Tables 5-6.



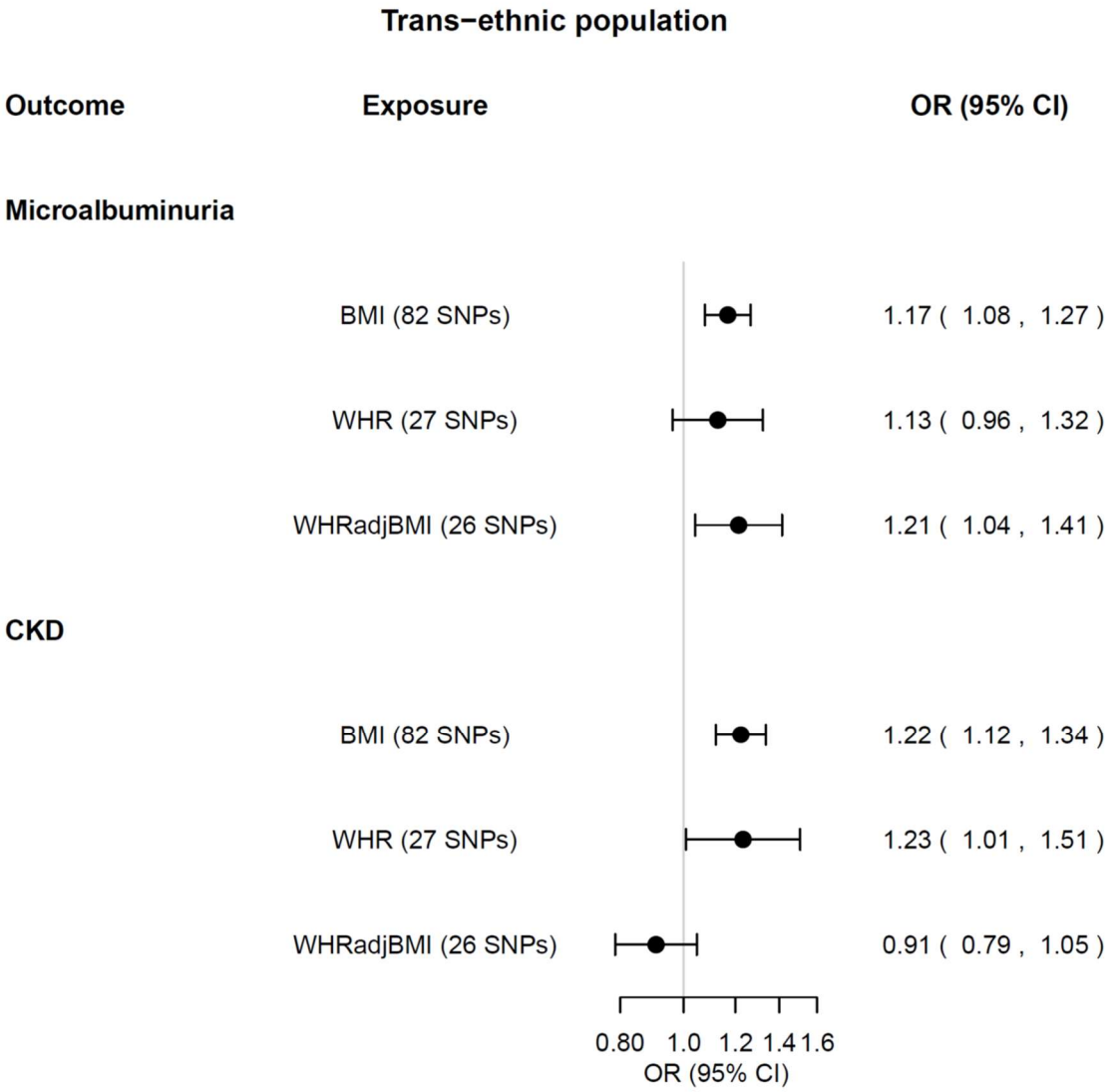
Supplemental Figure 2. Total causal effects of obesity on UACR in the trans-ethnic population.

Estimates (β coefficients and 95% confidence intervals [CIs]) are from the inverse variance weighted random-effects Mendelian randomization analysis, and expressed in log units per standard deviation increase in the relevant exposure.

Obesity exposures from the GIANT Consortium were BMI (body mass index, N=339,224), WHR (waist-to-hip ratio, N=212,191), and WHRadjBMI (WHR adjusted for BMI, N=224,459). For each obesity exposure, the number of single nucleotide polymorphisms (SNPs) included in the analysis is shown in parenthesis.

Kidney function outcome from the CKDGen Consortium was UACR (urinary albumin-to-creatinine ratio) in overall population and subpopulation with diabetes (UACR, $N_{overall}$=564,257, $N_{diabetes}$=51,541).

Sensitivity MR analyses are shown in Supplemental Tables 5-6.



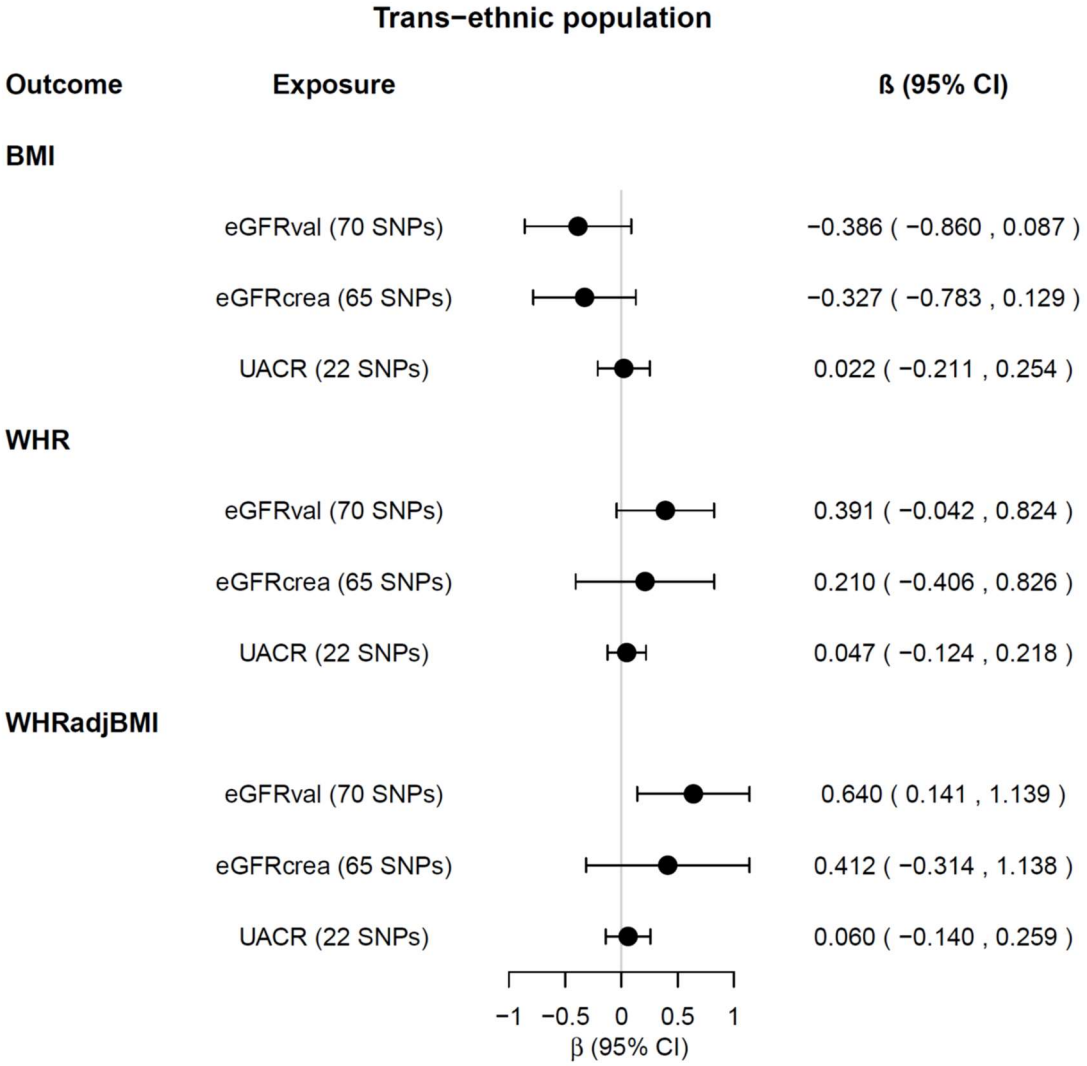
Supplemental Figure 3. Total causal effects of obesity on microalbuminuria and CKD in the trans-ethnic population.

Estimates are from the inverse variance weighted random-effects Mendelian randomization analysis, and expressed as odds ratios (ORs) and 95% confidence intervals (CIs).

Obesity exposures from the GIANT Consortium were BMI (body mass index, N=339,224), WHR (waist-to-hip ratio, N=212,191), and WHRadjBMI (WHR adjusted for BMI, N=224,459). For each obesity exposure, the number of single nucleotide polymorphisms (SNPs) included in the analysis is shown in parenthesis.

Kidney function outcomes from the CKDGen Consortium were microalbuminuria (N_{cases}=51,861, $N_{controls}$=297,093, defined as urinary albumin-to-creatinine ratio above 30 mg/g) and CKD (chronic kidney disease, N_{cases}=64,164, $N_{controls}$=561,055) defined as estimated glomerular filtration rate below 60 ml/min/1.73 m^2.

Sensitivity MR analyses are shown in Supplemental Tables 5-6.



Supplemental Figure 4. Total causal effects of kidney function on obesity in the trans-ethnic population.

Estimates (β coefficients and 95% confidence intervals [CIs]) are from the inverse variance weighted random-effects Mendelian randomization analysis, and expressed in log units per standard deviation increase in the relevant exposure.

Kidney function exposures from the CKDGen Consortium were eGFRval (estimated glomerular filtration rate based on creatinine and calculated by CKD-EPI equation, and validated by being associated with cystatin C and inversely associated with blood urea nitrogen [BUN], N=1,113,040), eGFRcrea (eGFR based on creatinine and calculated by CKD-EPI equation, and validated by being inversely associated with BUN, N= 765,348), and UACR (urinary albumin-to-creatinine ratio, N=564,257). For each kidney function exposure, the number of single nucleotide polymorphisms (SNPs) included in the analysis is shown in parenthesis.

Obesity outcomes from the GIANT Consortium were BMI (body mass index, N=339,224), WHR (waist-to-hip ratio, N=212,191), and WHRadjBMI (WHR adjusted for BMI, N=224,459).

Sensitivity MR analyses are shown in Supplemental Tables 9-10.