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BLACKS FACE A HIGHER RISK OF KIDNEY FAILURE THAN WHITES, REGARDLESS OF GENETICS

Racial disparities not explained by high-risk gene variants common in African Americans

Highlights

- Over nearly 25 years of follow-up, blacks had a higher risk of hypertension, diabetes, and kidney failure than whites, after adjustments.
- Most blacks with gene variants that have been linked to kidney disease experienced kidney function decline similar to blacks without the variants.

Washington, **DC** (March 10, 2016) — New research investigates the ties between certain genetic variants and kidney disease in African Americans. The findings, which appear in an upcoming issue of the *Journal of the American Society of Nephrology* (JASN), suggest that widespread screening for these variants in the black general population is not yet justified.

African Americans have an elevated risk for chronic kidney disease and kidney failure compared with European Americans. Studies have shown that much of this risk is due to genetic variations in a gene called apolipoprotein L1 (*APOL1*), which creates a protein that is a component of HDL, or good cholesterol. These variants arose tens of thousands of years ago in sub-Saharan Africa, and so are present in individuals who have recent sub-Saharan African ancestry. Approximately 5 million African Americans carry *APOL1* risk variants; however, not all persons with such variants develop kidney disease.

To getter a better sense of how *APOL1* genetic risk variants affect kidney disease and other aspects of health over the long term, Morgan Grams, MD PhD (Johns Hopkins University) and her colleagues evaluated the prognosis and *APOL1* status of participants in the Atherosclerosis Risk in Communities (ARIC) study. Among 15,140 ARIC participants followed from 1987–1989 to 2011–2013, 75.3% were white, 21.5% were black/*APOL1* low-risk, and 3.2% were black/*APOL1* high-risk. "Our study is a population-based cohort following participants over 25 years and thus well suited at assessing a fairly comprehensive set of outcomes among people with the high-risk genotype," said Dr. Grams.

In analyses that adjusted for differences in demographics, blacks had a higher risk for all assessed adverse health events: acute kidney injury, kidney failure, hypertension, diabetes, cardiovascular disease, hospitalization, and death; however, in analyses that also adjusted for comorbid conditions and socioeconomic status, blacks had a higher risk for hypertension, diabetes, and kidney failure only. When considering only blacks, the *APOL1* high-risk variants were linked with a higher risk of kidney failure, but there was high variability in kidney function decline among those with and without the variants.

"We found great variability in kidney function trajectory, such that most African Americans with the high-risk genotype experienced similar decline as African Americans with the low-risk genotype," said Dr. Grams. "We did find pervasive racial disparities in adverse health outcomes not explained by the *APOL1* risk variants, which suggests that interventions to improve health and health outcomes in African Americans are needed."

Study co-authors include Casey Rebholz, PhD; Yuan Chen, MS; Andreea Rawlings, MS; Michelle Estrella, MD MHS; Elizabeth Selvin, PhD; Lawrence Appel, MD; Adrienne Tin, PhD; and Josef Coresh, MD PhD.

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The article, entitled "Race, APOL1 Risk, and eGFR Decline in the General Population," will appear online at http://jasn.asnjournals.org/ on March 10, 2016, doi: 10.1681/ASN.2015070763.

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