## Black Women Have a Worse Cardio-Metabolic Risk Profile Compared to White Women with Polycystic Ovary Syndrome in the United States: A Systematic Review and Meta-Analysis

Maryam Kazemi. Cornell University

Maryam Kazemi, PhD, MSc, RD<sup>1</sup>, Joy Y. Kim<sup>1</sup>, Stephen A. Parry, MS<sup>2</sup>, Ricardo Azziz, MD, MPH, MBA<sup>3</sup>, Marla E. Lujan, PhD, MSc<sup>1</sup>.

<sup>1</sup>Division of Nutritional Sciences, Human Metabolic Research Unit, Cornell University, Ithaca, NY, USA, <sup>2</sup>Cornell Statistical Consulting Unit, Cornell University, Ithaca, NY, USA, <sup>3</sup>Department of Obstetrics and Gynecology, The University of Alabama at Birmingham, Birmingham, AL, USA.

**Abstract:** Health disparities may influence cardio-metabolic risk in women with polycystic ovary syndrome (PCOS). The magnitude and direction of differences in cardio-metabolic risk between Black and White women with PCOS remain uncertain due to inconsistent reports.

We conducted a systematic review and meta-analysis to evaluate evidence on cardio-metabolic health disparities between Black and White women with PCOS in the US in response to the call for further delineation of these disparities by the International Evidence-based Guideline for the Assessment and Management of PCOS. Databases of MEDLINE, Web of Science, and Scopus were searched (January 1990 to September 2020) to identify observational studies documenting cardio-metabolic risk profile (glucoregulatory, lipid profile, anthropometric, blood pressure status) in Black and White women with PCOS. The primary outcome was fasting glucose. Further, cardiovascular events (stroke, coronary heart disease, heart failure) and mortality rate (cardiovascular death, total mortality) data were evaluated between groups. Studies on children (< 17 yrs.), pregnant or menopausal-aged (> 50 yrs.) women were excluded. Data were pooled by random-effects models and expressed as weighted mean differences and 95% confidence intervals. Eleven studies (n = 2,821; [626 Black; 2,195 White women]) evaluated cardiometabolic risk profile, yet none reported on cardiovascular events/mortality rate. Black women had comparable fasting glucose (-0.61 [-1.69, 2.92] mg/dL; I2 = 62.5%), yet exhibited increased fasting insulin (6.76 [4.97, 8.56] μIU/mL; I2 = 59.0%); homeostatic model assessment of insulin resistance (HOMA-IR; 1.47 [0.86, 2.08]; I2 = 83.2%); systolic blood pressure (SBP, 3.32 [0.34, 6.30] mmHg; I2 = 52.0%) and decreased triglyceride (-32.56 [-54.69 , -10.42] mg/dL; I2 = 68.0%) when compared to White women with PCOS (All: P ≤ 0.03). Groups were comparable in total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and diastolic blood pressure (All:  $P \ge 0.06$ ). Paucity in the number of studies that evaluated cardiovascular events or mortality limits any conclusions about potential disparities.

Overall, Black women with PCOS have a greater tendency for an adverse cardio-metabolic risk profile (increased insulin, HOMA-IR, SBP), despite lower triglyceride levels than White women. Our observations support consideration of these disparities for diagnostic, monitoring, management, and public health practices, and for future guideline recommendations. Heterogeneity among studies warrants future research to address the relative contributions of biological, environmental, socioeconomic, and healthcare factors to the observed disparities. Longitudinal research should address cardiovascular events and mortality rate in Black women with PCOS (www.crd.york.ac.uk/PROSPERO ID, CRD42020183485).