Placental malaria exposure does not affect physical or cardiometabolic profile and aerobic fitness level in young Tanzanian Lajeunesse-Trempe F, Ramaiya K, Faurholt-Jepsen D, Nielsen J, Koch LS, Vaag A, Bygbjerg I, Mutabingwa T, Christensen DI University of Copenhagen, Copenhagen, Denmark

Objective
This study aimed at measuring if exposure to placental malaria during the foetal state affected clinical outcomes, cardio-metabolic profile or aerobic fitness of young men and women from Tanzania.

Methods
From a Tanzanian Birth Cohort, we traced 45 men and women aged 19.6 (± 1.1) years and used their birth files to determine the status of their placental malaria exposure (based on placental blood smear: PM+ / PM-). Eleven of the subjects had PM+. Anthropometric measures, body fat composition (visceral and subcutaneous fat thickness – VAT and SAT, respectively), blood pressure (BP), standard oral glucose tolerance test (OGTT) – 75 g with measurements of glucose (glucose tolerance), fasting insulin and c-peptide were performed. From blood sample, triglycerides, total cholesterol, LDL and HDL level were measured, as well as c-reactive protein (CRP). Standard HIV rapid test (capillus test), Hb-surface antigen test, and malaria peripheral smear microscopy were assessed. Aerobic fitness level (Wattmax test on stationary bicycle) were measured. Between-group differences of placental malaria exposure were assessed by ANOVA analyses for outcome variables.

Results
Mean BMI (kg/m2) was 20.1 (±0.3) for PM+ and 19.9 (±0.4) for PM-, with a difference of p=0.9. Waist circumference (cm) was 69.6 (±1) for PM+ and 72.8 (±0.9) for PM-, with a between-group difference of p=0.2. VAT (cm) was 5.2 (±0.3) for PM+ and 5.5 (±0.2) for PM-. SAT (cm) was 0.9 (±0.1) for PM+ and 1.6 (±0.2) for PM-, with differences of p=0.3 for both associations. Mean fasting plasma glucose (mmol/L) was 4.7 (±0.2) for PM+ and 4.5 (±0.1) for PM-; 2-h plasma glucose (mmol/L) was 6.2 (±0.3) for PM+ and 6.5 (±0.3) for PM-, with between PM group differences of p=0.2 and p=0.5, respectively. Mean fasting plasma insulin (pmol/L) was 50.3 (±5.6) for PM+ and 57.4 (±6.5) for PM-, with between difference of p=0.6. Plasma c-peptide (pmol/L) was 453.1 (±60.4) for PM+ and 446.5 (±27.7) for PM-, with a difference of p=0.9. For lipid profile, LDL level (mmol/L) was 1.6 (±0.5) for PM+, and 1.9 (±0.2) for PM-; HDL level (mmol/L) was 0.8 (±0.3) for PM+ and 1.0 (±1.1) for PM-, with a between difference of p=0.1 and p=0.2. Total cholesterol level (mmol/L) for PM+ was 3.0 (±0.5) and 3.6 (±0.2) for PM-, triglycerides level (mmol/L) was 0.8 (±0.2) for PM+ and 0.8 (±0.1) for PM-, with a group difference of p=0.05 and p=0.8 for both associations. C-reactive protein (pmol/L) was 4.5 (±0.01) for PM+ and 1.3 (±0.3) for PM- with a difference of p=0.01. Systolic BP (mmHg) was 124 (±5) for PM+, 126 (±3) for PM- and diastolic BP (mmHg) was 77 (±3) for PM+ and 75 (±1) for PM- with between differences of p=0.7 and p=0.5, respectively. Mean fitness (ml · kg-1 · min-1) was 26.0 (±3) for PM+ and 27.2 (±2) for PM-, with a between-group difference of p=0.7.

Conclusion
Placental Malaria exposure during the foetal state did not influence blood glucose, insulin and C-peptide, lipid profile, blood pressure, anthropometric measurements, body fat composition and aerobic fitness level in young Tanzanian men and women. The results do not exclude the possibility that the exposure may influence physiological and epigenetic mechanisms underlying metabolic diseases; health consequences might be noticeable and significant later in life. Long term follow up studies in larger cohorts of offspring exposed to Placental Malaria are needed.