Objective: To prospectively assess whether the infant psychosocial environment was associated with cardiometabolic risk as early as adolescence. Study design: Participants were recruited in Santiago, Chile, and have been followed from infancy. Inclusion criteria included healthy infants with birth weight \( \geq 3 \) kg and a stable caregiver. The psychosocial environment, including depressive symptoms, stressful life events, poor support for child development, father absence, and socioeconomic status, was reported by mothers at 6-12 months. Body mass index (BMI) z score was assessed at 5 and 10 years. BMI z score, waist-to-hip ratio, systolic and diastolic blood pressure, fat mass and body fat percentage, fasting glucose, total and high-density lipoprotein cholesterol, and homeostatic model of insulin resistance were tested in adolescence. Results: Adolescents ranged from 16 to 18 years of age (n = 588; 48.1\% female). A poorer infant psychosocial environment was associated with BMI z score at 10 years (\( \beta = 0.10, 95\% \text{ CI} = 0.00-0.19 \)) and in adolescence (\( \beta = 0.15, 95\% \text{ CI} = 0.06-0.24 \)) but not at 5 years. A poorer infant psychosocial environment was associated with higher blood pressure (\( \beta = 0.15, 95\% \text{ CI} = 0.05-0.24 \)), greater anthropometric risk (\( \beta = 0.13, 95\% \text{ CI} = 0.03-0.22 \)), greater biomarker (triglycerides, homeostatic model assessment of insulin resistance, total cholesterol) risk (\( \beta = 0.12, 95\% \text{ CI} = 0.02-0.22 \)), and a higher likelihood of metabolic syndrome in adolescence (aOR = 1.50; 95\% CI = 1.06-2.12). Conclusions: These findings demonstrate that a poorer infant psychosocial environment was associated with greater adolescent cardiometabolic risk. The results support
screening for infants? psychosocial environments and further research into causality, mechanisms, prevention, and intervention.