

0141 - EVIDENCE OF A LASTING PROTECTIVE LEGACY AFTER REVERSAL OF CHILDHOOD OBESITY DURING PUBERTY: A LONGITUDINAL ANALYSIS OF SUBCLINICAL CARDIOVASCULAR RISK

Mode: Research paper

Thematic Unit: Obesity

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Background/Objectives: The cardiometabolic (CDM) continuum begins early in life, yet the windows of biological reversibility remain understudied in Hispanic pediatric populations. It is important to understand whether remission of obesity around puberty can stop structural arterial damage, which is key for pediatric prevention strategies. This study investigates how childhood BMI trajectories influence cholesterol remnants, atherogenic ratios, the Remnant Cholesterol Inflammatory Index (RCII), and subclinical vascular damage from adolescence to adulthood at ages 16, 22, and 29.

Methods: Longitudinal design in a Chilean birth cohort (n=565; 50% females). BMI trajectories from birth to adolescence were modeled using cubic splines into 5 groups: always healthy (G1), adolescent-onset obesity (G2), childhood obesity reversed in puberty (G3), persistent childhood obesity (G4), and transient obesity (G5). Remnant cholesterol, remnant/HDL ratio, and the RCII (combining remnants and hsCRP) were assessed at 16y, 22y, and 29y. Pulse wave velocity (PWV) and left/right carotid intima-media thickness (cIMT) were measured at 29y. Data were analyzed using ANOVA with Tukey-Kramer post-hoc test.

Results: At 16y and 22y, persistent obesity (G4) and adolescent-onset obesity (G2) displayed significantly higher remnant cholesterol, remnant/HDL ratios, and RCII compared to G1 (Tukey, P.05). At 29y, structural vascular assessment revealed that G4 and G2 had significantly higher arterial stiffness (PWV) and cIMT than G1 (P

Conclusion: Reversing childhood obesity around puberty exerts a permanent protective "metabolic and vascular legacy" into adulthood, completely preventing the accumulation of remnant lipotoxicity, chronic inflammation, and structural arterial remodeling. Puberty represents a critical therapeutic window of opportunity to mitigate long-term cardiovascular disease. Funding: FONDECYT 1210283 - ACT210006 - NIH HL088530.

