

0139 - ADVERSE CHILDHOOD EXPERIENCES AMPLIFY IMMUNO-METABOLIC RISK DEPENDING ON LONGITUDINAL BMI TRAJECTORIES: A PRINCIPAL COMPONENT AND INTERACTION ANALYSIS

Mode: Research paper

Thematic Unit: Obesity

Thematic Unit 2: Insulin Resistance

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Background/Objectives: Background: Adverse childhood experiences (ACEs) induce chronic allostatic load, triggering metabolic dysregulation. Yet whether early-life stress acts as a synergistic amplifier of specific pediatric weight-gain trajectories into adulthood remains uncharacterized. We assessed the independent and interactive effects of ACEs and longitudinal BMI trajectories on immuno-metabolic and structural vascular phenotypes at 29y.

Methods: Methods: Longitudinal analysis in a Chilean birth cohort (n=565; 50% females). BMI trajectories from birth to adolescence were modeled (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). At 29y, principal component analysis (PCA) with Varimax rotation was applied to multi-systemic block: HOMA-IR, HOMA- β , MetS score (Gurka), Remnant Cholesterol Inflammatory Index (RCII), pulse wave velocity (PWV), and carotid intima-media

thickness (cIMT). This extracted 2 dimensions: Immuno-Metabolic (f1) and Structural Vascular (f2) factors. Linear regressions with interaction terms (trajectory#high_ace ≥ 4) were fitted.

Results: PCA consolidated 2 dimensions capturing 61.8% total variance: f1 (dominated by insulin resistance, pancreatic function, MetS severity and chronic inflammation) and f2 (dominated by cIMT remodeling). Interaction models revealed a massive synergistic effect for f1 within the transient obesity group (G5): while weight instability alone did not increase adulthood risk, the combination of G5 and high ACEs severely compounded the immuno-metabolic burden (G5#High_ACE Coef: 3.03, $P=.003$). Crucially, a trend emerged in G2, where high ACEs also tended to exacerbate immuno-metabolic damage (G2#High_ACE Coef: 1.01, $P=.052$). Conversely, for the vascular dimension (f2), an inverse trend appeared in G4, where high ACEs decelerated additional physical thickening (G4#High_ACE Coef: -0.79, $P=.055$). Reverted childhood obesity (G3) remained fully protected against stress-induced deterioration ($P>.05$).

Conclusion: Conclusion: ACEs is a critical second hit that dysregulates immuno-metabolic homeostasis, selectively magnifying risk in G5 and showing a deleterious trend in G2. Pubertal obesity remission successfully buffers against early life trauma. The inverse vascular trend in G4 suggests a blunted allostatic reactivity due to cumulative socio-



metabolic weathering, a pathophysiological phenomenon discussed herein.
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