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ANALYSIS OF KEY RISK FACTORS ASSOCIATED WITH THE DEVELOPMENT OF INSULIN RESISTANCE IN CHILDREN

ABDUMANNOBOVA RUSHANA OLIMJON QIZI
Scientific Supervisors:
Professor, Doctor of Sciences (DSc) **IRISKULOV B.U**
Doctor of Sciences (DSc) **AXMEDOVA D.B**
Tashkent state medical university, Tashkent, Uzbekistan

Annotation

The escalating global prevalence of pediatric obesity has precipitated a concurrent and alarming rise in metabolic disorders, most notably insulin resistance and metabolic syndrome. This comprehensive research article investigates the key risk factors driving the pathogenesis of insulin resistance among school-aged children, with a specific focus on the comparative impacts of urban and rural living environments. Drawing upon a cross-sectional clinical and laboratory analysis of 131 school children aged 10 to 11 years residing in Tashkent and the surrounding Tashkent region, this study elucidates the anthropometric, glycemic, lipemic, and endocrine markers indicative of early metabolic dysfunction. The analysis reveals a profound paradox in the regional epidemiological transition: while both urban and rural cohorts exhibit significant deviations in body mass index indicative of widespread overweight and obesity, rural children demonstrate a notably more severe degree of metabolic deterioration. Specifically, rural cohorts exhibited prolonged delayed glucose clearance during oral glucose tolerance testing, exacerbated dyslipidemia characterized by elevated low-density lipoprotein and severely depressed high-density lipoprotein, and catastrophic hyperleptinemia compared to their urban counterparts. These findings fundamentally challenge the traditional paradigm that rural environments inherently foster healthier metabolic profiles due to natural diets and physical labor. Instead, the data suggest that the globalization of ultra-processed diets, coupled with transitioning physical activity patterns and potential exposure to agricultural endocrine disruptors, has created a highly obesogenic and diabetogenic environment in rural settings. The findings underscore the urgent need for geographically tailored, socio-ecologically adapted public health interventions, highlighting that pediatric metabolic screening must become a universal standard regardless of population density.

Key words: pediatric obesity, insulin resistance, metabolic syndrome, hyperleptinemia, dyslipidemia, urban-rural disparity, endocrinology, HOMA-IR, carbohydrate metabolism, public health.

Introduction

The global epidemiological landscape of pediatric health has undergone a profound transformation over the last four decades, characterized by an exponential increase in the prevalence of overweight and obesity. What was once considered a physiological anomaly isolated to affluent adult populations has now permeated the pediatric demographic worldwide, establishing itself as one of the most pressing public health crises of the twenty-first century. According to data aggregated by the World Health Organization, 1.9 billion people worldwide are currently overweight or obese. Regionally, the prevalence of obesity reaches 60% of the population in the United States and Canada, 44% in Europe, approximately 50% in Russia, 27% in Asia, and 19% in Africa.

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The proportion of children and adolescents aged 5 to 19 presenting with overweight or obesity surged from a mere 4% in 1975 to over 18% in recent years, a trend distributed symmetrically across both sexes. In absolute terms, this represents hundreds of millions of children facing imminent metabolic compromise, with projections indicating that by 2025, an additional 167 million individuals globally will suffer from obesity. This morphological shift toward excessive adiposity is intricately linked to the early onset of a cluster of metabolic, hormonal, and clinical disturbances collectively termed metabolic syndrome, which serves as a potent precursor to cardiovascular disease, non-alcoholic fatty liver disease, and type 2 diabetes mellitus. Recent data indicate that metabolic syndrome is diagnosed in 27.2% of children and adolescents with obesity, with its prevalence reaching 20% in the 7-11 age group and a staggering 37.6% in the 12-18 age group.

At the pathophysiological core of metabolic syndrome lies insulin resistance, a complex multifactorial condition wherein normal physiological concentrations of insulin produce a subnormal biological response in target tissues, predominantly skeletal muscle, hepatic tissue, and adipose tissue. Under optimal homeostatic conditions, insulin acts as the master anabolic hormone. Upon binding to the extracellular alpha-subunits of the insulin receptor, it induces a conformational change that activates the intracellular beta-subunits, which possess intrinsic tyrosine kinase activity. This activation leads to the autophosphorylation of the receptor and the subsequent recruitment and phosphorylation of insulin receptor substrates, primarily IRS-1 and IRS-2. These substrates act as docking stations for downstream effector molecules, notably activating the phosphatidylinositol 3-kinase (PI3K) and Protein Kinase B (Akt) signaling pathways. In skeletal muscle and adipose tissue, this cascade culminates in the translocation of GLUT4 glucose transporters from intracellular vesicles to the plasma membrane, facilitating the rapid cellular uptake of circulating glucose. Simultaneously, in the liver, insulin signaling suppresses gluconeogenesis and glycogenolysis, while promoting glycogen synthesis and lipogenesis.

However, the accumulation of excess visceral adipose tissue drastically disrupts this elegant signaling architecture. Visceral fat is not merely an inert reservoir for triglyceride storage; it is a highly active, complex endocrine organ that secretes a multitude of bioactive peptides collectively known as adipokines, as well as classical inflammatory cytokines. As adipocytes undergo pathological hypertrophy in the face of sustained caloric surplus, they outstrip their vascular supply, leading to localized tissue hypoxia and cellular necrosis. This cellular stress recruits macrophages to the adipose depot, triggering a chronic, low-grade, systemic inflammatory response. Elevated circulating levels of pro-inflammatory cytokines, such as tumor necrosis factor-alpha and interleukin-6, alongside elevated free fatty acids, activate intracellular serine/threonine kinases including c-Jun N-terminal kinase and IkappaB kinase. These kinases erroneously phosphorylate IRS-1 on serine residues rather than tyrosine residues, effectively imposing a molecular blockade that prevents IRS-1 from associating with the insulin receptor. Consequently, the downstream PI3K/Akt pathway is blunted, GLUT4 translocation fails, and peripheral tissues become "deaf" to the insulin signal.

To maintain euglycemia in the face of this peripheral resistance, the pancreatic beta-cells undergo structural hypertrophy and functional hypersecretion, resulting in a state of chronic compensatory hyperinsulinemia. While this hyperinsulinemia initially succeeds in keeping blood glucose within normal parameters, it exerts widespread deleterious effects. High circulating insulin acts as a potent growth factor, driving further lipogenesis, exacerbating adiposity, promoting vascular smooth muscle proliferation, and stimulating sympathetic nervous system activity, thereby contributing to the hypertension and dyslipidemia

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characteristic of metabolic syndrome. Over time, the relentless demand placed on the pancreatic beta-cells leads to endoplasmic reticulum stress, amyloid deposition, and eventual beta-cell apoptosis, marking the irreversible transition from insulin resistance to overt type 2 diabetes mellitus.

Historically, insulin resistance and its attendant complications were diagnosed almost exclusively in middle-aged and elderly populations. Today, however, the temporal onset of these pathologies has shifted dramatically backward, with severe insulin resistance increasingly detected in prepubertal and pubertal children. In the context of Central Asia, and Uzbekistan specifically, rapid socioeconomic modernization, globalization of food supply chains, and sweeping lifestyle transitions have created a highly obesogenic environment. The etiology of pediatric insulin resistance is intrinsically tied to the environmental and socio-ecological microclimates in which children are raised, specifically the urban-rural divide. Traditionally, urbanization has been identified as the primary vector for metabolic disease, characterized by the displacement of physical labor, the proliferation of motorized transport, the density of digital entertainment promoting sedentary behavior, and continuous exposure to hypercaloric, ultra-processed, and heavily marketed fast foods.

Given the severe long-term consequences of childhood insulin resistance, identifying and understanding the precise environmental risk factors is of paramount importance. Therefore, the primary objective of this study is to conduct an exhaustive analysis of the key clinical, anthropometric, and biochemical risk factors associated with the development of insulin resistance in school-aged children, providing a robust evidence base for the formulation of targeted, effective, and socio-culturally adapted prophylactic strategies.

Material and Methods

To achieve a granular and comprehensive understanding of the metabolic health status among the target pediatric demographic, a cross-sectional clinical and laboratory investigation was meticulously designed and executed. The study population consisted of school-aged children, specifically targeting the prepubertal to early pubertal transition period. The research was conducted utilizing the educational and medical infrastructures of two distinct geographic locales to facilitate a robust comparative analysis of environmental impacts on metabolic health.

The total evaluated cohort comprised 131 children aged 10 to 11 years, corresponding to the fourth grade of the regional educational system. To assess the differential impacts of urbanization and agrarian lifestyles, the cohort was strategically divided into two primary subgroups. The urban cohort consisted of 63 students enrolled at Secondary School No. 302, situated within the highly developed, densely populated metropolitan matrix of Tashkent city. Conversely, the rural cohort comprised 68 students recruited from Secondary School No. 9, located in a less densely populated, traditionally agrarian sector of the broader Tashkent region.

Based on the initial clinical and laboratory screening, including the calculation of the HOMA-IR index, specific laboratory subgroups were formed for detailed physiological analysis. This included a main investigative group demonstrating clear clinical and biochemical markers of insulin resistance (23 children) and a normo-metabolic control group without signs of insulin resistance (20 children).

The study adhered to strict ethical and methodological protocols. Anthropometric measurements (height, weight, and BMI) were obtained utilizing highly standardized, calibrated equipment. Fasting venous blood glucose concentrations, Oral Glucose Tolerance Tests (OGTT), fasting serum insulin levels, lipid profiles (Total Cholesterol, LDL, HDL, and Triglycerides), and serum leptin concentrations were evaluated using automated biochemical

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analyzers and Enzyme-Linked Immunosorbent Assays (ELISA) according to manufacturer protocols.

Result and Discussion

The comprehensive clinical, anthropometric, and biochemical data generated from this investigation provide profound insights into the rapidly deteriorating metabolic health of the pediatric population.

Demographic Homogeneity and the Anthropometric Shift

The foundational demographic analysis of the cohort established a mean age of 10.6 ± 0.08 years, representing a highly homogenous sample critical for ensuring the validity of physiological comparisons. The analysis of anthropometric indicators—specifically body mass, height, and the resultant Body Mass Index (BMI)—revealed catastrophic deviations from established normative standards across both geographic settings.

Table 1

Anthropometric Characteristics and Growth Deviations in Pediatric Cohorts

Parameter	Overall Cohort	Urban Cohort	Rural Cohort
Mean Age (years)	10.6 ± 0.08	+2.56% above mean	+1.42% above mean
Mean Body Mass (kg)	34.37 ± 1.2	1.49x above norm	1.40x above norm
Mean Height (m)	1.439 ± 0.01	1.04x above norm (+4%)	1.00x (Baseline)
Mean BMI (kg/m²)	16.57 ± 0.5	23.39	23.36

As detailed in the anthropometric analysis table, children residing in the metropolitan center of Tashkent exhibited a body mass 1.49 times greater than standard normative projections, while rural children demonstrated a nearly identical degree of physical hypertrophy, presenting a mean body mass 1.40 times higher than normative expectations. The calculated BMI averages for this study were drastically elevated: 23.39 kg/m² for the urban cohort and 23.36 kg/m² for the rural cohort, demonstrating a negligible 1% variance between the two environments. This confirms that the environmental drivers of excess adiposity have achieved absolute geographic saturation.

To facilitate further data analysis and visualization of these anthropometric deviations in Excel, the following Python script utilizing pandas and matplotlib can be executed to generate Chart 1:

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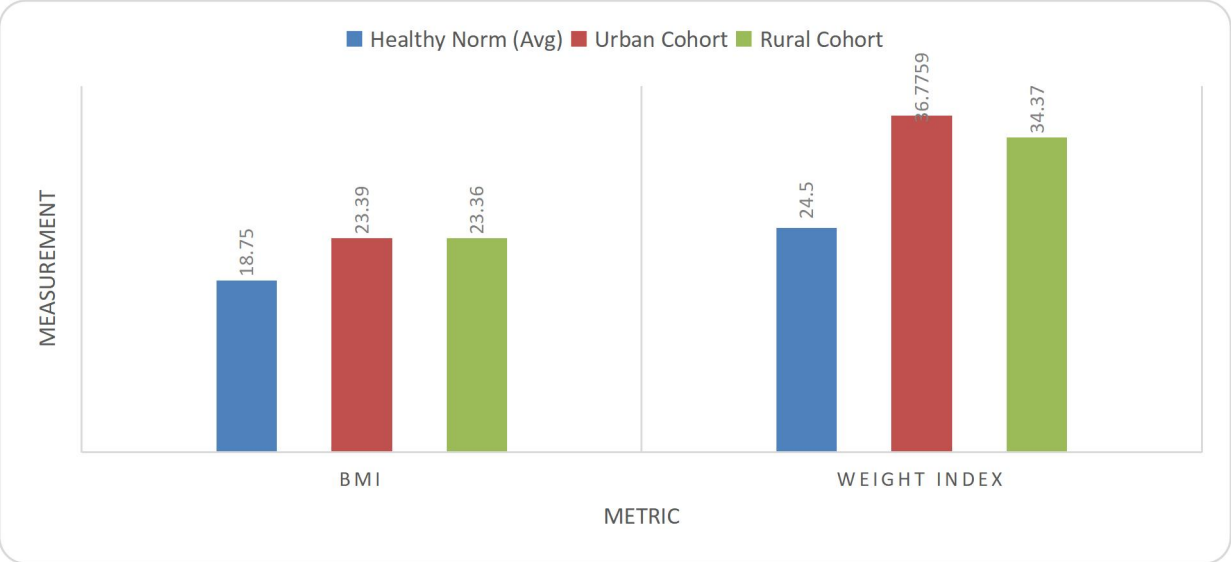

Chart 1: Anthropometric Shift in Pediatric Cohorts

Glycemic Dysregulation and Beta-Cell Exhaustion

The evaluation of blood glucose dynamics across the basal state and during the oral glucose tolerance test exposed severe, geographically distinct patterns of glycemic failure, alongside marked hyperinsulinemia.

Table 2

Glycemic Profile and Oral Glucose Tolerance Dynamics Across Cohorts

Glycemic Parameter	Control Baseline	Urban Cohort	Rural Cohort
Fasting Glucose (mmol/L)	4.8	6.2	6.1
2-Hour Post-OGTT (mmol/L)	< 7.8	10.8 - 11.0	11.4 - 11.8
3-Hour Post-OGTT (mmol/L)	Return to basal	13.5 - 14.0	14.5 - 15.0
Fasting Insulin (µU/mL)	7.0 ± 0.33	11.83	11.83

Both the urban and rural cohorts demonstrated marked basal hyperglycemia, recording mean fasting glucose levels of 6.2 mmol/L and 6.1 mmol/L, respectively, compared to a baseline of 4.8 mmol/L. Following a concentrated glucose load, the urban children sustained dangerous glycemic elevations between 10.8 and 11.0 mmol/L at two hours post-ingestion. The rural cohort exhibited an even more compromised clearance capacity, registering levels between 11.4 and 11.8 mmol/L. At the three-hour mark, both cohorts demonstrated an uncontrolled, continuous escalation of blood glucose. The urban children reached profoundly pathological levels of 13.5 to 14.0 mmol/L. Yet, the rural cohort suffered an exponential and near-total loss of glycemic control, skyrocketing to 14.5 to 15.0 mmol/L.

Furthermore, the laboratory analysis revealed a state of severe, systemic hyperinsulinemia across the entire investigated population. Both the urban and rural cohorts recorded an identical

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mean fasting insulin level of 11.83 µU/mL, representing a pathological 1.69-fold increase over normal values.

The following Python code generates the data tables and line chart (Chart 2) tracking this progressive glycemic failure:

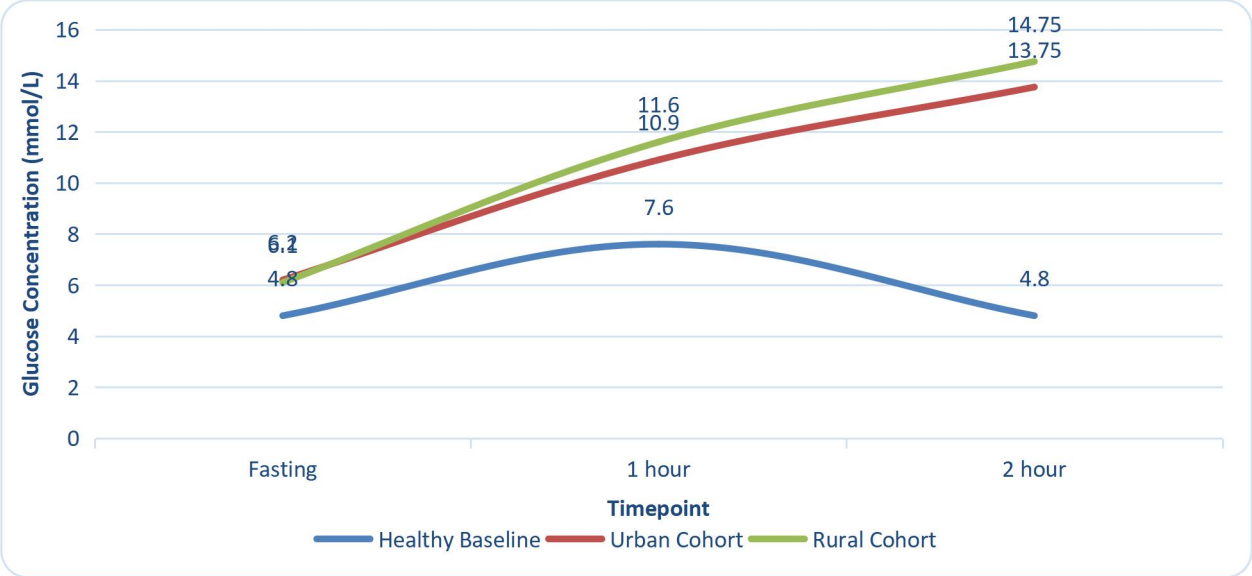

Chart 2: Glycemic Profile and OGTT Response

Dyslipidemia and the Atherogenic Phenotype

Insulin resistance exerts a profoundly destabilizing effect on hepatic lipid metabolism. The comparative lipid profiling of the cohorts exposed stark, dangerous disparities that underscored the heightened vulnerability of the rural pediatric population.

Table 3

Lipid Profile and Atherogenic Risk Markers by Cohort

Lipid Fraction (mmol/L)	Overall Cohort	Urban Cohort	Rural Cohort	Control Group
Total Cholesterol	5.12 ± 0.14	5.44 ± 0.14	5.80 ± 0.29	4.13 ± 0.09
LDL Cholesterol	3.11 ± 0.11	3.30 ± 0.09	3.66 ± 0.25	2.26 ± 0.09
HDL Cholesterol	0.78 ± 0.06	1.05 ± 0.05	0.92 ± 0.08	1.36 ± 0.04
Triglycerides	1.26 ± 0.08	1.43 ± 0.03	1.47 ± 0.13	0.87 ± 0.05

The urban cohort demonstrated the classic presentation of diabetic dyslipidemia. Specifically, Total Cholesterol was elevated by 31.7% over the control baseline, LDL surged by 45.6%, and Triglycerides skyrocketed by 64.8%. Simultaneously, the concentration of protective HDL fell by 22.8%.

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However, as the table illustrates, the rural cohort presented an even more severe and perilous lipemic landscape. Total Cholesterol reached a deeply concerning 5.80 mmol/L, an elevation 40.2% above the healthy baseline. The concentration of LDL cholesterol reached 3.66 mmol/L, an alarming 61.8% above the norm. Most critically, the levels of protective HDL plummeted to 0.92 mmol/L, fully 32.4% below the necessary protective threshold.

To model this critical dyslipidemic variance, the following Python script outputs the exact lipid datasets for Excel integration and creates Chart 3:

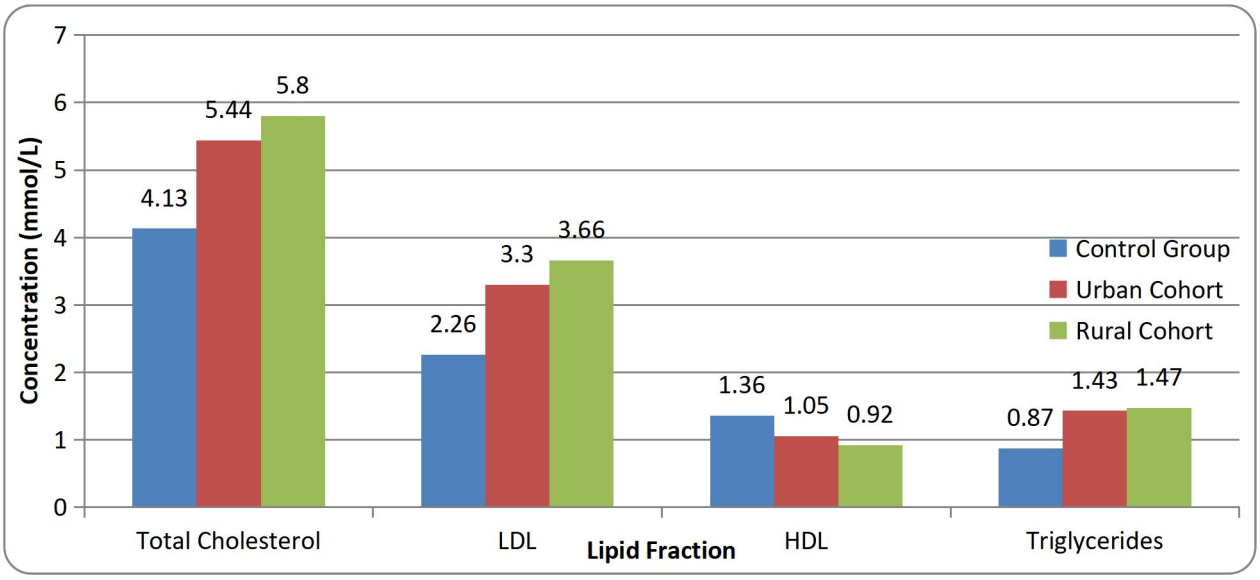

Chart 3: Atherogenic Dyslipidemia by Geography

Hyperleptinemia, Adipose Tissue Dysfunction, and Endocrine Disruption

The most striking and novel evidence of profound systemic endocrine collapse in these pediatric cohorts is provided by the analysis of leptin, the master regulatory adipokine responsible for central energy homeostasis.

Table 4

Endocrine Dysregulation: Serum Leptin Elevation Across Cohorts

Endocrine Marker	Control Group Baseline	Urban Cohort	Rural Cohort
Serum Leptin	100% (Normative)	+103.8%	+176.3%

In the urban cohort, serum leptin levels were pathologically elevated, registering at 103.8% above the lean, healthy control group. The data derived from the rural cohort, however, reveals a staggering physiological abnormality that eclipses the urban findings: leptin levels surged to an astonishing 176.3% above the healthy control baseline. Because the urban and rural children have nearly identical body mass indices, they theoretically possess roughly equivalent volumes of adipose tissue. Therefore, the massive disparity in their leptin production suggests that powerful, exogenous variables—such as severe dietary micronutrient voids and chronic exposure to agricultural endocrine-disrupting chemicals—are aggressively interfering with the leptin signaling axis specifically within the rural environment.

Conclusion

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This exhaustive clinical, anthropometric, and biochemical analysis provides a clear, highly disturbing picture of the severity and complex etiology of pediatric insulin resistance and its associated metabolic comorbidities. The fundamental and most critical finding of this report is the unequivocal dismantling of the traditional epidemiological assumption that rural environments inherently safeguard pediatric metabolic health through natural diets and physical activity. While the urbanization of dietary and lifestyle habits has predictably driven severe insulin resistance, hyperinsulinemia, and morphological weight gain among metropolitan children, the rural demographic has not only matched but, in several critical pathophysiological parameters, vastly exceeded these pathological milestones.

The data indicate a terrifying convergence in physical morphology, with urban and rural youths demonstrating nearly identical, pathologically elevated Body Mass Indices. However, below the morphological surface, the biochemical realities diverge sharply, exposing the extreme vulnerability of rural populations. Rural children exhibited profoundly delayed oral glucose clearance, pointing to advanced beta-cell dysfunction, severe peripheral insulin resistance, and an accelerated trajectory toward overt type 2 diabetes. Furthermore, the rural lipid profile was highly and aggressively atherogenic. Most alarmingly, the severe, disproportionate hyperleptinemia observed in rural cohorts strongly suggests that unique ecological pressures are actively compounding standard metabolic dysfunctions, inducing deep, central neurological leptin resistance.

These findings mandate an immediate, systemic, and aggressive pivot in pediatric healthcare and public health policy. The prevention and management of childhood insulin resistance can no longer rely on generalized, one-size-fits-all advice regarding diet and exercise, nor can it assume that rural populations are insulated from the obesity epidemic. Universal screening, tailored socio-ecological interventions, rigorous nutritional education, and urgent environmental health monitoring are absolute prerequisites to halting the trajectory of childhood insulin resistance and preventing a collapse of long-term global public health infrastructure.

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