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Research Article

Comparison of Prevalence of Pre-Diabetes and Insulin Resistance among Paediatric and Adolescent Individuals with Obese Parents in Pakistan

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Abstract: Urban familial obesity may program metabolic risk across generations, yet data comparing paediatric and adolescent vulnerability to pre-diabetes and insulin resistance in Pakistan remain limited. This cross-sectional analytical study evaluated the prevalence of pre-diabetes (impaired fasting glucose or HbA1c in the pre-diabetic range) and insulin resistance (HOMA-IR) among children (6–11 years, n = 210) and adolescents (12–18 years, n = 210) who each had at least one parent meeting criteria for obesity (BMI ≥30 kg/m2). Participants underwent fasting glucose, HbA1c, fasting insulin and anthropometry three months after screening; HOMA-IR >2.5 was used to define insulin resistance. Prevalence of pre-diabetes was 12.9% in children versus 28.6% in adolescents ($p < 0.001$). Mean HOMA-IR was significantly higher in adolescents (3.12 ± 1.05) than in children (2.21 ± 0.94), $p < 0.001$; adolescents had 2.7 times greater odds of insulin resistance (OR 2.70; 95% CI 1.75–4.18). After adjustment for participant BMI z-score, sedentary hours and household income, adolescent status remained an independent predictor of HOMA-IR (adjusted $\beta = 0.58$, $p = 0.002$). Findings indicate a markedly higher burden of pre-diabetes and insulin resistance in adolescents with obese parents compared with younger children, suggesting age-dependent amplification of inherited and environmental risk. These results highlight an urgent need for family-centred early screening and age-tailored prevention strategies that combine

lifestyle intervention with metabolic monitoring. Keywords: pre-diabetes, insulin resistance, parental obesity.

Introduction: Intergenerational transmission of metabolic risk is increasingly recognised as a major determinant of the early onset of dysglycaemia and cardiometabolic disease. Parental obesity confers risk to offspring through a constellation of mechanisms that include shared family behaviours, intrauterine metabolic programming and epigenetic modifications affecting adipose tissue function and insulin signalling. In low- and middle-income countries, rapid nutritional and lifestyle transitions have amplified both adult obesity and childhood excess adiposity, creating a milieu in which familial clustering of metabolic ill-health is common. Against this background, ascertainment of age-specific vulnerability — particularly the comparative risk of pre-diabetes and insulin resistance in younger children versus adolescents who have obese parents — is essential to inform targeted screening and prevention.1-4

Adolescence is a period of significant physiological, behavioural and hormonal change that often unmasks latent metabolic susceptibility. Pubertal insulin resistance is a well-described phenomenon that superimposes on any inherited or familial predisposition; thus, age stratification is critical in investigations of early dysglycaemia. In paediatric populations, early detection of impaired glycaemic states is clinically consequential because pre-diabetes in youth is associated with more rapid progression to type 2 diabetes and earlier emergence of cardiometabolic complications. Globally, prevalence estimates of youth pre-diabetes have risen over the last two decades, driven largely by the obesity epidemic and socio-environmental determinants that promote sedentary behaviour and caloric excess. This epidemiological shift underscores the need to characterise how parental obesity modifies risk across developmental windows.5-9

Mechanistic research highlights multiple pathways through which parental adiposity increases offspring risk. Maternal obesity during pregnancy produces a nutrient-rich intrauterine environment, placental inflammation and altered adipokine signalling, which together may programme offspring adiposity and insulin resistance. Emerging evidence implicates paternal obesity in shaping offspring metabolic trajectories via alterations in sperm epigenetic marks and small RNAs, suggesting that both parents contribute biologically beyond shared household behaviours. Moreover, family environments characterised by obesogenic diets and low physical

activity often reinforce genetic and epigenetic susceptibilities, leading to an accelerated path toward dysglycaemia in susceptible children and adolescents.10-13

Measurement of insulin resistance in paediatric research commonly uses surrogate indices such as the homeostatic model assessment for insulin resistance (HOMA-IR), which correlates with clamp measures and is feasible in epidemiological settings. While population-specific cut-offs for HOMA-IR vary by age, pubertal status and ethnicity, thresholds around 2.5–3.0 have been applied in adolescent cohorts to indicate clinically relevant insulin resistance. In Pakistan, a rising burden of adult diabetes and increasing childhood obesity have been documented in recent years; these trends make investigation into familial risk particularly urgent in the national context. The local health system's limited resources and competing priorities necessitate evidence that can efficiently identify high-risk subgroups — such as offspring of obese parents — for early intervention.

Previous regional studies have documented notable prevalence of impaired glucose tolerance and insulin resistance among overweight and obese youth; however, few studies have explicitly compared paediatric and adolescent subgroups within families where parental obesity is present. A focused comparison is valuable because it disentangles the contribution of developmental stage from shared familial exposures and may reveal windows of heightened susceptibility when interventions may yield maximal long-term benefit. The current research addresses this gap by comparing prevalence of pre-diabetes and insulin resistance in children and adolescents who have at least one obese parent, while accounting for participant adiposity, activity patterns, socio-economic status and household dietary characteristics.

The primary hypothesis posits that adolescents with obese parents will exhibit significantly higher prevalence of pre-diabetes and higher mean HOMA-IR compared with younger children with obese parents. Secondary objectives include quantifying the independent contribution of participant BMI z-score, sedentary behaviour and household socio-economic variables to insulin resistance, and estimating the odds of insulin resistance associated with adolescent developmental stage after covariate adjustment. By clarifying these relationships in a Pakistani urban/regional sample, the study aims to support targeted family-centred screening policies and age-tailored preventive programmes that prioritise metabolic health before irreversible disease develops.

Methods: A cross-sectional analytical study was conducted among paediatric and adolescent individuals identified through community screening and primary care registers in Allied Hospital-

2. The sample size was calculated using Epi Info StatCalc for comparing two proportions, assuming an expected pre-diabetes prevalence of 30% in adolescents versus 15% in children (based on regional pilot data), alpha 0.05, power 80% and a 1:1 allocation; this produced a required sample of 190 per group which was inflated to 210 per group (total n = 420) to allow for incomplete data and non-adherence. Eligible participants were aged 6–18 years, resided with at least one biological parent, and had documentation or measured evidence of parental obesity defined as BMI ≥30 kg/m2 measured at community screening within the prior six months; recruitment prioritized equal representation of both sexes and a mix of socio-economic strata. Exclusion criteria were known diagnosis of diabetes, chronic endocrine or genetic syndromes affecting growth, current use of systemic glucocorticoids, severe intercurrent illness, and inability to obtain verbal assent/consent. Ethical approval was secured from the institutional review board, and verbal informed consent from caregivers and verbal assent from minors (when appropriate) were obtained following a standardized script that explained study aims, voluntary participation, confidentiality, and the right to withdraw at any time; documentation of verbal consent was recorded by the interviewer. Clinical assessments were performed after an overnight fast of 10–12 hours: weight and height were measured using calibrated instruments and BMI z-scores computed against WHO reference charts; waist circumference was measured at the midpoint between the lowest rib and the iliac crest; blood pressure was measured using automated devices with appropriate cuff sizes; fasting venous blood samples were analysed for plasma glucose, HbA1c, fasting insulin and lipid profile by accredited laboratories; HOMA-IR was calculated as fasting insulin (μIU/mL) × fasting glucose (mmol/L) / 22.5, and a pre-specified HOMA-IR cut-off of >2.5 indicated insulin resistance for analytical comparisons. Pre-diabetes was defined using fasting glucose 100–125 mg/dL or HbA1c 5.7–6.4% in accordance with international paediatric practice. Structured questionnaires captured household income, parental education, physical activity (hours/day of moderate-vigorous activity and sedentary screen time), dietary patterns, and family history of diabetes. Data analysts blinded to participant group conducted statistical analyses using SPSS v26.0: descriptive statistics summarised demographic and metabolic variables; prevalence comparisons used chi-square tests; continuous measures were compared by independent-samples t tests and Mann–Whitney U tests where distributional assumptions failed; multivariable linear regression examined predictors of HOMA-IR and logistic regression estimated odds ratios for insulin resistance and pre-diabetes, adjusting for BMI z-score, sex, sedentary hours and household income. Statistical significance was

set at $p < 0.05$. All fieldwork followed ethical principles and quality-control procedures including double data entry and random audit of 10% of records.

Results

Table 1. Demographic and anthropometric characteristics (n = 420)

Variable	Children (6–11 yrs) n = 210	Adolescents (12–18 yrs) n = 210	p-value
Mean age (yrs)	9.1 ± 1.5	15.0 ± 1.8	<0.001
Female (%)	52.9	50.5	0.61
Mean BMI z-score	1.45 ± 0.78	1.89 ± 0.95	<0.001
Sedentary hours/day (median, IQR)	3 (2–4)	5 (3–6)	<0.001
Household low income (%)	38.6	41.0	0.58

Table 1 shows that adolescents had significantly higher BMI z-scores and sedentary time compared with children; sex distribution and household income were similar between groups.

Table 2. Prevalence of pre-diabetes and insulin resistance by age group

Outcome	Children (n=210) n (%)	Adolescents (n=210) n (%)	p-value ($\chi 2$)	Odds ratio (95% CI)
Pre-diabetes (FBG or HbA1c criteria)	27 (12.9)	60 (28.6)	<0.001	2.65 (1.64–4.29)
Insulin resistance (HOMA-IR >2.5)	48 (22.9)	112 (53.3)	<0.001	3.74 (2.47–5.67)

Table 2 demonstrates a significantly higher prevalence of both pre-diabetes and insulin resistance in adolescents compared with children; adolescents had markedly increased odds of insulin resistance.

Table 3. Metabolic measures (mean ± SD) and group comparisons

Measure	Children (n=210)	Adolescents (n=210)	p-value (t test)
Fasting glucose (mg/dL)	92.4 ± 8.6	98.1 ± 11.2	<0.001
HbA1c (%)	5.4 ± 0.3	5.7 ± 0.5	<0.001
Fasting insulin (μIU/mL)	9.6 ± 4.1	15.2 ± 6.8	<0.001
HOMA-IR	2.21 ± 0.94	3.12 ± 1.05	<0.001

Table 3 indicates significant between-group differences across glycaemic and insulin metrics, with higher fasting glucose, HbA1c, insulin and HOMA-IR in adolescents.

Table 1 characterises the study population and highlights higher adiposity and sedentary behaviour among adolescents. Table 2 reports categorical outcomes demonstrating significantly greater prevalence and odds of pre-diabetes and insulin resistance in adolescents. Table 3 provides continuous metabolic measures showing significantly higher glycaemic and insulin indices in the adolescent group.

Discussion: The findings reveal a pronounced age-related divergence in early dysglycaemia among offspring of obese parents: adolescents displayed substantially higher prevalence of pre-diabetes and insulin resistance than younger children. This pattern aligns with biological plausibility since puberty entails transient physiologic insulin resistance that may amplify any inherited or familial predisposition to impaired glucose regulation. The magnitude of difference observed — more than double the prevalence of pre-diabetes and nearly three- to four-fold higher odds of insulin resistance — is clinically meaningful and suggests adolescence as a critical period for emergence of measurable metabolic derangement in high-risk families.14-17

Adiposity accounted for part of the observed difference, as adolescents had higher BMI z-scores; however, adolescent status remained an independent predictor of HOMA-IR after adjustment for BMI z-score, sedentary behaviour and socio-economic indicators. This indicates that pubertal physiology and potentially age-dependent behavioural patterns (increased autonomy in diet, greater screen time) contribute beyond adiposity per se. The regression findings argue for screening strategies that incorporate age and developmental stage in addition to BMI alone when identifying at-risk youth from families with parental obesity.18

Family-level mechanisms likely include both prenatal programming and postnatal environmental reinforcement. Maternal metabolic milieu during gestation and paternal preconception health exert epigenetic and developmental influences that predispose offspring to adipocyte dysfunction and altered insulin signalling. Concurrently, household food environment, parental modelling of activity and socio-economic constraints shape daily exposures that interact with inherited risk. The study's design could not separate prenatal from postnatal contributions, but the stronger effect in adolescents supports a model where early predispositions are progressively unmasked by developmental processes and cumulative exposures.19-20

The high HOMA-IR values observed in adolescents translate into early cardiometabolic risk that may not be reversible without intervention. Early insulin resistance in youth is associated with dyslipidaemia, hypertension and non-alcoholic fatty liver disease; thus, the present results carry implications for long-term population health and healthcare resource planning. Interventions targeted at families with parental obesity — emphasising structured physical activity, dietary modification, sleep hygiene and reduction of sedentary time — could attenuate trajectory towards diabetes if implemented before or during early adolescence.

From a public-health perspective, findings support prioritising family-centred screening protocols in primary care and school health programmes, particularly in settings with high prevalence of adult obesity. Practical approaches include integrating fasting glucose/HbA1c and fasting insulin measurement in children and adolescents who have obese parents or multiple cardiometabolic risk factors, and deploying tiered interventions where elevated HOMA-IR triggers referral to multidisciplinary lifestyle counselling and follow-up monitoring.

Limitations include the cross-sectional design, which prevents inference on temporal causality or progression; reliance on single fasting measures that may be influenced by short-term variability; and potential selection bias from community recruitment. Ethnic and regional heterogeneity within the country may limit generalisability, and HOMA-IR cut-offs used are surrogate thresholds that necessitate validation in longitudinal cohorts. Strengths include rigorous anthropometric measurement, blinded laboratory analyses, sample size calculation with Epi Info and adjustment for key confounders. Future longitudinal and interventional studies should evaluate whether early family-based prevention during pre-adolescence can attenuate or reverse the metabolic divergence evident in adolescence.

Conclusion: Among offspring of obese parents, adolescence is associated with substantially higher prevalence of pre-diabetes and insulin resistance compared with childhood, independent of adiposity and socio-behavioural factors. These results underscore adolescence as a pivotal window for targeted family-centred screening and preventive interventions to mitigate progression toward type 2 diabetes. Longitudinal and intervention research is needed to establish effective age-specific strategies that disrupt intergenerational transmission of metabolic risk.

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