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Prevalence and Temporal Trends of Clinical Obesity Among Korean Youth, 2007–2024

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Abstract

Background/Objectives: To characterise 18-year trends in the prevalence of clinical obesity among Korean children and adolescents and to compare these trends with BMI-based classifications.

Subjects/Methods: This cross-sectional study included 13,230 participants aged 10–18 years from six survey periods of the Korea National Health and Nutrition Examination Survey (KNHANES; 2007–2024). The primary outcomes were the prevalences of clinical obesity, preclinical obesity, and no obesity, classified according to the Lancet Diabetes & Endocrinology Commission framework, which incorporates excess adiposity and obesity-related organ dysfunction. Temporal trends were assessed using survey-weighted linear regression, with analyses stratified by sex and age group.

Results: Among 13,230 Korean youth aged 10–18 years (mean [standard error] age, 14.09 [0.03] years; 52.81% male), the prevalence of clinical obesity increased from 9.79% in 2007–2009 to 13.91% in 2022–2024 (P for trend < 0.001), with a peak of 16.99% in 2019–2021. The increase was observed in both sexes and across all age groups; the largest absolute increases occurred among males (12.45% to 19.24%) and adolescents aged 16–18 years (8.26% to 17.52%). Among participants with BMI-defined obesity, 80.0% met the criteria for clinical obesity. Clinical obesity was also identified in 20.51% of participants with overweight and 0.81% of those with normal BMI. Dysglycaemia and elevated low-density lipoprotein cholesterol increased in both sexes, whereas low high-density lipoprotein cholesterol and asthma decreased.

Conclusions: These findings support further evaluation of screening strategies that consider obesity-related clinical manifestations and metabolic markers alongside BMI.

INTRODUCTION

Paediatric obesity is a major global health challenge, affecting approximately one in five children and adolescents worldwide. Its global prevalence increased 1.5-fold between 2000–2011 and 2012–2023. [1] Obesity is associated with cardiometabolic comorbidities, including type 2 diabetes mellitus (T2DM), dyslipidaemia, hypertension, and metabolic dysfunction-associated steatotic liver disease (MASLD), and often persists into adulthood, contributing to cardiovascular morbidity across the life course. [2, 3] In Korea, the prevalence of obesity among youth increased from 9.7% in 2012 to 19.3% in 2021, with a steeper increase following the coronavirus disease 2019 (COVID-19) pandemic. [4]

Paediatric obesity is conventionally classified using sex- and age-specific body mass index (BMI) percentile cutoffs. [5] Despite its widespread adoption, BMI has fundamental limitations. This measure fails to account for regional adipose distribution, does not capture metabolic health status, and cannot differentiate fat mass from lean mass. [6, 7] This distinction has prognostic significance because youth with metabolically unhealthy obesity face greater risks of adult metabolic syndrome and T2DM than their metabolically healthy peers with obesity, even after adjustment for BMI. [3, 8] Consequently, BMI-based classification alone cannot distinguish metabolically unhealthy from metabolically healthy phenotypes among youth with obesity or overweight, potentially limiting the identification of those at elevated cardiometabolic risk. [7]

To address these limitations, the Lancet Diabetes & Endocrinology Commission proposed a framework for defining obesity in 2025, distinguishing between clinical and preclinical obesity. Clinical obesity is defined as excess adiposity accompanied by objective evidence of illness resulting from impaired organ or tissue function. Preclinical obesity is defined as excess adiposity with preserved organ and tissue function but an increased risk of developing clinical obesity or other diseases. [9] Studies applying this framework have examined adult populations, including a nationally representative Korean cohort; [10, 11] however, to our knowledge, its application has not yet been evaluated in children or adolescents.

Therefore, we aimed to (1) characterise the prevalence and temporal trends of clinical and preclinical obesity among Korean children and adolescents and (2) assess the agreement and discordance between the clinical obesity framework and conventional BMI-based categories (obesity, overweight, and normal weight) using data from KNHANES 2007–2024.

METHODS

Study Design and Population

This cross-sectional study used data from KNHANES, a nationally representative, population-based survey conducted by the Korea Disease Control and Prevention Agency. We analysed data collected from 2007 through 2024 for participants aged 10–18 years. The participant selection process is shown in Supporting Fig. 1.

KNHANES is a nationally representative surveillance system established in 1998. After periodic implementation through 2005, the survey has been conducted annually since 2007. It uses a stratified, multistage cluster-sampling design to monitor the health and nutritional status of the Korean population through health interviews, physical examinations, laboratory assessments, and nutrition surveys. [12]

Study Variables

Height, weight, and waist circumference (WC) were measured directly by trained examiners using standardised protocols in mobile examination centres; none of these measurements was self-reported. Trained staff measured blood pressure (BP) using a standardised protocol.

BMI standard deviation scores (SDSs) and percentiles were derived using the 2017 Korean National Growth Charts. [13] WC percentiles were calculated using sex- and age-specific reference values. [14] Central obesity based on WC (CO-WC) was defined as WC ≥ 90th percentile for age and sex, and central obesity based on waist-to-height ratio (WHtR; waist circumference divided by height) (CO-WHtR) was defined as WHtR ≥ 0.50. [2, 14, 15] BP percentiles were determined using Korean age-, sex-, and height-specific reference values. [16] Blood samples for laboratory testing, including glucose, lipid profile, glycated haemoglobin (HbA1c), aspartate aminotransferase, alanine aminotransferase (ALT), and creatinine, were collected from the antecubital vein after an 8-hour fast, processed, and refrigerated until analysis.

Definition of Clinical Obesity

Clinical and preclinical obesity were defined according to the diagnostic framework proposed by the Lancet Diabetes & Endocrinology Commission. [9] Excess adiposity was defined as meeting either of the following criteria: (A) at least two of these three measures—BMI ≥ the 95th percentile, WC ≥ the 90th percentile, or WHtR ≥ 0.50—or (B) BMI ≥ 140% of the 95th percentile (Supporting Fig. 2). [2, 10, 17] Participants with excess adiposity and at least one obesity-related comorbidity were classified as having clinical obesity; those with excess adiposity but no comorbidities were classified as having preclinical obesity; all other participants were classified as having no obesity.

Obesity-related comorbidities included a history of asthma, diabetes mellitus, hypertension, or dyslipidaemia; cardiovascular dysfunction, defined as systolic or diastolic BP ≥ the 90th percentile; dysglycaemia, defined as HbA1c ≥ 5.7% and/or fasting glucose ≥ 100 mg/dL; dyslipidaemia, defined as triglycerides (TG) > 150 mg/dL, high-density lipoprotein cholesterol (HDL-C) ≤ 40 mg/dL, or low-density lipoprotein cholesterol (LDL-C) ≥ 130 mg/dL; hepatic impairment, defined as ALT ≥ 26 U/L in males or ≥ 22 U/L in females; and renal dysfunction, defined as estimated glomerular filtration rate (eGFR) < 90 mL/min/1.73 m² and an albumin-to-creatinine ratio ≥ 30 mg/g. [9, 10, 16, 18–21]

Statistical Analysis

All analyses were performed using R version 4.4.1 (R Foundation for Statistical Computing, Vienna, Austria) and accounted for the KNHANES complex sampling design by incorporating sampling weights, strata, and cluster variables. Continuous variables are presented as weighted means with standard errors, and categorical variables as weighted percentages with standard errors. Between-group differences were assessed using survey-weighted analysis of variance for continuous variables and the Rao–Scott chi-square test for categorical variables. Missing data were not imputed. For variables not collected in certain survey years, analyses were restricted to years with available data; all other missing observations were handled using complete-case analysis. Trends in obesity prevalence across survey periods were evaluated using survey-weighted linear regression, with calendar year treated as a continuous variable. Subgroup analyses were conducted by BMI category, sex, and age group (10–12, 13–15, and 16–18 years). Statistical significance was defined as $P < 0.05$.

RESULTS

Prevalence of Clinical Obesity Compared with Anthropometric Definitions

A total of 13,230 participants aged 10–18 years (7,009 boys and 6,221 girls) were included in the final analysis (Supporting Fig. 1). Baseline characteristics stratified by clinical obesity category are presented in Table 1. According to the Lancet Diabetes & Endocrinology Commission framework, 85.82% of participants were classified as having no obesity, 2.36% as having preclinical obesity, and 11.81% as having clinical

obesity. Participants with clinical obesity were predominantly male and had higher BMI SDS, WC, and TG and ALT levels, as well as lower HDL-C levels, than those with no obesity (all $P < 0.001$).

Table 1
Baseline characteristics of the participants according to definition of clinical obesity

Variable	No obesity (n = 11 395)	Preclinical obesity (n = 322)	Clinical obesity (n = 1 513)	*P*-value
Age, yr	14.10 (0.03)	14.01 (0.16)	14.06 (0.08)	0.779
Sex (male), %	51.07 (0.52)	47.63 (3.21)	66.43 (1.41)	< 0.001
BMI SDS	-0.35 (0.01)	1.87 (0.05)	2.28 (0.03)	< 0.001
BMI Categories, %				
Normal BMI	90.99 (0.31)	9.62 (2.00)	5.42 (0.64)	< 0.001
Overweight	8.13 (0.28)	24.95 (2.63)	16.52 (1.07)	< 0.001
Obesity	0.89 (0.10)	65.43 (3.03)	78.06 (1.23)	< 0.001
WC, cm	67.43 (0.09)	84.69 (0.46)	88.87 (0.30)	< 0.001
CO–WC, %	3.99 (0.21)	97.83 (0.90)	98.05 (0.47)	< 0.001
CO–WHtR, %	1.54 (0.13)	87.08 (2.01)	91.39 (0.84)	< 0.001
SBP, mmHg	106.50 (0.12)	108.14 (0.49)	114.81 (0.35)	< 0.001
DBP, mmHg	66.00 (0.11)	64.06 (0.46)	69.06 (0.30)	< 0.001
Fasting glucose, mg/dL	90.25 (0.10)	89.85 (0.36)	93.98 (0.45)	< 0.001
Total cholesterol, mg/dL	158.62 (0.33)	161.92 (1.23)	174.08 (1.02)	< 0.001
TG, mg/dL	81.28 (0.57)	83.67 (1.60)	129.30 (2.68)	< 0.001
HDL, mg/dL	52.79 (0.14)	50.45 (0.50)	44.71 (0.30)	< 0.001
LDL, mg/dL	91.17 (0.28)	96.68 (1.09)	107.76 (0.92)	< 0.001
HbA1c, %	5.36 (0.00)	5.31 (0.02)	5.51 (0.02)	< 0.001
AST, IU/L	18.96 (0.10)	17.68 (0.28)	25.53 (0.53)	< 0.001
ALT, IU/L	13.60 (0.13)	14.21 (0.31)	35.01 (1.11)	< 0.001
Ualb, µg/ml	18.78 (1.58)	13.19 (2.32)	13.03 (1.70)	0.024
Ucrea, mg/dL	199.67 (1.31)	208.38 (7.45)	194.51 (3.28)	0.142

Note: Values are presented as survey-weighted means ± standard errors (SE) or percentages (SE).

Abbreviations: BMI SDS, Body Mass Index Standard Deviation Score; BMI, Body Mass Index; WC, Waist Circumference; CO–WC, Central obesity based on Waist Circumference; CO–WHtR, central obesity based on Waist-to-Height Ratio; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; TG, Triglycerides; HDL, High-Density Lipoprotein; LDL, Low-Density Lipoprotein; HbA1c, Glycated Hemoglobin; AST, Aspartate Aminotransferase; ALT, Alanine Aminotransferase; Ualb, Urinary Albumin; Ucrea, Urinary Creatinine; eGFR, Estimated Glomerular Filtration Rate; ACR, Albumin-to-Creatinine Ratio.

Variable	No obesity (n = 11 395)	Preclinical obesity (n = 322)	Clinical obesity (n = 1 513)	*P*-value
Creatinine, mg/dL	0.71 (0.00)	0.70 (0.01)	0.71 (0.01)	0.480
eGFR, mL/min/1.73 m^2	97.77 (0.26)	99.21 (1.23)	99.20 (0.65)	0.053
ACR, mg/g	8.90 (0.58)	6.29 (0.79)	7.39 (1.60)	0.028
Clinical obesity-related comorbidities, %				
Asthma	4.04 (0.23)	0.00 (0.00)	5.85 (0.75)	< 0.001
High BP	13.00 (0.38)	0.00 (0.00)	36.47 (1.44)	< 0.001
Dysglycemia	14.16 (0.42)	0.00 (0.00)	29.51 (1.38)	< 0.001
High TG	6.28 (0.30)	0.00 (0.00)	29.40 (1.49)	< 0.001
High LDL	5.22 (0.25)	0.00 (0.00)	19.41 (1.32)	< 0.001
Low HDL	9.62 (0.36)	0.00 (0.00)	34.96 (1.61)	< 0.001
ALT elevation	6.19 (0.30)	0.00 (0.00)	50.46 (1.67)	< 0.001
Albuminuria with Low GFR	0.78 (0.15)	0.00 (0.00)	1.08 (0.56)	0.750
Nutrition				
Energy, kcal	2060.59 (10.75)	1984.86 (48.41)	2037.13 (28.17)	0.246
Carbohydrate, g	308.74 (1.60)	290.47 (6.87)	302.63 (4.24)	0.019
Protein, g	74.80 (0.50)	76.80 (2.57)	76.45 (1.19)	0.339
Fat, g	56.60 (0.45)	56.58 (2.31)	55.76 (1.14)	0.789

Note: Values are presented as survey-weighted means ± standard errors (SE) or percentages (SE).

Abbreviations: BMI SDS, Body Mass Index Standard Deviation Score; BMI, Body Mass Index; WC, Waist Circumference; CO–WC, Central obesity based on Waist Circumference; CO–WHtR, central obesity based on Waist-to-Height Ratio; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; TG, Triglycerides; HDL, High-Density Lipoprotein; LDL, Low-Density Lipoprotein; HbA1c, Glycated Hemoglobin; AST, Aspartate Aminotransferase; ALT, Alanine Aminotransferase; Ualb, Urinary Albumin; Ucrea, Urinary Creatinine; eGFR, Estimated Glomerular Filtration Rate; ACR, Albumin-to-Creatinine Ratio.

Supporting Table 1 presents baseline characteristics by BMI category. Overall, 78.95% of participants had normal BMI, 9.52% had overweight, and 11.53% had obesity. Supporting Table 2 presents baseline characteristics by sex. The prevalence of BMI-defined obesity was 13.21% among males and 9.65% among females, whereas the prevalence of clinical obesity was 14.86% and 8.40%, respectively.

Table 2
Trends in the prevalence of obesity and clinical obesity among the total participants

Variable	Total (n = 13 230)	2007–2009 (n = 2 971)	2010–2012 (n = 2 699)	2013–2015 (n = 2 232)	2016–2018 (n = 2 066)	2019–2021 (n = 1 771)	2022–2024 (n = 1 491)	*P* for trend
Age, yr	14.09 (0.03)	13.97 (0.06)	14.16 (0.07)	14.15 (0.06)	14.30 (0.07)	14.03 (0.07)	13.93 (0.08)	0.454
Sex (male), %	52.81 (0.48)	53.27 (1.01)	53.88 (1.14)	52.92 (1.14)	52.01 (1.13)	52.75 (1.30)	51.37 (1.39)	0.176
BMI SDS	0.01 (0.01)	-0.10 (0.03)	-0.10 (0.03)	0.05 (0.03)	0.00 (0.04)	0.19 (0.05)	0.13 (0.05)	< 0.001
BMI Categories, %								
Normal BMI	78.95 (0.42)	81.23 (0.83)	81.14 (0.87)	79.88 (0.93)	78.93 (1.05)	74.01 (1.41)	76.28 (1.32)	< 0.001
Overweight	9.52 (0.28)	10.16 (0.62)	9.33 (0.66)	9.29 (0.64)	9.30 (0.69)	9.67 (0.80)	9.21 (0.80)	0.487
Obesity	11.53 (0.35)	8.61 (0.65)	9.53 (0.70)	10.82 (0.75)	11.77 (0.85)	16.32 (1.17)	14.51 (1.12)	< 0.001
WC, cm	70.37 (0.12)	69.48 (0.24)	69.22 (0.23)	70.41 (0.27)	70.31 (0.29)	72.17 (0.37)	71.52 (0.38)	< 0.001
CO–WC, %	17.32 (0.40)	15.18 (0.75)	14.52 (0.83)	16.48 (0.89)	16.53 (1.00)	22.45 (1.30)	21.31 (1.27)	< 0.001
CO–WHtR, %	14.18 (0.37)	12.14 (0.68)	11.05 (0.72)	13.34 (0.85)	13.71 (0.90)	19.53 (1.23)	17.89 (1.18)	< 0.001
SBP, mmHg	107.53 (0.12)	105.45 (0.28)	107.09 (0.28)	108.24 (0.27)	108.47 (0.28)	108.54 (0.35)	108.31 (0.31)	< 0.001
DBP, mmHg	66.32 (0.11)	66.66 (0.27)	66.29 (0.27)	65.97 (0.23)	66.60 (0.24)	67.10 (0.31)	65.16 (0.23)	0.041
Fasting glucose, mg/dL	90.66 (0.11)	89.07 (0.24)	88.80 (0.20)	91.64 (0.31)	91.57 (0.22)	92.46 (0.27)	91.52 (0.29)	< 0.001

Note: Values are presented as survey-weighted means ± standard errors (SE) or percentages (SE).

Abbreviations: BMI SDS, Body Mass Index Standard Deviation Score; BMI, Body Mass Index; WC, Waist Circumference; CO–WC, Central obesity based on Waist Circumference; CO–WHtR, central obesity based on Waist-to-Height Ratio; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; TG, Triglycerides; HDL, High-Density Lipoprotein; LDL, Low-Density Lipoprotein; HbA1c, Glycated Hemoglobin; AST, Aspartate Aminotransferase; ALT, Alanine Aminotransferase; Ualb, Urinary Albumin; Ucrea, Urinary Creatinine; eGFR, Estimated Glomerular Filtration Rate; ACR, Albumin-to-Creatinine Ratio; BP, Blood Pressure; GFR, Glomerular Filtration Rate; NA, Not applicable.

Variable	Total (n = 13 230)	2007–2009 (n = 2 971)	2010–2012 (n = 2 699)	2013–2015 (n = 2 232)	2016–2018 (n = 2 066)	2019–2021 (n = 1 771)	2022–2024 (n = 1 491)	*P* for trend
Total cholesterol, mg/dL	160.47 (0.32)	157.82 (0.71)	157.98 (0.79)	158.59 (0.70)	164.86 (0.75)	163.73 (0.86)	161.50 (0.91)	< 0.001
TG, mg/dL	86.82 (0.63)	89.47 (1.46)	83.38 (1.43)	85.23 (1.43)	86.67 (1.38)	88.26 (1.61)	88.19 (1.99)	0.493
HDL, mg/dL	51.81 (0.13)	49.48 (0.25)	50.78 (0.32)	51.83 (0.27)	51.60 (0.28)	51.90 (0.32)	56.98 (0.47)	< 0.001
LDL, mg/dL	93.20 (0.28)	90.60 (0.59)	91.61 (0.66)	91.07 (0.63)	96.53 (0.65)	94.87 (0.74)	96.21 (0.81)	< 0.001
HbA1c, %	5.38 (0.00)	5.98 (0.39)	5.42 (0.01)	5.46 (0.01)	5.34 (0.01)	5.41 (0.01)	5.25 (0.01)	0.050
AST, IU/L	19.68 (0.11)	19.31 (0.18)	18.99 (0.19)	18.99 (0.26)	19.77 (0.24)	21.59 (0.30)	19.82 (0.43)	< 0.001
ALT, IU/L	16.06 (0.18)	15.49 (0.30)	14.98 (0.37)	15.36 (0.48)	16.52 (0.54)	18.09 (0.52)	16.55 (0.54)	< 0.001
Ualb, µg/ml	17.90 (1.36)	NA	15.73 (1.95)	17.28 (1.56)	NA	17.48 (1.06)	21.06 (4.80)	0.301
Ucrea, mg/dL	199.24 (1.22)	NA	198.67 (2.72)	204.64 (3.02)	204.64 (2.51)	198.49 (2.41)	188.46 (2.78)	0.002
Creatinine, mg/dL	0.71 (0.00)	0.76 (0.01)	0.71 (0.00)	0.73 (0.00)	0.70 (0.00)	0.67 (0.00)	0.67 (0.00)	< 0.001
eGFR, mL/min/1.73 m^2	97.97 (0.25)	91.02 (0.63)	96.71 (0.47)	95.53 (0.49)	99.94 (0.60)	104.35 (0.60)	103.92 (0.60)	< 0.001
ACR, mg/g	8.64 (0.54)	NA	6.80 (0.67)	8.58 (0.71)	NA	9.24 (0.77)	9.76 (1.74)	0.094
Clinical obesity-related comorbidities, %								

Note: Values are presented as survey-weighted means ± standard errors (SE) or percentages (SE).

Abbreviations: BMI SDS, Body Mass Index Standard Deviation Score; BMI, Body Mass Index; WC, Waist Circumference; CO–WC, Central obesity based on Waist Circumference; CO–WHtR, central obesity based on Waist-to-Height Ratio; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; TG, Triglycerides; HDL, High-Density Lipoprotein; LDL, Low-Density Lipoprotein; HbA1c, Glycated Hemoglobin; AST, Aspartate Aminotransferase; ALT, Alanine Aminotransferase; Ualb, Urinary Albumin; Ucrea, Urinary Creatinine; eGFR, Estimated Glomerular Filtration Rate; ACR, Albumin-to-Creatinine Ratio; BP, Blood Pressure; GFR, Glomerular Filtration Rate; NA, Not applicable.

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Asthma	4.16 (0.21)	4.11 (0.40)	5.16 (0.55)	4.91 (0.55)	3.90 (0.48)	2.89 (0.48)	2.08 (0.69)	0.002
High BP	15.48 (0.39)	15.52 (0.83)	15.15 (0.87)	15.12 (0.93)	16.10 (0.98)	18.93 (1.21)	12.16 (0.93)	0.693
Dysglycemia	15.60 (0.41)	5.33 (0.49)	14.09 (0.97)	23.86 (1.15)	16.72 (1.00)	23.80 (1.15)	13.27 (1.08)	< 0.001
High TG	8.75 (0.33)	9.67 (0.67)	8.61 (0.80)	7.64 (0.75)	8.23 (0.74)	9.65 (0.92)	8.49 (1.03)	0.646
High LDL	6.70 (0.27)	4.98 (0.50)	5.87 (0.61)	5.25 (0.56)	8.51 (0.72)	7.83 (0.78)	8.81 (0.88)	< 0.001
Low HDL	12.25 (0.38)	16.62 (0.97)	13.20 (0.93)	9.75 (0.75)	11.25 (0.79)	12.41 (1.10)	8.22 (0.91)	< 0.001
ALT elevation	11.09 (0.37)	11.06 (0.86)	8.17 (0.72)	9.10 (0.77)	12.34 (0.92)	15.16 (1.05)	11.71 (1.14)	< 0.001
Albuminuria with Low GFR	0.80 (0.15)	NA	0.97 (0.36)	1.23 (0.39)	NA	0.81 (0.26)	0.25 (0.15)	0.043
Nutrition								
Energy, kcal	2056.04 (9.92)	1943.80 (18.23)	2192.25 (24.93)	2204.21 (25.75)	2114.86 (26.92)	1981.78 (29.08)	1894.07 (23.61)	< 0.001
Carbohydrate, g	307.59 (1.48)	308.54 (2.91)	337.23 (3.67)	325.96 (3.51)	314.40 (4.28)	281.55 (3.87)	264.17 (3.28)	< 0.001
Protein, g	75.04 (0.46)	68.50 (0.78)	79.23 (1.22)	79.00 (1.28)	77.34 (1.22)	75.21 (1.35)	72.94 (1.11)	0.338
Fat, g	56.50 (0.42)	48.04 (0.77)	58.22 (1.09)	60.69 (1.02)	58.15 (1.10)	59.34 (1.27)	58.46 (1.09)	< 0.001
Clinical obesity categories. %								
No obesity	85.82 (0.37)	87.90 (0.69)	88.51 (0.75)	87.04 (0.81)	86.21 (0.91)	80.61 (1.24)	82.16 (1.16)	< 0.001

Note: Values are presented as survey-weighted means ± standard errors (SE) or percentages (SE).

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Preclinical obesity	2.36 (0.15)	2.32 (0.29)	2.11 (0.32)	2.02 (0.37)	1.73 (0.30)	2.40 (0.38)	3.93 (0.52)	0.020
Clinical obesity	11.81 (0.34)	9.79 (0.66)	9.38 (0.69)	10.94 (0.75)	12.06 (0.85)	16.99 (1.17)	13.91 (1.07)	< 0.001

Note: Values are presented as survey-weighted means ± standard errors (SE) or percentages (SE).

Abbreviations: BMI SDS, Body Mass Index Standard Deviation Score; BMI, Body Mass Index; WC, Waist Circumference; CO–WC, Central obesity based on Waist Circumference; CO–WHtR, central obesity based on Waist-to-Height Ratio; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; TG, Triglycerides; HDL, High-Density Lipoprotein; LDL, Low-Density Lipoprotein; HbA1c, Glycated Hemoglobin; AST, Aspartate Aminotransferase; ALT, Alanine Aminotransferase; Ualb, Urinary Albumin; Ucrea, Urinary Creatinine; eGFR, Estimated Glomerular Filtration Rate; ACR, Albumin-to-Creatinine Ratio; BP, Blood Pressure; GFR, Glomerular Filtration Rate; NA, Not applicable.

Among youth with clinical obesity, 78.06% had BMI-defined obesity, 16.52% were classified as having overweight, and 5.42% had normal BMI (Fig. 1A, Table 1). Among participants classified as having no obesity under the clinical obesity framework, 8.13% had overweight and 0.89% had BMI-defined obesity. Among youth with BMI-defined obesity, 80.00% met the criteria for clinical obesity, whereas 6.59% were reclassified as having no obesity under the clinical obesity framework (Fig. 1D, Supporting Table 1). Additionally, 20.51% of participants with overweight and 0.81% of those who had normal BMI also met the clinical obesity criteria. Among participants with BMI-defined obesity, 13.41% had preclinical obesity and 6.59% had no obesity. Among participants with CO-WC, 66.9% met the criteria for clinical obesity, 13.3% for preclinical obesity, and 19.8% for no obesity (Supporting Fig. 3A). When CO-WHtR was used, the corresponding values were 76.2%, 14.5%, and 9.3%, respectively (Supporting Fig. 4A).

Among participants with clinical obesity, ALT elevation was the most prevalent organ dysfunction (50.46%), followed by elevated BP (36.47%), low HDL-C (34.96%), dysglycaemia (29.51%), high TG (29.40%), high LDL-C (19.41%), asthma (5.85%), and albuminuria with reduced eGFR (1.08%) (Table 1).

Temporal Trends in Prevalence of Obesity and Organ Dysfunction

Temporal Trends in Total Population

Across the 18-year study period, the prevalence of all obesity indicators increased (Table 2, Fig. 2A, and Supporting Fig. 5A). Clinical obesity increased from 9.79% to 13.91%, peaking at 16.99% in 2019–2021 (P for trend < 0.001). Preclinical obesity increased from 2.32% to 3.93% (P for trend = 0.020), BMI-defined obesity from 8.61% to 14.51% (P for trend < 0.001), CO-WC from 15.18% to 21.31% (P for trend < 0.001), and CO-WHtR from 12.14% to 17.89% (P for trend < 0.001). Among obesity-related conditions and clinical indicators, dysglycaemia, elevated LDL-C, and ALT elevation increased over time, whereas low HDL-C and asthma decreased (Table 2 and Fig. 3A).

Temporal Trends According to Sex

The prevalence of clinical obesity and BMI-defined obesity increased in both males and females during the study period (Supporting Table 3, Figs. 2B–C, and Supporting Figs. 5B–C). Among males, clinical obesity increased from 12.45% to 19.24% (P for trend < 0.001), preclinical obesity from 1.93% to 4.10% (P for trend = 0.005), and BMI-defined obesity from 9.90% to 18.31% (P for trend < 0.001). Among females, clinical obesity increased from 6.74% to 8.27% (P for trend = 0.016), and BMI-defined obesity from 7.14% to 10.50% (P for trend = 0.003). Clinical obesity prevalence peaked in 2019–2021 in both males (22.27%) and females (11.10%). For obesity-related conditions and clinical indicators, dysglycaemia, elevated LDL-C, and ALT elevation increased among males, whereas low HDL-C, asthma, and albuminuria with low eGFR decreased (Fig. 3B). Among females, dysglycaemia and elevated LDL-C increased, whereas high TG, low HDL-C, and asthma decreased; ALT elevation showed no significant trend (Fig. 3C).

Temporal Trends According to Age

Clinical obesity and BMI-defined obesity increased across all age groups during the study period (Supporting Table 4, Figs. 2D–F, and Supporting Figs. 5D–F). Among participants aged 10–12 years, clinical obesity increased from 11.89% to 14.39% (P for trend = 0.002), and BMI-defined obesity increased from 7.36% to 12.78% (P for trend < 0.001). Among those aged 13–15 years, clinical obesity increased from 9.14% to 10.02% (P for trend = 0.038), and BMI-defined obesity increased from 9.72% to 11.96% (P for trend = 0.017). Among participants aged 16–18 years, clinical obesity increased from 8.26% to 17.52% (P for trend < 0.001), and BMI-defined obesity increased from 8.77% to 19.07% (P for trend < 0.001). Dysglycaemia and elevated LDL-C increased across all age groups (Fig. 3D–F). ALT elevation increased among participants aged 13–15 and 16–18 years (P = 0.016 and P < 0.001, respectively), whereas low HDL-C decreased among those aged 10–12 and 13–15 years.

DISCUSSION

In this nationally representative sample of Korean children and adolescents, the prevalence of clinical obesity increased from 9.79% in 2007–2009 to 13.91% in 2022–2024, with a peak of 16.99% in 2019–2021. This increase was observed in both sexes and across all age groups, with particularly large increases among males and adolescents aged 16–18 years. The clinical obesity framework and conventional BMI-based classification also yielded different classifications for some participants: 5.42% of those with clinical obesity had normal BMI, whereas 6.59% of those with BMI-defined obesity were classified as having neither clinical nor preclinical obesity.

The prevalence of BMI-defined obesity, abdominal obesity, clinical obesity, and preclinical obesity increased during 2007–2024, consistent with epidemiological trends previously reported among Korean youth. [22–24] Clinical obesity peaked in 2019–2021, coinciding with pandemic-related disruptions to lifestyle behaviours, including social-distancing measures, school closures, and lockdowns in Korea. [22, 25] During this period, the proportion of Korean youth meeting aerobic-exercise guidelines decreased from approximately 29.4% before the pandemic to 23.8% in 2020, [26] alongside a reduction in physical activity across the cohort. The increase observed before the pandemic may reflect longer-term changes in the

obesogenic environment, including a sustained rise in total fat and saturated fatty acid intake among Korean youth during the preceding decade. [27] These concurrent dietary and physical-activity trends underscore the importance of addressing structural determinants of paediatric obesity in Korea.

The present analysis identified discordance between conventional anthropometric classifications and the clinical obesity framework. Some participants with normal BMI met the clinical obesity criteria because they met alternative anthropometric criteria for excess adiposity and had qualifying clinical manifestations. Conversely, some participants with BMI-defined obesity were classified as having preclinical obesity or no obesity. Similar differences have been reported in adult populations. A US nationwide study found a lower prevalence of clinical obesity than of BMI-defined obesity, with substantial proportions of participants identified by only one of the two definitions. [10] A study of Korean adults likewise reported that 20.1% of adults with BMI-defined obesity had no obesity-related complications, whereas 19.4% of adults with overweight met the clinical obesity criteria under the newer framework. [11] This heterogeneity is consistent with the concept of metabolically healthy obesity, in which some individuals with obesity or overweight do not have cardiometabolic risk factors; thus, a single anthropometric threshold may not capture the full spectrum of obesity-related disease burden. [28] Population-based studies have also highlighted metabolic risk among youth with normal BMI, including adverse lipid profiles, prediabetes, and non-alcoholic fatty liver disease. [15, 29] Taken together, these findings suggest that BMI or WC alone may underestimate obesity-related comorbidity in youth. Future studies incorporating direct measures of body composition, limitations in daily activities, and functional impairment are warranted to characterise the clinical impact of obesity in paediatric populations more precisely.

Although the overall prevalence of any organ dysfunction remained statistically stable during the study period, this summary masked different trends among individual components. Dysglycemia and elevated LDL-C increased in both sexes. The increase in dysglycaemia may reflect worsening insulin resistance associated with increasing adiposity and declining physical activity. Elevated LDL-C is also a concern because adverse lipid profiles during adolescence can persist into adulthood and contribute to early atherosclerosis. [30–32] In contrast, asthma and low HDL-C became less prevalent. The decline in low HDL-C despite increasing obesity prevalence may be related to changes in dietary macronutrient composition; however, dietary intake was not assessed as an explanation for this trend in the present study. [33, 34] Changes in dietary intake may also have contributed to the increase in LDL-C through greater saturated fatty acid intake, illustrating the difficulty of interpreting individual lipid markers in isolation. [27, 35] Among males, ALT elevation increased, a finding that may be consistent with a rise in MASLD associated with greater visceral adiposity. [36] The absence of a significant trend in ALT elevation among females may reflect sex differences in fat distribution or a possible hepatoprotective effect of oestrogen during puberty. [37]

Among males, both preclinical and clinical obesity increased significantly, indicating increases in the prevalence of both categories alongside increasing adiposity. Among females, clinical obesity increased, whereas preclinical obesity showed no significant change. These differences may reflect changes over time in the distribution of clinical manifestations among females; however, the cross-sectional design does not allow assessment of transitions between obesity categories. Further research is needed to examine

potential sex differences in the relationships between adiposity and clinical manifestations. [38] The proportion of Korean adolescent girls meeting aerobic-exercise guidelines was substantially lower than that of boys (11.3% vs. 29.1%). Together with pubertal changes in insulin sensitivity, this difference may contribute to metabolic vulnerability. [26, 39] These findings support further evaluation of screening approaches that extend beyond BMI to include fasting glucose, HbA1c, and lipid measurements in adolescents with excess adiposity. Longitudinal studies are needed to clarify these patterns.

This study has several limitations. First, its cross-sectional design precludes causal inference and the assessment of individual trajectories over time. Second, BMI percentiles, although central to the classification, may not fully capture body composition. In addition, body-composition data were unavailable for trend analyses because these data were collected in only a limited number of survey years and established paediatric thresholds for excess adiposity were lacking. Finally, the classification of clinical obesity was limited by the absence of data on activity limitations in KNHANES and by reliance on a defined set of laboratory markers. Despite these limitations, to our knowledge, this is the first study to characterise the prevalence and 18-year temporal trends of clinical obesity, as defined by the Lancet Diabetes & Endocrinology Commission framework, in a nationally representative sample of Korean children and adolescents.

CONCLUSION

The prevalence of clinical obesity increased significantly among Korean children and adolescents over the 18-year study period. The clinical obesity framework identified a subgroup of participants not captured by BMI-based classification alone, underscoring the limitations of using anthropometric indices as the sole screening approach. Dysglycaemia and elevated LDL-C showed the most consistent adverse trends, whereas the increase in ALT elevation among males may indicate a growing burden of MASLD. Sex-specific differences in obesity trends warrant further investigation of screening strategies that incorporate metabolic markers. Addressing paediatric clinical obesity will require coordinated clinical and public-health efforts targeting structural determinants of the obesogenic environment affecting Korean youth.

Declarations

Author Contributions:

Y.J. Kwon and K. Song are co-corresponding authors.

Concept and design: Y. Choi, Y.J. Kwon, and K. Song

Acquisition, analysis, or interpretation of data: H. S. Lee and E. Lee

Statistical analysis: H. S. Lee and E. Lee

Drafting of the manuscript: Y. Choi, K. Song, and Y.J. Kwon

Critical revision of the manuscript for important intellectual content: All authors

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Ethics Statement

The study protocol was reviewed and approved by the Institutional Review Board (IRB) of Gangnam Severance Hospital (IRB No. 3-2026-0051). This study was conducted in accordance with the ethical standards of the Declaration of Helsinki. The IRB waived the requirement for informed consent because this study utilized publicly available, de-identified data from the KNHANES.

Competing Interest Statement:

The authors declare no conflicts of interest.

When available:

Data are available upon publication.

Supporting Documents

Document types: None.

Data Availability Statement:

Data

Data available: Yes.

Data types: De-identified participant data.

How to access data:

Data are publicly available through the Korea Disease Control and Prevention Agency (https://knhanes.kdca.go.kr).

Additional Information

Data access: Publicly available datasets; no additional approval required.

Types of analyses: For scientific research purposes only.

Mechanisms of data availability: Not applicable.

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