BMJ Paediatrics Open

Metabolically obese normal weight phenotype in children and adolescents aged 3 to 18 years: a systematic review

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To cite: Farooqui M, Selvam S, Kuriyan R. Metabolically obese normal weight phenotype in children and adolescents aged 3 to 18 years: a systematic review. *BMJ Paediatrics Open* 2026;**10**:e004412. doi:10.1136/bmjpo-2025-004412

► Additional supplemental material is published online only. To view, please visit the journal online (https://doi.org/10.1136/bmjpo-2025-004412).

Received 21 December 2025
Accepted 17 March 2026

Check for updates

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ABSTRACT

Background Children with normal Body Mass Index (BMI) may still exhibit adverse metabolic profile—termed as the metabolically obese normal weight (MONW) phenotype, placing them at increased risk of long-term cardiometabolic complications.

Objective To systematically synthesise the evidence of the MONW phenotype in children and adolescents aged 3–18 years, and to identify early-life determinants.

Methods This systematic review followed Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines and searched PubMed/MEDical Literature Analysis and Retrieval System On-Line (MEDLINE), Scopus, Web of Science (WoS), Google Scholar, Embase and Cochrane Library for studies from 2000 to January 2026. Observational studies of children and adolescents aged 3–18 years with normal BMI and at least one metabolic abnormality were included. Two reviewers independently screened the studies, extracted data and assessed risk of bias. Searches were conducted between March 2025 and January 2026, and 11 studies met the predefined inclusion and exclusion criteria.

Results The reported prevalence of MONW ranged from 10.6% to 56.2%, varying by diagnostic criteria and population. Children with MONW consistently showed higher visceral adiposity, insulin resistance (IR), dyslipidaemia, hypertension, impaired glucose metabolism and elevated markers of low-grade inflammation despite having normal BMI. Early-life determinants included extremes of birth weight, rapid infant weight gain, adverse maternal metabolic status, unhealthy dietary patterns and sedentary behaviour. Overall risk of bias across studies was judged to be low to moderate, with the main concerns relating to residual confounding and use of heterogeneous MONW definitions.

Conclusions MONW represents a clinically relevant but under-recognised paediatric phenotype. Reliance on BMI alone may delay identification of vulnerable children. Early risk stratification integrating metabolic proofing and central adiposity measures may enable timely prevention of long-term metabolic disease. Harmonised operational definitions of MONW and robust longitudinal life-course studies are required to inform evidence-based screening thresholds and preventive interventions.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Children and adolescents can have adverse cardiometabolic abnormalities, despite having normal Body Mass Index (BMI) (metabolically obese normal weight—MONW), but prevalence varies because of inconsistent definitions and limited synthesis of determinants.

WHAT THIS STUDY ADDS

⇒ This review provides the first integrated synthesis of MONW prevalence among children, demonstrating wide variation driven by divergent diagnostic criteria and measurement approaches. It further delineates key early-life, behavioural and socioeconomic determinants of MONW.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Findings support metabolic risk screening beyond BMI, incorporating body composition and cardiometabolic markers to detect high-risk MONW children, who could be missed by weight-based criteria alone.

⇒ This review highlights the need for harmonised paediatric MONW definitions and longitudinal research to guide early-life, socioeconomically.

INTRODUCTION

Childhood obesity, commonly identified using Body Mass Index (BMI)-for-age, is a major global public health challenge linked to adverse metabolic outcomes that often track into adulthood.[1] BMI, however, is a perfect surrogate of metabolic health. An emerging insidious concern is the metabolically obese normal weight (MONW) phenotype, characterised by dyslipidaemia, impaired fasting glucose, insulin resistance (IR) or elevated blood pressure (BP) in children whose BMI falls within the normal range.[2] Among children who do not meet conventional anthropometric cut-offs for overweight or obesity, cardiometabolic risk often may remain unrecognised for prolonged periods.[3] Evidence suggests that MONW is not a benign; affected children have substantially higher risk of future cardiometabolic disorders than metabolically healthy normal-weight peers, despite similar BMI profiles[2]. Over-reliance on BMI alone may delay identification of

high-risk children and limit opportunities for early, targeted prevention.

The MONW phenotype challenges traditional paediatric risk assessment and highlights the need to assess metabolic health using indices beyond body size. Clearer characterisation of its epidemiology, including prevalence, metabolic profiles and related body-composition patterns, is essential to inform targeted screening algorithms and prevention policies, particularly in low- and middle-income countries (LMICs), where rapid nutritional and lifestyle transitions, characterised by higher consumption of energy-dense ultra-processed foods, declining physical activity, increased sedentary behaviour and screen exposure, are reshaping childhood metabolic risk profiles[4] and contributing to a growing burden of non-communicable diseases (NCDs) in children. These shifts are accompanied by altered body composition, especially excess visceral adiposity, even among children with normal BMI[5] and can occur against a backdrop of the double burden of malnutrition (DBM), where undernutrition coexists with rising obesity and metabolic disease.[6] This systematic review will synthesise evidence on the prevalence and incidence, early-life determinants, and cardiometabolic risk of the MONW in children and adolescents to identify gaps and inform improved screening, prevention, and policy responses for this under-recognised yet clinically important phenotype.

METHODS

Protocol

Systematic review was conducted according to the guidelines of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020). The protocol for the review was registered with the International Prospective Register of Systematic Reviews (PROSPERO) (Registration No. CRD420261305686).

Databases and search strategy

The studies were systematically identified on MONW phenotype in children and adolescents across six databases: PubMed/ MEDical Literature Analysis and Retrieval System On-Line (MEDLINE), Scopus, Web of Science (Wos) (Core Collection), Google Scholar, Embase and Cochrane Library covering records from the year 2000 to January 2026, to ensure comprehensive capture of relevant literature.

The search strategy combined controlled vocabulary (where available) and free-text terms related to MONW and its equivalent phenotypes, the paediatric population, and associated metabolic abnormalities. Core MONW search concepts included: 'metabolically obese normal weight,' MONW, 'metabolically unhealthy normal weight,' MUNW, 'normal weight obesity,' NWO, 'metabolically abnormal normal weight,' 'Thin outside fat inside' TOFI and proxy definitions capturing metabolic dysfunction within normal-weight individuals defined by combinations of ('insulin resistance,' 'dyslipidaemia') AND ('normal weight' or 'normal BMI'). These were combined with paediatric population terms including 'child*,' 'adolescent*,' 'pediatric*,' 'paediatric*,' 'preschool,' and 'school-aged,' and metabolic outcome terms such as 'insulin resistance,' ' Homeostatic Model Assessment of Insulin Resistance (HOMA-IR),' 'hyperinsulinemia,' 'dyslipidaemia,' 'impaired fasting glucose,' 'hypertension,' 'blood pressure,' 'cardiometabolic risk,' and 'glucose intolerance.' Boolean operators, truncation, nesting and synonym expansion were applied to maximise sensitivity, while maintaining specificity. Full database-specific search strategies are provided in table 1.

A comprehensive literature search was conducted across electronic databases without applying restrictions on study design at the search stage to maximise sensitivity and capture all potentially relevant records. Final eligibility was determined during the screening process according to predefined inclusion and exclusion criteria.

All retrieved records were exported in Research Information Systems (RIS) or Comma Separated Values (CSV) formats and imported into a Microsoft Excel database for management and screening. Duplicate records were removed manually by comparing titles, authors, journal titles, publication years and digital object identifiers (DOIs); where uncertainty remained, abstracts were reviewed to confirm duplication prior to exclusion.

Inclusion and exclusion criteria

Studies were eligible for inclusion if they enrolled children and/or adolescents aged 3–18 years and evaluated the MONW phenotype, defined as normal BMI or BMI-for-age according to recognised standards such as World Health Organisation (WHO), Centers for Disease Control and Prevention (CDC) or International Obesity Task Force (IOTF) in the presence of one or more metabolic abnormalities. Given the evolving and heterogeneous definitions of MONW in paediatric populations, studies examining closely related phenotypes such as normal-weight obesity (NWO) defined by excess adiposity (eg, elevated body fat percentage (%BF)) among children with normal BMI—were also considered eligible, due to their conceptual overlap with MONW.

Eligible studies were required to report at least one outcome of interest, including (1) the prevalence or incidence of MONW, (2) early-life determinants (such as prenatal factors, birth weight, infant growth patterns, or early childhood exposures), or (3) cardiometabolic risk factors including IR, dyslipidaemia, impaired glucose metabolism or elevated BP. Observational study designs (cross-sectional, cohort, or case–control) conducted in any geographical setting and published in English were included.

Studies were excluded if they (1) were conducted exclusively in adults without separate reporting of paediatric data; (2) focused on children with chronic conditions known to affect growth or metabolism (type 1 diabetes, genetic or endocrine disorders); (3) defined obesity solely on the basis of BMI without assessment of

Table 1 Search strategy

Database		Search string
PubMed/MEDLINE	Jan-26	((‘metabolically obese normal weight’(Title/Abstract) OR MONW(Title/Abstract) OR ‘metabolically unhealthy normal weight’(Title/Abstract) OR MUNW(Title/Abstract) OR ‘normal weight obesity’(Title/Abstract) OR NWO(Title/Abstract) OR ‘metabolically abnormal normal weight’(Title/Abstract) OR ‘Thin Outside Fat Inside’(Title/Abstract) OR TOFI(Title/Abstract) OR ((‘insulin resistance’(Title/Abstract) OR dyslipidemia(Title/Abstract) OR ‘metabolic syndrome’(Title/Abstract)) AND (‘normal weight’(Title/Abstract) OR ‘normal BMI’(Title/Abstract)))) AND (child*(Title/Abstract) OR adolescent*(Title/Abstract) OR pediatric*(Title/Abstract) OR paediatric*(Title/Abstract) OR preschool*(Title/Abstract) OR ‘school aged’(Title/Abstract)))
Embase (Elsevier)	Jan-26	(‘metabolically obese normal weight’:ti,ab OR monw:ti,ab OR ‘metabolically unhealthy normal weight’:ti,ab OR munw:ti,ab OR ‘normal weight obesity’:ti,ab OR nwo:ti,ab OR ‘metabolically abnormal normal weight’:ti,ab OR ‘thin outside fat inside’:ti,ab OR ‘TOFI’:ti,ab OR ((‘insulin resistance’:ti,ab OR dyslipidemia:ti) AND (‘normal weight’:ti,ab OR ‘normal bmi’:ti,ab))) AND (child*:ti,ab OR adolescent*:ti,ab OR pediatric*:ti,ab OR paediatric*:ti,ab)
Web of Science (Core Collection)	Jan-26	TS= ((‘metabolically obese normal weight’ OR MONW OR ‘metabolically unhealthy normal weight’ OR MUNW OR ‘normal weight obesity’ OR NWO OR ‘metabolically abnormal normal weight’ OR ‘thin outside fat inside’ OR ‘TOFI’ OR ((‘insulin resistance’ OR ‘dyslipidemia’) AND (‘normal weight’ OR ‘normal BMI’))) AND (child* OR adolescent* OR pediatric* OR paediatric* OR preschool* OR ‘school aged’))
Scopus	Jan-26	TITLE-ABS-KEY ((‘metabolically obese normal weight’ OR MONW OR ‘metabolically unhealthy normal weight’ OR MUNW OR ‘normal weight obesity’ OR NWO OR ‘metabolically abnormal normal weight’ OR ‘thin outside fat inside’ OR TOFI OR ((‘insulin resistance’ OR ‘dyslipidemia’) AND (‘normal weight’ OR ‘normal BMI’))) AND (child* OR adolescent* OR pediatric* OR paediatric* OR preschool* OR ‘school aged’))
Cochrane Library	Jan-26	((‘metabolically obese normal weight’ OR MONW OR ‘metabolically unhealthy normal weight’ OR MUNW OR ‘normal weight obesity’ OR NWO OR ‘metabolically abnormal normal weight’ OR ‘thin outside fat inside’ OR ‘TOFI’ OR ((‘insulin resistance’ OR ‘dyslipidemia’) AND (‘normal weight’ OR ‘normal BMI’))) AND (child* OR adolescent* OR pediatric* OR paediatric*))
Google Scholar	Jan-26	‘Metabolically obese normal weight’ OR ‘metabolically unhealthy normal weight’ OR ‘normal weight obesity’ OR ‘thin outside fat inside’ OR ‘TOFI’ AND child OR adolescent

BMI, Body Mass Index; MEDLINE, MEDical Literature Analysis and Retrieval System On-Line; MONW, metabolically obese normal weight; MUNW, metabolically unhealthy normal weight; NWO, normal weight obesity; TOFI, thin outside fat inside.

metabolic health or adiposity; (4) evaluated metabolic risk without stratifying outcomes by normal-weight status; or (5) included only overweight or obese populations. Non-original research articles, conference abstracts without full-text availability, animal studies, duplicate publications, and studies for which the full texts could not be obtained despite reasonable efforts were also excluded.

Screening and study selection

Title and abstract screening were performed independently by two reviewers using the Excel database. Studies were marked as included, excluded or uncertain. Disagreements were resolved through discussion and consensus with the third reviewer. Full texts of potentially eligible studies were retrieved and assessed against predefined inclusion and exclusion criteria. Reasons for exclusion at the full-text stage were documented systematically.

A Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram was used to summarise the study selection process, including the number of records identified, screened, excluded and included in the final synthesis as represented in figure 1.

Data extraction and quality assessment

Data extraction

Data extracted are given below:

- Study characteristics (author, year, country, design, sample size, age range).
- Definitions of normal-weight and MONW.
- Metabolic outcomes assessed.
- Prevalence or incidence estimates with sexual dimorphism.
- Key findings relevant to cardiometabolic risk.
- Early-life exposures or determinants.

Risk of bias assessment

The methodological quality of included studies was independently assessed by two reviewers using the ROBINS-E (Risk Of Bias In Non-randomised Studies of Exposures) tool, developed by the Cochrane Bias Methods Group to evaluate the internal validity of non-randomised

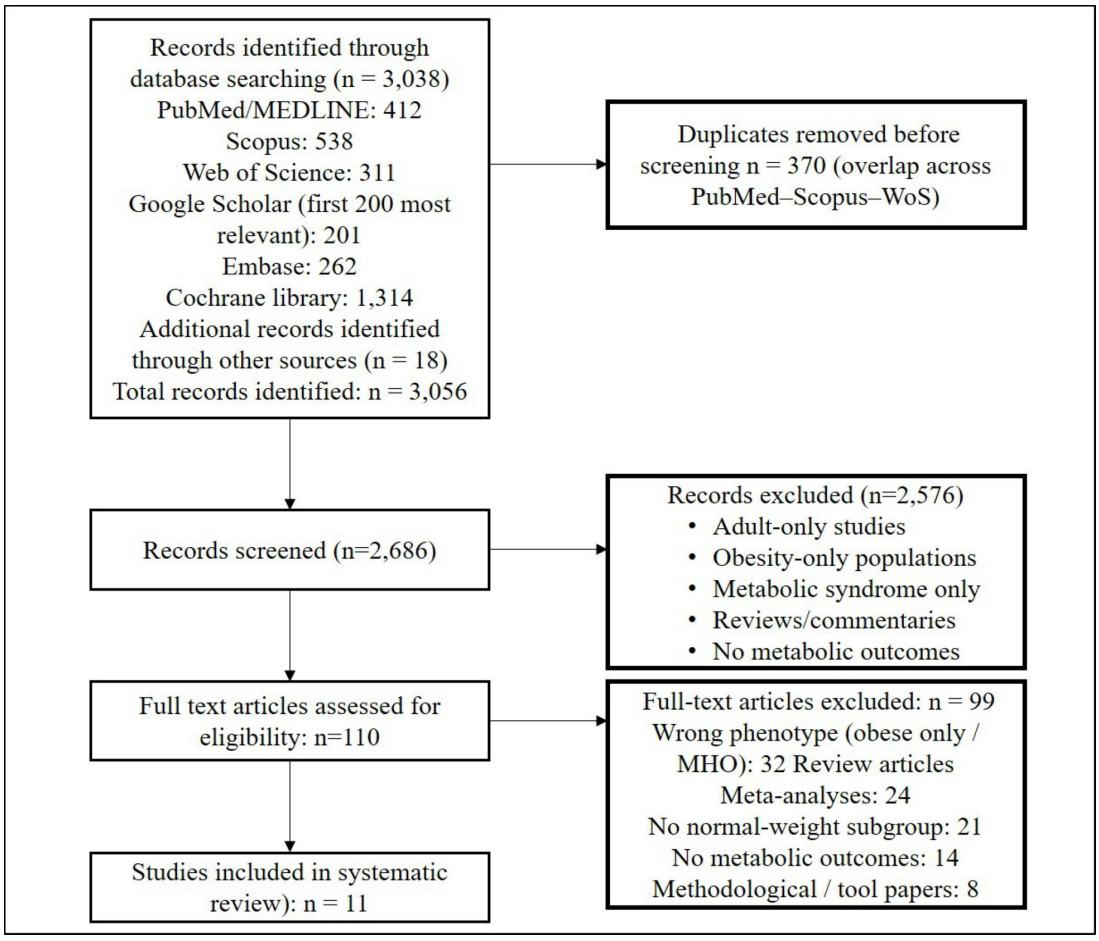

Figure 1 Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow. Flow diagram illustrating the identification, screening, eligibility assessment and inclusion of studies in the systematic review. Records were identified through database searching and other sources, duplicates were removed, and titles/abstracts were screened. Full-text articles were assessed for eligibility, with reasons for exclusion documented. The final number of studies included in qualitative and, where applicable, quantitative synthesis (meta-analysis) is shown. MEDLINE, MEDical Literature Analysis and Retrieval System On-Line; WoS, Web of Science; MHO, metabolically healthy obese.

exposure studies.[7] ROBINS-E assesses risk of bias across seven domains: (1) bias due to confounding; (2) bias arising from measurement of the exposure; (3) bias in selection of participants into the study; (4) bias due to post-exposure interventions; (5) bias due to missing data; (6) bias in measurement of the outcome; and (7) bias in selection of the reported result. Each domain was judged as low risk, some concerns, or high risk of bias using predefined signalling questions and domain-specific guidance. An overall risk-of-bias judgement was assigned for each study based on the highest level of bias observed across domains, in accordance with ROBINS-E guidance. Disagreements between reviewers were resolved through discussion and consensus.

Findings were synthesised and structured around:

- Prevalence and incidence of MONW.
- Early-life determinants.
- Treatment.

RESULTS

Study selection

The literature search identified 3038 records through electronic databases—PubMed/ MEDical Literature Analysis and Retrieval System On-Line (MEDLINE) (n=412), Scopus (n=538), Web of Science (Wos) (n=311), Google Scholar (first 200 most relevant records; n=201), Embase (n=262), and Cochrane Library (n=1314), plus additional 18 records from other sources, (total n=3056). After removal of 370 duplicates, 2686 unique titles and abstracts were screened, of which 2576 records were excluded according to predefined criteria (adult-only or obesity-only samples, metabolic syndrome-only studies, review articles/commentaries, or absence of relevant metabolic outcomes). Full texts of 110 articles were assessed; 99 were excluded due to incorrect phenotype classification (eg, obese-only or metabolically healthy obesity populations), review or meta-analyses, lack of a normal-weight subgroup, absence of relevant metabolic outcomes, or methodological/overview papers.

Finally, 11 studies met the inclusion criteria and were included in the final systematic review (figure 1).

Study characteristics

The characteristics of the included studies are summarised in online supplemental table 1. Three large

nationally representative cross-sectional surveys from China and India, including >35 000 children and adolescents aged 5–19 years,[6 8 9] were identified. Normal-weight was typically defined using WHO BMI-for-age criteria, and MONW was characterised as the presence of ≥1 cardiometabolic abnormality (lipids, glucose, Glycated Haemoglobin (HbA1c), insulin, or BP) among those with normal BMI, although BMI classification systems varied in one study (WHO, IOTF, Indian Academy of Pediatrics (IAP)).[6 8 9] Several additional cross-sectional studies from China, Mexico, Brazil and Germany[5 10–12] examined MONW or related phenotypes using heterogeneous definitions, ranging from ≥1 metabolic abnormalities to inclusion of dyslipidaemia, impaired glucose metabolism, HbA1c, IR, or elevated BP or %BF-based NWO, with some including non-traditional markers such as elevated serum uric acid and ultrasound-confirmed hepatic steatosis.[5 13] Sample sizes ranged from 45 to 19 000 participants.[5 10–12]

Four prospective cohort studies from Canada, Iran, China and Finland[3 13–15] followed children from early childhood or adolescence into later adolescence or young adulthood using broadly similar MONW definitions based on normal BMI and ≥1 metabolic abnormalities, although one study used Dual-Energy X-ray Absorptiometry (DXA)-measured body fat (≥30%) to define NWO.[3 13 15] Collectively, these studies highlight substantial heterogeneity in diagnostic criteria and study design.

The MONW phenotype was defined by using diverse criteria:

- Presence of ≥1 cardiometabolic abnormality—elevated fasting glucose or HbA1c, IR, dyslipidaemia and hypertension, despite having normal BMI.
- Clustering of metabolic risk factors.
- Excess adiposity or visceral fat within the normal BMI range.

Risk of bias

Risk of bias was assessed using ROBINS-E methodological domains[7] (D1–D7), and overall study-level judgements[16] are summarised in figure 2.

Domain-level findings

(D1) Bias due to confounding showed considerable variability: two studies (18.2%) were at high risk, three (27.3%) at low risk and six (54.5%) at some concerns, largely owing to incomplete adjustments for key sociodemographic and adiposity-related covariates.

(D2) Bias arising from measurement of the exposure was predominantly low risk (72.7%), with three studies (27.3%) rated as some concerns, mainly because of limited detail on exposure ascertainment.

(D3) Bias in selection of participants was low risk in six studies (54.5%) and some concerns in five (45.5%), reflecting unclear recruitment or restrictive eligibility criteria.

(D4) Bias due to post-exposure interventions was low (90.9%) aligning with observational design of included studies.

(D5) Bias due to missing data: eight studies (72.7%) were rated at some concerns and three (27.3%) at low risk, often due to incomplete reporting of attrition or unclear handling of missing data.

(D6) Bias in measurement of outcomes was generally robust, with nine studies (81.8%) at low risk and two (18.2%) at some concerns.

(D7) Bias in selection of the reported result showed heterogeneity: one study (9.1%) at high risk, and 10 at low or some concerns.

Overall, three studies (27.3%) were judged high risk, one (9.1%) low risk and seven (63.6%) as some concerns,[16] driven primarily by confounding and missing data (figure 3).

Prevalence and incidence of metabolically obese normal weight (MONW) in children and adolescents

Prevalence of MONW and metabolic abnormalities

Across population-based and cohort studies, a substantial proportion of normal-weight children exhibited cardiometabolic abnormalities consistent with MONW. In a large Chinese national survey of 12 346 participants aged 7–18 years, MONW, defined by ≥1 abnormalities in BP, triglyceride, High Density Lipoprotein (HDL) -cholesterol, or glucose value were 26.4%, with higher prevalence in boys.[9] A school-based Indian study reported MONW in 42.9% of normal-weight children and adolescents aged 6–19 years, with 73.7% of MONW cases occurring in lower-socioeconomic status (SES) public-school students with no sex difference.[8] Another Chinese study of 7926 children aged 6–17 years reported that 10.6% met criteria for metabolically unhealthy normal weight (MUNW), prevalence being higher in boys, and Dual-Energy X-ray Absorptiometry (DXA) assessment showed higher body fat, despite normal BMI.[10]

A large Indian national biomarker survey of 19 000 participants aged 5–19 years reported high metabolic abnormality prevalence, with 56% of normal-weight children having ≥1 abnormal lipid or glycaemic marker, indicating hidden metabolic risk independent of obesity.[6] Regional studies showed more moderate estimates, including 23% in Mexican children and adolescents aged 6–18 years based on combined lipid, glucose, BP and IR criteria[12]; 21% in an Iranian adolescent cohort defined by ≥2 cardiometabolic risk factors[15] and 26% in a Chinese ambidirectional cohort using lipid, glucose and uric-acid abnormalities[13]. Among Brazilian adolescents aged 10–19 years, 14% of adolescents with normal BMI had excess body fat as measured by DXA, consistent with the NWO phenotype.[11] Clinic-based studies further demonstrated metabolic dysfunction in lean children, with approximately 20% metabolically unhealthy German children aged 5–9 years[5] and 23% Canadian children with normal BMI but parental obesity risk exhibiting metabolic abnormality.[3]

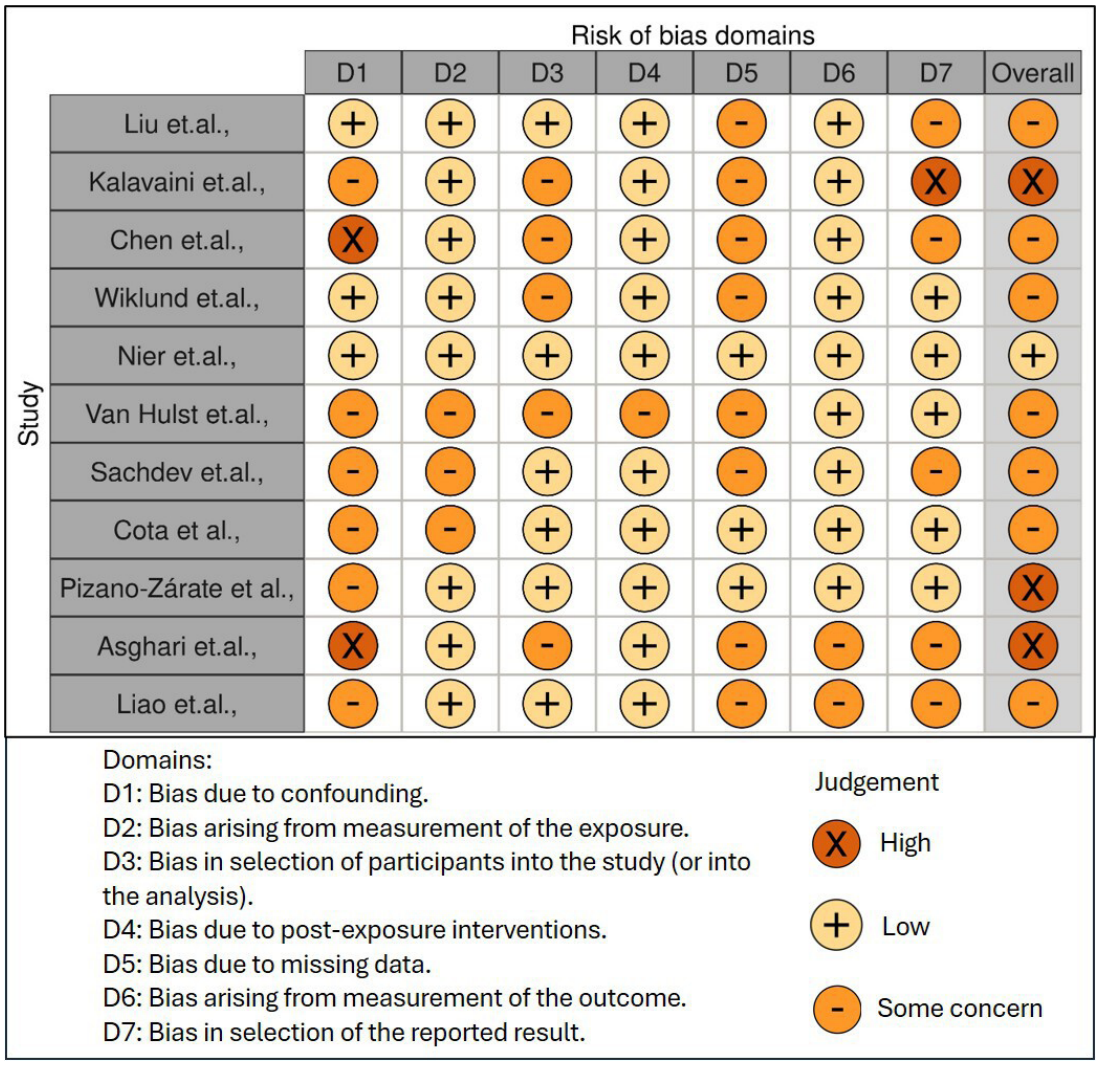

Figure 2 Assessment of risk of bias across seven methodological domains and overall study-level judgements by using ROBINS-E tool. This figure presents a ROBINS-E risk-of-bias traffic-light plot summarising the methodological quality of the included studies across seven bias domains (D1–D7). Each row represents an individual study, and each column corresponds to a specific bias domain: confounding (D1), exposure measurement (D2), participant selection (D3), post-exposure interventions (D4), missing data (D5), outcome measurement (D6) and selective reporting (D7). ROBINS-E, Risk Of Bias In Non-randomised Studies of Exposures.

Overall, reported MONW prevalence across studies ranged from 10.6% to 56.2%, varying by diagnostic criteria, adiposity measurement method, and population context. Table 2 presents the prevalence of MONW across the included studies.

Incidence and life-course emergence

Although most included studies were cross-sectional, limiting direct estimation of incidence, available longitudinal data provide important insight into the developmental dynamics of the MONW phenotype. Prospective cohort data indicate that MONW phenotype is not merely a static classification but can newly emerge over time. A Canadian longitudinal cohort demonstrated that approximately 23% of children with normal BMI developed cardiometabolic abnormalities within 2 years, while remaining within the normal weight range,[3] with increasing adiposity and greater screen time predicting this transition, suggesting that subtle changes in body fat accumulation and lifestyle behaviours may precede overt metabolic dysfunction.[3]

Evidence from Finland further highlights the persistence of this phenotype; excess body fat and adverse cardiometabolic profiles identified in childhood tracked into adolescence,[14] suggesting that early metabolic vulnerability in normal-weight children may become biologically embedded rather than resolving spontaneously with growth and maturation.[14] Similarly, prospective evidence from Iran showed that metabolically unhealthy normal-BMI adolescents were older and had higher hypertension prevalence than their metabolically healthy peers,[15] implying progression of cardiometabolic risk across adolescence and cumulative exposure to adverse metabolic influences.

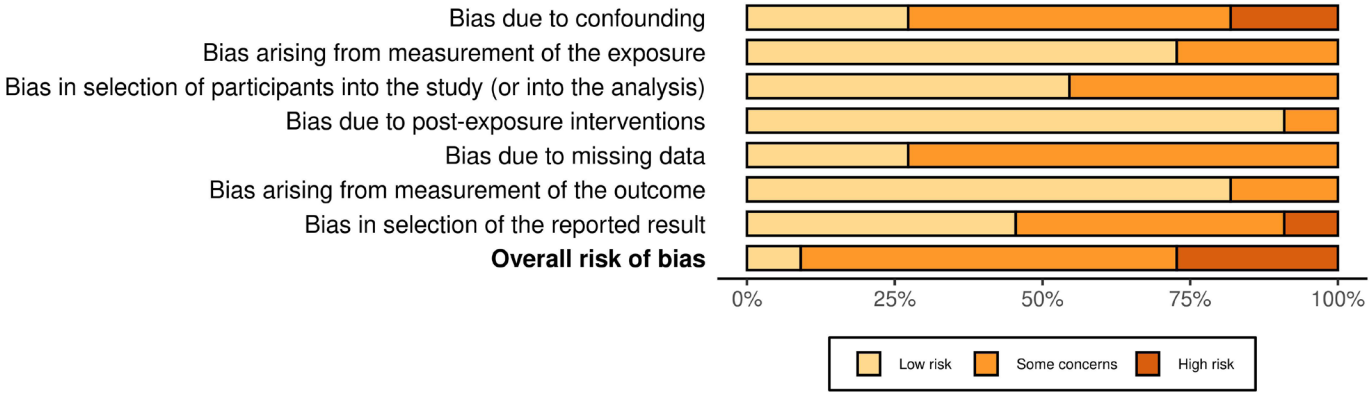

Figure 3 Evaluation of the risk of bias using the ROBINS-E tool. This figure summarises the risk of bias assessment across included studies using domain-level judgments. Most studies showed low risk of bias for measurement of exposure, outcome assessment and post-exposure interventions. Bias due to confounding and missing data was mainly rated as some concerns, with a small proportion at high risk. Selection of reported results also showed some concerns, with a few studies judged as high risk. Selection bias in participant inclusion was generally low to moderate. Overall, the majority of studies were rated as having some concerns, with only three studies classified as high risk of bias. ROBINS-E, Risk Of Bias In Non-randomised Studies of Exposures.

Collectively, these findings suggest that MONW can newly develop during childhood, persist across developmental stages and potentially worsen with age, supporting the concept of MONW as a dynamic, evolving phenotype rather than a transient metabolic state.

Determinants of MONW across the life course

While prevalence estimates confirm that MONW phenotypes are common across diverse populations, and longitudinal data demonstrate that this phenotype can emerge and persist over time,[10] the available evidence also highlights some determinants that may explain both its occurrence and progression.[3] Although most of the included studies were observational, consistent associations across cohorts implicate early-life, socioeconomic, behavioural, and biological factors as key contributors to the onset and maintenance of MONW. A detailed summary of socioeconomic and familial characteristics, body-composition assessment methods, and main outcomes of the included studies are presented in online supplemental table 2.

Early-Life and developmental determinants

The incidence findings described earlier suggest that MONW is developmentally dynamic rather than static. Evidence from a Chinese cohort showed that accelerated BMI-for-age gain between 6 and 24 months and maternal

Table 2 Prevalence of metabolically obese normal weight (MONW) in children and adolescents

Author	MONW prevalence	Prevalence with sexual dimorphism
Van Hulst *et al*[3]	23.0%	NA
Nier *et al*[5]	20.0%	Boys 16.7%; girls 23.8%
Sachdev *et al*[6]	56.2%	Similar overall; lipid higher in girls, glycaemia higher in boys
Kalavaini *et al*[8]	42.9%	Boys 41.65% (95% CI 38.31 to 45.04); girls 44.53% (95% CI 40.55 to 48.57). No significant difference
Liu *et al*[9]	26.4%	Boys 52.6%; girls 47.4%; girls lower odds (OR≈0.85)
Chen *et al*[10]	10.6%	Boys 11.7%; girls 9.5%
Cota *et al*[11]	13.8% (95% CI 10.8 to 17.0)	Boys 17.1 (OR 0.21 (95% CI 0.11 to 0.41); girls 82.9%
Pizano-Zárate *et al*[12]	22.9% (95% CI 16.9% to 29.8%)	Sex (male vs female): (22.3% girls vs 23.5% boys, p=0.86 (not statistically significant))
Liao *et al*[13]	25.9%	Sex (male vs female): OR 0.85; p=0.061 (not statistically significant)
Wiklund *et al*[14]	38.9%	Girls-only study
Asghari *et al*[15]	21.4%	Boys 18.9%; girls 23.4%

CI, Confidence Interval; MONW, metabolically obese normal weight; NA, not available or not reported in the original publication; OR, Odds Ratio.

obesity substantially increased the likelihood of unhealthy metabolic phenotypes in normal-weight children, highlighting infancy as a critical metabolic programming window.[13] This supports a life-course framework in which rapid early growth and maternal metabolic status predispose children to later metabolic vulnerability despite normal BMI. Consistent with this, Finnish longitudinal data demonstrated that persistent excess body fat from childhood onward was the key determinant of long-term cardiometabolic risk.[14] These findings indicate tracking of metabolic abnormalities across adolescence, whereby early adiposity patterns may establish a sustained trajectory of metabolic dysregulation.

Socioeconomic and family factors

The high prevalence observed in certain populations appears strongly shaped by social context.[9] Chinese national data showed that lower parental education, lower household income and parental overweight/obesity were independently associated with MONW.[9] This suggests intergenerational transmission of metabolic risk through both biological and environmental pathways.

Consistently, clustering of MONW in lower-SES Indian school populations reinforced the importance of structural socioeconomic determinants.[8] Brazilian adolescent data further linked lifestyle and socioeconomic context to hidden adiposity despite normal BMI.[11] These findings help explain why prevalence is often higher in low-income and middle-income settings and suggest that socioeconomic disadvantage may accelerate incidence through diet quality, physical inactivity, and limited health resources.

Lifestyle, diet, and behavioural exposures

Canadian longitudinal data identified higher screen time and increasing adiposity as predictors of MONW onset.[3] This provides direct prospective evidence that behavioural exposures contribute to incident cases.

Metabolic dysfunction in lean German children was associated with visceral adiposity, endotoxin exposure and monosaccharide-rich dietary intake.[5] An Indian study showed widespread dyslipidaemia and dysglycaemia in normal-weight adolescents further suggests dietary imbalance and metabolic transition as major contributors.[6] Together, these findings indicate that energy distribution, dietary composition and sedentary behaviour may promote metabolic dysregulation independent of BMI-defined obesity.

Metabolic risk clustering

Multiple cohorts confirmed clustering of hypertension, dyslipidaemia and impaired glucose metabolism within normal-BMI adolescents.[10 12 15] This reinforces that MONW is fundamentally defined by metabolic dysregulation rather than body size alone. The presence of cardiometabolic abnormalities in lean individuals supports the notion that body composition, fat distribution and IR are central pathophysiological determinants.[10 12 15]

Treatment

Currently, there is no treatment protocol specifically established for children and adolescents diagnosed with the MONW phenotype, largely because most interventions have focused on overweight/obese populations rather than normal-weight, but metabolically at-risk children.[17] Lifestyle modification remains the first-line strategy for metabolic risk reduction, emphasising increased physical activity, dietary changes and behavioural support, although direct evidence in MONW population is limited.[17 18] Meta-analyses of lifestyle programmes combining diet and physical activity in obese adolescents show significant reductions in body weight and improvements in cardiometabolic variables including lipids, BP and insulin sensitivity, particularly when behavioural and psychological support is integrated with dietary and exercise components.[19 20] School-based lifestyle interventions have demonstrated improved BP and High-Density Lipoprotein (HDL)-cholesterol in metabolically unhealthy obese children, and even transitions between unhealthy and healthier metabolic states, suggesting metabolic plasticity that might translate to MONW management.[21]

Family-centred multidisciplinary lifestyle programmes have been shown to foster modest long-term reductions in adiposity and physical fitness improvements, underscoring the importance of parental involvement in paediatric interventions.[17 22] Nutritional education can reduce abdominal obesity and may benefit cardiometabolic risk profiles, though evidence for dyslipidaemia, hypertension and glycaemia remains inconclusive.[17 23] More research is needed to test whether these lifestyle strategies are effective specifically in normal-weight, but metabolically at-risk children and adolescents, and to establish targeted interventions for the MONW phenotype.

DISCUSSION

Principal findings

This systematic review demonstrates that a substantial proportion of children and adolescents with normal BMI globally exhibit cardiometabolic abnormalities consistent with the MONW phenotype across diverse geographic and socioeconomic settings.[6 8 9] Reported prevalence varied widely, ranging from approximately 10% to more than 50%, reflecting large heterogeneity in diagnostic criteria, metabolic thresholds and adiposity assessment methods.[6 10 11] Despite this variability, all included studies consistently indicated that reliance on BMI alone fails to identify a clinically meaningful subgroup of metabolically at-risk children.[9 12]

Comparison with existing literature

The existence of metabolically unhealthy individuals with normal body weight was first described in adults several decades ago, characterised by excess body fat and IR despite normal BMI.[24 25] Subsequent paediatric research has demonstrated comparable metabolic heterogeneity during childhood and adolescence[10 14]. The prevalence

estimates observed in this review are broadly concordant with international data, which indicate that MUNW affects approximately 5%–30% of youth, depending on definition applied and population studied.[26 27] Higher prevalence observed in low-income and middle-income settings aligns with global evidence linking nutritional transition, dietary imbalance and socioeconomic disadvantage to early cardiometabolic risk.[6 8]

Pathophysiological interpretation

Findings across studies indicate that MONW is primarily a disorder of body composition and metabolic regulation, rather than body size.[11 14] Excess visceral or total adiposity within the normal BMI range appears central to cardiometabolic dysfunction, consistent with evidence linking visceral fat to IR, dyslipidaemia and inflammation.[10 28]

Dietary exposures and gut-derived inflammatory pathways may further contribute, as MUNW children show higher monosaccharide intake, endotoxin levels and visceral adiposity despite similar total energy intake.[5]

Behavioural factors such as screen time, sedentary lifestyle and rising adiposity during follow-up also predict MONW development in longitudinal cohorts.[3]

Developmental origins and early-life determinants

Evidence synthesised in this review supports a developmental origins framework in which maternal metabolic environment and early catch-up growth shape later cardiometabolic risk independent of BMI.[13 14] Accelerated weight gain in infancy and maternal obesity significantly increase the likelihood of unhealthy metabolic phenotypes among normal-weight children.[13] Such findings align with developmental-origins-of-health-and-disease theory linking intrauterine and early postnatal exposures to long-term metabolic programming.[29 30] Socioeconomic disadvantage and parental metabolic risk further amplify vulnerability, highlighting multilevel biological and social determinants of MONW.[8 9]

Life-course trajectory and clinical significance

Longitudinal evidence indicates that cardiometabolic abnormalities in normal-weight children can emerge during childhood and persist into adolescence or early adulthood.[3 14] Adolescents with MONW profiles demonstrate higher hypertension prevalence and worsening metabolic risk with age, suggesting progressive disease trajectories.[15] These observations are consistent with broader cohort evidence showing that childhood metabolic risk strongly predicts adult cardiometabolic disease independent of BMI.[31 32]

Implications for screening and prevention

Collectively, the evidence indicates that BMI-based screening alone is insufficient for identifying paediatric cardiometabolic risk.[10 11] Incorporating metabolic biomarkers, waist or body-fat assessment, and early-life risk profiling may improve detection of vulnerable children.[9 14] Preventive strategies should therefore target diet quality, physical activity, early growth patterns and maternal health, particularly in socioeconomically disadvantaged population.[6 8]

Strengths and limitations of the evidence base

This review synthesises evidence from diverse geographical regions, including national surveys and longitudinal cohorts, thereby enhancing the generalisability of findings across paediatric populations.[3 6 9 14] The global consistency of metabolically abnormal profiles among normal-weight children highlights the clinical relevance of the MONW,[2 24] and several studies incorporated validated laboratory biomarkers and advanced body composition techniques, including DXA, improving measurement precision.[5 10 11] Nonetheless, important limitations temper confidence in the evidence base. Substantial heterogeneity in MONW definitions particularly variation in metabolic cut-offs and adiposity thresholds limits cross-study comparability and reflects wider inconsistencies in defining metabolic phenotypes.[2 26] Considerable heterogeneity existed in the definition of MONW. One included study defined a related adiposity-based phenotype without mandatory metabolic abnormalities, which may limit comparability of prevalence estimates. Standardised paediatric definitions are urgently needed. The predominance of observational, often cross-sectional, designs limits causal inference and limits understanding of cardiometabolic trajectories into adulthood,[14 15 31 32] while residual confounding remains likely, given inconsistent adjustment for socioeconomic patterns, lifestyle behaviours and early-life determinants.[4 8 13 29 30] Variation in body composition assessment methods further complicates mechanistic interpretation and may contribute to phenotype misclassification.[10 11] Although emerging interventional studies in paediatric metabolic risk indicate modifiable pathways,[17–20] evidence specifically targeting MONW is sparse. Overall, the certainty of evidence is best characterised as moderate and findings should be interpreted cautiously pending harmonised MONW definitions and rigorously designed prospective studies.

CONCLUSIONS

This systematic review indicates that children with MONW represent a clinically important, yet, under-recognised phenotype, characterised by adverse metabolic risk despite normal BMI. Cardiometabolic abnormalities including IR, dyslipidaemia, impaired glucose regulation and excess visceral adiposity can occur independently of generalised obesity. Longitudinal evidence suggests that metabolic perturbations originating in childhood can track into adolescence and adulthood, increasing the risk of metabolic syndrome, subclinical atherosclerosis, type 2 diabetes and cardiovascular disease. The findings support a multifactorial aetiology wherein early-life influences (foetal programming, birth weight, rapid infant growth, maternal metabolic status and environmental exposures), sedentary behaviours, unhealthy dietary

patterns and excess refined sugar intake promote visceral adiposity and metabolic dysfunction. Socioeconomic and ethnic disparities may further modulate cardiometabolic risk profiles, through differences in adiposity distribution and visceral fat deposition. Collectively, the evidence challenges BMI-centric paediatric risk stratification, underscoring the need for broader screening frameworks.

Preventive strategies should prioritise optimisation of metabolic health through sustained physical activity, improved diet quality and behavioural modification, rather than weight reduction alone. However, substantial heterogeneity in MONW definitions, metabolic thresholds and phenotypic classifications, alongside predominately cross-sectional designs, limits comparability and causal inference. Harmonised paediatric MONW definitions, age-specific and sex-specific cut-offs, and well-designed longitudinal studies are urgently needed.

Contributors The conceptual framework was developed by RK and MF. RK is the guarantor. The review concept and design were developed by MF and RK. MF conducted the systematic literature search, study screening and data extraction, with methodological oversight from RK and SS. MF and SS assessed the risk of bias of the included studies. MF performed the data synthesis and drafted the initial manuscript. Subsequent revisions were undertaken by MF, RK and SS with RK and SS providing substantial critical input and overall supervision. All authors reviewed and approved the final manuscript.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests No, there are no competing interests.

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Patient consent for publication Not applicable.

Ethics approval Not applicable.

Provenance and peer review Not commissioned; internally peer reviewed.

Data availability statement Data sharing not applicable as no datasets generated and/or analysed for this study.

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