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SYSTEMATIC REVIEW AND META-ANALYSIS

Fructose Intake and Dietary Habits in Children with Overweight or Obesity, With and Without Steatotic Liver Disease: A Systematic Review and Meta-Analysis

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ABSTRACT

Background: Fructose and unhealthy eating are widely considered drivers of metabolic dysfunction-associated steatotic liver disease (MASLD) in children, but obesity confounds most comparisons.

Objective: To compare dietary intake between children with overweight or obesity with and without MASLD.

Methods: We searched PubMed, Europe PMC, OpenAlex and Scopus for observational studies reporting diet by liver status within overweight or obesity. Standardized mean differences (SMDs; Hedges' g) were pooled using random-effects models with Hartung-Knapp intervals. Certainty was rated with GRADE.

Result: Sixteen studies (1,867 children) from 13 countries were included; 15 gave diet by liver status (758 with and 960 without MASLD). Fructose intake did not differ significantly (SMD 0.20, 95% CI −0.17 to 0.56; k = 7; I^2 = 57.4%; prediction interval −0.57 to 0.97). In an exploratory subgroup comparison, higher intake appeared only in two studies at high risk of bias; the other five showed no difference (SMD −0.01, 95% CI −0.19 to 0.18; I^2 = 0.0%). Energy, carbohydrate, fat and protein were similar (all SMDs 0.06 to 0.10). Sugar-sweetened beverages (SMD 0.27, 95% CI −0.22 to 0.77) and fast food did not differ. Lower reported intake of sweets was not robust. Certainty was low or very low.

Conclusion: Within excess weight, energy and macronutrient intakes were similar, and reported fructose intake did not clearly differ, particularly in studies at low or moderate risk of bias. Reported diet should not be used to identify MASLD. Limiting free sugars remains appropriate on experimental grounds.

1. Introduction

The multisociety nomenclature replaced non-alcoholic fatty liver disease (NAFLD) with metabolic dysfunction-associated steatotic liver disease (MASLD).[1] MASLD is a common chronic liver disease of childhood. Global Burden of Disease analyses and body mass index-based modeling show a rising adolescent burden worldwide, with high body mass index as the main correlate.[2–5] Among children who already have excess weight, the condition is frequent: ultrasound or elastography surveys found it in 44% of overweight and obese Indian children and in up to 75% of children with obesity by the final primary school grade in another population, and high figures were also reported from African ambulatory settings.[6–8] Diagnosis requires hepatic steatosis plus at least one cardiometabolic criterion. Virtually every child with obesity meets it. In this group, then, the labels MASLD and NAFLD describe largely the same children.[1,9] Long-term data show that pediatric MASLD is not benign. In a cohort of 1,096 children the cumulative incidence of cirrhosis was 4.7%, with premature mortality and a heavy burden of extrahepatic disease.[10] In pediatric obesity, MASLD carried a hazard ratio of 2.71 (95% CI 2.14 to 3.43) for youth-onset type 2 diabetes,[11] a risk that also tracked with liver chemistry trajectories during follow-up.[12] Over two years of follow-up, children with MASLD were at higher risk of raised carotid intima-media thickness and left ventricular hypertrophy,[13] whereas normalization of MASLD was associated with lower carotid intima-media thickness and left ventricular mass development than persistent disease.[14] Standard care helps only some children: 31% improved over two years and about one-fifth worsened.[15] Treatment rests mainly on lifestyle change, so identifying a modifiable dietary driver in this group is important.

Diet is regarded as the principal modifiable target. Global intake of sugar-sweetened beverages among people aged 3 to 19 years rose by 23% over 28 years.[16] Fructose has drawn particular attention. The liver metabolizes most of it, and it supplies substrate for de novo lipogenesis. In adolescent boys with NAFLD, eight weeks of free-sugar restriction lowered hepatic de novo lipogenesis from 34.6% to 24.1% and hepatic fat from 25.5% to 17.9%.[17] Other trials in youth were less encouraging. A dietitian-led sugar reduction program did not improve liver outcomes in Latino youth with obesity.[18] Observational data linked sugary drinks in infancy[19] and a Western dietary pattern[20] to liver fat, and fiber intake in adolescence was associated with lower odds of steatotic liver disease.[21] These comparisons, however, cut across weight categories and cannot separate diet from adiposity. A cohort that followed free sugar intake from age three into adult life found that a weak association with severe steatosis

disappeared after adjustment for later body mass index.[22] Whether unhealthy habits such as fast food, sweet snacks and skipped breakfast add anything beyond energy excess is equally unclear.

The central gap is the separation of diet from adiposity. Obesity is the strongest determinant of MASLD and also a consequence of diet. Comparing children with MASLD against lean controls therefore mostly shows that heavier children eat differently. The useful contrast lies within excess weight. Why does one child with obesity develop steatotic liver disease while another does not, and does diet explain the difference? Single studies have compared these groups, but they were small and inconsistent: some reported clearly higher fructose intake with MASLD,[23,24] others no difference.[25,26] Recent syntheses addressed dietary treatment in pediatric MASLD[27] or ultra-processed food across ages,[28] not what affected children with excess weight actually eat. The question also matters locally. Overweight is rising among Indonesian children, and ultra-processed food has already been linked to poorer body composition in Indonesian primary school pupils.[29,30]

Eating behavior is thought to matter as much as nutrients. Sugar-sweetened beverages, fast food, fried food, sweet snacks and skipped breakfast tend to cluster, and later meal timing and higher energy intake at dinner were recently associated with MASLD in adolescents.[31] Such habits are also more practical to address in a clinic than a gram count of fructose. Dietary advice for this group is often built around them. Yet no one had summarized whether they separate children with MASLD from equally heavy peers. Experimental and cohort evidence alone cannot settle the matter. Trials test what happens when intake changes, but clinicians also need to know whether the diet of an affected child actually differs from that of an unaffected one.

This review restricts the comparison to children with overweight or obesity, which reduces confounding by adiposity by design but does not remove it, and pools fructose alongside sugars, macronutrients and specific eating habits on one standardized scale.

The aim was to determine whether dietary fructose intake and unhealthy dietary habits differed between children with overweight or obesity who had MASLD and those who did not.

2. Methods

2.1. Study Design

This systematic review and meta-analysis was reported according to the PRISMA 2020 statement.[32] The review question, eligibility criteria, outcomes and analysis plan were defined before the searches were run.

2.2. Search Strategy

PubMed/MEDLINE, Europe PMC, OpenAlex and Scopus were searched without language limits. The strategy combined three concept blocks: steatotic liver disease ("Non-alcoholic Fatty Liver Disease"[Mesh] OR "fatty liver" OR NAFLD OR MASLD OR MAFLD OR "steatotic liver" OR "hepatic steatosis" OR "liver fat"), children and adolescents ("Child"[Mesh] OR "Adolescent"[Mesh] OR "Pediatric Obesity"[Mesh] OR child *OR adolescen* OR pediatric *OR pediatric* OR youth *OR teen*) and diet ("Fructose"[Mesh] OR "Dietary Sugars"[Mesh] OR "Sugar-Sweetened Beverages"[Mesh] OR "Diet"[Mesh] OR "Feeding Behavior"[Mesh] OR "Fast Foods"[Mesh] OR "Energy Intake"[Mesh] OR fructose OR sugar *OR soda OR diet* OR "eating habit*" OR "fast food*" OR "food frequency"). No obesity terms were used, so that reports describing a clinic population without naming obesity were captured. Title, abstract and keyword fields were searched. The complete strings for each database are given in Supplementary Table S1. Reference lists of ten core reports and the articles citing them were also checked.

2.3. Eligibility Criteria

Eligibility followed the population, exposure, comparator, outcome and design framework detailed in Table 1. The defining rule was that both groups had overweight or obesity. Lean comparison groups were not used, even where a report also included one. MASLD, MAFLD and NAFLD labels were retained as reported, because the eligibility rule on excess weight meant that virtually all participants also met a cardiometabolic criterion for MASLD.[1]

Table 1. Eligibility Criteria.

Element	Inclusion	Exclusion
Population	Children and adolescents (mean age under 19 years) with overweight or obesity by age- and sex-specific criteria, from clinical, school or population settings	Mixed-weight samples without a separate overweight or obese stratum; steatosis from another cause (genetic, viral or drug-induced disease, intestinal failure, parenteral nutrition)
Exposure	Dietary fructose (total, free or added; absolute or energy-adjusted); total sugars; energy, macronutrients and fiber; sugar-sweetened beverages, fast food, sweets, fruit, vegetables and diet-quality indices, assessed by food record, dietary recall, food frequency or structured questionnaire	Maternal or infant diet alone; plasma or urinary biomarkers without dietary assessment; feeding challenges
Comparator	Children with overweight or obesity without MASLD from the same source population	Lean or normal-weight controls as the only comparator
Outcome	MASLD, or NAFLD or MAFLD under earlier definitions, diagnosed by histology, magnetic resonance imaging or spectroscopy, ultrasound or elastography	Liver disease inferred from aminotransferases or surrogate indices alone; hepatic fat analysed only as a continuous measure
Design	Cross-sectional, case-control and cohort studies in peer-reviewed journals, reporting dietary data by liver status	Reviews, case reports, conference abstracts, animal and laboratory studies, intervention effects; overlapping reports (the largest report per cohort and measure was retained)

Note: MASLD, metabolic dysfunction-associated steatotic liver disease; MAFLD, metabolic dysfunction-associated fatty liver disease; NAFLD, non-alcoholic fatty liver disease.

2.4. Study Selection and Data Extraction

Records were merged and de-duplicated on PubMed identifier, digital object identifier and normalized title. Rule-based triage removed records that could be identified as ineligible from metadata: reviews and other non-primary publications, conference abstracts, animal and laboratory studies, adult-only reports, other liver diseases, and supplement, drug or surgical treatments. The remaining titles and abstracts were screened against the criteria. One

reviewer screened the records and extracted the data. A second reviewer checked the inclusion decisions and every extracted value, and disagreements were resolved by discussion. For each study we extracted country, design, setting, age, sex, diagnostic method, dietary instrument, group sizes and every dietary measure by liver status. Where a study reported severity groups, they were combined into one group with the standard formula. Overlapping reports of a cohort were linked, and the largest report was used for each measure. When a study reported intake by more than one instrument, the instrument covering the most days was preferred. For fructose, total fructose (free fructose plus half of sucrose) was preferred to any single source. Measures were grouped in advance into sugars (fructose and total sugars), energy and macronutrients (carbohydrate, fat, saturated fat, protein and fiber) and foods and habits (sugar-sweetened beverages, fast or convenience food, sweets, fruit, vegetables and diet-quality indices). Reports were eligible when group-level dietary data could be extracted. Two Iranian cohorts from one university were treated as independent because they were recruited in different periods and different hospitals.[33,34] Unhealthy dietary habits were defined in advance as sugar-sweetened beverages, fast or convenience food, fried food, sweets and confectionery, and skipped breakfast. Each was pooled when at least two studies reported it, and otherwise described.

2.5. Risk of Bias

Risk of bias was assessed with a 10-point scale adapted from the nine-star Newcastle-Ottawa Scale for cross-sectional studies (NOS-xs),[35] covering selection (representativeness, sample size, non-respondents, dietary instrument), comparability (control of age, sex and adiposity) and outcome (liver assessment, statistical reporting). Blinded ultrasound, magnetic resonance and histology earned full points for liver assessment. Scores of 8 or more indicated low risk, 6 to 7 moderate risk and 5 or less high risk. Credit for blinded ultrasound reflected the fact that blinding addresses the main source of bias in a subjective reading, while its limited sensitivity was examined separately in the subgroup by diagnostic method.

2.6. Statistical Analysis

The primary outcome was the difference in dietary fructose intake between children with and without MASLD. Studies expressed fructose as grams per day, grams per week, energy-adjusted grams or grams per 1000 kcal, and energy and macronutrients as grams, as percentages of energy or as percentages of dietary reference values. Because units differed, effects were expressed as standardized mean differences (SMDs; Hedges' g), as defined in Equation 1;[36] positive values indicated higher intake with MASLD. Benchmarks of 0.2, 0.5 and 0.8 were used to describe small, moderate and large differences.

$$\begin{aligned} g &= J \times \frac{\bar{x}_{\mathrm{M}} - \bar{x}_{\mathrm{W}}}{S_{\mathrm{p}}}, \\ S_{\mathrm{p}} &= \sqrt{\frac{(n_{\mathrm{M}}-1)s_{\mathrm{M}}^2 + (n_{\mathrm{W}}-1)s_{\mathrm{W}}^2}{n_{\mathrm{M}}+n_{\mathrm{W}}-2}}, \\ J &= 1 - \frac{3}{4(n_{\mathrm{M}}+n_{\mathrm{W}})-9}, \\ v_g &= \frac{n_{\mathrm{M}}+n_{\mathrm{W}}}{n_{\mathrm{M}}n_{\mathrm{W}}} + \frac{g^2}{2(n_{\mathrm{M}}+n_{\mathrm{W}})} \end{aligned} \tag{1}$$

Standard errors and 95% confidence intervals (CIs) were converted to standard deviations with Equation 2, and medians with quartiles were converted to means and standard deviations with the Luo and Wan estimators in Equation 3.[37–40]

$$\begin{aligned} s &= \mathrm{SE} \times \sqrt{n}, \\ s &= \sqrt{n} \times \frac{U-L}{2t_{n-1,0.975}} \end{aligned} \tag{2}$$

$$\begin{aligned} \bar{x} &\approx w\frac{q_1+q_3}{2} + (1-w)m, \\ w &= 0.7 + \frac{0.39}{n}, \\ s &\approx \frac{q_3-q_1}{2\Phi^{-1}\left(\frac{0.75n-0.125}{n+0.25}\right)} \end{aligned} \tag{3}$$

Severity groups and weight strata were combined with Equation 4, and binary habits were placed on the same scale with Equation 5.

$$\begin{aligned} \bar{x} &= \frac{n_1\bar{x}_1 + n_2\bar{x}_2}{n_1+n_2}, \\ s^2 &= \frac{(n_1-1)s_1^2 + (n_2-1)s_2^2 + \frac{n_1n_2}{n_1+n_2}(\bar{x}_1-\bar{x}_2)^2}{n_1+n_2-1} \end{aligned} \tag{4}$$

$$\begin{aligned} g &= \ln(\mathrm{OR}) \times \frac{\sqrt{3}}{\pi}, \\ v_g &= \frac{3}{\pi^2}\left(\frac{1}{a}+\frac{1}{b}+\frac{1}{c}+\frac{1}{d}\right) \end{aligned} \tag{5}$$

Equation 5 assumes an underlying logistic distribution, so the habit analyses were repeated with continuous measures alone.

Effects were pooled with the inverse-variance random-effects model, using the DerSimonian-Laird between-study variance (Equation 6)[41] and the pooled estimate in Equation 7, with Hartung-Knapp confidence intervals (Equation 8)[42] when at least three studies contributed.[43,44]

$$\begin{aligned} \tau^2 &= \max\left\{0, \frac{Q-(k-1)}{\sum_i w_i - \frac{\sum_i w_i^2}{\sum_i w_i}}\right\}, \\ w_i &= \frac{1}{v_i} \end{aligned} \tag{6}$$

$$\begin{aligned} \hat{\mu} &= \frac{\sum_i w_i^* y_i}{\sum_i w_i^*}, \\ w_i^* &= \frac{1}{v_i+\tau^2} \end{aligned} \tag{7}$$

$$\hat{\mu} \pm t_{k-1,0.975}\sqrt{\frac{\sum_i w_i^*(y_i-\hat{\mu})^2}{(k-1)\sum_i w_i^*}} \tag{8}$$

Because the unmodified Hartung-Knapp interval could be narrower than the Wald interval when few homogeneous studies contribute,[43] Wald intervals were reported alongside for every measure, and a result was described as robust only when both intervals excluded no difference.[44]

Heterogeneity was described with Cochran's Q, I^2 and τ^2 (Equation 9),[45] and the 95% prediction interval was calculated with Equation 10.[46,47] It was calculated when at least three studies contributed, although prediction intervals are unreliable when few studies are pooled. The prediction interval estimated the range in which the true effect of a new comparable study would be expected to fall, which was the more relevant summary when heterogeneity was substantial.

$$\begin{aligned} Q &= \textstyle\sum_i w_i(y_i - \hat{\mu}_{\mathrm{FE}})^2, \\ I^2 &= \max\left\{0, \frac{Q-(k-1)}{Q}\right\} \times 100\% \end{aligned} \tag{9}$$

$$\hat{\mu} \pm t_{k-2,0.975}\sqrt{\tau^2 + \mathrm{SE}^2(\hat{\mu})} \tag{10}$$

Prespecified subgroups for the primary outcome were region, diagnostic method, age, dietary instrument, fructose metric and risk of bias. Between-subgroup tests used Wald standard errors, because Hartung-Knapp standard errors can be unstable in small homogeneous subgroups. These six comparisons were treated as hypothesis-generating, with no adjustment for multiplicity. Sensitivity analyses comprised alternative estimators, leave-one-out analysis and exclusions by data form and study quality. Funnel plots were inspected.

Egger's test was planned only for measures with at least 10 studies, and no measure reached that number.[48,49] Trim-and-fill was regarded as exploratory.

When group means were not consistent with the whole-sample mean and P value of a study, the effect was derived from the P value and the whole-sample SD, with the direction taken from the crude odds ratio for the highest tertile of intake. This rule applied to one study,[33] and both its exclusion and the use of its group means as reported were examined as sensitivity analyses.

Because several unhealthy-food measures came from the same children, a three-level model with random components for study and effect size summarized them jointly.[50] Analyses were run in R 4.5.3 with the meta (8.2.1) and metafor (4.8.0) packages.[51,52]

2.7. Certainty of the Evidence

Certainty was rated for each dietary measure with GRADE.[53] Because the question concerned an association and not a treatment effect, the rating started at high certainty, as in frameworks for prognostic factors,[54] and was then downgraded. One level was subtracted for risk of bias for every measure, because intake was self-reported and comparisons were largely unadjusted. For inconsistency, one level was subtracted when I^2 was 50% to 74% and two when it was 75% or more. One level was subtracted for imprecision when the confidence interval included no difference or fewer than 400 children contributed. Upgrading was not applied.

3. Results

3.1. Study Selection

The searches identified 5,644 records, of which 2,488 were duplicates and 1,486 were removed by rule-based triage. Of the 1,670 records screened, 57 reports were sought and 49 were assessed. Sixteen studies, described in 24 reports, met the criteria, as illustrated in Figure 1. Companion reports came from two Iranian cohorts[55–59] and one Greek centre,[60,61] and they were used only to supplement the main reports. The commonest reasons for exclusion at full text were the absence of dietary data by liver status and a comparator group not restricted to excess weight. Reports excluded at full text, with reasons, and the reports not retrieved are listed in Supplementary Tables S2 and S3.

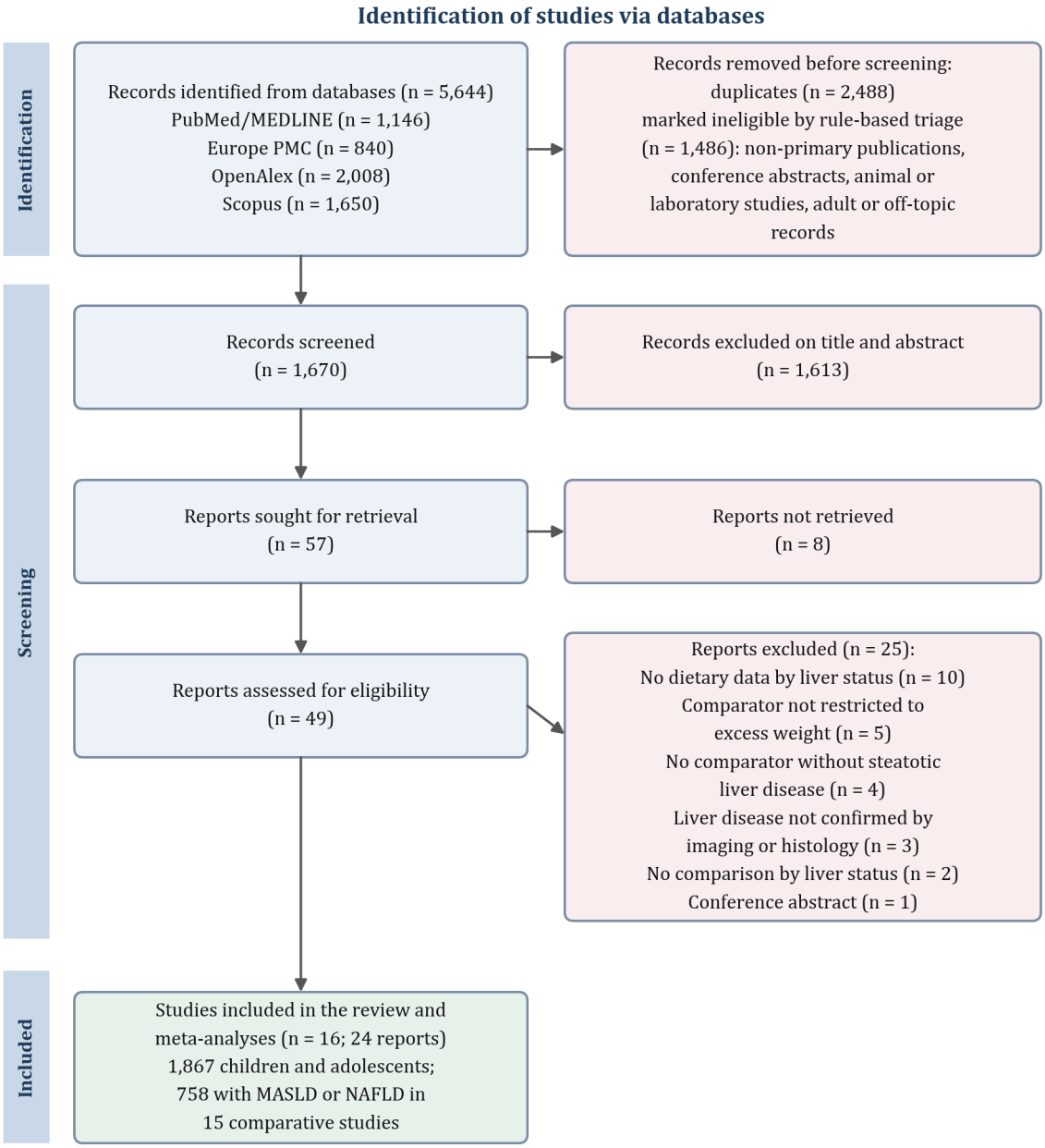


Figure 1. PRISMA 2020 flow diagram of the identification, screening and inclusion of studies.

3.2. Study Characteristics

The studies enrolled 1,867 children and adolescents in 13 countries, as detailed in Table 2. Fifteen gave dietary data by liver status (758 with and 960 without MASLD), and one Korean study reported odds ratios only.[62] Eleven studies used ultrasound, two histology and two magnetic resonance, and one did not state the method. Fourteen were cross-sectional or case-control comparisons from clinics or schools. One comprised the overweight and obese strata of a population-based birth cohort, with diet recorded at 14 years and liver ultrasound at 17 years, and one was a repeated-measures analysis of an intervention cohort. Mean age, where reported as a mean, ranged from 7.6 to 15.7 years. Eight studies used food records or recalls, four food frequency questionnaires and four structured questionnaires or the KIDMED index. Sample sizes ranged from 22 to 505 children. Fructose was reported by seven studies, while four others gave only foods, habits or diet-quality indices. Group sizes were markedly unbalanced in several studies, and the two Iranian cohorts contributed 66% of the children in the energy analysis. Thirteen reports used NAFLD or an equivalent term, one MAFLD and two MASLD.

Although every child had excess weight, adiposity was not identical between groups. Body mass index was significantly higher with liver disease in at least three reports,[23,33,63] whereas it was lower with NAFLD in the UK case-control study,[26] and similar in the Hispanic case-control study.[64] Diet was recorded three years before liver imaging in the Australian cohort;[65] in the other reports diet and liver status were assessed in the same period, and few stated whether families had already received dietary advice.

Table 2. Characteristics of the Included Studies.

Study	Country	Design	MASLD (n)	No MASLD (n)	Age (years)	Liver assessment	Dietary instrument	Measures	NOS (risk)
Hattar 2011 [64]	United States	Case-control	20	20	12.2 vs 11.9	Liver biopsy (controls: no liver disease)	School Physical Activity and Nutrition survey	Fr	5 (High)
Papandreou 2012 [63]	Greece	Cross-sectional	38	47	11.5 vs 11.0	Ultrasound	3-day food record	Sug, En, CHO, Fat, SFA, Pro, Fib	5 (High)
Mollard 2014 [25]	Canada	Cross-sectional	29	39	15.7 vs 15.2	Magnetic resonance spectroscopy (≥5.5%)	Harvard Youth/Adolescent FFQ	Fru, Sug, En, CHO, Fat, SFA, Pro, Fib, SSB, FF	8 (Low)
O'Sullivan 2014 [65]	Australia	Prospective cohort	42	87	14 at diet, 17 at ultrasound	Ultrasound	3-day food record	Fru, Sug	7 (Moderate)
Gibson 2015 [26]	United Kingdom	Case-control	24	8	13.5 vs 12.0 (median)	Liver biopsy (controls: ultrasound and liver tests)	7-day food diary	Fru, Sug, En, CHO, Fat, SFA, Pro, Fib	7 (Moderate)
Cakir 2016 [66]	Turkey	Case-control	106	21	12.4 vs 11.3	Not stated	KIDMED index	KIDMED	3 (High)
Félix 2016 [67]	Brazil	Cross-sectional	8	31	10.6 vs 8.3	Ultrasound	Two 24-h recalls and habit questionnaire	En, Sw, Fr, Veg	6 (Moderate)
Pawar 2016 [68]	India	Cross-sectional	62	38	13.5	Ultrasound, transaminases and elastography	Weekly food-habit questionnaire	SSB, FF, Sw	6 (Moderate)
Troisi 2017 [69]	Italy	Case-control	12	10	9.8 (whole sample)	Ultrasound and hypertransaminasemia	KIDMED index	KIDMED	4 (High)
Nier 2018 [23]	Germany	Cross-sectional	16	73	7.8 vs 7.6	Ultrasound	Two 24-h recalls	Fru, En, CHO, Fat, Pro, Fib, SSB, FF, Sw, Fr, Veg	5 (High)
Flores-Ramírez 2019 [70]	Mexico	Cross-sectional	67	16	9.2 (whole sample)	Ultrasound	Two 24-h recalls	Fru, En, CHO, Fat, Pro, SSB	6 (Moderate)
León-Plascencia 2021 [71]	Mexico	Cross-sectional	13	20	6 to 16 (range)	Ultrasound	Two 24-h recalls	Sug, En, CHO, Fat, SFA, Pro	4 (High)
Akhgarjand 2024 [33]	Iran	Cross-sectional	113	212	9.4 vs 9.0	Ultrasound	147-item FFQ	Fru, En, CHO, Fat, SFA, Pro, Fib, Fr, Veg	7 (Moderate)
Lee 2024 [62]	South Korea	Prospective analysis of an intervention cohort	NR	NR	12.2 (median, whole sample)	Magnetic resonance imaging (proton density fat fraction)	2-day or 3-day food diary	UPF	7 (Moderate)
Faienza 2025 [24]	Italy	Cross-sectional	12	29	8.6 vs 10.2	Ultrasound	IDEFICS FFQ, 24-h recall, fructose questionnaire	Fru	4 (High)
Kamrani 2026 [34]	Iran	Cross-sectional	196	309	10.5 vs 9.7	Ultrasound with MAFLD criteria	147-item FFQ	En, CHO, Fat, Pro	9 (Low)

Note: Age in years, MASLD versus no MASLD where reported. FFQ, food frequency questionnaire; NOS, adapted Newcastle-Ottawa Scale score out of 10 (risk of bias). Fru, fructose; Sug, total sugars; En, energy; CHO, carbohydrate; SFA, saturated fat; Pro, protein; Fib, fiber; SSB, sugar-sweetened beverages; FF, fast

or convenience food; Sw, sweets; Fr, fruit; Veg, vegetables; KIDMED, Mediterranean Diet Quality Index for children and adolescents; UPF, ultra-processed food; NR, not reported; the 149 children not allocated to groups in the table (1,867 – 758 – 960) belong to Lee 2024.

3.3. Risk of Bias

Risk of bias was low in two studies, moderate in seven and high in seven, with a median score of 6 of 10, as shown in Figure 2. The most common weaknesses were clinic-based sampling, no sample size justification and comparisons unadjusted for adiposity. Only four studies adjusted for, or stratified by, a measure of adiposity, and body mass index was higher with MASLD in several of the others. Dietary assessment was the strongest domain, with recalls, records or validated questionnaires in twelve studies, whereas four relied on short questionnaires or indices. The comparative evidence for fructose was mixed in quality: two of the seven studies were at high risk of bias, and the two with the strongest liver assessment found no difference. Scores by domain are given in Supplementary Table S4.



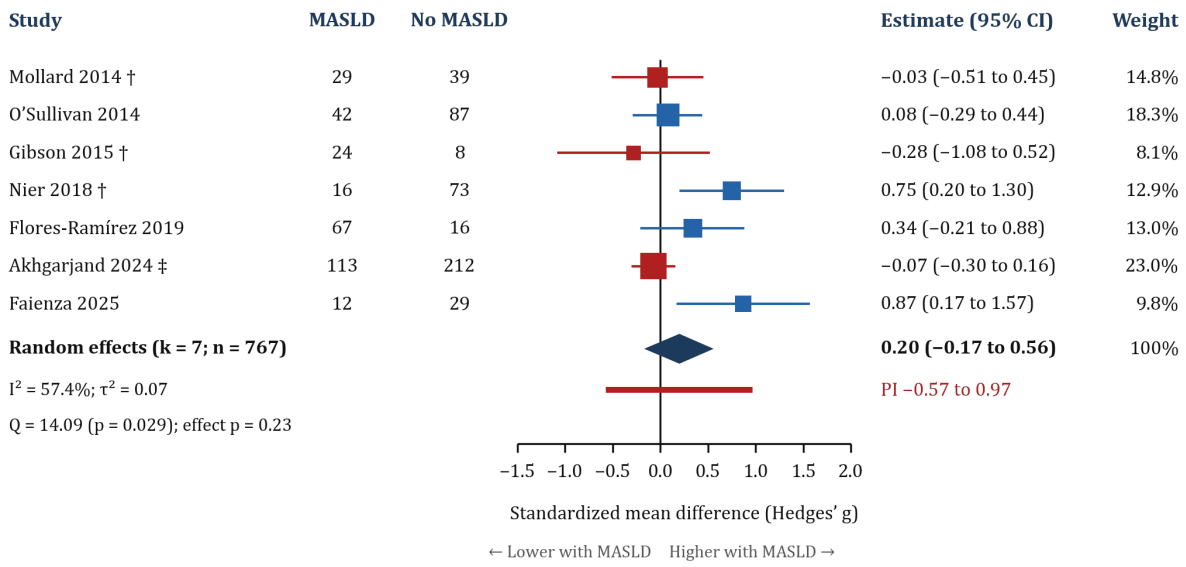
Figure 2. Risk of bias of the included studies on the adapted Newcastle-Ottawa Scale. Numbers are the points awarded in each domain and in total; green, low risk; amber, moderate risk; red, high risk.

3.4. Primary Analysis: Dietary Fructose

As illustrated in Figure 3, fructose intake did not differ significantly between the groups (SMD 0.20, 95% CI −0.17 to 0.56; $p = 0.23$; $k = 7$; $n = 767$). Heterogeneity was moderate ($I^2 = 57.4\%$; $\tau^2 = 0.07$; Cochran's Q $p = 0.03$), and the 95% prediction interval ran from −0.57 to 0.97. Two studies reported clearly higher fructose intake with MASLD: Nier 2018[23] (g = 0.75, 95% CI 0.20 to 1.30) and Faienza 2025[24] (g = 0.87, 95% CI 0.17 to 1.57). The other five showed little or no difference. They included the largest cross-sectional cohort,[33] the prospective Australian cohort[65] and the only studies using spectroscopy or histology.[25,26] In the Australian cohort the result differed by weight stratum: g = 0.95, 95% CI 0.17 to 1.73 in 28 adolescents with obesity against g = −0.11, 95% CI −0.55 to 0.32 in 101 with overweight, and the report found a significant interaction with obesity status (P = 0.032). Using the SD of fructose intake in the Mexican schoolchildren (22 g/day), the pooled estimate corresponded to about 4 g/day more fructose with MASLD, with an interval from about 4 g/day less to 12 g/day more. The data therefore remained compatible with no difference and with a moderate one.

Reported intakes varied widely. Children without MASLD reported a median of 16.7 g/day in the UK study,[26] about 29 g/day in Canadian adolescents,[25] a mean of 47 g/day in

German children[23] and 58 g/day in Mexican schoolchildren,[70] in whom fructose density was 0.028 g/kcal, about 11% of energy. Sources were described in only two studies: fresh fruit supplied most fructose in Italian children with obesity,[24] and sugar-sweetened beverages in Mexican schoolchildren.[70]



† Mean or SD converted from a median, standard error or confidence interval. ‡ Effect derived from the reported P value.
Red markers, estimates below zero; blue markers, estimates above zero. PI, 95% prediction interval.

Figure 3. Forest plot of dietary fructose intake in children with overweight or obesity, with versus without MASLD. Squares are study estimates (size proportional to random-effects weight) with 95% confidence intervals; the diamond is the random-effects pooled estimate (DerSimonian-Laird, Hartung-Knapp) and the red bar the 95% prediction interval.

3.5. Subgroup Analysis

As presented in Figure 4, risk of bias was the only variable associated with the variation, in a hypothesis-generating comparison of two studies with five. The pooled difference was 0.80 (95% CI 0.36 to 1.23; $k = 2$) in the studies at high risk of bias against −0.01 (95% CI −0.19 to 0.18; $k = 5$) in those at low or moderate risk (between-subgroup $p < 0.001$), and heterogeneity disappeared within both groups ($I^2 = 0.0\%$). Differences by diagnostic method were in the same direction but not significant: 0.31 (95% CI −0.19 to 0.81; $k = 5$) in ultrasound-based studies against −0.10 (95% CI −0.51 to 0.32; $k = 2$) with histology or magnetic resonance ($p = 0.14$). Region ($p = 0.15$), age ($p = 0.15$), dietary instrument ($p = 0.76$) and fructose metric ($p = 0.10$) did not explain the variation either, although these comparisons rested on few studies and had little power. Both studies at high risk of bias were ultrasound-based studies with younger children and absolute fructose intake, so these features could not be separated. Six subgroup comparisons were made, and these results were treated as hypothesis-generating.

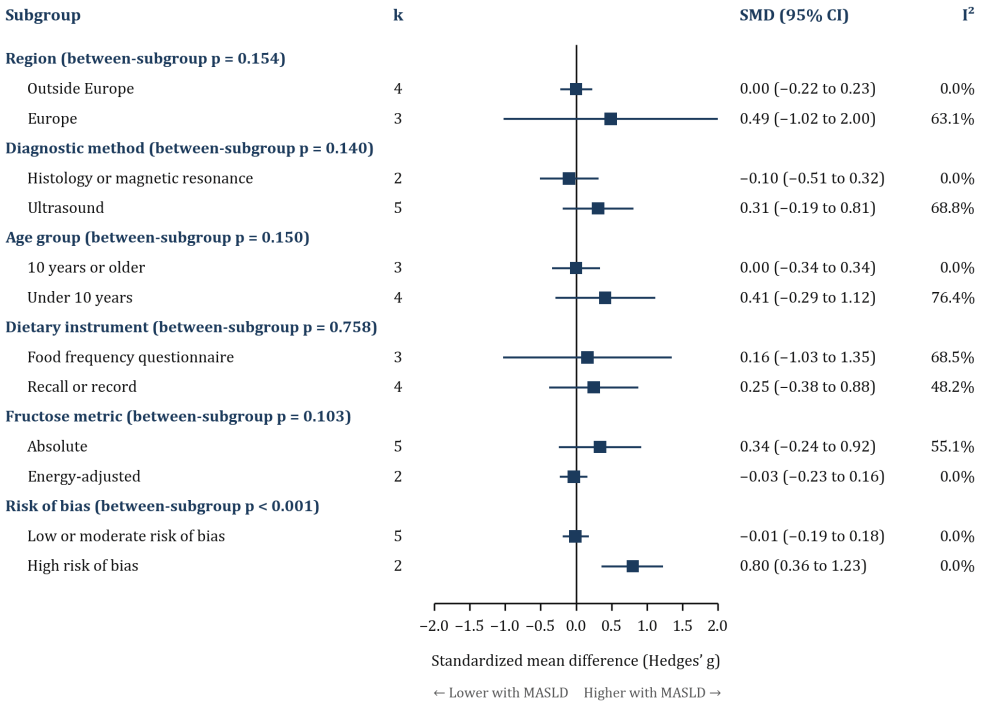
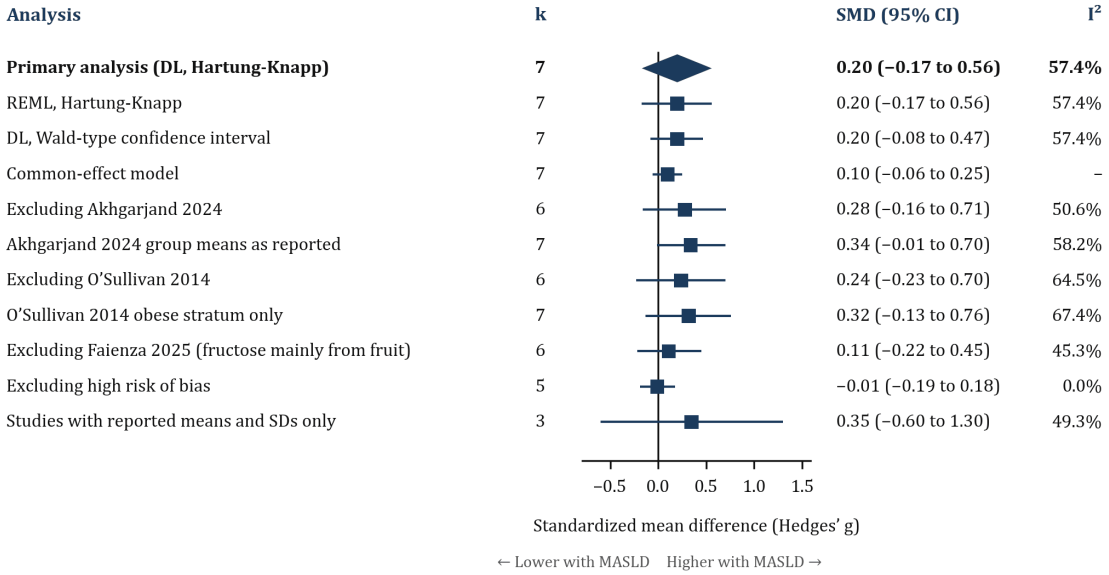


Figure 4. Subgroup analyses of the primary outcome. Squares are pooled standardized mean differences (95% confidence intervals) within each subgroup; I^2, heterogeneity within the subgroup.

3.6. Sensitivity Analysis

The result was stable to the choice of estimator, as shown in Figure 5. No single study changed the conclusion: leave-one-out estimates ranged from 0.09 to 0.28, and no analysis produced an interval excluding no difference. Excluding the two studies at high risk of bias reduced the estimate to SMD −0.01 (95% CI −0.19 to 0.18) with no heterogeneity. Excluding the largest cross-sectional cohort gave SMD 0.28 (95% CI −0.16 to 0.71), and using its group means as reported gave SMD 0.34 (95% CI −0.01 to 0.70). Using only the obese stratum of the Australian cohort gave SMD 0.32 (95% CI −0.13 to 0.76), and excluding the study in which fructose came mainly from fruit gave SMD 0.11 (95% CI −0.22 to 0.45). Excluding O'Sullivan 2014 gave SMD 0.24 (95% CI −0.23 to 0.70), and the three studies without converted or derived data (O'Sullivan 2014, Flores-Ramírez 2019 and Faienza 2025) gave SMD 0.35 (95% CI −0.60 to 1.30). The common-effect model gave SMD 0.10 (95% CI −0.06 to 0.25).



DL, DerSimonian-Laird; REML, restricted maximum likelihood.

Figure 5. Sensitivity analyses of the primary outcome under alternative estimators and study sets. The dark diamond is the primary analysis. DL, DerSimonian-Laird; REML, restricted maximum likelihood.

3.7. Small-Study Effects

The funnel plot in Figure 6 suggested that smaller studies reported larger differences, although with seven heterogeneous studies this impression was uncertain. No dietary measure reached the 10 studies needed for Egger's test. An exploratory trim-and-fill analysis imputed one study and gave an SMD of 0.11 (95% CI −0.29 to 0.52).

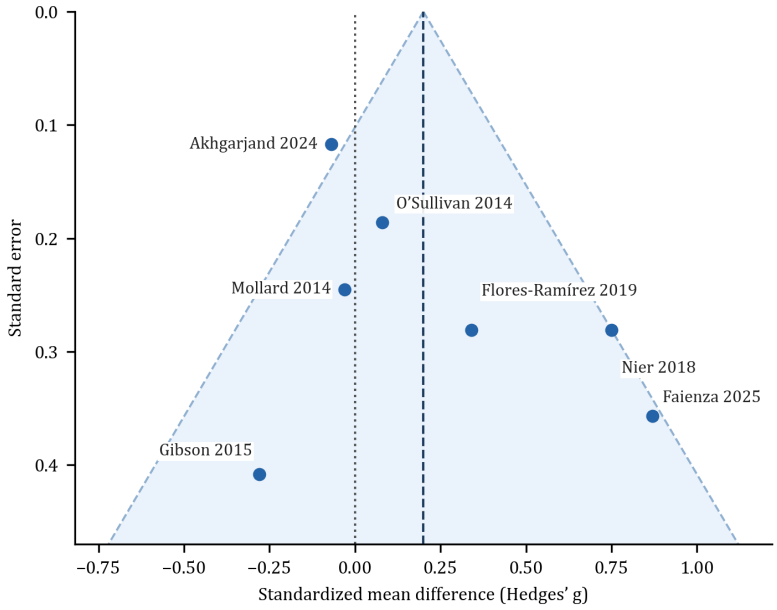


Figure 6. Funnel plot of study effects for dietary fructose against their standard errors. The dashed line marks the pooled estimate, the dotted line no difference and the shaded region the pseudo 95% confidence limits.

3.8. Sugars, Energy and Macronutrients

Pooled results for every measure are summarized in Table 3 and illustrated in Figure 7. Total sugars did not differ (SMD 0.16, 95% CI −0.29 to 0.61; I^2 = 50.4%). Energy (SMD 0.10, 95% CI −0.06 to 0.25; I^2 = 12.0%), carbohydrate (SMD 0.08, 95% CI −0.12 to 0.27; I^2 = 26.3%), total fat (SMD 0.06, 95% CI −0.08 to 0.19; I^2 = 0.0%) and protein (SMD 0.09, 95% CI −0.02 to 0.21; I^2 = 0.0%) were similar, with little heterogeneity. Saturated fat was inconsistent (SMD 0.31, 95% CI −0.49 to 1.10; I^2 = 84.3%), and fiber did not differ (SMD −0.09, 95% CI −0.57 to 0.40; I^2 = 58.8%). The saturated fat result was driven by one study reporting a large excess with NAFLD;[63] the others showed small differences in either direction. Energy was the most widely reported measure, and its prediction interval was narrow. The estimates for carbohydrate, fat and protein were all close to zero despite the large combined sample. Using the pooled SD of the larger Iranian cohort (957 kcal/day), the energy estimate corresponded to about 93 kcal/day more with MASLD, with an interval from about 58 kcal/day less to 244 kcal/day more. Wald intervals led to the same conclusions for these measures. Study-level forest plots for every measure are shown in Supplementary Figures S1–S10.

Table 3. Pooled Comparisons of Diet Between Children with Overweight or Obesity with and Without MASLD.

Dietary measure	*k*	Children	SMD (95% CI)	Wald 95% CI	*p*	I^2 (%)	GRADE certainty
Sugars							
Dietary fructose	7	767	0.20 (−0.17 to 0.56)	−0.08 to 0.47	0.23	57.4	Very low
Total sugars	5	347	0.16 (−0.29 to 0.61)	−0.17 to 0.49	0.38	50.4	Very low
Energy and macronutrients							
Energy	9	1,259	0.10 (−0.06 to 0.25)	−0.04 to 0.23	0.20	12.0	Low
Carbohydrate	8	1,220	0.08 (−0.12 to 0.27)	−0.08 to 0.24	0.38	26.3	Low
Total fat	8	1,220	0.06 (−0.08 to 0.19)	−0.06 to 0.18	0.36	0.0	Low
Saturated fat	5	543	0.31 (−0.49 to 1.10)	−0.23 to 0.85	0.34	84.3	Very low
Protein	8	1,220	0.09 (−0.02 to 0.21)	−0.03 to 0.21	0.10	0.0	Low
Dietary fiber	5	599	−0.09 (−0.57 to 0.40)	−0.40 to 0.23	0.64	58.8	Very low
Foods and eating habits							
Sugar-sweetened beverages	4	340	0.27 (−0.22 to 0.77)	−0.03 to 0.57	0.18	29.7	Low
Fast or convenience food	3	257	0.11 (−0.33 to 0.55)	−0.17 to 0.39	0.38	0.0	Low
Sweets and confectionery	3	228	−0.29 (−0.40 to −0.17)	−0.60 to 0.02	0.009	0.0	Low
Fruit	4	502	−0.01 (−0.80 to 0.77)	−0.48 to 0.45	0.96	65.2	Very low
Vegetables	3	462	−0.07 (−0.37 to 0.23)	−0.27 to 0.13	0.41	0.0	Low
Mediterranean diet quality (KIDMED)	2	149	−0.46 (−1.40 to 0.48)	−1.40 to 0.48	0.34	74.1	Very low

Note: SMD, standardized mean difference (Hedges' g), positive values indicating higher intake with MASLD; CI, confidence interval (Hartung-Knapp with k of 3 or more, Wald with k of 2); I^2, between-study heterogeneity; GRADE, certainty of the evidence. Positive values indicate higher reported intake with MASLD, which is unfavorable for sugars and unhealthy foods and favorable for fiber, fruit, vegetables and KIDMED scores. KIDMED, Mediterranean Diet Quality Index for children and adolescents. The p value comes from the Hartung-Knapp interval (Wald for k = 2).

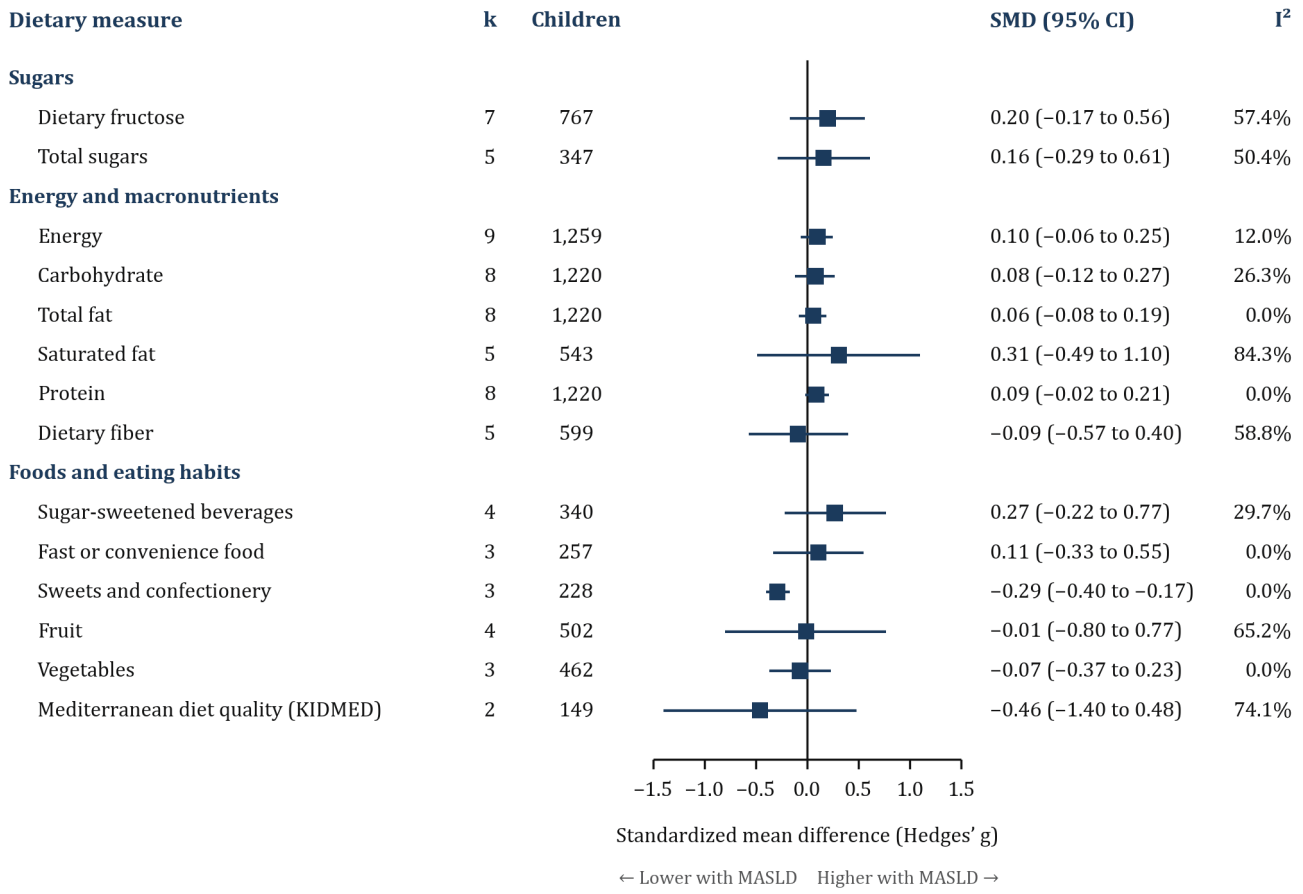
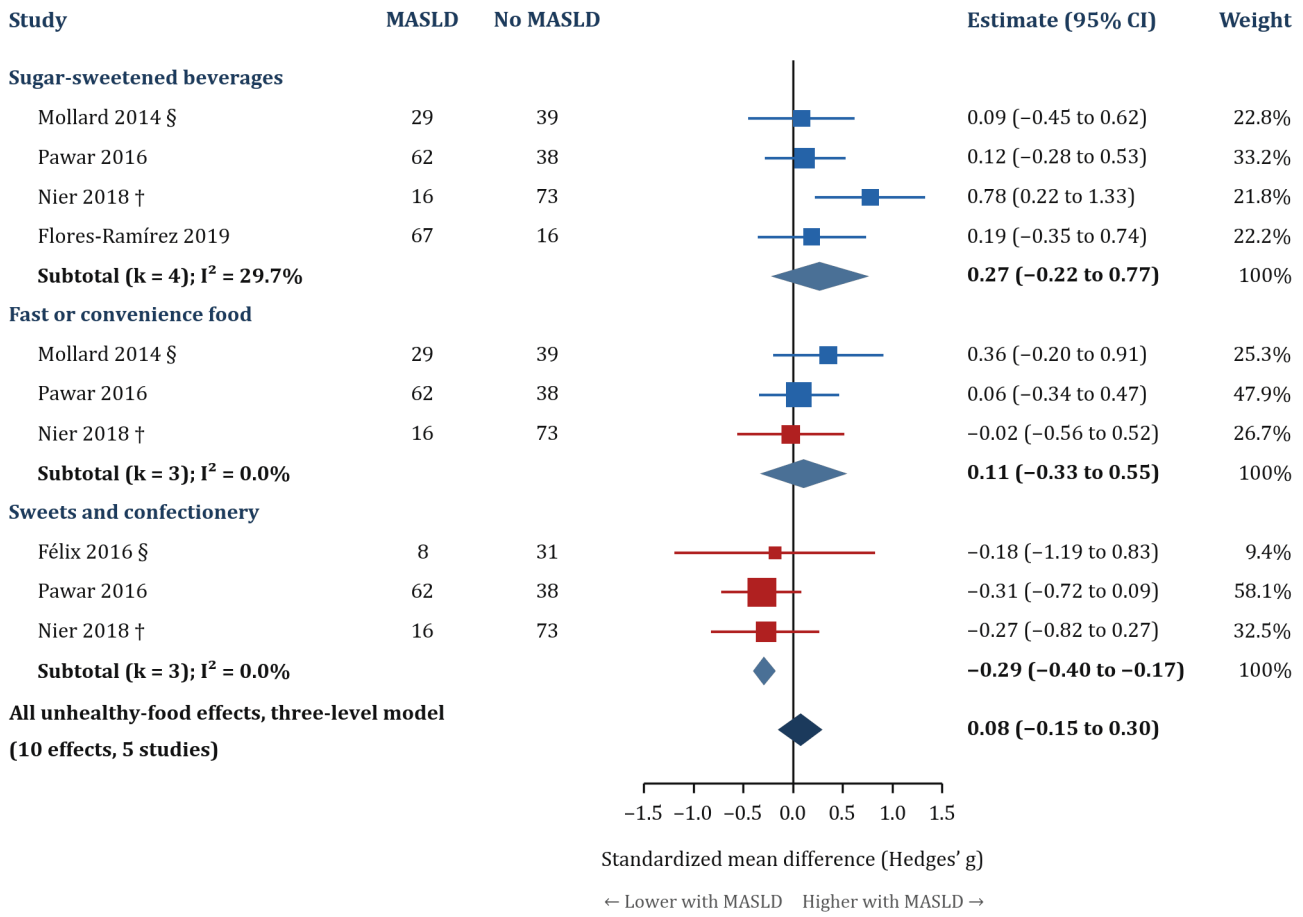


Figure 7. Pooled standardized mean differences (95% confidence intervals) for every dietary measure. Positive values indicate higher intake with MASLD; I^2, between-study heterogeneity. Positive values are unfavorable for sugars and unhealthy foods and favorable for fiber, fruit, vegetables and KIDMED.

3.9. Foods and Unhealthy Eating Habits

As illustrated in Figure 8, sugar-sweetened beverages (SMD 0.27, 95% CI −0.22 to 0.77; I^2 = 29.7%) and fast or convenience food (SMD 0.11, 95% CI −0.33 to 0.55; I^2 = 0.0%) did not differ. Sweets and confectionery appeared to be reported less often with MASLD (SMD −0.29, 95% CI −0.40 to −0.17; I^2 = 0.0%). This result rested on three studies with different measures, and it was not robust: the Wald interval ran from −0.60 to 0.02 (p = 0.07), whereas the Hartung-Knapp interval gave p = 0.009. With continuous measures alone the estimates were SMD 0.34 (95% CI −0.54 to 1.21) for beverages (k = 3), 0.03 (95% CI −0.29 to 0.36) for fast food (k = 2) and −0.30 (95% CI −0.62 to 0.03) for sweets (k = 2). Across 10 effects from 5 studies, the three-level model gave SMD 0.08 (95% CI −0.15 to 0.30), with no estimated between-study variance.

Fruit (SMD −0.01, 95% CI −0.80 to 0.77; I^2 = 65.2%), vegetables (SMD −0.07, 95% CI −0.37 to 0.23; I^2 = 0.0%) and scores on the KIDMED index, a measure of adherence to the Mediterranean diet (SMD −0.46, 95% CI −1.40 to 0.48; I^2 = 74.1%), did not differ significantly. Two other habits were reported by one study each: skipped breakfast was reported by 24% of Canadian adolescents with steatosis and 18% of those without it, and fried food by 41% and 18%.[25] Reported beverage energy was higher with NAFLD in the youngest cohort.[23] Ultra-processed food was examined within obesity by one study only, in which the third tertile of intake carried an adjusted odds ratio for MASLD of 1.75 (95% CI 1.03 to 3.00).[62] In Italy, children with MASLD reported more potato chips, jam and candies and fewer home-made snacks, without numerical data for pooling.[24]



† SD converted from a standard error. § Binary habit converted from a log odds ratio. Red markers, estimates below zero; blue markers, estimates above zero.

Figure 8. Forest plot of unhealthy foods and habits. Diamonds are random-effects subtotals; the dark diamond is the three-level summary of all effects.

Adjusted associations reported by individual studies, which were not pooled because the contrasts differed, are detailed in Table 4. They were mixed. Higher energy-adjusted fructose predicted NAFLD in the Australian obese stratum of 28 adolescents but not in the overweight stratum of 101,[65] and fructose showed no association in Canadian and Iranian adolescents.[25,33] A total fat intake above 35% of energy was strongly associated with steatosis in the Canadian study, with a very wide interval, and high refined carbohydrate intake was associated with NAFLD in a small Brazilian sample.[67]

Table 4. Adjusted Associations of Diet with MASLD or NAFLD in Children with Overweight or Obesity, as Reported by the Studies.

Study	Exposure	Association (95% CI)	Adjustment
O'Sullivan 2014 [65]	Energy-adjusted fructose, per g/day, obese stratum (*n* = 28)	OR 1.09 (1.01 to 1.19)	Sex
O'Sullivan 2014 [65]	Energy-adjusted fructose, per g/day, overweight stratum (*n* = 101)	OR 0.99 (0.97 to 1.02)	Sex
O'Sullivan 2014 [65]	Energy-adjusted total sugars, per g/day, obese stratum (*n* = 28)	OR 1.03 (0.999 to 1.07)	Sex
O'Sullivan 2014 [65]	Energy-adjusted total sugars, per g/day, overweight stratum (*n* = 101)	OR 1.00 (0.99 to 1.01)	Sex
Mollard 2014 [25]	Fructose, per g/day	OR 0.98 (0.93 to 1.03)	Fully adjusted model of the report
Mollard 2014 [25]	Total fat above 35% of energy	OR 11.76 (1.60 to 86.62)	Fully adjusted model of the report
Mollard 2014 [25]	Fried food	OR 2.75 (0.74 to 10.27)	Fully adjusted model of the report
Mollard 2014 [25]	Skipping breakfast	OR 3.13 (0.63 to 15.47)	Fully adjusted model of the report
Félix 2016 [67]	High refined carbohydrate	OR 2.17 (1.05 to 6.82)	Sex and physical activity
Akhgarjand 2024 [33]	Fructose, third versus first tertile	OR 0.67 (0.35 to 1.29)	Sex, maturity status, BMI z score and energy intake
Lee 2024 [62]	Ultra-processed food, third versus first tertile	OR 1.75 (1.03 to 3.00)	Age, sex, maternal education, birth weight, screen time, activity and energy intake

Note: OR, odds ratio with 95% confidence interval for MASLD or NAFLD, as reported by each study; the exposure contrasts differ and were not pooled. The odds ratio and interval for Félix 2016 are as printed in the source article.

3.10. Certainty of the Evidence

As summarized in Table 3, certainty was very low for fructose, total sugars, saturated fat, fiber, fruit and KIDMED scores, because of risk of bias, inconsistency and imprecision. Certainty was low for energy, carbohydrate, total fat, protein, sugar-sweetened beverages, fast food, sweets and vegetables, for which between-study consistency was better but the evidence remained self-reported and largely unadjusted. No measure reached moderate certainty. Ratings for the sweets result should be read with the lack of robustness described above. These ratings indicate that further research is very likely to change the estimates, and they apply to the comparison of reported intake, not to the causal effect of fructose on the liver. Reasons for rating down are listed in Supplementary Table S5.

4. Discussion

4.1. Main Findings

This meta-analysis compared the diet of children with excess weight with and without MASLD, and the results fell into two groups. For energy, carbohydrate, fat and protein, the intervals were narrow and centered near zero. Total intake and macronutrient balance therefore did not distinguish these children to any important degree. For fructose, the estimate favored higher intake with MASLD (SMD 0.20), yet the confidence and prediction intervals (−0.57 to 0.97) included no difference. Higher intake was confined to two studies at high risk of bias. The other five showed no difference and no heterogeneity (SMD −0.01, 95% CI −0.19 to 0.18). A modest difference could not be excluded overall, but the five studies at low or moderate risk of bias gave it no support.

Most unhealthy food habits did not separate the groups either. The apparent reduction in sweets ran against the expected direction and did not hold under a Wald interval, so it was more likely a reporting phenomenon than a real difference. Taken together, the data suggested that children who already had excess weight differed little in what they reported eating, compared with the dietary environment they shared and that produced the excess weight in the first place. They did not show that diet was unimportant to the disease.

4.2. Comparison with Other Evidence

Cross-age syntheses linked ultra-processed food to NAFLD, with considerable heterogeneity,[28] and a review of high-fructose corn syrup found very few focused studies.[72] Our narrower comparison gave weaker results. That would be expected if adiposity mediates part of the association between diet and liver disease; this review did not test mediation. Adolescent data pointed the same way. Higher ultra-processed food intake was linked to NAFLD mainly through body fatness,[73] and a later analysis found no clinically relevant association.[74] Among Korean children and adolescents with obesity, by contrast, the highest tertile of ultra-processed food intake carried higher adjusted odds of MASLD, a result based on magnetic resonance fat fraction.[62] Findings in adults are also inconsistent. In Finnish adults, higher fructose was associated with lower odds of MASLD, and fructose from fruit or beverages showed no association.[75] In a Korean analysis, soft drinks were associated with MASLD,[76] and in people without known liver disease they were associated with liver stiffness and steatosis.[77]

The trial evidence has to be reconciled with these findings. In adolescent boys, a diet low in free sugars reduced hepatic de novo lipogenesis and liver fat over eight weeks.[17] A dietitian-led clinical program to reduce sugar, in contrast, did not improve liver outcomes in Latino youth, and liver fat improved in those who lost fat mass whatever their allocation.[18] The later trial described the earlier study as an extreme restriction. This suggests that the intensity of the change matters, and that modest self-directed reductions may be too small to affect the liver unless they also reduce adiposity. A recent network meta-analysis of dietary treatment in pediatric MASLD found no dietary pattern significantly better than the others, with low to very low certainty.[27] Mediterranean-style diets improved steatosis and insulin resistance in children,[78–80] and well-controlled feeding trials of carbohydrate restriction are in progress.[81] None of this conflicts with our result. Treatment trials show what happens when diet changes; the present review asked whether diet already differed between affected and unaffected children.

4.3. Sources of Sugar and the Life Course

The comparison of total fructose may have hidden differences between sources. Liquid sugars were the main concern in most reports, and sugar-sweetened beverages were related to uric acid and insulin resistance in adolescents.[82,83] Whole fruit behaves differently. A large analysis from childhood to adulthood tied sugary drinks and juice, but not whole fruit, to later hypertension.[84] Only two of our studies described sources, and they pointed in opposite directions: fruit dominated in Italy and beverages in Mexico.[24,70] Removing the fruit-dominated study reduced the estimate but left the conclusion unchanged. The Italian authors attributed the excess of fruit partly to misreporting, which showed how hard total fructose was to interpret.

Timing is a further consideration. Sugary drinks in infancy predicted NAFLD at school age independently of later intake and body mass index,[19] and childhood nutrient intake and its changes were associated with hepatic fat in adolescence.[85,86] Free sugar intake and its sources shift through childhood,[87] and these trajectories were associated with cardiometabolic risk markers.[88] Sugar rationing early in life was associated with a lower later risk of MASLD.[89] Children's diets are a moving target. A single assessment in an established case may therefore miss the exposure window that matters. Early high intake of sugar-sweetened beverages and ultra-processed foods also predicted a rise in body mass index.[90] That is a route to liver disease that a comparison within excess weight cannot detect. The cohort that followed free sugar intake from age three into adult life supports this reading: the association with severe steatosis disappeared after adjustment for later body mass index.[22]

4.4. Biological and Clinical Plausibility

The mechanistic case for fructose remains strong. Controlled studies showed dose-dependent effects of fructose-containing beverages on hepatic lipid,[91] restriction lowered de novo lipogenesis in boys,[17] and fructose beverages increased oxidative stress in pediatric MASLD.[92] Fructose restriction also lowered ceramides in children with obesity.[93] Our findings do not contradict this evidence. Reported intake could fail to separate two groups with similar diets even if fructose contributes to disease in both, particularly if susceptibility differs. Several results support a role for susceptibility. In Latino adolescents, sugars were associated with liver stiffness only with the GG genotype of *PNPLA3*,[94] and arachidonic acid and unsaturated fat influenced fibrosis by genotype.[95] In the prediction of adolescent steatotic liver disease, childhood adiposity and fiber intake both contributed,[21] and gut microbiome features predicted MASLD

in children with obesity.[96] Endotoxin markers, however, were raised in adolescents with obesity whether or not they had MASLD, which suggests a shared exposure in both groups.[97] Differences in susceptibility, rather than in diet, may therefore partly separate the two groups, although susceptibility was not measured in the included studies.

4.5. Reverse Causation and Reporting Bias

Two features of this evidence argue against taking the null estimates at face value. The first is timing: most studies measured diet at or after the diagnosis, when families may already have received advice. Dietary change after diagnosis would lower sugar intake in the very group expected to have the highest exposure, and it could also explain the lower reported intake of sweets. The only prospective comparison recorded diet three years before the liver scan. Overall it showed no difference. Adolescents with obesity who did not develop NAFLD had, however, reported less fructose, a finding that rests on 28 adolescents.[65]

Second, reporting error is likely to be differential. In a food-record study, about two-thirds of adolescents under-reported energy, and under-reporting was associated with body weight and with lower reported intake of non-core foods.[98] A Chilean study found that under-reporting was more frequent with excess weight and that the bias was specific to certain foods.[99] Children with MASLD who were heavier and more conscious of their weight may have under-reported sugary foods more than their peers. A meta-analysis against doubly labeled water confirmed that food records underestimated energy intake in children.[100] Energy values above 3,000 kcal per day in the Iranian cohorts, on the other hand, showed that food frequency questionnaires could err in the opposite direction. Serum carbon isotope ratio was proposed as an objective marker of added sugar intake in youth with steatotic liver disease, but it did not track individual change over eight weeks.[101] Such error could bias the comparisons toward no difference and widen the gap between the experimental and observational evidence.

4.6. Heterogeneity

Heterogeneity for fructose followed study quality more than geography, age or diagnostic method, although these features overlapped in the two studies at high risk of bias and could not be separated. In one pediatric study, radiologists' sensitivity for steatosis on ultrasound ranged from 79.4% to 97.1%.[102] Misclassification was therefore likely, and it may have fallen preferentially on the heaviest children. Newer quantitative ultrasound and attenuation methods should reduce this problem.[103–105] Studies at high risk of bias were generally unadjusted. Residual confounding by adiposity was likely, because a heavier child reported more food in absolute terms. The estimate was smaller with energy-adjusted fructose (SMD −0.03) than with absolute intake (SMD 0.34), which would fit confounding by total intake. That comparison had little power, though, and standardization removed units, not differences in what was being measured. Fructose in grams, in grams per 1000 kcal and as an energy residual were different quantities. The instruments varied too, from 7-day diaries to brief weekly questionnaires. Age spanned early childhood to late adolescence, a period in which diet, puberty and insulin resistance all change.

4.7. Unhealthy Eating Habits

The habits named in the title of this review were the least well measured. Only sugar-sweetened beverages, fast or convenience food and sweets could be pooled, each with three or four studies and with measures ranging from weekly frequency to daily energy. Fried food and skipped breakfast were reported by a single study. For beverages the pooled estimate pointed to higher intake with MASLD, and its Wald interval only narrowly included no difference, so a modest effect was plausible; it was driven by one cohort of young German children,[23] and the other three studies gave small differences. The three-level summary of all unhealthy-food effects was close to zero, but it combined exposures that may have acted differently. These findings did not mean that the habits were harmless. Ultra-processed food and sugary drinks were linked to adiposity in children, including Indonesian pupils,[30,90] and meal timing was linked to adolescent MASLD.[31] They indicated only that the habits, as measured, did not separate affected from unaffected children who already had excess weight. The Korean result was a useful counterpoint. It came from a study with magnetic resonance measurement and a food diary, and it measured the degree of processing, not a single nutrient or food, so a whole-diet exposure may detect a difference that single items miss, although it did not adjust for adiposity.[62]

4.8. Strengths and Limitations

The main strength was the restriction to children with excess weight, which reduces the largest confounder of dietary studies in this field. The review also combined four databases with citation searching, included studies from 13 countries, applied a conservative interval method with a Wald check and reported prediction intervals and certainty ratings for every measure.

Limitations were considerable, and they bore directly on how the null estimates should be read. First, most data were cross-sectional, so MASLD could have changed diet or the reporting of it. Second, intake was self-reported, with the differential error described above. Third, few studies measured fructose, and sources were rarely given. Fourth, eight reports sought were not retrieved. At least one of them described higher fructose intake with a higher grade of NAFLD in obese children (Supplementary Table S3), so their absence may have biased the primary estimate toward no difference. Fifth, several effects came from converted or derived data, including the largest fructose study, and the pooled estimate ranged from 0.20 to 0.34 depending on the data used for that study. Sixth, adiposity was reduced as a confounder but not removed: children with MASLD were often heavier within the excess-weight range, and only four studies adjusted for, or stratified by, adiposity. Seventh, the subgroup analyses were observational comparisons between studies, the contrast between studies at high and at low or moderate risk of bias rests on two studies against five and is hypothesis-generating, and the secondary analyses were not adjusted for multiplicity. Eighth, screening and data extraction were done by one reviewer and verified by a second, which may have allowed some errors to pass unnoticed. Finally, no study came from Indonesia or Southeast Asia, so applying these findings locally requires caution.

4.9. Implications for Practice and Research

Three practical messages follow. First, reported diet should not be used to rule MASLD in or out in a child with excess weight, because energy and macronutrient intakes were similar and fructose intake did not clearly differ. The review tested no screening strategy, but the lack of dietary discrimination argues against selecting children for assessment on the basis of a diet history. Second, advice to limit sugar-sweetened beverages and free sugars should continue for all children with excess weight; the experimental evidence is supportive and the advice carries little risk. Third,

clinicians should expect fat-mass reduction to matter more than sugar restriction alone, and intensive dietary change to be needed before the liver improves.[17,18] Standard care improved fewer than one-third of children over two years, and improvement followed changes in body mass index and cholesterol.[15] Dietary advice should therefore be judged by what it does to these measures. Counseling should also stay sensitive to weight stigma. Children who feel judged may under-report what they eat, which would further reduce the value of a dietary history.

Future research needs within-excess-weight comparisons that record diet before the diagnosis, use objective biomarkers of sugar intake, quantify fructose by source, and measure the liver with magnetic resonance or calibrated quantitative ultrasound. Analyses should adjust for adiposity and report means, SDs and the numbers in each group. Southeast Asian data are a particular priority. Overweight is common among Indonesian schoolchildren,[29] and adolescents with obesity there report poor dietary intake,[106] yet no study in this review came from Indonesia. A school or clinic cohort of Indonesian children with excess weight, with imaging and repeated dietary assessment, would show whether these findings hold where beverage and snack patterns differ.

5. Conclusion

Children and adolescents with overweight or obesity reported similar intakes of energy, carbohydrate, fat and protein, whether or not they had MASLD. Most unhealthy food habits did not differ either. Only ultra-processed food was associated with MASLD, and a single study examined it. No clear difference in fructose intake emerged: the pooled estimate favored higher intake with MASLD, but its interval included no difference, higher intake was confined to two studies at high risk of bias, and the other five found none. Sweets were reported less often with MASLD. That result was not robust and may reflect reporting behavior. Certainty was low or very low for every measure.

Nothing here shows that fructose or unhealthy eating is harmless. Experimental studies support a causal role for fructose, whereas most of the observational evidence reviewed here was self-reported, collected after the diagnosis and affected by residual confounding. The data showed something narrower. Reported diet alone did not identify children with excess weight who had MASLD, and that finding is clinically useful.

The dietary history should not carry the assessment of a child with excess weight. Limiting sugar-sweetened beverages and free sugars should stay part of weight management for all such children on experimental and general nutritional grounds, and fat-mass reduction should remain a central aim. Future studies should measure diet before the diagnosis, using objective biomarkers; separate fructose by source; use standardized liver imaging; and adjust for adiposity. Southeast Asian children, absent from the evidence so far, should be included.

Declarations

Ethics Approval and Consent to Participate

Not applicable. This systematic review used published aggregate data and involved no human participants.

Consent for Publication

Not applicable.

Availability of Data and Materials

The extraction workbook, the analysis code and the completed PRISMA 2020 checklist are available from corresponding author on reasonable academic request.

Competing Interests

The authors declare no competing interests.

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Authors' Contributions

IWWA: Conceptualization, Methodology, Software, Investigation, Data curation, Formal analysis, Visualization, Writing - original draft. NNMN: Conceptualization, Methodology, Validation, Supervision, Writing - review and editing. IGNSP: Conceptualization, Methodology, Validation, Supervision, Writing - review and editing. ARA: Conceptualization, Investigation, Data curation. RAR: Conceptualization, Investigation, Data curation. All authors approved the final manuscript.

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None.

Use of artificial intelligence

None declared.

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