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Long-Term Metabolic Outcomes Following Bariatric Surgery: A Systematic Review, Meta-Analysis, and Meta-Regression

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ABSTRACT

Bariatric surgery (BS) has short- and medium-term benefits in improving metabolic syndrome (MetS) components, but its long-term impact remains incompletely characterized. This systematic review and meta-analysis aimed to quantify the long-term effects of BS (≥ 5 years) on fasting blood glucose (FBG), HDL cholesterol, triglycerides (TG), waist circumference (WC), and systolic and diastolic blood pressure (SBP and DBP). A comprehensive literature search was conducted across relevant databases. Eligible studies included randomized controlled trials and observational cohorts reporting pre- and postsurgical metabolic outcomes after at least 5 years of follow-up. Random-effects meta-analyses and meta-regressions were performed. Fifty-four articles (49 cohorts and five RCTs), including 37,840 baseline participants, were analyzed. BS was associated with significant improvements across all outcomes, with effect sizes varying by study design. Pooled estimates ranged from −22.97 to −44.68 mg/dL for FBG, +10.25 to +11.94 mg/dL for HDL, −54.11 to −59.59 mg/dL for TG, −20.55 cm for WC, −7.82 to −9.76 mmHg for SBP, and −4.80 to −6.48 mmHg for DBP. Meta-regression showed stronger effects in patients with higher baseline values. BS provides sustained, long-term improvements in all major MetS components. However, these findings are primarily driven by studies on Roux-en-Y gastric bypass, whereas long-term data on sleeve gastrectomy remain limited. High-quality longitudinal evidence on sleeve gastrectomy is needed to improve the generalizability of these results to contemporary surgical practice.

Abbreviations: BMI, body mass index; BPD, biliopancreatic diversion; BS, bariatric surgery; CI, confidence interval; CVD, cardiovascular disease; DBP, diastolic blood pressure; Dsw, duodenal switch; FBG, fasting blood glucose; FSO, International Federation for the Surgery of Obesity and Metabolic Disorders; GB, gastric bypass; Gband, gastric banding; GLP-1, glucagon-like peptide-1; HDL, high-density lipoprotein; IB, intestinal bipartition; Mesh, medical subject headings; MetS, metabolic syndrome; NHANES, National Health and Nutrition Examination Survey; PRISMA, preferred reporting items for systematic reviews and meta-analyses; PYY, Peptide YYCCKCholecystokininI; RCT, random clinical trial; RoB, risk of bias; RYGB, Roux-en-y gastric bypass; SBP, systolic blood pressure; SG, sleeve gastrectomy; T2DM, type 2 diabetes mellitus; TG, triglycerides; WC, waist circumference; WMD, weighted mean difference.

Bernardo Paz Barboza and Pietro Ferrara contributed equally to this work.

1 | Introduction

Metabolic syndrome (MetS) is a highly prevalent and complex condition defined by a cluster of interconnected metabolic abnormalities that increase the risk of cardiovascular disease (CVD), type 2 diabetes mellitus (T2DM), and all-cause mortality. It is characterized by the presence of abdominal obesity, insulin resistance, elevated blood pressure, reduced levels of high-density lipoprotein (HDL) cholesterol, and elevated serum triglycerides (TG) [1–4]. Its etiology is multifactorial, involving genetic predisposition, unhealthy dietary patterns, physical inactivity, and socioeconomic disparities [1, 5].

MetS represents a significant global health burden, with prevalence estimates ranging from 20% to over 30%, particularly among older and urbanized populations. In the United States, the National Health and Nutrition Examination Survey (NHANES, 2007 to 2014) showed a prevalence of 34.3% among adults, increasing to 54.9% in individuals aged 60 years and older [3]. Similar age-associated increases have been reported in European populations, where MetS prevalence exceeds 30% in adults over 70 years of age [2].

In particular, obesity plays a central role in the pathogenesis of MetS, acting as a key driver of insulin resistance, dyslipidemia, and elevated blood pressure [1, 6]. The growing prevalence of obesity worldwide has been closely paralleled by the rising incidence of MetS, underscoring the importance of addressing excess adiposity as a therapeutic target [1, 7]. The global rise in excess body weight has reached critical proportions, with over 2.5 billion adults classified as overweight and more than 890 million as obese as of 2022. Obesity now represents one of the most pervasive and multifaceted public health challenges worldwide, primarily due to its strong, well-documented association with numerous chronic conditions—most notably CVD [6, 7]. Although lifestyle modifications and pharmacological interventions remain first-line treatments, their long-term effectiveness is often limited, particularly in individuals with severe or treatment-resistant obesity [8–10]. In this context, bariatric surgery (BS)—also known as metabolic surgery—has emerged as the most effective treatment for achieving sustained weight loss and marked improvements in metabolic health. Robust evidence indicates that BS can induce remission of T2DM, normalize lipid profiles, lower blood pressure, and modulate inflammatory and hormonal pathways implicated in MetS. Beyond metabolic control, BS has also been associated with a 25%–50% reduction in overall mortality over long-term follow-up, highlighting its potential not only to improve comorbidities but also to enhance survival [8–11]. However, despite the robust evidence supporting the short- and medium-term benefits of BS, its long-term impact on the individual components of MetS remains incompletely characterized [12, 13]. Most existing studies differ by short follow-up durations, heterogeneous populations, and varying definitions of metabolic outcomes [12, 13], which hinder a clear understanding of the sustained metabolic effects of different surgical procedures. Given the global burden of MetS and the increasing reliance on BS as a therapeutic option, a comprehensive synthesis of long-term evidence is essential to inform clinical decision-making, refine patient selection, and optimize long-term management strategies. To address this gap, we conducted a systematic review and meta-analysis to evaluate the long-term effects of BS on key metabolic outcomes.

2 | Materials & Methods

2.1 | Study Design

This systematic review was conducted according to a predefined protocol registered in PROSPERO with number CRD42024625365 [14] and is reported following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines [15]. The review focused on evaluating the long-term metabolic effects of BS, specifically on key components of MetS: fasting blood glucose (FBG), HDL, TG, waist circumference (WC), and systolic blood pressure (SBP) and diastolic blood pressure (DBP).

2.2 | Search Strategy and Study Selection

A comprehensive systematic literature search was conducted across four major databases: MEDLINE/PubMed, EMBASE, Cochrane Library, and Web of Science. The search strategy incorporated a combination of free-text terms and Medical Subject Headings (MeSH) related to the intervention (e.g., *bariatric surgery* and specific surgical techniques), the follow-up duration (e.g., *long-term*), and the outcomes of interest. The complete search terms and strategies are detailed in Table S1. The search was performed on December 9, 2024. No restrictions were applied to the year of publication, and studies published in English, French, Italian, Portuguese, and Spanish were considered for the inclusion, by reviewers fluent in the respective languages or with documented proficiency in those languages. Eligible studies included randomized controlled trials (RCT), as well as observational studies (cohort, case–control, and cross-sectional designs) that assessed the long-term effects ($\geq$ 5 years) of BS, comparing follow-up outcomes with baseline values. No restrictions were applied regarding the type of BS procedure, study population, or age group, ensuring broad inclusion across surgical techniques and demographic profiles. Articles were excluded if they were conference abstracts, review papers, meta-analyses, expert commentaries, opinion pieces, or if they lacked sufficient methodological detail or extractable outcome data. Additionally, studies were excluded if follow-up outcomes were assessed exclusively via telephone, were based solely on participant self-reports, or if outcomes were defined only through medication use without accompanying biochemical evaluations. Inclusion criteria were developed according to the Participants, Interventions, Comparisons, Outcomes, and Study Design (PICOS) framework, as detailed in Table S2.

Study selection was performed in two stages. First, titles and abstracts were independently screened by two reviewers (B.P.B. and L.Y.). In the second stage, full texts of the studies selected by both reviewers were retrieved and assessed for eligibility. When full-text articles were unavailable or contained missing information, the corresponding authors were contacted for clarification. Disagreements between reviewers were resolved through consultation with a senior investigator (P.F.), who facilitated consensus.

2.3 | Data Extraction and Analysis

Key characteristics and outcome data were extracted from each included study, including study design, year of publication, country, baseline characteristics of the study population, inclusion and exclusion criteria, type of BS performed, duration of long-term follow-up, and outcome values at baseline and at follow-up. Continuous outcomes measures for FBG, HDL, and TG were standardized to mg/dL to improve interpretability [16, 17]. When necessary, parametric values were estimated from non-parametric data and pooled means and pooled standard deviations were calculated in accordance with the recommendations of the Cochrane Handbook [18] and Wan et al. [19]. The complete set of formulas used for these conversions is provided in Table S4.

Two reviewers (B.P.B. and L.Y.) assessed the possible risk of bias (RoB) using the Joanna Briggs Institute (JBI) Critical Appraisal tools for observational studies [20], and Revised Cochrane risk-of-bias tool for randomized trials (Rob-2) for RCTs [21].

For the quantitative synthesis of outcomes, random-effects meta-analyses were conducted for all models. Results were summarized as pooled estimates with 95% confidence intervals (95% CI). Heterogeneity across studies was assessed using the I^2 statistic and Cochran's Q-test. In addition, subgroup analyses were performed based on BS type, duration of follow-up, age, baseline BMI category, and risk of bias.

For meta-analysis including 10 or more studies, potential publication bias and small-study effects were evaluated through visual inspection of funnel plots and formally tested using Egger's test [22]. To explore the influence of baseline mean values, age, BMI, male sex proportion, and other potential moderators—such as surgical procedure, follow-up duration, and risk of bias—on pooled effect estimates, random-effects meta-regression analyses were conducted for outcomes with at least 10 included studies [22]. Potential moderators were initially assessed in univariable meta-regression models. Variables with p-values below 0.05 were subsequently included in a multivariable model to evaluate their combined contribution to between-study heterogeneity. Leave-one-out

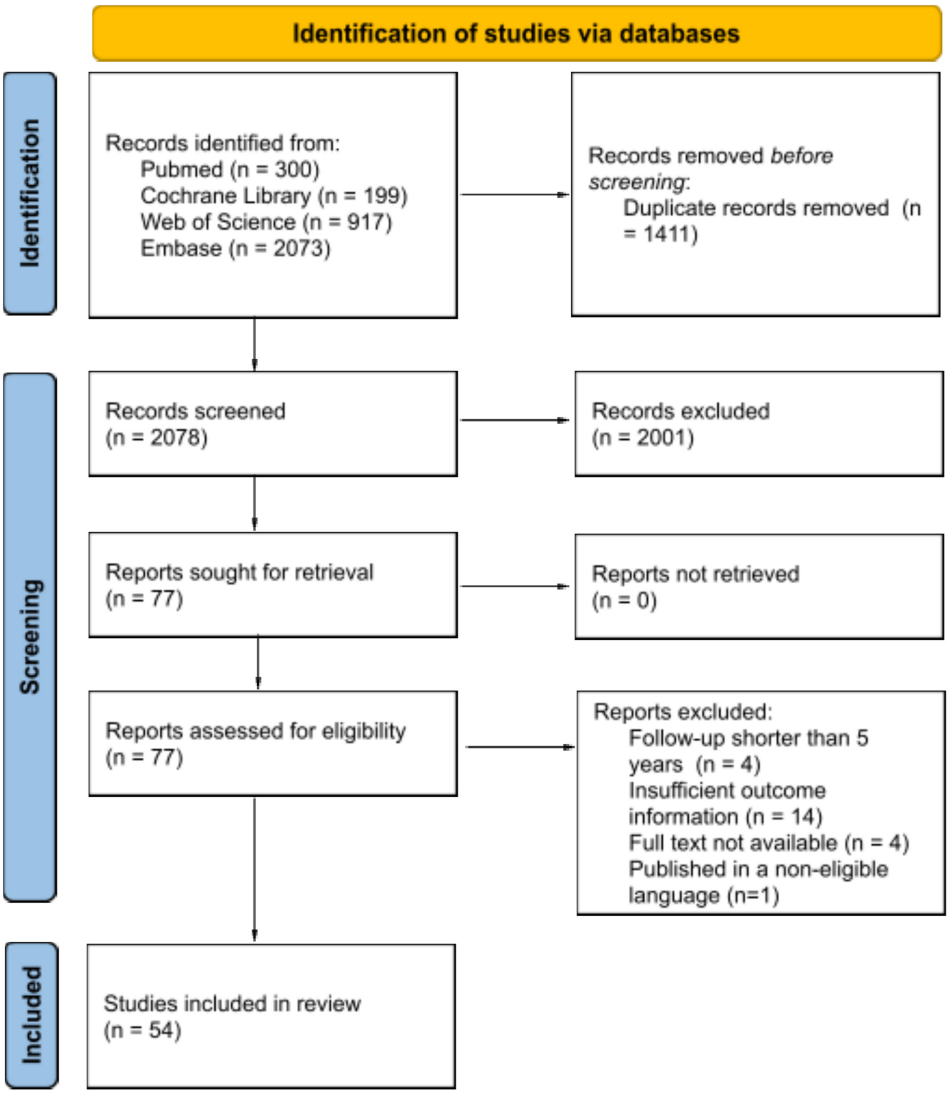

FIGURE 1 | PRISMA flow diagram of study identification, screening, and inclusion.

analyses and the trim-and-fill method were applied to assess the robustness and sensitivity of the results in meta-analyses including at least 10 studies. All analyses and graphical outputs were performed using STATA software, version 18.0 (StataCorp, College Station, TX, USA).

3 | Results

3.1 | Literature Search and Studies Overview

From the initial search, which yielded 3489 records, 51 cohorts of BS patients were included according to the predefined eligibility criteria. Data from these cohorts were sourced from a total of 54 individual articles, as in two cases [23, 24] it was necessary to refer to sister publications [25–27] to complete baseline and clinical information (Figure 1). Overall, the 54 articles comprised 49 cohort studies [10, 23–70] and five RCT [71–75]. With regard to the outcomes, the BS long-term effect on FBG was assessed in 36 studies, HDL cholesterol in 38, TG in 42, and SBP and DBP in 23. When distinct subcohorts could be identified within a study (e.g., according to BS type), they were analyzed separately. An overview of the number of subcohorts and participants contributing to each outcome is provided in Figure 2, together with a summary of the pooled estimates.

The included studies collectively enrolled a baseline sample of 37,840 participants, of whom more than 70% were female. Key baseline characteristics of the included cohorts are summarized in Table 1, whereas complete study characteristics, and baseline and long-term outcomes are reported in Table S3. Participant age ranged from 16.5 ± 1.3 years [40] to 54.2 ± 11.2 years [67], whereas BMI varied from 27.2 ± 3.2 kg/m^2 [10], in an Asian population, and 35.1 ± 2.6 kg/m^2 in non-Asian population [48], up to 56.7 ± 10.0 kg/m^2 [61]. In terms of geographical distribution, 40.7% of the studies (22/54) were conducted in Europe, 27.8% (15/54) in the Americas, 24.1% (13/54) in Asia, and 7.4% (4/54) in Oceania. Regarding the type of BS performed, the most frequently reported procedure was Roux-en-y Gastric Bypass (RYGB), included in 45.1% of studies. This was followed by Sleeve Gastrectomy (SG) in 23.5%, mixed procedures in 17.6%, gastric banding in 11.8%, Gastric Bypass (GB) in 9.8%, Biliopancreatic Diversion (BPD) in 5.9%, and Duodenal Switch (Dsw) and Intestinal Bipartition (IB) in 2% of studies each. Follow-up duration ranged from 5 to 15 years, with 52.9% of studies reporting a 5-year follow-up and 47.1% exceeding 5 years.

Participant attrition varied across outcomes, with ≥ 50% loss reported in 18.6% of studies for FBG, 16.3% for HDL, 14.6% for TG, 16.7% for WC, and 14.9% for SBP/DBP (Tables 1 and S3). Across studies, the overall direction of effect was consistent and indicated improvement in all evaluated outcomes. However, isolated studies reported no significant changes in FBG ($n = 3$) [48, 67, 74], HDL ($n = 1$) [10], TG ($n = 1$) [54], and SBP ($n = 1$) [71].

A few studies assessed outcomes categorically, confirming improvements in low HDL levels [29, 39], hypertension [33], and high TG levels [39].

Only two studies explicitly evaluated MetS diagnosis [37, 66], both showing postsurgical improvement despite using different criteria (the International Diabetes Federation and NCEP ATP III, respectively).

Overall, the risk of bias assessment indicated a moderate-to-high methodological quality across the included studies. Among cohort studies, nearly half (49%) achieved JBI conformity scores ≥ 70% [23–25, 28–30, 34, 36, 39, 41, 42, 44, 48, 51–53, 57, 65–70], suggesting good adherence to methodological standards. Among RCTs, only one study was classified as having a high risk of bias [73], whereas the remaining trials were judged as having some concerns, mainly related to deviations from intended interventions or

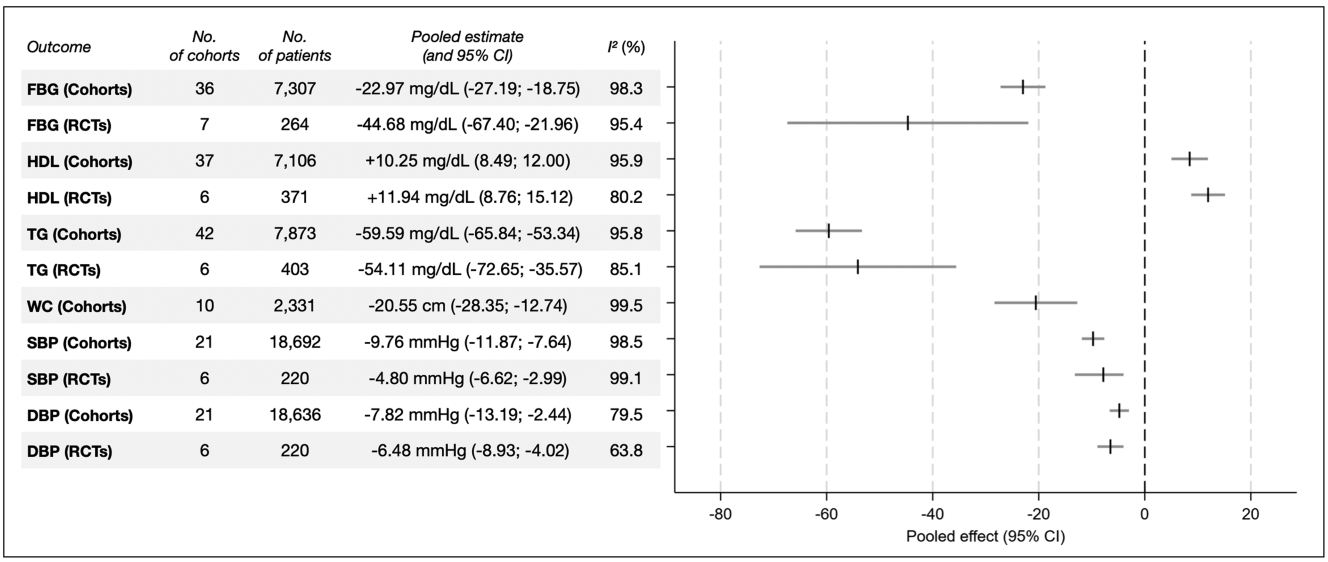

FIGURE 2 | Summary of pooled long-term metabolic outcomes following bariatric surgery, by study design. Pooled estimates were calculated using random-effects meta-analytic models. Abbreviations: 95%CI, 95% confidence interval; DBP, diastolic blood pressure (mmHg); FBG, fasting blood glucose (mg/dL); HDL, high-density lipoprotein cholesterol (mg/dL); RCT, randomized controlled trial; SBP, systolic blood pressure (mmHg); TG, triglycerides (mg/dL); WC, waist circumference (cm).

TABLE 1 | Summary characteristics of the included studies.

Author, year	Baseline population	Intervention	Follow-up (years)	Outcomes
Adams T. et al. 2015 Adams T. et al. 2017	$N = 418$ $M = 65$ (15.6%) $F = 353$ (84.4%) Baseline age = 42.5 (±10.9) Baseline BMI = 47.3 (±7.7)	RYGB	12	FBG HDL TG SBP DBP
Aftab H. et al. 2014	$N = 184$ $M = 46$ (25.0%) $F = 128$ (75.0%) Baseline age = 38.0 (±9.0) Baseline BMI = 46.0 (±5.0)	GByp	5.25	FBG HDL TG SBP DBP WC
Ahmed B. et al. 2018	Group SG $N = 57$ $M = 18$ (32.0%) $F = 39$ (68.0%) Baseline age = 50.0 (IQR 36.0 to 55.0) Baseline BMI = 56.4 (IQR 47.2 to 63.2) Group RYGB $N = 57$ $M = 18$ (32.0%) $F = 39$ (68.0%) Baseline age = 48.0 (IQR 38.0 to 54.0) Baseline BMI = 56.0 (IQR 47.7 to 63.2)	SG RYGB	5	Low HDL[1]
Aminiam A. et al. 2014 Aminiam A. et al. 2015 Aminiam A. et al. 2020	$N = 136$ $M = 46$ (37.8%) F = 90 (62.2%) Baseline age = 51.1 (±9.4) Baseline BMI = 42.5 (±9.0)	RYGB (87.2%) SG (12.8%)	8 (range 5–14)	FBG HDL TG SBP DBP
Andersson D. et al. 2017	Group nonomentectomy $N = 41$ $M = 0$ (0%) $F = 41$ (100%) Baseline age = 41 (±9.0) Baseline BMI = 43.1 (±5.4) Group omentectomy $N = 40$ $M = 0$ (0%) $F = 40$ (100%) Baseline age = 43.0 (±9.0) Baseline BMI = 44.3 (±4.7)	GByp	5	FBG HDL TG SBP DBP
Angrisani L. et al. 2015	Group 1 (Baseline BMI ≤ 50) $N = 61$ $M = 17$ (28.0%) $F = 44$ (72.0%) Baseline age = 40.0 (±10.0) Group 2 (Baseline BMI > 50) $N = 44$ $M = 21$ (48.0%) $F = 23$ (52.0%) Baseline age = 38.0 (±11.0)	SG	5	FBG
Artero A. et al. 2017	$N = 79$ $M = .$ $F = .$ Baseline age = 37.3 (±9.8) Baseline BMI = 55.0 (±10.2)	RYGB	10	FBG HDL TG

(Continues)

TABLE 1 | (Continued)

Author, year	Baseline population	Intervention	Follow-up (years)	Outcomes
Ballesteros-Pomar M. et al. 2015	$N = 299$ $M = 71$ (23.9%) $F = 228$ (76.1%) Baseline age = 43.0 (±10.7) Baseline BMI = 50.1 (±7.2)	BPD	5 to10	FBG HDL TG SBP DBP
Bhandari M. et al. 2019	Group GByp $N = 82$ $M = 44$ (53.6%) F = 38 (46.4%) Baseline age = 44.1 (±10.5) Baseline BMI = 44.0 (±7.2) Group RYGB $N = 90$ $M = 60$ (66.7%) $F = 30$ (33.3%) Baseline age = 44.0 (±10.9) Baseline BMI = 46.0 (±6.9)	GByp RYGB	5	Hypertension[2]
Bhasker A. et al. 2014	$N = 106$ $M = 43$ (40.6%) $F = 63$ (59.4%) Baseline age = 50.3 (±9.1) Baseline BMI = 45.0 (±7.9)	RYGB	5	HDL TG
Busetto L. et al. 2014	$N = 318$ $M = 58$ (18.2%) $F = 260$ (81.8%) Baseline age = 38.6 (±10.4) Baseline BMI = 46.7 (±7.2)	GBand	≥ 10	FBG HDL TG SBP DBP
Castro M. et al. 2020	Group SG $N = 83$ $M = 20$ (24.1%) $F = 63$ (75.9%) Baseline age = 43.5 (±10.2) Baseline BMI = 45.2 (±9.2) Group RYGB $N = 152$ $M = 32$ (21.1%) $F = 120$ (78.9%) Baseline age = 44.1 (±11.6) Baseline BMI = 43.6 (±8.9) Group GB $N = 123$ $M = 33$ (26.8%) $F = 90$ (73.2%) Baseline age = 42.4 (±11.0) Baseline BMI = 43.8 (±9.2)	SG RYGB GByp	5	FBG HDL TG
Chahal-Kummen et al. 2020	$N = 124$ $M = .$ $F = .$ Baseline age = . Baseline BMI = 45.6 (IQR 42.6 to 50.8)	RYGB	10	MS
Chang D. et al. 2018	$N = 1759$ $M = 533$ (30.3%) $F = 1226$ (69.7%) Baseline age = 35.2 (±10.3) Baseline BMI = 37.9 (±7.7)	SG	5 to10	WC

(Continues)

TABLE 1 | (Continued)

Author, year	Baseline population	Intervention	Follow-up (years)	Outcomes
Dewberry L. et al. 2020	*N* = 13 *M* = 2 (14.0%) *F* = 11 (86.0%) Baseline age = 18.2 (±0.4) Baseline BMI = 51.0 (±8.8)	Gband	5	Low HDL[3] High TG[4]
Elhag W. et al. 2021	*N* = 146 *M* = 74 (50.7%) *F* = 72 (49.3%) Baseline age = 16.5 (±1.3) Baseline BMI = 46.9 (±7.3)	SG	5,7 and9	FBG HDL TG SBP DBP
Gao X. et al. 2021	*N* = 24 *M* = 10 (41.7%) *F* = 14 (58.3%) Baseline age = 47.7 (±5.3) Baseline BMI = 27.6 (±2.3)	RYGB	5	FBG HDL TG WC
Gao X. et al. 2022	Group large pouch *N* = 42 *M* = 19 (45.2%) *F* = 23 (54.8%) Baseline age = 47.3 (±5.3) Baseline BMI = 29.4 (±1.9) Group small pouch *N* = 53 *M* = 20 (47.7%) *F* = 33 (62.3%) Baseline age = 48.1 (±5.0) Baseline BMI = 28.3 (±2.1)	RYGB	6	FBG HDL TG WC
Golomb I. et al. 2015	*N* = 443 *M* = . *F* = . Baseline age = 42.2 (±12.4) Baseline BMI = 43.9 (±6.6)	SG	5	HDL TG
Gómez-Abril A. et al. 2016	*N* = 66 *M* = 13 (19.7%) *F* = 53 (80.3%) Baseline age = 40.8 (±9.8) Baseline BMI = 48.0 (±7.2)	GByp	5	FBG HDL TG
Gottardi L. et al. 2023	*N* = 65 *M* = 13 (20.0%) *F* = 52 (80.0%) Baseline age = 18.6 (±1.3) Baseline BMI = .	SG (65.0%) RYGB (35.0%)	5	FBG HDL TG
Guimarães M. et al. 2021	*N* = 281 *M* = 40 (14.2%) *F* = 241 (85.8%) Baseline age = 42.8 (±10.6) Baseline BMI = 44.4 (±6.1)	RYGB	≥ 10	FBG HDL TG
Heald A. et al. 2024	*N* = 79 *M* = 32 (41.0%) *F* = 47 (59.9%) Baseline age = 51.5 (±8.3) Baseline BMI = 50.8 (±7.1)	GByp (85.0%) Gband (4.0%) SG (10.0%) Other (1.0%)	11.5 (IQR10.3 to 12.1)	HDL TG SBP DBP

(Continues)

TABLE 1 | (Continued)

Author, year	Baseline population	Intervention	Follow-up (years)	Outcomes
Heffron S. et al. 2014	*N* = 46 *M* = . *F* = . Baseline age = 43.8 (±.) Baseline BMI = 35.1 (±2.6)	Gband	5	FBG HDL TG SBP DBP WC
Huang Y. et al. 2020	*N* = 1999 *M* = 840 (42.0%) *F* = 1159 (58.0%) Baseline age = 41.5 (±11.1) Baseline BMI = 38.7 (±7.9)	SG (16.5%) GB (32.6%) RYGB (22.1%) GBand (9.2%) Duodenojejunal bypass with SG (9.7%)	5	FBG HDL TG SBP DBP
Inge T. et al. 2017	*N* = 58 *M* = 21 (26.0%) *F* = 37 (64.0%) Baseline age = 17.1 (±1.7) Baseline BMI = 58.5 (±10.5)	RYGB	8 (range 5.4 to 12.5)	FBG HDL TG SBP DBP
Ji G. et al. 2020	*N* = 52 *M* = 30 (57.7%) *F* = 22 (42.3%) Baseline age = 46.8 (±9.5) Baseline BMI = 27.2 (±3.2)	RYGB	6	FBG HDL
Jimenéz A. et al. 2019	Group RYGB *N* = 381 *M* = 94 (24.7%) *F* = 287 (75.3%) Baseline age = 43.6 (±10.3) Baseline BMI = 46.3 (±5.1) Group SG *N* = 123 *M* = 47 (38.2%) *F* = 76 (61.8%) Baseline age = 44.3 (±11.3) Baseline BMI = 53.7 (±8.1)	RYGB SG	10	FBG HDL TG SBP DBP
Joret M. et al. 2022	*N* = 65 *M* = 14 (21.5%) *F* = 51 (78.5%) Baseline age = . Baseline BMI = 49.9 (±5.3)	DSw	5	HDL TG
Kokkinos A. et al. 2024	*N* = 28 *M* = 5 (17.9%) *F* = 23 (82.1%) Baseline age = 50.3 (±8.1) Baseline BMI = 49.6 (±6.8)	SG (.%) RYGB (.%)	10	FBG HDL TG SBP DBP WC
Lee M. et al. 2015	*N* = 157 *M* = 52 (33.1%) *F* = 105 (66.9%) Baseline age = 35.7 (range 18 to 64) Baseline BMI = 39.8 (range 21.5 to 61.7)	RYGB (.%) GB (.%) SG (.%)	5	FBG TG SBP DBP WC
Lemanu D. et al. 2014	*N* = 55 *M* = 10 (18.2%) *F* = 45 (81.8%) Baseline age = 46.9 (95%CI 47.5 to 52.3) Baseline BMI = 50.7 (95%CI 49.0 to 52.4)	SG	5	HDL TG

(Continues)

TABLE 1 | (Continued)

Author, year	Baseline population	Intervention	Follow-up (years)	Outcomes
Magno F. et al. 2018	*N* = 323 *M* = 90 (28.0%) *F* = 233 (72.0%) Baseline age = 38.0 (±10.0) Baseline BMI = 45.4 (±6.1)	RYGB	5	FBG HDL TG
Mingrone G. et al. 2015	Group RYGB *N* = 20 *M* = . *F* = . Baseline age = . Baseline BMI = 44.0 (±4.6) Group BPD *N* = 20 *M* = . *F* = . Baseline age = . Baseline BMI = 44.7 (±7.7)	RYGB BPD	5	FBG HDL TG SBP DBP WC
Moriconi D. et al. 2024	Group T2DM-PA *N* = 15 *M* = 9 (60.0%) *F* = 6 (40.0%) Baseline age = 54.0 (±9.0) Baseline BMI = 46.0 (±6.1) Group T2DM-noPA *N* = 61 *M* = 14 (23.0%) *F* = 47 (77.0%) Baseline age = 56.0 (±7.0) Baseline BMI = 46.2 (±6.0) Group noT2DM-PA *N* = 29 *M* = 5 (27.0%) *F* = 24 (83.0%) Baseline age = 45.0 (±10.0) Baseline BMI = 43.2 (±5.7) Group noT2DM-noPA *N* = 43 *M* = 13 (30.0%) *F* = 30 (70.0%) Baseline age = 51.0 (±12.0) Baseline BMI = 45.3 (±7.4)	RYGB	5	FBG HDL TG
Obeid N. et al. 2015	*N* = 328 *M* = 57 (17.0%) *F* = 271 (83.0%) Baseline age = 41.4 (±9.8) Baseline BMI = 47.5 (±7.1)	RYGB	5 to 10	FBG HDL TG SBP DBP
Otake R. et al. 2022	*N* = 230 *M* = 127 (55.2%) *F* = 103 (44.8%) Baseline age = 46 (range 19 to 72) Baseline BMI = 40.6 (range 30.3 to 77.7)	SG	5	FBG HDL TG SBP DBP
Pajecki D. et al. 2007	*N* = 75 *M* = 18 (24.0%) *F* = 57 (76.0%) Baseline age = . Baseline BMI = 56.7 (±10.0)	RYGB	5 to 9	HDL TG

(Continues)

TABLE 1 | (Continued)

Author, year	Baseline population	Intervention	Follow-up (years)	Outcomes
Pata G. et al. 2012	$N = 874$ $M = 397$ (45.4%) $F = 477$ (54.6%) Baseline age = 39 (range 18 to 69) Baseline BMI = 52.0 (IQR 49.0 to 54.4)	BPD with DSw	5 to 15	FBG TG
Pontirolli A. et al. 2009	Group GBand $N = 78$ $M = 10$ (22.8%) $F = 68$ (87.2%) Baseline age = 44.2 (±1.2) Baseline BMI = 44.8 (±0.7) Group BPD $N = 23$ $M = 7$ (30.4%) $F = 16$ (69.6%) Baseline age = 45.3 (±1.8) Baseline BMI = 48.6 (±1.4)	Gband BPD	> 5	FBG TG SBP DBP
Salminen P. et al. 2017	Group RYGB $N = 121$ $M = 39$ (32.8%) $F = 80$ (67.2%) Baseline age = 48.4 (±9.3) Baseline BMI = 46.4 (±5.9) Group SG $N = 121$ $M = 34$ (28.1%) $F = 87$ (71.9%) Baseline age = 48.5 (±9.6) Baseline BMI = 45.5 (±6.2)	RYGB SG	5	FBG HDL TG
Sánchez-Pernaute A. et al. 2022	$N = 164$ $M = 65$ (39.6%) $F = 99$ (60.4%) Baseline age = 47.0 (range 22 to 71) Baseline BMI = 45.8 (range 34.0 to 67.0)	Duodenoileal bypass with SG	5 to 10	FBG HDL TG
Sarkis R. et al. 2018	$N = 7$ $M = 2$ (28.6%) $F = 5$ (71.4%) Baseline age = 51.0 (±6.0) Baseline BMI = 36.7 (±1.6)	IB	5	TG
Schauer P. et al. 2017	Group RYGB $N = 50$ $M = 21$ (42.0%) $F = 29$ (58.0%) Baseline age = 48.3 (±8.4) Baseline BMI = 37.0 (±3.3) Group SG $N = 50$ $M = 21$ (42.0%) $F = 29$ (58.0%) Baseline age = 47.9 (±8.0) Baseline BMI = 36.2 (±3.9)	RYGB SG	5	FBG HDL TG SBP DBP
Schiavon C. et al. 2024	$N = 50$ $M = 12$ (24.0%) $F = 38$ (76.0%) Baseline age = 43.8 (±9.2) Baseline BMI = 36.9 (±2.7)	RYGB	5	SBP DBP

(Continues)

TABLE 1 | (Continued)

Author, year	Baseline population	Intervention	Follow-up (years)	Outcomes
Steffen R. et al. 2009	*N* = 388 *M* = 89 (33.0%) *F* = 299 (77.0%) Baseline age = 42.7 (±10.0) Baseline BMI = 42.6 (±4.0)	GBand	7	MS
Sundbom M. et al. 2017	*N* = 26,119 *M* = 6323 (24.2%) *F* = 19,796 (75.8%) Baseline age = . Baseline BMI = 42.8 (±5.5)	RYGB	5	FBG HDL TG SBP DBP WC
Tan M. et al. 2022	*N* = 65 *M* = 30 (46.2%) *F* = 35 (53.8%) Baseline age = 54.2 (±11.2) Baseline BMI = 45.8 (±8.8)	SG (80.0%) GBand (10.8%) GB (9.2%)	5 to 6	FBG TG SBP DBP
Wentworth J. et al. 2017	*N* = 60 *M* = 29 (48.3%) *F* = 31 (51.7%) Baseline age = 50.0 (IQR 44.0 to 53.0) Baseline BMI = 40.2 (IQR 37.5 to 47.1)	GBand	10	FBG HDL TG SBP DBP WC
Xing Y. et al. 2024	*N* = 37 *M* = 14 (37.8%) *F* = 23 (62.2%) Baseline age = 45.3 (±10.7) Baseline BMI = 30.9 (±5.0)	RYGB	5	FBG HDL TG
Zetu C. et al. 2020	*N* = 60 *M* = 10 (16.7%) *F* = 50 (83.3%) Baseline age = 41.7 (±12.5) Baseline BMI = 44.6 (±9.9)	SG	5	HDL TG WC

Note: Complete information on the included studies is reported in Table S3.
Abbreviations: ±, standard deviationl; 1, < 40 mg/dL (male)/ < 50 mg/dL (female); 2, hypertension was defined as blood pressure > 140/90 mmHg; 3, < 40 mg/dL; 4, > 130 mg/dL; 95%CI: 95% confidence interval; BMI, body mass index (kg/m^2); BPD, biliopancreatic diversion; DBP, diastolic blood pressure (mmHg); DSw, duodenal switch; F, female; FBG, fasting blood glucose (mg/dL); GBand, gastric banding; GByp, gastric bypass; HDL, high-density lipoprotein cholesterol (mg/dL); IB, intestinal bipartition; IQR, interquartile range; M, male; MS, metabolic syndrome; PA, physical activity; RCT, randomized clinical trial; RYGB, Roux-en-y gastric bypass; SBP, systolic blood pressure (mmHg); SG, sleeve gastrectomy; T2DM, type 2 diabetes mellitus; TG, triglycerides (mg/dL); WC, waist circumference (cm).

outcome measurement, rather than to major issues in randomization or selective reporting (Tables S5, S6; Figure S1) [71, 72, 74, 75].

Leave-one-out sensitivity analyses and trim-and-fill method were applied for all meta-analyses with 10 studies or more. No substantial changes in the pooled estimates or improvements in heterogeneity were observed for any of the outcomes (Figures S3–S6).

3.2 | Fasting Blood Glucose

Long-term FBG data were available for 7571 patients across the included studies at follow-up. In cohort studies, the pooled weighted mean difference (WMD) was −22.97 mg/dL (95%CI −27.19 to −18.75 mg/dL; $I^2 = 98.3\%$, Q-test $p < 0.001$), with no significant heterogeneity reduction after BS subgroup analyses (Figure S7). Meta-regression indicated that higher baseline FBG and a greater proportion of male participants were significantly associated with greater post-BS reduction in univariate analyses ($\beta = -0.61$ mg/dL; $p < 0.001$ and $\beta = -65.61$ mg/dL; $p < 0.05$, respectively). However, in the multivariable model, only baseline FBG retained statistical significance ($p < 0.001$), indicating that the postsurgical reduction increased by 0.68 mg/dL for each 1 mg/dL increase in the mean baseline FBG (Table S7). No publication bias was detected (Egger's test $p = 0.153$) (Figure S7). In RCTs, the pooled effect was −44.68 mg/dL (95%CI −67.40 to −21.96 mg/dL; $I^2 = 95.4\%$, Q-test $p < 0.001$) (Figure S9). Stratification by baseline BMI, improved heterogeneity, and revealed a stronger effect in participants with BMI 35–39.9 kg/m^2 (−70.58 mg/dL; 95%CI −84.33 to −56,84 mg/dL; $I^2 = 31.8\%$, Q-test $p = 0.231$) compared to those with BMI 40–49.9 kg/m^2 (−23.36 mg/dL; 95%CI −42.40 to −4.31 mg/dL) (Figure S10). No further subgroup analyses yielded substantial improvements.

3.3 | HDL Cholesterol

HDL cholesterol was assessed in 7477 individuals. In cohort studies, the pooled effect was +10.25 mg/dL (95%CI 8.49 to 12.00 mg/dL), with high heterogeneity ($I^2 = 95.9\%$, Q-test $p < 0.001$) (Figure S11). Each 1 kg/m^2 increase in baseline BMI was significantly associated with greater postsurgical increases in HDL cholesterol ($\beta = 0.48$ mg/dL; $p < 0.001$) at univariable meta-regression analysis (Table S7). The funnel plot was symmetric (Figure S12), and no evidence of small-study effects was detected (Egger's test $p = 0.426$). Subgroup analyses did not reduce heterogeneity. In RCTs, the pooled effect was +11.94 mg/dL (95%CI 8.76 to 15.12 mg/dL), with high heterogeneity ($I^2 = 80.2\%$, Q-test $p < 0.001$), which remained unchanged after subgroup analysis (Figure S13).

3.4 | Triglycerides

This outcome was assessed in 8276 individuals at follow-up. In cohort studies, the pooled WMD was −59.59 mg/dL (95%CI −65.84 to −53.34 mg/dL) with high heterogeneity ($I^2 = 95.8\%$, Q-test $p < 0.001$). Among studies evaluating gastric banding procedures, the effect size was smaller −41.39 mg/dL (95%CI −43.68 to −39.11 mg/dL) but showed no heterogeneity ($I^2 = 0.0\%$, Q-test $p = 0.785$) (Figure S14). Meta-regression analyses identified mean baseline TG as a significant moderator of the pooled WMD, with each 1 mg/dL increase in baseline TG associated with a 0.55 mg/dL reduction in long-term TG levels ($\beta = -0.55$ mg/dL; $p < 0.001$; Table S7). No subgroup analyses reduced heterogeneity. Although the funnel plot appeared symmetrical, a small-study effect was detected (Egger's test $p < 0.001$) (Figure S15). In RCTs, the pooled effect was −54.11 mg/dL (95%CI −72.65 to −35.57 mg/dL), with high heterogeneity ($I^2 = 85.1\%$, Q-test $p < 0.001$) (Figure S16) that persisted after analysis.

3.5 | Waist Circumference

Long-term WC data were available for 2369 patients after follow-up. When analyzed in cohort studies, the pooled WMD was −20.55 cm (95% CI: −28.35 to −12.74 cm; $I^2 = 99.5\%$) (Figure S17). Among the surgical techniques, SG showed the greatest reduction (WMD: −31.14 cm; 95%CI −32.74 to −29.53 cm), followed by mixed procedures (WMD: −26.22 cm; 95%CI −27.93 to −24.51 cm) and gastric banding (WMD: −15.36 cm; 95%CI −17.36 to −12.77 cm). Studies reporting SG and gastric banding outcomes showed no heterogeneity ($I^2 = 0.0\%$, and respective Q-test $p = 0.905$ and Q-test $p = 0.654$). Analyses indicated that baseline WC ($\beta = -0.61$ cm per 1 cm increase, $p < 0.01$), baseline BMI ($\beta = -1.15$ cm per 1 kg/m^2, $p < 0.01$), and male sex proportion ($\beta = +66.24$ cm per 1 unit increase, corresponding to +6.62 cm per 10% increase, $p < 0.05$) were significant moderators of the pooled outcome in univariable meta-regression. However, none of these associations remained significant in the multivariable model (Table S7). Only one RCT was identified for this outcome, which evaluated the effects of RYGB and BPD, reporting a WC reduction of −22.4 ± 9.3 cm and −28.8 ± 12.4 cm, respectively [75].

3.6 | Systolic and Diastolic Blood Pressure

SBP data were available for 18,912 individuals, and DBP data for 18,856. In cohort studies, the pooled WMD for SBP was −9.76 mmHg (95% CI −11.87 to −7.64 mmHg; $I^2 = 98.5\%$; Q-test $p < 0.001$) and for DBP −4.80 mmHg (95% CI −6.62 to −2.99 mmHg; $I^2 = 99.1\%$; Q-test $p < 0.001$) (Figure S18; Figure S20). Univariable meta-regression showed that for each 1 mmHg increase in baseline SBP and DBP values were significantly associated with greater post-BS reductions (SBP: $\beta = -0.59$ mmHg, $p < 0.001$; DBP: $\beta = -0.56$ mmHg, $p < 0.01$). However, in the multivariable meta-regression model, the association remained significant only for SBP, with a β coefficient of −0.40 mmHg per each 1 mmHg increase in baseline SBP ($p < 0.05$) (Table S7). Subgroup analyses did not reduce heterogeneity. Funnel plots were symmetrical (Figure S19; Figure S21), though a small-study effect was identified for SBP (Egger's test $p = 0.032$), but not for DBP (Egger's test $p = 0.144$). In the RCTs, the pooled effect was −7.82 mmHg (95%CI −13.19 to −2.44 mmHg) for SBP and −6.48 mmHg (95% CI −8.93 to −4.02 mmHg) for DBP, both indicating moderate-to-high heterogeneity (SBP: $I^2 = 79.5\%$, Q-test $p < 0.001$; DBP: $I^2 = 63.8\%$, Q-test $p = 0.017$) (Figure S22; Figure S23). Subgroup analyses based on surgery type and baseline BMI reduced heterogeneity but did not significantly alter the overall effect measure (Figures S22–S25).

4 | Discussion

This systematic review and meta-analysis provides comprehensive evidence of the long-term (≥ 5 years) metabolic benefits of BS across key components of MetS. Significant and sustained improvements across all six core components FBG, TG, HDL, WC, SBP, and DBP. These results support an association between BS and sustained long-term improvements in metabolic control and cardiovascular risk reduction.

The long-term metabolic benefits of BS are likely mediated by a combination of sustained hormonal and metabolic adaptations, as well as behavioral changes [8–10, 70, 76]. Among the most consistently supported mechanisms are persistent changes in gut hormone secretion—particularly GLP-1, glucagon-like peptide-1 (GLP-1), postprandial levels of peptide YY (PYY), and cholecystokinin (CCK)—and bile acid signaling, which contribute to improved insulin sensitivity, glycemic control, and lipid metabolism over time [10, 70]. These effects are complemented by durable alterations in energy balance and appetite regulation, which support long-term weight maintenance and metabolic stability [8, 56, 77–79]. Although changes in gut microbiota composition have also been described, their independent contribution to long-term metabolic outcomes remains less clearly defined and is likely context-dependent. Overall, the available evidence suggests that the durability of metabolic improvements after BS is driven primarily by sustained endocrine and metabolic adaptations rather than by short-term weight loss alone. A conceptual summary of the main pathways potentially contributing to the sustained metabolic benefits of BS is provided in Figure 3.

FBG exhibited one of the most pronounced reductions among all outcomes, and meta-regression showed that individuals with higher baseline FBG experienced greater improvements,

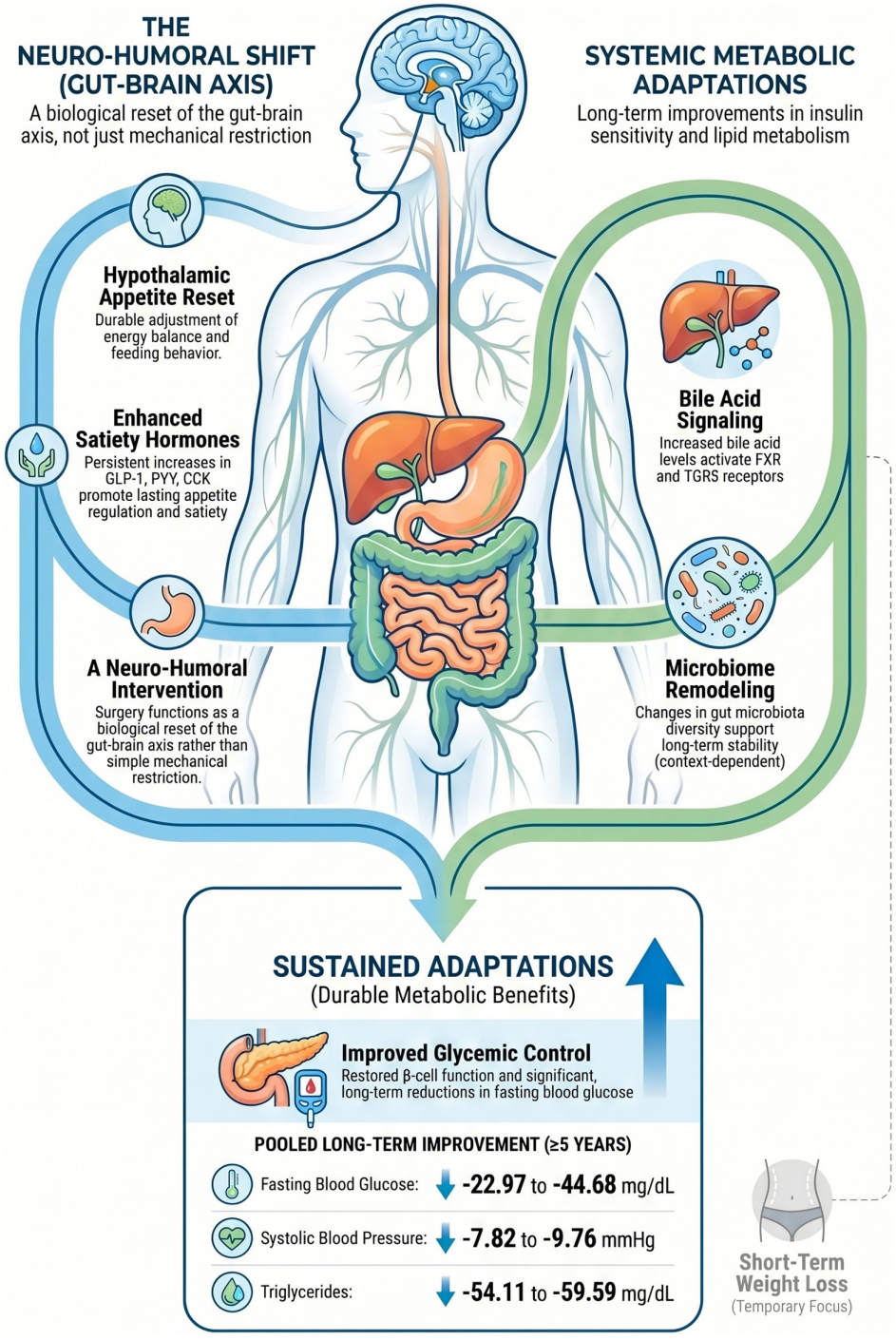

FIGURE 3 | Conceptual model of the main mechanisms contributing to sustained metabolic benefits after bariatric surgery. Bariatric surgery may induce long-term metabolic improvements through multiple interconnected pathways, including altered gut hormone secretion (e.g., GLP-1, PYY, and CCK), changes in bile acid signaling, modifications of gut microbiota composition, reduced appetite and energy intake, and improved insulin sensitivity and lipid metabolism. These mechanisms may jointly contribute to durable improvements in glycemic control, lipid profile, blood pressure, and waist circumference. Abbreviations: CCK, cholecystokinin; FXR, farnesoid X receptor; GLP-1, glucagon-like peptide-1; mg/dL, milligrams per decilitre; mmHg, millimeters of mercury; PYY, peptide YY; TGR5, Takeda G protein-coupled receptor 5; β-cell, pancreatic beta cell.

suggesting that BS may act as a potent corrective intervention in patients with poor glycemic control. These findings are consistent with previous evidence indicating that BS can induce remission of T2DM, especially when performed earlier in the disease trajectory [8, 9, 76]. Moreover, subgroup analyses revealed that participants with BMI between 35 and 39.9 kg/m^2 benefited the most, challenging traditional BMI-based eligibility thresholds and supporting more personalized patient selection criteria.

From a clinical perspective, the magnitude of the observed long-term metabolic improvements is meaningful. A sustained reduction in FBG of approximately 20–45 mg/dL is within a range that

has been associated with substantial reductions in microvascular complications and increased likelihood of diabetes remission, as demonstrated in landmark research [77, 78]. Similarly, long-term reductions in SBP of approximately 8–10 mmHg are clinically relevant, as they are associated with meaningful decreases in cardiovascular morbidity and mortality and may translate into reduced need for antihypertensive therapy, in line with established blood pressure risk thresholds [79].

HDL levels improved by more than 10 mg/dL. These findings confirm that BS has a positive impact on HDL, a key protective factor against atherosclerosis. Interestingly, the magnitude of HDL increase appears to be directly influenced by baseline BMI. After BS, patients with higher baseline BMI may experience more substantial metabolic improvements—such as increased insulin sensitivity, reduced inflammation, and improved lipid metabolism—which can lead to greater increases in HDL levels. Their greater visceral fat loss may also help restore HDL production and reduce its catabolism [56, 80, 81]. Similarly, TG levels were significantly reduced, supporting a long-term association between BS and improved atherogenic dyslipidemia [56, 80, 81]. Meta-regression identified baseline TG levels as a significant predictor of response. Notably, studies involving gastric banding procedures yielded smaller effects, underscoring the superior efficacy of malabsorptive or mixed procedures in lipid regulation [35, 48, 63, 68]. However, the presence of a small-study effect suggests that smaller trials might have overestimated the benefit, warranting cautious interpretation.

Among all anthropometric outcomes, WC showed the highest variability. The pooled WMD of −20.55 cm indicates a substantial visceral fat reduction, a critical component of MetS. Stratified analysis revealed SG to be the most effective procedure, followed by mixed techniques and gastric banding. However, the paucity of RCTs on this outcome limits the strength of causal inference.

Several considerations emerge from our findings. First, meta-regression analyses suggest that higher baseline metabolic values were associated with greater postsurgical improvements, reflecting a potential homeostatic compensatory response in individuals with more severe metabolic imbalance. Second, although our review included heterogeneous surgical techniques, RYGB was overrepresented relative to SG, which is currently the most performed procedure worldwide [82]. This likely reflects historical trends in surgical practice and highlights the need for more long-term data on SG, especially given its different physiological effects and safety profile [82]. This may shift in future years as more data from recent SG cohorts become available. Third, the long-term metabolic benefits demonstrated in this review must be interpreted in light of real-world access to BS. Although BS has been shown to be cost-effective over the long term [83, 84], socioeconomic and health system factors continue to influence who is able to benefit from these sustained metabolic improvements. Population-based evidence indicates disparities in access by sex, income, and healthcare setting, suggesting that the long-term reductions in cardiometabolic risk observed in clinical studies may not be equitably realized across all patient groups [85, 86]. From a translational perspective, these findings underscore the importance of aligning clinical evidence with health policy and service organization to ensure broader and more equitable access to BS and its long-term metabolic benefits. Fourth, moderate-to-substantial heterogeneity was observed across several outcomes and was only partly explained by subgroup and meta-regression analyses. This likely reflects differences in patient populations, surgical procedures, follow-up duration, outcome measurement, and postoperative care. Therefore, pooled estimates should be interpreted as average long-term metabolic changes across diverse clinical settings. Nevertheless, pooling was considered appropriate given the common clinical question, comparable endpoints, and generally consistent direction of effect across studies. Fifth, the present work focused on metabolic surrogate endpoints, including glycemic, lipid, anthropometric, and blood pressure measures, rather than hard clinical outcomes such as major adverse cardiovascular events, cardiovascular mortality, or all-cause mortality. Although these outcomes are clinically meaningful and closely related to cardiometabolic risk, they should not be interpreted as direct evidence of reduced clinical events. Future long-term studies should integrate metabolic endpoints with hard clinical outcomes to better define the overall clinical impact of BS. A further methodological consideration is that several included studies were based on pre–post comparisons and did not include a nonsurgical comparator group. Accordingly, the pooled estimates should be interpreted as long-term metabolic changes observed after BS, rather than as definitive causal effects. Although the inclusion of randomized trials, subgroup analyses, and sensitivity analyses strengthens the evidence base, residual confounding related to lifestyle modifications, medication adjustments, regression to the mean, or differences in long-term follow-up care cannot be fully excluded. Moreover, ethnic, genetic, and epigenetic factors may contribute to inter-individual variability in metabolic response but remain largely unaccounted for in the current evidence base.

Evidence suggests that preoperative weight loss through lifestyle interventions and pharmacotherapy is associated with better postoperative outcomes, potentially due to improvements in glycemic control, lipid profile, and body composition [83, 87–89]. However, a recent systematic review by MacVicar et al. [84] identified inconsistent evidence on whether the magnitude of preoperative weight loss consistently correlates with long-term outcomes.

Although the mechanisms underlying weight loss and comorbidity resolution after BS are well documented, several hypotheses still require confirmation in human studies [8–10, 85, 86, 90]. Dietary restriction, reduced gastric volume, and hormonal changes—such as decreased orexigenic and increased anorexigenic signals—contribute to reduced energy intake [9, 10, 85, 86, 90]. Postprandial levels of peptide YY GLP-1, and CCK likely influence central appetite regulation via circulatory and vagal pathways [90].

Emerging evidence also highlights the role of gut microbiota [85, 86, 90, 91]. Both RYGB and SG significantly alter microbial composition and bile acid profiles, with RYGB inducing more pronounced shifts. Specific genera involved in secondary bile acid metabolism, such as *Blautia* and *Veillonella*, may play a role in glucose and HDL regulation and have been proposed as potential predictors of T2DM remission [86, 90].

This is one of the most comprehensive evaluations of long-term metabolic outcomes following BS to date, encompassing over 37,000 participants from over 50 studies. Methodologically, we

adhered to rigorous PRISMA guidelines, employed multiple sensitivity analyses, and used meta-regression to explore heterogeneity. Nevertheless, limitations remain. These include variability in outcome definitions, missing data on concomitant pharmacologic or lifestyle interventions, limited information on surgical technique nuances (e.g., pouch size, limb length, or whether the procedure was a primary, revisional, or conversion surgery), and residual confounding. Additionally, the persistence of high heterogeneity despite subgroup and regression analyses suggests substantial individual variability in response to BS. This variability is likely influenced by factors not captured in the current evidence base, including genetics, epigenetics, or behavioral determinants.

5 | Conclusion

Bariatric surgery was associated with sustained long-term improvement in all major components of metabolic syndrome, with consistent benefits observed across FBG, lipid profile, blood pressure, and WC. Although the magnitude of improvement varied across procedures and populations, the overall direction of effect was uniformly favorable. However, considerable heterogeneity remains, partly due to differences in surgical techniques, patient profiles, and study methodologies. Future research should prioritize the standardized reporting of concurrent interventions, follow-up protocols, and metabolic endpoints to improve comparability and refine pooled estimates. Notably, although SG is currently the most widely performed bariatric procedure globally, it remains underrepresented in the available long-term evidence. This limits the generalizability of current findings to contemporary surgical practice and highlights the urgent need for dedicated, high-quality longitudinal studies on SG to inform clinical decision-making and health policy.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Table S1:** Search strategy. **Table S2:** PICOS framework and inclusion/exclusion criteria. **Table S3:** Detailed baseline and long-term outcomes of the included studies. **Table S4:** Set of formulas used for parameters' conversion. **Table S5:** Risk of bias (RoB) of randomized controlled trials. **Table S6:** Risk of bias (RoB) of nonrandomized studies (cohort studies). **Table S7:** Random-effects meta-regression results of the long-term effects of bariatric surgeries in metabolic syndrome indicators. **Figure S1:** Visual representation of risk of bias using ROB-2 Tool. **Figure S2:** Leave-one-out sensitivity analysis plot of fasting blood glucose in cohort studies. **Figure S3:** Leave-one-out sensitivity analysis plot of tryglicerides levels in cohort studies. **Figure S4:** Leave-one-out sensitivity analysis plot of waist circumference in cohort studies. **Figure S5:** Leave-one-out sensitivity analysis plot of systolic blood pressure in cohort studies. **Figure S6:** Leave-one-out sensitivity analysis plot of diastolic blood pressure in cohort studies. **Figure S7:** Long-term effects of bariatric surgery on fasting blood glucose: meta-analysis of cohort studies (Forest plot). **Figure S8:** Funnel plot: assessment of publication bias for long-term fasting blood glucose changes after bariatric surgery (cohort studies). **Figure S9:** Long-term effects of bariatric surgery on fasting blood glucose: meta-analysis of fasting blood glucose. **Figure S10:** Long-term effects of bariatric surgery on fasting blood glucose, stratified by baseline BMI: meta-analysis of random clinical trials (Forest plot). **Figure S11:** Long-term effects of bariatric surgery on high-density lipoprotein: meta-analysis. Of cohort studies (Forest plot). **Figure S12:** Funnel plot: assessment of publication bias for long-term high-density lipoprotein changes after bariatric surgery (cohort studies). **Figure S13:** Long-term effects of bariatric surgery on high-density lipoprotein: meta-analysis of random clinical trials (Forest plot). **Figure S14:** Long-term effects of bariatric surgery on tryglicerides levels: meta-analysis of cohort studies (Forest plot). **Figure S15:** Funnel plot: assessment of publication bias for long-term tryglicerides levels changes after bariatric surgery (cohort studies). **Figure S16:** Long-term effects of bariatric surgery on tryglicerides levels: meta-analysis of random clinical trials (Forest plot). **Figure S17:** Long-term effects of bariatric surgery on waist circumference: meta-analysis of cohort studies (Forest Plot). **Figure S18:** Long-term effects of bariatric surgery on systolic blood pressure: meta-analysis of cohort studies (Forest plot). **Figure S19:** Funnel plot: assessment of publication bias for long-term systolic blood pressure changes after bariatric surgery (cohort studies). **Figure S20:** Long-term effects of bariatric surgery on diastolic blood pressure: meta-analysis of cohort studies (Forest plot). **Figure S21:** Funnel plot: assessment of publication bias for long-term diastolic blood pressure changes after bariatric surgery (cohort studies). **Figure S22:** Long-term effects of bariatric surgery on systolic blood pressure: meta-analysis of random clinical trials (Forest plot). **Figure S23:** Long-term effects of bariatric surgery on systolic blood pressure, stratified by baseline BMI: meta-analysis of random clinical trials (Forest plot). **Figure S24:** Long-term effects of bariatric surgery on diastolic blood pressure: meta-analysis of random clinical trials (Forest plot). **Figure S25:** Long-term effects of bariatric surgery on diastolic blood pressure, stratified by baseline BMI: meta-analysis of random clinical trials (Forest plot).