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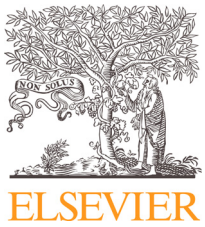

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Review

Metabolic changes after surgical fat removal: A dose-response meta-analysis

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KEYWORDS
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Lipid profile

Abstract *Background:* Bariatric surgery averts obesity-induced insulin resistance and the metabolic syndrome. By contrast, surgical fat removal is considered merely an esthetic endeavor. The aim of this article was to establish whether surgical fat removal, similar to bariatric surgery, exerts measurable, lasting metabolic benefits.
Methods: PubMed, Embase, and Scopus were searched using the Polyglot Search Translator to find studies examining quantitative expression of metabolic markers. Quality assessment was done using the MethodologicAl STandard for Epidemiological Research scale. The robust-error meta-regression model was employed for this synthesis.
Results: Twenty-two studies with 493 participants were included. Insulin sensitivity improved gradually with a maximum reduction in fasting insulin and homeostatic model assessment for insulin resistance of 17 pmol/L and 1 point, respectively, at postoperative day 180. Peak metabolic benefits manifest as a reduction of 2 units in body mass index, 3 kg of fat mass, 5 cm

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of waist circumference, 15 μg/L of serum leptin, 0.75 pg/ml of tumor necrosis factor-alpha, 0.25 mmol/L of total cholesterol, and 3.5 mmHg of systolic and diastolic blood pressure that were observed at day 50 but were followed by a return to preoperative levels by day 180. Serum high-density lipoproteins peaked at 50 days post-surgery before falling below the baseline. No significant changes were observed in lean body mass, serum adiponectin, resistin, interleukin-6, C-reactive protein, triglyceride, low-density lipoproteins, free fatty acids, and fasting blood glucose.

Conclusion: Surgical fat removal exerts several metabolic benefits in the short term, but only improvements in insulin sensitivity last beyond 6 months.

Contents

Introduction

Obesity is a global health crisis and, after smoking, the leading cause of avoidable death in the developed world. Comorbidities include diabetes mellitus, hypertension, obstructive sleep apnea, ischemic heart disease, cancer, osteoarthritis, and depression.[1] Lifestyle changes are largely ineffective in the long term, and there are no efficacious pharmacological interventions. Bariatric surgery aims to induce weight loss by altering intestinal absorption and/or inducing changes in perceived satiety. Categories include gastric bypass, gastric banding, and pancreatico-biliary diversion. The efficacy of bariatric surgery in the management of morbid obesity is well established.[2]

While bariatric surgery addresses the physiological root causes of obesity, surgical fat removal (SFR) tackles the physical manifestations.[3] Options for SFR include en-bloc excision of skin and fat to the level of the muscle fascia, known as body contouring surgery (e.g., abdominoplasty, belt lipectomy, brachioplasty, thigh lift, and breast reduction) and the percutaneous avulsion and aspiration of fat (e.g., liposuction).[4] Bariatric surgery-induced weight loss results in the depletion of fat stores from both subcutaneous deposits and the viscera (accounting for approximately 20% of fat stores),[5] while SFR selectively depletes the subcutaneous stores.

Fat is an endocrine organ.[6] The long-term metabolic impact of fat loss by bariatric surgery is well documented.[2,7] Attempts have been made to evaluate the comparable metabolic impact of selective loss of subcutaneous fat,[8–12] but uncertainties persist owing to the heterogeneity of variables and study parameters. It is important to seek clarity

here, for, while the metabolic benefits of bariatric surgery are well established, SFR continues to be considered cosmetic in nature and subject to health-care rationing. This article describes a systematic review and dose–response meta-analysis (DRMA) of observational studies pertaining to the metabolic impact of body contouring surgery with a view to establishing how these procedures impact patient physiology over time.

Methods

Search strategy

A search string was initially designed in PubMed, then translated and run in Embase and Scopus using the Polyglot Search Translator.[13] The search string was designed by an experienced information specialist and was run across all databases on 8 November 2021. The search string was comprised of both medical subject heading terms and free-text terms. Additionally, the online trials register ClinicalTrials.gov and the national research register were scrutinized for completed, discontinued, and ongoing trials relating to body contouring surgery and physiological and/or metabolic parameters. The search strategy was performed in accordance with the Cochrane Highly Sensitive Search Strategy guideline in the *Cochrane Handbook for Systematic Reviews of Interventions*.[14] The review is reported in line with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA). Full search strings for all databases and PRISMA checklist are available in Supplementary Figures 1 and 2.

Inclusion criteria

Papers were included if they provided quantitative data permitting analyses of the effect of SFR (abdominoplasty or suction lipectomy) on physiology and/or metabolism. Only human studies were considered. No date, language, or publication limits were applied to the search.

Exclusion criteria

Nonhuman (in vivo) studies were excluded from consideration as were studies that used non-SFR procedures.

Quality assessment

The quality assessment of the eligibly included articles was independently done by two reviewers using the MethodologicAl STandard for Epidemiological Research (MASTER) scale.[15] This scale evaluates each included study against 36 safeguards across seven domains that, if present, may mitigate systematic error in the trial. Then, a quality rank for each assessed article was computed and reported qualitatively. The MASTER scale provided a unified framework for the assessment of the methodological quality of quasi-experimental and randomized controlled trials included in this synthesis.

Outcome measures

The outcome measures sought encompassed six domains. These included anthropometrics/body composition, serum adipokines, inflammatory cytokines, glucose homeostasis, lipid profile, and blood pressure. Data units were unified to the Systeme International d'Unites (SI) units. Specifically, the quantitative data extracted (before and after SFR) included the following:

1. Anthropometrics/ body composition: body mass index (BMI), fat mass (FM), lean body mass (LBM), and waist circumference (WC).
2. Serum adipokines: leptin, adiponectin, and resistin.
3. Markers of glucose homeostasis: fasting blood glucose (FBG), fasting insulin, and homeostatic model assessment for insulin resistance (HOMA-IR).
4. Inflammatory cytokines: tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and C-reactive protein (CRP).
5. Lipid profile: low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglycerides (TG), total cholesterol (TC), and free fatty acids (FFA).
6. Blood pressure: systolic blood pressure (SBP) and diastolic blood pressure (DBP).

Other metabolic variables that were reported in less than 5 studies were excluded, such as waist–hip ratio, body fat percentage, free FM, bone mineral content, IL-10, 2-hour postprandial glucose, very low-density lipoprotein-cholesterol, whole-body glucose disposal, glucose oxidative metabolism, nonoxidative glucose metabolism, lipid oxidative metabolism, and glycerol.

Data extraction

Data were retrieved from all full-text articles by two authors. Where necessary, clarification was sought with the senior author (SD).

Statistical methods

To establish an "average" dose–response relationship between the outcome parameters (metabolic changes) and time based on the data of all available studies, the robust-error meta-regression (REMR) model was employed in this study.[16] This is a one-stage approach that treats each study as a cluster and uses robust error variance to address the potential correlations among the within-study effects as these effects share the same reference within the study. A nonlinear curve was fitted using restricted cubic splines with three knots. The Wald test was used to test potential nonlinearity by assuming the coefficient of the nonlinear terms was zero. All analyses were performed using the *remr* module in Stata version 15, College Station, TX, USA.

Results

The literature review yielded a total of 444 studies. Duplicate studies were excluded, leaving 258 studies, of which

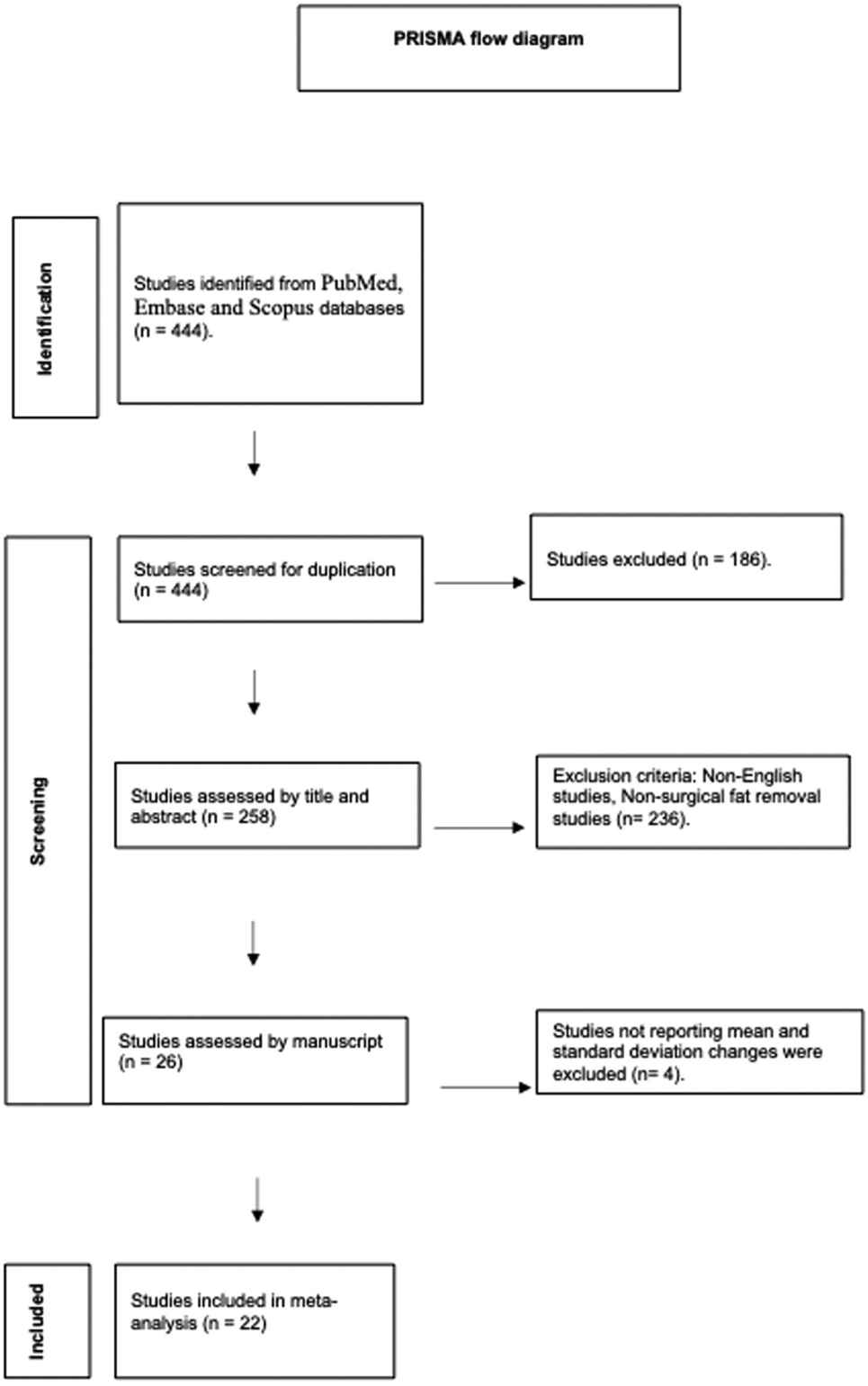

Figure 1 PRISMA flow diagram for selection of studies.

236 were excluded by abstract review. Eventually 22 studies with a total of 493 participants were selected as relevant to this synthesis.[17-38] The conduct of the literature review is summarized in Figure 1.

Characteristics of included studies

Characteristics of the selected studies are summarized in Table 1 and include study identifier, country, design, num-

Table 1 Characteristics of included studies.

Study	Country	Study design	No. of sub-jectss	Population (gender, age, comorbidities)	Baseline BMI ($kg/m^{2)}$	Type of SFR	Outcome measures	Follow-up time points (days)	Average fat mass (kg) or lipoaspirate (L) removed
Vinci et al., 2016	Italy	Case-control	13	-males - age (18-55) yrs	30.3 ± 3.06	abdomino-plasty	Adiponectin, IL6, CRP, TNF-a, FBG, LDL, HDL, TG, TC	0 40	NR
Gibas-dorna et al., 2016	Poland	Case-control	17	-males - age (37.15 ± 9.60) yrs −35% are diabetic type 2	29.16 ± 4.02	lipo-suction	BMI, WC, leptin, adiponectin, FBG, insulin, HOMA-IR	0 60 180	2.208 ± 0.562 L
Cuomo et al., 2015	Italy	Quasi-experiment	64	-females -age (32–48) yrs	33.44 ± 2.3	abdomino-plasty	Leptin, adiponectin, resistin, FBG, insulin, TG, TC	0 180 360	1.6 kg
Solis et al., 2014	Brazil	RCT	18	-females -age (20 −35) yrs	23.1 ± 1.6	lipo-suction	FM, LBM, leptin, adiponectin, IL6, TNF-a, FBG, insulin.	0 60 180	1.240 ± 0.363 L
Ramos-gallardo et al., 2013	Mexico	Quasi-experiment	26	-female -age (26 −56 yrs - all are dyslipidemic	27.4 ± 1.1	abdomino-plasty	BMI, FBG, insulin, HOMA-IR, LDL, HDL, TG, TC	0 90	1.7 kg
Benatti et al., 2012	Brazil	RCT	18	-females -ages (20–35) yrs	23.2 ± 1.3	lipo-suction	FM, LBM	0 60 180	1.240 ± 0.363 L
Yabarra et al., 2008	Spain	Quasi-experiment	20	−18 female −2 males -age (24 −52 yrs	19.8 -36	lipo-suction	BMI, WC, adiponectin, CRP, FBG, insulin, HOMA-IR, LDL, HDL, TG, TC, FFA, SBP, DBP	0 120	5.494 ± 5.297 L
Mohammed et al., 2008	USA	Quasi-experiment	7	-females - 43% diabetic type 2	39± 2	lipo-suction	BMI, FM, FBG, HOMA-IR, LDL,HDL, TG, SBP, DBP	0 70 189	18 L
Robles-cervantes et al., 2007	Mexico	RCT	6	-females -age (30-40) yrs	31.9 ± 1.2	lipo-suction	Leptin, FBG, HDL, TG, TC	0 30	NR

(*continued on next page*)

Table 1 (*continued*)

Study	Country	Study design	No. of sub-jectss	Population (gender, age, comorbidities)	Baseline BMI ($kg/m^{2)}$	Type of SFR	Outcome measures	Follow-up time points (days)	Average fat mass (kg) or lipoaspirate (L) removed
Chang et al., 2007	USA	Quasi-experiment	15	-females -age (21 - 39) yrs	18- 25	lipo-suction	Leptin, adiponectin, IL6, CRP	0 1	NR
Martinez-abundis et al., 2007	Mexico	RCT	6	-females -age (20 - 50) yrs	30.7 ± 0.9	abdomino-plasty	Leptin, LDL, HDL, TG, TC	0 21	3.2 kg
Busetto et al., 2006	Italy	Quasi-experiment	15	- females -pre-menopausal	30.7 - 53.6	lipo-suction	FM,LBM, leptin, adiponectin, resistin, IL6, CRP, FBG, insulin, HOMA-IR, FFA	0 1 3 28 180	16.3 ± 4.3 L
Hong et al., 2006	Korea	Quasi-experiment	11	-age (19 —40) yrs	23.8 ± 4.4	lipo-suction	BMI, LDL, HDL, TC	0 60	6790 L
Andrea et al., 2005	Italy	Quasi-experiment	123	-females -age (32 - 40) yrs	32.8 ± 0.8	lipo-suction	BMI, leptin, adiponectin, resistin, IL6, TNF-a, FBG, insulin, HOMA-IR, TG, TC, FFA, SBP, DBP	0 21 90	4.984 ± 0.821 L
Rizzo et al., 2005	Italy	Quasi-experiment	20	-females -age (25–40) yrs	31·1 ± 0·7	abdomino-plasty	BMI, FM, leptin, adiponectin, resistin, IL6, TNF-a, FBG, insulin, TG, TC, FFA, SBP, DBP	0 40	2·3 ± 0·2 kg
Davis et al., 2005	USA	Quasi-experiment	15	-females -age (23–45) yrs	25–35	lipo-suction	BMI, leptin, adiponectin, IL6, TNF-a, FBG, insulin, HOMA-IR, TG, FFA	0 1 30	1.88 ± 0.213 L

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Table 1 (*continued*)

Study	Country	Study design	No. of sub-jectss	Population (gender, age, comorbidities)	Baseline BMI ($kg/m^{2)}$	Type of SFR	Outcome measures	Follow-up time points (days)	Average fat mass (kg) or lipoaspirate (L) removed
Klein et al., 2004	USA	Quasi-experiment	15	-females −46% diabetic type 2	nonDM 35.1 ± 2.4, DM 39.9 ± 5.6	lipo-suction	BMI, WC, FM, leptin, adiponectin, IL6, CRP, TNF-a, FBS, insulin, LDL, HDL, TG, TC, SBP, DBP	0 84	17±2
Robles-cervantes et al., 2004	Mexico	Quasi-experiment	15	-females -age (28.8) yrs	26.35	lipo-suction	BMI, FBG, insulin, HOMA-IR, TC	0 21	3.570 ± 1.543 L
Esposito et al., 2004	Italy	Quasi-experiment	45	-females -pre-menopausal	35.1 ± 2.9	lipo-suction	BMI, WC, adiponectin, HOMA-IR, TG, TC	0 90 180	NR
Gonzalez-Ortiz et al., 2002	Mexico	RCT	6	-females -age (20 - 40) yrs	31.7 ± 1.7	lipo-suction	BMI, FBG, insulin, LDL, HDL, TG, TC	0 28	4.308 ± 1.126 L
Chen et al., 2001	China	Case series	4	-females - age (34.0 ± 3.7) yrs.	23.6 - 42.7	lipo-suction	leptin	0 1 2 14	range 1.25 - 12.78 L
Enzi 1979	Italy	Quasi-experiment	14	−12 females - 2 males -age (34 −58) - all are diabetic type 2	34.5	abdomino-plasty	FM, FBG, TG	0 30	6.0 ± 0.5 kg

RCT; randomized controlled trial. *BMI;* body mass index. *FM*; fat mass. *LBM*; lean body mass. *WC;* waist circumference. *TNF-α*; tumor necrosis factor-alpha. *CRP*; C-reactive protein. *IL6*; interleukin 6. *FBG*; fasting blood glucose. *HOMA-IR*; homeostatic model assessment for insulin resistance. *SBP;* systolic blood pressure. *DBP;* diastolic blood pressure. *LDL;* low-density lipoprotein cholesterol. *HDL*; high-density lipoprotein cholesterol. *TC*; total cholesterol. *FFA*; free fatty acids. *L*; liters. *NR*; not reported.

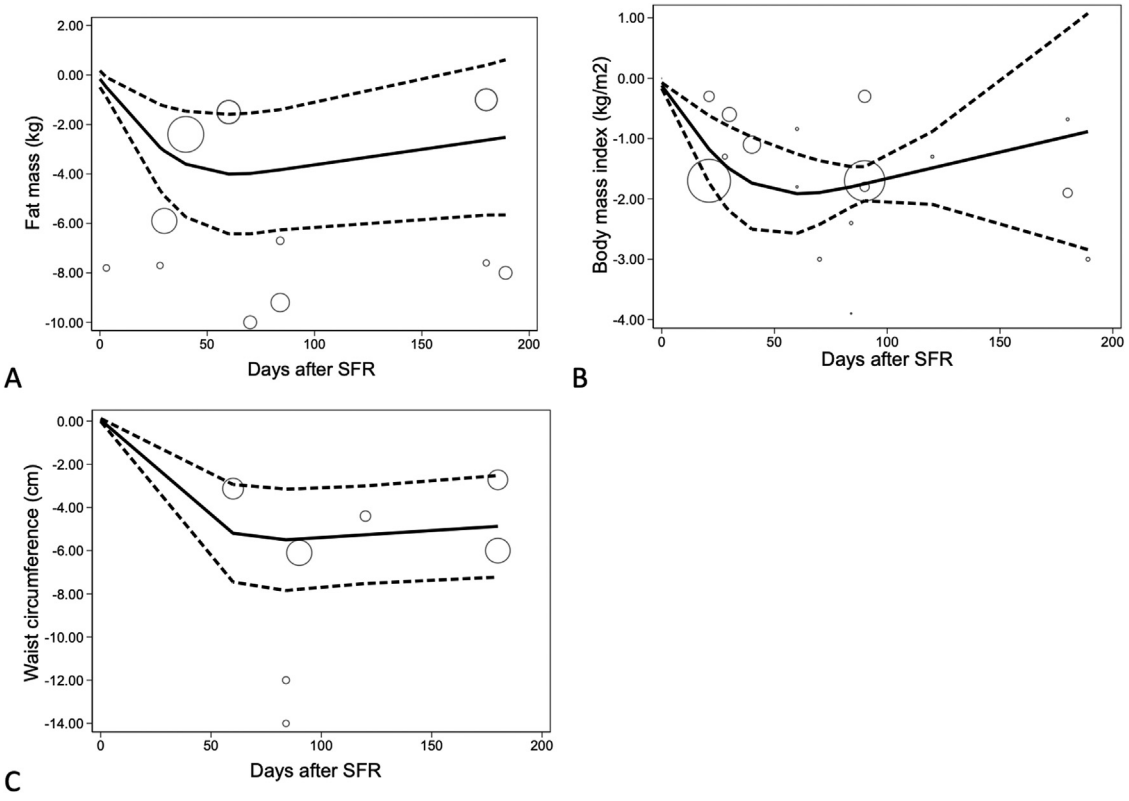

Figure 2 Changes in (A) body mass index, (B) fat mass, and (C) waist circumference over time since surgical fat removal (SFR). The "dose" is time in days after surgical fat removal (SFR). The circles represent the weighted mean difference in each individual study at this time point, with the marker size reflecting the weight of the corresponding study.

ber of participants (sample size), population demographics, preoperative (baseline) BMI, type of SFR (abdominoplasty versus liposuction), follow-up time points after surgery (in days), and FM (in kg) removed in abdominoplasty or lipoaspirate (in liters) (which consists of infiltrated solution plus removed FM) in liposuction procedures.

Metabolic changes after SFR

Anthropometrics/body composition

BMI (kg/m^2), FM (kg), WC (cm), and LBM (kg) were measured. There was significant heterogeneity in BMI and FM changes across studies; however, the DRMA suggested that postsurgical weight reduction was maximal at fifty days (2 BMI units and 3 kg of FM, respectively), after which there was a return toward the average presurgical weight (Figure 2). Because of the paucity of studies, confidence intervals were wide and the trend could not be confirmed more precisely as this was driven by the bigger studies. Nevertheless, the effect of SFR on BMI and related parameters persisted for at least 50 days. The WC showed a clear reduction of around 5 cm after surgery, which was maintained till the end of follow-up. LBM showed no significant change after SFR.

Serum adipokines

Serum leptin (μg/L), adiponectin (μg/ml), and resistin (μg/L) were measured before and after SFR. Leptin exhibited a significant postoperative reduction that peaked at postoperative day 50 (average of 15 μg/L) and returned to preoperative levels by day 180 (Figure 3). The DRMA yielded no significant differences in serum adiponectin and resistin over time.

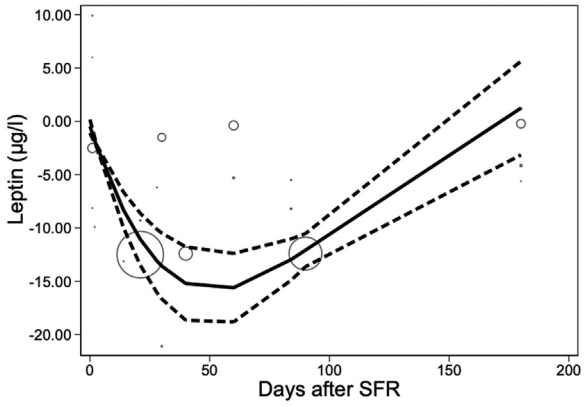

Figure 3 Changes in leptin over time since SFR. The "dose" is time in days after surgical fat removal (SFR). The circles represent the weighted mean difference in each individual study at this time point, with the marker size reflecting the weight of the corresponding study.

Markers of glucose homeostasis

FBG (mmol/L), fasting insulin (pmol/L), and HOMA-IR levels were measured. The DRMA suggested that postsurgical

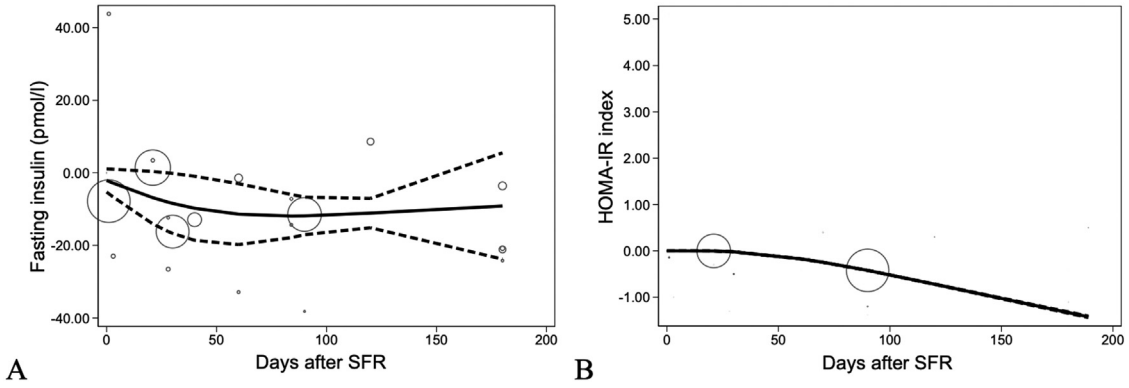

Figure 4 Changes in (A) fasting insulin and (B) homeostasis model assessment for insulin resistance (HOMA-IR) over time since SFR. The "dose" is time in days after surgical fat removal (SFR). The circles represent the weighted mean difference in each individual study at this time point, with the marker size reflecting the weight of the corresponding study.

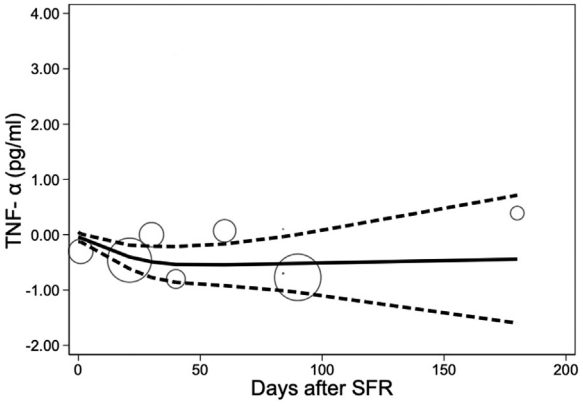

Figure 5 Changes in tumor necrosis factor-alpha (TNF-α) over time since SFR. The "dose" is time in days after surgical fat removal (SFR). The circles represent the weighted mean difference in each individual study at this time point, with the marker size reflecting the weight of the corresponding study.

insulin resistance reduction was a lasting feature of SFR for the duration of the study. Peak reductions were 17 pmol/L and 1 point for fasting insulin and HOMA-IR, respectively. There was no change seen with FBG (Figure 4).

Inflammatory markers

TNF-α (pg/ml), IL-6 (pg/ml), and CRP (mg/L) were measured. While there was substantial heterogeneity across studies, the DRMA suggested that postsurgical reduction in serum TNF-α peaked at day 50 (0.75 pg/ml) and thereafter exhibited a return to presurgical levels (Figure 5). No significant differences were observed in serum levels of IL-6 or CRP over the course of the study.

Lipid profile

LDL cholesterol (mmol/L), HDL cholesterol (mmol/L), serum fasting TGs (mmol/L), TC (mmol/L), and FFA (g/L) were measured. Serum HDL increased post-surgically, peaking at day 50. However, by day 100, expression had returned to the baseline and thereafter continued to fall to the end of the study period at day 180. TC reduced by 0.25 mmol/L post-surgically to day 50; however, the trend thereafter is obscured by wide confidence intervals owing to the paucity of data (Figure 6). No significant differences were observed in serum levels of TG, LDL, and FFA.

Blood pressure

Following SFR, there was a mean reduction in both SBP and DBP of 3.5 mmHg by day 50, which thereafter exhibited a return to presurgical levels at day 180 (Figure 7).

Quality assessment of included studies

Most of the studies were ranked in the 4th quartile of the count of safeguards. Moreover, the most deficient standards across articles were equal ascertainment and equal prognosis. In contrast, equal implementation, equal recruitment, equal retention, sufficient analysis, and temporal precedence were found to be the least deficient standards. See Supplementary Figure 3.

Discussion

We examined the influence of SFR on body anthropometrics/body composition measurements, serum adipokines and inflammatory cytokines, glucose homeostasis, lipid profile, and blood pressure by means of a systematic review of clinical data and subjected these data to DRMA. We observed that SFR resulted in a significant and lasting improvement in insulin resistance as evidenced by serum fasting insulin and HOMA-IR index and transient improvements in BMI, FM, SBP, and DBP and in serum leptin, TNF-α, HDL, and TC concentrations. There were no observable improvements in LBM, serum adiponectin, resistin, IL-6, CRP, LDL, FFA, or FBG.

Weight loss after SFR peaked at day 50 post-surgery, but thereafter weight gain was observed with BMI and FM, returning to near-preoperative levels after a period of 6 months. This might be because of the loss of the negative energy balance after surgery, or increased energy intake, particularly if it was not accompanied by physical exercise after SFR.[9,12] Another possibility is that this return toward baseline has an underlying hormonal basis such as residual

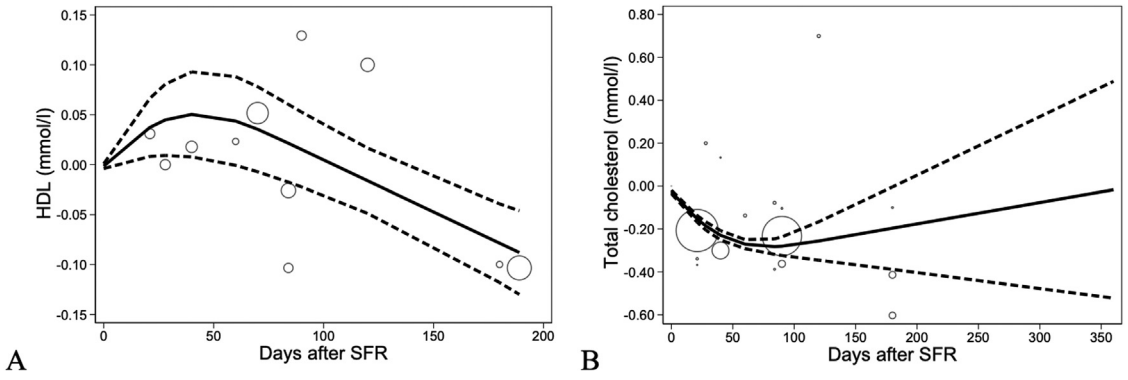

Figure 6 Changes in (A) high-density lipoprotein (HDL) cholesterol and (B) total cholesterol over time since SFR. The circles represent the weighted mean difference in each individual study at this time point, with the marker size reflecting the weight of the corresponding study.

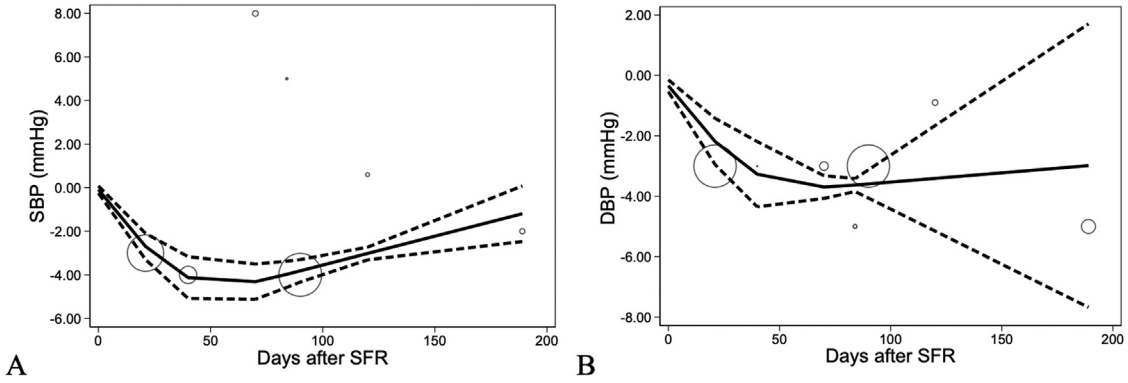

Figure 7 Changes in (A) systolic blood pressure (SBP) and (B) diastolic blood pressure (DBP) over time since SFR. The "dose" is time in days after surgical fat removal (SFR). The circles represent the weighted mean difference in each individual study at this time point, with the marker size reflecting the weight of the corresponding study.

fat cells hypertrophy.[39-41] This has also been noted in similar animal studies, where surgical fat reduction was followed after weeks to months by a compensatory increase in the FM elsewhere.[42,43] Using dual-energy x-ray absorptiometry scans and magnetic resonance imaging, a clinical trial observed compensatory abdominal FM deposition in a 12-month period after thigh liposuction.[44] Other retrospective human studies reported an increase in breast size after abdomen and thigh liposuction surgeries, which was postulated to be due to an altered ratio of androgen-to-estrogen levels,[39-41] but this may not be the only explanation.

Leptin was the only hormone derived from adipose tissue that exhibited an expression pattern altered by SFR. This was similar to the findings of a meta-analysis on the effect of large-volume liposuction on serum leptin and adiponectin levels. In this study, too, leptin, but not adiponectin, was reduced after SFR. Because leptin is secreted mainly from fat cells and correlates with FM,[45] the transient fall in serum leptin levels is understood in the context of the postsurgical reduction in FM. More interestingly, we may speculate that the rebound rise in serum leptin to presurgical levels may involve hypersecretion and/or hypertrophy by the residual FM.[39-41] The physiological adaptation underpinning this phenomenon remains obscure, but the fact that the postsurgical BMI mirrors the postsurgical temporal expression profile of leptin (which governs satiety) suggests a homeostatic mechanism.

There is no clear relationship in the literature between SFR and variations in the expression profiles of the inflammatory cytokines TNF-α and IL-6.[9,11,46] That said, the transient reduction in serum TNF-α identified in this study has been observed before.[46] Compared with leptin, the synthesis of TNF-α occurs mainly in the monocyte lineage.[47] The accumulation of resident macrophages in the adipose tissue correlates with the degree of obesity.[48,49] Animal models suggest that in morbid obesity, macrophages (responsible for most of the overall secretion of TNF-α) may account for up to 40% of the cellular mass of adipose tissue.[50] Our analysis suggests that SFR-mediated removal of the resident macrophages in the adipose tissue results in the initial reduction in TNF-α levels. The underlying mechanisms for the recovery in TNF-α level after the first two months of the SFR remain unclear. However, toll-like receptors-induced synthesis of TNF-α from existing resident macrophages and/or through recruitment of circulating myeloid-derived blood monocytes that give rise to adipose tissue-resident macrophages are potential pathways for TNF-α recovery.

Accumulation of adipose tissue-resident macrophages is facilitated by IL-6 secreted from adipocyte, and obesity is associated with elevated circulating IL-6 levels.[51,52] During the inflammatory phase, macrophages promote the return to homeostasis by removing apoptotic cells and cell debris and contributing to damage repair.[53] Circulating IL-6 plays an important role in mediating inflammation and is a central stimulus for the acute-phase inflammation response.[54] Our analysis found no significant changes in serum IL-6 and CRP (a known marker for acute inflammation), suggesting the absence of systemic inflammatory response after SFR. IL-6 stimulates CRP synthesis in the liver,[55] and, thus, the stable serum CRP level after SFR is consistent with a stable IL-6 level for the same period. Our analysis could not exclude the possibility of an increase in IL-6 levels at the surgery site.

Several syntheses have examined the changes in insulin sensitivity after SFR, and a trend toward improvement in insulin sensitivity has been described without elucidation of the magnitude of effect or clinical significance thereof.[8-11] Moreover, results were inconsistent because of the heterogeneity in the design and analysis of studies. The present synthesis demonstrates a gradual and steady decrease in the fasting insulin, which reaches an average decline of 17 pmol/L by 6 months. The HOMA-IR showed a similar trend with a 1-unit reduction by 6 months. There have so far been reports of more accurate measurements for insulin sensitivity, such as the oral glucose tolerance test.[56] Interestingly, the return of BMI toward baseline after 50 days, as shown above, was not coupled with a similar return in insulin sensitivity toward baseline values. This finding may reflect extra-abdominal postsurgical fat deposition, which might be less harmful.[57]

There is a strong positive relationship between body mass and blood pressure. A reduction in body mass of between 5 and 10% can reduce blood pressure in both hypertensive and normotensive cohorts.[58] Indeed, a reduction of 1 kg of body mass in obese patients results in a sustained decrease of 1.2 mmHg and 1 mmHg in SBP and DBP, respectively.[59] Additionally, chronic hyperleptinemia as seen among the obese population is also correlated with blood pressure.[60] Loss-of-function mutations in leptin and leptin receptors are associated with decreased blood pressure despite severe obesity.[61] The effect of leptin is mediated by the neurons in the dorsomedial hypothalamus. Inhibiting leptin receptor-expressing neuronal activity in the hypothalamus leads to a rapid decrease in blood pressure in obese mice, independent of changes in body mass.[61] In this study, the correlation between postsurgical blood pressure and serum leptin may be understood in these terms.

Subcutaneous FM plays a causative role in obesity-linked dyslipidemia.[62] Thus, SFR may have a positive effect on lipid profile,[38] particularly in the absence of morbid obesity.[63] However, the present study failed to demonstrate a clinically significant clear correlation between SFR and postsurgical lipid profile.

Several small and heterogeneous studies have measured changes in body composition, adipokines, and inflammatory marker,[17-38] and have been followed by systematic reviews and meta-analyses in an attempt to examine the effect of SFR on body metabolism. Only one synthesis looked at these changes in terms of time since surgery,[12] but even then, the latter study only reported the differences in physical biometrics such as body weight and FM. The remaining syntheses combined several heterogeneous studies with different follow-up durations,[8-11] resulting in contradicting and unclear conclusions regarding the metabolic benefit or harm of SFR. The ideal approach to the synthesis of the existing body of evidence required a DRMA because this is the only way to reduce the existing clinical heterogeneity.

A major limitation of this study is the small number of eligible studies, many of which had recruited a small number of patients. Thus, when the margin for error was taken into consideration, few obvious trends emerged. While we considered the inclusion of different types of SFR to be a strength of our meta-analysis, it is possible that the technical differences of each approach bequeathed unique and dissimilar physiological legacies on the patient that manifest as different changes in postsurgical metabolic parameters. For example, abdominoplasty surgery for obesity or weight loss often includes, as an operative step, correction of divarication of the rectus muscles. This, in turn, results in an increase in the abdominal pressure, myocardial preload, and compresses visceral fat.[64] It is clear from the synthesis that metabolic changes after SFR need further study in a well-designed prospective design, and this in turn will help us not only to identify the changes and the safety of these procedures but also broaden our knowledge about the metabolic effects of obesity.

Conclusion

This study shows that body contouring surgery correlates with enhanced insulin sensitivity for at least 6 months after surgery. Transient benefits were observed in BMI, blood pressure, serum leptin, and TNF-α. An evaluation of the metabolic benefits of body contouring surgery beyond 6 months is hampered by lack of data.

Conflict of interest

The authors declare no competing interests.

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Category

Meta-analysis.

Declaration

This article has not previously been presented at any national or international meeting.

Ethical Approval

Not required.

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.bjps.2022.10.055.

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